A Retrospective Study on Coronary Artery Disease Patients to Calculate the Number of Vessel Blockage

N.S.Harini[1], Naufal Rizwan[2], Sankari M[3]

Graduate Student[1], Associate Professor[2], Department of General Medicine,

Professor[3], Department of Periodontics.

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: Coronary artery disease plays major role in the European and developing countries. Indian prevalence also shows a high risk of coronary artery disease. Change in the sedentary life style, unhealthy diet, stress etc. leads to high cholesterol levels with added risk factors such as smoking, diabetes mellitus thus making the individuals more prone for coronary artery disease. With the current scenario change in your working habits improper eating habits, decreased physical inactivity, stressful lifestyle have led to early evolution of coronary artery disease. Thus the aim of this article was to retrospectively analyze 50 CAD patients to reassess the risk factors for its development and to calculate the number of vessels involved in blockage through echo cardiogram.

Materials and Methods: The clinical data of 50 CAD Patients who underwent treatment at Saveetha Medical College was used to derive at the number of vessel blockage with gender, age, diabetes, obesity and habit of smoking through Echo Cardiogram.

Results:Our study reveals that out of 50 CAD patients with 31 men and 19 women. 25 patients are affected with single vessel disease, 9 patients are affected with double vessel disease, and 16 patients are affected with triple vessel disease. Smokers are the most affected with 22 in numbers amongst the male. The age group spread across 35 to 60 years(13). In total 45 patients are with diabetics

Conclusion: Cardiovascular diseases and CAD remain the leading cause of mortality in the world. Thus, preventing ourselves from such diseases rather than treating the deadly ones is much better and we have to join hands in abolishing such emerging deadly diseases in the future.

Key Words: *Coronary Artery Disease, atherosclerotic plaque, thrombus, Smoking, , Diabetes Mellitus, Hypertension, Obesity, , Stress, Genetics.*

Introduction

Indian youngsters are on the high risk of Coronary Artery Disease according to various research and articles published in the recent times which was mainly due to factors of Smoking(11), high cholesterol levels, Diabetes Mellitus, Hypertension(16), Obesity, Unhealthy food habits, Physical inactivity(17), Stress, Genetics.(1) This has driven me to choose a study on the above CAD to evaluate the major contributors which can be an eye opener and also help to create an awareness to the prospective CAD patients, which will help them to change their life style to avoid major threat to their life. This article highlights the significance of the Coronary artery disease where we have chosen 50 CAD patients to calculate the number of vessels involved with the aid of Echo Cardiogram.

CAD refers to disease of the coronary arteries. Coronary arteries carry blood and oxygen to the heart; therefore any disease which affects the coronary arteries deprives the heart of oxygen — the extreme form of this deprivation occurs during myocardial infarction (heart attack)(9).Built of fatty material is called as atherosclerotic plaque which transforms in to thrombus which stays in the arteries and blocks the movement of blood platelets (2). The precious asset of our nation is its young population; ironically, it is becoming the most vulnerable to CAD and myocardial infarction (MI).

Four people die of heart attack every minute in India and the age group is mainly between 30 and 50.Twenty-five per cent of heart attack deaths occur in people less than 40.Nine hundred people under 30 die due to heart disease in India every day (2).Lack of awarenessin rural and urban poor are more susceptible because they tend to ignore the disease due to poor access to health care, high cost of treatment, social stigma and illiteracy(6).

Seeking treatment will also mean missing wages and reduced productivity.The statistics in our country may represent only the tip of the iceberg. Because of poor reporting of cases and incomplete collection of statistical data, the actual figures for young Indians suffering from coronary artery disease may be much more than that reported in studies. Several lines of evidenced suggests that clinical depression may be a risk factor for coronary artery disease (12).

Salient Features of the Coronary Artery Disease (CAD) Epidemic in India

- India topped the world with 1 531 534 cardiovascular disease-related deaths in the

year 2002

- Median age of first heart attack in Indians is 53 years.
- Incidence of CAD in young Indians is about 12%–16%, which is higher than any other ethnic group(8).
- About 5%–10% of heart attacks occur in Indian men and women younger than 40 years
- Age-standardized estimates for disability-adjusted life-years lost due to CAD per 1000 population in India are three times higher than in developed countries(1).
- Depression has been found to be an important cause for CAD among women(14).
- The chances of CAD due to genetic is estimated at 50 – 60%(15)
- Dyslipidemia is one of the major contributor in CAD(18)

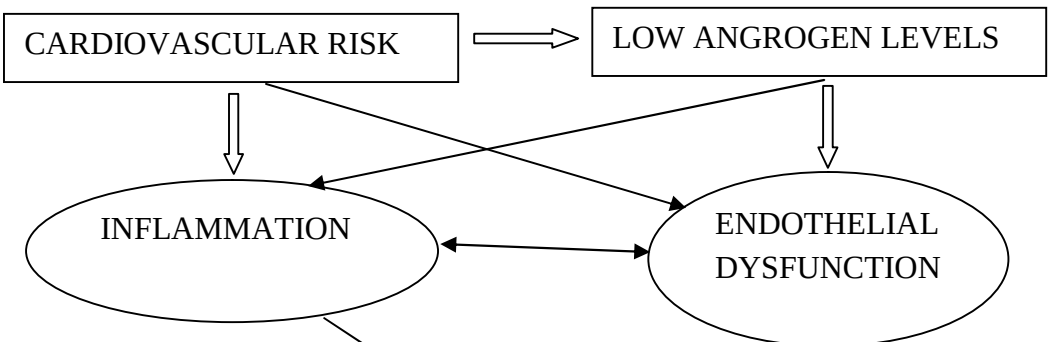
Materials and Methods

The clinical data of 50 CAD Patients who underwent treatment at Saveetha Medical College was used to derive at the number of vessel blockage with gender, age, diabetes, obesity and habit of smoking through Echo Cardiogram.

The clinical data of 50 CAD patients were taken for this research which comprises patients with background of Smoking, high cholesterol levels, Diabetes Mellitus, Hypertension, Obesity, Unhealthy food habits, Physical inactivity, Stress, Genetics in the age group of 35 – 60 years of both genders. These patients were diagnosed from SaveethaMedical College and Hospitals.

The patients were grouped based on demographic data of age, gender, men with habit of smoking, diabetes, obesity and age group. The vessel blockage identification was done through Echo Cardiogram. Obesity was calculated using body mass index(BMI), the age and gender were estimated through graph and pie chart estimation. The smoking habit and diabetes mellitus risk factors were estimated through pie charts.

Pathophysiology



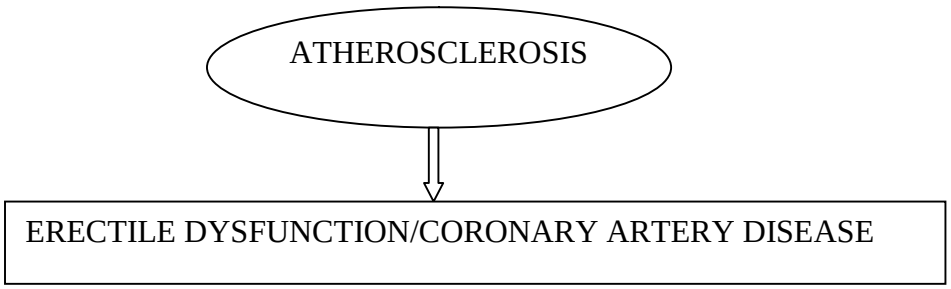


Figure 1: Pathophysiology of Coronary Artery Disease

Pathogenesis:

ENDOTHELIUM

⇓

BACTERIAL PRODUCTS

⇓

RISK FACTORS

⇓

PRO INFLAMMATORY PRODUCTS

⇓

ADHESION

⇓

CHEMO INFLAMATORY CYTOKININS

⇓

ARTERIAL INTIMA

⇓

INFLAMMATORY REACTIONS

⇓

Figure 2: Pathophysiology of Atherosclerosis

The heart muscle relies on a steady flow of oxygen-rich blood to nourish it and keep it pumping. During a heart attack, that blood flow is interrupted by a blockage in an artery. Without blood, the area of heart fed by the affected artery begins to die and scar tissue forms in the area.(43) Atherosclerosis is an immuno-inflammatory disease which occurs mainly in the large and medium sized arteries. Endothelial injury is the prime step in the formation of atheroma. After endothelial injury, the surface adhesion molecules are increased and it leads to monocyte adhesion by the expression of monocyte chemoattractant protein-1 (MCP-1).These monocytes are converted into tissue macrophages in the intima layer. The activated macrophages secrete various cytokines. Cytokines in turn stimulate the endothelial cells. Activated macrophages also have the property of capturing low density lipoproteins which undergo progressive oxidation and are no longer available for β oxidation of fatty acids to produce energy. Subsequently, there is a formation of lipid peroxidises and accumulation of cholesterol esters which leads to the formation of foam cells. Further accumulation of these cells by modified LDL leads to the formation of fatty streak which gets calcified to form atherosclerotic plaque. The features of atherosclerotic plaque include a lipid rich core with common focal calcification detected by the coronary calcium score, smooth muscle necrosis, neovascularisation and intra-plaque haemorrhage, vascular remodelling and luminal stenosis(39).

Signs and Symptoms

Chest pain that occurs regularly with activity, after eating, or at other predictable times is termed stable angina and is associated with narrowing of the arteries of the heart. Angina that changes in intensity, character or frequency is termed unstable. Unstable angina may precede myocardial infarction. In adults who go to the emergency department with an unclear cause of pain, about 30% have pain due to coronary artery disease. Cohort studies have shown an increased risk of cerebrovascular disease (stroke) in individuals with signs and symptoms of CAD compared with non affected individuals. Symptoms and signs of cerebrovascular

disease, particularly transient ischemic attack, carotid bruit, and completed stroke also identify individuals with a greater-than-normal likelihood of developing a coronary event(3).

Risk Factors

Coronary artery disease has a number of well determined risk factors. The most common risk factors include smoking, family history, hypertension, obesity, diabetes, lack of exercise, stress, and high blood lipids.Smoking is associated with about 36% of cases and obesity 20%.Lack of exercise has been linked to 7–12% of cases.Exposure to the herbicide Agent orange may increase risk.Both rheumatoid arthritis and systemic lupus erythematous are independent risk factors as well. A plethora of risk factors can predispose to IHD in women, ranging from Diabetes and Hypertension to Dyslipidemia to smoking and Obesity and the Stress of a multi-faceted and simultaneous multitasking personality, typical of a woman(41).

Some of the risk factors of CAD are mentioned below:

1)Cholesterol

2)Hypertension

3)Cigarette smoking

4)Family history

5) Diabetes mellitus where Diabetes imposes lifelong threats on the individuals and their families(40).

Results

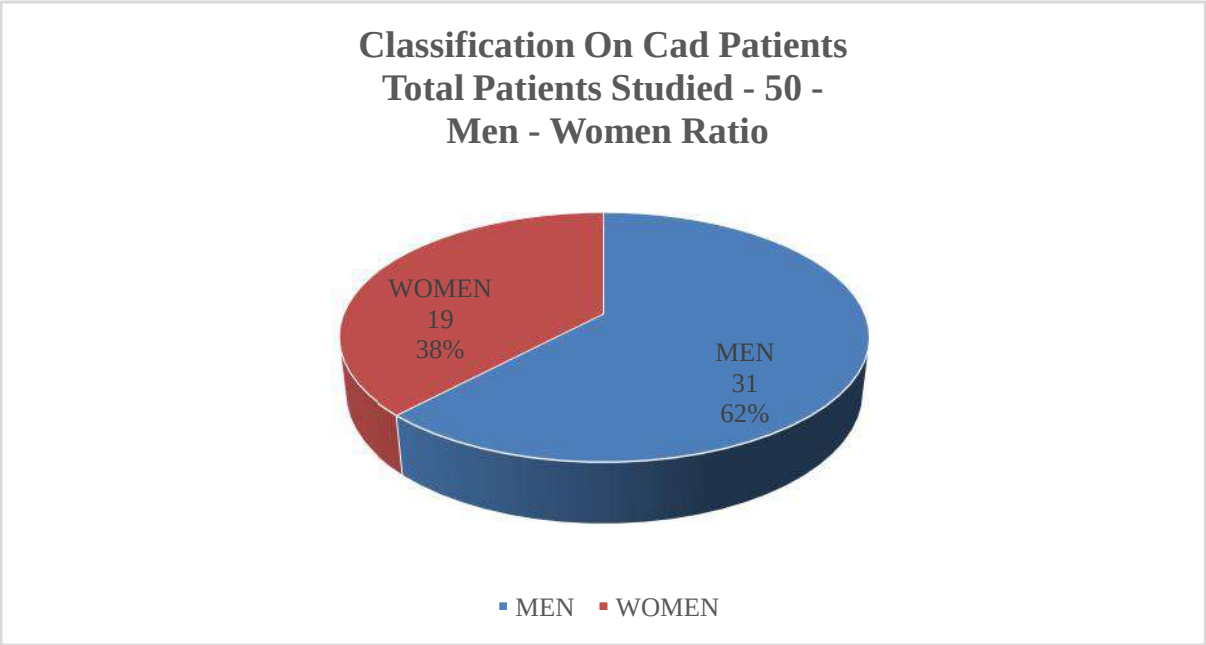


Figure 3: Pie Chart Showing Classification of Cad Patients Based on Men Women Ratio

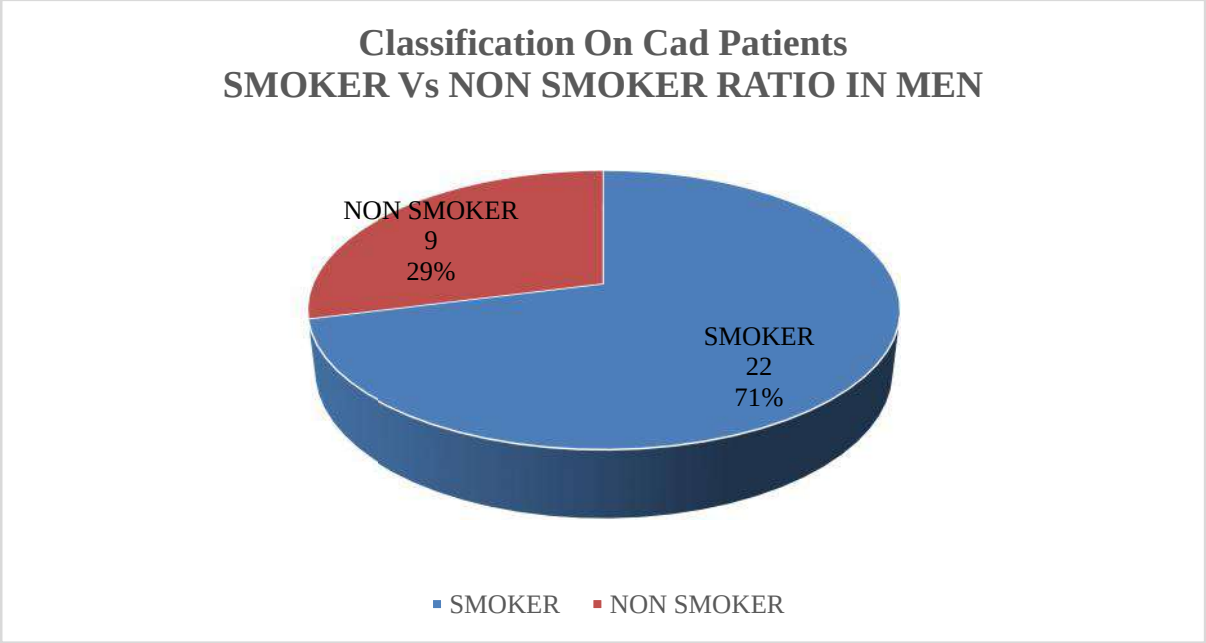


Figure 4: Pie Chart Showing Classification Of Cad Patients Based On Smoking Habits

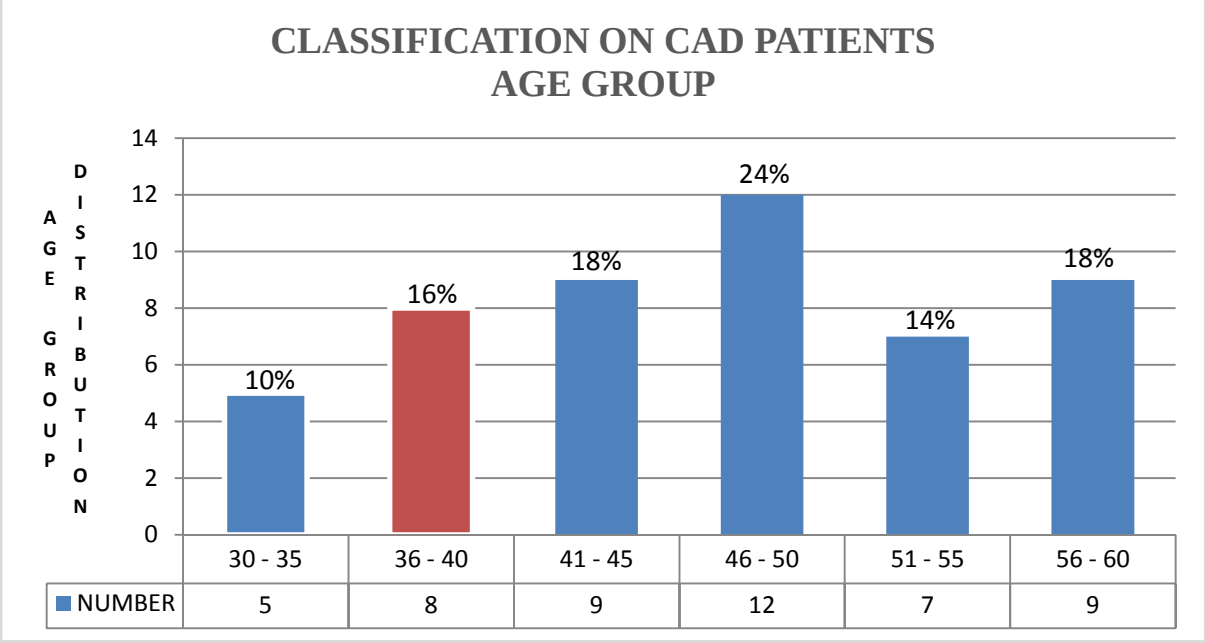


Figure 5: Bar Graph Showing Demographic Data among Cad Patients

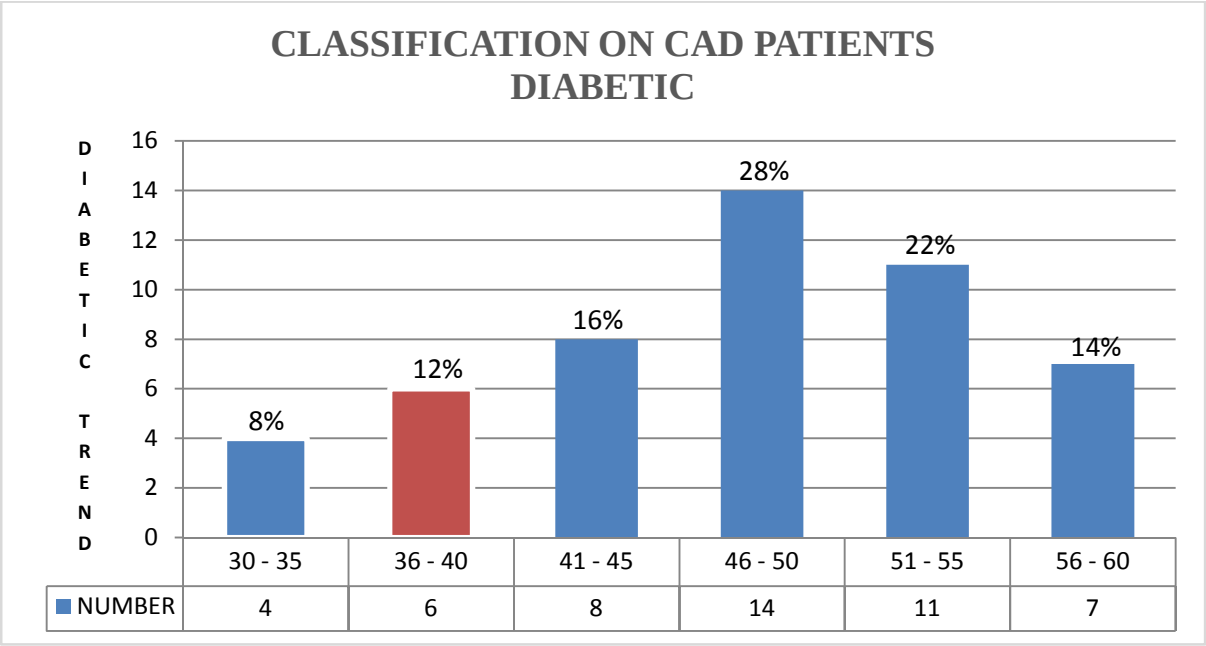


Figure 6: Bar Graph Showing the Diabetes Level among CAD Patients

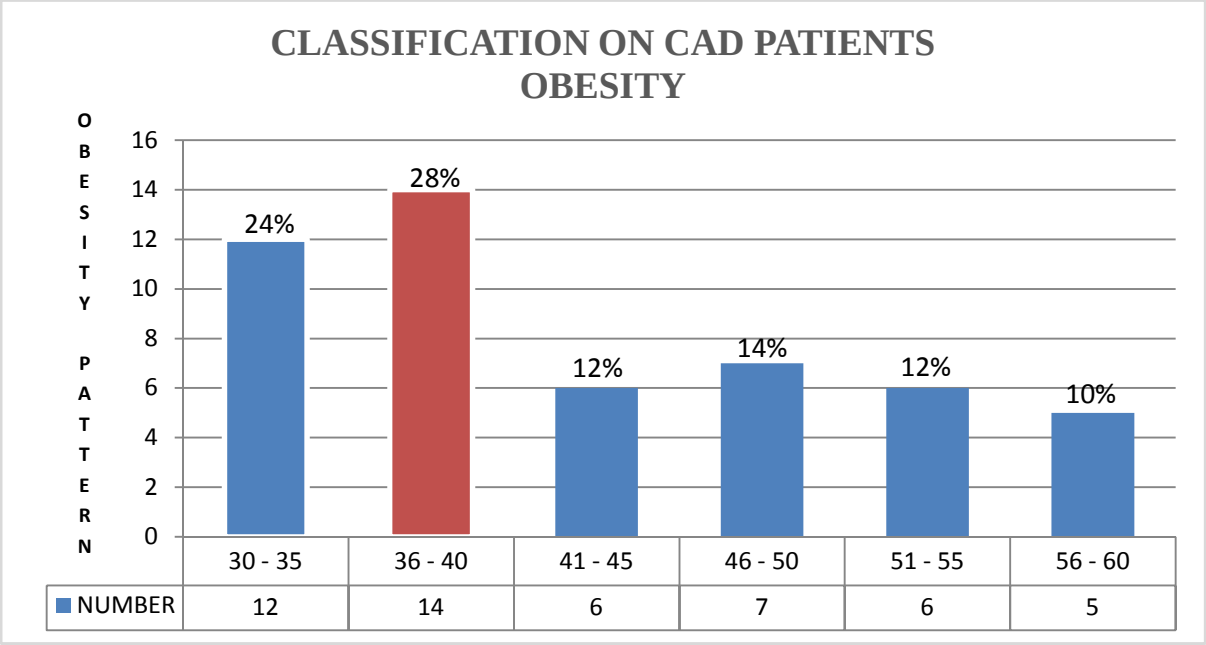


Figure 7: Bar Graph Showing Obesity Status among Cad Patient

Discussion

CAD is a disease which is most common worldwide,with high mortality rate. This mortality rate depends upon the number of vessels involved. Various studiesreveal that single vessel disease has the lowest mortality rate, and triple vessel disease has the highest mortality

rate. Coronary heart disease is the leading cause of death in the US general population. With the obesity pandemic and the expected worsening of cardiovascular risk factors in the general population, the incidence and the prevalence of heart disease is expected to rise. Coronary artery disease (CAD) is the leading cause of death in patients with chronic kidney disease (CKD). Of the more than 320,000 patients with ESRD that requires dialysis or kidney transplantation in the United States, half will die from cardiovascular causes, and patients with milder degrees of CKD are more likely to die of CAD than to develop kidney failure that requires renal replacement therapy (20).

The incidence of coronary artery disease (CAD) is apparent from community surveys. The Bromley Coronary Registry is a contemporary community registry used to identify all symptomatic medical presentations of CAD in one population (21). All incident (first) presentations of exertional angina, acute coronary syndromes, and sudden cardiac death were recorded for the Bromley Health Authority in South East London (population 186 053, in men and women aged 25–74 years) for the period from 1996 to 1998. In the ARIC study in participants aged from 45 to 64 years, the average age-adjusted CAD incidence rates per 1000 person-years were 12.5 in white men and 10.6 in black men.(22)

According to AHA Heart Disease and Stroke statistics, it is estimated that 770 000 Americans had a new coronary attack in 2008, and 430 000 had a recurrent attack. It is estimated that 190 000 additional silent first acute myocardial infarctions (MIs) occur each year. Approximately every 26 s, an American will have a coronary event, and about every minute someone will die of one.(23)

There are marked variations in the epidemic of CAD among regions of the world, nations, and even between regions within a country (24).The age-standardized death rates from CAD are declining in many developed countries, but are increasing in developing and transitional countries, partly as a result of demographic changes, urbanization, and lifestyle changes. Nowadays ~3.8 million men and 3.4 million women worldwide die each year from CAD.(25) According to the Global Burden of Disease Study,(26) the developing countries contributed 3.5 million of the total number of 6.2 million deaths from CAD in 1990. The projections estimate that these countries will account for 7.8 million of the 11.1 million deaths due to CAD in 2020. According to global and regional projections of mortality and burden of disease, CAD will remain the leading cause of death for the next 20 years.(27)

In the USA and in most countries in the European Union, the age-standardized CAD mortality rates have decreased significantly. This may lead paradoxically to an increase in the prevalence of CAD; indeed a better survival of CAD patients and demographic changes result in more elderly people suffering from CAD. Today CAD is the most important major killer of both American men and women, causing approximately one of every five deaths in the USA in 2005.(28) Approximately 37% of the people who have a coronary event in a given year will die of it. In 2005 the overall CAD death rate was 144.4 per 100 000 population.

The death rates were 187.7 for white males and 213.9 for black males; for white females the rate was 110.0 and for black females 140.99. In the European Union, CAD is also the single most common cause of death. One in five to one in seven women die of CAD; in men CAD accounts for one in four to one in six of all deaths. Age-standardized and gender-specific CAD mortality rates have significantly decreased during recent decades in many countries in the north, west and south of Europe.

However, the decline was less apparent or absent in central and eastern Europe. Thus, the Russian Federation, Belarus, Ukraine, and Central Asian republics show the highest CAD mortality rates ever seen, significantly higher even than recognized peaks in the USA, Australia, New Zealand, Finland, and Scotland (29,30). Furthermore, population aging represents a major challenge. Thus, even if age-specific mortality rates continue to decline, the absolute number of cardiovascular disease (CVD) deaths will increase. Predictions up to 2030 suggest that even with an annual decline in mortality rates of about 1%, the absolute number of deaths will increase, attributable solely to population aging (27).

It has been estimated that 30–43% of patients who were asymptomatic after an MI had silent myocardial ischaemia in the initial 30-day period after the infarct, based on stress test data or Holter monitoring.(31,32). A recently published analysis in 937 outpatients with stable CAD from the Heart and Soul Study demonstrated that 14% of outpatients had angina alone, 20% had inducible ischaemia alone, and 4% had both angina and ischaemia. Recurrent CAD events occurred in 7% of participants without angina or inducible ischaemia, 10% of those with angina alone, 21% of those with inducible ischaemia alone, and 23% of those with both angina and inducible ischaemia.(33)

Coronary atherosclerosis most commonly presents in the community as angina pectoris, followed by acute coronary syndromes (MI and unstable angina), and finally as sudden

cardiac death. When the acute manifestations of CAD—sudden cardiac death and MI—are considered together, one in two patients with new or recurrent disease die within 30 days of their acute clinical presentation.(34-37) . About 69% die in the community, 29% die in hospital, and the other 2% die within 30 days of discharge. Congestive heart failure (CHF) is the result of the heart's inability to function effectively as a pump.(42)

The prognosis of patients with chronic CAD is not uniform. It depends on several factors, including the underlying coronary anatomy, left ventricular (LV) function, and comorbidities. The data concerning prognosis in clinical trials are of limited value due to the highly selective nature of populations included in such studies. Large population-based studies could help to increase our understanding of the differences in prognosis between CAD manifestations.

The recently published data on cardiovascular event rates from the REACH (Reduction of Atherothrombosis for Continued Health) registry in stable CAD outpatients (n = 38 602 patients) has confirmed that patients with established stable CAD had the highest non-fatal MI rate and the highest non-fatal stroke rate. The registry reported annual event rates of 15.2% for death, stroke, MI, or hospitalization for an atherothrombotic event, 6.4% for unstable angina, 4.5% for death, acute MI, and stroke, and 3.8% for revascularization by percutaneous coronary intervention (PCI).(38)

Thus, ~3 of 20 patients with established CAD had a major event or had been hospitalized within a year of follow-up. The high event rates observed in the subgroup of patients with established CAD in this large contemporary cohort indicate that continued efforts are needed to improve secondary prevention and clinical outcomes.

Our study reveals that out of 50 CAD patients 25 patients are affected with single vessel disease,, 9 patients are affected with double vessel disease,, and 16 patients are affected with triple vessel disease. The annual mortality rate of single vessel disease is less than 4%. The mortality rate of double vessel disease is 7-10%. The mortality rate of triple vessel disease is 10-12%.

The study results shows that diabetic and obesity amongst the young population as per the trend charts indicates the increase in the CAD. The chart represent the vessel blockage categorizing them in to single, double and triple where single blockage being a major one followed by triple. Gender wise, smokers and non-smokers among the male patients in

numbers and percentage are also represented through pie charts.

From the research study based on the clinical data provided it is evident that the current day life style poses a major threat to the young population being the victim of CAD. To emphasize for a healthy life, counselling on the life style, food habits, the need for exercises are to be carried out in preventing the young victims to CAD.

Conclusion

Cardiovascular diseases and CAD remain the leading cause of mortality in the world. In addition to the lifestyle and pharmacological approaches that are available to prevent and treat CAD, MI is emerging as a potential risk factor for CAD-related outcomes. Ongoing randomized trials are assessing the role of selective HR reduction in improving outcomes in patients with CVDs. Thus, preventing ourselves from such diseases rather than treating the deadly ones is much better and we have to join hands in abolishing such emerging deadly diseases in the future.

Corresponding Author

M.Sankari,

Professor, Department of Periodontics,

Saveetha Dental College,Saveetha University, Saveetha Institute of Medical and Technical Sciences,

162, Poonamallee High Road, Chennai-600077, Tamil Nadu,India.

Phone Number: 9840285905

Email Id: msankari@gmail.com

References

1. Dr. A. Kader Sahib Ashraf; Wanner C, Krane V, Marz W, Olschewski M, Mann JF, Ruf G, Ritz E; German Diabetes and Dialysis Study Investigators: Atorvastatin in patients with type 2 diabetes mellitus undergoing hemodialysis.N Engl J Med 353: 238 –248, 2005
2. Gorank,Hansson,M.D.,PhD;Mechanism of coronary artery disease: inflammation, atherosclerosis, and coronary artery disease;The New England Journal of Medicine, 2005;352:1685-95.
3. Peter Libby, MD; Pierre Theroux, MD;Basic science for clinicians; pathophysiology ofcoronary artery disease;Circulation,June28,2005; 111:3481-3488.
4. Timothy E. Craven, MSPH, Jacqueline E. Ryu, PhD, Mark A. Espeland, PhD, Frederic R. Kahl, MD, William M. McKinney, MD, James F. Toole, MD, Mary R. McMahan, PhD, Corleen J. Thompson, MSPH, Gerardo Heiss, MD, PhD, and John R. Crouse III, MD; Evaluation of the Associations Between Carotid Artery Atherosclerosis and Coronary Artery Stenosis; A Case-Control Study;; (Circulation 1990;82:1230-1242).
5. Joseph A. Vita, MD, Charles B. Treasure, MD, Elizabeth G. Nabel, MD, James M. McLenachan, MD, R. David Fish, MD, Alan C. Yeung, MD, Vladimir I. Vekshtein, MD, Andrew P. Selwyn, MD, and Peter Ganz, MD;Coronary Vasomotor Response to Acetylcholine Relates to Risk Factors for Coronary Artery Disease; Circulation Vol 81, No 2, February 1990.
6. Jassim Al Suwaidi, MB, ChB; Shuichi Hamasaki, MD; Stuart T. Higano, MD; Rick A. Nishimura, MD; David R. Holmes, Jr, MD; Amir Lerman, MD;Long-Term Follow-Up of Patients with Mild Coronary Artery Disease and Endothelial Dysfunction Endothelial Function and non-obstructive Arteries; Circulation March 7, 2000.
7. Robert 0. Bonow, MD; VaskenDilsizian, MD; Alberto Cuocolo, MD; and Stephen L. Bacharach, PhD;Identification of Viable Myocardium in Patients with Chronic Coronary Artery Disease and Left Ventricular Dysfunction Comparison of Thallium Scintigraphy with Reinjection and PET Imaging With '8F-Fluorodeoxyglucose; (Circulation 1991;83:26-37).
8. Peter Libby, MD; Paul M. Ridker, MD; Attilio Maseri, MD;Inflammation and AtherosclerosisCirculation March 5, 2002; 98(7);45-48.

9. Janet M. Torpy, MD; Alison E. Burke, MA; Richard M. Glass, MD; Coronary heart disease risk factors and its complications; JAMA. 2009;302(21):2388. doi:10.1001/jama.302.21.2388.
10. Nainar, Madhu Sankar, SallaSweta Ramani, Rajaram Anantharaman; Coronary artery disease in women; Indian Heart Journal 69 (2017) 532–538.
11. Robert A. Harrington, M.D; Targeting Inflammation in Coronary Artery Disease. N Engl J Med 2017; 377:1197-1198;September -21, 2017DOI: 10.1056/NEJMe1709904.
12. Danier E Ford MD,MPH; Lucy A Mead, SCM; Patricia P. Chang, MD; Lisa Cooper-Patrick, MD,MPH; Nac-Yuh Wang, MS; Michael J. Klag,MD,MPH – Depression is a risk factor for coronary artery disease in Men Arch Intern Med/Vol 158 ,July 13, 1998.
13. Meenakshi Sharma and Nirmal Kumar Ganguly – Premature Coronary Artery Disease in Indians and its associated risk factors – Vascular Heath and Risk Management – 2005 Sep; 99:(6); 67-72.
14. Fahad Aziz, Penn State Hershey Medical Center; Coronary Artery Disease in Women: An unsolved dilemma – Journal of Clinical Medical Research 2014 Apr, 6(2); 86-90
15. Xuming Dai, Szymon Wiernek, James P Evans and Marshall S Runge; Genetics of Coronary artery disease; 2009; 34(8);54-60.
16. Graham I, Atar D, Borch-Johnsen K et al; European guidelines on cardiovascular disease prevention in clinical practice:executive summary.Eur Heart J 2007;28:2375–2414
17. Manisha Nair, Dorairaj Prabhakaran; Why do south Asians have high risk for CAD; Global Heart Vol 7, Issue 4, Dec 2012 Pages 307-314.
18. V Mohan, K Gokulakrishnan, R Deepa, S C Shanthirani and M. Datta; Association of Physical Inactivity with components of metabolic syndrome and coronary artery disease – the Chennai Urban Population study (CUPS no.15) DiabeticMedicine Vol 22; Issue 9 Sept 2005; Pages 1206-1211.
19. Cyrill James – Department of Cardiology , Lourdes heart Institute and Neuro Center, Cochin – Risk factors for coronary artery diseases; a study among patients with Ischemic heart disease in Kerala; Heart India –Vol 1, Issue 1, year 2013 pages 7-11.

20. McCullough PA, Soman S;Cardiovascular calcification in patients with chronic renal failure: Are we on target with this risk factor?Kidney Int 66[Suppl 90]: S18 –S24, (2004); 76:22; 108-115.
21. Sutcliffe SJ, Fox KF, Wood DA, Sutcliffe A, Stock K, Wright M, Akhras F, Langford E;Incidence of coronary heart disease in a health authority in London: review of a community register.Br Med J 2003;326:20;182-87.
22. Jones DW, Chambless LE, Folsom AR, Heiss G, Hutchinson RG, Sharrett AR, Szklo M, Taylor H Jr; Risk factors for coronary heart disease in African Americans: the Atherosclerotic Risk in Communities Study,1987–1997. Arch Intern Med 2002;162:2565–2571.
23. Chobanian AV, Bakris GL, Black HR et al; Seventh report of the joint national committee on prevention, detection, evaluation, and treatment of high blood pressure. Hypertension2003;42:1206–1252
24. Allender S, Peto V, Scarborough P, Boxer A, Rayner M; Coronary Heart Disease Statistics.London: British Heart Foundation; (2007):466:28;33-37.
25. Leal J, Luengo-Ferna´ndez R, Gray A, Petersen S, Rayner M; Economic burden of cardiovascular diseases in the enlarged European Union.Eur Heart J 2006;27:1610–1619.
26. Murray CJL, Lopez AD;The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Disease, Injuries and Risk Factors in 1990 and Projected to 2020.Boston, USA: Harvard University Press;1996; 2:56:1–52.
27. Fraker TD, Fihn SD; Chronic angina focused update of the ACC/AHA 2002 guidelines for the management of patients with chronic stable angina. A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines writing group to develop the focused update of the 2002 guidelines for the management of patients with chronic stable angina.J Am Coll Cardiol 2007;23:2264–2274.
28. Heron MP, Hoyert DL, Xu J, Scott C, Tejada-Vera B. Deaths: preliminary data for 2006. Natl Vital Stat Rep 2008;56:1–52.
29. Rea TD, Pearce RM, Raghunathan TE;Incidence of out-of-hospital cardiac arrest.Am J Cardiol 2004;93:1455–1460.
30. Allender S, Peto V, Scarborough et al.; Coronary Heart Disease Statistics: 2007 edition. London: British Heart Foundation;2007;89(5);56-67.

31. Greenland P, Gidding SS, Tracy RP;Commentary: lifelong prevention of atherosclerosis: the critical importance of major risk factor exposures.Int J Epidemiol 2002;31:1129–1134.
32. Cohn PF, Fox KM;Silent myocardial ischemia. Circulation 2003;108: 1263–1277.
33. Gehi AK, Ali S, Na B, Schiller NB, Whooley MA; Inducible ischemia and the risk of recurrent cardiovascular events in outpatients with stable coronary heart disease.The Heart and Soul Study. Arch Intern Med 2008;168:1423–1428.
34. Norris RM, on behalf of the United Kingdom Heart Attack Study Collaborative Group. Fatality outside hospital from acute coronary events in three British health districts,1994–95. Br Med J 1998;316: 1065–1070.
35. Norris RM;Sudden Cardiac Death and Acute Myocardial Infarction in Three British Health Districts: The UK Heart Attack Study.London: British Heart Foundation; 1999. p61–64.
36. Tunstall-Pedoe H, Kuulasmaa K, Amouyel P, Arveiler D, Rajakangas AM, Pajak A;Myocardial infarction and coronary deaths in the World Health Organization MONICA Project. Registration procedures, event rates, and case-fatality rates in 38 populations from 21 countries in four continents.Circulation 1994;90:583–612.
37. Volmink JA, Newton JN, Hicks NR, Sleight P, Fowler GH, Neil HA;Coronary event and case fatality rates in an English population: results of the Oxford myocardial infarction incidence study.The Oxford Myocardial Infarction Incidence Study Group. Heart 1998;80:40–44.
38. Steg PG, Bhatt DL, Wilson PW; One-year cardiovascular event rates in outpatients with atherothrombosis.J Am Med Assoc 2007; 297:1197–1206.

39. Kritika Jangid, Jayakumar Nadathur Doraiswamy, Sheeja Varghese and Sankari Malaiappan; Periodontitis as a Risk Factor for Cardiovascular Diseases- Hype or a Fact?Journal of Cardiovascular Disease Research; Jan-Mar 2015; Vol 6 ● Issue 1; 24-27.
40. Dr.S.Aruna, M. Anagha;Assess the knowledge on insulin therapy among patients with diabetes mellitus; Int J Pharm Bio Sci 2017 Apr; Special issue SP - 01 “Exhilaration of Nursing Sorority: Maneuvering the Quality” 2005; 76(8)32-53.
41. Meenakshi Kadiyala; Ischemic Heart Disease in Women-changing perspective; Journal of Heart Lung and Circulation 2017; Vol.1 No.1:e105;p:1-2.

42. Sharmraaj Subramaniam, Prasanna Neelakantan; Local anesthesia in dentistry - Clinical Considerations; Int. J. Drug Dev. and Res., October -December 2013, 5 (4): 30-36.
43. Sanchana and Sai vignesh; Stem Cell Therapy for Heart; International Journal of Pharma and Bio Sciences; 27th april 2013; ISSN 0975 – 6299;p:52.

A Study Of 50 Patients Presenting With Stroke To Assess Whether Hypertension Is A Risk Factor

Shasmitha.R[1], Naufal Rizwan [2], Madhusudhan Vasantharajan[3]
Graduate Student [1],Senior Lecturer [2],Department of General Medicine,
Senior Lecturer [3], Department of Pedodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: Stroke occurs when the supply of blood to the brain is either interrupted or reduced. When this happens, the brain does not get enough oxygen or nutrients which cause brain cells to die. High blood pressure is a risk factor for stroke recurrence. Mean Arterial pressure is a stronger predictor of cerebrovascular events than systolic or diastolic blood pressure. It is well known that patients with acute stroke, even without the history of previous hypertension, often have a very high blood pressure on arrival at the hospital. Mechanisms and effects of the BP elevation are unclear. On the other hand, high blood pressure may increase edema in areas surrounding a cerebral infarction or haemorrhage. In patients with acute stroke and very high BP, their blood pressure reduction should be gradual in order to preserve cerebral auto regulation to prevent further extension of the injury. So a study of 50 stroke patients and their blood pressure control is observed. The aim of the study is to assess whether hypertension is a risk factor of stroke.

Material and Method:50patients admitted with stroke in the medical wards in Saveetha Medical College from December 2015 till May 2016 was observed for hypertension.

Result: This study reveals that the signs and symptoms of stoke increases with the incidence of hypertension for that particular patient. Here, from the 50 stroke patients examined, 14 patients were reported with accelerated hypertension at the time of report, at Saveetha Medical College.

Conclusion:50 stroke patients were admitted with a complaint of dizziness and unconsciousness in the general ward at Saveetha Medical College have been diagnosed and treated with complete care. Out of which 14 patients have reported with accelerated hypertension at the time of report, which have no history of systemic hypertension. Maximum incidence of intra cerebral bleed was in above 60yrs. Females have highs systolic pressure compared to males which leads to increased intracerebral bleeding or hematoma. Under which their status were constantly monitored and maintained. From the above discussion it is clear that hypertension is a significant risk factor of stroke.

Keywords: *Stroke, Hypertension, Cerebrovascular accident, intracerebral bleeding*

Introduction

Stroke is the third leading cause of death in the world. The incidence of stroke approximately doubles each decade after the age of 45, with a 2% annual risk over 80 years of age and a 3–7% annual risk over 95 years of age. It is the cause of one in eight deaths and constitutes a formidable burden of disability for the patients and their relatives and the wider community [1]. Stroke is defined as an abrupt onset of a neurological deficit that is attributable to a focal vascular cause and lasts more than 24 hours. It is manifested either, as brain infarction or haemorrhage. A variety of causes result in stroke which comes under two sub groups: One, resulting from ischemia and the other from brain haemorrhage. After thrombosis and embolism intracranial haemorrhage is the 3rd most frequent cause of stroke. Acute ischemic stroke, intracerebral hemorrhage and subarachnoid hemorrhage were the three main emergencies regarding stroke [1, 2]. However recent studies were stressing that untreated hypertension is a significant risk factor for hemorrhagic stroke [3, 4].

Materials and Methods

An observational study is carried out with duration of 6 months from December 2015 till May 2016. Fifty patients admitted with stroke in the medical wards, Saveetha Medical College were taken up for this study. A detailed history including symptoms, occupational history, address, history of diabetes mellitus, hypertension, ischaemic heart disease, cerebrovascular accident and family history of hypertension, diabetes mellitus, ischaemic heart disease, and cerebrovascular accident were taken from the conscious patients and from reliable care giver in unconscious patients. The patients had been subjected to detailed clinical examination including blood pressure, complete blood count, computed tomography scan were obtained in all subjects. Patient was constantly checked for blood pressure every 4 hours. Informed consent from the patients and approval from Ethics committee were also obtained.

Inclusion criteria: All stroke patients above the age of 35, with accelerated hypertension at the time of report and no history of systemic hypertension or under anti-hypertensive medications.

Exclusion criteria: All stroke patients below the age of 35 and history of hypertension and who are under anti-hypertensive medications

Results

The eldest stroke patient was 87 years old. Mean age of hypertensive bleed was 60 years. Of 50 stroke patients, 12 were females and 38 were males. 8 patients were Type II Diabetic, 14 patients have accelerated hypertension, 6 patients with the history of cerebrovascular events, 16 patients reported with hemeplegia. Of the 50 stroke patients examined, 14(28%) patients reported with accelerated hypertension at the time of report.

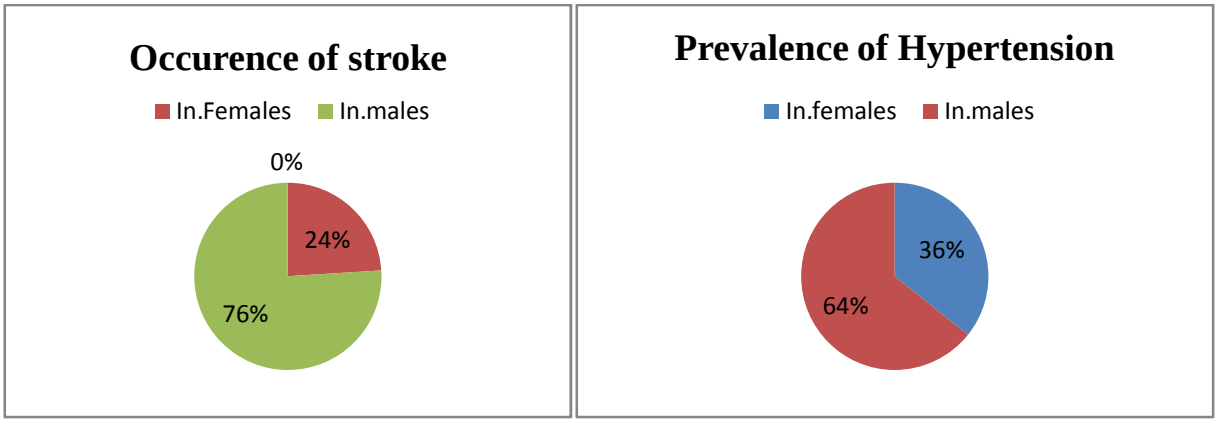


Figure 1 and 2: Shows prevalence of stroke and hypertension

Discussion

In this study, 28% of the patient has a history of hypertension regardless of the sex. CT brain scans of all the patients have been confirmed with stroke. On constant blood pressure check-up at every 4 hours, the initial blood pressure were greater than 190/140mmHg, the highest systolic pressure reaching 220mmHg and the diastolic pressure reaching 140mmHg recorded in a 47 year old female[8]. 47% of the female patients have a history of hypertension and an increased systolic blood pressure ranges between 220-190mmHg and also diastolic blood pressure ranges between 140-100mmHg. 40% of male patients has ischemic stroke. As the duration of hypertension increased the incidence of intra cerebral bleed also increased. High systolic and diastolic blood pressure is associated with increased risk of intra cerebral bleed. The incidence of intra cerebral bleed was found to increase stepwise with elevation of either systolic or diastolic blood pressure [5, 6]. Since the pressure inside the haematoma is equal to the arterial blood pressure, a high BP might be predisposing to large haematoma or re-bleeding. Hence patients with high blood pressure causes increased haematoma [7]. High blood pressure is evident in hypertensive patient; in such case 90%

of the female patients develop increased haematoma [9, 10]. Majority of the female patients with intra cerebral bleed had sedentary life style compared to males [11, 12]. Majority of the males were manual labourers. Even during the treatment hypertensive patients show varied blood pressures with took quite some time to stabilize them. Particularly in resource-limited settings, aside from BP control, stroke prevention efforts should be targeted towards these identified risk factors for stroke among hypertensive patients. Carotid diameter should be further explored as an intermediate risk marker for stroke [13-17].

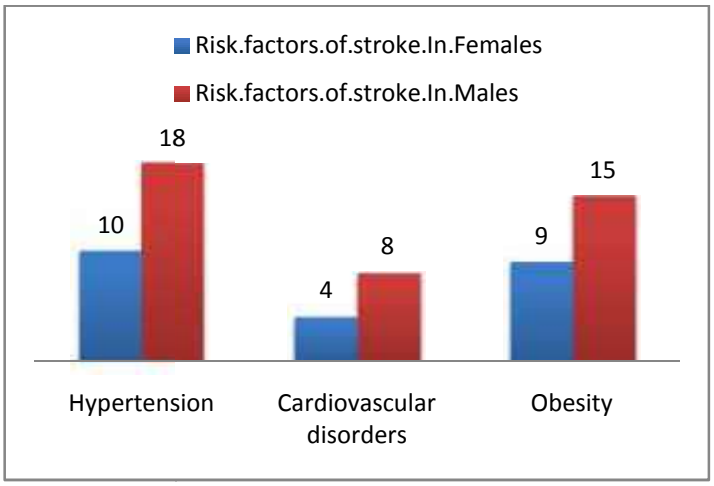


Figure 3: Graph showing percentage of risk factors among males and females

Conclusion

50 stroke patients were admitted in the general ward in Saveetha Medical College have been diagnosed and treated with complete care. Out of which 14 patients have reported with accelerated hypertension at the time of report, which have no history of systemic hypertension. Maximum incidence of intra cerebral bleed was in above 60yrs. Females have highs systolic pressure compared to males which leads to increased intracerebral bleeding or hematoma [18-20]. Under which their status were constantly monitored and maintained. From the above discussion it is clear that hypertension is a significant risk factor of stroke.

Corresponding Author

Madhusudhan Vasantharajan

Senior Lecturer, Department of Pedodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences

Saveetha University,
162, Poonamallee High Road,Chennai 600077,TamilNadu, India
Email: dr.madhusudhan24@gmail.com
Telephone Number:+91 9790166987

Reference

1. Ali L, Jamil H, Shah M.A. Risk factors and stroke. J Coll Phys Surg Pak 1997; 7(1):7-10.
2. Sushiba Nechikkat, Chandni R, Sasidharan PK. Hypertension as a Risk Factor for Haemorrhagic Stroke in Females. JAPI 2014; 62: 23-25
3. Woo D, Sauerbeck LR, Kissela BM, Khoury JC, Szaflarski JP, Gebel J, Shukla R, Pancioli AM, Jauch EC, Menon AG, Deka R, Carrozzella JA, Moomaw CJ, Fontaine RN, Broderick JP. Genetic and environmental risk factors for intracerebral hemorrhage: preliminary results of a population-based study. Stroke. 2002; 33:1190–1196.
4. Kissela BM, Sauerbeck L, Woo D, Khoury J, Carrozzella J, Pancioli A, Jauch E, Moomaw CJ, Shukla R, Gebel J, Fontaine R, Broderick J. Subarachnoid hemorrhage: a preventable disease with a heritable component. Stroke. 2002; 33:1321–1326.
5. Omar T, Ueda K. Risk factors of cerebral stroke in Japan. Prospective epidemiological study in Hisayama community. Excerpta Medica 1982; 119-135.
6. Hubert HB, Feinleib M, McNamara PM, Castelli WP. Obesity is an independent risk factor for cardiovascular disease: a 26-year follow-up of participants in the Framingham Heart Study. Circulation 1983; 67:968-77.
7. Jong Ku Park. Risk factor for hemorrhagic stroke in Wonju Korea 1998; 3:229-235.
8. Harshfield GA, Pickering TG, Kleinert HD, Blank S, and Laragh JH: Situational variations of blood pressure in ambulatory hypertensive patients. Psychosometric Med 1982; 44 (3): 237–245.
9. Devereux RB, Pickering TG, Harshfield GA, Kleinert HD, Denby L, Clark L, Pregibon D, Jason M, Sachs I, Borer JS, and Laragh JH: Left ventricular hypertrophy in patients with hypertension: Importance of blood pressure response to regularly recurring stress. Circulation 1983; 68(3): 470–476.
10. Jason M, Devereux RB, Borer JS, Pickering T, Fisher J, Harshfield G, and Berkowitz A, Laragh J: 24-Hour arterial pressure measurement: Improved prediction of left ventricular dysfunction in essential hypertension. Am J Cardiol 1983; 1(2): 599.

11. West JE, Busch-Visniac IJ, Harshfield GA, and Pickering TG: Foil electret transducer for blood pressure monitoring. J Acoust Soc Am 1983; 74: 680–686
12. Marion RM, Sullivan PA, Harshfield GA, Kleinert HD, Pickering TG, Laragh JH: The value of home blood pressure vs. physician office blood pressure. Circulation 68: III - 37 (1983).
13. Owolabi MO, Agunloye AM. Risk factors for stroke among patients with hypertension: A case–control study. Journal of the neurological sciences. 2013 Feb 15; 325(1):51-6.
14. Wilber JA, Barrow JG. Hypertension–a community problem. Am J Med. 1972; 52:653–663.
15. Pessah-Rasmussen H, Engstrom G, Jerntorp I, Janzon L. Increasing stroke incidence and decreasing case fatality, 1989–1998: a study from the stroke registers in Malmo, Sweden. Stroke. 2003; 34:913–918.
16. Manjer J, Carlsson S, Elmstahl S, Gullberg B, Janzon L, Lindstrom M, Mattisson I, Berglund G. The Malmo Diet and Cancer Study: representatively, cancer incidence and mortality in participants and nonparticipants. Eur J Cancer Prev. 2001; 10:489–499.
17. Jerntorp P, Berglund G. Stroke registry in Malmo¨. Stroke. 1992; 23: 357–361.
18. The sixth report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. Arch Intern Med. 1997; 157:2413–2446.
19. Janzon E, Hedblad B, Berglund G, Engstrom G. Changes in blood pressure and body weight following smoking cessation in women. J Intern Med. 2004; 255:266 –272.
20. Haider AW, Chen L, Larson MG, Evans JC, Chen MH, Levy D. Antecedent hypertension confers increased risk for adverse outcomes after initial myocardial infarction. Hypertension. 1997; 30:1020 –1024.

Aesthetic Assessment of the Effect of Gingival Exposure in Smile of Patient among the General Public

Affrin. J[1],A.V. Arun[2], Sreedevi Dharman[3]
GraduateStudent[1], Professor[2],Department of Orthodontics,
Senior Lecturer[3], Department of Oral Medicine
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The smile plays an importantrole in facial expression. Facial attractiveness and smile aesthetics arestrongly related toeach other. An attractive, well-balanced smile is a highly regarded treatment objective, along with creating a functional occlusion. The aim is to assess the aesthetics by studying the influence of gingival exposure in the smile of patients.

Materials and Method: The study included 100 individuals. Colour photographofa smilingfemale changed on the computer,displaying thedifferent gingival exposures, from 5mm of gingival exposure to 5mm of upper lip incisor coverage was assessed by laypersons. The individuals were asked to rate the attractiveness of the smiles with altered gingival display. Statistics was done in SPSS software.

Results: 19% of laypersons have chosen 0mm as best smile which is no gingival exposure during smile .18% felt 1mm as best smile which is little amount of gingival exposure. 8% felt,-1mm as best smile which covers some portion of central incisors. 1% felt
-5mm as best smile.Age or gender didn't show any sort of variation.

Conclusion: The perception of laypersons in evaluating gingival display may be different from that of orthodontists.There is influence of gingival exposure in smile and thus contributing to aesthetics. Therefore, it is important for publicto participate inthe decision-making process of orthodontic treatment planning.

Key Words*: smile, gingival exposure,orthodontic treatment, attractiveness of smile, general public.*

Introduction

Smile is what gives beauty to a face. Everybody wants to have a beautiful smile as compliments, the personality, and appearance of a person. So however there is no definition for a beautiful smile. This is because the perception of a beautiful smile varies from person to person. What looks very good for somebody or even for the certain population may not be acceptable for people from different population or even within the same population. So this brings us to a very vital question on. Whether our perception of a good smile actually matches what they actually want.The smile is the most effective means by which people convey their emotions.[1].One smile can make all the difference in the world. One smile has the power to release stress, calm you down, make you attractive, make someone else happy and believe it or not, smiling can actually cause happiness. That is why; Smile at Life has become the personal motto for someday smile is the prettiest thing one can wear. This is absolutely true. Whether you are female or male, a smile is all you need to complete your beauty. There is nothing more attractive than a man with a smile. Besides, smiling also shows people that you are person with positive attitude and easier to get along with.Aesthetics is often the main complaint in the dental office and patients usually evaluate treatment results based on the positive changes in their smile.[2]. Each person has a particular way to self-assess his/her own appearance and the beauty of other persons. [3]. Physical attractiveness is an important social issue in our culture, and the face is one of its key features. [4].The demand for improved dental appearance is one of the major motives of orthodontic treatment.Aesthetic perception varies from person to person and is influenced by each person's experiences and social environment.The search for improved dent facialaesthetics persists in modern society. Thus, pretty faces and beautiful smiles were inspired and patients have sought treatment modalities to improve dent facialaesthetics' and so to the yieldpositive changes in their smile.[5-10].Once we realize the importance of the smile in a facial context, we are able to extrapolate even further. It is determining not only in the perception of facial attractiveness, but also with the perception of one's psychological characteristics.

Gummy smile has a negative effect on the aestheticsof smile. But we can correct the gummy smile easily through various treatment options. An overexposure of gum tissue in upper jaw can result from the abnormal eruption of the teeth. Teeth covered by gum tissue appear short, even though they might actually be the proper length.The manner in which the upper jaw bone grew and developed could cause the appearance of a gummy smile. For example, if

there was an excessive over protrusion of the upper jaw within the gum tissue, one might experience an obvious gummy appearance when they smile causing anaesthetic appearance. Smile analysis is the most important stage as far as diagnosis; treatment planning and treatment prognosis is concerned for any dental treatment involving aesthetics as their objective.

The evaluation of the intrinsic characteristics of the smile is a necessary procedure to achieve consistent form in orthodontic treatments, which in turn makes it necessary to recognise the components and factors that affect these characteristics. Evaluating beauty is always subjective. However, we need adequate tools to overcome the challenge of this subjectivity. In orthodontics, it is not enough only to recognize what is interfering with the smile—it requires a diagnosis of what is not normal, in order to establish a treatment plan.The smile line is the determining factor in the evaluation of mouth aesthetics. The aesthetic results of orthodontic treatments always maintain a strong relationship to smile line. It is not uncommon for the conclusion of orthodontic treatments to be compromised by the smile line. Either high or low smiles aid in compromising the results. This is the great challenge of contemporary orthodontics in its search for its excellence. This study is a attempt to solve this problem and to find out which type of smile is most liked by the patient. So the aim was to assess the aesthetics by studying the influence of gingival exposure in the smile of patients.

Materials and Methods

A set of smile photographs showing well aligned teeth were chosen. The teeth wereexposed at different levels in each picture. There were 11photos that were prepared for the study the teeth alignment were the same in all the pictures.The only difference in these pics was the amount of upper incisor crowns exposure below the lips. The exposure varied from -5 mm below the lip margin to +5mm above the gum margin. That is, The first smile had 5 millimetres of Gingival exposure below the central incisors , second smile picture had 4mm of gingival exposure, the third smile picture had the 3mm of gingival exposure, the fourth picture had to 2mm of gingival exposure, fifth picture had 1mm of gingival exposure ,Sixth picture had no gingival exposure ,Seventh picture had 5mm of only central incisor exposure Eight picture had only 4mm of central incisor exposure ,Ninth picture had only 3mm of central incisor exposure ,Tenth picture had only 2mm of central incisor exposure. Eleventhpicture had only 1mm of central incisor exposure, and the last picture had no central

incisor exposure. The pictures were randomly arranged and were shown to 100 general public. Among the 11 pictures displayed in front of them, the people engaged in the study were asked to select the best picture in their opinion and grade 1for the best smile and 5 being for the worst smile. The results obtained were statistical analysed and the conclusions were derived from the same.

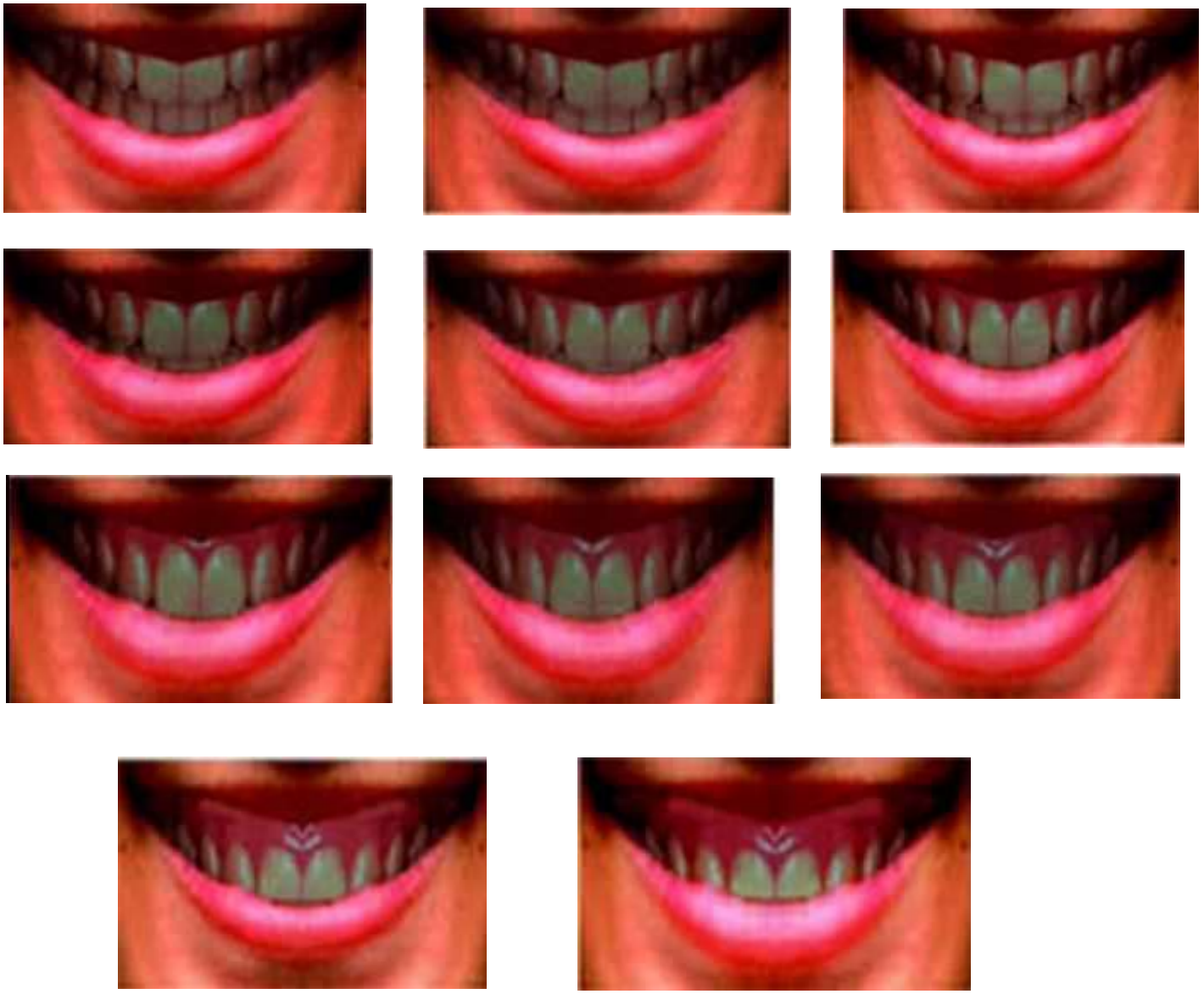


Figure: Photograph Showing Various Smiles with Gingival Exposure from -5mm To +5mm Respectively

Results

Among 200 individuals,19% felt 0mm as best smile which is no gingival exposure during smile. 18% feltt1mm as best smile which is little amount of gingival exposure was Seen. 13% felt 2mm as best smile in which gingival exposure was slightly increased compared to 1mm.12% felt 3mm as best smile which was slightly more exposure compared to 2mm.

11% felt 4mm as best smile which is more compared to 3mm and ,8% felt -1mm as best smile which covers some portion of central incisors. 7% felt -2mm as best smile which was covering more portion of central incisor compared to -1mm and ,5% felt -5mm as best smile,3% felt -3mm as best smile,3% felt -3mm as best smile,1% felt -5mm as best smile. And Age or gender didn't Show any sort of variation. (Table 1)(Fig1)

Table 1: FrequencyOf Gingival Exposures From -5mm To +5mm Influence On Smile Aesthetics

gingival exposure					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	-5.00	1	1.0	1.0	1.0
	-4.00	3	3.0	3.0	4.0
	-3.00	3	3.0	3.0	7.0
	-2.00	8	8.0	8.0	15.0
	-1.00	7	7.0	7.0	22.0
	.00	19	19.0	19.0	41.0
	1.00	18	18.0	18.0	59.0
	2.00	13	13.0	13.0	72.0
	3.00	12	12.0	12.0	84.0
	4.00	11	11.0	11.0	95.0
	5.00	5	5.0	5.0	100.0
	Total	100	100.0	100.0	

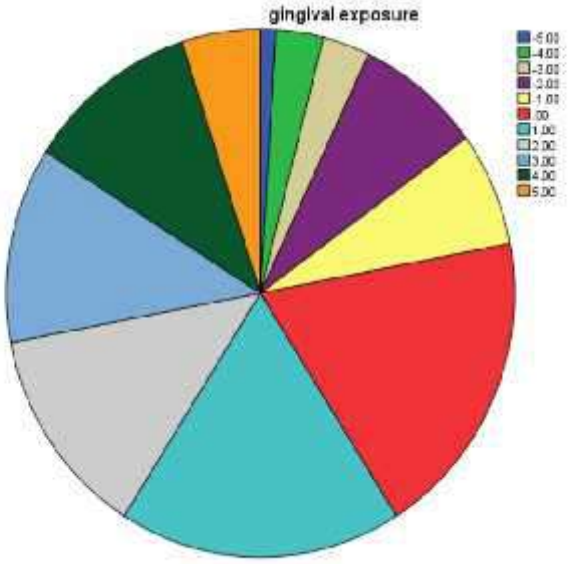


Figure2:Pie Chart Depicting On Gingival Exposures From -5mm To +5mm And Its Influence On Smile Aesthetics

Discussion

The smile line or the aestheticzone is nothing but teeth which are when we smile is determined by various factors, including: The shape and size of lip, facial muscles, the shape and size of the teeth and the gum tissue. The optimal smile line appearance should reveal the least amount of gum tissue possible. Gum tissue which is visible in the smile line should b balanced, even that are in harmony with the upper lip. It is for this reason that many people with a gummy smile or excessive gingival display feel their smile to be unattractive, oftentimes feeling reluctant to smile at all. However, more serious underlying dental conditions could be present. The disproportionate amount of gum and tooth showing when you smile can be caused by the overactive muscles that control the top lip, which causes the lip to curl up and expose the excess gum tissue above the teeth. Showing ‘too much gum’ when smiling is often noticed when people look at photos of themselves.

A smile demonstrating minimal gingival display has been deemed to be more aesthetic than a smile with excessive gingival display. [11-13]The most important aesthetic goal in orthodontics is to achieve a balance smile which can be achieved by appropriate positioning of the teeth and gingival scaffold within the dynamic display zone. Smile develops when

person seeks happiness, humour or greetings. The amount of gingival display of the patients should be considered as an important parameter as far as aesthetics is concerned. Smile acts as a method of communication. The various etiological factors which have been described for an aesthetic smile is not only determined by tooth position, size, shape, and colour but also by the amount of gingival tissue revealed and the framing of lips [14]. Various conditions like malpositioning of teeth, altered passive eruption, recession, loss of inter proximal papilla leads to an anaestheticappearance. [15].Among the different involved factors, the aesthetics of the smile has an important influence in the perception about the individual appearance and personality [16, 17].

In some studies, post orthodontic treatment, there was different perception among different people. Kokich[18] reported that orthodontists classified smiles as least attractive only when midline shifts reached 4.0 mm. On the contrary, Pinho[19] reported that orthodontists and prosthodontists were less tolerant of dental midline shifts, and rated 1.0 mm and 3.0 mm shifts as less attractive, respectively. Ker [20] also found that laypersons accepted as much as 4 degrees of canting (equivalent to 3.0 mm).Our study as just focused on the best smile among the general public with no age or gender category. And gummy smile showed the least score and 0mm gingival exposure showed the maximum score.

Three important aspects are involved in analyzing smile aesthetics, such as, gingival display, curvature formed by the incisal edge of anterior superior teeth and the upper lip, and width of the buccal corridor [21]. From a strictly orthodontic perspective, dentogingival display and transverse dimension are usually considered the most important factors in smile analysisPreference is given to a smile line where the elevation of the upper lip is close to the gingival margin of maxillary incisors [22-26]. Most of the females were more concerned about their smiles, they were inspired by the patients already benefited by Orthodontic treatment and so they felt that they will look better after Orthodontictreatment [27]. In general the dentists were more critical than the patients as reported in other studies and more patients were satisfied with their appearance than were unsatisfied [28]. Position of lip during smile also influences the clinical and technical procedures required for aesthetic restorations.

Assessment of smile provides information about the relationship between the teeth and surrounding pink tissue, and is a key element of diagnosis and treatment planning in cosmetic

dentistry. The relationship between upper lip and display of gingival tissues and teeth denotesthat periodontal outlook depends on the smile architecture. An imaginary line following the lower margin of the upper lip, with a convex appearance is de Ned as smile line [29,30].

Dental attractiveness plays an important role in seeking orthodontic treatment.[31].Brisman's study [32]he segregated the opinions of patients, dental students and dentist, concluded that all groups preferred teeth of a similar shape, but the dentists preferred relatively longer and thinner teeth than the patients. Mehl (2015) showed a difference between students and dentists and emphasised the need to teach aesthetics at dental school [33]. Prahl-Andersen's study [34] compared the opinions of laypeople, general dentists and orthodontists, finding that the general dentists and orthodontists essentially agreed in their responses but as a combined group, they were much more critical than the laypeople that were more likely to accept increased or reverse overjets and mild crowding [34].Shiyan et al. (2016) stressed the need to be aware of the difference in perception between orthodontists and patient [35]. Others agreed and found orthodontists were found to be more critical than general dentists [36] [37]. Geographic variation in opinions also exists [38].

Conclusion

Gingival display of 0mm and 1mm showed highest score on our study. Knowledge about the intrinsic characteristics of the smile also helps in the aesthetic perception. Being able to evaluate the smile of each patient assures the professional of the possibility of seeing what needs to be done, what can be done, and what should be accepted. Which aids in interpreting the nuances of a smile gives each orthodontist the opportunity to act in a conscious manner in the mouth aesthetic treatment of their patients, allowing the diagnosis to be integrated with the prognosis and giving a realistic outlook of the results than can be obtained. Buccal corridor, number of exposed teeth during smiling, frontal, oblique and profile facial analyses, relationship between resting and speech positions and the smile are the factors that should also be observed in order to achieve a better diagnosis of mouth aesthetics. Theperception of laypersons in evaluating gingival display may be different from that of orthodontists. Importantly, treatment should be discussed with patients so as to individualize treatment planning and, as a result, fulfil their desires. Therefore, it isimportant for public to participate in the decision-making process of orthodontic treatment planning.

Corresponding Author

Sreedevi Dharman

Senior Lecturer,Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University

162, Poonamallee High Road,Chennai-600077,Tamil Nadu, India

E-Mail: sanjamrut@gmail.com, Telephone Number: 9841009003

Reference

1.Hulsey CM. An Aesthetic evaluation of lip-teeth relationships present in the smile. Am J Orthod 1970;57:132-44.

2. Isiksal E, Hazar S, Akyalçin S. Smile aesthetics: perception and comparison of treated and untreated smiles. Am J Orthod Dent facialOrthod 2006; 129:8-16.

3. Czarnecki ST, Nanda RS, Currier GF.Perceptions of a balanced facial profile. Am J Orthod Dento facial Orthod1993; 104:180-187.

4. Flores-Mir C, Silva E, Barriga MI, Lagravere MO, Major PW. Layperson's perception of smile aesthetics and facial views. J Orthod. 2004; 31:204–209.

5. Machado AW, Moon W, Gandini Jr LG. Influence of maxillary incisor edge asymmetries on the perception of smile aesthetics among orthodontists and laypersons. Am J Orthod Dentofacial Orthop. 2013; 143(5):658-64.

6. Machado AW, Macomb R, Moon W, Gandini Jr LG. Influence of the vertical position of maxillary central incisors on the perception of smile aesthetics among orthodontists and laypersons. J Aesthetic Rest Dent. 2013; 25(6): 392-401.

7. Machado AW, Moon W, Campos E, Gandini Jr LG. Influence of spacing in the upper lateral incisor area on the perception of smile aesthetics among orthodontists and laypersons. Journal of the World Federation of Orthodontists. 2013; 25(2): e169e174.

8.Camara CALP. EstheticalemOrthodontia: Diagrams de Reference'sEstéticasDentárias (DRED) e Faciais (DREF). RDental PressOrtodonOrtop Facial. 2006; 11(6):1308-56.

9. Camara CA. EstéticasemOrthodontia:seislinhashorizontais do sorriso. Dental Press J Orthod. 2010; 15(1):118-31.

10. Machado AW, Santos TC, Araujo TM, Gandini Jr LG. The role of orthodontics as an auxiliary tool to lip augmentation.An Bras Dermatol 2011; 86(4):773-77.

11. Moore T, Southard KA, Casko JS, Qian F, Southard TE. Buccal corridors and smile aesthetics. Am J Orthod Dentofac Orthop 2005; 127: 208-13.

12. Mackey RJ. An evaluation of smiles before and after orthodontic treatment. Angle Orthod 1993;63:183-9.

13. Kokich VG. Aesthetics: The orthodontic-periodontics restorative connection. Semin Orthod 1996;2:21-30.

14. Jensen J, Joss A, Lang NP. The smile line of different ethnic groups in relation to age and gender. Acta Med Dent Helv. 1999; 4:38–46

15. Dolt AH, 3rd, Robbins JW. Altered passive eruption: An aetiology of short clinicalcrowns. Quintessence Int.1997; 28:363–72.

16. Beall AE. Can a new smile make you look more intelligent and successful? Dent Clin North Am 2007; 51:289-297.

17. Abu Alhaija ES, Al-Shamsi NO, Al-Khateeb S. Perceptions of Jordanian laypersons and dental professionals to altered smile aesthetics. Eur J Orthod 2011; 33:450-456.

18. Kokich VO, Jr, Kiyak HA, Shapiro PA.Comparing the perception of dentists and lay people to altered dental aesthetics. J Esthet Dent. 1999; 11:311–324.

19. Pinho S, Ciriaco C, Faber J, Lenza MA. Impact of dental asymmetries on the perception of smile aesthetics. Am J Orthod Dentofacial Orthop. 2007; 132:748–753.

20. Ker AJ, Chan R, Fields HW, Beck M, Rosenstiel S. Aesthetics and smile characteristics from the layperson's perspective: a computer-based survey study. J Am Dent Assoc. 2008; 139:1318–1327.

21. Camara CA. Estéticaemorthodontiaseislinhashorizontais do sorriso. Rev ClínOrtodon Dental Press. 2010 Jan-Feb; 15(1):118-31.

22. Sarver DM. The importance of incisor positioning in the aesthetics smile: the smile arc. Am J Orthod Dentofacial Orthop.2001; 120:98-111.

23. Peck S, Peck L, Kataja M. Some vertical lineaments of lip position.Am J Orthod Dentofacial Orthop.1992; 101:519-24.

24. Geron A, Atalia W. Influence of sex on the perception of oral and smile aesthetics with different gingival display and incisal plane inclination. Angle Orthod. 2005; 75(5):778-84.

25. Caetano SRO, Telles CS. Estética do sorriso: relação dos lábios com osdentes e alinha do sorriso. J Bras OrtodonOrtop Facial. 2005; 10(58):421-9.

26. Desai S, Upadhyay M, Nanda R. Dynamic smile analysis: Changes with age. Am J Orthod Dentofacial Orthop. 2009; 136:310.e1-310.e10.

27. RathnaSubhashiniM.H and Ravindra Kumar Jain.Thomas, M., Reddy, R. and Reddy, B.J.Motivational Factors Influencing Orthodontic Treatment among South Indian Adult Population.(2011) Perception Differences of Altered Dental Aesthetics by Dental Professionals and Laypersons. Indian Journal of Dental Research, 22, 242-247.

28. Crispin BJ, Watson JF. Margin placement of Aesthetic veneer crowns. Part II: Posterior tooth visibility. J Prosthet Dent 1981; 45:389-91.

29. Towsend CL. Resective surgery: An esthetic application. Quintessence Int 1993; 24:535-42.

30.Jayachandar D, Dinesh SS. Factor's affecting patient's desire for seeking orthodontic treatment. International Journal of Orthodontic Rehabilitation. 2016 Jul 1;7(3):89.

31. Brisman AS. Aesthetics: a comparison of dentists' and patients' concepts. The Journal of the American Dental Association. 1980 Mar 1; 100(3):345-52.

32. Mehl CJ, Harder S, Wolfart S, Vallarta O, Trinkler A, Wenz HJ, Kern M. Influence of dental education on esthetic perception. The International Journal Of Esthetic Dentistry. 2015;10(3):486-99.

33. Prahl-Andersen B, Boersma H, Moore AW. Perceptions of dentofacial morphology by laypersons, general dentists, and orthodontists.Journal of the American Dental Association (1939). 1979 Feb; 98(2):209-12.

34. Huang S, Xu Q, Xu S, Rao N, Li X. Orthodontists' and laypeople's perception of smile height aesthetics in relation to varying degrees of transverse cant of anterior teeth. Australian Orthodontic Journal.2016 May; 32(1):55.

35.Cotrim ER, VasconcelosJúnior ÁV, Haddad AC, Reis SA. Perception of adults' smile aesthetics among orthodontists, clinicians and laypeople. Dental Press Journal
Of Orthodontics. 2015 Feb; 20(1):40-4.

36, 37.Thomas M, Reddy R, Reddy BJ. Perception differences of altered dental aesthetics bydental professionals and laypersons.Indian Journal of Dental Research. 2011 Mar 1; 22(2):242.

38.Mehl C, Harder S, Lin J, Vallarta O, Kern M. Perception of dental aesthetics: influence of restoration type, symmetry, and colour in four different countries. International Journal of Prosthodontists.2015 Jan 1; 28(1).

Analysis Of Angulation Of Impacted Third Molars To Second Molars And Relation To Nerve Canals – A Radiographic Study

Priyanka Shenoy.B[1], Jayanth Kumar [2], Sri Sakthi.D [3]

Graduate Student[1], Reader[2],Department of oral medicine and radiology,

Reader[3],Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute Of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The third molar is the most commonly impacted tooth in the whole dentition because of the eruption timing and decrease in the length of Jaw .The mandibular third molars are the frequently impacted teeth in the human. They are often associated with pericoronitis, periodontitis, cystic lesions, neoplasm, pathologic root resorption and can cause detrimental effects on adjacent tooth. Also, many researches have shown that impacted third molar weakens the angle of mandible. It makes it susceptible to fracture and is implicated in the etiology of crowding of the lower arch. The various types of relationship of Inferior Alveolar Root canal to third Molar root tips are loss of roof of canal,Narrowing, Deflection, and dilaceration of roots.

Materials and Methods In this Observational study, up to 100 orthopantomograms (OPG) of the patients who were referred to the radiology clinics of the Saveetha Dental College were evaluated. Data including the gender, Pattern, the angulation type of mandibular third molar to second molar and its relation of third molar were evaluated by statistical tests.

ResultsOut of the 100 orthopantomograms, the most common type of impaction is the mesioangular(77%) and the least common type of impaction is distoangular. The winters classification was used for determining the type of impaction. The radiographis signs were assessed in this present study. The presence/absence of the four radiographic signs previously mentioned were examined and from the results it is seen that radiographic signs which can lead to nerve damage or associated with nerve damage are present in majority of the radiographs. The most common radiographic sign observed here is Narrowing/diversion of the nerve canal(46.1%) followed by interruption of lamina dura of the nerve canal(45.5%) and outline distortion/deviation of the canal.

Conclusion The absence of radiographic signs associated with a relationship between the LTM and the canal indicates a minimum risk of nerve damage. In this present study, only the presence/absence of the four radiographic signs previously mentioned were examined and from the results it is seen that radiographic signs which can lead to nerve damage or associated with nerve damage is present in majority of the radiographs.

Keywords : *Impaction, third molars, OPG's, angulation, nerve canals.*

Introduction

Tooth impaction is a pathological situation in which a tooth can not or will not erupt into its normal functioning position. This problem can be solved by dental treatment.[1] The removal of the impacted third molars can be simply performed using elevators and/or forceps, but may require surgical intervention. This increases the risk of complications, such as nerve paresthesia, alveolar osteitis, hemorrhage, or even fracture of the jaw.[2]Removal of mandibular impacted third molars are routine surgical procedure performed by maxillofacial surgeons in dental clinics as well as hospital setups. To prevent complications a useful diagnostic tool is needed that can determine the relationship between inferior alveolar nerve and the impacted third molar.Panoramic radiography is suggested as the technique of choice to evaluate impacted third molars as well as to estimate the pre-operative risk for inferior alveolar nerve injury associated with third molar surgery.[3] The mandibular third molars are the most frequently impacted teeth that can be found in human.[4] The prevalence of third molar impaction ranges from 16.7% to 68.6% [1] .Studies have shown that patients with retained impacted third molars are significantly more susceptible to mandibular angle fracture of the mandible,and is implicated in the etiology of lower arch crowding, Temporomandibular Joint (TMJ) disorders, vagueorofacial pain and neuralgias.[5,1]. Teeth which are impacted may cause detrimental effects on the adjacent tooth. They are most commonly associated withfactors like periodontitis, cystic lesions, neoplasm, pericoronitis, root resorption.[6] Patients with the impacted third molars may present with caries, pain, gingivitis and oral infections . [5] Various methods have been used for classification of

impaction, in which impaction is described based on the impaction level,[7] the angulations of the third molars,[8] and the relationship to the anterior border of the ramus of the mandible.

Various classifications have been given on impacted teeth such as Winters classification, Pell and Gregorys classification, Killey and Kay, Archers classification of impacted maxillary teeth, etc. Winter's classification is classified based on the inclination of the impacted tooth to the long axis of the second molar into distoangular, mesioangular, horizontal, vertical and transverse. This classification is used for the study as it is simple and easily understandable [9] Currently, the panoramic radiograph is the technique of choice to evaluate impacted mandibular third molars. It permits an initial evaluation of any problems related to impacted mandibular third molar.[2] The proximity of the lower third molar (LTM) to the inferior alveolar canal in the preoperative radiographic study is considered a risk factor for damage to the inferior alveolar nerve,the incidence of which is approximately 1.1%. [10]The reported frequency of inferior alveolar nerve injury associated with third molar removal ranges from 0.6% to 5.3% but the risk of permanent IAN injury is less than 1% . [3] Injuring the inferior alveolar nerve is rare but typical of specific surgical procedure. The patient’s age along with the experience of the surgeon and how deeply the tooth is impacted are influential factors.[11]

A radiographic examination is an imaging exam is an essential tool for diagnosis and surgical management. So accurate preoperative radiographic examination is therefore considered indispensable before extraction of mandibular third molar. It is one of the conventional diagnostic aids used in impactions. The ramus, distal bone covering the impacted tooth, relation of mandibular canal to the third molars, etc can be assessed.[12]

Thus, the objective of this study is to evaluate the pattern of third molar impaction and its relation to nerve canals using panoramic radiograph in a sample of hundred patients. The presence or absence signs which may cause damage to the canal were assessed in OPG'S (root contacting the nerve canal, narrowing/diversion of the canal, outline distortion, interruption of laminadura with the canal) in the study. The purpose of taking up this observational study is because of the paucity of literature and to throw a light upon the topic.

Materials and methods

Study Design An observational radiographic study.

This study was conducted on 100 patients. One hundred Orthopantomograms (OPG'S) of patients who presented to the outpatient Dental centre were selected for the study. Only the patients with the impacted third molars were included from the records.Third molar was considered impacted if it did not have any functional occlusion and at the same time roots were fully formed.

For the purpose of this study, OPG X-rays were selected randomly which meant that not all patients included in the study had attended the Clinic for the management of impacted wisdom teeth. The gender and the angulation of the lower impacted third molars were determined separately for the 3rd and the 4rth Quadrant from the OPG records and its relationship with the nerve canal is assessed by four questions – Q1)Whether the root contacting the nerve canal, Q2)narrowing/diversion of the canal, Q3) Outline distortion of the canal, Q4) Interruption of lamina dura. The radiographs were taken from the digital panaromic machine. The third molar was considered impacted when it was not fully erupted to the assumed normal functional position in the occlusal plane. The angulation of the impacted third molar was recorded using Winter's classifcation with reference to the angle formed between the intersected longitudinal axes of the second and third molars.

Study Area Saveetha Dental College, Chennai, Tamil Nadu.

Study Population Patients reporting to the radiology department of Saveetha Dental College.

Inclusion Criteria

The criteria for selection of radiographs are

- either unilateral or bilateral lower third molar impaction.
- Clinically asymptomatic or symptomatic patients

Exclusion Criteria

Records of patients aged younger than 17 years, records with any pathological dentoalveolar condition, any craniofacial anomaly or syndrome such as Down syndrome, cleidocranialdysostosis,with the presence of incomplete records and poor quality OPG, incomplete root formation of third molar, or absence of adjacent second molar, were excluded.

Results

The radiographic signs were evaluated using four questions

Q1) Whether the root contacting the nerve canal

Q2) Narrowing/diversion of the canal

Q3) Outline distortion of the canal

Q4) Interruption of lamina dura. The type of impaction was determined.

To compare proportions Chi-Square test is applied, if any expected cell frequency is less than five then Fisher's exact test is used. SPSS version 22.0 is used to analyse the data. Significance level is fixed as 5% ($\alpha = 0.05$). If P-Value is <0.05 then statistically significant.

A total of 100 panoramic radiographs of patients aged not younger than 17 years were examined. In this study, clinical data were collected from the only dental teaching hospital in Saveetha Dental College, which has a policy of using DPT for all new patients. It was seen that the younger years age group had the highest prevalence of tooth impaction but this decreased with increasing age. This may reflect increased dental awareness in this group of patients. However, the relatively high proportion of patients in their third decade may also have increased the overall prevalence of impacted teeth in this study. Of the 100 panoramic radiographs, mandibular third molars were most commonly encountered.The radiograph was analysed based on the angulations using winters classification. Table 1 shows questionwise distribution of the data based on the type of impaction. In this study, more than 50% of impacted mandibular third molars were mesially angulated (Graph 1).

Graph 1 – Distribution of study subjects based on type of Impaction

Table 1 questionnaire wise distribution of data

Q	QUADRANT	IMPACTION YES(n) %		HORIZONTAL n(%)	MESIOANGULAR n(%)	VERTICAL n(%)	P VALUE
Q1	3rd quadrant	36	(44.4%)	8(29.6%)	21(56.7%)	7(41.1%)	0.05(>0.059)
	4rth quadrant	48	(55.8%)	18(58.0%)	20(50.0%)	10(66.6%)	0.5(>0.05)
	TOTAL	84	(50.2%)	26(44.8%)	41(53.2%)	17(53.1%)	0.3(>0.05)
Q2	3rd quadrant	38	(46.9%)	11(40.7%)	19(51.4%)	8(47.1%)	0.7(>0.05)
	4rth quadrant	39	(45.3%)	15(48.4%)	14(35.0%)	10(66.7%)	0.1(>0.05)
	TOTAL	77	(46.1%)	26(44.8%)	33(42.9%)	18(56.3%)	0.4(>0.05)
Q3	3rd quadrant	45	(55.6%)	13(48.1%)	23(62.2%)	9(52.9%)	0.5(>0.05)
	4rth quadrant	29	(33.7%)	14(45.2%)	9(22.5%)	6(40.0%)	0.1(>0.05)
	TOTAL	74	(44.3%)	27(46.6%)	32(41.6%)	15(46.9%)	0.8(>0.05)

Q 4	3rd quadrant	35	(43.2%)	8(29.6%)	22(59.5%)	5(29.4%)	0.02(<0.05)
	4rth quadrant	41	(47.7%)	14(45.2%)	17(42.5%)	10(66.7%)	0.2 (>0.05)
	TOTAL	76	(45.5%)	22(37.9%)	39(50.6%)	15(46.9)	0.3(>0.05)

Discussion

The position of the impacted lower third molars have been evaluated from the radiographs. The data were analysed using Pearson chi square test. The significance level is fixed as 5% ($p<0.05$).

In the present study, the most common angulation of impaction in the mandible was Mesioangular(77%) followed by horizontal (58%) and vertical(32%) .The present study is in agreement with those of Queket al,(13) Kramer and Williams,[14] and Moris and Jerman [15]Hemamalinibalaji et al,[3]MP Santosh kumar et al,[9]which says that the mesioangular impaction is the most common angulation in mandible. Linden et al., Hattab et al., Knutsson et al. and Sedaghatfar et al. in their study found maximum number of third molars to be mesioangular. [16,17,18,19]

However, the present study is in contrast to those of Hugoson and Kugelberg,[20] who found the vertical angulation to be the most common. This could be due to the fact that a different method of classification of angulation was used in this study.Hazza'a et al in their study also found highest number of vertically placed third molars followed by mesioangular, distoangular, and horizontal third molars. These variations in angulation of mandibular third molars may be because of the reason that the studied population in each study was quite different from each other.

It appears that mesioangular impactions are probably the commonest type and this may be due to their late development and maturation, path of eruption and lack of space in mandible at later age.[1]

It was mentioned above that the proximity of the mandibular third molar to the mandibular canal is considered a risk factor for damage to the inferior alveolar nerve.

In this present observational study, the third molar root was contacting the nerve canal in 50.2% of the cases. The most common radiographic sign observed here is Narrowing/diversion of the nerve canal(46.1%) followed by interruption of lamina dura of the nerve canal(45.5%) and outline distortion/deviation of the canal. This is in agreement with that of *Costa FWG et al* where the narrowing of the mandibular canal was present in 86 of the 126 studied cases (68.25%).[21] It also agrees withValmaseda-Castellón et al. [22], where diversion of the canal was the only radiographic sign statistically associated with nerve damage. For Blaeser et al. [23], darkening of the root, interruption in the white line of the canal and the diversion of the canal were all statistically related with nerve damage.

This study is also in agreement with that of Hemamalinibalaji et al[3] where the most significant radiological sign noticed was the diversion of the mandibular canal.In the literature review conducted by Leung and Cheung, [24] the incidence of IDN deficit was highest in lower wisdom teeth showing radiographic sign of diversion of ID canal by its root (30%), followed by darkening of root (11.6%) and defected root by the ID canal (4.6%).

For gaikwad PT et al,[25] the radiographic sign most consistently associated with nerve involvement was the interruption of white line of the canal (10%), followed by the diversion of the canal(5%). The presence/absence of risk factors observed in panoramic radiographs is not always accurate, since it is a bidimensional-imaging feature. For better observation of these signs is necessary to obtain an image in three dimensions, such as computed tomography, and thus enable a better and more accurate visualization of anatomical structures related to the third molar.

However, in the present study,only the presence/absence of the four radiographic signs previously mentioned were examined and from the results it is seen that radiographic signs which can lead to nerve damage or associated with nerve damage are present in majority of the radiographs/cases. For Sandhya et al[12]in their study, out of 100 cases of impacted teeth, 82 cases had radiographic findings associated with the impacted teeth.

It can be concluded that the absence of radiographic signs associated between the LTM and the canal indicates a minimum risk of nerve damage and viceversa.

Conclusion

Panaromic radiograph can be used as a valuable predictor of outcome of the impacted mandibular third molars position, as they have quite good cost-information ratio. In the

present study, the area of concentration was on the angulation of the lower third molars to second molars and its relation to nerve canals. Mesioangular type and horizontal type of impaction were observed most common and it should be taken into consideration for high frequency of complications after extraction. More precise studies are necessary to evaluate the impaction of third molars in a randomized sample representative of Indian population as they help to evaluate the pattern of third molars impaction in India. The radiographic signs in the OPG that indicate a relationship between the LTM and the inferior alveolar canal are considered a risk factor for nerve damage. The absence of radiographic signs associated with a relationship between the LTM and the canal indicates a minimum risk of nerve damage. In this present study, only the presence/absence of the four radiographic signs previously mentioned were examined and from the results it is seen that radiographic signs which can lead to nerve damage or associated with nerve damage is present in majority of the radiographs. The absence of radiographic signs associated with a relationship between the LTM and the canal indicates a minimum risk of nerve damage.

Further studies should also be conducted to determine how many patients with impacted third molars are symptomatic or actively seek treatment. Further studies are also needed to assess the pattern of third molar impaction in other regions. More precise studies are necessary to evaluate the impaction of third molars in a randomized sample representative of Indian population as they are required to evaluate the pattern of third molars impaction in India.

Corresponding author

Sri Sakthi. D

Reader,Department of Public Health Surgery

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University,Chennai,India

162, Poonamallee High Road,Chennai – 600077,Tamil Nadu, India.

Mobile Number: +91 – 9962787531

Email ID: drsrisakthiphd@gmail.com

References

1. Maryam-AlsadatHashemipour, MehrnazTahmasbi-Arashlow , FarnazFahimi-Hanzaei. Incidence of impacted mandibular and maxillary third molars: a radiographic study in a Southeast Iran population. Med Oral Patol Oral Cir Bucal. 2013 Jan 1;18 (1):e140-5.
2. Siddharth Gupta, Rahul R. Bhowate et al. Evaluation of Impacted Mandibular Third Molars by Panoramic Radiography. International Scholarly Research Network ISRN Dentistry.2011.8 pages.
3. HemamaliniBalaji et al. Evaluation of Impacted Mandibular Third Molar using Panaromic Radiographs. J.Pharm. Sci. & Res. Vol.7(11),2015, 940-945.
4. Lima CJ, Silva LC, Melo MR, Santos JA, Santos TS. Evaluation of the agreement by examiners according to classifications of third molars. Med Oral Patol Oral Cir Bucal. 2012;17:e281-e216.
5. Rosfaima Othman Jaffar, Mon Mon Tin-Oo. Impacted mandibular third molars among patients attending Hospital UniversitiSains Malaysia. Archives of Orofacial Sciences. Vol 4(1),2009, 7-12.
6. Ma'aita J, Alwrikat A. Is the mandibular third molar a risk factor for mandibular angle fracture? Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2000;89:143-6.
7. Pell GJ, Gregory BT. Impacted mandibular third molars: classification and modified techniques for removal. Dent Digest. 1933;39:330–338.
8. Winter GB. The Principles of Exodontia as Applied to the Impacted Third Molar. St. Louis, MO: American Medical Book Co; 1926.
9. M.P. Santhosh Kumar et al. Angulations Of Impacted Mandibular Third Molar: A Radiographic Study in Saveetha Dental College. J.Pharm. Sci. & Res. Vol.7(11),2015,981-983.
10. Cristina Palma-Carrio et.al. Radiographic signs associated with inferior alveolar nerve damage. Med Oral Patol Oral Cir Bucal. 2010 Nov1;15(6):e886-90.
11. Thomas Schneider, Katharina Filo et al. Variations in the anatomical positioning of impacted mandibular wisdom teeth and their practical implications. Swiss dental Journal. Vol 124 5/2014.
12. Sandhya, R.,Dr. SreedeviDharman. Radiographic Findings associated with impacted third molars. International Journal of Recent Advances in Multidisciplinary Research. Vol. 03, Issue 03, pp.1334-1338, March, 2016.

13. Kramer RM, Williams AC. The incidence of impacted teeth. Oral Surg Oral Med Oral Pathol.1970;29(2):237–241.
14. Quek SL, Tay CK, Tay KH, Toh SL, Lim KC. Pattern of third molar impaction in a Singapore Chinese population: a retrospective radiographic survey. Int J Oral Maxillafac g. 2003;32(5):548–552.
15. Morris CR, Jerman AC. Panoramic radiographic survey: a study of embedded third molars. J Oral Surg.1971;29(2):122–125.
16. M. Sedaghatfar, M. A. August, and T. B. Dodson.Panoramic radiographic findings as predictors of inferior alveolar nerve exposure following third molar extraction. Journal of Oral and Maxillofacial Surgery,vol.63,no.1, pp. 3–7, 2005.
17. W. V. D. Linden, P. Cleaton-Jones, and M. Lownie. Diseases and lesions associated with third molars. Review of 1001 cases. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and, vol.79, no.2, pp. 142–145, 1995.
18. F. N. Hattab, M. A. Rawashdeh, and M. S. Fahmy. Impaction status of third molars in Jordanian students. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and, vol.79, no.1, pp. 24–29,1995.
19. K. Knutsson, B. Brehmer, L. Lysell, and M. Rohlin. Pathoses associated with mandibular third molars subjected to removal. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontics, vol.82, no.1, pp. 10–17, 1996.
20. Hugoson A, Kugelberg CF. The prevalence of third molars in a Swedish population. An epidemiological study. Community Dental Health. 1988;5(2):121–138.
21. Costa FWG et al. Correlation between radiographic signs of third molar proximity with inferior alveolar nerve and postoperative occurrenceof neurosensory disorders. A prospective, double-blind study. ActaCirúrgicaBrasileira - Vol. 28 (3) 2013.
22. Valmaseda-CastellónE,Berini-AytésL,Gay-Escoda C. Inferior alveolar nerve damage after lower third molar surgical extraction: a prospective study of 1117 surgical extractions. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2001;92:377-83.
23. Blaeser BF, August MA, Donoff RB, Kaban LB, Dodson TB. Pan- oramic radiographic risk factors for inferior alveolar nerve injury after third molar extraction. J Oral Maxillofac Surg. 2003;61:417-21.

24. Leung YY, Cheung LK. Risk factors of neurosensory de cits in lower third molar surgery: an literature review of prospective studies. Int J Oral Maxillofac Surg. 2011;40(1):1-10.
25. Gaikwad PT, Datarkar AN . Radiographic signs associated with damage to inferior alveolar nerve: a diagnostic tool to perform coronectomy. Journal of Indian academy of oral medicine and radiology, July-September 2011;23(3):S320-322.

Antibacterial Activity of Tea Tree Oil against Clinical Isolates of Escherichia Coli

Sai Chaitanya Raj [1], Gopinath P [2], Meenakshi Krishnan[3],

Graduate Student [1],Senior Lecturer [2], Department of Microbiology,

Senior Lecturer [3], Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: Complementary and alternative medicines such as tea tree oil have become increasingly popular in recent years. This essential oil has been used for several decades in European countries but have now become increasingly popular in a wide array of products. The primary uses of tea tree oil is mainly because of the antibacterial and antiseptic properties of the oil itself. Antibacterial activity of tea tree oil on clinical isolates of Escherichia coli has been reviewed in this study.

Materials and Methods: A total of 20 non repetitive urinary isolates of Escherichia coli were collected followed by qualitative method of Antibiotic Susceptibility testing.

Results: We have observed that, clinical isolates of E. coli were inhibited from 0.03-0.25% of tea tree oil. The MIC of tea tree oil was appeared to be 0.03% for E. coli

Conclusion: Tea tree is found to have antibacterial activity against clinical isolates of E. coli. However, the studies on toxic and irritant properties of essential oils are imperative, especially when considering any new products for human administration. This can be used as alternative and complementary antibacterial agents for controlling the infections.

Keywords: *Antibacterial, Escherichia, Antibiotic, Susceptibility, Infections*

Introduction

Escherichia coli, one of the most common and frequent etiological agents of urinary tract infection (UTI) and is also responsible for wide array of other infections such as bacteremia, pneumonia, soft-tissue infection and neonatal meningitis. Multiple antibiotic resistance exhibited by such E. coli strains is a major concern, as they are showing resistance even to the carbapenems, which are considered to be the last resort of antibiotics for such stubborn E. coli infections. Tea Tree Oil (TTO) or melaleuca oil is known since long for many of its medicinal uses and also for other uses [1]. It is an essential oil obtained from the leaves and terminal branches of M. alternifolia through steam distillation method. It is available all over the world and is used for its antimicrobial properties [2]. It should be handled with care as it is toxic on oral consumption and for injections but can be used for its topical application use [3] studies have shown that TTO is not genotoxic in vitro in mammalian cells [4]. It is incorporated as an active substance in many topical applications used for treatment of cutaneous infections for controlling dandruff, lice, herpes, acne and other skin infections [5]. After a study on Tea tree oil samples [6] about 100 compounds were identified in tea tree oil. Antibacterial activity of Tea tree oil has been noted and has been reported to be broad-spectrum inhibiting bacteria. The minimum inhibitory concentration (MIC) for tea tree oil for most of the susceptible bacteria has been reported ranging from 0.003% for Prevotellaintermedia [7,8] and maximum greater than 8% (v/v) for Enterococcus faecalis strains of bacteria [9]. A recent study on ATCC reference and clinical strains [10] of different bacteria namely, Pseudomonas aeruginosa ,Staphylococcus aureus, E. faecalis, Salmonella Enteritidis, S. Typhimurium and escherichia coli. Tea tree oil is bacteriostatic in low concentration but bactericidal at higher concentrations. Though exact mechanism of action is yet to be understood it is hypothesized and proved to some extent [11,12] that TTO act through increasing permeability of liposomal systems causing lysis and the loss of membrane integrity and manifested by the leakage of ions and the inhibition of respiration and ultimately death of the bacterium [13].In early studies on the antimicrobial activity of TTO, it was hypothesized to be more active against antibiotic-resistant bacteria [14] thus attracted considerable interest. Thereafter, several studies were conducted but only on a limited number of strains specifically using mupirocin resistant and methicillin-resistant strains of *Staphylococcus aureus* (MRSA) and of other bacteria [15,16,17]. Most of the studies concluded an insignificant

difference in TTO sensitivity of antibiotic-resistant and sensitive strains. Resistance to TTO in clinical isolates has not yet been reported and resistance of bacteria to conventional antibiotics has not been correlated with susceptibility to TTO, suggesting that cross resistance does not occur. Tea tree oil is seen as important alternative as a topical antimicrobial for antibiotics. Search for "alternatives to antibiotics" is identified as one of the most important goals to combat the emerging antibiotic resistant pathogens [18]. It is often claimed that TTO can replace antibiotics at least for topical applications [19]. The present study aimed to test the sensitivity of variety and number of the bacterial strain, isolated from clinical cases with the potential to cause a wound or cutaneous infections, to TTO and simultaneously to common use topical antimicrobials. Data, interpreted and was analyzed for contact and association between Tea tree oil sensitivity and antimicrobial sensitivity and multi-drug resistance in different bacteria different origins. Thus the aim of the study was to determine the antibacterial activity of tea tree oil against clinical isolates of E. coli.

Materials and Methods

Bacterial Isolates

A total of 20 non repetitive urinary isolates of Escherichia coli were collected from Saveetha Medical College and Hospitals, Chennai. They were processed for standard biochemical tests and confirmed. Isolates were preserved in semisolid trypticase soy broth stock and were stored at 4 ºC until further use. Antibiotic susceptibility testing was done.

Antibiotic Susceptibility Testing

Antibiotics/antimicrobial agents are the major drugs of choice of the physician's desk to treat the host of various infections. This prescribing pattern may be one of the reasons for the development of resistance for the antibiotics [20]. Therefore, Antibiotics susceptibility testing plays an important role to check the effectiveness and efficacy of a drug against a bacterial strain or several bacterial strains and select the best drug that acts against the strains. The main objectives of the testing are to find out drug resistance in common microorganisms and the susceptibility to drug of choice for a particular infectious

organism or bacterium can be found out and can be assured. Mechanism of antimicrobial resistance: There are number of ways by which microorganisms are resistant to antimicrobial agents. These includes: Bacteria produce enzymes which can destroy the antimicrobial agents before it reaches the targets e.g. Beta lactamase enzyme hydrolyses beta lactam drugs which develop resistance to it. Impermeable cell for antimicrobial drugs e.g. Gram negative bacteria may become resistant to Beta lactam antibiotics by developing a permeability barrier. Mutation develops macrolide resistance. Bacterial efflux pump that expels antimicrobial drugs from cell before they can reach their targets. Specific Metabolic pathways in the bacteria are genetically altered so that antibacterial agents cannot exert an effect [21,22]. Antimicrobial susceptibility testing has several important purposes which can include a) A laboratory test which can determine how effective antibiotic therapy is against a bacterial infection or infections b) Antibiotic Susceptibility testing can control the use of antibiotics in clinical practice. c) Antibiotic susceptibility testing will help the clinicians in choosing the choice of drugs for the treatment of infection or infections d) Antibiotic Susceptibility Testing can help the local sequence of antibiotic prescriptions. e) Antibiotic Susceptibility Testing can be used to reveal the changing trends in local isolates.

Methods of Antimicrobial Susceptibility Testing

There are several methods of antibiotic susceptibility testing. The method followed in this study was the qualitative method of Antibiotic Susceptibility testing.

Qualitative Method: This method is used for testing of isolates from healthy patients with an intact immune defense system, in less serious infections such as Urinary Tract Infection. There are two qualitative methods.

Disk diffusion test : The disk diffusion sensitivity test also known as the Kirby Bauer disk diffusion method. It is a simple and practical test which uses the antibiotic- impregnated wafers or disks to test whether particular bacteria is susceptible to specific antibiotic or not [23,24] The bacterial inoculum was uniformly spread using sterile cotton swab on a sterile Petridish Mueller Hinton agar. The antibiotic disks were placed on top of the previously inoculated Mueller Hinton agar medium surface with the help of sterile instruments like forceps. Each disc must press down on one another to ensure complete contact

with agar surface. The plates were incubated for 18–24 h at 35-37 degree C temperature in bacteriological incubator before an interpretation and arrival of the result. The antibiotic is found to diffuse from the disc into the agar in decreasing amounts, the further it is away from the disk. This states that the antibiotic that is furthest away from the disks has a better permeability in the disks. If the organisms were killed or inhibited by the concentration of the antibiotic, there will be no growth in the immediate area around the disks will be represented as the zone of growth inhibition. The diameter of the zone of inhibition is directly proportional to the sensibility of the isolate and to the diffusion rate of antibiotics through the agar medium. A zone of inhibition was measured in millimeters by either measuring the Radius: Measure half the distance of the zone and then multiply by 2. This method was used when part of the zone is not clear or has grown into another zone. Diameter: Measure the entire length of the zone and subtract the disk diameter . The standard disk size was found to be about 5 to 6 millimeters. The result of the test can be interpreted by using the criteria published by Clinical and Laboratory Standard Institute (CLSA formerly the National Committee for the Clinical Laboratory Standard or NCCLS) [25]. The results of the disk diffusion test are “qualitative” and will be reported out as: Susceptible: ‘The term “susceptible” represent that isolates are inhibited by the usually recommended dosage of an antimicrobial agents. However, this term doesn’t fully assure clinical success in all cases. Predicting clinical outcome based on susceptibility testing and the use of drugs shown to be in the susceptible category is very imprecise. This imprecision is due to the effect of host responses, site of infection, toxin production by bacteria that is independent of antimicrobial susceptibility, the presence of biofilm, drug pharmacodynamics and other factors. As an outcome, there can be two categories, namely Intermediate and resistant categories.

A. **Intermediate:** ‘The “intermediate” category includes isolates with antimicrobial MICs that approach usually attainable blood and tissue levels and for which response rates might be lower than for other susceptible isolates. The intermediate category implies clinical efficacy in body sites where the drugs are physiologically concentrated, quinolones or beta lactams in the urine or when a higher than normal dosage of a drug can be used (e.g. Beta lactams).

B. **Resistant:** 'The category indicates that the isolates are not inhibited or stopped by the usually achievable concentrations of the antibiotics with normal dosage schedules, which demonstrate an existence of the specific microbial resistance mechanisms (e.g. Beta lactamase). The merits of the disk diffusion methods are simplicity in test, most patient cost friendly, a wide variety of disks can be chosen during disk selection, and the result can be easily interpreted by the clinicians. However, the demerits of this can include manual test, lack of automation and all slow growing bacteria cannot be accurately tested by this method. The limitation of this testing show that the microbiologist and clinician both should be cautious and should not forget that the response therapy in vivo may not always reflect the result of testing the sensitivity of patient's pathogen in vitro. A study conducted by Rakesh Kumar to study antimicrobial sensitivity pattern of Escherichia coli from urine samples of UTI patients used the resistant test method [26].

Well diffusion method: In this agar well diffusion method, a suitable agar medium was prepared, once the agar is solidified the medium was fully inoculated and was swabbed with bacterial suspension of approximately 1-2 X 108 CFU/mL using a cotton swab. The wells were prepared by punching with a six millimeters diameter standard sterile cork borer made up of stainless steel . These wells were filled up with 25 – 50 µL of the antimicrobial solution/s to be tested. Well diffusion test has been used for susceptibility testing of antifungals like fluconazole, itraconazole. The plates were incubated at 35 ± 2°C for 18 – 24 h. The antimicrobial activity is calculated in millimeter by using the expression: ZOI = Total Diameter of growth inhibited zone minus diameter of the well, where, ZOI is Zone of inhibition. The factors which may affect the result of AST included Density of an inoculum, Disk application time, Temperature of incubation, Potency of drug, inappropriate storage conditions, pH the agar medium, Moisture on the surface of the medium and effects of Thymidine or Thymine containing agar medium [20]

Results:

Sample wise distribution of clinical isolates of E.coli:

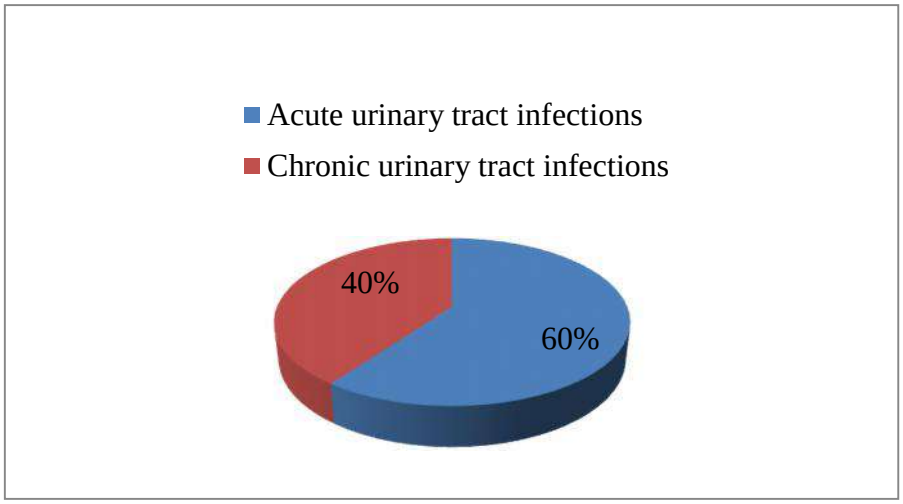


Figure 1: Sample wise distribution of urinary isolates of *E.coli*

Antibiotic Susceptibility Testing

Table 1: Antibiotic sensitivity pattern of E.coli

Antibiotics	Sensitivity(20) (%)	Intermediate (20) (%)	Resistant(20) (%)
Ampicillin	5	0	95
Amoxicillin	5	0	95
Ceftazidime	10	10	80
Cefotaxime	5	5	90
Amikacin	70	10	20
Gentamicin	45	20	35
Norfloxacin	15	15	70
Ciprofloxacin	20	5	75
Imipenem	70	0	30

We have observed that, clinical isolates of E. coli were inhibited from 0.03-0.25% of tea tree oil.

The MIC of tea tree oil was appeared to be 0.03% for E. coli.

Table 2: MIC of Tea Tree Oil

Dilutions of Tea tree oil	0.03%	0.06%	0.125%	0.25%	0.5%	1%	2%
No. of organisms	4 (20)	2 (10)	6 (30)	8 (40)	0	0	0

Discussion

Antibiotic susceptibility test fixed to routinely used antibiotics such as ampicillin, amoxicillin, norfloxacin, ceftazimide, cefotaxime, ciprofloxacin and gentamicin, imipenem by the Kirby Bauer disc diffusion method. Complementary and alternative medicines such as tea tree oil have become increasingly popular in recent decades. This essential oil has been used for almost 100 years in Australia but is now available worldwide both as neat oil and as an active component in an array of products. Tea tree oil have been historically used for their antiseptic and antiinflammatory actions. Food processors, food safety researchers, and regulatory agencies have been increasingly concerned with the growing number of food borne illness outbreaks caused by some pathogens. The increasing antibiotic resistance of some pathogens that are associated with foodborne illness is another concern. Hence, there has been increasing interest in the development of new types of effective and nontoxic antimicrobial compounds. Plant essential oils are a potentially useful source of antimicrobial compounds. Although numerous studies have been published on the antimicrobial activities of plant compounds against many different types of microbes, including food borne pathogens a review of the earlier literature reveals that the results reported for these different studies are difficult to compare, presumably because of the different test methods, bacterial strains, and sources of antimicrobial samples used.

Detection of antibacterial activity of tea tree oil against clinical isolates of E. coli:

Anti-bacterial activity of tea tree oil was tested against E. coli isolates by minimum inhibitory concentration method.

Mueller Hinton broth was supplemented with 0.002% (V/V) tween 80 (HiMedia, Mumbai) to enhance the dispersion of the essential oil. Agar dilution method was performed to attain the different concentrations of essential oil such as 0.03%, 0.06%, 0.125%, 0.25%, 0.5%, 1% and 2% in Mueller Hinton Agar (MHA). Media containing various concentrations of essential oil were poured over the sterile petridishes and allowed to dry. Media without essential oil was served as control plate.

Spot inoculation of 0.5 McFarland standard turbidity adjusted isolates were made on the plates and incubated at 37ºC for overnight.

The lowest concentration of the essential oils that completely inhibited the growth of isolates was considered as MIC. The lowest concentration of the essential oil, here tea tree oil that completely inhibited the growth of the isolates of Escherichia coli was found to be about 0.03 % in this study. This is found to be a much lower MIC when compared to the MIC achieved by other plant essential oils in a similar study which covers a whole spectrum of antibiotics. The MIC achieved in this study conducted in Australia was found to be about 0.23%.[21]. Another study performed confirms that many essential oil plant extracts possess in-vitro antibacterial and antifungal properties. The MIC as per this study was found to be roughly the same as in our study.[22] Another study, aimed at studying the membrane integrity of e coli bacteria when exposed to several different antibiotics upon tea tree oil, found that e coli lost its surface cell membrane integrity when it was exposed to around 0.5 % MIC of the antibiotic. [23]

Conclusion

Tea tree is found to have antibacterial activity against clinical isolates of E. coli. Nevertheless, the studies on toxic and irritant properties of essential oils are imperative, especially when considering any new products for human administration. This can be used as alternative and complementary antibacterial agents for controlling the infections.

Corresponding Author

Meenakshi Krishnan,

Senior Lecturer, Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

162, Poonamallee High Road, Chennai -600077,Tamil Nadu, India.
E-mail: drmeenakshisdc@gmail.com

Telephone Number: 8754415200

Reference

1. Auddy B, Ferreira M, Blasina F, Lafon L, Arredondo F, Dajas F, Tripathi PC, Seal T, Mukherjee B. Screening of antioxidant activity of three Indian medicinal plants, traditionally used for the management of neurodegenerative diseases. Journal ofEthnopharmacology. 2003 Feb 1;84(2-3):131-8.
2. Costa DC, Costa HS, Albuquerque TG, Ramos F, Castilho MC, Sanches-Silva A. Advances in phenolic compounds analysis of aromatic plants and their potential applications. Trends in Food Science & Technology. 2015 Oct 1;45(2):336-54.
3. de Sousa Barros A, de Morais SM, Ferreira PA, Vieira ÍG, Craveiro AA, dos Santos Fontenelle RO, de Menezes JE, da Silva FW, de Sousa HA. Chemical composition and functional properties of essential oils from Mentha species. Industrial Crops and Products. 2015 Dec 15;76:557-64.
4. Hale AL, Reddivari L, Nzaramba MN, Bamberg JB, Miller JC. Interspecific variability for antioxidant activity and phenolic content among Solanum species. American journal of potato research. 2008 Oct 1;85(5):332.
5. De Billerbeck VG. Huilesessentiellesetbactériesrésistantes aux antibiotiques. Phytothérapie. 2007 Dec 1;5(5):249-53.

6. Fisher K, Phillips C. Potential antimicrobial uses of essential oils in food: is citrus the answer?. Trends in food science & technology. 2008 Mar 1;19(3):156-64.
7. Safaei-Ghomi J, Ahd AA. Antimicrobial and antifungal properties of the essential oil and methanol extracts of Eucalyptus largiflorens and Eucalyptus intertexta. Pharmacognosy magazine. 2010 Jul;6(23):172.
8. Astani A, Reichling J, Schnitzler P. Comparative study on the antiviral activity of selected monoterpenes derived from essential oils. Phytotherapy Research. 2010 May 1;24(5):673-9.
9. Kaloustian J, Chevalier J, Mikail C, Martino M, Abou L, Vergnes MF. Étude de six huilesessentielles: composition chimiqueetactivitéantibactérienne. Phytothérapie. 2008 Jun 1;6(3):160-4.
10. Benjilali B, Tantaoui-Elaraki A, Ismaili-Alaoui M, Ayadi A. Méthoded'étude des propriétésantiseptiques des huilesessentielles par contact direct en milieugélosé. Plant. med. phytother. 1986;20(2):155-67.
11. Burt S. Essential oils: their antibacterial properties and potential applications in foods—a review. International journal of food microbiology. 2004 Aug 1;94(3):223-53.
12. Stefanakis MK, Touloupakis E, Anastasopoulos E, Ghanotakis D, Katerinopoulos HE, Makridis P. Antibacterial activity of essential oils from plants of the genus Origanum. Food control. 2013 Dec 1;34(2):539-46.
13. Devi KP, Nisha SA, Sakthivel R, Pandian SK. Eugenol (an essential oil of clove) acts as an antibacterial agent against Salmonella typhi by disrupting the cellular membrane. Journal of ethnopharmacology. 2010 Jul 6;130(1):107-15.
14. Djenane D, Yangueela J, Gomez D, Roncales P. perspectives on the use of essential oils as antimicrobials against Campylobacter jejuni CECT 7572 in retail chicken meats packaged in microaerobic atmosphere. Journal of Food Safety. 2012 Feb 1;32(1):37-47.
15. Balouiri M, Sadiki M, Ibnsouda SK. Methods for in vitro evaluating antimicrobial activity: A review. Journal of Pharmaceutical Analysis. 2016 Apr 1;6(2):71-9.
16. Balouiri M, Sadiki M, Ibnsouda SK. Methods for in vitro evaluating antimicrobial activity: A review. Journal of Pharmaceutical Analysis. 2016 Apr 1;6(2):71-9.

17. Canillac N, Mourey A. Antibacterial activity of the essential oil of Piceaexcelsa on Listeria, Staphylococcus aureus and coliform bacteria. Food Microbiology. 2001 Jun 1;18(3):261-8.
18. Nikolić M, Jovanović KK, Marković T, Marković D, Gligorijević N, Radulović S, Soković M. Chemical composition, antimicrobial, and cytotoxic properties of five Lamiaceae essential oils.Industrial Crops and Products. 2014 Nov 1;61:225-32.
19. Martucci JF, Gende LB, Neira LM, Ruseckaite RA. Oregano and lavender essential oils as antioxidant and antimicrobial additives of biogenic gelatin films. Industrial Crops and products. 2015 Sep 1;71:205-13.
20. Ashok Kumar, Gopinath P. Antibacterial activity of ginger oil against clinical isolates of Escherichia coli. IJSDR. 2018:3(2);14-17
21. Friedman M, Henika PR, Mandrell RE. Bactericidal activities of plant essential oils and some of their isolated constituents against Campylobacter jejuni, Escherichia coli, Listeria monocytogenes, and Salmonella enterica. Journal of food protection. 2002 Oct;65(10):1545-60.
22. Hammer KA, Carson CF, Riley TV. Antimicrobial activity of essential oils and other plant extracts. Journal of applied microbiology. 1999 Jun;86(6):985-90.
23. Cox SD, Mann CM, Markham JL, Bell HC, Gustafson JE, Warmington JR, Wyllie SG. The mode of antimicrobial action of the essential oil of Melaleuca alternifolia (tea tree oil). Journal of applied microbiology. 2000 Jan 1;88(1):170-5.
24. Reller LB, Weinstein M, Jorgensen JH, Ferraro MJ. Antimicrobial susceptibility testing: a review of general principles and contemporary practices. Clinical infectious diseases. 2009 Dec 1;49(11):1749-55.
25. Pasteran F, Mendez T, Guerriero L, Rapoport M, Corso A. Sensitive screening tests for suspected class A carbapenemase production in species of Enterobacteriaceae. Journal of clinical microbiology. 2009 Jun 1;47(6):1631-9.
26. Sindhu Priya K, Gopinath P. Antibacterial activity of Eucalyptus oil against clinical isolates of pseudomonas aeruginosa. Int J of current and advanced research. 2017:6(3);2944-2947.

Anti-Fungal Activity of Ginger Oil against Clinical Isolates of Candida Species

Prasanna Guru[1], Gopinath P[2], Meenakshi Krishnan[3]
Graduate Student[1], Senior Lecturer [2],Department of Microbiology,
Senior Lecturer [3], Department of Oral Medicine and Radiology,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: The use of essential oils from herbs and spices is a novel antimicrobial treatment to reduce the initial microorganism loads. In herbs and spices, there are many antimicrobial compounds exhibiting a wide range of activities against bacteria, yeasts and fungi. Essential oils from plants have been suggested as natural preservatives.Thus the aim of the present study is to assess the antifungal activity of ginger oil against clinical isolates of candida species.

MaterialsandMethods:A total of 20 non-repetitive clinical isolates of Candida species were collected from different samples of immunocompromised individuals attending a medical hospital, in Chennai. They were characterized by carbohydrate fermentation and assimilation tests and confirmed. Isolates were preserved in semisolid Sabouraud chloramphenicol semi solid stock and stored at 4˚C until further use. Candida species were further characterized by using Hichrom agar (Himedia, Mumbai).

Results: Clinical isolates of Candida sp. were inhibited from 0.125-1% of ginger oil. The MIC of ginger oil was appeared to be 0.25% for Candida spp**.**

Conclusion: The ginger oil is found to have antifungal activity against Candida spp. Since, any toxic or irritant property of ginger oil has to be under consideration, ginger oil can be used as alternative and complementary agent for controlling fungal infections.

Key Words: *Ginger oil, Candida species, Antifungal, Sabouraud agar, chloramphenicol, Medicinal plants, Cytoprotective*

Introduction

Medicinal plants are used as pharmaceuticals, nutraceuticals, cosmetics and also food supplements [1]. Plant derived products have been used for medicinal purposes for centuries. In traditional Indian medicine or Ayurveda, Zingiber officinale and many other herbs have been used as medicine. One of the most common infectious diseases of humans is urinary tract infections (UTIs), caused by Candida species, which responsible for more than 80% of cases worldwide. [2] About 6 to 20% of the human populations are experiencing asymptomatic bacteriuria (ABU), depending on age and gender and nearly 50% of women suffer from cystitis in their lifetime. Many ABU Candida isolates are phylogenetically associated to virulent uropathogenic Candida (UPEC) strains and some may have evolved from pathogenic strains by virulence attenuation. [3]Epidemiological studies also revealed rise in local fungal infections of chronic wounds (diabetic foot, burn, bed- sore, cancer ulceration), with the involvement of C. albicans biofilm.In the past two decades increased prevalence of fungal infections has been reported. Fungi cause both superficial and internal mycoses. Systemic fungal infections constitute a major community health problem in many parts of the world, both in developed and developing countries. Along with other potential infections, candida affecting oral environment is become common leading to oral thrush, denture stomatitis. Candida species are capable of causing a variety of deep seated and superficial infections. Candida sp are considered as one of the most hospital acquired infection. Due to increase in multi-drug-resistant strains of Candida, it is become necessary to search for new sources of antifungal agents and compounds that inhibit these resistance mechanisms. This has led to a search for therapeutic alternatives, particularly among medicinal plants and compounds isolated from them used for their empirical antifungal properties. The in vitro antimicrobial evaluation of lozenges containing extract of garlic and ginger was carried out. Results indicated that there was inhibition of growth by nystatin tablet but garlic and ginger combination only inhibited growth of laboratory strains of C. albicans.The use of essential oils from herbs and spices is a novel antimicrobial treatment to reduce the initial microorganism loads. In herbs and spices, there are many antimicrobial compounds exhibiting a wide range of activity [4].Hence, there is a great demand for novel antifungals belonging to a wide range of structural classes, selectively acting on new targets with fewer side effects. One approach might be the testing of plants traditionally used for their antifungal activities as potential sources for drug development. Medicinal plants are not only important to the millions of people for whom traditional medicine is the only

opportunity for health care and to those who use plants for various purposes in their daily lives, but also as a source of new pharmaceuticals.[5]

Ginger is an important spice in Thailand. In 2001, Thailand grew more than 30,000 million tons of ginger. It is widely used as an ingredient in the food, pharmaceutical, cosmetic and other industries. Garlic and ginger have been known to possess immunological and cytoprotective effects in the biological host. Ginger contains a unique flavour derived from both non-volatile and volatile oils. The pungent compounds are gingerol and shagaol, while zingiberene is a pre-dominant component of oils. Some volatile compounds having antimicrobial properties present in gingerare α-pinene, borneol, camphene and linalool.[6]The medicinal properties have been mainly used for treating the symptoms of vomiting, diarrhea, light-headedness, blurred vision, dyspepsia, tremors, decrease in body temperature and high blood pressure. Furthermore, 6-gingerol and 6-shagaol can reduce viability of gastric cancer cells.[7].[8] Ginger extracts have been reported to inhibit growth of Listeria monocytogenes, Salmonella typhimurium, Staphylococcus aureus, Bacillus cereus, B. subtilis, F. moniliforme and Mycobacterium sp.[9] The garlic and ginger lozenge demonstrated pronounced antifungal activity against the laboratory isolate of C. albicans but not against the clinical isolates of the same organism while nystatin gave inhibition zone diameter values of 21 to 41 mm. Escherichia coli and Staphylococcus aureus strains were resistant. Ginger oils showed very good inhibition of Salinococcus roceus, H. turkmenicus and Halococcus morrhuae isolated from salt cured fish.[10] Thus, the present study indented to determine the antibacterial activity of ginger oil against clinical isolates of Candida sp.

Materials and Methods

Candida Isolates

A total of 20 non-repetitive clinical isolates of Candida species were collected from different samples of immunocompromised individuals attending a medical college, in Chennai. They were characterized by carbohydrate fermentation and assimilation tests and confirmed. Isolates were preserved in semisolid Sabouraud’s chloramphenicol semi solid stock and stored at 4˚C until further use.

Characterization of Candida Species

Candida species were further characterized by using Hichrome agar (Himedia, Mumbai).

Preparation of Hichrome Agar

CHROM agar Candida (HiMedia, Mumbai) was prepared following manufacturer's instructions. About 21.02 gram of HiChromeCandida differentiation agar base (modified) was suspended in 500 ml of distilled water. It was heated to boiling gently to dissolve the medium completely. Then it was allowed to cool to 50^{o}C and rehydrated (one vial) contents of HichromeCandida selective supplement was added under aseptic precautions. It was mixed well and poured into petri dishes. Isolates were identified on Hichrome agar based upon the characteristic colour of the colony by sub culturing from Sabouraud's chloramphenicol agar plates and the Candida Hichrome plates were incubated at 37oC for 24- 48 hours. [8] Based on colour produced by the isolates speciation have been made.

Table 1: Isolated Candida Species

***Candida* species**	**Color**
C.albicans	Green
C.tropicalis	Blue
C.krusei	Pink dry colonies
C.kefyr	Pale
C.parapsilosis	Pale

Detection of Antifungal Activity of Ginger Oil against Clinical Isolates of Candida Spp

Antifungal activity of ginger oil was tested against Candida isolates by minimum inhibitory concentration method. Mueller Hinton broth was supplemented with 0.002% (V/V) tween 80 (HiMedia, Mumbai) to enhance the dispersion of the essential oil. Agar dilution method was performed to attain the different concentrations of essential oils such as 0.03%, 0.06%, 0.125%, 0.25%, 0.5%, 1% and 2% in Mueller Hinton Agar (MHA).

Viability controls were performed on the fungal strains in the liquid medium under the same assay conditions. The plates were sealed and incubated at 35°C ± 2°C for 24 to 48 h. The MICs of the products used in the biological assays were defined as the lowest concentrations capable of visually inhibiting the fungal growth in the wells, as compared to the growth under control conditions

Media containing various concentrations of essential oils were poured over the sterile petridishes and allowed to dry. Media without essential oil was served as control plate. Spot

inoculation of 0.5 McFarland standard turbidity adjusted isolates were made on the plates and incubated at 37ºC for overnight. The lowest concentration of the essential oils that completely inhibited the growth of isolates was considered as MIC. [11]

Results

Characterization of Candida Species by HichromeCandida Agar

A total of 20 clinical isolates of Candida sp were seeded on to Hichrome Candida agar and results were tabulated based on pigment production.

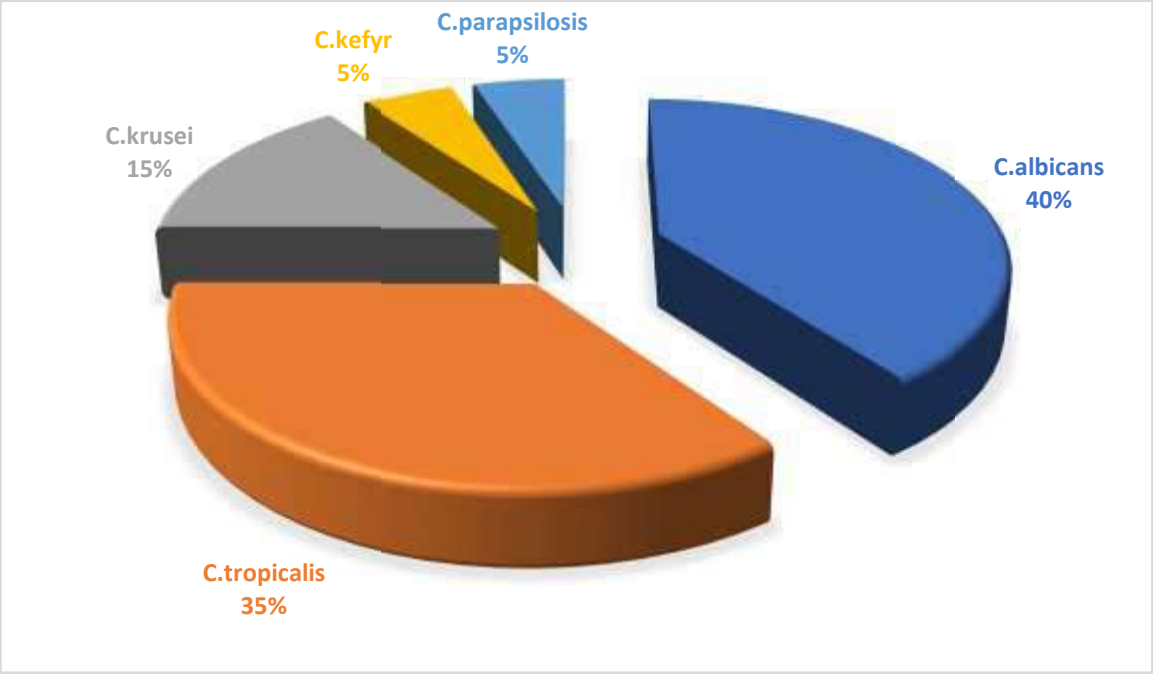


Figure 1: Pie chart showing distribution of Candida species

Figure 2: Representative picture showing Candida species on Hichrome Candida agar

Sample wise Distribution of Candida Species

Of the 20 clinical isolates of Candida sp, 6/20 (30%) were from oral thrush, 5/20 (25%) from urine, 3/20 (15%) from sputum, 3/20 (15%) from vaginal swab, 2/20 (10%) from ear swab and one (5%) from wound swab.

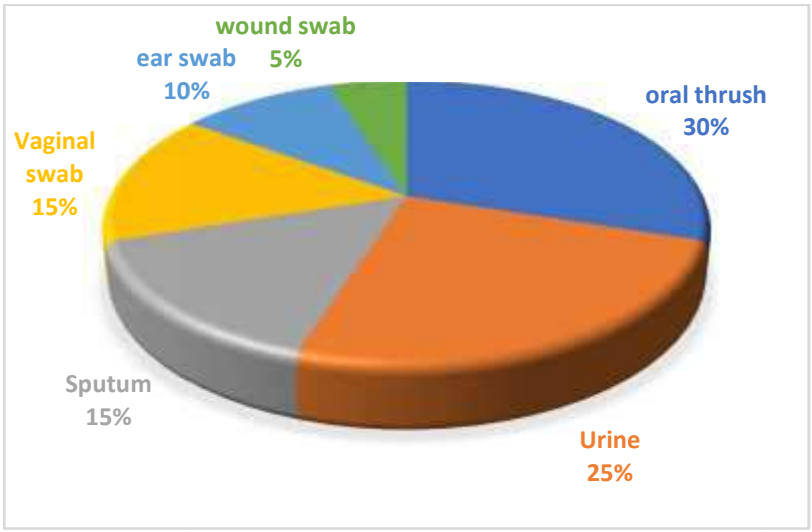


Figure 3: Result of antifungal activity of ginger oil against clinical isolates of Candida species

We have observed that, clinical isolates of Candida sp were inhibited from 0.125-1% of ginger oil.

The MIC of ginger oil was appeared to be 0.25% for Candida spp**.**

Table 2: The MIC of ginger oil against isolated Candida Species

Dilutions of ginger oil	0.03%	0.06%	0.125%	0.25%	0.5%	1%	2%
No. of organisms	0	0	3 (15)	2 (10)	9 (45)	6 (30)	0

Discussion

Plant oils used as cooking and flavouring agents are increasingly claimed to have broad spectrum antimicrobial activity. Selected oils have been suggested to have potent antimicrobial activity against skin infections, insect bites, chicken pox, colds, flu, measles, sinus congestion, asthma, bronchitis, pneumonia, tuberculosis and cholera, probably due to their phenolic, alcoholic and terpenoid constituents.

The various therapeutic effects of ginger has been reported which includes anti-emetic activity, anti- ulcer, antiplatelet, antipyretic, anti-inflammatory and antioxidant activity[11]. The antifungal activity of ginger has been attributed to gingerol and shagelol derived from the ethanolic extracts of ginger [12]

The study results are comparable with other studies suggesting that different antifungal agents are present in the Ginger extract. In the ginger rhizome there are several components which have antibacterial and anti-fungal effects.(13] The study done by Supreetha.S on antifungal therapy has shown that ethanol itself has antifungal activity against candida albicans. However, there was an increased inhibition zone when ethanolic extract of ginger was used when compared to ethanol alone and this was statistically significant.(14)

According to the study by Anupama N. Devkatte Gajanan B. Zore S. Mohan Karuppayil, these oils required more than 1.0% to exert fungicidal effect. MFC of these oils ranged from 1.0% to 3% except in the case of Jasmine, Ginger and Juniper oil, which were not fungicidal up to the 3% concentration.[15]

The higher content of geranial and other oxygenated compounds makes fresh ginger oil more potent than dry ginger oil. The content of hydrocarbon compounds are more in dry ginger oil compared to fresh ginger oil. [16] Earlier studies have reported that monoterpene compounds are more active than sesquiterpene compounds. Dry ginger oil hadhigher content of sesquiterpene hydrocarbons.Especially when this is compared to the results obtained for the standard nystatin tablet where the C. albicans strains showed susceptibility. It is worthy to note that in most cases of infection, a combination of antimicrobial activity and

one or more other biological effects, such as immunomodulation, could be responsible for overall effect of a natural product [17]

Study conducted by Prakasam et al from Chennai in 2014 demonstrated that, Acinetobacter strains were inhibited from 0.06 to 0.25%, 0.25-1% and 0.125-1% for clove, peppermint and eucalyptus oils respectively. In clove oil, 14/50 (28%) isolates were inhibited at 0.06%, 25/50 (50%) at 0.125% and 11/50 (22%) at 0.25% of clove oil. In peppermint oil, 34/50 (68%) isolates were inhibited at 0.25%, 12/50 (24%) and 4/50 (8%) were at 0.5% and 1% concentrations of peppermint oil respectively. In eucalyptus oils, 10/50 (20%) isolates were inhibited at 0.125%, 18/50 (36%) at 0.25%, 16/50 (32%) and 6/50 (12%) were at 0.5% and 1% respectively. Thus, the MIC of clove oil was found to be 0.06%, 0.25% for peppermint oil and 0.125% for eucalyptus oil. [19,18] In contrast, in our study, we used ginger oil against Candida sp isolates. 15% of isolates were inhibited at 0.125%, 10% were at 0.25%, 45% were at 0.5% and 30% were at 1% of essential oil.

Thus, the MIC of ginger oil against Candida sp was found to be 0.125%.The resistance of clinical strains of C. albicans isolated from prosthesis users to the antifungal standard nystatin, which is used for the treatment of Candida-associated denture stomatitis, demonstrates the importance of investigating the efficacy and mode of action of novel products on this clinically relevant pathogen. Such research will facilitate the discovery of new antifungal agents. [20]

In some essential oils such as eucalyptus and clove oils, there has been much research and documented some toxic and irritant properties. In spite of this, most of these oils are available as whole oils or as part of pharmaceutical and cosmetic products. However, the studies on toxic and irritant properties of essential oils are imperative, especially when considering any new products for human administration.[21]

The addition of citral to the growth medium at a sub-inhibitory concentration of 4 µg/mL (MIC/8) did not alter the resistance of the clinical strains or the standard to nystatin, as assessed by proliferation. Furthermore, there are no studies in the literature investigating the ability of citral to modulate nystatin activity. The ability of unconventional compounds to increase the antimicrobial activity or reverse the resistance of microorganisms to drugs classifies the compounds as modifiers of antifungal activity [22].

It is therefore likely that a combination of these biological effects of garlic and ginger and the demonstrated antimicrobial effect may explain its usefulness in the management of oropharyngeal infections, especially those of fungal origin in folklore medicine.

Conclusion

The ginger oil is found to have antifungal activity against Candida spp. However, the studies on toxic and irritant properties of essential oils are imperative, especially when considering any new products for human administration. This can be used as alternative and complementary agent for controlling the infections.Nowadays, the indiscriminate use of commercial antimicrobial drugs has caused multiple drug resistance in human pathogenic microorganisms. The resistance strains of C. albicans have become a cause of major health concerns and require novel antifungal agents to tackle this problem.

Corresponding Author

Meenakshi Krishnan

Senior Lecturer, Department of Oral Medicine and Radiology,

Saveetha University,Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

162, Poonamallee High Road, Chennai - 600077,Tamil Nadu, India

Email id: drmeenakshisdc@gmail.com

Phone Number: +91 8754415200

References

1. Sharma S, Vijayvergia R, Singh T. Evaluation of antimicrobial efficacy of some medicinal plants. J. Chem.Pharm. Res. 2010;2(1):121-124.
2. Ronald A. The etiology of urinary tract infection: traditional and emerging pathogens. Disease-a-month. 2003 Feb 1;49(2):71-82.
3. Foxman B. Epidemiology of urinary tract infections: incidence, morbidity, and economic costs. The American journal of medicine. 2002 Jul 8;113(1):5-13.
4. Mabbett AN, Ulett GC, Watts RE, Tree JJ, Totsika M, Cheryl-lynn YO, Wood JM, Monaghan W, Looke DF, Nimmo GR, Svanborg C. Virulence properties of asymptomatic bacteriuria Escherichia coli. International Journal of Medical Microbiology. 2009 Jan 1;299(1):53-63.
5. Roller S, editor. Natural antimicrobials for the minimal processing of foods. Elsevier; 2003 Jul 1.

6. Ravindran PN, Babu KN, editors. Ginger: the genus Zingiber. CRC press; 2016 Apr 19.
7. Ashok Kumar M.D, Gopinath P. Antibacterial activity of ginger oil against clinical isolates of Escherichia coli. International Journal of Scientific Development and Research. 2018;3(2):14-17.
8. Ishiguro K, Ando T, Maeda O, Ohmiya N, Niwa Y, Kadomatsu K, Goto H. Ginger ingredients reduce viability of gastric cancer cells via distinct mechanisms. Biochemical and biophysical research communications. 2007 Oct 12;362(1):218-23.
9. Yamada Y. Identification of antimicrobial gingerols from ginger (Zinglber officinale Roscoe). J. Antibact. Antifung. Agent. 1992;20:309-11.
10. Prasad MM, Seenayya G. Effect of spices on the growth of red halophilic cocci isolated from salt cured fish and solar salt. Food Research International. 2000 Jan 1;33(9):793-8.
11. Gopinath Prakasam, Manju Bhashini, Lakshmipriya, Srivani Ramesh S. 2014. In-vitro antibacterial activity of some essential oils against clinical isolates of Acinetobacter baumannii. Indian J Med Microbiol. 32:90-91.
12. Jain J, Tikare S, Mahuli A. Antifungal activity of ginger extract on Candida albicans: an in-vitro study. Journal of Dental Sciences and Research. 2011;2(2):18-21.
13. Atai Z, Atapour M, Mohseni M. Inhibitory effect of ginger extract on Candida albicans. American Journal of Applied Sciences. 2009;6(6):1067-9.
14. Wayne PA. National committee for clinical laboratory standards. Performance standards for antimicrobial disc susceptibility testing. 2002;12:01-53.
15. Anil S, Shukla YN, Sushil K. Recent development in plant derived antimicrobial constituents-a review. Journal of Medicinal and Aromatic Plant Sciences. 2000;22(2/3):349-405.
16. Sinha GK, Gulati BC. Antibacterial and antifungal study of some essential oils and some of their constituents. Indian perfumer. 1990;34(2):126-9.
17. Shuford JA, Piper KE, Steckelberg JM, Patel R. In vitro biofilm characterization and activity of antifungal agents alone and in combination against sessile and planktonic clinical Candida albicans isolates. Diagnostic microbiology and infectious disease. 2007 Mar 1;57(3):277-81.
18. Bonjar S. Evaluation of antibacterial properties of some medicinal plants used in Iran. Journal of ethnopharmacology. 2004 Oct 1;94(2-3):301-5.

19. Hammer KA, Carson CF, Riley TV. In-vitro activity of essential oils, in particular Melaleuca alternifolia (tea tree) oil and tea tree oil products, against Candida spp. The Journal of antimicrobial chemotherapy. 1998 Nov 1;42(5):591-5.
20. Lawless J. The illustrated encyclopedia of essential oils: the complete illustrated guide to the use of oils in aromatherapy and herbalism. Shaftesbury, Dorset, UK: Element. 1995.
21. Gupta S, Ravishankar S. A comparison of the antimicrobial activity of garlic, ginger, carrot, and turmeric pastes against Escherichia coli O157: H7 in laboratory buffer and ground beef. Foodbourne Pathogens & Disease. 2005 Dec 1;2(4):330-40.
22. Coutinho HD, Costa JG, Lima EO, Falcão-Silva VS, Siqueira-Júnior JP. Enhancement of the antibiotic activity against a multiresistant Escherichia coli by Mentha arvensis L. and chlorpromazine. Chemotherapy. 2008;54(4):328-30.

Assessment of Degree of Sexual Dimorphism using Diagonal Tooth Measurements

Fazeelath Banu M.A[1] , Sreedevi Dharman[2], Suresh.V[3]

Graduate Student[1],Senior Lecturer[2], Department of Oral Medicine and Radiology

Reader[3], Department of Prosthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Teeth are the structures which are highly resistant to higher temperatures, fragmentation and destruction. These characters make it possible to be used in various forensic and anthropological studies. In this study we use the mesiobuccal-distolingual (MBDL) and distobuccal-mesiolingual (DBML) crown diameters of maxillary canine and maxillary molar teeth on the study models measured using digital Vernier calipers.

Aim and objective: To study degree of sexual dimorphism in teeth of using crown diagonal diameters.

Materials and Method: Mesiobuccal-distolingual (MBDL) and distobuccal-mesiolingual (DBML) measurements of the crown and cervix of maxillary canine and maxillary molar were taken using digital vernier callipers in 120 upper dental casts obtained from 60 males and 60 females of the age group of 17-30 years.

Results: The stepwise discriminant functional analysis showed percentageof sexual dimorphism maximum for left canine crown MBDL, right canine crown MBDL and left canine crown DBML. Accuracy rate of gender determination is 68.3% in males and 65.0% in females.

Conclusion: Sex identification of a person with teeth remains is an inexpensive and easy method which is useful in forensic odontology. Diagonal measurements are preferred over linear measurements in case of attrition, crowding, malposition of teeth.

Key Words: *Sexual dimorphism, Diagonal measurements, Canine, Molar, Forensic odontology*

Introduction

Identification of sex of unrecognised skeletons is an important step in building the biological profile in forensics which helps in establishing identity of the individual [1] and one of the oldest approaches in forensic anthropology and medico-legal examinations [2]. Sexual dimorphism refers to a group of characteristics that differentiate male and female[3]. These are the differences in size, stature and appearance between male and female that can be applied to dental identification because no two mouths are alike[4].In massive disasters, even when other structures of body are destroyed, teeth are most often preserved[5].The unique pattern of tooth enables antemortem and postmortem analysis of dental variables.They provide higher degree of resistance to damage such as bacterial decomposition, fracture, fire etc. [6]

They are used in genetic, anthropological, forensic and odontologicalinvestigations as they are the hardest and chemically stable tissue in the body[7,8].

Tooth size has been evaluated in various populations in forensic and anthropological applications[3]. They show some differences in sexes and population[9] and is determined by cultural, environmental, genetic and racial factors[10].

Traditionally gender determination using tooth dimensions are determined using mesiodistal and buccolingual crown diameters of teeth. These dimensions get affected by attrition, cervical abrasion, crowding and presence of dental calculus in cervical third[11,12].As a result alternative dental measurements were developed to overcome these problems. Hilson et al., proposed diagonal diameters Mesiobuccal-distolingual crown diameter, Mesiolingual-distobuccal crown diameter, Mesiobuccal-distolingual cervical diameter and Mesiolingual-distobuccalcervical dimeters[13].

In this study diagonal measurements of maxillary canines and first molars are used. Canines are least affected than any other teeth by periodontal disease and are last teeth to be extracted. They have been found to be recovered from hurricanes and air disasters and withstand extreme conditions[14].

Molars are the first teeth to erupt in oral cavity. Hence they are available for sex assessment at an early age when compared to other permanent teeth[15].

The aim of this study is to determine the degree of sexual dimorphism using upper canines and first molars using diagonal measurements.

Materials and Methods

Study samples included 120 upper dental casts obtainedafter informed consent from 60 males and 60 females of the age group of 17-30 years in Chennai population.

Selection criteria: It included fully erupted right and left permanent maxillary canine and first molar, healthy state of periodontium, caries free teeth and with no history of orthodontic treatment. Any fillings, extractions, crowns, orthodontic anomalies that could affect the odontometric measurements were excluded from the study.

After taking informed consent, alginate (irreversible hydrocolloid) impressionsof the upper and lower jaws were taken followed by immediate preparation of their models with dental stone to prevent dimensional change.

Using the casts, Mesiobuccal-distolingual and Distobuccal-mesiolingual measurements were taken in dental casts using digital Vernier calliper of 0.01mm resolution.

Two crown and two cervical diagonal measurements of upper canine and first molar of right and left sides were taken. Each dimension was measured twice at different time interval by the same observer. After placing the calliper parallel to the occlusal surface, the following points were taken as guide during measurements[13,16].

Mesiobuccal-distolingual crown diameter: Defined as maximum distance from the mesiobuccal corner of the crown to the distolingual corner.

Mesiolingual-distobuccal crown diameter: Defined as maximum distance from the mesiolingual corner of the crown to the distobuccal corner

Mesiobuccal-distolingual cervical diameter: Defined as maximum distance from the mesiobuccal corner of cemento-enamel junction to the distolingual corner.

Mesiolingual-distobuccal cervical diameter: Defined as maximum distance from the mesiolingualcorner of cemento-enamel junction to the distobuccal corner.

The measurements were taken by one person. All values were rounded to two decimal places. All samples were analysed using student T test. Data obtained were further analysed using stepwise discriminant functional analysis.

Results

A total of 16 measurements of each cast was taken in this study. Four diagonal measurements of each tooth was taken with two cervical and two crown measurements of both the sides.

Each measurement was taken twice by the same observer at different intervals of time to check for inter observer error. There was no statistically significant difference between the first and second observations.

An independent two sample t-test was done to test whether males were significantly different from females.

Table 1 shows percentage of sexual dimorphism, P values, t values for MBDL and DBML crown and cervical diameters for all the teeth of both males and females included in the study. In MesiobuccalDistolingualdiameter,right canine crown, right molar crown, right molarcrown cervix, left canine crown, left molar crown, left molar crown cervix were statistically significant between males and females ($P < 0.05$)

In DistobuccalMesiolingualdiameter right canine crown,left canine crown, left molar crown and left molar crown cervix were statistically significant between males and females ($P < 0.05$).

Percentage of sexual dimorphism was maximum for left canine crown MBDL, followed by right canine crown MBDL, left canine crown DBML, and right canine crown DBML.

Accuracy of classification results was 68.3% for males and 65% females and the overall was 66.7% (Table 2)

Stepwise Discriminant Function Analysis

Discriminant functional analysis is used to determine which continues variables discriminate between two or more naturally occurring groups.

Discriminant function score (Z) for gender assessment = 0.084(RtCanCrMBDL) + 0.458(RtCanCrDBML) + 0.062(RtCanCerMBDL) – 1.230(RtCanCerDBML) + 0.493(RtMolCrMBDL) + 0.018(RtMolCrMBDL) – 0.279(RtMolCerMBDL) – 0.501(RtMolCerDBML) + 1.353(LtCanCrMBDL) + 0.282(LtCanCrDBML) – 1.135(LtCanCerMBDL) + 0.364(LtCanCerDBML) + 0.064(LtMolCrMBDL) + 0.202(LtMolCrDBML) + 0.603(LtMolCerMBDL) + 0.210(LtMolCerDBML) – 11.229

If the discriminant score is (Z) is below the sectioning point, individual is classified as female and if the score is above sectioning point, individual is classified as male.

Discriminant function analysis (Table 3) provides coefficients and sectioning points for each function to determine sex. The group centroids provides the average discriminant scores for each sex. Sectioning point is the average of male and female group centroids. The sectioning point for male and female was calculated to be 0.0. Raw coefficients are the discriminant function coefficients used to calculate the discriminant score.

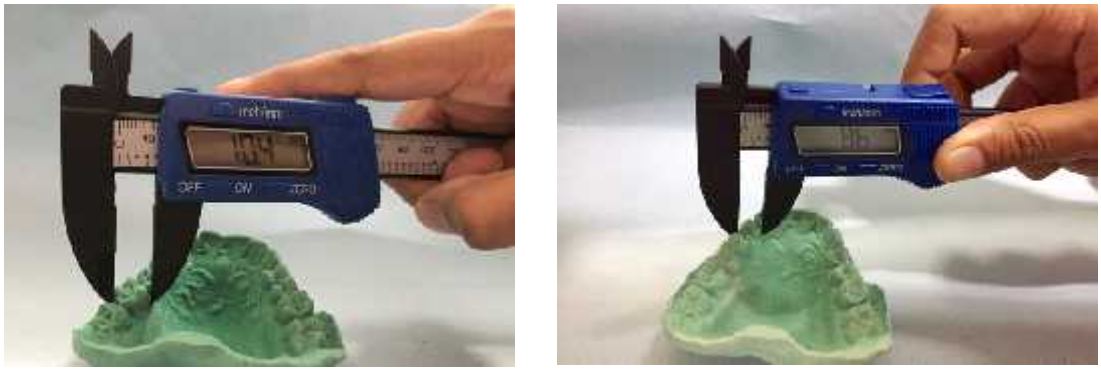


Figure 1: Recording diagonal measurements of molar and canine using vernier calipers

Table 1: Descriptive statistics, % sexual dimorphism, t values and P value

Measurements	Gender	N	Mean	Std. Dev	% of sexual dimorphism	t-Value	P-Value
RtCanCrMBDL	Male	60	7.4300	.71812	4.28	2.412	0.017
	Female	60	7.1250	.66603			
RtCanCrDBML	Male	60	6.9717	.64626	3.46	2.025	0.045
	Female	60	6.7383	.61537			
RtCanCerMBDL	Male	60	7.5067	.52169	1.90	1.432	0.155
	Female	60	7.3667	.54886			
RtCanCerDBML	Male	60	6.9700	.53908	0.77	0.542	0.589
	Female	60	6.9167	.53873			
RtMolCrMBDL	Male	60	11.9367	.71828	2.98	2.606	0.010
	Female	60	11.5917	.73214			
RtMolCrDBML	Male	60	10.2633	.78782	2.51	1.900	0.060
	Female	60	10.0117	.65719			
RtMolCerMBDL	Male	60	12.6917	.68180	2.41	2.442	0.016
	Female	60	12.3933	.65636			
RtMolCerDBML	Male	60	11.1333	.72968	1.69	1.431	0.155
	Female	60	10.9483	.68556			
LtCanCrMBDL	Male	60	7.4533	.67534	5.08	3.205	0.002
	Female	60	7.0933	.54861			
LtCanCrDBML	Male	60	6.9073	.60923	3.92	2.165	0.032
	Female	60	6.6467	.70602			
LtCanCerMBDL	Male	60	7.4633	.53677	1.98	1.537	0.127
	Female	60	7.3183	.49557			

LtCanCerDBML	Male	60	7.0000	.53043	2.41	1.805	0.074
	Female	60	6.8350	.46898			
LtMolCrMBDL	Male	60	11.9733	.67845	3.11	2.801	0.006
	Female	60	11.6117	.73510			
LtMolCrDBML	Male	60	10.2117	.74858	2.89	2.295	0.024
	Female	60	9.9250	.61331			
LtMolCerMBDL	Male	60	12.7733	.62460	2.65	2.831	0.005
	Female	60	12.4433	.65208			
LtMolCerDBML	Male	60	11.2483	.63339	2.47	2.363	0.020
	Female	60	10.9767	.62582			

Table 2: Accuracy of classification of sample

Accuracy of Classification Results		
Male	Female	Total
68.3%	65.0%	66.7%

Table 3: canonical discriminant function

Gender	Group Centroids	Sectioning
Male	0.434	0.0
Female	-0.434	0.0

Discussion

Sex determination is one of the important steps in building the biological profile of the skeletal remains [16].It is of significance in mass fatality cases where bodies are damaged beyond recognition. In situations where only jawbone fragments with teeth are found, sex determination is possible only with the help of teeth [17]. Teeth being resistant to postmortemdestruction,can be used as a tool of reconstructive identification particularly in cases of major calamity [10].

Even though DNA profile gives accurate results, measurement of linear and diagonal dimensions such as anthropometric or odontometric parameters can be used for sex determination in a large population because they are simple, reliable, inexpensive, and easy to measure [14].As most teeth complete development before skeletal maturation, dentition makes a valuable sex indicator especially in young individuals [18].

Teeth are the most durable structures and highly resistant to post-mortem insults in the body which make them to be used in various forensic odontological studies for sex determination. Since the other skeletal parameters like pelvis and skull used as an indicator of sex are destroyed or fragmented, dentition is used in gender determination[12,19,20].

It is known that differences in odontometric features are seen in specific populations and also within the same population. So it is necessary to determine specific population values to make identification possible based on dental measurements[21]. In the present study maxillary canine and first molar are used in the assessment of sex in Chennai population.

Linear measurements have been routinely used in forensics for gender determination. Previous study done with linear measurement showed mandibular canine more dimorphic, followed by maxillary canine and maxillary central incisors in all the three dimensions[22].Since linear measurements are affected by conditions like attrition, crowding, tooth rotations, any mesio-occlusal, disto-occlusal fillings, this prevents one from taking accurate linear measurements. In these situations diagonal measurements can be used in determining gender[13].It was proposed that one of the advantages of diagonal measurementsof molars is that the diagonal axes do not conincide the contact points of the crown [12].

In this study, the diagonal tooth measurements were higher in males than females. The results were in accordance with previous studies by Prabhu S et al[16], JL Anderson et al[23], MA Hashim et al[24].In mesiodistal dimensions Khangura *et al*. [25] and Howe *et al.*[26]found that of male dentition are greater than those of females.This could be because males typically have larger dentine and pulp dimensions than females, while marginal enamel thickness was similar in both sexes [27].

The canine crown measurement was found to be more sexually dimorphic than the other measurements taken in this study. In a study conducted by Hashim HA, Murshid ZA where they evaluated 720 teeth in Saudi population they found that canines were the only teeth to exhibit dimorphism[24]. In another study conducted in Japanese population Minzuno reported that maxillary canine showed a higher degree of sexual dimorphism compared to the mandibular canine [28].Otuyemi and Noarshowed dimorphism in maxillary canine bilaterally [29].

In a study done in Turkish population, diagonal measurements of canine showed greater accuracy rate with MBDL measurements being more reliable[30]. The percentage of sexual

dimorphism was maximum for MBDL crown diameter of left canine (5.08%) followed by MBDL crown diameter of right canine (4.28%) and least for DBML cervical dimeter of right canine (0.77). The results were different in previous studies which showed Cervical diagonal diameters of molars to be more sexually dimorphic than crown diagonal diameters [12,15].

After analysing with discriminant functional analysis it is seen that the accuracy rate of gender determination in this study was higher in males (68.3%) than females (65%). And the overall accuracy rate of this study is 66.7%. In a study by Rao *et al*. 88% accuracy of sex identification was reported [31].

It's always seen that tooth size or odontometricsis under considerable influence of the environment. Such measurements are, therefore, population specific, and do not apply to the world at large [32].

Conclusion

Forensic odontology is an inexpensive and easy way of identification of persons from dental remains. Teeth are considered as supplement and adjunct in sex identification and is not recommended as the sole indicator of sex.

Diagonal measurements can be taken in situations where linear measurements cannot be taken as in attrition, crowding and orthodontic anomalies.

To conclude MBDL and DBML crown and cervical diameters are reliable indicators and can be used in sex determination. In future studies, larger samples from diverse populations should be analysed to enhance the reliability of this parameter.

Corresponding Author

Suresh.V

Reader,Department of Prosthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai-600077,Tamil Nadu, India

Mobile: +91 9543192858

Email: sureshvbhat@gmail.com

Reference

1. Payal sharma, Tushita Singh, Puish kumar, Pavan kumar chandra, Rakesh sharma.Sex determination potential of permanent maxillary molar widths and cusp diameters in a North Indian population. J Orthod Sci. 2013; 2(2): 55–60.
2. J.D. Raj and S. Ramesh. Sexual Dimorphism in Mandibular Ramus of South Indian Population. Antrocom Online Journal of Anthropology 2013; 9(2):1973 – 2880.
3. Ayoub F. Journal of Forensic Odonto- Stomatology Dec 2007; 25(2).
4. Garn SM, Lewis AB, Kerewsky RS. The relationship between sexual dimorphism in tooth size and body size as studied within families. Arch Oral Biol 1967;12:299-301.
5. Ermenc B, Rener K. Possibilities for dental identification in the case of mass disaster in Slovenia. Forensic Sci Int. 1999;103(Suppl 1):S67–75.
6. Rai B, Dhattarwal SK, Anand SC. Sex determination from tooth. Med Leg Update. 2008;8:3–5.
7. Dayal PK, Srinivasan SV, Paravathy RP. Determination of Sex Using Tooth. 1st ed. Hyderabad: Paras Medical Publishers; 1998. Textbook of Forensic Odontology.
8. Kieser JA, Groeneveld HT. The unreliability of sex allocation based on human odontometric data. J Forensic Odontostomatol 1989;7:1-12.
9. Dempsey PJ, Townsend GC. Genetic and environmental contributions to variation in human tooth size. Heredity (Edinb) 2001;86:685–93.
10. M Malik. Sex determination by diagonal teeth measurements.Dissertation submitted to the Rajiv Gandhi Institute Of Health Sciences, Karnataka Bangalore 2008.
11. E Zorba, K Moraitis, C Elipoulos, C Spiliopoulou. Sex determination in modern Greeks using diagonal measurements of molar teeth. Forensic Science International 2012; 217:19-26.
12. S Hillson, C FitzGerald, Flinn Helen. Alternative Dental Measurements: Proposals and Relationships with Other Measurements. American Journal Of Physical Anthropology 2005; 126:413-26.
13. Kaushal S, Patnaik VV, Agnihotri G. Mandibular canines in sex determination. J Anat Soc India. 2003;52:119–24.
14. Ramandeep Singh Narang, Adesh S.Manchanda, and Balwinder Singh. Sex assessment by molar odontometrics in North Indian population. J Forensic Dent Sci. 2015 Jan-Apr; 7(1):54–58.

15. Ahmed B.R. Mujib, RatnaKumari V. N. Tarigoppula, Pavan G Kulkarni, Anil BS. Gender Determination Using Diagonal Measurements of Maxillary Molar and CanineTeeth in Davangere Population. Journal of Clinical and Diagnostic Research. 2014 Nov; 8(11):ZC141-ZC144.
16. Prabhu S, Acharya AB. Odontometric sex assessment in Indians. Forensic Sci Int. 2009;192:129.e1–5.
17. V Sonika, K Harshaminder, GS Madhushankari, J Kennath. Sexual dimorphism in the permanent maxillary first molar: a study of the Haryana population. J Forensic Odontostomatol 2011; 29(1):37-43.
18. Lund H, Mornstand H. Gender determination by odontometrics in a Swedish population. J Forensic Odontostomatol. 1999; 17:30-4.
19. Spradley MK, Jantz RL. Sex estimation in forensic anthropology: Skull versus postcranial elements. J Forensic Sci. 2011; 56:289–96.
20. Iscan YM, Kedici SP. Sexual variation in bucco-lingual dimensions in Turkish dentition. Forensic Sci Int. 2003;137:160–4.
21. Sreedevi Dharman, Gnanasundaram N, Maragathavalli Gopal, Arvind Muthukrishnan. Phenotypic differences in teeth dimensions among Chennai population: An aid in sex determination. Journal of Indian Academy of Oral Medicine and Radiology 2015; 27(2):171-177.
22. K Boaz, G Gupta. Dimorphism in maxillary and mandibular canines in establishment of gender. J of forensic Dental Sciences 2009; 1(1):42-44.
23. JL Anderson, GW Thompson. Interrelationship and sex differences of dental and skeletal measurements. J Dent Res 1973; 52:431.
24. MA Hashim, ZA Murshid. Mesiodistal tooth width: A comparison between Saudi males and females. Part 1 Egypt Dent J 1993; 39:343-46.
25. Khangura RK, Sircar K, Singh S, Rastogi V. Sex determination using mesiodistal dimension of permanent maxillary incisors and canines. J Forensic Dent Sci 2011;3:81-5.
26. Howe RP, McNamara JA Jr, O'Connor KA. An examination of dental crowding and its relationship to tooth size and arch dimension. Am J Orthod 1983;83:363-73.
27. Harris EF, Hicks JD, Barcroft BD. Tissue contributions to sex and race: Differences in tooth crown size of deciduous molars. Am J Phys Anthropol 2001; 115:223-37.
28. Minzuno O. Sex determination from maxillary canine by fourier analysis. Nihon Univ Dent J 1990;2:139-42.

29. Otuyemi OD, Noar JH. A comparision of crown size dimensions of the permanent teeth in a Nigerian and British population. Eur J Orthod 1996;18:623-8.
30. F Karaman. Use of diagonal teeth measurements in predicting gender in a Turkish population. Journal of Forensic Sciences 2006; 51(3):630-85.
31. Rao NG, Pai ML, Rao NN, Rao KT. Mandibular canine in establishing sex identity. J Indian Forensic Med 1988;10:5-12.
32. Gupta B, Gupta M. Sex identification in forensic odontology- a review of various methodology. Int J Forensic Odontol 2016; 1:9-13.

Assessment Of Mandibular Fractures Patterns In Orthopantogram- A Retrospective Study.

Janani.M[1] ,Sreedevi Dharman[2]

Graduate Student [1], Senior Lecturer [2], Department of Oral Medicine

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Background: Mandibular fracture is the most common site of fracture involved in facial bones following nasal bone. Despite many reports, about the incidence, diagnosis and treatment of mandibular fractures there is limited knowledge about the specific type or pattern of mandibular fractures in India. This study aims to evaluatethe percentage of fractures occurring in various regions of mandible and to examine the etiology of mandibular fractures using Orthopantomogram (OPG).

Objective: The etiology and pattern of mandibular fractures vary considerably among different study population. This study presents to delineate the etiology and predictable patterns of mandible using orthopantomogram. The reason for the present study is to correlate the etiology of mandibular fractures with anatomic location of fracture and to gain knowledge to guide surgeons for timely management of mandibular fractures.

Materials and methods: This study comprised radiographic evaluation of 70 mandibular fractured OPGS were taken for routine investigation. The type of mandibular fracture present in each OPG was identified and the cause for the fracture was retrospectively analysed. The results obtained were tabulated and recorded.

Result: The present study is done to evaluate the probability of occurrence of mandibular fractures in different anatomic location and identifying the type of mandibular fracture with proper history taking. Out of 70 fractured OPGs assessed, the most commonest site of fracture in mandible is parasymphysis region(34%) followed by body(15%),sub condylar(10%) and angle(2%) respectively. On tracking the medical history of patient, it was

found that most mandibular fractures occurred due to road traffic accidents(50%) and fall(35%). Condylar fractures are common due to physical force of injury.

Conclusion: In centres where OPG or extra oral radiograph facility is unavailable, a proper history taking with clinical examination could assist the clinicians for effective diagnosis and timely management of orofacial fractures.

Introduction

Injuries of the maxillofacial region represent one of the most arising health issue throughout India[1]. Maxillofacial injuries include soft tissue injuries, maxillary fractures, mandibular fractures and zygomatic fractures which are the common group of injuries treated by oral and maxillofacial surgeons [2]. The mandible is one of the most unique bone having a complex role in aesthetics of face and functional occlusion[3]. Because of the most prominent position of lower jaw in face, mandibular fractures tend to be the most common fractures of facial skeleton.[4] . In general, mandibular fractures have a higher adolescent and young adult age distribution and male predilection of occurrence which is attributed due to work in outdoor occupation thus becoming exposed to more Road Traffic Accidents(RTA)[5].The other causes of mandibular fractures include assaults, falls and the other miscellaneous causes include attack by animals, sporting injuries and industrial accidents[6]. Mandibular fractures can involve only one site or can often involve multiple regions simultaneously [7].

Therefore for each patient the combination of above mentioned factors determine the possibility of a mandibular fracture. Hence a clear understanding of different patterns and etiology of mandibular fractures is necessary for maxilla facial surgeons to plan the treatment for these kind of maxillofacial injuries.

This study aims to delineate the aetiology, frequency of anatomic distribution and the correlation between aetiology and anatomic location of fracture as an retrospective analysis.

Materials and methods

This study involves a retrospective analysis of medical records of patients who reported to Saveetha Dental College with the history of trauma wherein they are advised to take an OPG to detect the intensity and anatomic location of mandibular fracture. The medical records and

OPGs of these patients with facial trauma were retrieved and reviewed over the last two years. A total of 70 fractured OPGs were identified to have some form of mandibular fracture out of 1200 OPGs taken over a period of two years. The complete medical records of these 70 patients who had mandibular fractures were obtained that included case history, radiographs taken, photographs and if any surgical procedures performed were noted. Then data was analysed based on parameters of age, sex, mechanism of trauma, number and anatomic location of fractures.

Results

70 patients included in the present study were divided into groups based on age (1-10 years;11-20 years;31-40 years;41-50 years and 51-60 years) and according to sex male and female. Mechanism of trauma was recorded and classified as Road Traffic Accidents(RTA); fall; assault and miscellaneous. Anatomically the mandibular fractures were classified into Condylar; Coronoid; Sub condylar; Body ; Angle and Parasymphysis regions.

Out of 70 patient's OPGs assessed 50 were males(71.4%) and 20 were females(28.57%) with a male : female ratio of 20:1. The age of the patients ranged from 1 to 57 years with the mean of 27.09 years. The mean age formales was 26.39 years and for females it was 32.5years.

We found a peak occurrence of mandibular fractures in younger adults of age 21-30 years which was followed by 31-40 years,11-20 y3ears,41-50 years and 1-10 years of age group. Amongst the males the most common age of occurrence was 21-30 years while in females it was 31-40 years.

Incase of aetiology, RTAs were most common(34%) followed by fall(25%) which is in turn followed by assault(9%) which is followed by miscellaneous causes of(2%) respectively that included attack due to animals, industrial accidents etc. The data for causes of fractures categorized by gender showed that RTA(65% in males and 70% in females) was the most favourable etiological factor irrespective of gender. The second most commonest cause of fracture in males was fall(25%) and assault(9%) while in females the second most commonest cause was fall(30%) alone.

Amongst the anatomic location of mandible,the most frequently involved site is Parasymphysis region with(34%) of total mandibular fractures. This was followed by Body(15%);Sub condylar(10%);Angle (9%) and Condylar(2%) fractures respectively. The coronoid fractures are least common to occur which accounts for 0% of total mandibular fractures.

Out of patients with RTA, the parasymphysis fracture was predominant combination of fracture site involvement, followed by body. In case of fall patients the most common part of fracture in mandible was body followed by angle region whereas in case of patients with assault the most common region of fracture involved was Condylar and Sub Condylar region respectively.

Figure 1: Opg Showing Fracture In Angle Of Mandible

Figure 2: Opg Showing Fracture In Condyle And Parasymphysis Region In Mandible.

Figure 3: Opg Showing Fracture In Condyle And Sub Cobdylar Region In Mandible.

Figure 4: Opg Showing Fracture In Body Of Mandible.

Figure 5: Opg Showing Unfavourable Fracture Patterns.

Figurure 6: Opg Showing Fractures In Multiple Regions Of Mandible

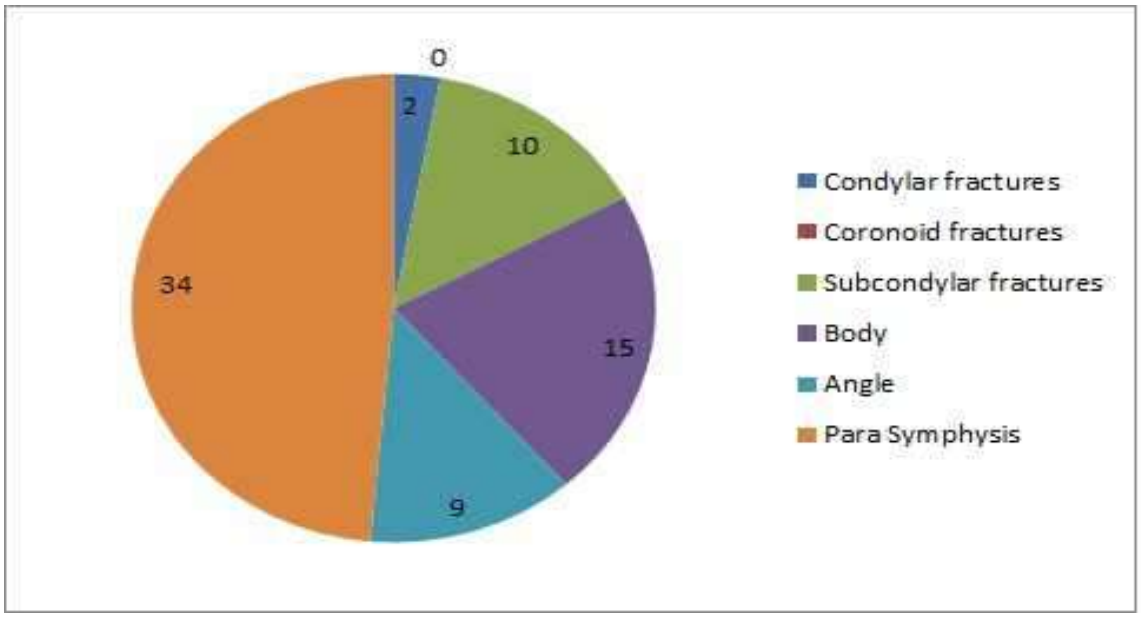


Figure 6: Distribution Of Different Types Of Fractures In Mandible (In %) According To Anatomic Location

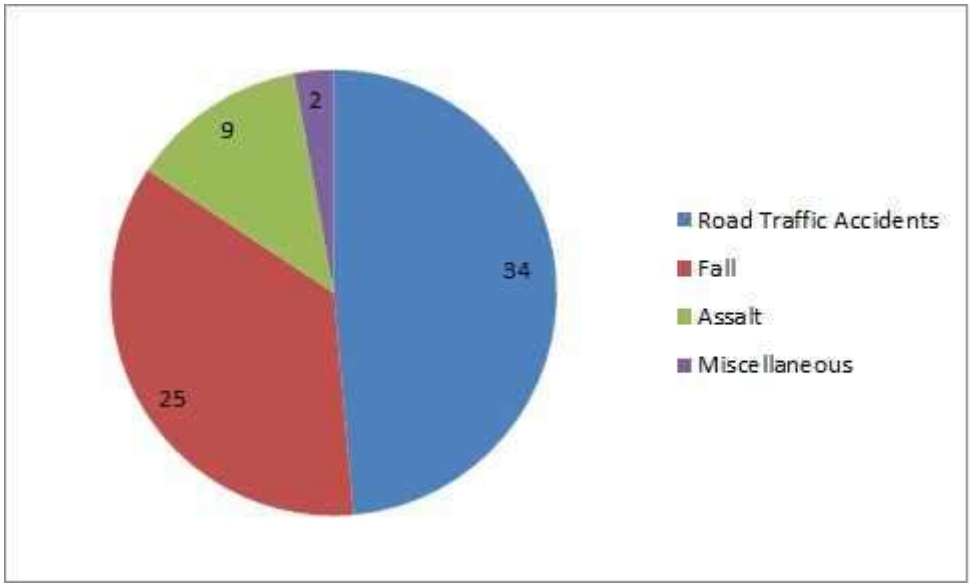


Figure 7: Distribution Of Etiology Of Mandibular Fractures (In %)

Discussion

Regardless of the geographic areas, injuries of body are more common to occur [9]. Maxillofacial injury occurs in approximately in 5-33% of patients experiencing severe trauma which is often associated with loss of function, disfigurement, severe morbidity and significant financial cost for management [10].

Amongst the facial bone injuries the second most common fractures tend to occur in mandible following nasal bone [11]. The mandible is the only facial bone that has mobility amongst the other facial bones [12]. Hence the fracture in this region is never left unnoticed because it is very painful, pain that worsens with masticatory and phonation movements [13]. The patient may also complain of facial asymmetry that may lead to deformation of face whichis caused by displacement of dental occulusion or Tempero Mandibular Joint [14]. If not identified and treated these lesions may lead to severe aesthetic and functional dysfunction [14].

Mandibular fractures occur in all ages and all races in a wide range of geographic boundaries [15]. This result of present study of patients with mandibular fractures, who were treated in Saveetha Dental College are largely in agreement with those previous reports, particularly with regard to age and gender of the patient.

The finding was that the age group 21-30 years constituted the highest frequency of mandibular jaw fractures. Trauma is considered as the most common cause of fracture in these age group as their aggressive nature and careless driving on roads(Figure No 8). It has been also estimated that there is a male predilection of mandibular fractures than compared to the females. This is due to the fact that this group is more prone to road traffic accidents since they drive vehicle carelessly and more likely to be involved in violence.

In present study, the most commonest site of fracture is parasymphysis region (Figure1) which is generally due to Road Traffic Accidents .They are followed by Body, Sub condylar fractures , Angle, Condylar fractures and coronoid fractures respectively. It is found that Condylar fractures are common due to physical force of injury. In addition to that Condylar fractures occurs in combination with parasymphysis fractures. In case of fall patients the most commonly involved site of fracture was parasymphsis followed by body and angle of mandible.

Mandibular fractures are common in all races and all age groups of people. It is hoped that such assessments as the one presented here will give a valuable piece of information to government agencies and healthcare professionals involved in planning future prevention of these kind of injuries.

Conclusion

On correlating the etiology with the anatomic site of mandible, a clear history taking is necessary by clinician for immediate management to elicit the area of fracture in mandibular region before an Orthopantomogram could be taken where in the patient is pshycologically affected by pain. Furthermore etiology also aids in planning of surgical treatment.

Since the main cause of these fractures proved to be Road Traffic Accidents, any efforts made to enforce traffic and safety rule in roads and improve traffic culture can be made effective to promote the present situation.

References

1. Haug RH, Prather J, Indresano AT. An epidemiologic survey of facial fractures and concomitant injuries. J Oral Maxillofac Surg. 1990;48:926–32. [PubMed]
2. Adebayo ET, Ajike OS, Adekeye EO. Analysis of the pattern of maxillofacial fractures in Kaduna, Nigeria. Br J Oral Maxillofac Surg. 2003;41:396–400. [PubMed]

3. Deogratius BK, Isaac MM, Farrid S. Epidemiology and management of maxillofacial fractures treated at Muhimbili National Hospital in Dar es Salaam, Tanzania, 1998-2003. Int Dent J. 2006;56:131–4. [PubMed]
4. Fasola AO, Obiechina AE, Arotiba JT. Incident and pattern of maxillofacial fractures in the elderly. Int J Oral Maxillofac Surg. 1990;28:200–2.
5. Thorn JJ, Møgeltoft M, Hansen PK. Incidence and aetiological pattern of jaw fractures in Greenland. Int J Oral Maxillofac Surg. 1986;15:372–9. [PubMed]
6. Scherer M, Sullivan WG, Smith DJ, Jr, Phillips LG, Robson MC. An analysis of 1,423 facial fractures in 788 patients at an urban trauma center. J Trauma. 1989;29:388–90. [PubMed] Brasileiro BF, Passeri LA. Epidemiological analysis of maxillofacial fractures in Brazil: A 5-Year prospective study. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2006;102:28–34. [PubMed]
7. Yokoyama T, Motozawa Y, Sasaki T, Hitosugi M. A retrospective analysis of oral and maxillofacial injuries in motor vehicle accidents. J Oral Maxillofac Surg. 2006;64:1731–5. [PubMed]
8. Shah AA, Salam A. Pattern and management of mandibular fractures: A study conducted on 264 patients. Pakistan Oral Dent J. 2007;27:103–5.
9. Sirimaharaj W, Pyungtanasup K. The epidemiology of mandibular fractures treated at Chiang Mai University Hospital: A review of 198 cases. J Med Assoc Thai. 2008;91:868–74. [PubMed]
10. Olasoji HO, Tahir A, Arotiba GT. Changing picture of facial fractures in northern Nigeria. Br J Oral Maxillofac Surg. 2002;40:140–3. [PubMed]
11. Subhashraj K, Ramkumar S, Ravindran C. Pattern of mandibular fractures in Chennai, India. Br J Oral Maxillofac Surg. 2008;46:126–7. [PubMed]
12. Al-Khateeb T, Abdullah FM. Craniomaxillofacial injuries in the United Arab Emirates: A retrospective study. J Oral Maxillofac Surg. 2007;65:1094–101. [PubMed]
13. Subhashraj K, Nandakumar N, Ravindran C. Review of maxillofacial injuries in Chennai, India: A study of 2748 cases. Br J Oral Maxillofac Surg. 2007;45:637–9. [PubMed]
14. Dongas P, Hall GM. Mandibular fracture patterns in Tasmania, Australia. Aust Dent J. 2002;47:131–7.[PubMed]

15. King RE, Scianna JM, Petruzzelli GJ. Mandible fracture patterns: A suburban trauma center experience. Am J Otolaryngol. 2004;25:301–7. [PubMed]

Assessment of Neurosensory Disturbances of Lingual Nerve Following Surgical Removal of Impacted Lower Third Molar

Bharadwaj.B [1], Kathiravan.S [2], Balakrishna R.N [3]

Graduate Student [1], Senior Lecturer [2], Department of Oral and Maxillofacial Surgery

Senior Lecturer [3], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: To determine the incidence of Neurosensory disturbances of lingual nerve following surgical removal of impacted lower third molar and compare the type of impaction to the incidence and duration of Neurosensory disturbances.

Materials and Methods: 20 patients were selected and surgical removal of impacted lower third molar was done using Conventional Ward's incision and elevating the buccal flap. Neurosensory disturbances of lingual nerve were recorded periodically over telephonic conversations with the patients.

Result: Out of 20 patients, two patients were diagnosed having prolonged Neurosensory disturbances in lingual nerve. It was found that one patient had recovered while another is still under follow up. Hence, the overall incidence rate was found to be 5%.

Conclusion: It can be concluded that neurosensory disturbances of lingual never mainly depends upon the anatomical variations of lingual nerve, surgeon's inexperience and the type of impaction.

Key Words: *Mucoperiosteal flap, Odontomy, Ward's incision, Paraesthesia, Lingual nerve*

Introduction

Impacted teeth can be defined as those teeth whose normal eruption is prevented by adjacent tooth, overlying bone or soft tissue, malpositioning and lack of space in the arch, or other impediments. Impacted mandibular 3rd molar is one of the most common findings which are detected on routine dental checkup. However the patient seeks treatment whenever there is pain, swellings or another discomfort [1].Although the overall complication rate is low and most complications are minor, third molar removal being most common procedure that those minor complications may be significant. Therefore, efforts made to minimise intraoperative or postoperative complications will have a significant effect in terms of enhancing patient Outcome [2].Mandibularthird molar is situated at the distal end of the body of the mandible where is connection with relatively thin ramus. In mandible, there is a weak region where the fracture can occur if the applied force exceeds the limit during impacted wisdom tooth elevation without preliminary and adequate removing of surrounding bone.Impacted mandibular third molar teeth are in close proximity to the lingual, inferior alveolar, mylohyoid, and buccal nerves. During surgical removal of impacted lower third molar, both lingual nerve and inferior alveolar nerve are at great risk of damage [3]. If the damage occurs it may result in serious complications. Majority of these injuries result in transient sensory disturbances. In some cases, permanent paraesthesia (abnormal sensation), hypoesthesia (reduced sensation), dysesthesia (unpleasant abnormal sensation) can occur. These sensory disturbances as a result of injury can cause irritation for the patient, may result in speech and mastication and thus adversely affect the patient’s quality of life. They also constitute as one of the most frequent causes of complaints and litigation [4].Surgical removal of impacted lower third molar is one of the most common procedures performed in maxillofacial surgery clinics. The surgical removal of impacted third molar is often associated with some complications like pain; swelling, bruising and trismus.These complications are usually unexpected and unacceptable for the patients who weren't warned before the procedure. Hence, incidences of neurosensory disturbances in Inferior alveolar nerve and lingual nerve are also noted commonly [5].The lingual nerve is morphologically very different from the inferior alveolar nerve. The lingual nerve being a posterior division of the mandibular nerve provides somatosensory innervation of the lingual mucosa through its wide range of mechanosensitive, nociceptive and thermosensitive afferent fibres. Jointly with the chorda tympani nerve fibres, a branch of the facial nerve, it provides information to the anterior two

thirds of the dorsum of the tongue and preganglionic parasympathetic innervation of submandibular and sublingual secretory glands [6]. Its location is medial and anterior to the inferior alveolar nerve in its passage between the medial pterygoid muscle and the ramus of the mandible, where the fibres meet the chorda tympani nerve. Lingual nerve then goes via the gingival sulcus of the lingual mucosa superficially to the surface of the gland and submandibular ganglion. It finally ends as a sublingual nerve located immediately below the mucosa of the tongue. During surgical removal of impacted lower third molar oral and maxillofacial surgeons should consider the following factors such as the lingual nerve's proximity to the third molar lingual cortical plate, separated from the cortex only by the periosteum and its variable anatomy [7]. The exact mechanism of lingual nerve damage during third molar surgery is controversial and amongst the most studies causes are lingual plate perforation and lingual flap trauma during ostectomy or tooth sectioning, usage of lingual flap retractor and supra-crestal incision because the nerve can be located in this region in some cases and may be sectioned.During surgical removal of impacted lower third molar the nerve is covered with a thin layer of soft tissue whereas inferior alveolar nerve runs within a bony canal[8]. When sectioned and seen the cut nerve ends retract apart, adjacent soft tissue is distorted and the nerve ends may become malaligned and trapped. Regeneration of axons across a gap will be less successful than if the nerve ends remain in apposition[9].Neurosensory disturbances in lingual nerve usually occurs because of depth of impaction, position of tooth, age of the patient, duration of procedure, surgeon's inexperience and raising lingual and distal Mucoperiosteal flap. Incidence of lingual nerve damage was found to be 0.9% in a study conducted by Carmichael FA et al and McGowan DA et al[10] and 1.1%, a study conducted by Jeevan Lata and Arunesh K. Tiwari[11]. This article aims at emphasising the importance of relation between the type of impaction, duration of procedure and neurosensory disturbances in the lingual nerve.

Materials and Methods

20 patients were selected from Oral and Maxillofacial Surgery Department of Saveetha Dental College. Healthypatients of age group between 18 and 50 were included and those who are medically compromised are excluded from our study.

The surgical removal of impacted lower third molar was done using conventional Ward's incision. To minimise the risk of neurosensory disturbances, only buccal flap was raised and

gutter in the distobuccal bone for maximum exposure of the tooth was performed. The bone removal was done with the help of motor-driven surgical bur under the constant irrigation of normal saline. Odontotomy procedure was done depending on the type of impaction and the path of removal of impacted tooth[12].Patient's discomfort was assessed by telephonic conversations after 6hours, 24 hours and 7days following surgical removal of impacted lower third molar. Patients complaining neurosensory disturbances were advised for regular follow up and observed until paraesthesia was resolved.

Results

Out of 20 patients, 6 patients were found to have neurosensory disturbances for more than 24 hours and 2 patients had it for more than 7days on postoperative evaluation. The overall incidence rate is 5% since a patient is still under follow up for recovery of neurosensory disturbances. However other factors may influence neurosensory disturbances the level and the type of impaction play a major role in determining the duration of neurosensory disturbances [13].

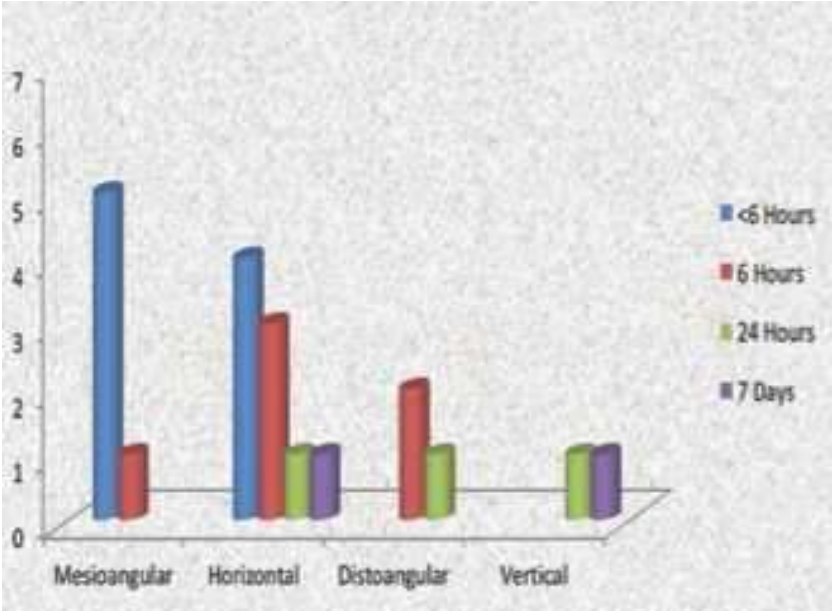
Table 1: Comparison of type of impaction and duration of neurosensory disturbances

Sl No	Age (Years)	Sex	Tooth Number	Type of Impaction	Duration of procedure (minutes)	Incidence & Duration of Neurosensory disturbances		
						6 Hours	24 Hours	7 Days
1	29	M	38	Mesioangular	14	Present	Absent	Absent
2	23	M	38	Mesioangular	11	Absent	Absent	Absent
3	29	F	48	Horizontal	16	Present	Absent	Absent
4	26	F	38	Horizontal	19	Absent	Absent	Absent
5	36	F	38	Vertical	22	Present	Present	Present
6	38	F	38	Horizontal	11	Absent	Absent	Absent
7	43	F	38	Mesioangular	10	Absent	Absent	Absent
8	39	M	38	Mesioangular	22	Absent	Absent	Absent
9	24	F	38	Distoangular	13	Present	Present	Absent
10	52	F	48	Horizontal at Root Apices of adjacent molar	45	Present	Present	Present
11	34	M	38	Horizontal	11	Present	Absent	Absent
12	21	M	38	Horizontal	9	Absent	Absent	Absent
13	45	M	48	Mesioangular	15	Absent	Absent	Absent
14	25	F	38	Distoangular	25	Present	Absent	Absent
15	20	M	48	Horizontal	15	Present	Absent	Absent
16	26	M	38	Distoangular	16	Present	Absent	Absent
17	26	M	38	Vertical	17	Present	Present	Absent
18	30	M	38	Horizontal	11	Present	Present	Absent
19	30	M	48	Mesioangular	9	Absent	Absent	Absent
20	45	M	38	Horizontal	8	Absent	Absent	Absent

Distribution of Type of Impaction

Out of 20 patients, most common type of impaction was horizontal(9) and mesioangular(6) impactions while vertical(2) and distoangular(3) cases have been recorded.

Graph 1: Comparison of type of impaction and duration of neurosensory disturbances



Discussion

The surgical removal of impacted mandibular third molars is the most commonprocedure performed by oral and maxillofacial surgeons. Sometimes surgeons may face various complications associated with the surgical removal of impacted mandibular 3rd molars. Among these complications most common postoperative complication is neurosensory deficit. It may affect either the inferior alveolar nerve or more commonly the lingual nerve that leads to numbness of the ipsilateral anterior two-thirds of the tongue and taste disturbance [14].

Neurosensory disturbances in lingual nerve are one of the most common complications experienced by dental surgeons in surgical removal of impacted lower third molar. They can have many serious psychological and legal implications [15]. The factors that contribute to these complications are type of impaction, depth of impaction, duration of procedure, sex and age of the patient.In our study, patient's age ranged from 20 to 55 years. Out of 20 patients, 60% of them were male and 40% were female. It had been found that more difficult the type of impaction more is the neurosensory disturbance. The overall incidence rate was found to

be 5% which is higher when compared to other studies. Initially Rood (1983) reported an initial incidence of 6.6%of neurosensory disturbances in lingual nerve[16]. In another study by Zuniga, the incidence of permanent injury to the IAN and LN has been mentioned to fall in the range between 0.4% and 25% and 0.04% and 0.6%, respectively [17]. In 2004, Tay and Go carried out a research study to determine the incidence of inferior alveolar nerve paraesthesia patients. In that study, an exposed inferior alveolar nerve bundle wasseen during third molar surgery and they concluded that such a situation indicates a high probability of an intimate relationship of the nerve with the tooth and thus carried a 20% risk of paraesthesia with a 70% chance of recovery by one year from surgery [18].Recently Cheung et al. carried out a study in which it was seen that of all the lower third molar extractions performed by various grades of operators, 0.35% developed IAN deficit and 0.69% developed LN deficit[19]. On comparing with radiological studies it has been concluded that mesioangular impactions are most common type and on surgical removal of those molars seldom causes paraesthesia [20]. It concluded that distoangular impaction was found to increase the risk of LN deficit significantly, wherein the depth of impaction was related to the risk of IAN deficit. It was also found that sex, age, raising of a lingual flap, protection of LN with a retractor, removal ofdistolingual cortex, tooth sectioning, and difficulty in tooth elevation were significantly related to IAN or LN injury.

Conclusion

Mandibular third molar extraction is a very commonly carried out procedure in day-to-day dental practice and is undoubtedly associated with few risks especially neural injuries and therefore in the light of the existing evidence, adequate preoperative evaluation of the patient and meticulous surgical technique with minimum handling of the lingual flap are of paramount importance to diminish the incidence of nerve injury. Although third molar surgery is a secure and low morbidity procedure, the risk of complications will always exist and it increases with increased surgical difficulty; hence, the patient should always be educated about the risks and benefits of surgery in order to ensure adequate surgical management of impacted mandibular third molar.

The lingual nerve injury after third molar removal is an important complication to consider before subjecting the patient to the intervention due to its remarkable incidence, its unpredictable cause and the significant discomfort it can generate. Depending on the severity

or chronicity of the injury, everyday life can be altered considerably. It is therefore vital to know the different risk factors involved in order to minimize the possible damage and report in detail to the patient before surgery to avoid medico-legal issues.

From our study and review of literature, it can be concluded that lingual nerve paraesthesia can occur with or without reflection of lingual flap and in spite of all the measures taken to protect it. It may be contributed to the fact of anatomical variations of lingual nerve. However if on clinical examination or radiographic presentation, it is pre-assessed that lingual nerve can be injured during surgical procedure, it should be well explained to the patient to avoid any legal litigation.

Corresponding Author

Balakrishna R.N

Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

E-mail Id – balakrishdr8394@gmail.com

Mobile number: 8939896729

References

1. Shehanaz A. The incidence of types of mandibular third molar impactions in different skeletal face types: A clinical study. Int J Orofac Res. 2017; 2(1):24.
2. Rothamel D, Wahl G, d'Hoedt B, Nentwig GH, Schwarz F, Becker J. Incidence and predictive factors for perforationof the maxillary antrum in operations to remove upper wisdom teeth: prospective multicentre study. Br J Oral Maxillofacial Surg. 2007; 45(5):387-91.
3. Benediktsdóttir IS, Wenzel A, and Petersen JK, Hintze H. Mandibular third molar removal: risk indicators for extended operation time, postoperative pain, and complications. Oral Surg Oral Med Oral Pathol Oral Radiol Endod2004; 97(4):438-46.

4. Valmaseda-Castellón E, Berini-Aytés L, Gay-Escoda C. Inferior alveolar nerve damage after lower third molar surgical extraction: a prospective study of 1117 surgical extractions.Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001; 92(4):377-83.
5. Robinson PP, Loescher AR, Smith KG. A prospective, quantitative study on the clinical outcome of lingual nerve repair.Br J Oral Maxillofacial Surg. 2000; 38(4):255-63.
6. Pogrel MA, Renaut A, Schmidt B, Ammar A. The relationship of the lingual nerve to the mandibular third molar region: an anatomic study. J Oral Surg. 1995; 53(10):1178-81.
7. Grau-Manclús V, Gargallo-Albiol J, Almendros-Marqués N, Gay-Escoda C. Mandibular fractures related to the surgical extraction of impacted lower third molars: a report of 11 cases. J Oral Surg. 2011; 69(5):1286-90.
8. Smith AC, Barry SE, Chiong AY, Hadzakis D, Kha SL, Mok SC, Sable DL. Inferior alveolar nerve damage following removal of mandibular third molar teeth. A prospective study using panoramic radiography. Aus Dent J. 1997; 42(3):149-52.
9. Moss CE, Wake MJ. Lingual access for third molar surgery: a 20-year retrospective audit. Br J Oral Maxillofacial Surg. 1999; 37(4):255-8.
10. Carmichael FA, McGowan DA. Incidence of nerve damage following third molar removal: a West of Scotland Oral Surgery Research Group study.Br J Oral Maxillofacial Surg. 1992; 30(2):78-82.
11. Lata J, Tiwari AK. Incidence of lingual nerve paraesthesia following mandibular third molar surgery. Natl J Maxillofac Surg. 2011; 2(2):137.
12. Gomes AC, do EgitoVasconcelos BC, e Silva ED, da Silva LC. Lingual nerve damage after mandibular third molar surgery: a randomized clinical trial. J Oral MaxillofacSurg. 2005; 63(10):1443-6.
13. Juodzbalys G, Daugela P. Mandibular third molar impaction: review of literature and a proposal of a classification.J Oral MaxillofacRes. 2013; 4(2).
14. McGurk M, Haskell R. Wisdom tooth removal and lingual nerve damage.Br J Oral Maxillofac Surg. 1999; 37(4):253-4.

15. Kim JC, Choi SS, Wang SJ, Kim SG. Minor complications after mandibular third molarsurgery: type, incidence, and possible prevention.Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2006; 102(2):e4-11.
16. Rood JP. Permanent damage to inferior alveolar and lingual nerves during the removal of impacted mandibular third molars. Comparison of two methods of bone removal. Br Dent J 1992; 17(2):108-110.
17. Zuniga JR. Management of Third Molar–Related Nerve Injuries: Observe or Treat? Alpha Omegan. 2009; 102(2):79-84.
18. Tay AB, Go WS. Effect of exposed inferior alveolar neurovascular bundle during surgical removal of impacted lower third molars.J Oral Surg. 2004; 62(5):592-600.
19. Kumar SM. Efficacy ofarticaine over lidocaine–A review. J Pharm Sci Res. 2015; 7:956-1.
20. Kumar MS, Aysha S. Angulations of impacted mandibular third molar: a radiographic study in Saveetha Dental College. J Pharm Sci Res. 2015; 7:981-3.

Association between Serum TriglyceridesLevel and Type 2 Diabetes

Vidhya.T.K[1],Naufal Rizwan[2],Saravana Pandian[3],

Graduate Student[1],Senior Lecturer[2], Department of General Medicine,Senior Lecturer[3], Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The association between changes in triglycerides concentration over time and diabetics is unknown. The aim of this work is to find out an association between serum triglycerides level and type 2 diabetics.

Material and Methods: 50 type 2 diabetic patients are examined at saveetha medical college and serum triglyceride is measured in these patients.Triglycerides are bundle of fat found in bloodstream. The normal triglyceride level is around 100 to 150mg/dl. In diabetic patients triglyceride level was found to be elevated. The presence of hyperglycaemia stimulates the production of triglycerides and hence triglycerides levels are usually high in type 2 diabetes. The combination of high triglycerides, low HDL and central obesity are the hallmarks of the metabolic syndrome, which occurs in 80 percent of people with type 2 diabetes.

Result: Of the 50 patients examined, 41(82%) patients were found to have high serum triglyceride level. Lifestyle interventions such as diet, physical activity, weight loss, and smoking cessation are the integral part of diabetic management plan. There are several classes of medications used in the treatment of lipid and lipoprotein abnormalities associated with insulin resistance and type 2 diabetics.

Conclusion: In this study, there is a significant correlation between glycemic control and triglyceride levels in patients with type 2 diabetes in this population. Familiarity with this concept helps to diagnose lipid abnormalities in patients with poor glycemic control and preventing cardiovascular events in the high-risk patient.The raised TG levels leads to increased blood viscosity and altered fibrinolytic activity which leads to formation of hard exudates.

Key Words:*Triglycerides, Diabetic, Blood, Viscosity,Glycemia.*

Introduction

Type 2 diabetes mellitus is one of the prevalent diseases increasing health burden in both developed and underdeveloped countries. Prevalence of diabetes is noted to be higher in Asians (people from Pakistan, India, and China) as compared to Caucasians [1-2]. The significant factors responsible for this outstanding prevalence in Asians as compared to Caucasians are the sedentary lifestyle changes, rapid urbanization & adoption of industrialized food culture which leads to obesity and insulin resistance. In Pakistan, a recent study showed that the prevalence of non-communicable diseases like type 2 diabetes mellitus has doubled as compared to data collected in 2004. The health burden of diabetes in some of the semi urban area was found to be 14.6% [3]. Diabetes Mellitus is an array of metabolic dysfunction secondary to decreased insulin secretion or insulin resistance. The common threat of this disease is poor glycemic control predisposing to micro- and macro-vascular complications. Micro vascular complications include neuropathy, retinopathy, and nephropathy. Macro vascular complications are coronary artery and peripheral artery disease. Type 2 diabetes is linked with high cardiovascular morbidity and mortality. Cardiovascular risk in diabetes is determined by dyslipidemia and hypertension [4-5]. Dyslipidemia is one of the common condition associated with a poor glycemic control in type 2 DM. The pathogenesis of dyslipidemia in type 2 DM is a decrease in activity of lipoprotein lipase due to insulin deficiency or resistance. Under the action of insulin, enzyme lipoprotein lipase metabolizes lipids in a healthy individual. In type 2 DM, the relative insulin deficiency and decreased adiponectin causes decrease lipoprotein lipase activity resulting in high levels of low-density lipoprotein (LDL), triglyceride and low levels of high-density lipoprotein (HDL). Qualitative defects in LDL are also seen in type 2 diabetes including atherogenic, glycated or oxidized LDL further amplifying the risk of atherogenesis. [6-7]. Dyslipidemias is one of the modifiable risk factors for coronary artery disease in type 2 diabetes. Atherogenic or diabetic dyslipidemia is defined by a profile of low- and high-density lipoprotein and high triglycerides. It is an independent predictor of coronary artery disease or silent myocardial ischemia [3,8]. High triglycerides can be dictated by many factors including genetic or acquired [9]. To rule out other causes, we only include patients without any familial dyslipidemia or history of alcohol intake. As these two factors also play a role in increasing triglycerides as compared to polygenic etiologic like obesity, insulin resistance or diabetes mellitus [10-11]. The literature review was evident that hypertriglyceridemia is linked to high

glucose levels and increased risk of type 2 diabetes [12-13]. In our study, correlation of HbA1c with high triglycerides signifies HbA1c as a direct marker of hypertriglyceridemia and an indirect marker of risk assessment of coronary artery disease. It is important to understand the concept of insulin resistance and dyslipidemia predisposing to atherosclerosis [14-15]. Cholesterol lowering through secondary prevention by lifestyle changes or statin therapy has tremendously improved cardiac outcome in diabetes [5].

Type 2 Diabetes

Diabetes is a problem with your body that causes blood glucose (sugar) levels to rise higher than normal. This is also called hyperglycemia. Type 2 diabetes is the most common form of diabetes. If you have type 2 diabetes your body does not use insulin properly. This is called insulin resistance. Diabetes mellitus type 2 (also known as type 2 diabetes) is a long-term metabolic disorder that is characterized by high blood sugar, insulin resistance, and relative lack of insulin.[6] Common symptoms include increased thirst, frequent urination, and unexplained weight loss. Symptoms may also include increased hunger, feeling tired, and sores that do not heal. Often symptoms come on slowly. Long-term complications from high blood sugar include heart disease, strokes, diabetic retinopathy which can result in blindness, kidney failure, and poor blood flow in the limbs which may lead to amputations. The sudden onset of hyperosmolar hyperglycemic state may occur; however, ketoacidosis is uncommon. Type 2 diabetes primarily occurs as a result of obesity and lack of exercise. Some people are more genetically at risk than others. Type 2 diabetes makes up about 90% of cases of diabetes, with the other 10% due primarily to diabetes mellitus type 1 and gestational diabetes. In diabetes mellitus type 1 there is a lower total level of insulin to control blood glucose, due to an autoimmune induced loss of insulin-producing beta cells in the pancreas. Diagnosis of diabetes is by blood tests such as fasting plasma glucose, oral glucose tolerance test, or glycated haemoglobin (A1C). Type 2 diabetes is partly preventable by staying a normal weight, exercising regularly, and eating properly. Treatment involves exercise and dietary changes. If blood sugar levels are not adequately lowered, the medication metformin is typically recommended. Many people may eventually also require insulin injections. In those on insulin, routinely checking blood sugar levels is advised; however, this may not be needed in those taking pills. Bariatric surgery often improves diabetes in those who are

obese.Rates of type 2 diabetes have increased markedly since 1960 in parallel with obesity. As of 2015 there were approximately 392 million people diagnosed with the disease compared to around 30 million in 1985.(7,8)Typically it begins in middle or older age, although rates of type 2 diabetes are increasing in young people. Type 2 diabetes is associated with a ten-year-shorter life expectancy. Diabetes was one of the first diseases described. The importance of insulin in the disease was determined in the 1920s. The development of type 2 diabetes is caused by a combination of lifestyle and genetic factors. While some of these factors are under personal control, such as diet and obesity, other factors are not, such as increasing age, female gender, and genetics. (9,10) Management of type 2 diabetes focuses on lifestyle interventions, lowering other cardiovascular risk factors, and maintaining blood glucose levels in the normal range.

Serum Triglyceride

Triglycerides are a type of fat in your blood that is formed when you consume more calories than you need. Our body uses these fats for energy in between meals, storing them in your fat cells until they are needed. Normal triglyceride levels are less than 150 milligrams per decilitre. High triglycerides tend to show up along with other problems, like high blood pressure, diabetes, obesity, high levels of "bad" LDL cholesterol, and low levels of "good" HDL cholesterol (11,12). So it’s hard to know for sure which problems are caused by high triglycerides alone. Triglycerides are important organic compounds. Most of the fat we consume in our diet is triglyceride and so too is most of the fat we store in our body. Fatty acids contained in triglycerides are an essential source of energy for our cells. Triglyceride concentration can be measured in blood and may provide valuable information about metabolism and general health.(13) High levels may reflect underlying metabolic disorders and evidence shows that high blood triglycerides are associated with increased risk of heart disease. Making changes to our lifestyle can have a dramatic benefit. Some suggestions: Get more physical activity. Exercise can have a big impact on triglyceride levels. Experts recommend that everybody get at least 30 minutes of exercise at least five times a week. If you're out of shape, start slowly. Begin with a quick walk three times a week and then build up from there. Lose some weight. If you're heavy, shed a few pounds and try to maintain an ideal body weight. Exercise will help, but you also need to focus on diet. The key is to eat

fewer calories -- whether they come from fats, carbs, or protein. Focus on a diet that's high in fruits, vegetables, lean proteins, and low-fat dairy products. Cutting down on sugary foods -- like sodas -- could really help, too.Choose better fats. Pay more attention to the fats you eat. Eat fewer foods with unhealthy fats (found in meat, butter, and cheese) and trans fats (in processed foods and margarines), as well as cholesterol. Boost your intake of healthy monounsaturated and polyunsaturated fats, which are found in olive oil, nuts, and some fish. Studies have found that the omega-3s in fatty fish - like tuna, salmon, mackerel, and sardines -- are particularly good at lowering triglyceride levels. Because even healthy fats are high in calories, you still need to eat these foods in moderation.Cut down on alcohol. Even small amounts of alcohol seem to cause big spikes in triglyceride levels. Limit alcohol intake to one drink a day.(15) Triglycerides are a type of fat (lipid) found in your blood. Your liver makes triglycerides. You also get them through many of the foods you eat. In fact, after you eat, any extra calories that your body doesn't need right away are converted into triglycerides. These fatty substances are stored in fat cells. They are later released and circulate the bloodstream to be used as energy by cells. But as with LDL cholesterol, too much can be harmful. Most dietary fats are triglycerides. (14)

Material and Methodology

In this cross-sectional analytical study, we included 50 consenting patients of either gender from outpatient diabetes clinic Medical Unit(Saveetha Medical College), suffering from type 2 diabetes. Our age limit was 45 and above. Demographic information was collected from all patients included age, gender, body mass index (BMI), active tobacco use, active alcohol use, hypertension status, employment status and marital status. Relevant medical history including the presence of the type of diabetes, hypertension, pulmonary comorbidities, cardiac comorbidities (i.e. arrhythmia, myocardial infarction and coronary artery disease), thyroid abnormalities and the presence of familial dyslipidemia was also obtained. Our exclusion criteria were patients with missing data, taking statins or suffering from renal, hepatic, cardiac or thyroid diseases, and familial hypercholesterolemia.

Results

Of the 50 patients examined, 41(82%) patients were found to have high serum triglyceride level. Lifestyle interventions such as diet, physical activity, weight loss, and smoking cessation are the integral part of diabetic management plan. There are several classes of medications used in the treatment of lipid and lipoprotein abnormalities associated with insulin resistance and type 2 diabetics.

Discussion

Diabetes is a multifactorial disorder having a wide range of lipid abnormalities. In type 2 diabetes mellitus, there is an increased incidence of hypertriglyceridemia as compared to other lipid abnormalities [16-17]. This study evaluated the correlation between glycated hemoglobin (HbA1c) and triglycerides level and the results showed that there is a significant correlation between high HbA1c and high triglyceride. This may in turn help in predicting the triglyceride status of type 2 diabetics from the degree of glycemic control and therefore identifying patients at increased risk from cardiovascular events [18]. Diabetic dyslipidemia is associated with microvascular and macrovascular complications of diabetes, as has been shown in the studies conducted past few decades. High TG/HDL ratio in diabetic state is a significant risk factor for arterial stiffness and carotid atherosclerosis. Hence, atherosclerosis (due to high Triglycerides or TG/HDL) may contribute to the development of DKD. Hence, clinical efforts should be done to reduce triglycerides and to elevate HDL-C to reduce diabetic related complications contributed by dyslipidemia. Excretion of albumin (or microalbumin) in the urine is an early marker of renal dysfunction or damage in diabetes, causing proteinuria or nephropathy, which may lead to stage renal disease (ESRD) with high incidence .

Lebovitz suggested that there is a lipotoxic mechanism by triglyceride which interferes with gastric or neural pathway which regulates glycemic control [19]. In most of the studies, there is a correlation found between glycemic control and dyslipidemia [20]. In a recent study, it was evident that there was a positive correlation between HbA1c and high triglycerides and HbA1c can be used as a potent marker for dyslipidemia and mitigate the macro- and micro-vascular complications of disease [21]. Diabetes is an independent risk factor for developing

cardiovascular risk. Cardiovascular events are also the most common cause of death in diabetes [22-23]. Gluco-centric medications might help in improving glycemic control but their role in preventing cardiovascular disease is limited. According to the records mandated by American Association of Clinical Endocrinology (AACE). In 2016, approximately 660,000 United States residents will have a new coronary event as myocardial infarction or atherosclerotic cardiovascular event (ASCVD). It further mentions dyslipidemia as major risk factor for ASCVD. The contribution of triglycerides to CVD risk has been much debated in the past, with many important prospective studies observing an association between elevated triglycerides levels and CVD risk (in particular coronary risk), but also risk for microangiopathy, nevertheless, more recent studies gave different results, suggesting that this association might be weakened when adjustment for other risk factors was made. The pathogenetic mechanism undergoing these findings remains uncertain, and could be mainly related to triglycerides induced endothelial dysfunction through oxidative stress. By Cox analysis we demonstrated a direct association between long-term mortality risk and triglycerides levels; the association was strong and significant even after multivariate adjustment for traditional CVD risk factors including BMI, HbA1c, LDL-C, and medication use. This independent association with long term all-cause mortality support the idea that serum triglycerides could play a role in type 2 diabetic patients mortality risk.

Chronic complications of DM includes macro vascular complications like coronary artery disease, cerebrovascular disease and peripheral vascular disease along with micro vascular complications like retinopathy, nephropathy and neuropathy. Risk factors like duration of diabetes, glycaemic control (HbA1c),systolic blood pressure, dyslipedemias, smoking and microalbuminurias have been linked with complications of DM. Various studies have shown a positive correlation between elevated serum lipids (TG, LDL,TC) and macro vascular complications like ischemic heart disease. However, studies of association of elevated serum lipids with micro vascular complications like diabetic retinopathy (DR) have shown varying results. Serum lipids were measured in the fasting blood sample. Serum triglycerides (TG) and cholesterol were measured by enzymatic methods, Glycerol 3 phosphate oxidase N-ethyl sulphopropyl anisidine and cholesterol oxidase-peroxidase end point methods respectively. Serum high density lipoprotein (HDL) was estimated by precipitation method Polyethylene glycol precipitation method. Serum low density lipoprotein (LDL) was calculated using Fried

Wald's formula (LDL=TC-HDL-TG/5). TG is associated with the increased incidence of diabetic retinopathy.

Conclusion

In this study, there is a significant correlation between glycemic control and triglyceride levels in patients with type 2 diabetes in this population. Familiarity with this concept help to diagnose lipid abnormalities in patients with poor glycemic control and preventing cardiovascular events in the high-risk patient.The raised TG levels leads to increased blood viscosity and altered fibrinolytic activity which leads to formation of hard exudates. Also, TG incorporates into the cell membrane, altering its fluidity and permeability which leads to haemorrhage and oedema. This also leads to endothelial cell dysfunction and local inflammatory response releasing cytokines and growth factors which are responsible for neovascularisation in retina. So this article relates the risk of increased triglyceride level in diabetic patients.

Corresponding Author

Saravana Pandian,

Senior Lecturer, Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id- sporthoresearch@gmail.com

Telephone Number: +91 9003101627

References

1. Ozer PA, Unlu N, Demir MN, Hazirolan DO, Acar MA,Duman S: Serum lipid profile in diabetic macular edema.*Journal of Diabetes and its Complications*, 2009;23(4): 244-248.
2. People's Journal of Study of Association of Serum Lipids with Diabetic Retinopathy in Type 2 Diabetes Mellitus- A. Mathur & R. Mathur Scientific Research Vol. 6(1), Jan. 2013 28
   244-248.
3. Rema M, Srivastava BK, Anitha B, Deepa R, Mohan V: Association of serum lipids with diabetic retinopathy in urban South Indians-the Chennai Urban Rural Epidemiology Study (CURES) Eye Study-2. Diabetic Medicine, 2006;23(9):1029-1036
4. Gyesi A. Living a healthy and normal life with diabetes. In: Health care tit-bits. Accra: Health Care Services Limited; 1992.
5. Amoah AG, Owusu SK, Adjei S. Diabetes in Ghana: a community based prevalence Study in greater Accra. Diab Res Clin Pract.2002; 56(3):197-205.
6. Harris M I: Undiagnosed NIDDM: Clinical and public health issues. Diabetes Care. 1993; 16: 642- 652.
7. Lyons TJ, Jenkins AJ, Zheng D, Lackland TD, Mc Gee D, Garvey TW, Klein LR. The DCCT/EDIC Research Group. Diabetic retinopathy and serum lipoprotein subclasses in the DCCT/EDIC cohort. Invest Ophthalmol Vis Sci. 2004;45: 910–918.
8. Rema M, Srivasta BK, Anitha B, Deepa R, Mohan V. Association of serum lipids with diabetic retinopathy in Urban South Indians-the Chennai Urban Rural Epidemiolog Eye Study (CURES). Diabetic Med. 2006; 23:1029-1036.
9. Mahalle N, Garg MK, Naik SS, Kulkarni MV. Study of pattern of dyslipidemia and its correlation with cardiovascular risk factors in patients with proven coronary artery disease. Indian journal of endocrinology and metabolism. 2014 Jan;18(1):48.
10. Rahman S, Kumar P, Mahto SK, Tonk RS, Taneja RS. Light Chain Myeloma induced Severe Hypertriglyceridemia. Journal of clinical and diagnostic research: JCDR. 2017 Mar;11(3):OD01.
11. Kushner PA, Cobble ME. Hypertriglyceridemia: the importance of identifying patients at risk. Postgraduate medicine. 2016 Nov 16;128(8):848-58.

12. Quispe R, Martin SS, Jones SR. Triglycerides to high-density lipoprotein–cholesterol ratio, glycemic control and cardiovascular risk in obese patients with type 2 diabetes. Current Opinion in Endocrinology, Diabetes and Obesity. 2016 Apr 1;23(2):150-6.
13. Ren Y, Luo X, Wang C, Yin L, Pang C, Feng T, Wang B, Zhang L, Li L, Yang X, Zhang H. Prevalence of hypertriglyceridemic waist and association with risk of type 2 diabetes mellitus: a meta- analysis. Diabetes/metabolism research and reviews. 2016 May 1;32(4):405-12.
14. Chen GY, Li L, Dai F, Li XJ, Xu XX, Fan JG. Prevalence of and risk factors for type 2 diabetes mellitus in hyperlipidemia in China. Medical science monitor: international medical journal of experimental and clinical research. 2015;21:2476.
15. Roy S, Sherman A, Monari-Sparks MJ, Schweiker O, Jain N, Sims E, Breda M, Byraiah GP, Belecanech RG, Coletta MD, Barrios CJ. Association of comorbid and metabolic factors with optimal control of type 2 diabetes mellitus. North American journal of medical sciences. 2016 Jan;8(1):31.
16. Ozder A. Lipid profile abnormalities seen in T2DM patients in primary healthcare in Turkey: a cross-sectional study. Lipids in health and disease. 2014 Dec;13(1):183.
17. Patel VI, Patel KP, Makadia MG, Shah AD, Chaudhari KS, Nilayangode HN. Levels of apolipoprotein a1, b100 and lipoprotein (a) in controlled and uncontrolled diabetic patients and in non-diabetic healthy people. Journal of clinical and diagnostic research: JCDR. 2017 Feb;11(2):BC01.
18. Mortensen LS, Hartvigsen ML, Brader LJ, Astrup A, Schrezenmeir J, Holst JJ, Thomsen C, Hermansen K. Differential effects of protein quality on postprandial lipemia in response to a fat-rich meal in type 2 diabetes: comparison of whey, casein, gluten, and cod protein–. The American journal of clinical nutrition. 2009 May 20;90(1):41-8.
19. Lebovitz HE, Ludvik B, Yaniv I, Haddad W, Schwartz T, Aviv R, Metacure Investigator Group. Fasting plasma triglycerides predict the glycaemic response to treatment of type 2 diabetes by gastric electrical stimulation. A novel lipotoxicity paradigm. Diabetic Medicine. 2013 Jun;30(6):687-93.
20. Parhofer KG. Interaction between glucose and lipid metabolism: more than diabetic dyslipidemia. Diabetes & metabolism journal. 2015 Oct 1;39(5):353-62.
21. Hussain A, Ali I, Ijaz M, Rahim A. Correlation between hemoglobin A1c and serum lipid profile in Afghani patients with type 2 diabetes: hemoglobin A1c prognosticates

dyslipidemia. Therapeutic advances in endocrinology and metabolism. 2017 Apr;8(4):51-7.

22. Valensi P, Avignon A, Sultan A, Chanu B, Nguyen MT, Cosson E. Atherogenic dyslipidemia and risk of silent coronary artery disease in asymptomatic patients with type 2 diabetes: a cross-sectional study. Cardiovascular diabetology. 2016 Dec;15(1):104.
23. Schofield JD, Liu Y, Rao-Balakrishna P, Malik RA, Soran H. Diabetes dyslipidemia. Diabetes Therapy. 2016 Jun 1;7(2):203-19.

Audio Distraction Technique in Management of Anxious Pediatric Dental Patients

RathnaSubhashini M.H[1], Sam Jebaraj. A[2], Kanthawsamy A.C[3], Raghu Sandhya[4]

Graduate Student[1],Graduate Student[2],Senior Lecturer[3],Department of Pediatric Dentistry,Reader[4],Department of Conservative Dentistry and Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Dental anxiety results in undesirable effects like turning away of dental treatment and increase stress among doctors that consequently have an effect on the treatment quality.Several studies have shown that, in medical practice audio-distraction was commonly usedto reduce the patients' pain and anxiety for short invasive procedures.Hence the aim of this study was to evaluate the effectiveness of listening to music using headphones as an audio distraction technique on behavior and anxiety management in children receiving dental treatment.

Materials and Method: The presentinterventional prospective study comprised of 60 subjects within the age group of6-12 who were advised for dental procedures. The patients were selected from the outpatient department and grouped randomly into group A and group B with 30 subjects in each group. The study group Acomprised of 30 pediatric patients who were given audio distraction aids. In the control group B, the treatment procedure were carried out without any distraction aids .The anxiety level was measured before and after the treatment for both the studyand control group using Wong Baker's anxiety rating scale and statistical analysis were made.

Result: Intergroup comparison showed highly significant difference in anxiety levels after the treatment.Intragroup comparison showed significant decrease of anxiety level from pre-operative and post-operative in both control and study group but it was more significant in study group. The statistical analysis showed significant decrease in anxiety level after the treatment in study group than in control group.

Conclusion: Within the limitations of the study, it was found that audio distraction technique proved to distract and reduce anxiety during dental procedures in pediatric patients.

Key Words: *Pediatric patients, Anxiety, White noise, Head Phones,Wong baker's anxiety rating scale, Distraction technique.*

Introduction

Dental anxiety among pediatric patients is a great challenge faced by every dentists in everyday dental practice. The child's uncooperative behavior may restrain the effective delivery of dental care which in turn may compromise the quality of treatment provided [1,2]. Behavior management techniques are meant to reduce the need for excessive and unsafe use of medications. Dentists have a wide variety of techniques available to them to assist in management of child with anxiety [3], such as tell-show-do, relaxation, distraction, systemic desensitization, modeling, audio analgesia, hypnosis and behavioral rehearsal[4]. Among this, traditional management such as papooseboard and hand over mouth technique can be successful. Management of children in the dental clinic is complex balancing act involving the child, parent and the dentist. To provide dental care for children good communication skills are necessary. A considerable percentage of children do not co-operate in the dental chair, causing an obstruction to delivery of quality dental care. This gives rise to behavior management techniques or alternatives to communicative management. Behavior management of the child patient is an intrinsic component of pediatric dental practice. Parents and care takers also play an important part in reducing the child's anxiety, allowing the dentist to perform the treatment and manage them [5,6]. Now no aversive techniques such as distraction are more popular. The success of distraction technique in medical setting and in adult patients is well organized, but literature reports limited data to assess the efficacy of distraction methodology in pediatric dental patients [1].Hence the aim of this study was to evaluate the effectiveness of listening to music using headphones as an audio distraction technique on behavior and anxiety management in children receiving dental treatment.

Materials and Method

Study Population

The present interventional prospective study comprised of 60 subjects within the age group of 6-12 years selected from the outpatient department and grouped randomly into group A as study group and group B as control group with 30 subjects in each group.

Inclusion Criteria

No previous dental treatment experience.

No systemic disease.

No learning disability.

Exclusion Criteria

Uncooperative patients.

Childrenwith any mental and physical disability.

Children with any trauma.

Materials

Wong baker's anxiety rating scale

Headphones

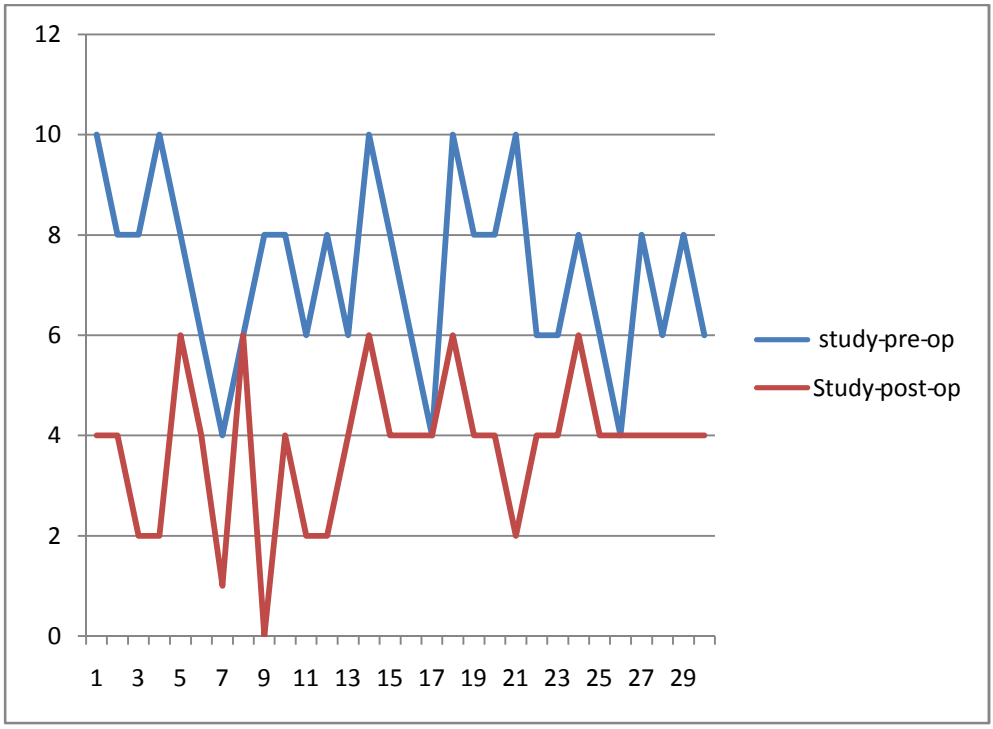
MP3 player

Method

The study was approved by the ethical committee of the institution and informed consent was obtained from the patients and their parents/guardians before the commencement of the study. The study group Acomprised of 30 pediatric patientswho were given audio distraction aidsi.e. Headphones with white noise a few minutes before the commencement of treatment.In the control group B, the treatment procedure were carried out without any distraction aids.

The volume of the music was maintained at 75 db to reduce the audible sound of hand piece to the patient. Whenever there was a need to communicate with the patient the volume was reduced. The anxiety score was measured before and after the treatment using Wong Baker's anxiety rating scale. Statistical analysis were made with unpaired t-test for intergroup comparison of anxiety scores and paired t-test for intragroup comparison of anxiety scores.

Graph 1: Anxiety score using audio distraction technique measured by Wong Baker's anxiety scale before and after the treatment



Graph 2: Anxiety score without using audio distraction technique measured by Wong Baker's anxiety scale before and after the treatment

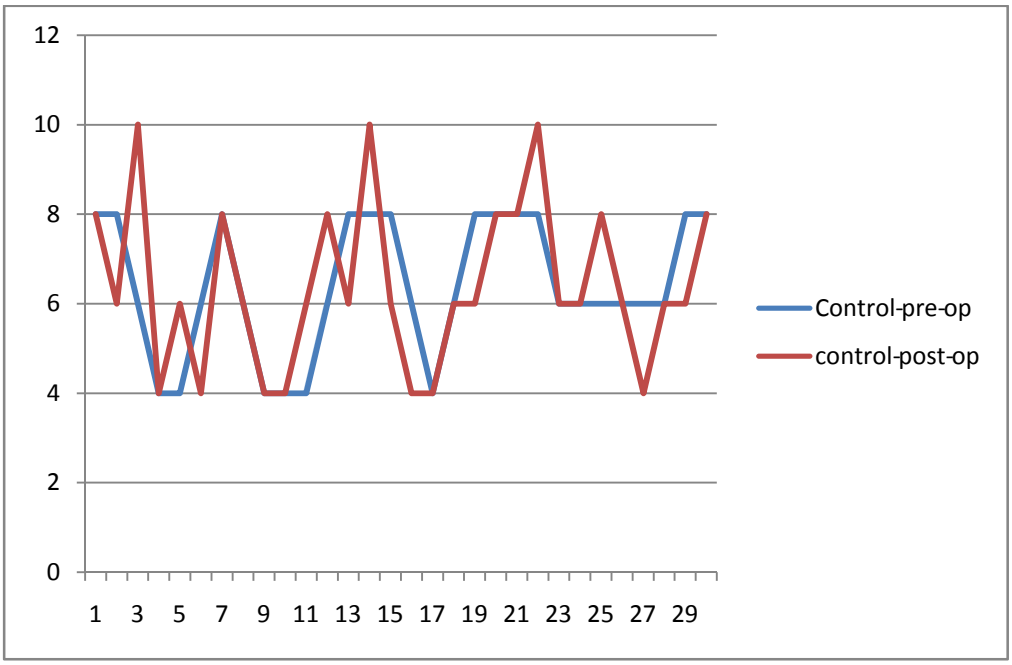


Table1: Inter group comparison of anxiety score

	Group	N	Mean	Std. Deviation	Mean diff	t value	p value
Pre-op	Study group	30	7.2667	1.77984	.66667	1.495	.140
	Control group	30	6.6000	1.67332			
Post-op	Study group	30	3.7667	1.47819	-2.36667	-5.841	**.000
	Control group	30	6.1333	1.65536			

Table 2: Intra group comparison of anxiety score

Group		N	Mean	Std. Deviation	Mean diff	t value	p value
Study group	Pre-op	30	7.267	1.780	3.500	8.69	*<0.001
	Post-op	30	3.767	1.478			
Control group	Pre-op	30	6.600	1.673	.466	2.97	*0.006

Post-op	30	6.133	1.655			

*- significant

**- Highly significant.

Result

Table 1 shows the mean scores, standard deviation and mean difference of pre-operative and post- operative anxiety levels between the groups (Unpaired t test), whereas, Table 2 shows within the groups (Paired t test). There was no much difference in distribution of subjects(based on anxiety levels) in study and control group ($p>0.05$).

Intergroup comparison showed highly significant difference in anxiety levels after the treatment. (Table.1)

Intragroup comparison showed significant decrease of anxiety level from pre operative and post operative in both control and study group but it was more significant in study group (Table.2)

The statistical analysis showed significant decrease in anxiety level after the treatment in interventional group than in control group (Fig 1, Fig 2).

Discussion

Dental fear of the patients is one of the most common reason to delay or avoid dental visits. Number of fearful people regularly cancel or fail to visit dentists. Patients with high dental fear, both children and adults, may prove difficult to treat. They require more time, and theirbehavioral problems which can result in a stressful and unpleasant experience for both the patient and the treating dental practitioner. If patients are not managed appropriately, it is more likely possible to establish what has been referred to as a ‘vicious cycle of dental fear’[6].

There are various pharmacological and non-pharmacological approaches in helping patients cope with dental anxiety. A caring and patient-centered approach in combination with various behavioral and psychological approaches play a vital role. This will yield superior short-term results as well as better long-term patient retention than the use of pharmacological methods.

Few non-pharmacological methods used for anxiety management includes, tell-show-do, rest breaks, signaling, positive reinforcement, diaphragmatic breathing, progressive muscle relaxation, cognitive restructuring, hypnosis[7]. These techniques have been in practice for many years and recent advancement in introduction of new distraction techniques includes music distraction and visual distraction techniques. A very few studies have been undertaken to evaluate the efficacy of distraction technique used for treating pediatric patients. Audio analgesia is one among the non-pharmacological distraction technique used to treat pediatric patients. In this study we have used white noise to reduce the pain and anxiety level of the pediatric patient. Objective measurement was done using Wong Baker's anxiety rating scale. The study results showed the effectiveness of using white noise in distraction and reducing anxiety in pediatric dental patients. The study result demonstrated that the anxiety levels were significantly reduced both with and without use of audio distraction. Interestingly,the use of audio distraction had reduced anxiety levelwhen compared with no usage.

White noise is a mixture of sounds of various frequencies [8]. It is also believed that White noise helps to increase the concentration and improve memory.This white noise of various frequencies have effect on mesolimbic midbrain, and this region of brain corresponds to the dopamine pathway [9]. Thus, white noise was reporedto reduceanxiety[9-10]. Moreover, the volume was kept at 75 decibel which was at 3/4 of the volume in the MP3 device which helped to decrease unpleasant noisecreated by dental hand piece or other anxiety inducing stimuli. Naithani et al evaluated audio- visual distraction in the managing anxious pediatric dental patients and reported an obviousdecrease in anxiety scores [9]. Jindal et al also found that audio distraction aids decreased level of anxiety in pediatric patients[10].Most children experience anxiety purely on the basis of psychological,social and environmental influences. Parents face special challenges because children with anxiety tend to be nervous, avoidant, annoying or exhausting[11-16].Ram et al reported that, audiovisual distraction technique provide as an effective distraction tool for the management of unpleasant behavior and distress that arises during dentalprocedures[17]. Singh et al reported that decrease the anxiety in pediatric patients to a significant extent, moreover patients had an overwhelming response to music presentations and wanted to hear them in their subsequent visits[18]. Prabhakar et al from his study conferred that, music reduced anxiety to some extent but not very significant, and stated that music distraction may be helpful

as an adjunct along with other techniques therefore further research needs to be done in this field using other non- aversive techniques and newer strategies should be devised to manage anxious pediatric dental patient [3]. Behavioral management of pediatric patients during dental treatment is most commonly used by almost all the dentists in which distraction technique plays a crucial role[19-22].Non- pharmacological methods of behavior management, particularly with difficult children proved to be more effective in reducing the complications caused due to anxiety[23-24].

However further research is required with greater sample size, involving different age group, giving choice of audio to the subjects and efficacy of other distraction methods such as visual and audio-visual techniques.

Conclusion

Within the limitationsof this study, it was found that the anxiety level of pediatric dental patients reduced with and without the usage of audio distraction after the treatment. Reduction in anxiety level with audio distraction proved more beneficial. Thus, it was concluded that managing pediatric patients with audio distraction aids especially white noise was an effective method for comfortable handling of anxious patients in dental clinic. Further more researches can be conducted with more invasive procedures to evaluate other better techniques in management of pediatric dental patients.

Corresponding Author

Raghu Sandhya

Reader,Department of Conservative Dentistry and Endodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Mail Id: drsandhyaendo@gmail.com

Contact Number: +91 9884610410

References

1.Singh R K, Gupta V K, Kumar A, Singh A, Shetty R, Pandey V. Effectiveness and comparison of various audio distraction aids in management of anxious pediatric dental patients. International Journal of Contemporary Medical Research 2016; 3(5): 1532-1534.

2. Navit S, Johri N, Khan S A, Singh R K, Chandha D, Navit P, Sharma A, Bahuguna A. Effectiveness and comparison of various distraction aids in management of anxious dental patients. Journal of Clinical and Diagnostic Research 2015;9(12): ZC05-ZC09.

3.Prabhakar A.R, Marwah N,Raju O.S. Music distraction-its efficacy in managemant of anxious pediatric dental patients.Journal of Indian Society Pedodontics and Preventive Dentistry 2007;25 (4): 177-82.

4. Venham L, Bengston D, Cipes M. Children's response to sequential dental visits. Journal ofDentistry 1977;56(5):454-9.

5. J Chandrapooja,Selvarasu K, Behavioural management techniques in pediatric clinic. International Journal of Pharmacy and Biological Sciences 2016;6(3);10-15

6.Freeman,R. The case for mother in the surgery.British Dental Journal 1999;186(10): 610-613.

7. Armfield J M, Heaton. U. Management of fear and anxiety in the dental clinic: a review. Australian Dental Journal 2013; 58(4): 390–407.

8. Rausch V H, Bauch E M, Bunzeck N. White noise improves learning by modulating activity in dopaminergic midbrain regions and right superior temporal sulcus. Journal of Cognitive neuroscience 2014; 26(7):1469- 1480.

9.Naithani M, Vishwanth D.Child's dental anxiety management by audio and audio- Visual distraction Technique- a comparative study. University of Research Journal of Dentistry 2014; 4(2):101-7.

10. Jindal R, Kaur R. Can we tune our pediatric patients?.International Journal of Clinical Pediatric Dentistry 2011; 4(3):186-9.

11.Muppa R, Bhupatiraju P, Duddu M, Penumatsa NV, Dandempally A, Panthula P. Comparison of anxiety levels associated with noise in the dental clinic among children of age group 6-15 years. Noise and Health2013;15 (64):190-3.

12. Eitner S, Wichmann M, Paulsen A, Holst S. Dental anxiety – an epidemiological study on its clinical correlation and effects on oral health. Journal of Oral Rehabilitation. 2006;33(8):588-93.

13. Baldwin DC. An investigation of psychological and behavioral response of dental extractions in children. Journal of Dental Research. 1966;45(6):1637-51.

14. Mc Knight-Hanes C, Myers, D R., Dushku J C et al. The use of Behavior management techniques by dentist across practitioner type,age,and geographic region. Pediatric Dental Journal 1993;15:267-271.

15. Carson P, Freeman R. Tell show do: reducing anticipatory anxiety in emergency pediatric dental patients. International Journal of Health promotion and Education 1998; 36(3): 87-90

16. Ingersoll B D, Nash D A, Blount R, Gamber,C. Disraction and contingent reinforcement with pediatric dental patients. Journal of Dentistry for children 1984; 51(3):203-207.

17. Ram D, Shapira J, Holan G, Magora F, Cohen S, Davidovich E. Audiovisual video eyeglass distraction during dental treatment in children. Quintessence international. 2010 Sep 1;41(8).

18. Singh D, Samadi F, Jaiswal JN, Tripathi AM. Stress reduction through audio distraction in anxious pediatric dental patients: an adjunctive clinical study. International journal of clinical pediatric dentistry. 2014 Sep;7(3):149.

19. Ingersoll B D, Nash D., Bloun, R,GamberC.The use of contingent audio taped material with pediatric dental patients. Journal of American Dental Association 1984; 109(5): 717-719.

20.Sabarathinam J, Maheswari U. Assessment of Behavioural Management Techniques of Paediatric Patients Among the Dentist in Chennai: A Questionnaire Study, Research journal of pharmacy andtechnology 2016;9(2):145-148.

21.Adair SM, Waller JL, Schafer TE, Rockman RA. A survey of behavior management teaching inpredoctoral pediatric dentistry programs. Pediatric Dental Journal 2004; 26(2):143-50

22.Acs G, Musson C W, Burke M J. Current teaching of restrain and sedation in pediatric dentistry: A survey on program directors. Pediatric Dental Journal 1990; 12(6):364-367.

23.Allen K.D, Stonley RT, McPherson K. Evaluation of Behavior management technology dissemination in pediatric dentistry. Pediatric Dental Journal 1990; 12(2):79-82.

24. BelangerG.K ,Tilli, T S. Behavior management techniques in predoctoral and postdoctoral pediatric dentistry programs. Journal of Dental Education 1993 ; 57(3):232-238.

BMI Eating Habits and Related Factors among Students of Saveetha Dental College

Nathasha A/P Sivakumar[1], Gowri Sethu [2], Dhanalakshmi Ravikumar [3]

Graduate Student [1], Professor and Head [2], Department of Physiology

Senior Lecturer [3],Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction

The last two decades have witnessed tremendous changes in the eating pattern of Indian population. There has been a revolution in adolescent's life style and eating pattern which can be largely attributed to changes in family and social environment. The changes in eating pattern may adversely affect their health. The aim of this research is to evaluate the BMI and eating habits among students in Saveetha Dental College.

Materials and Methods

100 students of Saveetha Dental College in the age group of 17-26 years were selected for the study and were divided into five age groups respectively. Age 17-18(group 1), 19-20(group 2), 21-22(group 3), 23-24 (group 4), 25-26 (group 5). The questionnaires were distributed to them and their food intake for 1 whole week was noted down, the collected data is tabulated for further interpretation of the study.

Result

Results were assessed statistically. Almost every fourth male student was overweight. Strikingly, 15% of female students were underweight. Highly-significant difference was found between average body mass index (BMI) of male and female students. Students' BMI did not correlate with average family income or with the frequency of taking breakfast.

Conclusion

Increasing the proportion of adolescents meeting recommended dietary and physical activity guidelines has been identified as an important strategy to contrast the epidemic increase in obesity, especially in western Countries. These students are more concerned about their appearance then their healthy diet intake. They are keener on finishing their works then taking proper care on their health.

Key Words: BMI, eating habit, healthy diet, obesity, underweight, carbonated drinks.

Introduction

The prevalence of obesity and overweight is quite high in the world population. According to a 2008 World Health Organization report, one billion of the world population is overweight and an additional 300 million are obese. Changing eating habits can be responsible for the rapid increase in these conditions, particularly among the young population [1].Previous studies have demonstrated that the adoption of an unhealthy lifestyle after becoming a university student, including smoking and alcohol consumption, and poor food preferences may be attributed to moving away from home, poor cooking skills, low income, and little time availability to engage in physically active recreation, and so forth. Such lifestyle changes lead to weight gain and an increase in body fat composition during the first year of the university education [2].

Obesity is associated with a variety of adverse health outcomes. An important risk factor for cardio metabolic disorders, cardiovascular diseases and premature mortality is obesity. There was evidence that suggest that diabetes, heart disease, osteoarthritis and high blood pressure may mediate the association between being overweight and health-related quality of life. In the last 20 years obesity has increased in prevalence worldwide. The majority of studies focused on overweight, obesity and less on underweight [3]. However, it is necessary to pay more attention to the negative effects that being too skinny have on health, being noted that the mortality risk is associated with underweight. A person may be underweight because of genetic or metabolic causes, lack of food, or diseases like hyperthyroidism, cancer, tuberculosis, gastrointestinal or

liver problems etc. But a person can be underweight because he/she wants it, as a consequence of the emphasis society puts on "thinness" (thinness, „silhouette“, the fact of not being fat). It can even lead to anorexia nervosa and bulimia nervosa. In case of anorexia nervosa, there is a loss of appetite, but sometimes the appetite stays while the patient refuses to eat and the body weight drastically drops. In case of bulimia nervosa, hyperphagia of large quantities of food in a short period of time (eating binge) take place, followed by self-induced vomiting, laxatives or diuretics, by periods of feeding cease (fasting) or intense physical exercises in order to prevent weight gain (binge and purge, approx. “satiation and deprivation”).Underweight individuals are prone to infections, osteoporosis. For women with severe underweight amenorrhea, infertility, complications during pregnancy, anemia, hair loss etc. can occur [4].

In 1993, the WHO assembled an Expert Consultation Group with a charge of developing uniform categories of the BMI. The results were published as a technical report in 1995.Four categories was established: underweight, normal, overweight, and obese. An individual would be considered to be underweight if his/her BMI was in the range of 15 to 19.9, normal weight if the BMI was 20 to 24.9, overweight if the BMI was 25 to 29.9, and obese if it was 30 to 35 or greater. Using linear regression, a BMI of 16.9 in men and 13.7 in women represents a complete absence of body fat stores.

The above 4 categories are similar to those suggested by John S. Garrow in 1981, but the terminology was changed. The terminology he used was “desirable” for a BMI up to 25, “grade I obesity” between 25 and 29.9, “grade II obesity,” between 30 and 40, and “grade III obesity” for BMI greater than 40.

The latter classification was based on Rosenbaum and colleagues’ own data obtained in a survey of an adult to 39.9 is class II obesity and a BMI of 40 or greater is class III obesity.

Over the past several decades, there has been an increase in BMI in the general population. This has resulted in predictions of a public health disaster. It should be recognized that in the United States during the period from 1960 to 2002 not only has the mean weight increased by 24 lbs. among men aged 20 to 74 years, but also the height has increased by about 1 in. We can then calculate that the weight increase per year has only been 0.57 lb. and as indicated above, this

could be due to an increase in lean mass rather than fat mass, or it may be a combination of the two. In women, there was a similar increase in weight and height.

In an earlier report, life-insured men up to age 40 years were reported to be 0.5 to 1.5 inches taller and 2 to 9 lbs. heavier for the same height in 1959 than those studied 50 to 60 years prior to 1959. Also, in the earlier study, the mortality rate was lowest in those with higher weight-to-height ratios. This was attributed to the presence in the population of wasting diseases such as tuberculosis that resulted in an increased death rate. Previously, a secular upward trend in height in adults in the United Kingdom also was reported. In addition, in a twin study in the United Kingdom, children in 2005 were not only heavier but also taller than 1990 norms, whereas their BMIs were essentially the same.

Overall, the history of changes in height and weight in Western European men and probably women has been that of an increase in both weight and height. In the 17th century, the average height of men in Northern Europe was ~5 ft. 3 in. It now approaches 6 ft. These data suggest that the BMI categories should be adjusted upward periodically to accommodate population-based changes. Improvements in mortality rates also suggest an adjustment would be useful.

The new terminology appears to be a bit presumptuous and careless because the BMI is not a direct measure of percent of fat mass, and the dynamic concept that those in the former “overweight” category are now in the “pre obesity” category invariably going on to “obesity” is not the case. Also those with a lower BMI initially, but with a dynamic weight gain over time, would have to transition through this category in order to become classified as “obese” regardless of the terminology.

There exists at present enormous concern amongst the population of the developed world regarding being overweight and obesity. This concern is generally justified by alleging health motives. The deeper motivation is, however, to a considerable extent, aesthetic. It is a slim body that is considered an attractive body in our society, amongst other things because, in a context of nutritional overabundance, it is more and more difficult to maintain a normal weight. In this cultural context all kinds of strategies to lose weight have flourished: hypo caloric diets, restrictive eating habits, nutritional compounds, pharmaceutical products and such extreme

measures as skipping certain meals. Thus, we find ourselves in the situation where more than 70% of the population of the western world admits that they are attempting either to lose weight or to maintain their present weight [5].

However, despite all these attempts to lose weight, figures show that the prevalence of obesity and being overweight has grown continually and alarmingly since the eighties. All of this would lead one to suspect that, although for many these strategies may be of use in the short term, this is not the case in the long term. Several studies seem to show this. Half of the people tend to give up hypo caloric diets within two months of starting them [6]. Furthermore, 95% of people tend to recover their initial weight between one and five years after finishing the diet. Similarly, it has been observed that some subjects may gain a surplus of additional weight with respect to their weight before the diet. Recent prospective studies show that attempts to lose weight seem to be associated in some subjects with a subsequent greater increase in weight (equal to or greater than 2 kg). Finally, weight and food can be the cause of great worry in some people [7].

The body mass index (BMI) is a value gained from the calculation of weight and height of an individual. A student's eating habits depends on their lecture schedules and availability of food inside or in the vicinity of the university area. As a result of the increase in the fast-food market and lack of appropriate food courts, students usually face meal skipping, inadequate variety of foods, and snacking [8].

Because calculation requires height and weight, it is cheaper and easy to use for clinicians and for the general public [9]. BMI can be used as a screening tool for body fatness but is not diagnostic. BMI does not only measure body fat directly, but BMI is slightly correlated with more direct measures the body fat obtained from skin thickness measurements, bioelectrical impedance, densitometry, dual energy x-ray absorptiometry (DXA) and other methods[10].

The purpose of this study was to quantify the prevalence of overweight and obesity among a sample of students in Saveetha Dental College and to describe their main eating habits. This study is part of a larger analytical investigation among the students of Saveetha Dental College, which estimated factors associated with quality of life.

A particular problem with BMI as an index of obesity is that it does not differentiate between body lean mass and body fat mass; that is, a person can have a high BMI but still have a very low fat mass and vice versa.

From an anatomical and metabolic perspective, the term obesity should refer to an excessive accumulation of body fat (triacylglycerol), and upon these grounds, the accuracy of the BMI as a determinant of body fat mass has been repeatedly questioned, because it clearly has limitations in this regard. Gender, age, ethnic group, and leg length are important variables. It should be noted that in population-based studies women generally have a BMI that is lower than that in men, even though their fat mass relative to their body build or BMI is considerably greater (~20% to 45%+).

The relatively poor correlation between percent of body fat mass and BMI in males has been known for many years and was clearly shown in a study in which percent of body fat was determined by a densitometric method. For men with a BMI of 27 in that study, the 95% confidence intervals for percent of body fat were 10% to 32%; that is, in this group, the percent of body fat varied from very little to that considered to be in the obesity range. (NIH-suggested criterion for obesity based on percent of body fat for men is ≥25%, and that for women is ≥35%)

Materials and Methods

- **Ethical Approval**

The present cross-sectional study was carried out after obtaining Ethical approval from the Institutional review board of Saveetha Dental College.

- **Study Design**

The survey was conducted in a single dental school in Chennai. This was one of the first study to assess the knowledge of BMI, eating habits and its related factors among the dental students. Since it was easy to recruit the study population from a single dental school, purposive sampling technique was chosen. A sample of 100 undergraduate students in a Saveetha Dental School, Chennai, India was included in the study.

The study was undertaken in two stages stage 1 and Stage 2. Stage 1 comprised formulating, designing, and validating the questionnaire, whereas Stage 2 tested the validated questionnaire among 100 undergraduate dental students.

- **Stage 1 (Designing and Validation of Questionnaire)**

A standardized self-constructed questionnaire was formulated by two investigators (NS, DR,). Both the investigators (NS, DR,) independently formulated the questionnaire, and after a consensus, they arrived at a final list of 15 questions. Initially, content validation of the questionnaire was performed by circulating the questionnaire to 100 students of Saveetha Dental College. A panel discussion was conducted among 10 qualified General dentists, and they had rated the questionnaire using content validity ratio.[14-16] There was a good agreement between the investigators, with a rating of >0.7. Finally, the questionnaire was distributed to 10 random General dentists for face validation, and it was evaluated using 5-point Likert scale.

- **Stage 2 (Testing of Validated Questionnaire)**

After the content and face validation, the questionnaire was distributed to 100 undergraduate students to complete the questionnaire. Distribution and collection of the questionnaire were done by one Graduate dentist (NS).

- **Statistical Analysis**

Data were collected and entered into SPSS software version (SPSS Inc., Chicago, IL, USA) 20.0 for percentages.

Results

Basic demographic data were shown. Male students were, in average, significantly taller and weighed more than females BMI 23.4±2.7 and 21.0±2.6 respectively. Mean BMI across genders varied within the normal weight range. According to BMI categories, three-quarters had normal weight (Table 1). Almost every fourth male student was overweight. In contrast, 15% of females were underweight. Obese students were the least common category with prevalence of only 1.5%. Students' BMI did not correlate with average family income or with the frequency of

taking breakfast. Mean BMI did not differ between those who ate breakfast and those who skipped it. In addition, BMI did not differ according to students' residence (with parents/alone/student dormitory. More than half of the students (57.3%) ate their breakfast regularly. One-third of subjects drank coffee every day, opposed to 27.3% who never drank it. Majority of students (80.1%) ate meat at least two times a week. Half of them ate one portion of fruits and vegetables daily (Table 2). There were no gender differences between consumption of fruits, vegetables, and meat.

TABLE 1: Distribution of Students According to BMI Categories

	MALE (NUM)	MALE (%)	FEMALE (NUM)	FEMALE (%)	TOTAL (NUM)	TOTAL (%)
UNDERWEIGHT	1	2.17	9	16.67	10	10
NORMAL	34	73.91	42	77.78	76	76
OVERWEIGHT	10	21.75	2	3.7	12	12
OBESE	1	2.17	1	1.85	2	2
TOTAL	46	100	54	100	100	100

TABLE 2: Distribution of Students According to Daily Consumption of Fruits And Vegetables

DAILY PORTION	FRUITS%	VEGETABLES(%)
ONE	46.8	46.6
TWO	22.7	25.6
THREE	8.9	9.8
FOUR	2	1.2
FIVE	1.9	0.9
MISSING	17.7	15.9

Discussion

Most of the Saveetha Dental College students were of normal nutritional status. Similar mean BMI was documented in students of different cultural backgrounds [11]. Obese students accounted for only 1.5% in our sample [12]. During 1997-2007, eating habits in the population of Serbia have been associated with irregular meals, decrease in fruits and vegetables consumption, along with related augmented intake of high-energy ingredients. In 2006, only half of the adults had had three regular meals per day while more than half were overweight [13].

In contrast, striking overall prevalence of underweight among Serbian students was recorded to be 10.6%. Proportion of females who were underweight was even higher (15.3%). Bodily proportions and BMI are certainly subject to emotional and physical well-being[14]. which is particularly addressed in concomitant eating disorders[15]. Also, university setting may have an important role in weight loss in the young adults as well. Academic pressures and time limits might often prevent students from regular meal consumption, leading to unintentional weight loss. Another possible explanation for such a high prevalence of underweight in female students might be related to eating disorders [16].

In this study, BMI in students did not differ among diverse place of residence during university schooling, implicating that parental involvement in meal regularity does not have impact upon weight change [17]. On the other hand, individuals who take care of meal frequency on their own, accomplish this in a responsible manner without experiencing dramatic weight losses or gains [18].

The importance of taking breakfast was emphasized because of the local circumstance that university schooling schedule covers the period from 8 am to 3 pm. Therefore, it is crucial that students have regular morning meal before the start of daily duties [19].

Since the students were filling in the questionnaire independently, we may consider reliability of data and potential information as well as recall bias. Additionally, there were no food intake diaries to quantify exact consumption of the ingredients [20].

Conclusion

Nutritional interventions among students have already shown positive results [21]. Therefore, promotion of healthy food consumption, with abundant fibers, whole grains, dairy products, and low energy-dense foods is needed. In addition, there is a growing demand for global health strategies which would encourage healthy body-image and figure; thus, these initiatives should mobilize the society on a national and international level [22].

Corresponding Author

Dhanalakshmi Ravikumar

Senior Lecturer,Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

E-Mail: dhana9677@gmail.com

Telephone number - 9597011442

References

1. Van Gaal LF, Mertens IL, Christophe E. Mechanisms linking obesity with cardiovascular disease. Nature. 2006 Dec 13;444(7121):875.
2. Hou X, Liu Y, Lu H, Ma X, Hu C, Bao Y, Jia W. Ten-year changes in the prevalence of overweight, obesity and central obesity among the Chinese adults in urban Shanghai, 1998–2007—comparison of two cross-sectional surveys. BMC public health. 2013 Dec;13(1):1064.

3. Kearns B, Ara R, Young T, Relton C. Association between body mass index and health-related quality of life, and the impact of self-reported long-term conditions–cross-sectional study from the south Yorkshire cohort dataset. BMC Public Health. 2013 Dec;13(1):1009.

4. Diouf I, Charles MA, Ducimetière P, Basdevant A, Eschwege E, Heude B. Evolution of obesity prevalence in France: an age-period-cohort analysis. Epidemiology (Cambridge, Mass.). 2010 May;21(3):360.

5. Roh L, Braun J, Chiolero A, Bopp M, Rohrmann S, Faeh D. Mortality risk associated with underweight: a census-linked cohort of 31,578 individuals with up to 32 years of follow-up. BMC Public Health. 2014 Dec;14(1):371.

6. Rada C. Body mass index and eating habits in young adults from Romania. International Journal of Medical Research and Health Sciences. 2016 Jan 1;5(5):42-50.

7. Nurul Husniyah binti Che Soh The effect of stress on eating habit and consequence health problem among individuals of Saveetha Dental College-A Questionnaire based study,. IJAR,10.21474/IJAR01/432;123-127

8. Nivashini.G.S.V, Vishnu priya.V, Awareness of eating disorder – A Questionnaire based survey, IJPSR33 (1), July-August 2015; 168-170.

9. A.Ashik Ahmed, Jothi Priya General Knowledge about proper eating habits among local population, JPSR2016; 8(8):909-913

10. Shrimpton R, Rokx C. The double burden of malnutrition.

11. Black RE, Bhutta ZA, Bryce J, Morris SS, Victora CG. The Lancet's Series on Maternal and Child Undernutrition. The Lancet. 2013; 2013:1-2.

12. Driskell JA, Kim YN, Goebel KJ. Few differences found in the typical eating and physical activity habits of lower-level and upper-level university students. Journal of the American Dietetic Association. 2005 May 1;105(5):798-801.

13. Pekmezovic T, Popovic A, Tepavcevic DK, Gazibara T, Paunic M. Factors associated with health-related quality of life among Belgrade University students. Quality of life research. 2011 Apr 1;20(3):391-7.

14. Sira N, Pawlak R. Prevalence of overweight and obesity, and dieting attitudes among Caucasian and African American college students in Eastern North carolina: A cross-sectional survey. Nutrition research and practice. 2010 Feb 1;4(1):36-42.

15. Sakamaki R, Amamoto R, Mochida Y, Shinfuku N, Toyama K. A comparative study of food habits and body shape perception of university students in Japan and Korea. Nutrition Journal. 2005 Dec;4(1):31.

16. Gazibara T, Tepavcevic DB, Popovic A, Pekmezovic T. Eating habits and body-weights of students of the University of Belgrade, Serbia: a cross-sectional study. Journal of health, population, and nutrition. 2013 Sep;31(3):330.

17. Vasiljevic N, Ralevic S, Marinkovic J, Kocev N, Maksimovic M, Milosevic GS, Tomic J. The assessment of health-related quality of life in relation to the body mass index value in the urban population of Belgrade. Health and quality of life outcomes. 2008 Dec;6(1):106.

18. Jenkins PE, Hoste RR, Conley CS, Meyer C, Blissett JM. Is being underweight associated with impairments in quality of life in the absence of significant eating disorder pathology?. Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity. 2011 Mar 1; 16(1):e61-4.

19. Ha EJ, Caine-Bish N. Effect of nutrition intervention using a general nutrition course for promoting fruit and vegetable consumption among college students. Journal of nutrition education and behavior. 2009 Mar 1; 41(2):103-9.

20. Lee HS, Lee J, Paik JJ. A Study of Food Habits, Physical Status and Related Factors of College Students in Chuncheon. Korean Journal of Community Nutrition. 1998 Feb 1;3(1):34-43.

21. Timlin MT, Pereira MA, Story M, Neumark-Sztainer D. Breakfast eating and weight change in a 5-year prospective analysis of adolescents: Project EAT (Eating among Teens). Pediatrics. 2008 Mar 1; 121(3):e638-45.

22. G.Lakshmanan Correlation between Body Mass Index, waist-hip length and Menstrual abnormalities, IJST, Vol 10(24), June 2017;90-95

Body Mass Index and Severity of Dental Caries among South Indian Children

Aslin Sanofer[1], Mahesh.R[2], Jayanth Kumar V[3]

Graduate Student[1], Reader[2], Department of Pedodontics, Reader[3], Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

Abstract

Introduction: Obesity is a medical condition in which excess body fat has accumulated to the extent that it has an adverse effect on health. Relative weight and body mass index (BMI) are nearly identical and are reasonable estimates of body fatness as measured by percentage body fat.The aim of this study was to find out the impact of body mass index and severity of dental caries among South Indian population. This study also assess the correlation between severity of caries in relation to body mass index among children.

Materials and Method: The prevalence of dental caries using dmft/DMFT index and BMI was assessed among South Indian school children population of age 8-12 years. A sample of 100 children werechosen.

Results: There was no significant correlation shown between BMI and Dental caries with a weak positive relationship between them among South Indian Children with the r value being 0.09.

Conclusion: This study showed that the obese children were prone to risk of more dental caries than normal individuals.

Key Words*: Dental caries, body mass index (BMI),dmft/DMFT , Children, Obesity.*

Introduction

The Body Mass Index (BMI) is a measure which relates patient weight and height.BMI is the metric currently in use for defining anthropometric height/weight characteristics in adults and for categorizing them into groups. The common interpretation is that it represents an index of an individual's fatness. BMI isalso widely used as a risk factor for the development of or the prevalence of several health issues (1). It is most ordinarily employed in decisive public health

policies.The BMI has been accepted in population-based studies and employed in process specific classes of body mass as a health issue.In 1972, Keys et al severely criticized the validity of Metropolitan Life Insurance published data per se, and the then-published tables of desirable weight for height, as well as the tables used to define people who were underweight or overweight (2). The term “obese” was rarely used in that era. Instead, Keys et al, using better documented weight for height data, popularized the Quetelet Index in population-based studies. They referred to it as the body mass index (BMI).

A higher BMI score indicates greater risk for developing serious health problems(3). WHO estimates that obesity is the fifth leading cause of mortality worldwide and now the rates have doubled within the last 20 years (4).The dietary changes are considered to be one of the potential causes for the observed increase in the prevalence of both overweight and obesity. Dental caries is multi-factorial disease and affects most of the world population.Dental disease ranks as the second most expensive disease. Recently, caries prevalence has increased in developing nations due to an array of factors, such as intake of sugary foods, low socio-economic status, exposure to fluorides, ethnicity, age, the limited access to oral health services and other lifestyle factors(5).

The decay-missing-filled (DMF) index or decayed, missing, and filled (restored) teeth (DMFT) index is one in every of the foremost common ways in oral medical specialty for assessing caries prevalence also as dental treatment wants among populations and has been used for concerning seventy five years (6). This index relies on in-field clinical examination of people by employing a probe, mirror and cotton rolls, and easily counts the quantity of decayed, missing (due to tooth decay only) and restored teeth. Another version projected in 1931 counts every affected surface, yielding a decayed, missing, and stuffed surfaces (DMFS) index.

Principle and rules in coding –DMFT are:

1- A tooth could have many restorations however it counted together tooth, F.

2- A tooth could have restoration on one surface and tooth decay on the opposite, it should be counted as decayed D .

3- No tooth should be counted over once, D M F or sound.

The maximum score of dmft is thirty two and minimum score of dmft is zero .The maximum score of primary teeth index is twenty .In mixed dentition, each child index is taken , one for permanent teeth and another for primary teeth.

This study gives details about prevalence and severity of dental caries among young children and its associated correlation with Body mass index.

Material and Method

The sample no of children was 100 whose age range were 8-12 years. Both the male and female were included .Data were collected through interview and a self-report questionnaire. The prevalence of dentalcaries were obtained using the WHO standard criteria. Weight and height were measured and BMI was calculated according to the formula: weight / height (Kg/ m2) units. BMI was categorized into underweight (BMI < 18.5), normal (BMI 18.5 - 24.9), overweight (BMI 25 - 30), and obese (BMI > 30). Information about relevant eating habits and brushing habits were added in the survey .The DMFT/ dmftand BMI values were noted(Table no : 1) and statistical analysis were calculated. The statistical analysis was done using SPSS version 17 and Pearson's correlationcoefficientwas calculated.

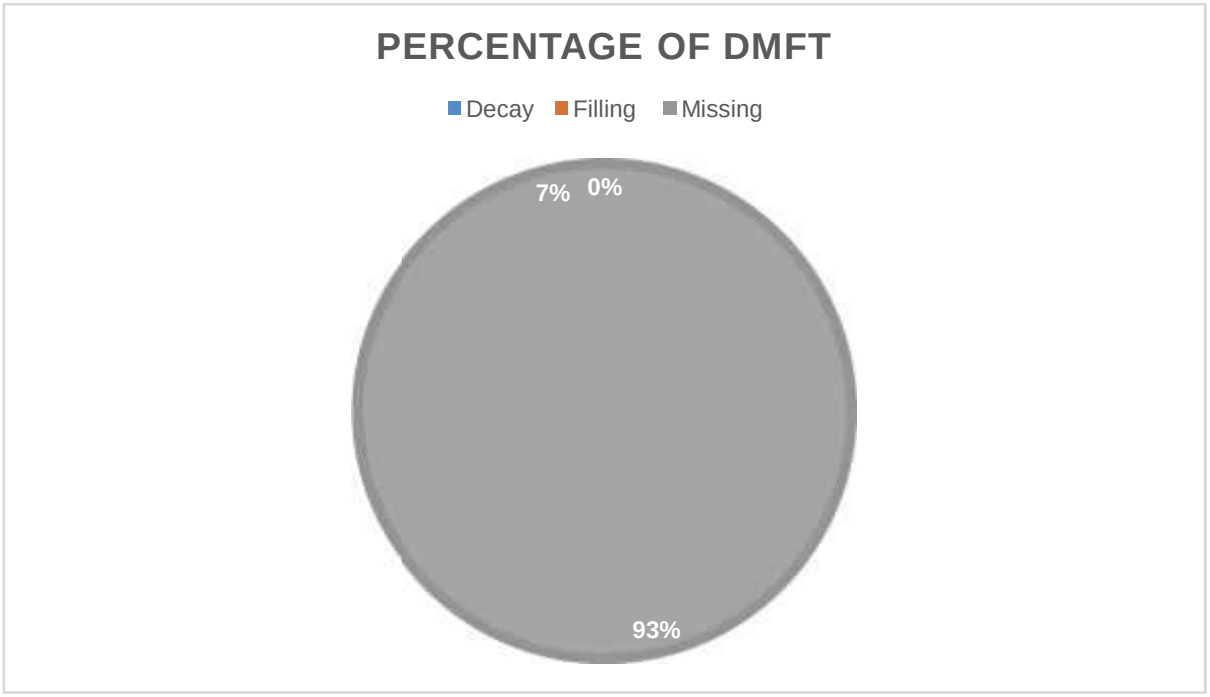
Distribution of study subject based on gender, BMI and dmft / DMFT

No,of,samples		100
	Males	43
Sex		
	Females	57
	DMFT,/,dmft	0.148
Mean,value		
	BMI	19.86

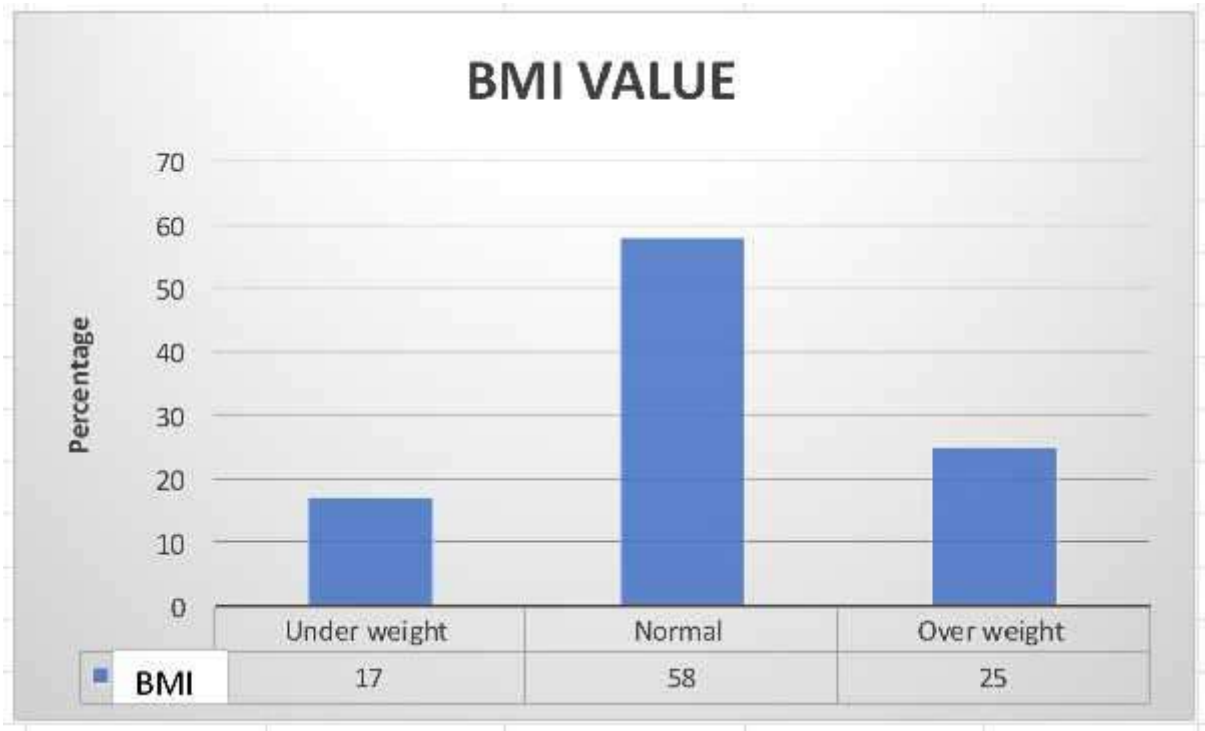
(**Table no : 1)**

Result:

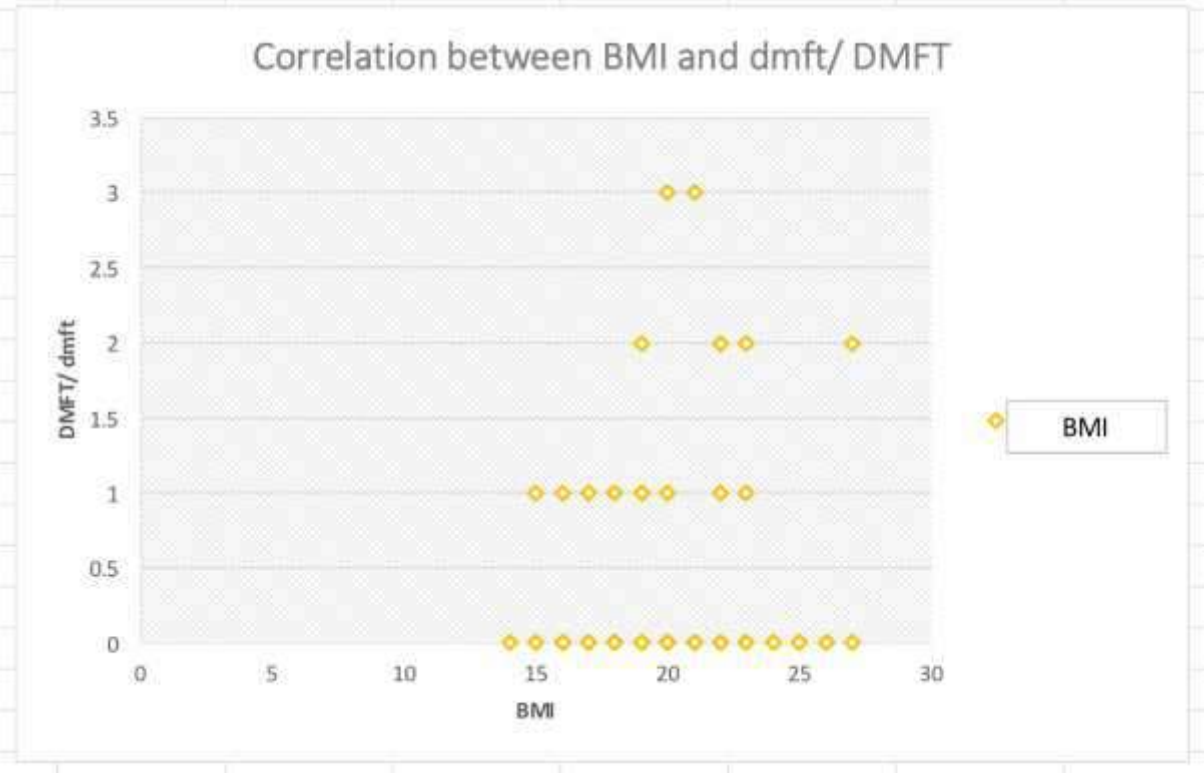
The prevalence of caries were found to be more in posterior teeth than anterior teeth . The mean DMFT/dmft was calculated as 0.148 and the mean BMI was 19.86. From the data the underweight children (BMI <18.5) was 35 and the mean DMFT/dmft score was 0.071. The normal weight children were 58 (BMI 18.5 - 25) and the mean score was 0.19. The overweight children with BMI > 25 were 5 with the mean score at 0.2. Pearson's correlation coefficient between DMFT/dmft and BMI was 0.09 indicating a weak positive correlation. High risk of caries were found to be more in permanent dentition than primary dentition.Both the gender was affected in the equal population.The obese or overweight children have more risk of caries due to mid snacking habits during school time and holidays.The missing and filling were found to be comparatively less than decay.



Graph 1 –Pie chart showing frequency of decay, missing and filled tooth/ teeth



Graph 2 - Bar graph showing average ideal Body weight percentage



Graph 3 – Scatter plot showing correlation between BMI and dmft / DMFT

Discussion:

In this study, we noticed a weak correlation between BMI and dmft / DMFT which was similar to other previous studies (7).The r value evaluated in this study were 0.09. (Graph no : 3). In this study, Dmft / dmft rate showed less risk in normal and under weightschool children where as higher risk in over weight school children (Graph no : 1& 2) .

In one of the recent studies , it is considered that clinical variables (dmft, DMFT) can be relevant predictors for the occurrence of dental caries in mixed dentition and permanent dentition.Caries lesions of primary teeth along with the consumption of carbohydrates (sweets) were associated with the occurrence of proximal lesions of permanent teeth in adolescents, and especially first permanent molars were affected.The combination of a large number of incipient caries, Streptococcus mutans and Lactobacillus acidophilus presence, represent a predictive model for

carious lesions in permanent dentition.The bacterial level (Streptococcus mutans, Lactobacillus acidophilus) is included with the highest accuracy in prediction models of caries in the permanent dentition.Increased frequency of tooth brushing associated with a higher socioeconomic level,education level and reducing snacks, are considered unfavourable elements for caries development in the permanent dentition.Mentioned aspects can become effective conceptual and lucrative opportunities to improve the child's dental management (8).

Forty-eight percent of studies reviewed found no association between dental caries and BMI; 35% found a positive association and 19% found an inverse association.Children who are overweight or obese have relatively high levels of dental caries.They also consume high levels of soda (9) and other energy-dense foods, many of which are cariogenic and obesogenic. Modeer et al. suggest that obese children are at risk of dental caries because they have reduced salivary flow which is associated with protein-deficient malnutrition(10). Obese children may well suffer from protein deficient malnutrition if their energy intake is made up of high carbohydrate, highly processed foods. Malnutrition could also predispose to dental caries; deficiencies in protein or energy foods may lead to protein-energy malnutrition, decreased salivary flow, calculus formation, high levels of caries and reduced growth(11).

Also Costacurta et al (12) suggest that BMI may not be the best measure of body fat composition when testing the association between dental caries and obesity. They suggest that misclassification of childhood obesity using BMI might account for the failure of studies to detect the association between child adiposity and dental caries. Present studies were found to be show a positive significant relationship between BMI and dental caries.The association between DMFT/dmft and BMI were also significant.

In a study conducted in India (13) among 350 school children revealed a negative correlation of $r=-0.023$ which was not statistically significant ($p>0.05$). Further their study had reported that mean DMFT values 4.9 ± 4.0, 4.6 ± 3.3, 2.7 ± 1.9, and 4.4 ± 3.0 for normal, overweight and obese respectively.

A similar study carried out by Bhayat et al (14) among 12 year old boys in fur geographical location in Medina, Saudi Arabia. The results showed that the mean BMI was 22.17 kg/m² (± 5.15); 41% had normal BMI, 25% were overweight and 30% were obese. The mean DMFT Score 1.46 (± 2.04). The participants in the normal BMI group had a higher prevalence of Caroes (57%)and mean DMFT was 1.92. Using a linear regression analysis it was found that overweight and obese participants had twice the risk of developing caries.

In a study America at al (15) in a cross sectional study of 1326 children who took part in the 2010 survey found that there was no significant difference in the DMFT and BMI. In a study conducted in Kerala (16) among 2000 school going children the mean BMI recorded was 26.87 ± 2.26 for the overweight children and 20.82 ± 1.48 for the normal-weight children. The mean DMFT for the overweight group was 3.90 ± 2.95 while that in normal weight was 3.36 ± 2.73. There was no statistically significant correlation between BMI and DMFT. In a broad study of 32461 pupils in China had reported a positive correlation between BMI and DMFT/dmft (24) . A study carried out in Japan showed that 23% of participants had no caries (DMFT=0),17% had few caries (DMFT =<3) 23% had many caries (DMFT=3.1-6.9) and 22% had many caries (DMFT>7). There was no siginificant correlation between DMFT and BMI
(17) . In a study done in Korea the mean DMFT had no correlation with BMI (18).In a study conducted in Chandigarh among 4493 school going children (19) it was found that a majority of children required restorative procedures for dental caries. There are reports in the literature to indicate that high DMFT score has a significant impact in the patient's perception of dental health (20).

Conclusion

The correlation between dmft / DMFT in relation to BMI shows a weak correlationof 0.09 which was similar to other previous studies.In our studies, the association between BMI and dmft/DMFT we're not statistically significant in the South Indian population. The dental checkup and medical health care awareness should be present for each individual or family functioning to prevent the early childhood caries and malnutrition or any multifactorial disease.

Corresponding Author

Mahesh.R

Senior Lecturer, Department of Pedodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai -600 077, Tamil Nadu, India.

Email Id: geosdch@gmail.com,

Telephone Number: 7358433246

Reference

1. Jayashri prabakar, Joseph John, Srisakthi D.Prevalence of Dental caries and treatment needs among school going children of Chandigarh.Ind J Dent Res.2016; 27 :547-52.
2. Ganesh R, John J. A correlation between dental caries and dental impact on daily living: A cross sectional study. Indian J Oral Sci.2013 ; 4: 70-74.
3. J.L. Winston, P.F. Cavanaugh Jr., A.R. Biesbrock, C.M. Cerone, H. Liu, J.D. Hyde, R.E. Singer. Relationship Between the Body Mass Index and Oral Health.General Session of the IADR, 2002 ;24 : 6-9.
4. Alm A, Isaksson H, Fahraeus C, Koch G, Andersson-Gare B, Nilsson M, Birkhed D, et al. BMI status in Swedish children and young adults in relation to caries prevalence. Swed Dent J. 2011;35(1):1-8.
5. Hobdell M, Petersen PE, Clarkson J, Johnson N. Global goals for oral health 2020. Int Dent J. 2003;53(5):285-288
6. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. The Lancet. 2004 ;363 (9403): 157–63.
7. Laura mariaGavrila. Mixed dentition caries predictor of carious activity in permanent dentition. Romanian Journal of Oral Rehabilitation. 2013; Vol. 5 : 1-3.
8. Turnbaugh,P.J.et al. Structure,function and diversity of the healthy human microbiome. Journal of the human microbiome project consortium. 2012 ; vol 486: 207 – 214.
9. Hobdell M, Petersen PE, Clarkson J, Johnson N. Global goals for oral health 2020. Int Dent J. 2003;53(5):285-288.
10. Hooley M, Skouteris H, Millar L. The relationship between childhood weight, dental caries and eating practices in children aged 4–8 years in Australia 2004–2008. Pediatric obesity, 2012 ;vol. 7 : 461-470.
11. Modéer T, Blomberg CC, Wondimu B, Julihn A, Marcus C: Association between obesity, flow rate of whole saliva, and dental caries in adolescents. Obes 2010, 18:2367–2373.
12. Cinar AB, Murtomaa H: Interrelation between obesity, oral health and lifestyle factors among Turkish school children. Clin Oral Investig 2011, 15:177–184

13. Costacurta M, Di Renzo L, Bianchi A, Fabiocchi F, De Lorenzo A, Docimo R: Obesity and dental caries in paediatric patients. A cross-sectional study. Eur J Paediatr Dent 2011, 12:112–116.
14. Krishna HVNS, Manaswini E, Kumar VY et al.Association between Nutritional Status and Early Childhood Caries in Indian Children. J Int Soc Prev Community Dent. 2017;7(3):131-35.
15. Bhayat A, Ahmad MS et al.Association between body mass index, diet and dental caries in Grade 6 boys in Medina, Saudi Arabia.EastMediterr Health J. 2016 ;22(9):687-93.
16. Almerich-Torres T, Montiel-Company JM, Bellot-Arcís C et al. Relationship between caries, body mass index and social class in Spanish Children. Gac Sanit. 2017;31:499-504.
17. Kottayi S, Bhat SS, Hegde KS et al. A Cross-sectional Study of the Prevalence of Dental Caries among 12- to 15-year-old Overweight Schoolchildren.JContemp Dent Pract. 2016;17(9):750-54.
18. Liang JJ, Zhang ZQ, Chen YJ et al. Dental caries is negatively correlated with body mass index among 7-9 years old children in Guangzhou, China. BMC Public Health. 2016;16:638
19. Mitrakul K, Asvanund Y, Arunakul M et al. Assessing associations between caries prevalence and body mass index and nutritional data among children aged 6-12 years. Southeast Asian J Trop Med Public Health. 2016;47(1):152-9.
20. Kim YS(1), Kim JH(1). Body mass index and oral health status in Korean adults: the Fifth Korea National Health and Nutrition Examination Survey.nt J Dent Hyg. 2017;15(3):172-78.

Brine Shrimp Lethality Assay of Grape Seed Extract

N.S.Harini[1], Lakshmi T[2], Sankari M[3]

Graduate Student[1], Reader[2], Department of Pharmacology, Professor[3], Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: *V. vinifera* contains many phenolic compounds,which includes Anthocyaninsthat can be found in the skin of the berries, hydroxycinnamic acids in the pulp and condensed tannins of theproanthocyanidins type in the seed. The aim of the study is to evaluate the brine shrimp lethality assay of ethanolic extract of grape seed.The objective is to correlate the cytotoxicity results with the known pharmacological actions of the plant.

Materials and Methods: The present study was conducted to evaluate invitro brine shrimp lethality assay of ethonolic extract of grape seed and correlate cytotoxicity results with known pharmacological actions of the plant. Novel cytotoxic, pesticidal compounds can be isolated from plant based sources through the investigation of cytotoxic activity against brine shrimps. Twenty nauplii were added into three replicates of each concentration of the plant extract. After 24,48 hours, the surviving brine shrimps were counted and percentage of mortality was assessed.

Results: It shows that grape seed extract is highly toxic to brine shrimp with increase in concentration. It was observed that at a concentration of 1mg per ml 25% of mortality is seen in brine shrimp in 24 and 48 hours. At 2mg concentration 35% of brine shrimp were killed in 24 hours and 55% were killed in 48 hours, at 3 mg per ml concentration 60% of the brine shrimp were killed and at 48 hours 80% mortality was seen. At a concentration of 4 mg per ml 80%mortality of brine shrimp was noticed in 24 hours and at 48 hours about 85% mortality was observed. Finally, at 5mg per ml concentration about 100% of eggs (Larvae) of brine shrimp were killed and this concentration proves the larvicidal efficacy of the grape seed ethanolic extract.

Conclusion: Brine shrimp lethality assay is an economical and cost effective method for investigating the cytotoxic activity. Hence from the results it can be concluded that grape seed is a promising candidate against brine shrimp and potents cytotoxic activity.

Key Words:*Eggs of Brine shrimp, grape seed extract, cytotoxicity, lethality assay, concentration, larvicidal activity*

Introduction

Vitis vinifera(grape seed extract) is a species of *Vitis*, which is native to the Mediterranean region, central Europe, and southwestern Asia[1]. There are currently between 5000 and 10,000 varieties of *Vitis vinifera* grapes available in nature. The fruit is a berry, which is known as a grape; in the wild species it is 6 mm (0.24 in) diameter and ripens dark purple to blackish with a pale wax bloom; in cultivated plants it is usually much larger, up to 3 cm (1.2 in) long, and can be green, red, or purple (black).[8] The species typically occurs in humid forests and in streamsides.[2-3]

V. vinifera contains many phenolic compounds,which includes Anthocyanins that can be found in the skin of the berries, hydroxycinnamic acids in the pulp and condensed tannins of theproanthocyanidins type in the seeds[4].

Stilbenoids can be found in the skin and in wood.[9-10].Stilbenoids*Trans*-resveratrol is a phytoalexin produced against the growth of fungal pathogens such as *Botrytis cinerea* and delta-viniferin are another grapevine phytoalexin produced following fungal infection by *Plasmopara viticola*Anthocyanins*Vitis vinifera* red cultivars are rich in anthocyanins that impart their colour to the berries (generally in the skin).[5-6] The 5 most basic anthocyanins found in grape are:Cyanidin-3-O-glucoside **,** Delphinidin-3-O-glucoside**,**Malvidin-3-O-glucoside**,**Petunidin-3-O-glucoside**,**Peonidin-3-O-glucoside[7,14,15].

Innovation of antimicrobials has long paved the way for human health [8].Its well-known fact that various cell line studies are there to prove the cytotoxicity efficacy of the herbal extract [16].

Aim

The aim of the study is to evaluate the brine shrimp lethality assay of ethanolic extract of grape seed.

Objective

The objective is to correlate the cytotoxicity results with the known pharmacological actions of the plant.

Materials and Methods

Grape seed extract was obtainedfrom green stem Chem. Herbel extracts and formulations, Bangalore.

Brine Shrimp Lethality Assay

The eggs of Brine shrimp were procured from Philadelphia, USA. In a small water tank containing sea water, the eggs were incubated for 48 hours for hatching. Required light was provided with Philips 40 Watts lamp for 12 hours cycle. After 48 hours, the larvae were used for the experiments. The nauplii of Brine shrimp were challenged in different test tubes containing 10 mL of sea water and 20 larvae. To this, extracts of leaves at different concentrations (1, 2 3, 4 and 5 mg/mL) were added. After 48 hours, the viability of larvae was observed and mortality was recorded. Nauplii were considered dead when they were immobile and stayed at the bottom of the test tubes. The percent mortality of brine shrimp was calculated with the formula given below.

Results

[% mortality = No. of brine shrimp dead /No. of brine shrimp introduced x 100]

Table 1: Brine shrimp lethality assay of grape seedextract

Sample	Incubation Period	Concentration(mg/ml)				
		1 mg/ml	2 mg/ml	3mg/ml	4 mg/ml	5mg/ml
Grape seed extract	24 h	25	35	60	80	100
	48 h	25	55	85	85	100

The results obtained from different concentrations of brine shrimp lethality assay are given in table1. It shows that grape seed extract is highly toxic to brine shrimp with increase in concentration. It was observed that at a concentration of 1mg per ml 25% of mortality is seen in brine shrimp in 24 and 48 hours. At 2mg concentration 35% of brine shrimp were killed in 24 hours and 55% were killed in 48 hours, at 3 mg per ml concentration 60% of the brine shrimp were killed and at 48 hours 80% mortality was seen. At a concentration of 4 mg per ml 80%mortality of brine shrimp was noticed in 24 hours and at 48 hours about 85%

mortality was observed. Finally, at 5mg per ml concentration about 100% ofeggs(Larvae) of brine shrimp were killed and this concentration proves the larvicidal efficacy of the grape seed ethanolic extract.

Discussion

The brine shrimp lethality assay is considered a useful tool for preliminary assessment of toxicity. It has also been suggested for screening pharmacological activities in plant extracts. However, we think that it is necessary to evaluate the suitability of the brine shrimp methods before they are used as a general bio-assay to test natural marine products for pharmacological activity [17].

The shrimp lethality assay was proposed by Michael et al. [18], and later developed by Vanhaecke et al. [19], and Sleet and Brendel [20]. It is based on the ability to kill laboratory-cultured *Artemia* nauplii brine shrimp. The assay is considered a useful tool for preliminary assessment of toxicity [21], and it has been used for the detection of fungal toxins [22], plant extract toxicity [23], heavy metals [24], cyanobacteria toxins [25], pesticides [26], and cytotoxicity testing of dental materials [27].

On the other hand, although most researchers have made use of the hatched nauplii, other assays based on the inhibition of hatching of the cyst (encased embryos that are metabolically inactive) have also been used [28]. Brine shrimp lethality bioassay is a standard method to investigate cytotoxicity of herbal extracts. This method is cost effective hence easily employed in vitro to check the larvicidal efficacy of the plant extract[29].***Vitis vinifera*** is a species of *Vitis*, which is native to the Mediterranean region, central Europe, and southwestern Asian region[30,31].

The grapevine (*Vitis vinifera*) belongs to the family Vitaceae, which comprises about 60 inter-fertile wild *Vitis* species distributed in Asia, North America and Europe under subtropical, Mediterranean and continental–temperate climatic conditions. It is the single *Vitis* species that acquired significant economic interest over time; some other species, for example the North American *V. rupestris*, *V. riparia* or *V. berlandieri*, are used as breeding rootstock due to their resistance against grapevine pathogens, such as *Phylloxera*, *Oidium* and mildews. Indeed, a great majority of cultivars widely cultivated

for fruit, juice and mainly for wine, classified as *Vitis vinifera* L. subsp. *vinifera* (or *sativa*), derive from wild forms [*Vitis vinifera* L. subsp. *sylvestris* (Gmelin) Hegi][32]

The wild grapevine is a heliophilous liana growing generally along river banks, and in alluvial and colluvial deciduous and semi-deciduous forest [33]. It is distributed in a wide area from western Europe to the Trans-Caucasian zone and around the Mediterranean Basin, except the most southern infra-Mediterranean and non-Mediterranean [34].

The present distribution of the wild grapevine is highly fragmented, in disjoint micro-populations or metapopulations, with few individuals, at least in the western part of the Mediterranean Basin. Anthropogenic pressure on their natural habitats and pathogens introduced from North America during the second part of the 19th century, may explain the progressive decline of wild grape populations[34].

The '*Phylloxera* crisis' that affected European vineyards had a considerable impact on both cultivated varieties and wild grapes. As a result, modern wild grapevines are endangered and threatened with extinction [34]. The future of *Vitis vinifera* subsp. *sylvestris* represents a major stake in biodiversity conservation.

The cultivation and domestication of the grapevine appears to have occurred between the seventh and the fourth millennia BC, in a geographical area between the Black Sea and Iran[35]. From this area, cultivated forms would have been spread by humans in the Near East, Middle East and Central Europe. As a result, these areas may have constituted secondary domestication centres[36]

Indirect evidence of ancient winemaking[37] is provided by the discovery of significant quantities of vinification residues (tartaric acid) with terebinth resin in clay jars, dating back to the end of the seventh millennium BC. In the Near East, numerous archaeological grape seeds attributed to the cultivated grapevine were found in Chalcolithic and mid Bronze Age archaeological levels[38].

From the eastern Mediterranean areas, grape cultivation seems to have spread gradually westwards. In Greece and Crete, the beginnings of viticulture would have started during the fifth millennium BC[39].

In Italy, the most ancient testimonies of grapevine cultivation date back to the ninth century BC[40]. In Spain and in the Maghreb, the Phoenician influence during the first part of the last

millennium BC appears to have played a significant part in the establishment and development of viticulture and viniculture[41]. Finally, it is currently thought that the emergence of viticulture in France was concomitant with the foundation of Marseille (600 BC) by the Greek Phocaeans [39]. Viticulture seems to have extended rapidly in southern France from the fifth century BC. Important coastal exchanges and trading centres such as Lattes (Hérault) would have also played an important role in this process. After the Roman conquest, viticulture was well established in the Languedoc region, reaching its height between the end of the first century and the second century AD.

By way of the 'Narbonnaise' route, viticulture extended into Aquitaine (western France) during the first century AD. During this same period it also spread northwards, in the Rhône, Loire and Seine valleys [40]. When the wine production of the Narbonnaise area started threatening the hegemony of Italian wines, the Emperor Domitian ordered the destruction of half of the region's vineyards. However, it appears that the Emperor's edict (92 AD) was not applied[40, 41].

Archaeological investigations have revealed a considerable development of viticulture and viniculture between the first and the end of the second century AD. It is only approx. 300 AD that Emperor Probus withdraws Domitien's prescriptions. From the fourth century onwards, while the Christian faith spread its influence throughout Europe, viticulture and viniculture again experienced a geographical expansion.

Although this model of development, expansion and diffusion of viniculture and viticulture in France (and more generally for the north-western Mediterranean areas) is well documented from archaeological and historical points of view, the ancestral cultivars and the varietal diversification process through time and space are not well known.

In spite of the important corpus of bio-archaeological, morphological, historical and genetic data available, the identity of former cultivars, history, biogeography and mechanisms of grapevine domestication remain obscure.Morphological criteria for the identification of archaeological remains (seeds and wood) attributed to *Vitis vinifera* are highly incomplete, and those ancient texts mentioning various cultivated grapevines are not exploitable to characterize ancient varieties.

In the present state of research, in France and elsewhere, studies on seeds are still too limited to allow a precise discrimination between wild and cultivated grapes [41].Although

informative, the main limits of these former studies concern the absence or the inadequacy of modern reference collections on which they are based. In every case, they represent local or regional studies and concern a reduced number of cultivars and wild specimens.The present study is based on the geometrical analysis of grape seed structure. It aims to test shape criteria in order to discriminate between wild forms and modern cultivars, interpret changes that have occurred during domestication, quantify the phenotypic diversity in the wild and the cultivated compartments and interpret shape diversity in relation to the supposed geographical origin (country or region) and parentage evidenced by genetic approaches.

Although the analysis of genetic diversity based on living material is very successfulon the first analyses based on ancient DNA, using a few microsatellite markers, though encouraging, have not yet provided significant results for the identification of ancient grapes. Shape characterization of cultivated varieties combined with genetic data should allow a better understanding of the changes that have occurred during domestication and finally identification of ancestral forms of current cultivars, based on the analysis of archaeological grape seeds.

Conclusion

The grapevine (*Vitis vinifera*) belongs to the family Vitaceae, which comprises about 60 inter-fertile wild *Vitis* species distributed in Asia, North America and Europe under subtropical, Mediterranean and continental–temperate climatic conditions. It is the single *Vitis* species that acquired significant economic interest over time; some other species, for example the North American *V. rupestris*, *V. riparia* or *V. berlandieri*, are used as breeding rootstock due to their resistance against grapevine pathogens, such as *Phylloxera*, *Oidium* and mildews.

Indeed, a great majority of cultivars widely cultivated for fruit, juice and mainly for wine, classified as *Vitis vinifera* L. subsp. *vinifera* (or *sativa*), derive from wild forms [*Vitis vinifera* L. subsp. *sylvestris* (Gmelin) Hegi]Grape seed extract possess lots of medicinal value[11-12]. Brine shrimp lethality assay is an economical and cost effective method for investigating the cytotoxic activity[13]. Hence from the results it can be concluded that grape seed is a promising candidate against brine shrimp and potents cytotoxic activity.

Corresponding Author

M.Sankari,

Professor, Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai-600077, Tamil Nadu,India.

Phone Number: 9840285905

Email Id: msankari@gmail.com

References

1. Meyer BN, Ferrigni NR, Putnam JE, Jacobsen LB.Brine shrimp: A convenient general bioassay for active plant constituents. Planta Med. 1982;45:31-4.
2. Persoone G.Proceeding of the international symposium on brine shrimp; Artemia salina. Belgium: University press. 1988;1-3.
3. Bagchi D, Bagchi M, Stohs SJ, Das DK, RS D, Charles A.Free radicals and grape seed proanthocyanidin extract importance in human health and disease prevention. Toxicol. 2000;148:187-97.
4. Almeida PA, Silva TMS, Echevarria A. Mesoionic5-alkyl-1,3-dithiolium-4-thiolates: Synthesis and brine shrimp toxicity.Heterocycle Comm. 2002;8:593-600.
5. Yamakoshi J, Kataoka S, Koga T, Ariga T. Proanthocyanidin-rich extract from grape seeds attenuates the development of aortic atherosclerosis GSE and Endothelial Function 277 in cholesterol-fed rabbits. Atherosclerosis. 1999;142 (1):139–149.

6. Brooker S, Martin S, Pearson A; Double-blind, placebo-controlled, randomised phase II trial of IH636 grape seed proanthocyanidin extract (GSPE) in patients with radiation-induced breast induration. Radiotherapy and Oncology. 2006;79(1):45-51
7. Boik J.Biochemical assay brine shrimp;. Minnesota, USA: Oregon Medical Press; 2001:1;17
8. Ozçelik B, Kartal M, Orhan I.Cytotoxicity, antiviral and antimicrobial activities of alkaloids, flavonoids, and phenolic acid,grapeseedextract. Pharm Biol. 2011;49(4):396-402.
9. Sawer IK, Berry MI, Ford JL. Biochemical mutations of brine shrimp. Lett Appl Microbial. 2005;40:24-9.
10. Lisgarten JN, Coll M, Portugal J, Wright CW, Aymami J.Brine shrimp lethality assay. Nat Struct Biol. 2002;9:57-60.
11. Dassonneville L, Lansiaux A, Wattelet A, Wattez N, Mathieu C, Van MS;Cytotoxicity and cell cycle effect of the grape seed extract Eur J Pharmacol. 2000;409:9-18.
12. Chung KT, Wong TY, Wei CI, Huang YW, Lin Y. Tannins and human health: a review. Crit Rev Food Sci Nutr.,biochemical reactions of grape seed extract1998;38:421-464.
13. Akiyama H, Fujii K, Yamasaki O, Oono T, Iwatsuki K.Antibacterial action of several tannins against grape seed extract. J Antimicrob Chemother. 2001;48:487-91
14. Waikedre J, Dugay A, Barrachina I, Herrenknecht C, Cabalion P, Fournet A.Chemical composition and antimicrobial activity of the essential oils of grape seed extract from New Caledonian Citrus macroptera and Citrus hystrix. Chem Biodivers.2010;7:871-7.
15. Uddin N, Hasan MM, Hossain MM, Sarker A, Hasan AHMN, Islam AFMM;In vitro 艹-amylase inhibitory activity and In vivo hypoglycemic effect of methanol extract of grape seed extract . Asian Pac J Trop Biomed. 2014;4(6):473-9.
16. Priyangaa Sathasivam, T. Lakshmi Department of Pharmacology, Saveetha Dental College, Saveetha University, Chennai, Tamil Nadu, India; Brine shrimp lethality assay of Sesbania grandiflora ethanolic extract – in vitro study;2017 Journal of Advanced Pharmacy Education & Research ;

17. José Luis Carballo*, Zaira L Hernández-Inda, Pilar Pérez and María D García-Grávalos; A comparison between two brine shrimp assays to detect in vitro cytotoxicity in marine natural products; BMC Biotechnology 2002, 2:17 p. 1-5.
18. Michael AS, Thompson CG, Abramovitz M: Artemia salina as a test organism for a bioassay. Science. 1956, 123: 464.
19. Vanhaecke P, Persoone G, Claus C, Sorgeloos P:Proposal for a short-term toxicity test with Artemia nauplii. Ecotoxicol Env Safety. 1981, 5: 382-387.
20. Sleet RB, Brendel K:Improved methods for harvesting and counting synchronous populations of Artemia nauplii for use in developmental toxicology. Ecotoxicol Env Safety. 1983, 7: 435-446.
21. Solís PN, Wright CW, Anderson MM, Gupta MP, Phillipson JD:A microwell cytotoxicity assay using Artemia salina. Plant Med. 1993, 59: 250-252.
22. Harwig J, Scott P:Brine shrimp (Artemia salina L.) larvae as a screening system for fungal toxins. Appl Microbiol. 1971, 21: 1011-1016.
23. McLauglin JL, Chang CJ, Smith DL:Bench top" bioassay for the discovery of bioactive natural products: an update. In: Studies in Natural Products Chemistry. Edited by: AU Rahman. 1991, Elsevier, 383-409.
24. Martínez M, Del ramo J, Torreblanca A, Díaz-Mayans J:Effect of cadmium exposure on zinc levels in the brine shrimp Artemia partenogenética. Aquaculture. 1998, 172: 315-325. 10.1016/S0044-8486(98)00431-1.
25. Jaki B, Orjala J, Bürji HR, Sticher O:Biological screening of cyanobacteria for antimicrobial and molluscicidal activity, brine shrimp lethality, and cytotoxicity. Pharm Biol. 1999, 37: 138-143. 10.1076/phbi.37.2.138.6092.
26. Barahona MV, Sánchez-Fortún S:Toxicity of carbamates to the brine shrimp Artemia salina and the effect of atropine, BW284c51, iso-OMPA and 2-PAM on carbaryl toxicity. Env Pollut. 1999, 104: 469-476. 10.1016/S0269-7491(98)00152-3.
27. Pelka M, Danzl C, Distler W, Petschelt A:A new screening test toxicity testing of dental materials. J Dent. 2000, 28: 341-345. 10.1016/S0300-5712(00)00007-5.

28. Migliore L, Civitareale C, Brambilla G, Di delupis GD: Toxicity of several important agricultural antibiotics to Artemia. Wat Res. 1997, 31: 1801-1806. 10.1016/S0043-1354(96)00412-5.

29. Indhumathi.M , Lakshmi.T;Brine Shrimp Lethality Assay of Acacia catechu Bark Ethanolic Extract; Int. J. Pharm. Sci. Rev. Res., 41(2), November - December 2016; Article No. 03, Pages: 13-14.

30. Dhinahar.S, Lakshmi.T.Role of botanicals as antimicrobial agents in management of dental infections .International journal of pharma and biosciences., 2(4), 2011, B 691-703.

31. Lakshmi.T, Geetha R.V, Anitha Roy"In vitro Evaluation of Anti-bacterial Activity of Acacia catechu willd Heartwood Extract." International journal of Pharma and Biosciences. 2(1), 2011, B188-192.

32. Rossetto ,Sefc , Crespan;Evolution and history of grapevine (Vitis vinifera) under domestication: new morphometric perspectives to understand seed domestication syndrome and reveal origins of ancient European cultivars;Ann Bot. 2010 Mar; 105(3): 443–455.

33. Levadoux;Thermal thresholds as predictors of seed dormancy release and germination timing: altitude-related risks from climate warming for the wild grapevine Vitis vinifera subsp. Sylvestris; Ann Bot. 2012 Dec; 110(8): 1651–1660.

34. Arnold ;Disinfection of Nauplii of Artemia franciscana by Ozonation; The Progressive Fish-Cttlturist 6ll:1.19 l5l, 1998

35. Châtaignier,McGovern and Rudolph, Zohary and Hopf;Genetics of berry colour and anthocyanin content variation in grapevine (Vitis vinifera L. subsp. vinifera; Oeiras, January 2011; chapter-1; p-3-338.

36. Grassi ,Arroyo-Garcia 2006;The Current Status of Wild Grapevine Populations (Vitis vinifera ssp sylvestris) in the Mediterranean Basin; chapter 3; p-2-23.

37. Valamoti ; Grape-pressings from northern Greece: The earliest wine in the Aegean?; Antiquity 81(311):54-61 · March 2015.

38. Di Vora and Castelletti**; Carving out a new market niche: Historic world of wines;** BIO Web of Conferences, 5 (2015) 03002**.**

39. Brun and Laubenheimer; Bioarchaeological Insights into the Process of Domestication of Grapevine (Vitis vinifera L.) during Roman Times in Southern France; 8(5); May 2013; p: 1-13.

40. Laubenheimer and Brun; Archaeobotany, vine growing and wine producing in Roman Southern France; Journal Of Archeological Science; Volume 37, Issue 1, January 2010, Pages 139-149

41. Stummer, Kislev, Di Vora and Castelletti, Mangafa and Kotsakis, Marinval, Jacquat and Martinoli, Bouby and Marinval, Terral;Evolution and history of grapevine (Vitis vinifera) under domestication: new morphometric perspectives to understand seed domestication syndrome and reveal origins of ancient European cultivars; Annals of Botany, Volume 105, Issue 3, 1 March 2010, Pages 443–455.

CarabelliTraitinMaxillaryMolars

Hansini B. Ajantha[1], Abilasha Ramasubramanian[2], S. Pavithra Priyadarshoni[3]

Graduate Student[1], Senior Lecturer[2],Department of Oral pathology,

Tutor[3], Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: Carabelli's trait occurs on the palatal surface of the mesiopalatal cusp of permanent upper molars and deciduous upper second molars. The distribution of the carabelli trait is highly variable in different regions and races of the world. The trait is entirely absent in some individuals, while present in others, in a variety of forms. To determine the prevalence of carabelli trait in a group of people visiting saveetha dental college.

Materials and Methods: In this descriptive study, 200 maxillary molars were examined. Examination of maxillary molars was done by direct intra oral examination. Morphological details of the maxillary molar crown, gender of the subjects were recorded and Dahlberg classification was used. Photographs were taken.

Results: The prevalence of the trait was 76.5%, with significantly different distribution between males and females. Class 5 was the most frequent configuration (17%) and Class 7 was the least frequent(2%). The prevalence of carabelli trait is seen more in first maxillary molars than second maxillary molars.

Conclusion: The incidence of cusp of carabelli is lower compared to the European studies, showing a frequency of 76.5% with Class 5 (small tubercle) being more prominent and Class 7 being less prominent and more prevalence of bilateral symmetry and a bit of male prediction.

KeyWords*: Carabelli trait, Dahlberg classification, Prevalence, Maxillary molars, Cusp of carabelli*

Introduction

Teeth are an excellent object for the study of human variation and are used to address questions in numerous disciplines including anthropology. Morphological features of teeth are controlled to a greater extent by genes than skeletal features. In contrast to bone, hard dental tissues do not remodel once they have formed. Consequently, tooth morphology changes through life only as a result of pathological (e.g. caries, resorption, trauma) and age-dependent processes (e.g. attrition, abrasion). Teeth are very resistant to post-mortem degradation and to mechanical trauma. So they are generally better preserved than bones in archaeologically derived human remains. An additional advantage of teeth is that their morphological features are observable on extracted specimens, on specimens from skeletal-dental remains, on dental impressions or cast reproductions, and directly in the mouth of a living person. Tooth morphology has been known to show variation from one ethnic group to another. One such morphological characteristic which has warranted a lot of investigations in different ethnic groups of the world is the trait of Carabelli [1]. George carabelli in 1842 first described a tubercle on the lingual surface of the mesiolingual cusp of maxillary molars. This protuberance goes by other denominations like an accessory cusp, a fifth lobe, a supplemental elevation, a fifth cusp, molartubercle, carabelli's cusp, a carabelli's anomaly and a carabelli's tubercle [2]. The phenotypical appearance of the trait is attributed to a dominant Mendelian gene and also to the intake of fluorides, nutrients and the size of the jaws. Therefore, it s a result of interaction between genetic and environmental factors [3]. Carabelli cusp is expressed in several degrees and different frequencies between different populations. The variation of molar tubercle is significant in the diagnosis of carious lesion and in cases of anthropological studies and those related to forensic dentistry [4]. The aim of this study is to evaluate the prevalence and symmetrical trait of carabelli in permanent dentition of individuals visiting saveetha dental college.

Materials and Methods

Patients, who came for treatment to Saveetha Dental College, were examined for this study. After obtaining permission from the institutional research committee, the study was carried out for a period of 4 months from February 2015 to may 2015. Healthy permanent maxillary molars, devoid of dental caries and attrition found during intra oral examination of the patients were

considered in this study. The required consent and assent from the patients were obtained and those willing to participate in this study were included. During the study period, 200 permanent maxillary molars were examined of age group from 20 years old to 40 years old people. On intra oral examination, symmetrical appearance of carabelli trait and prevalence of carabelli trait in first permanent maxillary molars and second permanent maxillary molars were recorded. Prevalence of carabelli trait in maxillary first molars and maxillary second molars, according to gender was also recorded. Dahlberg's classification [5] was also used for the determination of degree and expression of carabelli trait.

Results

In this study, 76.5% of the maxillary molars examined have shown the carabelli trait. Out of 200 maxillary molars examined in this study, 132 were first maxillary molars and 68 were second maxillary molars. The carabelli trait is more in maxillary first molars (93.9%) than maxillary second molars (42.6%). [Table 1] Bilateral symmetry (76%) of carabelli trait is more than unilateral symmetry (24%) among the examined individuals. [Table 2]. On using Dahlberg's classification for determining the degree and expression of carabelli cusps, Class 5 is the most common configuration (23.5%) and Class 7 is the most least configuration (1%). [Table 3] Out of 200 maxillary molars examined, 130 were from male individuals and remaining 70 from female individual. Based on gender predilection, the incidence of carabelli trait is bit higher in males (80%) than females (70%). [Table 4].

Table 1: Prevalence of carabelli trait in first and second maxillary molars

	FREQUENCY	PERCENTAGE
Maxillary first molars	124/132	93.9%
Maxillary second molars	29/68	42.6%
TOTAL	153/200	76.5%

Table 2: Prevalence of symmetrical trait of carabelli

SYMMETRY	FREQUENCY	PERCENTAGE
Bilateral symmetry	152	76%
Unilateral symmetry	48	24%
Total	200	100%

Table 3: Dahlberg's scale for the determination of degree and expression of the carabelli trait

-

DAHLBERG'S SCALE	FREQUENCY	PERCENTAGE
0	47	23.5%
1	10	5%
2	25	12.5%
3	12	6%
4	29	14.5%
5	48	24%
6	27	13.5%
7	2	1%
TOTAL	200	100%

Table 4: Presence of carabelli trait based on gender predilection

	PRESENCE	ABSENCE	TOTAL
MALES	104 (80%)	26	130 (65%)
Maxillary first molars	82	4	86
Maxillary second molars	22	22	44
FEMALES	49 (70%)	21	70 (35%)
Maxillary first molars	42	4	46
Maxillary second molars	7	17	24

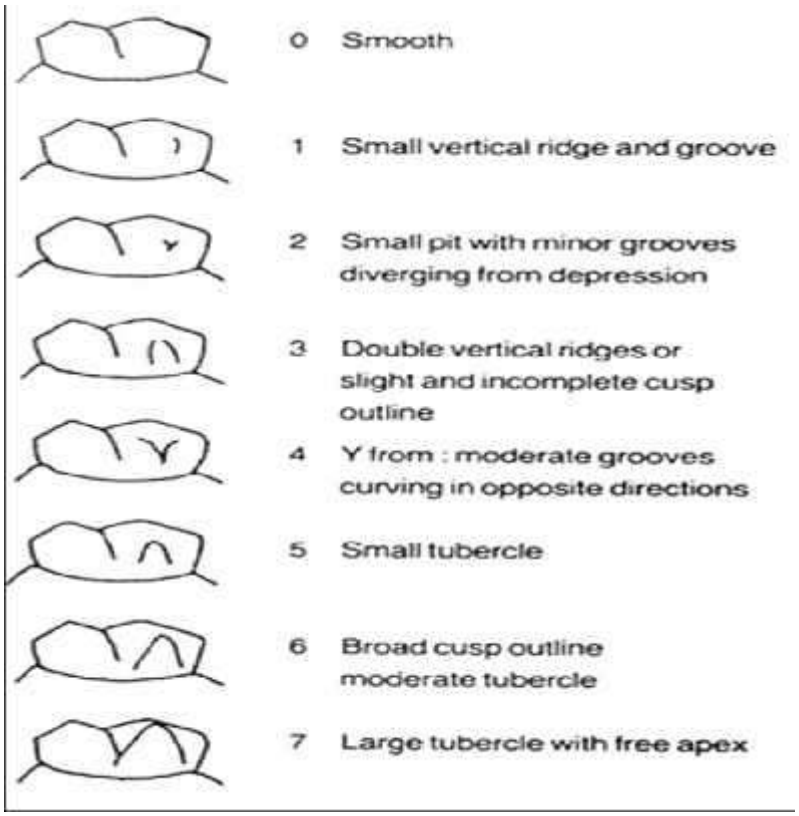


Figure 1: Dahlberg's scale for Carabelli trait

Limitations

This study was not without any limitations. Sample size was small compared to other studies done related to cusp of carabelli or molar tubercle in maxillary molars. Furthermore, the participants were selected from only one institute and the age group of the participants is limited, making generalization of the results difficult. Cross-sectional nature of the study and absence of any control groups might not have served our purpose of assessing the attitude and ascertaining the importance of undergraduate education in Psychiatry. Some observed differences on group wise and sex-wise comparison may be attributed solely to the cohort effect.

Discussion

Carabelli's trait occurs on the palatal surface of the mesiopalatal cusp of permanent upper molars and deciduous upper second molars. It is a quasi continuous variable, i.e. it can be either present or absent, but when present, it exhibits continuous variation in expression. The expression of the

trait varies from a slight or distinct single furrow, pit, double furrow, y-shaped furrow, or slight protuberance lacking a free apex, to a small, moderate or large cusp, which occasionally equals in size the main occlusal cusp. A pit and a furrow - single, double, y-shaped are negative expressions of the trait, whereas a protuberance and a cusp are its positive expressions. The frequency of the carabelli trait varies from population to population or within the population groups [6], [7]. The cusp of carabelli has never been reported to interfere with occlusion, no matter how large it is, but, has a great potential for the development of tooth decay. The cusp expression has a tubercle in addition to the groove; this tubercle may act as a physical barrier against the cleansing mechanisms of a tooth brush and physiological cleaning by the tongue movement and the salivary flow. In cases where the carabelli trait was absent, caries was not detected which supports the idea that the carabelli structure presents a foci for plaque accumulation and caries development. The method by which caries was examined, i.e., avoiding apically directed force, might have contributed to the low caries prevalence [8], [9], [10].

The presence of the carabelli trait might be due to both genetic and environmental factors [11]. There is still a controversy in the literature with regards the degree of genetic control in the prevalence and expression of this trait. While a field theory suggests that the trait is induced, and therefore, is subjected to environmental stress, clonal model theory suggests that the trait is intrinsic, and therefore, it would be less responsive to environmental factors [12]. Being a distinguishing feature or characteristic of an individual, carabelli trait is frequently used in anthropological, clinical and forensic studies [13]. The significance of carabelli cusp for evolutionary biology is that it provides a well-documented glimpse at the origin of a new cusp. Over the evolutionary history of mammals, which has experienced a large-scale increase in dental complexity, major dental traits have evolved that began at early evolutionary stages as peripheral features, low on a tooth crown, and developed presumably late in ontogeny. Transforming a small, low peripheral cusp into a centrally located, large cusp must be accomplished by shifting the initiation of that cusp earlier in ontogeny. However, origin of a new cusp in the first place, to use carabelli expression as a model, can occur as a byproduct of natural variation in the spacing of enamel knots and offset of morphogenesis, which impacts inter cusp spacing and tooth size. It is rarely possible to study population level variation in the early evolutionary stages of the origin of a new cusp in extinct species.

The findings of the present study showed an overall prevalence of 76.5% in the surveyed group, with Class 5 (23.5%) being more prominent and Class 7 (1%) being less prominent. Based on the previous studies of carabelli trait on racial basis, it is proved that, the Mongoloids had a low prevalence whereas Caucasians had a high prevalence [14], [15]. Among the Caucasians, Asiatic population showed a lower trait, whereas, European population showed higher trait with a prevalence of 96.6% with class 6 predominance [16], [17]. When the trait symmetry is considered, bilateral symmetry (76%) is more predominant than unilateral symmetry (24%) and the prevalence of carabelli trait is more in maxillary first molars (93.9%) than maxillary second molars(42.6%). Bilateralism of the trait, in this study, was very high. This supports the findings of other researchers [18],[19], which they agree that bilateralism of the trait is more frequent. The presence of the trait can't be used as a sole criterion for identification of maxillary molars as it is also found to occur in maxillary second molars. In this study, a bit of higher prevalence is seen in males (80%) than females (70%), which may be due to the reduction of crown size in females. Many studies [20], [21], [22], [23] failed to show any sex dimorphism in the occurrence of the trait, although some investigators [24], [25] observed a sex-linked pattern. Thus, it is verified that the sexual dimorphism related to the molar tubercle changes among the different studies and populations.

Conclusion

The incidence of cusp of carabelli is lower compared to the European studies, showing a frequency of 76.5% with Class 5 (small tubercle) being more prominent and Class 7 being less prominent and more prevalence of bilateral symmetry and a bit of male prediction. Dentists are advised to perform a careful examination to the palatal surfaces of the maxillary first and second molars to rule out the presence of the cusp of carabelli, as they are way more prone to plaque accumulation, which in turn can lead to development of dental caries. Future studies should be made to assess the relationship of the carabelli trait to genetics and its heritability.

Corresponding Author

S. PavithraPriyadarshoni

Tutor, Department of Public Health Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id: drpavithraphd88@gmail.com

Contact Number: 9176222104

References

1. Salako NO, Bello LL. Prevalence of the carabelli trait in Saudi Arabian children. Tropical dental journal. 1998 Dec:11-4.
2. Falomo OO. The cusps of Carabelli: frequency, distribution, size and clinical significance in Nigeria. West African journal of medicine. 2002;21(4):322-4.
3. Syed S, Odusanya S, Abdelbagi M, Razak P, Wahab M, Meer Z. The prevalence of fifth cusp (Cusp of Carabelli) in the upper molars in Saudi Arabian school students. International journal of morphology. 2012;30(2):757-60.
4. Mavrodisz K, Rózsa N, Budai M, Soós A, Pap I, Tarján I. Prevalence of accessory tooth cusps in a contemporary and ancestral Hungarian population. The European Journal of Orthodontics. 2007 Feb 22;29(2):166-9.
5. Dahlberg AS Analysis of American indain dentition. Dental Anthropology. Oxford: Pergamon Press; 1963. pp. 149-78
6. Scott GR. Population variation of carabelli's trait. Hum Biol 1980;52:63-78.
7. Mosharraf R. Prevalence of the carabelli trait in Iranian adolescents. SRM Journal of Research in Dental Sciences. 2013 Jan 1;4(1):12.

8. Van Dorp, C.S., Exterkate, R.A., Ten Cate, J.M., 1988. The effect of dental probing on subsequent enamel demineralization. ASDC J. Dent. Child. 55 (5), 343–347.
9. Warren, J.J., Levy, S.M., Wefel, J.S., 2003. Explorer probing of root caries lesions: an in vitro study. Spec. Care Dentist. 23 (1), 18–21.
10. Yassin, O.M., 1995. In Vitro Studies of the Effect of a Dental Explorer on the Formation of an Artificial Carious Lesion. ASDC J Dent Child 62 (2), 111–117.
11. Ferreira MA, Hespanhol LC, Capote TS, Gonçalves M, Campos DB, Alvares J. Presence and morphology of the molar tubercle according to dentition, hemi-arch and sex. International Journal of Morphology. 2010 Mar 1:121-5.
12. Keiser J.A, An Analysis of the Carabelli trait in the mixed deciduous and permanent human dentition; Archives of Oral biology 1984, 29: 403-406
13. Baskarraj M, Gupta YM, Kumari RR, Samuel AV, Kannan SS, Mahesh R. Forensic odontology: Supernumerary teeth, their importance, and a radiographic study in identifying supernumerary teeth. International Journal of Forensic Odontology. 2016 Jul 1;1(2):39.
14. King NM, Tsai JS, Wong HM. Morphological and numerical characteristics of the southern chinese dentitions. part II: traits in the permanent dentition. The Open Anthropology Journal. 2010.
15. Hsu JW, Tsai PL, Hsiao TH, Chang HP, Lin LM, Liu KM, Yu HS, Ferguson D. The effect of shovel trait on Carabelli's trait in Taiwan Chinese and Aboriginal populations. Journal of Forensic Science. 1997 Sep 1;42(5):802-6.
16. Kannapan JG, Swaminathan S. A study on a dental morphological variation. Tubercle of Carabelli. Indian journal of dental research: official publication of Indian Society for Dental Research. 2001;12(3):145-9.
17. RaminMoaharrak; Prevalence of the carabelli trait in Iranian adolescents; srmjds, vol 4, issue 1, jan-mar 2013.
18. Alvesalo, L., Nuutila, M., Portin, P., 1975. The cusp of Carabelli Occurrence in first upper molars and evaluation of its heritability. ActaOdontol. Scand. 33 (4), 191–197.
19. Joshi, M.R.,Carabelli trait on maxillary second deciduous and first permanent molars in Hindus. Arch. Oral Biol. 1975. 20, 699–700.

20. Rusmah, M., 1992. The cusp of Carabelli in Malaysians. Odontostomatol. Trop. 15 (1), 13–15.
21. Saunders, S.R., Mayhall, J.T., 1982. Development patterns of human dental morphological traits. Arch. Oral. Biol. 27, 45–49.
22. Thomas, C.J., Kotze Jr., T.W., Nash, J.M., The Carabelli trait in the mixed deciduous and permanent dentition of five South African populations. Arch. Oral. Biol.1986. 31, 145–147.
23. Gupta B, Gupta M. Sex identification in forensic odontology-a review of various methodology. International Journal of Forensic Odontology. 2016 Jan 1;1(1):9.
24. Kaul, V., Prakash, S., 1981. Morphological features of jat dentition. Am. J. Phys. Anthrop. 54, 123–127.
25. Kieser, J.A., Preston, C.B., 1981. The dentition of the Lengua Indians of Paraguay. Am. J. Phys. Anthrop. 55, 485–490.

Change in Angulation of Mandibular Third Molars after Orthodontic Treatment- A Cross-Sectional Study

Surabhi.R.Jain[1], A V Arun[2], Meenakshi Krishnan[3]

Graduate Student[1], Professor[2], Department of Orthodontics and DentofacialOrthopeadics

Senior Lecturer[3], Department of Oral Medicine & Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Background: The mandibular third molar is an unusual tooth in the oral cavity. The angulation and position keeps changing as the tooth erupts. The orthodontist need to be aware of the relationship of the mandibular third molars to the remaining teeth in the dental arch, as it can cause complications like impaction, mandibular anterior crowding. Thus theaim and objective of this study is to analyse the changes in mandibular third molar angulation, pre and post orthodontic treatment and also to evaluate the positional changes of the mandibular 3rd molars.

Materials and Method: The study was a cross sectional analytical study and data was retrospectively retrieved from archival records of patients visiting Saveetha dental college for orthodontic treatment. Pre and post Lateral cephalograms and orthopantomograph records of 20 orthodontic patients were selected. The mean age of the patients ranged from 20-35 years and all the cases had undergone extraction of mandibular first premolars for the orthodontic treatment. The angulation of the mandibular third molars was measured using OPG and the positional change was measured with lateralcephalograms after tracing the radiographs.

Result:A significant change in mandibular third molar angulation was observed on both right (10.4º) and left sides (13.3º) of the mandible, pre and post orthodontic treatment and positional change of1.04 and 1.31 cm was observed pre and post orthodontic treatment.

Conclusion: The present study suggests that premolar extraction therapy has a favourable effect on third molar angulation.

Keywords: *third molar, pre and post orthodontic, lateral cephalograms, orthopantamographs, impaction*

Introduction

Mandibular third molar is an atypical tooth, due to its variability in formation, variations in crown and tooth morphology and most posterior location in the jaw. Varies studies have been done on differences in presence or absence of the tooth, variations in morphology and size. But recent studies concentrate more on the calcification, position and angulation of mandibular third molars.

Mandibular third molar calcification begins at 8 to 10 years of age and root formation starts at the age of 15 and the tooth erupts between 17 to 30 years of age. Of all the teeth present in the oral cavity, third molar eruption presents the most complications. They are often impacted or retained or embedded, which can cause conditions such as pericoronitis (inflammationofpericoronal flap covering the coronal aspect of a developing tooth), cavities due to difficulty in brushing as it is in the posterior most location, root resorptions, alveolar bone loss leading food impaction and periodontitis, cystic processes (eg. dentigerous cysts most commonly associated with impacted third molars), or tumours.

Insufficient space in jaw leads to more distal eruption of third molars rather than erupting in its normal location. The difference in morphology like root curvature will also lead to displacement of the tooth.

When drawing orthodontic treatment plan, sometimes the mandibular third molars are ignored, as it is always assumed that, the tooth will be impacted and will be indicated for extraction. But as a dentist, the key treatment objective is to conserve all dental organs and to maintain maximum masticatory function. Nevertheless, it has been estimated that 54% of mandibular third molars are removed prophylactically, even though they do not present any subjective symptoms

The development of third molars, their influence on the dental arches and its eruption potential is an important consideration for both the dentist and the orthodontist when planning active treatment plan and long-term maintenance [1].The impaction of mandibular third molar is the most common problem [2].Approximately 43% of mandibular third molar

impactions may be classified as mesial impactions while maxillary third molars are mostly distally impacted [3]. The mandibular third molar tooth buds begin their formation at approximately 8 to 9 years of age and also show a great variation in in size, position, shape, root shape, and development time [4,5,6,7].One explanation for the third molar impaction is inadequate retro molar space due to limited resorption at the anterior aspect of the mandibular ramus [8,9,10,11].

The orthodontist need to be aware of the relationship of the mandibular third molars to the remaining teeth in the dental arch. The main aspects to be analysed are whether the third molars will erupt or become impacted, whether they will cause crowding of the mandibular anterior teeth, and whether the extraction of other teeth will prevent crowding and influence their eruption [12].Developing third molars continually change their angular positions and undergo important pre-eruptive rotational movements [11, 13,14].The causes for mandibular third molar impaction and prediction of its eruption have also been studied extensively. In contrast, the effect of orthodontic treatment on the developing third molars has not been subjected too much investigation. The purpose of this study is evaluate the change in the angulation of mandibular third molars pre and post orthodontic treatment andalso to evaluate the positional changes of the mandibular third molars with reference to the occlusal plane.

Materials and Method

The study was a cross sectional analytical study.Dental records of patients who had undergone fixed orthodontic treatment at the Department of Orthodontics, Saveetha Dental College and Hospital, Chennai, India, were obtained from the year 2014 to 2016. Out of 600 records collected, 20 records were randomly selected with inclusion criteria as meanagefrom 20-35 years,had undergone extraction of mandibular first premolars for the purpose of orthodontic treatment, and absence of any congenitally missing right and left mandibular third molars, with pre-treatment (T1) and post treatment (T2) Lateral cephalogramsand panoramic radiographs (orthopanmographs). Rest 580 records were excluded from the study. Tracing of pre and post treatment lateral cephalograms and orthopantamograms were done on a tracing paper. The angulation of the mandibular third molars was measured by tracingOPG with lines drawn from the midpoint of the ramus of the mandible as the horizontal reference point and long axis of the tooth as vertical reference point and compared with pre and post treatment radiograph for both right and left mandibular third molar. The positional change of the right and left mandibular 3rd molar was measured and compared using pre-treatment and

post treatment lateral cephalograms, with a line drawn from the anterior most part of the ramus of the mandible tothe mesial cusp tip of 3rd molar.The measurements were recorded and tabulated and was subjected to statistical analysis to find the mean difference in angulation and mean difference in position of the mandibular third molars on both right and left side of the jaw.

Results

The mean angular changes in mandibular third molars, pre andpost orthodontic treatment are depicted in table1. A mean difference of 10.4^0 and 13.3^0 was found in right and left mandibular third molars respectively. Graph 1 represents the right side angular changes of third molar seen in all the samples, while graph 2 represents the changes seen in left side of mandible.Table 2 represents mean positional changes of mandibular 3rd molar estimated from lateral cephalograms. The positional changes of mandibular third molars taking place before treatment was found to be 1.04cmand after orthodontic treatment it was found to be1.31cm and is represented in graph 3.

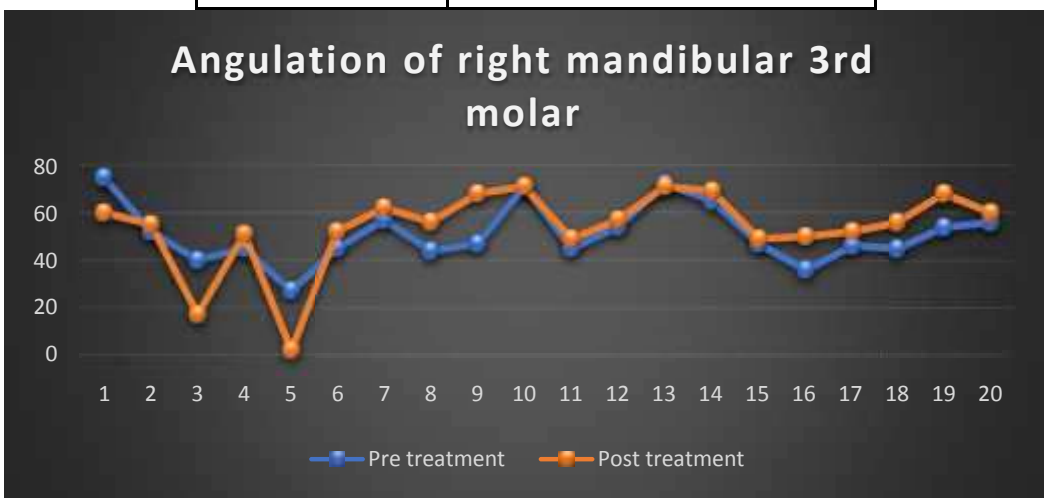
Table 1: Mean Angular changes pre and post orthodontic treatment (in degrees)

	Pre treatment	Post treatment	Mean Difference in angulation(in degrees)
Right mandibular 3rd molar	46.7	57.1	10.4
Left mandibular 3rd molar	43.7	51.3	13.3

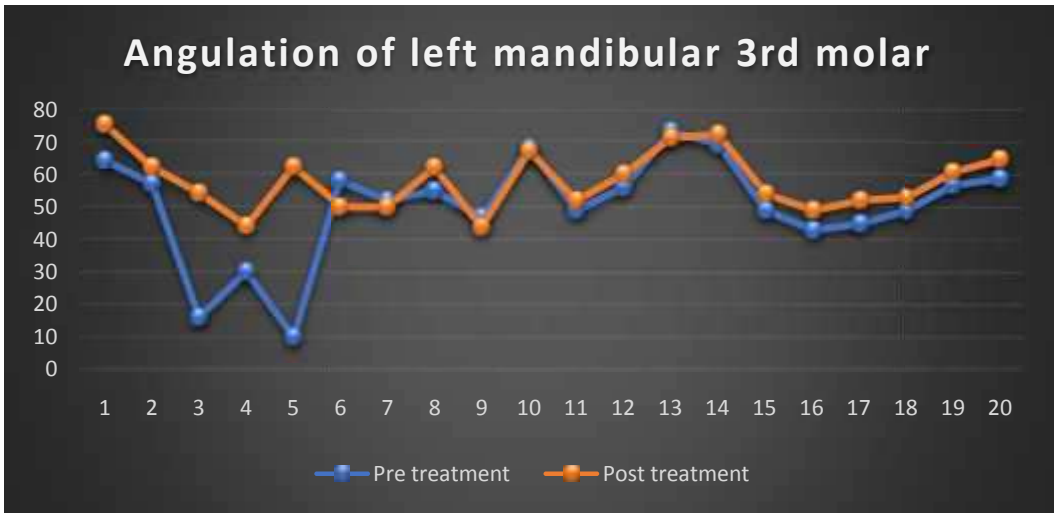
Table 2: Mean positional change of mandibular third molars in lateral cephalograms pre and post orthodontic treatment (in cms)

	Mean Distance (in cms)
Pre treatment	1.04

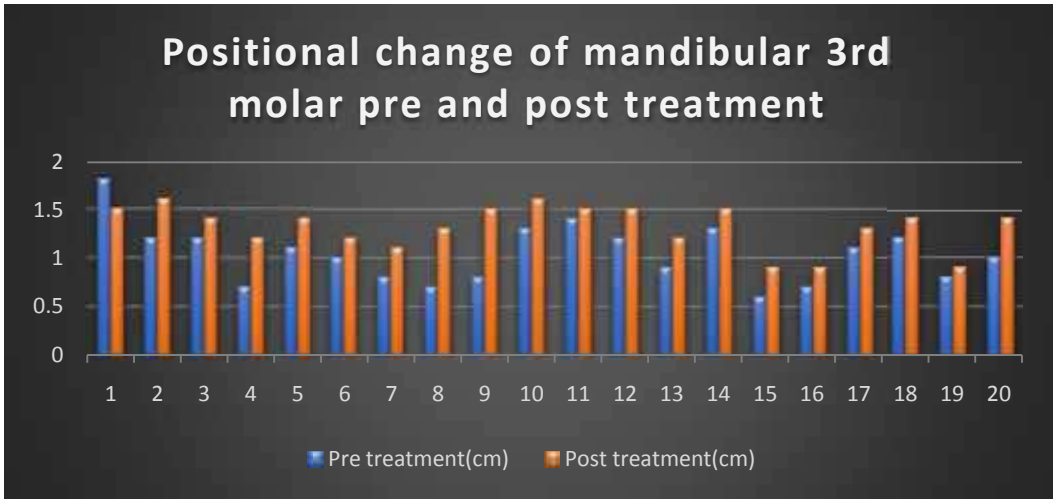
Post treatment	1.31



Graph 1: Right side angular changes of third molar pre and post orthodontic treatment (in degrees)



Graph 2: Left side angular changes of third molarpre and post orthodontic treatment(in degrees)



Graph 3: Positional changes of mandibular 3rd molarspre and post orthodontic treatment (in cms)

Discussion

Third molar impaction is a major clinical problem and if its eruption can be predicted at an early age during the course of the orthodontic treatment, then the occurrences of difficult impactionscan be avoided. In the study, a significant change in mandibular third molar angulation is seen on both right (10.4) and left sides (13.3) of the mandible, pre and post orthodontic treatment. Previous studies have used the occlusal plane and mandibular plane as the horizontal plane of reference to measure the angular changes of 3rdmolar. However, the changes in the occlusal plane, with treatment and modelling of the lower border of the mandible during growth may cause misinterpretations of third molar angulation [15].Measurements of third molar angulation on lateral cephalograms may be biased because of differences in angulation between the superimposed images [16].

Richardson has proved that measurement of third molar angulation on lateral cephalograms may be biased and can only be overcome if measurements are made on 60 degree head films of right and left sides [13, 14]. Studies have shown that panoramic radiographs are reliable tool for evaluation of angular changes and hence they were used in the present study. It also showed that there was not much significant difference in angulation in right and left mandibular third molars.

The findings of the present study also suggests that premolar extraction therapy has a favourable effect on third molar angulation. The mechanism may be that mesial movement of first molar, associated with extraction site closure,promotes mesial tipping of the third molar

bud.Elsey and Rock have stated that the closure of mandibular extraction sites commonly allows improvement in the position of unerupted third molars. [17, 18, 19]

Conclusion

Impaction or failure of mandibular third molars to erupt is mostly due to lack of space in the alveolar arch between the distal of the second molar and the ascending ramus, most posterior location, aberrant anatomy and abnormal eruption pattern, due to which extraction remains the treatment of choice. Contemporary dental practice aims at minimal intervention and retaining every functional component of dental arch. Orthodontic treatment mostly requires extraction of premolars for the required space management [20, 21].This, can be useful for movement of third molar mesially for normal eruption. This study aimed at estimating movement of third molar post orthodontic treatment.

The study reveals that:

- A change in angulation of third molar by 10.4^0 is seen on the right side and 13.3^0 is seen on right side of mandibular third molar.
- If there is a mesio angular impaction of $<10^0$ before the start of the orthodontic treatment, then there is a high chance that the tooth would get upright automatically by the time the orthodontic treatment is done.

Hence, post – orthodontic treatment can be useful for movement of third molar into eruption. Further studies can be done to orthodontically move third molar into functional occlusion.

Corresponding Author

Meenakshi Krishnan

Senior Lecturer, Department of Oral Medicine & Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University,

162, Poonamallee High Road, Chennai - 600077,Tamil Nadu, India

Email: drmeenakshisdc@gmail.com

Telephone Number: +91 8754415200

References

1. Staggers JA, Germane N, Fortson WM. A comparison of the effects of first premolar extractions on third molar angulation. The Angle Orthodontist. 1992 Jun; 62(2):135-8.
2. Richardson ME. The etiology and prediction of mandibular third molar impaction. The Angle orthodontist. 1977 Jul; 47(3):165-72.
3. Peterson LJ. Principles of management of impacted teeth. Contemporary oral and maxillofacial surgery. 1998; 2:223-57.
4. Richardson ME. Some aspects of lower third molar eruption. The Angle Orthodontist. 1974 Apr; 44(2):141-5.
5. Kaplan RG. Some factors related to mandibular third molar impaction. The Angle orthodontist. 1975 Jul; 45(3):153-8.
6. Kim TW, Årtun J, Behbehani F, Artese F. Prevalence of third molar impaction in orthodontic patients treated nonextraction and with extraction of 4 premolars. American journal of orthodontics and dentofacialorthopedics. 2003 Feb 28; 123(2):138-45.
7. Hattab FN, Alhaija ES. Radiographic evaluation of mandibular third molar eruption space. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 1999 Sep 30; 88(3):285-91.
8. Björk A, Jensen E, Palling M. Mandibular growth and third molar impaction. ActaOdontologicaScandinavica. 1956 Jan 1; 14(3):231-72.
9. Björk A. Variations in the growth pattern of the human mandible: longitudinal radiographic study by the implant method. Journal of Dental Research. 1963 Jan; 42(1):400-11.
10. Alling CC, Alling RD. Indications for Management of Impacted Teeth. Impacted Teeth. Philadelphia, Pa: WB Saunders Co; 1993:46–49.
11. Silling G. Development and eruption of the mandibular third molar and its response to orthodontic therapy. The Angle Orthodontist. 1973 Jul; 43(3):271-8.
12. Haavikko K, Altonen M, Mattila K. Predicting angulational development and eruption of the lower third molar. The Angle Orthodontist. 1978 Jan; 48(1):39-48.
13. Richardson M. Pre-eruptive movements of the mandibular third molar. The Angle Orthodontist. 1978 Jul; 48(3):187-93.

14. Richardson M. Late third molar genesis: its significance in orthodontic treatment. The Angle Orthodontist. 1980 Apr; 50(2):121-8.
15. CapelliJr J. Mandibular growth and third molar impaction in extraction cases. The Angle orthodontist. 1991 Sep; 61(3):223-9.
16. Erdem D, Özdiler E, Memikoglu UT, Baspinar E. Third molar impaction in extraction cases treated with the Begg technique. The European Journal of Orthodontics. 1998 Jun 1; 20(3):263-70.
17. Kim TW, Årtun J, Behbehani F, Artese F. Prevalence of third molar impaction in orthodontic patients treated nonextraction and with extraction of 4 premolars. American journal of orthodontics and dentofacialorthopedics. 2003 Feb 28; 123(2):138-45.
18. Behbehani F, Årtun J, Thalib L. Prediction of mandibular third-molar impaction in adolescent orthodontic patients. American journal of orthodontics and dentofacialorthopedics. 2006 Jul 31; 130(1):47-55.
19. Årtun J, Thalib L, Little RM. Third molar angulation during and after treatment of adolescent orthodontic patients. The European Journal of Orthodontics. 2005 Jul 11; 27(6):590-6.
20. Sandra Sager, Saravana Kumar, Sagar Moses. Variation in Anatomy and Position of Mandibular Third Molars in South Indian Population. Journal Pharm. Sci. & Res. 2015; 7(6):385-386.
21. Shehanaz A. The incidence of types of mandibular third molar impactions in different skeletal face types: A clinical study. International Journal of Orofacial Research. 2017 Jan 1; 2(1):24.

Change in the Angulations of Second Molar after First Premolar Extraction

Sangavi [1], A.V.Arun [2], Aravind Kumar [3]
Graduate Student[1], Professor [2], Department of Orthodontics
Professor [3], Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: In modern populations, the impaction rate is higher for third molars than for any other tooth. Mesial movement if present it can sometimes unlock a mild mesioangular impaction of third molar so it is reported to analyse whether there is any change in the second molar angulation and if so what is the average change in the angulation following first premolar extraction

Material and Methods: 58 patients who have undergone all four extractions werectaken. Pre-treatment and immediate post treatment record of the patients was traced. The armamentarium used for this research are orthopantomograms, panorex machine,Tracing sheets, 30cm scale, pencil, eraser, protractor.

Result: The average pre treatment angle is 64.24 and the average post treatment angle is 62.40. So the change in angle is shown to be 2 degree.

Conclusion: In cases such as bimaxillary protrusion and class II malocclusion all four extraction has become inevitable. Drifting of the adjacent tooth is common in such cases. Due to the recent advancement in anchorage system like TAD helps to overcome the anchorage loss. Sometimes angulations of second molar helps to unlock a mild mesioangular impaction of third molar

Key Words: *Premolar extraction, angulation, second molar, anchor loss, first Premolar*

Introduction

The most common malocclusion encountered by a orthodontist in Indian patients is bimaxillary protrusion and class IImalocclusion. The commonest treatment plan for these patients would be extraction of all first premolars. The most preferable anchorage unit in these cases is the is the first molar. A maximum anchorage or group an anchorage is preferred in these cases while extracting all the first premolars the most common side effect is anchorage loss. It is a reactionary effect of space closure following the first premolars extraction in orthodontic treatment. While taking the molars as anchorage unit with maximum anchorage75% of retraction takes place by anterior teeth and the remaining 25 % takes place due to posterior protraction. When there is a posterior protraction the molars may have mesial migration. There are various research and studies regarding the anchor loss and mesial migration of first molars due to first Premolar extraction however not many studies have been done in terms of measuring the angulation changes in second molar . The development of third molars and its influence on the dental arches has been a major concern to the dental profession [1]. Mandibular third molar impaction is a major issue in modern dentistry. [2] The path of development of third molars in human beings is not regular and the formation, calcification timing, and the position and course of eruption of these teeth show variability. Often, third molars are impacted or congenitally missing. [3]In modern populations, the impaction rate is higher for third molars than for any other tooth. [4] Mesial movement if present it can sometimes unlock a mild mesioangular impaction of third molar so it is reported to analyse whether there is any change in the second molar angulation and if so what is the average change in the angulation following first premolar extraction

Inclusion

Orthodontic patients treated with all four extractions

Retraction performed with regular friction mechanics

Patients with the presence of second molar in normal position.

Exclusion

Cases with friction less mechanism and micro implants aided retraction

Patients with immediate post treatment records not available

Patients with Temperomandibular disorder

Material and Method

Case records of 58 patients who have undergone all four extractions were taken. Pre-treatment and immediate post treatment record of the patients was traced. The line joining the midpoint of right and left rami is taken as the reference point (XI point). The angle which the long axis of the second molar makes with this reference plane in pre-treatment and post treatment radiographs is recorded.The difference between the pre treatment and the post treatment angulations gives the change in angulation of the second molar.

The armamentarium used for this research are orthopantomograms, panorex machine ,Tracing sheets, 30cm scale, pencil, eraser, protractor(figure 1)

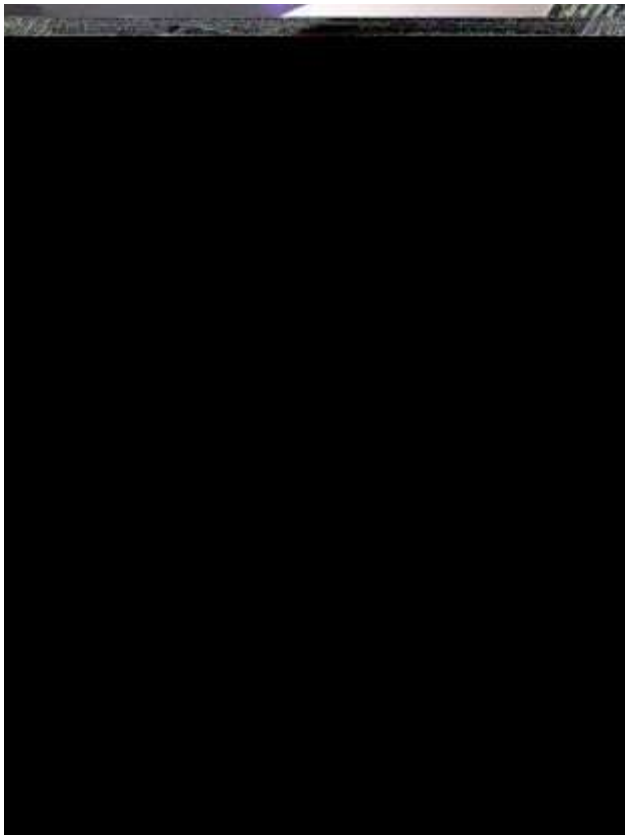


Figure 1: Panorex machine was used for taking orthopantomograms for the study.

Figure 2: Tracing sheets, metal scale 30cm, pencil , eraser , sharpener ,protractor .

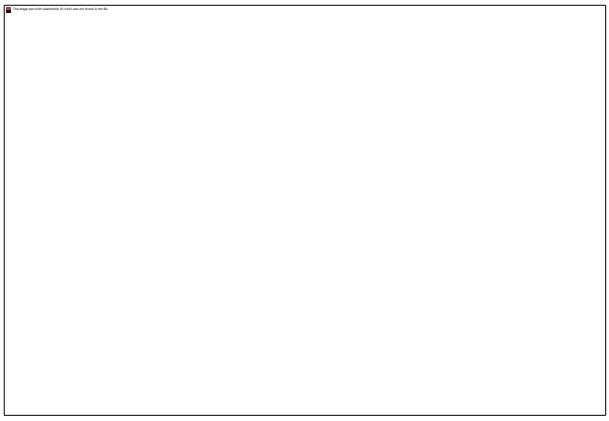


Figure 3: Tracing of the orthopantomogram done in a tracing sheet. The line joining the midpoint of right and left rami is taken as the reference point (XI point) . The angle which the long axis of the second molar makes with this reference plane in pre-treatment and post treatment radiographs is recorded.

Result

This study included Case records of 58 patients who have undergone all four extractions were taken. Pre-treatment and immediate post treatment record of the patients was traced. Orthodontic patients treated with all four extractions, Retraction performed with regular friction mechanics, Patients with the presence of second molar in normal position were included in the study. Patient with Cases with friction less mechanism and micro implants

aided retraction, Patients with immediate post treatment records not available, Patients with Temperomandibular disorder were exculded from the study. The data obtained from the study was statistically analysed using paired t test. In this research it has been considered that if the pre operative angle is greater than post operative angle then the change in angulations is considered as positive. If post operative angle is greater than preoperative angle then the change in angulation is considered as negative. Out of 58 cases 41 cases have shown positive results while the rest 17 has shown negative results. It has been concluded that 70.68% 0f cases shows greater pre operative angle than post operative angle the rest 29.31% of cases shows greater post operative angle than pre operative angle. The average pre treatment angle is 64.24 and the average post treatment angle is 62.40. So the change in angle is shown to be 2. This shows the mesial tilt of 2 degree.

(If P-Value < 0.05 then statistically Significant)

Table: 1 Paired T-Test to compare pre and post mean values

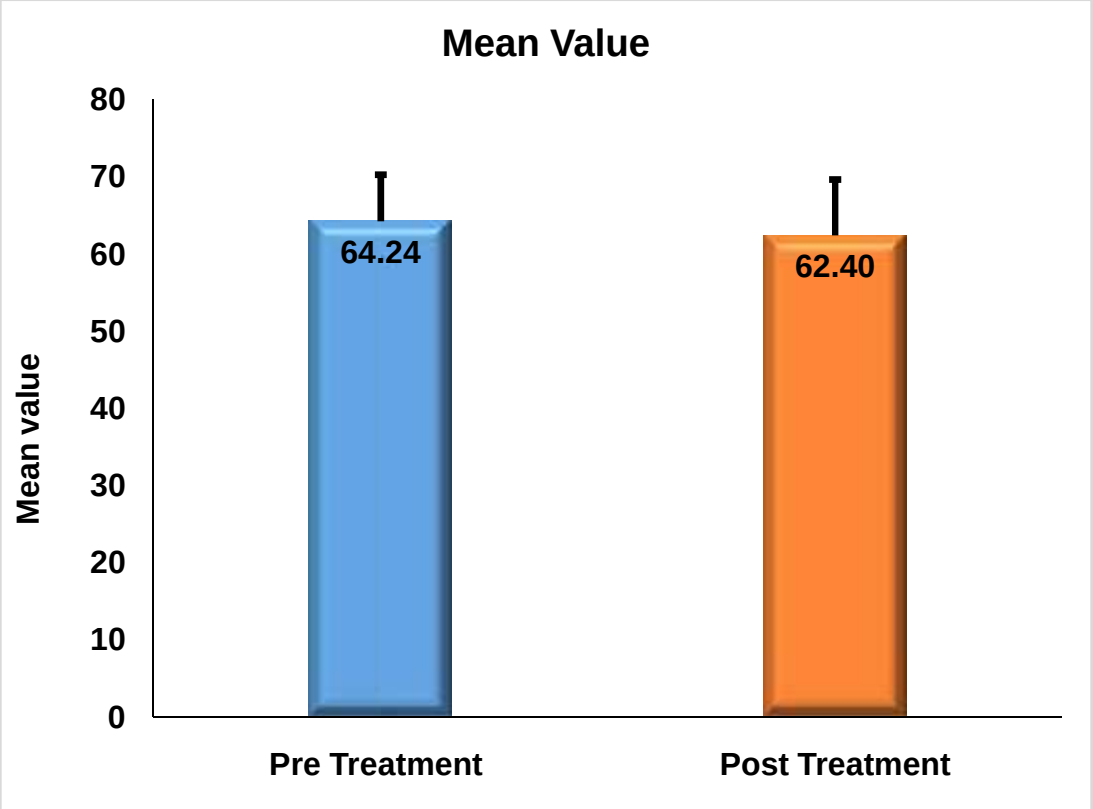
	N	Mean	Std. Dev	t-Value	P-Value
Pre Value	58	64.24	6.039	2.047	0.045
Post value	58	62.40	7.235		

Table: 2 One sample T-Test to compare mean differences (from "0") for positive and negative differences

Differences (Pre – Post)	N	Mean	Std. Dev	t-Value	P-Value
Positive (Pre > Post)	41	5.37	4.476	7.675	<0.001
Negative (Pre < Post)	17	-6.65	3.061	8.954	<0.001

In our study the P Value is less than 0.001 hence our study is statistically significant.

Graph: 1 Showing the mean value of pre treatment and post treatment values



Discussion

The purpose of this study is to analyse the change in the angulation of lower second molar after the extraction of lower first Premolar. This study shows mesial movement of second molars up to 2 degrees in all four extraction. Anchorage loss is the reactionary effect of space closure after the extraction of premolars .It is generally accepted that a strong relationship exists between root surface area and anchorage potential, the choice of teeth to be extracted should have a direct influence on the amount of anterior segment retraction .Anchorage is the resistance to unwanted tooth movement. [5, 6]. Major concern while correcting severe

crowding, excessive over jet, and bimaxillary protrusion is control of Anchor loss. Stability following orthodontic treatment continues to be a challenge to all orthodontists [3, 4]the ability to maintain long-term alignment following orthodontic treatment involving the extraction of premolars has unfortunately also been unpredictable. [7]

The role of extractions in the treatment of orthodontics is being a controversy in the history This controversy exists even today and, the extraction decision is still the most critical decision made by orthodontists during treatment planning .This is complicated further by the fact that the relative efficacy of extraction or non extraction strategies, in either the short or long term, has yet to be fully established.

Various studies have documented that premolars are the most commonly extracted teeth for orthodontic treatment located between the anterior and posterior segments of teeth premolar extractions seems to allow a straightforward relief of crowding or the correction of interincisor relationship. It has been suggested that improvements in techniques for controlling the tooth movement in dimensions and improvements in the correlation of these movements with anticipated facial growth changes have both increased the number of extraction options.

Different authors, for various reasons, have suggested variations in extraction sequences, including maxillary and mandibular first and/or second premolars. Its accepted that, during the treatment involving the extraction of teeth, dimensional changes in arch occur and these dimensions changes continue following active treatment. Furthermore, the ability for extraction spaces in the maxillary arch to be used in a predictable fashion has not yet been widely stated in the literature. In one such study, Williams and Hosila24 found that, in cases involving the extraction of 4 first premolars, approximately 66.5% of the available extraction space was taken up by the retraction of the anterior segment. In cases involving extractions of maxillary first and mandibular second premolars, 56.3% of the available extraction space was taken up by the retraction of the anterior segment.

It is generally accepted that a strong relationship exists between root surface area and anchorage potential, the choice of teeth to be extracted should have a direct influence on the amount of anterior segment retraction. For instance, Creek more stated, as a rule-of-thumb, that when first premolars are extracted, one can expect the posterior teeth to move forward

approximately one-third of the space, leaving two-thirds of the space for the relief of crowding and incisor retraction. When second premolars are extracted, one can expect the posterior teeth to move forward approximately half the extraction space, leaving the remaining half for the relief of crowding and the retraction of anterior teeth.

The concept of physiology drift commonly referred to as 'driftodontics'. It is been gaining acceptance orthodontic community after the Premolar extraction. The accurate drift and change in the inclination of the adjacent tooth has not be adequately documented. This study is done to evaluate the amount of inclination of the second molars that takes place after he extraction of first Premolar. Study done by Christos Livasa stated that Orthodontic treatment with unilateral maxillary M1 extraction resulted in a significant increase in the mesial inclination of maxillary second [8]. Similarly study done by D. M. Swessi C. D. Stephens stated that teeth tended to tip towards the extraction space, the amount of tipping was small (not exceeding 15 degrees in the majority of cases). The tipping was found to be greatest during the first 6 months [9]

In this study orthopantomograms of 58 patients were traced. The pre operative and post operative orthopantomograms were traced separately. The study included Orthodontic patients treated with all four extractions ,retraction performed with regular friction mechanics, Patients with the presence of second molar in normal position and patient with temperomandibular disorder were excluded from the study . Reference plane and reference angle were marked .The midpoint of right and left rami was connected by a line this is considered as the reference point. The angle formed by the long axis of the second molar makes with this reference plane in pre-treatment and post-treatment. These reference angle was measured from the pre operative and post operative radiograph . These values are then statistically analysed using paired t test . In this study the change in the angle between pre and the post treatment is around 2 degree with a standard deviation of 1 which is statistically significant.

A study done by Tarvade SMT has concluded that the difference in the mean and standard deviation between pre and post treatment is found to be 3.11which shows improvement in the angulation which had a distal tilt which was not statistically significant [10] Similarly a study done by a Avinash Gohilot, Tejashri Pradhan , Kanhoba Mahabaleshwar keluskar show an improvement in the angulation around 0.6 degree this shows a distal tilt.[11]Another study

done by Suruchi Jain , Ashima valiathan they show a difference in angulation around 6 degree which also shows a distal tilt [12-16]Study done by Luz Victoria showed that Verticalization of the third molar in the group of female subjects, represented by the α angle (.033), and the β angle (.016) on the left side.[17]In men, unlike women, third molar angulation showed an increase to mesial inclination; changes to the α angle were significant on the right side (.030). Study done by Tae-Min-You on effect of Effect of premolar extraction and presence of the lower third molar on lower second molar angulation in orthodontic treatmentshows . The angular difference and ratio of M2 to M1, the angulation change of M2 between pre- and postorthodontic treatment, and the distal bone level of M2 were higher in the nonextraction group than in the control and extraction groups[18]. Study done by Huda shows significant improvement in the third molars angulation in the extraction orthodontic therapy group compared to non- extraction group, when compared to the previous study this study shows greater angulation of lower second molar[19].Orthodontic treatment with premolars extraction during third molar development, and subsequent improvement of its angulation and path of eruption still remain controversy in previous literatures. [20]

does the extraction therapy has an impact on the eruption of my wisdom teeth is often asked by orthodontic patients to the orthodontist . In general this situation is uncertain among orthodontists, and in most of the cases, we have very few data on whether the extraction of premolars has beneficial effect on the eruption of third molars.

Third molars exhibit variation in their size, shape, position, root formation, time of development, and path of eruption. [21] The prevalence of impaction of at least one impacted lower third molar has been reported as 72.7% in a cohort aged 20–30 years. [22] Today, third molar impaction has been reported as the mostcommon of all tooth impactions.[23] In addition excessive interproximal attrition allowed mesial drift of the posterior teeth which made the incidence of third molar impaction relatively low.[24, 25]

The mandibular third molar is the most frequently impacted tooth after the maxillary third molar. For the lack of space between the second molar and the ramus has long been a major etiologic factor of mandible third molar impaction.[26-28] Björk et al. stated that the alveolar arch space behind the second molar is deficient in 90% of mandibular third molar impaction cases.[29] In their study, they examined 243 cases to investigate the correlation between mandibular third molar impaction and some cephalometric parameters. They reported that factors that affect the space of third molars were mandibular length, backward direction of eruption of the dentition, and vertical direction of condylar growth, which were associated

with the degree of resorption on the anterior aspect of the mandibular ramus.Mesial movement of second molars up to 2 degrees in all four extractions. Premolar extractions had a positive influence on the second molar angulations both on right and left .

Increased use of readjusted appliances, have replaced closing loop arches to various forms of sliding mechanics Sliding mechanics have great advantages , such as minimal wire-bending time and adequate space for activations.[30]

The retraction of four incisors post canine retraction is accepted as a method to reduce the mesial movement of the posterior teeth segment, whereas complete retraction of six anterior teeth may create anchorage problems. Added to this the tipping action built into anterior brackets in readjusted appliances may produce problems of anchorage. These problems may be adjusted by the use of a transpalatal bar and extra oral appliances.[31] However, intraoral anchorage devices provide unacceptable anchorage, whereas extraoral appliances provide a suitable anchorage but they are completely dependent on patient compliance.

Skeletal anchorage using dental implants,[33-34]manipulates,[35] miniscrews, [36] and micro screws[37-39] provides a complete anchorage for tooth movement. Miniscrew or micro screw implants are easy to place and remove and economical. Most importantly, because of their reduced size, they can be placed in the intra-arch alveolar bone without any damage to tooth roots. Added to this in contrast to dental implants orthodontic force applications can begin immediately after placement, . Therefore, these advantages have expanded the use of mini- or microscrew implants for various orthodontic problems.

By using microscrew implants in the mechanics of complete retraction of six anterior teeth, time required for the treatment can be reduced effectively and clinicians can move teeth to satisfy the treatment goal without patient compliance for anchorage devices.

Conclusion

In cases such as bimaxillary protrusion and class II malocclusion all four extraction has become inevitable . Drifting of the adjacent tooth is common in such cases .Due to the recent advancement in anchorage system like TAD helps to overcome the anchorage loss .Sometimesangulation of second molar helps to unlock a mild mesioangular impaction of third molar further study is required with larger number of samples to analyse the changes that happens to the second molar after the Premolar extraction.

Corresponding Author

Aravind Kumar

Professor, Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Mobile Number: +91 9841299939

Email id:dr.aravind.s@gmail.com

Reference

1. Staggers JA, Germane N, Fortson WM. A comparison of the effects of first premolar extractions on third molar angulation. The Angle Orthodontist.1992; 62:135-8.
2. Richardson ME. The etiology and prediction of mandibular third molar impaction. The Angle orthodontist. 1977;47:165-72
3. Forsberg CM. Tooth size, spacing, and crowding in relation to eruption or impaction of third molars. American journal of orthodontics and dentofacial orthopedics. 1988 1; 94:57-62.
4. Kim TW, Årtun J, Behbehani F, Artese F. Prevalence of third molar impaction in orthodontic patients treated nonextraction and with extraction of 4 premolars. American journal of orthodontics and dentofacial orthopedics. 2003 28; 123:138-45.
5. Geron S, Shpack N, Kandos S, Davidovitch M, Vardimon AD. Anchorage loss—a multifactorial response. The Angle orthodontist. 2003; 73:730-7.
6. Geron S, Shpack N, Kandos S, Davidovitch M, Vardimon AD. Anchorage loss—a multifactorial response. The Angle orthodontist.2003; 73:730-7.
7. Little RM. Stability and relapse of dental arch alignment. British Journal of Orthodontics. 1990 1; 17:235-41.
8. Ong HB, Woods MG. An occlusal and cephalometric analysis of maxillary first and second premolar extraction effects. The Angle Orthodontist. 2001; 71:90-102.
9. Sampson P, Little RM, Årtun J, Shapiro PA. Long-term changes in arch form after orthodontic treatment and retention. American Journal of Orthodontics and Dentofacial Orthopedics. 1995 31; 107:518-30.
10. Livas C, Pandis N, Booij JW, Halazonetis DJ, Katsaros C, Ren Y. Influence of unilateral maxillary first molar extraction treatment on second and third molar

inclination in Class II subdivision patients. The Angle Orthodontist.2015 12; 86:94-100.

11. Swessi DM, Stephens CD. The spontaneous effects of lower first premolar extraction on the mesio-distal angulation of adjacent teeth and the relationship of this to extraction space closure in the long term. The European Journal of Orthodontics. 1993 1; 15:503-11.
12. Tarvade SM, Biday S. Influence of first premolar extraction on second and third molar angulation-A clinical study. 2015;2(4):87-98.
13. Gohilot A, Pradhan T, Keluskar KM. Effects of first premolar extraction on maxillary and mandibular third molar angulation after orthodontic therapy. Journal of oral biology and craniofacial research. 2012 31; 2:97-104.
14. Jain S, Valiathan A. Influence of first premolar extraction on mandibular third molar angulation. The Angle Orthodontist. 2009; 79:1143-8.
15. Chua ES, Felicita A. The orthodontic extraction of permanent molars: A literature review. Australian orthodontic journal. 2015; 31:69.
16. Felicita A, Chandrasekar S, Shantha Sundari KK. Management of severe Class II division 1 malocclusion: a case report. Australian orthodontic journal.2011; 27:181.
17. Mendoza-García LV, Vaillard-Jiménez E, García-Rocha A, Bellot-Arcís C, Paredes-Gallardo V. Effect of orthodontic treatment involving first premolar extractions on mandibular third molar angulation and retromolar space. Journal of Clinical and Experimental Dentistry. 2017; 9:e333.
18. You TM, Ban BH, Jeong JS, Huh J, Doh RM, Park W. Effect of premolar extraction and presence of the lower third molar on lower second molar angulation in orthodontic treatment. Oral surgery, oral medicine, oral pathology and oral radiology. 2014 30; 118:278-83.
19. Huda M. Al Kuwari.Influence of orthodontic treatment with rst premolar extraction on the angulation of the mandibular third molar .Saudi Med J 2013; Vol. 34.
20. Forsberg CM, Vingren B, Wesslen U. Mandibular third molar eruption in relation to available space as assessed on lateral cephalograms. Swedish dental journal. 1989;13(1-2):23-31.

21. Hattab FN, Alhaija ES. Radiographic evaluation of mandibular third molar eruption space. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 1999 30; 88:285-91.
22. Hugoson A, Kugelberg CF. The prevalence of third molars in a Swedish population. An epidemiological study. Community dental health. 1988; 5:121.
23. Bishara SE. Third molars: a dilemma! Or is it?.American journal of orthodontics and dentofacial orthopedics. 1999 30; 115:628-33.
24. Godfrey K, Dent KK. Prophylactic removal of asymptomatic third molars: a review. Australian dental journal. 1999 1; 44:233-7.
25. Kim TW, Årtun J, Behbehani F, Artese F. Prevalence of third molar impaction in orthodontic patients treated nonextraction and with extraction of 4 premolars. American journal of orthodontics and dentofacial orthopedics. 2003 28; 123:138-45.
26. Björk A. Variations in the growth pattern of the human mandible: longitudinal radiographic study by the implant method. Journal of Dental Research. 1963 Jan; 42:400-11
27. Behbehani F, Årtun J, Thalib L. Prediction of mandibular third-molar impaction in adolescent orthodontic patients. American journal of orthodontics and dentofacial orthopedics. 2006 31; 130:47-55.
28. Abu Alhaija ES, AlBhairan HM, AlKhateeb SN. Mandibular third molar space in different antero-posterior skeletal patterns. The European Journal of Orthodontics. 2010 27; 33:570-6.
29. Björk A, Jensen E, Palling M. Mandibular growth and third molar impaction. Acta Odontologica Scandinavica. 1956 1; 14:231-72.
30. McLaughlin RP, Bennett JC. The transition from standard edgewise to readjusted appliance systems. Journal of clinical orthodontics: JCO. 1989; 23:142.
31. McLaughlin RP, Bennett JC. Anchorage control during leveling and aligning with a preadjusted appliance system. Journal of clinical orthodontics: JCO. 1991; 25:687-96.
32. Shapiro PA, Kokich VG. Uses of implants in orthodontics. Dental Clinics of North America. 1988; 32:539-50.
33. Roberts WE. Rigid implant anchorage to close a mandibular first molar extraction site. J. clin. Orthod.. 1994;28:693-704.
34. Umemori M, Sugawara J, Mitani H, Nagasaka H, Kawamura H. Skeletal anchorage system for open-bite correction. American Journal of Orthodontics and Dentofacial Orthopedics. 1999 28; 115:166-74.

35. Creekmore TD. The possibility of skeletal anchorage. J Clin Orthod. 1983;17:266-9.
36. Kanomi R. Mini-implant for orthodontic anchorage. J clin Orthod. 1997;31:763-7.
37. Deguchi T, Takano-Yamamoto T, Kanomi R, Hartsfield Jr JK, Roberts WE, Garetto LP. The use of small titanium screws for orthodontic anchorage. Journal of dental research. 2003; 82:377-81.
38. Park HS. The skeletal cortical anchorage using titanium microscrew implants. KOREA J (ORT-OD 1999, 29. 1999 1; 699:706.
39. Park HS. The use of micro-implant as orthodontic anchorage. Seoul. Korea: Nare Publishing. 2001:257-88.

Changes in Total andDifferential Leukocyte Count of Post Therapeutic Cancer Patients

Bharadwaj.B [1], Brundha.M.P [2], Balakrishna R.N [3]
Graduate Student [1], Senior Lecturer [2], Department of General Pathology
Senior Lecturer [3], Department of Oral and Maxillofacial Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction : infectious diseases and foreign bodies. Sudden decrease and increase in their count causes marked change in the immune system.The present study aims to highlight the total and differential leukocyte Leukocytes are known as cells of immune system that are involved in protecting the body against both count in post therapeutic cancer patients

Materials and Method:A sum of 15 blood smears were made from peripheral blood of post therapeutic cancer patients in Saveetha Dental College and staining was done to determine the total and differential leukocyte count using leishman's stain.

Result: The total and differential count varied significantly in few post operative patients and few had various diseases like lymphocytosis, neutrophilia, neutropenia and eosinophilia.

Conclusion: All smears showed hypersegmentation and hypergranulation due to depletion of vitamin B_{12} and folic acid through radiation and surgery.

Key Words: *Hypersegmentation, Hypergranulation, Leukocytosis,Leucopenia, VitaminB_{12}, Folic acid.*

Introduction

Cancer is a group of diseases characterized by uncontrolled growth and spread of abnormal cells. If the spread is not controlled, it can result in death [1]. Cancer is caused by both external and internal factors. The external factors are tobacco, infectious organisms, chemicals and radiation. The internal factors are inherited mutations, hormones immune conditions and mutations that occur from metabolism. These casual factors may act together or in sequence to initiate or promote the development of cancer [2]. Cancer is treated by surgery, radiation, chemotherapy, hormone therapy, biological therapy and targeted therapy [3]. The extravasation of chemotherapeutic drug and radiation can be serious adverse event that can damage internal tissues. After this treatment there may be a marked increase or decrease in leukocyte count. Leukocytes are produced from bone marrow and they are found throughout the body including blood and lymphatic system[4]. There are 5 different and diverse types of leukocytes [5]. They are: Neutrophils(60-70%) also called as polymorphonuclear cells and their main function is phagocytosis which results in digestion of bacterial organisms and debris. Lymphocytes (20-30%) measurement reflects a combination of the T and B cells. Their function is to combat acute viral infections and chronic bacterial infections. Monocytes (2-5%)also called as monos and play a vital role in phagocytosis of bacteria. Monocytes generally last longer in circulation than neutrophils.Eosinophils (2-3%) also called as eosand their main function is during allergic reactions and parasitic infections involved in inflammatory process and allergic reactions. Basophils (0-1%) also called as mast cells or basos act as an anticoagulant. These leukocytes continuously circulate throughout the body and their count increases in certain infections [6].The Infiltrating inflammatory cells have an important character in the rapidity and spread of tumors [7, 8]. With the help of specific chemokines actions when it’s released, the tumor microenvironment monitors the migration of the leukocyte and many important functions of these cells when they arrive at the tumor site. The autocrinesecretion of chemokines by the tumor cells brings in the action of inflammatory cells such as monocytes and neutrophils, where its survival rate increases with rapid proliferation and dissemination [8]. The inflammatory part of a developing tumor involves a variety in the leukocyte population such as the neutrophils, dendritic cells, macrophages, eosinophils, mast cells and lymphocytes, where all these cells has the potential in developing more number of different mediators. [9]. However, macrophages and lymphocytes are the most common cell types found infiltrated in the stroma and in neoplastic fluids [10, 11].Thechemokines such as MCP-1 attracts these leukocytes and they are regulated

during activation, the expression of normal T-cell occurs, and there is secretion (RANTES). The communication between the increased levels of MCP-1 and RANTES expression and the action of tumor-associated macrophages is increasingly reported in cases of breast cancer [12] and in cervical cancer [13]. Certain Clinical studies have studied the correlation relating the increased number of infiltrated macrophages and the poor prognosis rate for breast, prostate, ovarian, cervical, endometrial, esophageal and bladder cancers [14].The infiltrating inflammatory cells are capable of having pleiotropic functions: inhibition of tumor growth by non-specific cytotoxic mechanisms and initiation of cell lysis, or conversely promotion of tumor development by the release of inflammatory mediators [15].Pro-inflammatory cytokines promotes thedevelopment of migration, differentiation and activity of antigen-presenting cells and may favorably affect survival of cancer patients. Tumor-infiltrating leukocytes can produce vascular endothelial growth factor (VEGF), IL-8 and matrix metalloproteinases that mediate tumor invasion and angiogenesis [16].The prostaglandins and NO, produced by cyclooxy-genase-2 and inducible NO synthase, influences human tumor development [17]. NO is consistently evaluated at nanomolar concentrations in the microenvironment of the tumour and has been found to aid in tumor genesis [18]. The factors determining the outcome of these competing effects of the inflammatory response during tumor development are still undefined [15].The host response to malignant tumors includes not only the local changes in tumor microenvironment, but also involves the systemic effects. In terms of inflammatory response, neoplasia is a paradox. Tumors produce factors such as the chemokineswhich recruit leukocytes, but a deficient inflammatory response is seen at sites remote from the tumor [8, 19]. The Circulating chemokines factors have the capacity to desensitize leukocytes to migratory stimuli or, the tumor may produce anti-inflammatory mediators. Therefore, a systemic deficiency in inflammatory response could coexist with continued leukocyte recruitment to the tumor site [8].The most common systemic alterations assessed in patients with malignant solid tumors are leukocytosis and neutrophilia. These are the hematological conditions which is significantly correlated with advanced disease and, consequently, with poor prognosis [20]. Leukocytosis is defined as a condition often occurring in a clinical setup, usually caused by an elevation in the number of neutrophils, which represent 50 to 60% of total leukocytes. Etiologically, leukocytosis is a primary pathological condition affecting the white blood cells, but it frequently arises as a reaction to infection, chronic inflammation and cancer [20]. Neutrophilia is a condition

found to be an independent prognostic factor, associated with reduced survival in human metastatic melanoma, pancreatic carcinoma, and renal carcinoma [16, 21, 22]. A high pretreatment count of neutrophils in blood was confirmed as an independent prognostic factor for short overall survival in stage IV melanoma patients undergoing interleukin-2 immunotherapy. Furthermore,a high overall leukocyte count was a predictor of short overall survival. Since immunotherapy with cytokines induces adverse effects in almost every patient, it is important to avoid treating patients who are predicted not to benefit from the treatment [23].The correlation between the lymphocyte count and the clinical response to chemotherapy was assessed in lung, colorectal, breast and prostate cancer patients. Independent of tumor histotype and chemotherapeutic regimen, the average number of lymphocytes was significantly reduced in patients with progressive disease following chemotherapy. Conversely, lymphocytosis was observed in patients who achieved an objective tumor regression in response to chemotherapy, and lymphocyte count which at the end of treatment was significantly increased than values which was calculated before the beginning of treatment. A possible mechanism for this increase in lymphocyte count may be modulation of the cytokine network by chemotherapy, that corrects increased endogenous production of immunosuppressive cytokines [24]. In patients with locally advanced cervical carcinoma, univariate and multivariate analysis showed that a greater baseline lymphocyte count was the best predictor of a complete clinical response and progression-free survival after chemoradiation [25].

A study of patients with metastatic renal carcinoma during immunotherapy with interleukin-2 and interferon-alpha showed that changes from baseline in the total lymphocyte counts 4 weeks after treatment were significantly higher in the responding patients than in the non-responding patients, whereas no difference was observed in eosinophil counts [26]. An accumulation of immature or defective circulating leukocytes could facilitate a systemic immune dysfunction, while the presence of those cells at the tumor site could facilitate tumor growth. In several cancers breast, prostate and glioma; it was found that systemic distribution of immature dendritic cells was more evident in patients with advanced disease [27] . Dendritic cells are antigen presenting-cells that are potent initiators of a primary immune response. They originate from bone marrow progenitors, which circulate in peripheral blood and subsequently give rise to immature dendritic cells that reside in peripheral

tissues [31]. This study helps to identify the change in leukocyte count and also its morphological changes due to radiation.

Materials and Methods

Total leukocyte count and differential Leukocyte count can be done from same smear preparation. For about 15 post therapeutic cancer patients in Saveetha Dental College peripheral blood was drawn and smear was made on glass slide. The materials used for staining leukocytes are blood sample, glass slide, spirit, cotton, lancet, leishman stain, distilled water and stop watch. The count was done manually.For total count, the slide is focused under 45x and the number of leukocytes in 10 successive fields was noted. From this the total Leukocyte is counted by using the following formula:

$$\text{Total count} = \frac{\text{Total number of cells counted}}{10} \times 2000 \text{ cubic mm}$$

For differential count cedar wood oil is used, the slide is focused under 100x, the Leukocyte count is made to complete 100 boxes. This is repeated for all the patients and the count is determined.

Result

Staining was done on blood smears collected from the cancer patients depicted various changes in total and differential leukocyte count in post therapeutic cancer patients.

Table 1: Changes in TC & DC in peripheral blood of post therapeutic Cancer patients

S.No	Patient Name	Age/Sex	Type of Cancer	Total Leukocyte count	Differential count				
				(Cells/cmm)	N	L	E	M	B
1	Mohandas	36/Male	Oral Cancer	16800	76	21	2	1	-
2	Lakshmi	60/Female	Uterine Cancer	4600	50	24	19	6	1
3	Kolleshwaran	38/Male	Lung Cancer	9600	68	29	3	-	-
4	Gopal	47/Male	Neck Cancer	7600	75	21	3	-	1
5	Kalyani	45/Female	Intestine Cancer	8600	62	35	3	-	-
6	Kurian	80/Male	Oral Cancer	17200	65	32	3	-	-
7	Kanchana	38/Female	Stomach Cancer	7200	65	33	2	-	-
8	Murali	63/Male	Tongue Cancer	5400	56	38	4	-	2
9	Chiranjeevi	22/Male	Liver Cancer	9400	48	49	3	-	-
10	Ravi	38/Male	Stomach Cancer	8200	48	46	4	2	-
11	Krishnaveni	50/Female	Uterine Cancer	4500	53	43	3	1	-
12	Selvi	48/Female	Uterine Cancer	2800	64	28	7	-	1
13	Malliga	47/Female	Uterine Cancer	6000	64	33	2	-	1
14	ConskaleDiwakar	48/Female	Breast Cancer	6400	50	50	-	-	-
15	Anjali	42/Female	StomachCancer	7400	48	49	2	1	-

In the above table, N L E M B refers toN – Neutrophil L- lymphocyte E- Eosinophil

M-Monocyte B- Basophil

From the above table we can say that oral cancer patients sufferedleucocytosis and are said tohave normal differential count. Uterine cancer patients mostly have leucopenia and differential count was normal except one who had eosinophilia. Other cancer patients have a normal total leukocyte count. Stomach cancer, breast cancer and liver cancer patients suffer from lymphocytosis and neutropenia.

Discussion

Aquestion which arises in cancer biology is how tumor development leads to changes in the number of circulating leukocytes . A possible mechanism is the development of soluble factors such as the granulocyte and macrophage colony stimulating factor (GM-CSF) by tumor cells, which has the potential of mobilizing precursors in the bone marrow; or vascular endothelial growth factor (VEGF) and interleukin-6, both of which alter cell differentiation [28].White blood cell count has been suggested as an significant indicator in disease activity which is to be closely evaluated in patients with G-CSF-producing renal cell carcinoma. Pronounced neutrophilia is a manifestation of the strong host immune response elicited by photodynamic therapy of tumors. The cause of this neutrophilia was shown to be complement activation, which triggers the release of complement factors and at least a dozen secondary mediators including tumor necrosis factor, IL-1 , IL-6, IL-10, GCSF, prostaglandins and leukotrienes [29] The circulating neutrophils are consistently higher in mice deficient in the small GTPaseRac2 than in wild-type mice. The depletion of lymphocyte with consequent reduction of innate cellular immunity is a very severe clinical problem that can develop during cancer progression and cytoreductive therapies. Lymphopenia is a result of tumor-induced mechanisms involving the impairment of antigen presentation, activation of negative co-stimulators signals, and production of immunosuppressive factors, resulting in a marked reduction in T-helper lymphocytes [30,31] Along with this occurrence cancer cells may develop the expansion and recruitment of regulatory cell populations that plays a role in the immunosuppressive network; these include regulatory T cells (Tregs), myeloid suppressor cells and distinct subsets of immature regulatory dendritic cells. Development ofimmunosuppressive mediators such as IL-10, NO and transforming growth factor by tumor cells are responsible for evasion of immune surveillance; reduced production of these mediators restored a lymphoproliferative response [32]. The innate immunesystem triggers an anti-tumor response. The thymic pathway ismostly compromised in adults recoveringfrom

lymphopenia. Due to this , T cells depends upon peripheral expansion to restore cell numbers, through the stimulation of high cytokine levels [33].

The count done manually may vary from person toby person but gives an idea on the changes in count of post therapeutic cancer patients. The patients suffering from leukocytopenia, leukocytosis, neutrophilia, lymphocytosis, neutropenia and eosinophilia must be treated immediately as it may even result in death [34]. In all patients smear a characteristic features were seen, hypersegmentation and hypergranulation of neutrophil [35]. Hypersegmentation refers to neutrophils having more than 5 nuclei and hypergranulation refers to increased number of granules in the cytoplasm [36] This occurs due to deficiency of vitamin B12 or folic acid [37]. We all know that during surgery and radiation exposure vitamin B12 and folates are destroyed thus resulting in hypersegmentation and hypergranulation of neutrophils [38].

Conclusion

A pilot study has been made with 15 patients from Saveetha Dental College. All smears had hypersegmentation and hypergranulation of neutrophils clearly. This indicates that vitamin B12 and folic acid are very much necessary which has been depleted due to surgery and radiation. Many patient suffer from leukocytopenia, leukocytosis, neutrophilia, lymphocytosis, neutropenia and eosinophilia .There are many marked changes in total and differential leukocyte count of post therapeutic patients which has to be considered.

Corresponding Author

Balakrishna R.N

Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamalle High Road,Chennai 600077,Tamil Nadu, India

E-mail Id – balakrishdr8394@gmail.com

Mobile number: 8939896729

References

1. World Health Organization. Ten statistical highlights in global public health. World health statistics. 2007;9420:657-660.
2. Orlando G, Pilone V, Vitiello A, Gervasi R, Lerose MA, Silecchia G, Puzziello A. Gastric cancer following bariatric surgery: a review. Surgical Laparoscopy Endoscopy & Percutaneous Techniques. 2014;24(5):400-5.
3. Roland KB, Rodriguez JL, Patterson JR, Trivers KF. A literature review of the social and psychological needs of ovarian cancer survivors. Psycho- Oncology. 2013;22(11):2408-18.
4. Maton A. Human biology and health. Prentice Hall; 1997;6(7):8.
5. LaFleur-Brooks M. Exploring medical language: a student-directed approach, St. Louis, Mosby Elsevier. 2008;78(8):9.
6. Handin RI, Lux SE, Stossel TP, editors. Blood: principles and practice of hematology. Lippincott Williams & Wilkins. 2003;9(10):8.
7. Zak M, Rezný Z, Uhlirova A, Bednarova Z, Lintner L, Benes J. Dependence of the postirradiation lymphopenia and hypersegmentation of neutrophil nuclei on the therapeutic irradiation exposure in patients with breast carcinoma--some ways of its use for biological detection of irradiation. Strahlentherapie. 1978;154(12):852-7.
8. Westerman DA, Evans D, Metz J. Neutrophil hypersegmentation in iron deficiency anaemia: a case-control study. British journal of haematology. 1999;107(3):512-5.
9. Bills T, Spatz L. Neutrophilic hypersegmentation as an indicator of incipient folic acid deficiency. American journal of clinical pathology. 1977;68(2):263-7.
10. Edwards MJ, Penny RH. Effects of hyperthermia on the myelograms of adult and fetal guinea- pigs. British journal of haematology. 1985;59(1):93-101.
11. Wakatsuki S, Hirokawa M, Horiguchi H, Kanahara T, Manabe T, Sano T. Hypersegmentation of inflammatory cells in Langerhans cell granulomatosis. Diagnostic cytopathology. 2000;23(4):238-41.
12. Ben-Baruch A. Inflammation-associated immune suppression in cancer: the roles played by cytokines, chemokines and additional mediators. InSeminars in cancer biology. 2006;16(1):38-52.
13. Zijlmans HJ, Fleuren GJ, Baelde HJ, Eilers PH, Kenter GG, Gorter A. Role of tumor- derived proinflammatory cytokines GM- CSF, TNF- α, and IL- 12 in the migration and differentiation of antigen- presenting cells in cervical carcinoma. Cancer. 2007;109(3):556-65.

14. Shih JY, Yuan A, Chen JJ, Yang PC. Tumor-associated macrophage: its role in cancer invasion and metastasis. J Cancer Mol. 2006;2(3):101-6.
15. Schmidt H, Bastholt L, Geertsen P, Christensen IJ, Larsen S, Gehl J, Von Der Maase H. Elevated neutrophil and monocyte counts in peripheral blood are associated with poor survival in patients with metastatic melanoma: a prognostic model. British journal of cancer. 2005;93(3):273.
16. Kong G, Kim EK, Kim WS, Lee KT, Lee YW, Lee JK, Paik SW, Rhee JC. Role of cyclooxygenase- 2 and inducible nitric oxide synthase in pancreatic cancer. Journal of gastroenterology and hepatology. 2002;17(8):914-21.
17. Pervin S, Singh R, Hernandez E, Wu G, Chaudhuri G. Nitric oxide in physiologic concentrations targets the translational machinery to increase the proliferation of human breast cancer cells: involvement of mammalian target of rapamycin/eIF4E pathway. Cancer Research. 2007;67(1):289-99.
18. Fernandes PC, Garcia CB, Micheli DC, Cunha FQ, Murta EF, TAVARES- MURTA BM. Circulating neutrophils may play a role in the host response in cervical cancer. International Journal of Gynecological Cancer. 2007;17(5):1068-74.
19. Ruka W, Rutkowski P, Kaminska J, Rysinska A, Steffen J. Alterations of routine blood tests in adult patients with soft tissue sarcomas: relationships to cytokine serum levels and prognostic significance. Annals of oncology. 2001;12(10):1423-32.
20. Fogar P, Sperti C, Basso D, Sanzari MC, Greco E, Davoli C, Navaglia F, Zambon CF, Pasquali C, Venza E, Pedrazzoli S. Decreased total lymphocyte counts in pancreatic cancer: an index of adverse outcome. Pancreas. 2006;32(1):22-8.
21. Donskov F, von der Maase H. Impact of immune parameters on long-term survival in metastatic renal cell carcinoma. J Clin Oncol. 2006;24(13):1997-2005.
22. Schmidt H, Suciu S, Punt CJ, Gore M, Kruit W, Patel P, Lienard D, von der Maase H, Eggermont AM, Keilholz U. Pretreatment levels of peripheral neutrophils and leukocytes as independent predictors of overall survival in patients with American Joint Committee on Cancer Stage IV Melanoma: results of the EORTC 18951 Biochemotherapy Trial. Journal of Clinical Oncology. 2007;25(12):1562-9.
23. Huang ZS, Chien KL, Yang CY, Wang CH, Chang TC, Chen CJ. Peripheral differential leukocyte counts and subsequent mortality from all diseases, cancers, and cardiovascular diseases in Taiwanese. Journal of the Formosan Medical Association= Taiwan yi zhi. 2003;102(11):775-81.
24. Zahorec R. Ratio of neutrophil to lymphocyte counts-rapid and simple parameter of systemic inflammation and stress in critically ill. Bratislavske lekarske listy. 2001;102(1):5-14.

25. Del Prete M, Giampieri R, Loupakis F, Prochilo T, Salvatore L, Faloppi L, Bianconi M, Bittoni A, Aprile G, Zaniboni A, Falcone A. Prognostic clinical factors in pretreated colorectal cancer patients receiving regorafenib: implications for clinical management. Oncotarget. 2015;6(32):33982.
26. Choi CH, Kang H, Kim WY, Kim TJ, Lee JW, Huh SJ, Lee JH, Kim BG, Bae DS. Prognostic value of baseline lymphocyte count in cervical carcinoma treated with concurrent chemoradiation. International Journal of Radiation Oncology Biology Physics. 2008;71(1):199-204.
27. Jeong IG, Han KS, Joung JY, Choi WS, Hwang SS, Yang SO, Seo HK, Chung J, Lee KH. Analysis of changes in the total lymphocyte and eosinophil count during immunotherapy for metastatic renal cell carcinoma: correlation with response and survival. Journal of Korean medical science. 2007;22:122-8.
28. Rovati B, Mariucci S, Manzoni M, Bencardino K, Danova M. Flow cytometric detection of circulating dendritic cells in healthy subjects. European journal of histochemistry: EJH. 2008;52(1):45.
29. Kah-Wai L, Jacek T, Jacek R. Dendritic cells heterogeneity and its role in cancer immunity. Journal of cancer research and therapeutics. 2006;2(2):35.
30. Hirashima M, Higuchi S, Sakamoto K, Nishiyama T, Okada H. The ratio of neutrophils to lymphocytes and the phenotypes of neutrophils in patients with early gastric cancer. Journal of cancer research and clinical oncology. 1998;124(6):329-34.
31. Yamanaka T, Matsumoto S, Teramukai S, Ishiwata R, Nagai Y, Fukushima M. The baseline ratio of neutrophils to lymphocytes is associated with patient prognosis in advanced gastric cancer. Oncology. 2007;73(3-4):215-20.
32. Gwak MS, Choi SJ, Kim J, Ko JS, Kim TH, Lee SM, Park JA, Kim MH. Effects of gender on white blood cell populations and neutrophil-lymphocyte ratio following gastrectomy in patients with stomach cancer. Journal of Korean medical science. 2007;22:104-8.
33. Walsh SR, Cook EJ, Goulder F, Justin TA, Keeling NJ. Neutrophil- lymphocyte ratio as a prognostic factor in colorectal cancer. Journal of surgical oncology. 2005;91(3):181-4.
34. Cook EJ, Walsh SR, Farooq N, Alberts JC, Justin TA, Keeling NJ. Post-operative neutrophil–lymphocyte ratio predicts complications following colorectal surgery. International Journal of Surgery. 2007;5(1):27-30.
35. Halazun KJ, Aldoori A, Malik HZ, Al-Mukhtar A, Prasad KR, Toogood GJ, Lodge JP. Elevated preoperative neutrophil to lymphocyte ratio predicts survival following hepatic resection for colorectal liver metastases. European journal of surgical oncology. 2008;34(1):55-60.
36. Cho H, Hur HW, Kim SW, Kim SH, Kim JH, Kim YT, Lee K. Pre-treatment neutrophil to lymphocyte

ratio is elevated in epithelial ovarian cancer and predicts survival after treatment. Cancer immunology, immunotherapy. 2009;58(1):15-23.

37. Jerusha Santa Packyanathan. Awareness of Vitamin B12 Deficiency among the General Population. Int J Recent Sci Res. 2017;8(3):16149-16152.
38. Hanshika R. Comparison of Morphological Analysis of RBC through Peripheral Smear and Automated Method. Glob J Pharmaceu Sci. 2017; 1(5):555-571

Colonisation of Candida and its Species among Diabetic Denture Wearers VsNon-diabetic Denture Wearers

Umme Salma Durbar[1] , NithyaJagannathan [2], Gopinath [3], GurumoorthyKaarthikeyan[4]

Graduate Student [1],Reader [2],Department of Oral Pathology

Professor [3],Department of Microbiology,Professor [4] ,Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Human oral cavity in health is normally colonised by different microbiological flora predominantly with Candida.*Candida*is recognized as a normal commensal but can become a pathogen during favourableconditions such as immuno-compromised status like diabetes, ill filling dentures, nocturnal wear of the denture, age of the dentureand unhygienic maintenance of the dentures. Diabetics have an increased predisposition to the manifestation of candidiasis, which is associated with poor glycemic control and therapeutic dentures. Denture stomatitis is a common inflammatory reaction, multifactorial etiology, which is usually associated with Candida species, particularly Candida albicans, due to its high virulence, ability to adhere and form biofilms on oral cavity tissues and denture surfaces. Acrylic dentures which are usually ill fitting with suboptimal hygiene, act as reservoirs of infection.The aim of the study was to assess the prevalance of candidiasis among diabetic and nondiabetic denture wearers and the variety of candida species seen.

Materials and Method;A total of 30 individuals were enrolled in the study. Of the subjects, 15 were diabetic denture wearers and 15 were diabetic non-denture wearers. The salivary samples were collected in sterile containers and processed in Sabouraud dextrose agar (SDA) and incubated at 37 C for 48 hours.

Result: Out of the 30 subjects the prevalence of candida species was 86% in diabetic denture wearers and 46% in diabetic non-denture wearers.

Conclusion: The rate of candidal growth was higher in denture wearers with Diabetes when compared to non-diabetic denture wearers.

Keywords*: candida, denture wearers, diabetesmellitus,oppurtunistic, poor oral hygiene*

Introduction

In healthy individuals, oral cavity harbors many different spectra of micro-organisms. There are about 300-400 species of micro-organisms and 20 species of Candida in the oral cavity [1]. Most of the yeast infection in the oral cavity is caused by the genus Candida. Any changes in the oral cavity, such as tooth loss and denture wearing will cause changes in the oral micro flora [2]. *Candida* is recognized as a normal commensal of almost half of the population which does not cause any harmful effects [3,4]. But *Candida* can become a pathogen during unfavourable conditions and immuno-compromised status such as diabetes, trauma due to ill filling dentures, nocturnal wear and age of the denture, other systemic conditions andunhygienic maintenance of the dentures by the patient. These makethe oral cavity a suitable environment for Candida to turn pathogenic [5].*Candida* species also have the affinity to adhere to, and subsequently colonize, the denture acrylic resin material[6]. Oral Candidiasis is an opportunistic infection which causes pathological changes in the mucosal surface of the oral cavityThis is due to the prosthesis that acts as a focus and trauma caused by the denture facilitates the infection. Candidiasis may appear as various symptoms including pain, burning sensation, difficulty in swallowing, change of taste, but often they are asymptomatic [7,8].

Candida in the oral cavity may be present in two distinct forms,either as floating planktonic cells (blastopores, blastoconidia) and/or in an organized biofilm. Poor oral hygiene, practices such as failure to remove the denture whilst sleeping and poor denture cleansing allows the accumulation of biofilm [9], which is defined as structured microbial community that is attached to a surface, consisting of more than 10^{11} microorganisms per gram of dry weight and surrounded by a self-produced extracellular matrix. It has been recognized that unlike dental biofilm, the biofilm that forms on denture materials harbours a much larger population of yeasts [10]. Proliferation of bacteria and fungi can cause bad breath, acrylic resin pigmentation and staining, formation of

calculus deposits and the development of chronic atrophic candidiasis, also known as denture stomatitis. Candida albicans is able to utilize special mechanisms that allow it to be able to switch back and forth from one form to another (from budding yeast form to pseudohyphal, to hyphal forms) [11,12]. Morphologic transition from the yeast to hyphal state is one of the key factors in the pathogenic activity of Candida albicans.

Diabetes mellitus is a chronic metabolic disease, marked by high levels of blood glucose[13].Immunodeficiency and increased susceptibility to opportunistic infections are seen in diabetic patients. Diabetes have an increased predisposition to the manifestations of oral diseases like candidiasis, which is associated with poor glycemic control and therapeutic dentures[14]. This also contributes to xerostomia, which may be due to increased glucose levels in oral fluids or immune dysregulation[15].

Wearing complete denture is a risk factor, which can promote colonization of *Candida*, produce Candidal biofilm and result in oral candidiasis. About 50- 65 % of patient wearing dentures have been reported to have over growth of oral Candida, leading to Candida associated denture stomatitis (inflammation of the denture bearing site of the mucosa).Additionally, Candida has affinity for the acrylic surface of dentures and non- renewing surfaces such as teeth, dental fillings [16,17]. Surface characteristics of denture base acrylic resins, such as hydrophobicity, have generally been acknowledged to be one of the factors contributing to the adhesion, which is a crucial step in biofilm formation.

Wearing dentures is a contributingfactor which can affect the oral health and hygiene. This should be taken into consideration while making predictions and assessing oral health management. Association of denture and diabetes can increase the incidence of oral *Candida* disorders in diabetic patients.Candidiasis may appear asvarious symptoms including pain, burning sensation, difficulty in swallowing, change oftaste, but often they are asymptomatic[18].

Many studies have been done previously regarding prevalence of candidiasis among diabetic denture vs non-diabetic denture wearers but not much is known about the variety of species seen in them. The aim of the present study was to assess the prevalence of *Candida* and species diversity on the denture of diabetic and non-diabetic denture wearers.

A total of 30 subjects participated in the study. Fifteen subjects were denture wearers and fifteen subjects were non-denture wearers. All subjects were patients selected from the outpatient section of Saveetha Dental College and Hospital, Chennai. This study has been approved by the scientific review board of Saveetha dental college and hospitals. Patients included in the study were diabetic denture wearers. The controls included non-diabetic denture wearers. The subjects belonging to the age group between 40-80 years were included in this study.The exclusion criteria includes smokers, patient who are already treated or currently taking antibiotics, antifungal, steroids or immunosuppressive drugs in the past six months.

Patients were clearly explained about the purpose of the study, followed by a detailed information on demographics(name, age, sex),history of denture wearing, history of diabetes, type of diabetes, medications, duration, family history and history of other drugs was obtained using a questionnaire.

The diabetic status of the patients was determined by the history and the dentures worn included removable partial dentures and complete dentures. The salivary samples were collected in sterile containers. The samples were immediately transported to the Microbiology laboratory, Saveetha Dental College and inoculated in Sabouraud's dextrose agar (SDA) and incubated at 37 C for 48 hours for the recovery of *Candida* species.

Results

Out of the 30 subjects, the prevalence of *Candida* was 86% (n=13/15) in diabetic denture wearersand 46% (n=7/15) in non-diabetic denture wearers (Graph 1).

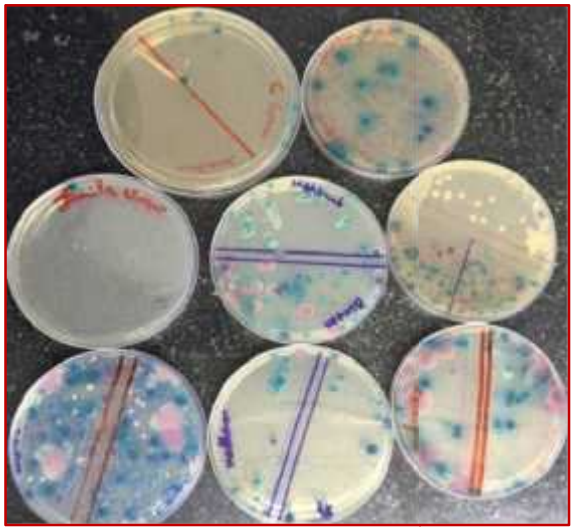


Figure 1: *Candida* colonization in diabetic denture wearers

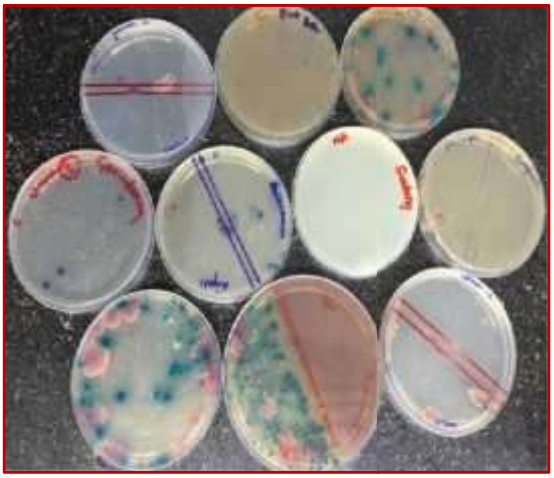
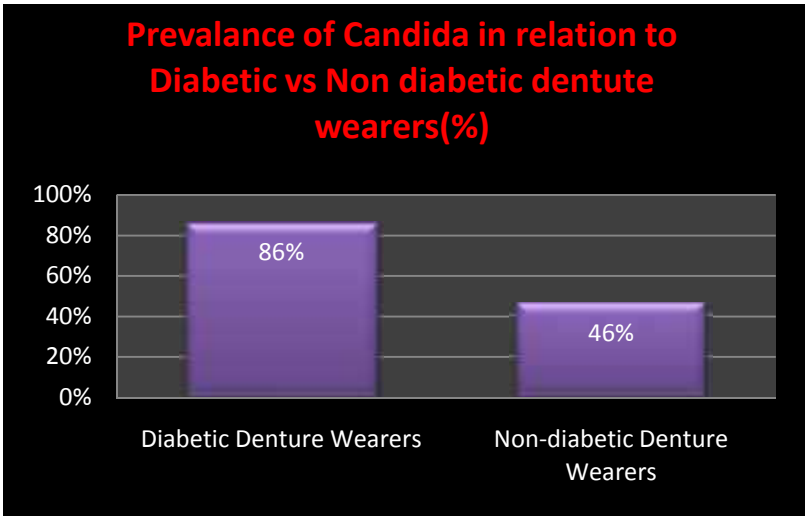


Figure 2: *Candida* colonization in non-diabetic denture wearers

C. albicans was the most commonly isolated species in both groups followed by *C. tropicalis, C. kefyr, C. krusei,* and *C. parapsilosis.*



Graph 1: Prevalence of Candida(%) in relation to diabetic denture & non-diabetic denture wearers.

Discussion

The sole factor of this study was denture wearing increases the candidalcolonizationin the oral cavity and its growth rate was found higher in diabetic denture wearers whencompared to non-diabetic denture wearers due to their immuno-compromised status. The obtained results were similar to the studies reported by Mohammad HosseinLotfi-Kamran et al [19]and Divya Rani M et al. in 2014 [20].

Oral candidiasis, also known as oral thrush, is one of the most common oral mucosal infections. Symptoms of oral candidiasis include oral discomfort, pain, loss of taste, and aversion to food. *Candida albicans* carriage and a history of oral candidiasis are other significant risk factors for oral candidiasis[21]. The infection is caused by *Candida albicans*, a dimorphic fungal organism that typically is present in the oral cavity in a nonpathogenic state in about one- half of healthy individuals. Normally present as a yeast, the organism, under favorable conditions, can transform into a pathogenic hyphael form. Conditions that favor this transformation include broadspectrumantibiotic therapy, acidic saliva, dry mouth, tobacco, carbohydrate rich diets, patients receiving radiotherapy and chemotherapy, xerostomia, immune dysfunction, or the presence of removable prostheses.

Historically, the ancient Greek physician Hippocrate (460–370BC) described oral candidiasis as “disease of diseased” [22]. This infection was classified according to Newton in 1962, based exclusively on clinical criteria, into three clinical types: type I is characterized by localized simple inflammation or pin-point hyperaemia. Diffuse erythema and oedema of palatal mucosal areas covered by dentures are included in Type II which is the most common type.. Type III is described as granular surface or inflammatory papillary hyperplasia in central palate [23]. Classic study has shown that this oral inflammation may occur on the maxilla and mandible; however, it is more often associated with the maxilla, sometimes found under maxilla partial dentures, but only rarely beneath mandibular dentures [24].

Infection with *Candida Albicans*may present in any form [25,26].

Pseudomembranous candidiasis commonly known as ‘thrush’ presents as soft, multiple,raised white plaques of material resembling milk curds that can easily be wiped away. These plaques consist of tangled aggregates of hyphae.

Acute erythematous candidosis is a clinical form of Candida albicans characterized by localized erythema of the oral mucosa, with or without associated symptoms.

Hyperplastic candidiasis has also been named as candidalleukoplakia. It clinically appears as a well demarcated, slightly elevated, adherent white lesion of the oral mucosa ranging from small translucent lesions to large, dense opaque plaques, that cannot be wiped away by the clinician.
Angular cheilitis form presents as cracking, peeling, or ulceration involving the corners of the mouth. Patients with a reduced vertical dimension of occlusion, secondary to severe attrition or worn dentures, are particularly susceptible to the development of angular cheilitis. This is due to the increased folding of the soft tissue that is frequently seen at the corners of the mouth, creating a haven for the organism.
C. tropicalis is the most virulent of the non-Candida albicans Candida species. This may be due to its ability to adhere toepithelial cells in vitro and its ability to secrete moderate levels of proteinase [27].

*Candida parapsilosis*has become an emerging fungal pathogen.Individuals at the highest risk for severe infection include neonates and patients in intensive care units and patients with catheters or other implanted devices.*C. parapsilosis*has capacity to grow in total parenteral nutrition and to form biofilms. Factors involved in disease pathogenesis include the secretion of hydrolytic enzymes, adhesion to prosthetics, and biofilm formation[28].
Candida krusei,isatransient,infrequent isolate of minor clinical significance inhabiting the mucosal surfaces. Morerecently it has emerged as a notable pathogen with a spectrum of clinical manifestations suchasfungaemia, endophthalmitis, arthritis and endocarditis, most of which usually occurincompromised patient groups in a nosocomial setting [29].
Candida kefyr (formerly *Candida pseudotropicalis*) is a yeast with its teleomorph currently recognized as Kluyveromycesmarxianus [30].*Candidapseudotropicalis* is a species of fungus belonging to the genus *Candida* and order *Saccharomycetales*. Alongside other members of the genus, is, in due course, that causes candidiasis. It is also very infectious, particularly in immunodeficient individuals [31].

Oral thrush occurs when your immune system is weakened. Diabetes is an illness that affects the immune system and can contribute to oral thrush. Also in diabetes, saliva may contain enormous amounts of sugar, which encourages the growth of *Candida*.Higher incidence of candida infections affecting various tissues and organs is seen in diabetic people.. Excess sugars are an excellent source of fuel that can rapidly increase the growth of candida.Candidaspeciesplays a role in shaping of the intestinal flora, which has been found to regulate blood sugar levels through effects on organs such as the pancreas and liver.The ability of the body to eliminate candida depends upon the effectiveness of the white blood cells. High or low levels of sugar in the blood reduces the effectiveness of the white blood cells, allowing candida to progress the ongoing disturbances and imbalances.Most of these effects are in regard to Type II diabetes but candida has also shown to create autoimmune conditions like Type I diabetes.

Frequently, when the host defence system suffers because of any alterations, like immune deficiency, Candida albicans become virulent and generate candidiasis, that can be manifested through various clinical forms, involving one or more oral sites, up to affect the whole oral cavity and to disseminate into invasive forms [32]. Many studies have demonstrated that there is an increased oral colonisation of Candida albicans among the diabetic subjects [33], comparable studies by Manfredi et al. and Al-Karaawi et al. present contradictory findings with decreased rates of candidalpresence in diabetic subjects compared with healthy controls. The level of glycaemic control appears to be a more significant factor than the presence of the disease itself, because poor control can reduce salivary ow and pH, increase salivary glucose level. These factors facilitate oral candidal growth and colonization. The saliva of diabetics favors the growth of Candida albicansand it has been shown that there are more elevated counts of colonies of the yeast on the denture surfaces of diabetic in comparison with the nondiabetic subjects. Thus, diabetic patients are more likely to acquire oral candidal infections than non-diabetic patients [34].

Acrylic dentures act as a predisposing factor in the occurrence of oral candidal infection. The dentures can act as a reservoir of infection, because of the surface irregularities, micro porosities, improper fit and suboptimal hygiene[35]. Acrylic resins are pervious and exhibit more water sorption. This may help the candidial cells to adhere or to even penetrate the surface of acrylic

resin.Surface irregularities of acrylic resin acts as a factor in the entrapment of microorganisms.*Candida* species possess surface free energy closer to that of polymethyl methacrylate. This makes possible for the yeasts to nest and make difficult to eliminate bacteria by mechanical and chemical hygiene maneuver [36]. Denture age is shown to be an important factor as a result of poor fit, roughness, inadequate hygiene, and accumulation of plaque due to aging of denture. It was reported, that aging of the denture and release of residual monomer with time results in poorer fit which affects the contamination of the denture. Moreover, age of dentures was proportional to Candida colonization. 25% of the individuals using dentures for less than 1 year were diagnosed with denture stomatitis, whereas more than 84% of those using dentures for more than 5 years had the disease [37].

Unclean dentures are a chronic source of potentially harmful bacteria and fungi that may be associated with oral and systemic diseases. Good hygiene control of the oral cavity and the denture is important, because the porous material and surface biofilm formed can serve as a reservoir of fungal microorganisms and lead to relapse or reinfection [38]. Both the prosthesis that oral mucosa in contact with it must be involved in procedures for oral hygiene through brushing them after each meal with water or an effective, nonabrasive chemical agents.The most commonly used mechanical methods for cleaning of dentures are brushing with dentifrice or neutral soap and use of ultrasonic devices. The chemical methods involve denture immersion in chemical products, such as sodium perborate, sodium hypochlorite and chlorhexidinedigluconate [39]. The patients should be instructed to remove the denture during night and to immerse in a suitable antimicrobial cleansing agent.Instead of dry and water preservation, using cleaning tablets for acrylic dentures for overnight storage reduces denture biofilm and pathogenicity of the mass which will contribute to the overall hygiene [40]. Prior to reinsertion into the oral cavity, dentures should be thoroughlycleaned by soaking and brushingusing denture-cleansing solutions.

Conclusion

The present study concludes that*Candidal* infection is more common among denture wearers and it is strongly associated with diabetes mellitus. Hence controlling serum glucose level, drinking enough water in diabetic patients with xerostomia is essential to prevent candidiasis.

Corresponding author

GurumoorthyKaarthikeyan

Professor ,Department of Periodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

SaveethaUniveristy,

162,Poonamallee High Road ,Chennai 600077,Tamil Nadu, India.

Email :drkarthik79@yahoo.co.in

Telephone Number: +91 9841222027

References

1. Hiroyuki Mizugai, Emiko Isogai, Kimiharu Hirose, Itsuo Chiba. J App Res 2007; 7(3).143-46
2. Sumi Y, Nagaosa S, Michiwaki Y, et al. J JGerodont 2001;16:171-178.Arenforf TM, Walker DM. Brit Dent J. 1979; 147:267-272.
3. Arenforf TM, Walker DM. Arch Oral Biol. 1980; 25:1-7.
4. E Emami, P de Grandmont, PH Rompré, J Barbeau. J Dental Res 2008;87(5):440- 444.
5. Darwazeh AM, Al-Refai S, Al-Mojaiwel S. Isolation of Candida species from the oral cavity and fingertips of complete denture wearers. J Prosthet Dent 2001;86:420-3.
6. NivedhaaChezhian, GeethaR.V.Prevalence of Oral Candidiasis in Periodontal Patients with Diabetes. J. Pharm. Sci. & Res. Vol. 7(9), 2015, 767-768.
7. Gonsalves WC, Chi AC, Neville BW. Am FAM Physician 2007; 75:501-7.
8. Ben-Aryeh H, Berdicevsky I, Gutman D, Szargel R. Int J Oral Surg 1980; 9:113-5.
9. Cruz PC, Andrare IM, Peracini A, Souza-Gugelmin MCM, Silva-Lovato CH, Souza RF. The effectiveness of chemi- cal denture cleansers and ultrasonic device in bio lm removal from complete dentures. J Appl Oral Sci2011;19:668-73.
10. Offenbacher S, Barros, Altarawneh S, Beck JD, Loewy ZG.Impact of tooth loss on oral and systemic health. J Gen Dent 2012; 6:494-500.

11. Farah CS, Lynch N, McCullough MJ. Oral fungal infections: an update for the general practitioner. Aust Dent J 2010;55:48-54.
12. Darwazeh AMG, Darwazeh TA. What makes oral Candidiasis recurrent infection? a clinical view. J Mycol2014;2014:758394.
13. Soell M, Hassan M, Miliauskaite A, Haikel Y, Selimovic D. The oral cavity of elderly patients in diabetes.DiabetesMetab. 2007;33(Suppl 1):S10–S18.
14. Barbeau J, Seguin J, Goulet JP, de Koninck L, Avon SL, Lalonde B, et al. Reassessing the presence of Candida albicans in denture-related stomatitis. Oral Surg Oral Med Oral Pathol Oral RadiolEndod.2003;(95(1)):51–9.
15. Samaranayake LP. Host factors and oral candidiasis. In: MacFarlane TW, Samaranayake LP, editors. Oral candidosis. London: Butterworth & Company Ltd; 1990. P.66-103.
16. Salim N, Moore C, Silikas N, Satterthwaite J, Rautemaa R. Candidacidal effect of uconazole and chlorhexidine released from acrylic polymer. J AntimicrobChemother 2013; 68:587-92.
17. Kanaguchi N, Narisawa N, Ito T, Kinoshita Y, Kusumoto Y, Shinozuka O. Effects of salivary protein ow and indigenous microorganisms on initial colonization of Candida albicans in an in vivo model. BMC Oral Health 2012;12:36.
18. Mohammad HosseinLotfi-Kamran,Abbas Ali Jafari,AbbasFalah-Tafti,EhsanTavakoliandMohammadHosseinFalahzadeh. Candida Colonization on the Denture of Diabetic and Non-diabetic Patients.Dent Res J (Isfahan). 2009 Spring; 6(1): 23–27
19. Divya Rani M, Surya Prabha P, LakshmipriyaandChitralekhaS.Prevalence of Candida among Denture and Non Denture Wearers. Research Journal of Pharmaceutical, Biological and Chemical Sciences. Jan 2014,11;2:123-6
20. Macphiual LA, Hilton JF, Dodd CL, Greenspan D. Prophylaxis with nystatin pastilles for HIV associated oral candidiasis. J Acquir Immune DeficSyndr 1996:12;470-6.
21. Yuvrajsinghdangi, Murarilalsoni, Kamtaprasadnamdeo. Oral Candidiasis; A Review. International journal of pharmacy & pharmaceutical sciences.Vol.2 issue 4, 2010.36-41.
22. Williams DW, Kuriyama T, Silva S, Malic S, Lewis MAO. Candida bio lms and oral candidosis: treatment and preven- tion. Periodontology 2000 2011; 55:250-65.

23. Naik AV, Pai RC. A Study of factors contributing to denturestomatitis in a North Indian Community.Int J Dent 2011, 2;11:589064.
24. Dandekeri S, Prasad K, Shetty M, Hegde C, Sowmya MK, Jagadeesh M. Occurrence of streptococcus and Candida spe- cies and salivary ph in patients wearing complete denture. Int J Health RehabilSci2013;2:198-203.
25. Hemashree J, Geetha VR. Oral candida species in patients with periodontitis and diabetes mellitus.International Journal of Recent Scientific Research 2017; 8(3):16222-16224.
26. G.P. Moran, D.J. Sullivan, D.C. Coleman. Emergence of non-*Candida albicans*species as pathogens. In: Calderone RA. *Candida* and Candidiasis. 4th Edition (ASM Press, Washington 2002; 4:37-53.
27. David Trofa, Attila GácserandJoshua D. Nosanchuk.*Candidaparapsilosis*, an Emerging Fungal Pathogen.ClinMicrobiol Rev. 2008 Oct; 21(4): 606–625.
28. Yuthika H. Samaranayakeand L. P. Samaranayake.*Candidakrusei:* biology, epidemiology, pathogenicity and clinical manifestations of an emerging pathogen.Review Article. Clinical Mycology. J. Med. Microbiol. 1994; 41:295-31.
29. Simon F. Dufresne , Kieren A. Marr, Emily Sydnor, Janet F. Staab, Judith E. Karp,Kit Lu, Sean X. Zhang, Christian Lavallée, Trish M. Perl and DionysiosNeofytos. Epidemiology of Candida kefyr in Patients with Hematologic Malignancies. Journal Of Clinical Microbiology 2014,5;1:121-22
30. José A. L. Fernandes, Tâmara H. R. Prandini, Maria da Conceiçao A. Castro, Thales D. Arantes, Juliana Giacobino, Eduardo Bagagli and Raquel C. Theodoro. Evolution and Application of Inteins in Candida species: A Review.Front. Microbiol.2016, 5:45-54
31. Lotfi-Kamran MH, Jafari AA, Falah-Tafti A, Tavakoli E, FalahzadehMH.*Candida* colonization on the denture of diabetic and non-diabetic patients. Dent Res J 2009;6:23-7.
32. Hoshing C, Dixit S, Mootha A, Diwan N. Role of Candida albicans in denture stomatitis. J Indian Acad Oral Med Radiol2011;23:617-9.
33. Javed F, Klingspor L, Sundin U, Altamash M, Klinge B, Engstrom PE. Periodontal conditions, oral Candida albicans and salivary proteins in type 2 diabetic subjects with emphasis on gender. BMC Oral Health 2009 9;12:23-7.
34. Farah CS, Lynch N, McCullough MJ. Oral fungal infections: an update for the general practitioner. Aust Dent J 2010;55:48-54.

35. Pereira-Cenci T, Del BelCury AA, Crielaard W, Ten Cate JM. Development of *Candida*-associated denture stomatitis: New insights. J Appl Oral Sci2008;16:86-94.
36. Radford DR, Sweet SP, Challacombe SJ, Walter JD. Adherence of *Candida albicans* to denture-base materials with different surface finishes. J Dent 1998;26:577-83.
37. Silva MM, Mima EGO, Colombo AL, Sanita PV, Jorge JH, Massucato EMS. Comparison of denture microwave disinfection and conventional antifungal therapy in the treatment of denture stomatitis: a randomized clinical study. Oral Surg Oral Med Oral Pathol Oral Radiol2012;114:469-79.
38. Salerno C, Pascale M, Contaldo M, Esposito V, Busciolano M, Milillo L. Candida-associated denture stomatitis. Med Oral Patol Oral Cir Bucal 2011; 16:139-43.
39. Pellizzaro D, Polyzois G, Machado AL, Giampaolo ET, Sanitá PV, Vergani CE. Effectiveness of mechanical brushing with different denture cleansing agents in reducing *in vitro Candida albicans* biofilm viability.Braz Dent J 2012;23:547-54.
40. Dantas APFM, Consani RLX, Sardi JCO, Mesquita MF, Silva MCVS, SinhoretiMAC.Bio lm formation in denture base acrylic resins and disinfection method using microwave. J Res Pract Dent 2014;4:1122-24.

Comparative Study of the Effect of Mcinnes Bleaching Agent and Commercially Available Bleaching Agent on Shear Bond Strength of Orthodontic Metal Brackets- In Vitro Study

Nishanthi.R[1],Saravana Dinesh [2],Antonette Rhea [3]

Graduate Student[1],Reader[2],Deprtment of Orthodontics,Senior Lecturer[3],Deprtment of Prosthodontics.

Saveetha Dental college,Saveetha Institute of Medical and TechnicalSciences, Saveetha University, Chennai, India

Abstract

Introduction:Tooth enamel is the most mineralized tissue of the human body. Teeth whitening is done by bleaching using in house preparations like Mcinnes solution or commercially available bleaching agents. This process could have deleterious effect on bond strength of orthodontic metal brackets when used in patient wearing fixedappliances.The objective of this research is to compare the effect of Mcinnes bleaching agent and commercially available bleaching agent on shear bond strength of orthodontic metal brackets.

Material and Methods: 15 human teeth bonded with orthodontic metal brackets were divided into three groups with 5 in each.Group 1: Teeth exposed to commercially available bleaching agent. Group 2: Teeth exposed to Mcinnes solution. Group 3: Control group without bleaching treatment is taken. Selected teeth were tested for shear bond strength using Instron universal testing machine. Data were statistically analyzed .

Result: We found that for enamel, the traditional approach of etching with a 35% phosphoric acid gel for 10 seconds and application of a primer led to accurate bonding of an orthodontic bracket.

Conclusion:The reason of the study is to determine whether bleaching can be done while patientis under orthodontic treatment .

***Key Words**: Orthodontic metal brackets,Mcinnes solution, Bleaching ,Shear bond strength.Enamel,Commercially available bleaching agent.*

Introduction

Bleaching of discoloured teeth may avoid invasive treatments such as prosthetic procedures that require the wear of a large quantity of dental tissue when they are performed.Sobleaching is considered a more conservative alternative,which significantly changes the appearance of the teeth and increases the patient's self esteem[1].Especially in patients who are undergoing orthodontic treatment,prosthetic procedures should be avoided or delayed until tooth movement has been performed[2]. Concentrated solutions of hydrogen peroxide are the most common agents used to bleach discoloured teeth.

The need for rebonding has resulted from the bond failure. One of the controversies that clinicians face with the bond failure is to decide if rebonding the same bracket would provide a sufficient bonding or a new bracket should be preferred. There are several commercial recycling methods available, but these are impractical to perform at the chairside. As a result, several in-office bracket recycling methods have been introduced. It has been found that the tensile bond strengths of the reused brackets were significantly lower than new brackets in vitro when a green stone was used to remove the surface layer of the residual resin on the bracket base.

Heating in flame was also found to decrease the bond strength dramatically.On the contrary, when sandblasting techniques were evaluated, it was found that sandblasting could increase the bond strength. Furthermore, articles tried to find a rapid office method of recycling brackets and found that sandblasting provided the simplest, most efficient way.

Nearly 30% of people benefit from orthodontic treatment, and the recent popularity of tooth whitening or bleaching also has a significant impact on the practice of dentistry and orthodontics. Although bleaching of vital teeth has been known since 1800's, the use of peroxide-based tooth whitening materials has increased recently. Some patients who are

interested in orthodontic treatment might have had their teeth bleached thus, it seems important to determine whether bleaching would significantly influence the bonding strength of orthodontic bracket adhesives to the enamel surface and how to reverse the effect in case it does.Previous studies have shown changes in the enamel structure and composition when exposed to 30.35% hydrogen peroxide for in-office vital bleaching. Some authors identified a substantial reduction in the bond strength. Acid etching of enamel is recommended with many dentinal bonding systems to improve the delivery of orthodontic treatment[3]. Claims of comparable bond strengths to enamel and dentin with conventional methods of bonding have meant that these adhesives are also suitable for orthodontic bonding .The development of an adhesive bond requires establishing intimate contact between the liquid adhesive and the solid adherent, minimizing the stress concentration at the interface and reducing the influence of environmental factors on the interface integrity .

The use of peroxide-based tooth-whitening materials haveincreased substantially in the past few years, despite many unanswered questionsabout their use.[4]The direct bonding of orthodontic brackets to the teeth is one of the most important advancements in modern dentistry. Three factors affecting the success of bonding are the tooth surface, the design of attachment base and the bonding material itself[5].The most common recommendation is to delay any bonding procedures after bleaching because the reduction of composite resin bond strength has shown to be transient.However, the recommended post-bleaching period for bonding procedures varies between 24 h and 4 weeksand hence it may be confusing for the clinician.[5,6]

Thus, this study was conducted to assess the changes in the shear bond strength (SBS) of metal brackets bonded with composite resins onto extracted teeth and exposed to bleaching solutions and also to compare the effect of Mcinnes bleaching agent and commercially available bleaching agent on shear bond strength of orthodontic metal brackets.

Several bonding protocols for effective bonding on enamel, composite, and ceramic surfaces have been introduced, but there is no agreement on the most effective procedure[6].Precoating of orthodontic brackets (with adhesive on the bracket mesh pad) was introduced to reduce the

number of operations needed for bonding as well as bonding time and bond failures (APC coated brackets, Unitek/3M, Monrovia, California, US). If precoated brackets are used, they should be stored in dark containers and the adhesive of these brackets should be cured using a dental light. Removal of adhesive flush around the bracket base is necessary for easier cleaning, and less white spot formation around the bracket base can be expected[7]. Prior studies reported conflicting results when using precoated brackets on different tooth surfaces. In this study, the accuracy of different procedures for bonding traditional metal brackets and precoated orthodontic brackets on different tooth surfaces was evaluated[8].

The cause of failures could be the alteration of enamel that follows previous bonding procedures. Bishara et al stated that the changes in bond strength after repeated bonding may be related to changes in the morphologic characteristics of the tooth surface caused by adhesive remnants[9].Various methods have been used to analyze tooth demineralization and remineralization when acidic bleaching is used, including techniques ranging from direct measures of mineral gain/ loss as in microradiography to indirect measures like iodide permeability and surface microhardness.

Material and Method

This study was done in the Department of Orthodontics and Dentofacial Orthopaedics,in Saveetha Dental College, Chennai .The bondstrengthanalysis was carried out at Central Institute of plastic engineering technology, Chennai. This study was carried out with an objective of assessing the effect of in-office hydrogen peroxide containing bleaching agent, and commercially available bleaching agent on shear bond strength of orthodontic metal brackets. Fifteen non-carious premolars extracted for orthodontic treatment were used for the study[10].Teeth with hypoplastic areas, cracks or gross irregularities of the enamel structures were excluded. No pretreatment with chemical agents such as alcohol, formalin or hydrogen peroxide or any other form of bleaching was allowed.The teeth were cleaned of blood and tissue debris and stored in normal saline after extraction. The saline was changed daily to avoid bacterialcontamination[11].

Teeth were divided into three experimental groups according to the bleaching agent used, and a control group, each containing 5 specimens.

-Group1 (n=5):deionised water(control).

-Group 2(n=5):Teeth bleached with mcinnes solution.

- Group 3 (n=5):Teeth bleached with commerciallyavailablebleaching agent.

Bonding Agent used:

Bonding agent used in the study was the Transbond XT (3M Unitek, Monrovia, USA) light cure orthodontic bonding system.

Bleaching Agentused :

A 35% hydrogen peroxide Pola office tooth whitening system (SDI Ltd., Victoria, Australia) was used to bleach the teeth .Each Group, the teeth were etched with 37% phosphoric acid gel for 30 seconds,then rinsed with water and dried till the enamel surface of the teeth showed frosty white appearance of etched enamel[12]

For those teeth that did not show white frosty appearance the above mentioned procedure was repeated. Then orthodontic metal brackets where bonded to tooth surface using light cure composite resin excess material was removed using a sharp probe and cured, using a light emitting diode (LED) light source and cured for 30 seconds. Group 2 was exposed to Mcinnes solution,Group3 was exposed to commercially available bleaching agent(Polo office bleaching agent).The teeth in all the groups were mounted individually on to a universal testing machine[13] for the debonding procedure.The data was statistically analysed.

Brackets:

One hundred stainless steel preadjusted edgewise upper bicuspid brackets (Ormco mini 2000 series, Ormco Corporation, Glendora, CA, USA) with 0.022 inch slot and micro etched base with a surface area of 9.63 mm2 were used for the study. The brackets were kept in the manufacturer's packaging until immediately prior to bonding and were handled at all times with bonding tweezers to avoid contamination[14].

The teeth used for the study were cleaned and then decontaminated with a 0.5% chlorine-T (Hi Media Laboratories, Mumbai) for 1 week at room temperature. The teeth were cleaned and stored in distilled water, which was changed twice weekly to prevent bacterial contamination. The root of each tooth was grooved in apical third with a diamond bur and then mounted in self-curing acrylic till cemento-enamel junction with long axis vertical[15]

Teeth were then divided into 5 groups randomly with 20 teeth in each group. The buccal surface of each tooth was polished with fluoride free pumice slurry, rinsed, and dried.

The buccal surface was then etched with 37% phosphoric acid for 30 s, rinsed for 15 s, dried, and checked for the frosted appearance[16].Transbond XT primer was applied to the enamel surface in a thin film with an applicator brush and light cured for 10 s. Transbond XT adhesive was applied to the bracket base and then, the bracket was positioned lightly on the buccal surface of the tooth. Excess adhesive was removed with an explorer and then, cured for 10 s on each side[17]

Group 2 (Bleached Group)

A 35% hydrogen peroxide bleaching agent was mixed and applied with a brush onto the tooth surface in a layer of approximately 1 mm in thickness, ensuring no enamel was visible. It was then exposed to the curing light twice for 10 s and the bleaching agent was kept in place for 16 min before washing away. Brackets were then immediately etched and bonded as in the control group.

Group :3

Teeth bleached with commercially available bleaching agent(Polo office bleaching kit)

Results

Table 1:Shear bond strength values of the different groups.

Group 1(Control group)	Group 2	Group 3
90.21	80.20	59.99
49.31	36.71	48.12
30.46	12.76	23.76

15.17	8.88	12.33
11.23	5.76	10.16

- Group 1(n=5):deionised water(Control)

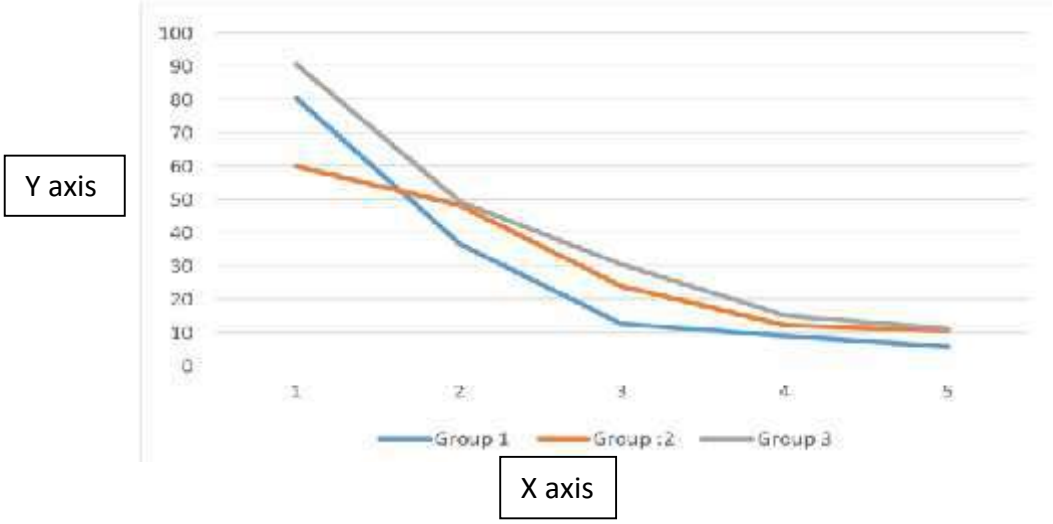
-Group 2(n=5):Teeth bleached with mcinnes solution.

-Group 3 (n=5):Teeth bleached with commercially available bleaching agent(Polo office)

Table 2 :- Statistically analysed values

Test groups	n	Mean	Sd	P value
Group 1	5	34.965	42.806	
Group 2	5	28.826	31.109	0.027
Group 3	5	30.872	22.185	

Graph shows that there is decrease in the shear bond strength of orthodontic metal brackets after treatment with Mcinnes solution and Commercially available bleaching agent(Polo office bleaching agent).



The value of the Walkthrough statistical test and the p = 0.027, indicates a statistically significant difference.

Discussion

Many patients are keeping their teeth for longer time but dislike the physiological darkening process that follows the laying down of secondary dentine. The use of chemical agents to remove certain intrinsic stains from the enamel or the dentin of vital teeth is not new. Hydrochloric acid and hydrogen peroxide have been used alone and in combination to remove stains associated with fluorosis, tetracycline and more recently trauma related injuries.The purpose of this in vitro study was to test an optimal protocol for bonding orthodontic brackets on various dental surfaces.

We found that for enamel, the traditional approach of etching with a 35% phosphoric acid gel for 10 seconds and application of a primer led to accurate bonding of an orthodontic bracket. For bonding orthodontic brackets onto composite surfaces, roughening of the surface with a drill and cleaning with water and air spray, followed by drying of the surface and application of a primer seems to be sufficient for effective bonding of brackets.[18] For bonding on ceramic surfaces, surface preparation using etching with hydrofluoric acid to roughen the ceramic surface for a mechanical retention followed by application of a thin layer of a silane-coupling agent (porcelain activator) was needed for a chemical bonding between the adhesive and the ceramic surface.[19]

It was expected that an increased roughness of the dental surface should increase the mechanical interlocking between the dental surface and the adhesive. In this study, we found an increase in surface roughness did not lead to an increase in bond strength. As permanent damage caused by the surface treatment required for bonding should be avoided, it can be concluded that roughening of the dental surface should and can be reduced without reduction of bonding efficiency. Studies evaluating differences in the efficiency of non-precoated and precoated brackets had different outcomes.

Theoretically, the placement of adhesive in the factory precoating should result in an effective connection between the bracket mesh and reduce bracket failures. In this study, no significant differences in bonding strength were found between these two groups, except when bonding brackets on a sandblasted composite surface. In general, the advantages of precoated brackets such as increased bonding efficiency, shorter bonding times, and less bond failures should be evaluated. The use of extracted premolars as tooth surfaces for bonding orthodontic brackets could have introduced some variables, such as the fluoride concentration in the enamel and the storage of the tooth after extraction at the start of the study. Finally, the results of this study are only applicable for the materials and methods used.

The use of chemical agents to remove certain intrinsic stains fromthe enamel or the dentin of vital teeth is not new.Hydrochloric acid and hydrogen peroxide have been used alone and in combination to remove stains associated with fluorosis, tetracycline and more recently trauma related injuries[20].

It was in the year 1966 that McInnes reported a technique that combined hydrochloric acid and hydrogen peroxide to remove fluorosis stains.He used a solution of five parts 30% hydrogen peroxide, five parts 36% hydrochloric acid and one part ethyl ether and applied the solution with a cotton wrapped toothpick to the areas of the teeth affected by the stain. After 10 to 15 minutes the teeth were washed with water and neutralized with a sodium bicarbonate paste.

In the present study McInnes bleaching agent and commercially available bleaching agent wasused.Articlehas reported that McInnes bleaching technique were specifically recommended for the treatment of teeth exhibiting endemic dental fluorosis because of its superficial nature, easy manipulation.All the samples used in the study were treated with Mcinnes bleaching agent and commercially available bleaching agent showed decreased value of Shear bond strength immediately after bleaching.

It is not uncommon for patient undergoing orthodontic treatment to develop stains on their teeth.The duration of Orthodontic treatment on an average is not less than 18 months.And it is not unfair from the patients point of view to desire a teeth whitening procedure such as bleaching.

There are number of studies regarding the interaction between the bleaching agents such as carbamide peroxide, hydrogen peroxide and bond strength of the composite resin to enamel.

The studies conducted by Adanir, etalin 2007 and Dishman in 1994 has found that there is reduced bond strength when the teeth is treated with the 35% hydrogen peroxide and immediately bonded with the light cure composite for bonding procedure[21]. Several authors have reported significant reduction in the bond strength of brackets bonded to enamel, immediately after bleaching.

One of the reasons suggested for the reduced bond strength is because of the release of free oxygen radical of the bleaching agents on the tooth surface[22].

The present study was conducted to find in-office bleaching agent on the bond strength of orthodontic bonding adhesive to enamel. In the present study, Group 3showed the least mean Shear bond strength among all the groups. Thus, the result of this study demonstrated that the reduction in Shear bond strength of composite resin to enamel treated with Mcinnes solution.

Conclusion

The present study shows that bleaching reduced the Shear bond strength significantly when compared to the control group.The Shear bond strength was drastically reduced when orthodontic metal brackets was exposed to commercially available bleaching agent compared to that of Mcinnes solution.

Coressponding Author

Antonette Rhea,

Senior Lecturer[3]

Deprtment of Prosthodontics.

Saveetha Dental College,Saveetha University ,

Saveetha Institute of Medical and Technical Sciences.

162,Poonamallee High Road,

Chennai 600077,

Tamilnadu ,India.

Reference

1. Uysal T, Bascici FA, Usumez S, Sari Z, Buyukerkmen A. Can previously bleached teeth be bonded safely? Am J Orthod Dentofacial Orthop. 2003 Jun;123(6):628-32.

2. Haywood VB, HeymannHO.Nightguard vital bleaching. Quin- tessence Int 1989;20:173-6.

3. Larson TD. The effect of peroxides on teeth and tissue review of the literature. Northwest Dent 1990;69:29-31.

4. Socratic D, Hailer AW, Takagi S, Antonucci JM, Eanes ED. Quantitative assessment of the efficacy of amorphous calcium phosphate/methacrylate composites in remineralizing caries-like lesions artificially produced in bovine enamel. J Dent Res. 1996;75:1679–86.

5. Chen JH, Xu JW, Shing CX. Decomposition rate of Hydrogen Peroxide bleaching agent under various chemical and physical condition.J Prosthet Dent. 1993;69:46–8.

6. Lai SC, Tay FR, Cheung GS, Mak YF, Carvalho RM, Wei SH, et al. Reversal of compromised bonding in bleached enamel. J Dent Res. 2002;81:477–81.

7. Bulut H, Turkun M, Kaya AD. E ect of an antioxidizing agent on the shear bond strength of brackets bonded to bleached human enamel. Am J Orthod Dentofacial Orthop. 2006 Feb;129(2): 266-72.

8. Miyazaki M, Sato H, Sato T, Moore BK, Platt JA. E ect of a whitening agent application on enamel bond strength of self-etching primer systems. Am J Dent. 2004 Jun;17(3):151-5.

9. Suleiman M, Addy M, Macdonald E, Rees JS. The bleaching depth of a 35% hydrogen peroxide based in-office product: A study in vitro. J Dent. 2005;33:33–40.

10. Harshitha. Effects of Tooth Whitening Agents in Non VitalTeethJ. Pharm. Sci. & Res. 2014; 6(3):124 – 126.

11. Deepa, S Aruna, R Ganesh, M Joyson Journal of Indian Society of Pedodontics and Preventive Dentistry, Comparison of the bleaching efficacy of three different agents used for intracoronal bleaching of discolored primary teeth: An in vitro study, ijbps. 2013; 31(1):21

12. Y Madhukar Rao, V Srilakshmi, K KarpagaVinayagam, L Lakshmi Narayanan.An evaluation of the color stability of tooth-colored restorative materials after bleaching using CIELAB colortechnique 2009;20 (1) :60-64

13. McEnoy SA. Chemical agents' for removing intrinsic stains from vital teeth Technique development. Quintessence Int. 1989;20:323–8.

14. McCabe JF, Buckley JG. A critique of bond strength testing in orthodontics.Br J Orthod. 1994;21:33–43.

15. Eslamian L, Borzabadi-Farahani A, Mousavi N, Ghasemi A. The effects of various surface treatments on the shear bond strengths of stainless steel brackets to artificially-aged composite restorations. Aust Orthod J. 2011;27:28–32.

16. MazzaouiSA, Burrow MF, Tyas MJ, Dashper SG, Eakins D, Reynolds EC. Incorporation of caseinphosphopeptide-amorphous calcium phosphate into glass-ionomer cement.J Dent Res. 2003;82: 914–8.

17. Featherstone JD, Cutress TW, Rodgers BE, Dennison PJ. Remineralization of artificial caries like lesions invivo by a self administered mouth rinse or paste.Caries Res. 1982; 16: 235–42.

18. White DJ, Faller RV, Bowman WD. Demineralization and remineralization evaluation techniques--added considerations. J Dent Res. 1992;71: 929–33.

19. Purdell-Lewis DJ, Groeneveld A, Arends J. Hardness test on sound enamel and artificiallydemineralized white spot lesions. Caries Res. 1976;10: 201–15.

サ

Co-Relation between Consumption of Milk and Oral Streptococcus and Lactobacillus Count- A Study

Anirudha Kabilan[1], Muralidharan[2], Subash Sharma[3]
Graduate student[1], Professor[2], Department of Microbiology,
Reader[3], Department of Conservative Dentistry and Endodontics
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Milk is found to be one of the naturally available foods that have a beneficial role in oral and periodontal diseases. Milk and milk products are said to be rich in bioactive peptides and calcium which have important functions such as enamel-protection and anticaries effects.Milk product like cheese exhibit cariostatic effect. Casein, a phosphoprotein, is a major constituent of bovine milk .This phosphoprotein is actively involved with caries control. It was previous noted that increased consumption of casein rich food decreased the dental caries risk. This article is aimed to detect the co-relation between the intake of milk and milk products to the oral bacterial count.To determine the co-relation between consumption of milk and oral Streptococcus and Lactobacillus count.

Materials and Methods: To carry out this study, a screening questionnaire was prepared in order to separate the participants into 2 groups, one who consumed plain milk and the other group contained people who took milk with the addition of sugar. Following this screening, unstimulated saliva samples were collected from the participants half an hour after consumption of their respective variants of milk. After collection, these samples were transported to the laboratory. The samples were diluted in a dilution of 1 in 20 with saline after and they were cultured in mutants and lactobacillus agar respectively. They were kept overnight in an incubator which was maintained at 37 degree C. The bacterial colonies were counted in the next dawn.

Result : On comparing the bacterial count present on either side of the spectrum it was noted that the amount of Streptococcus in the test and control group was nearly equal , indicating that the sugar consumption does not instantaneously affect bacterial number. When the lactobacillus counts were scrutinized, difference that was sighted was noteworthy. Thereby the leading to conclusion that intake of sugar tends to alter the oral environment to a conducive state where the lactobacillus can actively thrive.

Conclusion: Diet has a local effect on oral health, primarily on the integrity of the teeth, pH, and composition of the saliva and plaque. Alterations in nutrient intake secondary to changes in diet intake, absorption, metabolism, or excretion can affect the integrity of the teeth, surrounding tissue and bone as well as the response to wound healing.

Key Words*: Milk, streptococcus, cariostatic, lactobacillus, sweeteners.*

Introduction

Milk is one of the naturally available sources of complete nutrition. With recent advancements in the field of food technology increasing importance is being received by products of less nutritive value. Flavouring agents which claim to submerse the undesirable taste of raw milk are being widely used in the past decades. Apart from all these addition to milk, the most important additive added are sweeteners be it sugar, honey, dates or artificial sweeteners. During the recent times many articles have claimed the cariostatic effect exhibited by milk and cheese. Various components of milk have been considered to be protective against dental caries. The elements like minerals, casein and other lipids present in milk exhibit a cariostatic action [1]. Articles have stated that long term consumption of non sweetened milk is linked with lower caries risk. Hence as a confirming study we desired to compare the immediate changes in the oral Streptococcusmutants and lactobacillus count in people who consume plain milk regularly and people who consume milk with the addition of sugar.

Materials and Methods

To carry out this study, a screening questionnaire was prepared in order to separate the participants into 2 groups, one who consumed plain milk and the other group contained people who took milk with the addition of sugar. Following this screening, unstimulated saliva samples were collected from the participants half an hour after consumption of their respective variants of milk. After collection, these samples were transported to the laboratory. The samples were diluted in a dilution of 1 in 20 with saline after and they were cultured in mutants and lactobacillus agar respectively. They were kept overnight in an incubator which was maintained at 37 degree C. The bacterial colonies were counted in the next dawn.

Results

The salivary samples collected from 15 individuals who consumed plain milk and 15 individuals who consumed milk with the addition of sugar which were cultured for S.mutants and lactobacillus were counted after overnight incubation[2]. On comparing the bacterial count present on either side of the spectrum it was noted that the amount of Streptococcusmutants in participants who had consumed plain milk and milk with the addition of sugar(half an hour before sample collection) that is the test and control group was nearly equal , indicating that the sugar consumption does not instantaneously affect bacterial number. When the lactobacillus counts were scrutinized, difference that was sighted was

noteworthy. Thereby the leading to conclusion that intake of sugar tends to alter the oral environment to a conducive state where the lactobacillus can actively thrive. The above mentioned information are presented in the form of a pie chart for quick cognition.

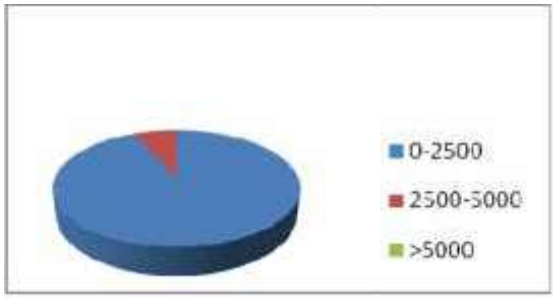


Figure 1: Shows the Lactobacillus count in subjects who consumed

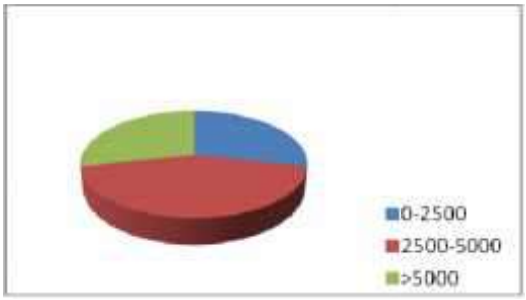


Figure 2: Shows the Lactobacillus count in subjects who consumed milk with the addition of sugar.

Discussion

Milk, one of the most commonly consumed foods, under certain circumstances exerts cariostatic effect. Bovine milk contains several proteases including plasmid, plasminogen, plasminogen activators, thrombin, cathepsin D, acid milk proteases, and aminopeptidase.

Milk and other dairy products not only have a low cariogenic potential but are also claimed to exhibit cariostatic potential. It has been culminated in many studies, involving in rats and *in situ*, that bovine milk and cheese safe guard against caries even under caries-susceptible conditions. The active constituents caseins and peptides, more precisely casein glycomacropeptide (CGMP) and casein phosphopeptide (CPP), is said to reduce the adhesion of cariogenic bacteria and reduce colonization [3]. CGMP binds to the tooth as micelle-like structures, which do not bind bacteria. CPP binds as minor complexes with calcium and phosphate, buffering calcium and phosphate when hydroxyapatite solubility increases by decreasing pH, and possibly explaining the acid-buffering effect from milk and cheese. Further proteose-peptones,provide protection against tooth tissue demineralization. In addition other milk peptides namely kappacin, lactoferrin, lactoperoxidase and lysozyme, possess innate immunity-like functions. Using milk components as a caries-prophylactic measure has not been studied in humans, but epidemiological studies confirm associations between milk and cheese intake and protection against caries [4] .

.

Milk contains 4–5% of the disaccharide lactose, which can be fermented by oral flora.

However, unless the bacteria are adapted to lactose, fermentation is signicantly less than from sucrose. Physiologically sucrose lowers pH of the oral cavity below 5.0, whereas lactose lowers pH to an approximate value of 6 . Inspite of variations among the homosapeins , it is a known fact that a pH lower than 5.5 is extremely injurious to the enamel of the tooth and lower than 6.2 to root tissues [5].Thus it is noteworthy that under normal circumstances the carbohydrate content of milk confers a low cariogenic potential to milk and dairy products but the act of drinking milk or chewing on cheese[6]. Removal of casein, fat or lactose from the milkdoes not modify the protective capacity on demineralization as the existence of free calcium and phosphate ions in the dairy products are have some favourable effect[7]. Apart from the above mentioned components milk still encompasses certain robust protective factors that are named as water-soluble proteose-peptones,amounting to 1 g l¼1 milk .However the action of milk is not exhibited in case of increase uptake of cariogenic diet in human beings. The cells of the oral epithelial and the secretory cells in the salivaryglands produce antibacterial peptides, commonly known as innate immunity peptides, that act immediately in the oral cavity upon the onset of an insult or challenge [8]. These peptides are formed as a result of proteolytic cleavage from larger precursor proteins, namely lactoferricin, which is both bacteriostatic and fungistatic. Recentlycaseino-macropeptide (CMP), a C-terminalfragment from bovine k-casein, has shown to inhibit the growth of *S. mutans* and *Porfyromonasgingivalis* ,responsible for caries production and periodontal diseases respectively. The other bioactive milk polypeptides, Lactoperoxidase andlysozyme, reduce the metabolism of *S.mutans*.

The beneficial activity of milk in the oral cavity is masked due to the addition of sugars and other types of artificial flavours and hence the caries activity is increased due to the creation of favourable environment. The oral flora plays a pivotal role in the initiation and propagation of dental caries[9].

Dental caries a disease of microbial origin which causes dissolution of the calcified part of the tooth structure is initiated by Streptococcus mutant's .Lactobacillus a causative organism for pit and fissure caries and smooth surface caries. The saliva present in the oral cavity acts as indictors for various disease conditions. The extensive presence of certain bacteria in saliva may act as markers for various pathological states

Many studies suggest that increase in Streptococcus and lactobacillus count is associated with increased caries risk and high DMFT and DMFS scores [10]. The immediate effect of sugar added milk in the oral cavity was to create a friendly environment for the lactobacillus to

survive thus confirming the risks associated with long term consumption .Apart from increased caries risk increase in lactobacillus count is found to be closely associated with conditions like nervous breakdown, hyposalivationetc . The cariogenic properties of bacteria is linked to sucrose metabolism are implicated in two pathogenic properties namely adherence and acid production.It has been shown that lactobacillus were able to adhere to various cell cultures and that the mechanisms of adhesion involved a certain level of specificity.

Lactobacilli cell surfaces have acrystalline protein layer which is responsible for the surface hydrophobicity[11]. The bacteria are able to adapt their surface hydrophobicity and to environmental changes like pH. The production of exopolysaccharides is a key factor in the adherence of dental biofilm.

The property of cariogenicity in latobacilli is due to their capacity to produce acids and their ability to grow and survive in acidic environment. These bacteria have a fermentative metabolism. Thus it results in an acidification of the environment, lowering the pH until values are less than 4.5. [12] the cariogenic properties of bacteria is linked to sucrose metabolism are implicated in two pathogenic properties namely adherence and acid production. It has been shown that lactobacillus were able to adhere to various cell cultures and that the mechanisms of adhesion involved a certain level of specificity.

The cariogenic properties of bacteria is linked to sucrose metabolism are implicated in two pathogenic properties namely adherence and acid production. It has been shown that lactobacillus were able to adhere to various cell cultures and that the mechanisms of adhesion involved a certain level of specificity.

Milk and dairy products are an inevitable, substantial part of the food consumed by Indians [13], though mean intake of dairy products is high , individual variations are ought to be taken into consideration in this diverse population. An array of studies have negatively correlated the development of dental caries and consumption of milk in children and adolescents. The strongest association was detected among children with high intake of any form of sucrose [14]. Further the development of root caries among gediatric people was halved on consumption of cheese and other probiotic products. Furthermore xerostomia , caused due to, head and neck irradiation, or primary and secondary Sjogren’s syndrome.

Overdose or adverse reaction of medication are often associated with either an experience of mild to severe discomfort in the oral cavity or rampant caries as a result of elevated numbers of mutans streptococci. Hyposalivation is also another concern which is repeatedly associated with issues during mastication, inadequate energy and nutrient intake With regards to lactobacillus, at present, there are no indications that the bacteria used in yoghurt and fermented products colonize the mouth[15]. Lactobacilli-containing products have reported to significantly decrease the number of mutans streptococci in saliva Thus a drink based on milk or milk components might can be used to serve both purposes. Milk and milk based drinks seem to possess a bunch of the biological and physical properties that are desired for a saliva substitute. Hence the need for frequent intake of liquid in order to lubricate and provide adequate moisture to the mouth is met, at the same time as providing caries protection to highly susceptible individuals [16].

Conclusion

The ultimate focus of the present study was to describe, seed in the minds of health care professionals, the potential anticariogenic properties of milk and cheese. This naturally available bioactive peptide which can be exploited in the field of dentistry due to its cariostatic properties is being pointed out as an important causative agent of caries due to the addition of certain products which may tend to alter its desirable property. The main focus is to notify the instantaneous effect of sugar on oral cavity when added along with a potential caries suppressing agent[17].The above mentioned effect of milk in the oral cavity provides nothing but a short term overview of the future benefit that can essentially be obtained by the consumption of non sweetened milk. Although forms of cariogenic, cariostatic, and anticariogenic food exists , the frequency of sugars consumption and other fermentable carbohydrates, retention time, nutrient composition, the potential of the food to stimulate saliva do exist as caries altering factors[18]. Caries risk which also depends on individual host factors like the presence of altered salivary pH, use of medications, incidence of systemic or local diseases that affect the immune system, andpersonal hygiene habit also play an essential role in the associated caries risk[19][20].

The cariogenic risk associated with individual foods is challenging to determine but regular consumption of plain milk can or may have innate potential to arrest the cavity or atleast inhibit the adsorption of sugar rich entities onto the tooth surface.[20]As mentioned various studies the remineralizing capacity of the tooth may also be exhibited. Thus it can be

concisely said that consuming plain milk on a regular basis without any addition can be beneficial. This beneficial activity of milk should not be ignored but should be exploited to the utmost level.

Corresponding author

Subash Sharma

Reader, Department of Conservative Dentistry and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Phone Number**:** +91 9884533118

Email Id**:**drsubashsharma@gmail.com

Contact Number: 9176222104

Reference

1. Keene HJ, Shklair IL. Relationship of Streptococcus mutans carrier status to the development of carious lesions in initially cariesfree recruits. Journal of dental research. 1974 Sep;53(5):1295-.
2. Kashket S, DePaola DP. Cheese consumption and the development and progression of dental caries. Nutrition Reviews. 2002 Apr 1;60(4):97-103.
3. Krobicka A, Bowen WH, Pearson S, Young DA. The effects of cheese snacks on caries in desalivated rats. Journal of dental research. 1987 Jun;66(6):1116-9.
4. Aguirre M, Collins MD. Lactic acid bacteria and human clinical infection. Journal of Applied Microbiology. 1993 Aug 1;75(2):95-107.
5. Littleton NW, Kakehashi S, Fitzgerald RJ. Recovery of specific “caries-inducing” streptococci from carious lesions in the teeth of children. Archives of oral biology. 1970 May 1;15(5):461-3.
6. Tanzer JM. Virulence of mutants defective in glucosyltransferase, dextran-mediated aggression, or dextranase activity. Molecular Basis of Oral Microbial Adhesion. 1985:204-11.
7. Heller KJ. Probiotic bacteria in fermented foods: product characteristics and starter organisms–. The American journal of clinical nutrition. 2001 Feb 1;73(2):374s-9s

8. Panesar PS, Panesar R, Singh RS, Kennedy JF, Kumar H. Microbial production, immobilization and applications of β- D- galactosidase. Journal of Chemical Technology and Biotechnology. 2006 Apr 1;81(4):530-43.
9. Touger-Decker R, Van Loveren C. Sugars and dental caries. The American journal of clinical nutrition. 2003 Oct 1;78(4):881S-92S.
10. Beighton D. The complex oral microflora of high- risk individuals and groups and its role in the caries process. Community dentistry and oral epidemiology. 2005 Aug 1;33(4):248-55.
11. Johansson I. Milk and dairy products: possible effects on dental health. Scandinavian journal of Nutrition. 2002 Jan 1;46(3):119-22.
12. Bowen WH, Pearson SK, VanWuyckhuyse BC, Tabak LA. Influence of milk, lactose-reduced milk, and lactose on caries in desalivated rats. Caries Research. 1991;25(4):283-6.
13. Fitzgerald RJ, Fitzgerald DB. The microbiologic status of test animals in relation to caries research. InAnimal models in cariology: proceedings of a Symposium and Workshop on Animal Models in Cariology 1980 Apr 21:89-91.
14. Saravanan S, Madivanan I, Subashini B, Felix JW. Prevalence pattern of dental caries in the primary dentition among school children. Indian Journal of Dental Research. 2005 Oct 1;16(4):140.
15. Kuramitsu HK. Virulence factors of mutans streptococci: role of molecular genetics. Critical Reviews in Oral Biology & Medicine. 1993 Jan;4(2):159-76.
16. Chokshi A, Mahesh P, Sharada P, Chokshi K, Anupriya S, Ashwini BK. A correlative study of the levels of salivary Streptococcus mutans, lactobacilli and Actinomyces with dental caries experience in subjects with mixed and permanent dentition. Journal of oral and maxillofacial pathology: JOMFP. 2016 Jan;20(1):25.
17. Holbrook WP, De Soet JJ, De Graaff J. Prediction of dental caries in pre-school children. Caries research. 1993;27(5):424-30.
18. Woods WA. Fermentation of carbohydrates and related compounds. The bacteria: a treatise on structure and function. 1961:59-100.
19. Loesche WJ, Syed SA. The predominant cultivable flora of carious plaque and carious dentine. Caries research. 1973;7(3):201-16.
20. Smith AV, Bowen WH. The Effects of Milk and Kappa–Casein on Salivary Pellicle Formed on Hydroxyapatite Discs in situ. Caries research. 2000;34(1):88-93.

CorrelationofClinicalandRadiographicDiagnosisofPulpoperipicalPathologies – A CohortStudy

Hansini B. Ajantha[1], Laliytha K.B[2], Pavithra Priyadarshoni S[3]
Graduate Student[1], Senior Lecturer[2],Department of Oral Medicine and Radiology,
Tutor[3], Department of Public Health Dentistry
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Diagnosis in dentistry is defined as the process by which the data collected from history taking, clinical examination, special tests and radiographic examination are combined to identify deviations from the normal status and render an appropriate treatment. Vitality testing and radiographic examination aids in an appropriate diagnosis of pulpal and periradicular diseases. The aim of the present study is to find out periapical radiographic changes involving vital teeth with acute pulpitis, chronic pulpitis and chronic hyperplastic pulpitis.

Materials and Methods: The present study was conducted in SaveethaDentalCollege, in the department of Oral Medicine and Radiology. Ethical committee clearance and the patient's consent were obtained.A clinical and radiographic examination was done on a total of 200 teeth diagnosed of pulpitis.Clinical diagnosis was made by correlating the history, heat test, cold test and electric pulp test. Radiographic diagnosis was made with an intraoralperiapical radiograph.The results obtained were subjected to descriptive statistical analysis.

Results: In a total of 200 teeth with pulpitis, 119 (59.5%) had chronic pulpitis, 58 (29%) had acute pulpitis and 23 (11.5%) had chronic hyperplastic pulpitis.

Conclusion: Thus to conclude, an acurate clinical diagnosis of pulp status and radiographic diagnosis of periapical changes of tooth with pulpitis is of paramount importance to render an appropriate treatment.

Key Words*: Periradicular changes, Pulpitis, Intraoral Periapical Radiograph, Heat test, Cold test*

Introduction

Dental caries is considered the chief cause of pulpal injury. The pulp reactions in deep carious lesions have been attributed to bacterial toxins, not to a direct result of bacterial invasion (1). Diagnosis in dentistry is defined as the process by which the data collected from history taking, clinical examination, special tests and radiographic examination are combined to identify deviations from the normal status and render an appropriate treatment. Vitality testing and radiographic examination aids in an appropriate diagnosis of pulpal and periradicular diseases (2). If a vital tooth with pulpitis is left untreated for a longer period, the infection may extend into periradicular tissues, such cases are referred to as pulpoperiapical periodontitis. The diseases of the pulp and periradicular tissues are progressive. The signs and symptoms vary depending on the stage of the disease. There are some limitations associated with pulp testing modalities and radiographic techniques when done individually (3). A complete interpretation of both pulpal and periradicular tissues must be done so as to provide an appropriate treatment. The aim of this study is to find out periapical radiographic changes in vital tooth with acute pulpitis, chronic pulpitis and chronic hyperplastic pulpitis.

Acute, Reversible Pulpitis: A mild to moderate inflammatory condition of the pulp caused by noxious stimuli, in which the pulp is capable of returning to the non-inflamed state following removal of the stimuli.

Chronic, Irreversible Pulpitis: A persistent inflammatory condition of the pulp, symptomatic or asymptomaticin nature with pulp becoming incapable of healing.

Chronic Hyperplastic Pulpitis: A productive pulpal inflammation due to an extensive various exposure of young pulp, characterised by the development of granulation tissue, covered at times with epithelium and resulting from long standing, low grade irritation.

Materials and Methods

The present study was conducted in Saveetha dental college from October 2016 to Decemberr 2016. Ethical committee clearance and patient’s consent was obtained. A clinical and radiographic examination was done on total of 200 teeth diagnosed with pulpitis. Vital tooth with acute pulpitis, chronic pulpitis and chronic hyperplastic pulpitiswas included and

tooth without opposing tooth, pathologically migrated tooth, mobile tooth, tooth with severe periodontitis, non vital tooth, tooth with periapical pathologies like abscess and cyst, maxillary molars and premolars was excluded. Clinical diagnosis was made by correlating the history, heat test, cold test and electric pulp test. Depending on history taking, clinical examination and pulp testing, clinical diagnosis of three types of pulpitis was made: Acute reversible pulpitis, Chronic irreversible pulpitis and Chronic hyperplastic pulpitis. Radiographic examination was done using intraoral periapical radiograph. Radiographic interpretation was done for the following findings: discontinuity in lamina dura (fig 1), thickening of lamina dura (fig 2), widening of periodontal ligament space (fig 3), periapical rarefying osteitis (fig 4), periapical condensing osteitis (fig 5), periapical condensing and rarefying osteitis (fig 6). The results obtained were subjected to statistical analysis.

Results

In a total of 200 teeth with pulpitis (table 1), 119 (59.5%) had chronic pulpitis, 58 (29%) had acute pulpitis and 23 (11.5%) had chronic hyperplastic pulpitis.

In 23 teeth with chronic hyperplastic pulpitis (table 2), 5 (21.7%) had widening of periodontal ligament space, 2 (8.7%) had discontinuity in lamina dura, 4 (7.3%) had thickening of lamina dura, 3 (13.1%) had periapical rarefying osteitis, 6 (26.1%) had periapical condensing osteitis and 3 (13.1%) had periapical condensing and rarefying osteitis.

In 119 teeth with chronic pulpitis (table 3), 27 (22.6%) had widening of periodontal ligament space, 16 (13.4%) had discontinuity in lamina dura, 21(17.4%) had thickening of lamina dura, 15 (12.5%) had periapical rarefying osteitis, 19 (16%) had periapical condensing osteitis and 11 (9.1%) had periapical condensing and rarefying osteitis.

In 58 teeth with acute pulpitis, 17 (29.3%) had widening of periodontal ligament space, 14 (24.1%) had discontinuity in lamina dura, 3 (5.2%) had thickening of lamina dura and 24 (41.4%) had no periapical changes.

Table 1: Types of Pulpitis

	N=200	**Percentage(%)**
Chronic hyperplastic pulpitis	23	11.5
Chronic pulpitis	119	59.5
Acute pulpitis	58	29

Table 2: Radiographic Changes in Chronic Hyperplastic Pulpitis

Chronic hyperplastic pulpitis N=23	**Radiographic changes**	**Percentage (%)**
5	Widening of periodontal ligament space	21.7
2	Discontinuity in lamina dura	8.7
4	Thickening of lamina dura	17.3
3	Periapical rarefying osteitis	13.1
6	Periapical condensing osteitis	26.1
3	Periapical condensing and rarefying osteitis	13.1

Table 3: Radiographic Changes in Chronic Pulpitis

Chronic pulpitis n=119	**Radiographic changes**	**Percentage (%)**
27	Widening of periodontal ligament space	22.6
16	Discontinuity in lamina dura	13.4
21	Thickening of lamina dura	17.4
15	Periapical rarefying osteitis	12.5
19	Periapical condensing osteitis	16
11	Periapical condensing and rarefying osteitis	9.1

Figure 1: Discontinuity of Lamina Dura

Figure 2: Thickening of Lamina Dura

Figure 3: Widening of Periodontal Ligament

Figure 4: Periapical Rarefying Osteitis

Figure 5: Periapical Condensing Osteitis

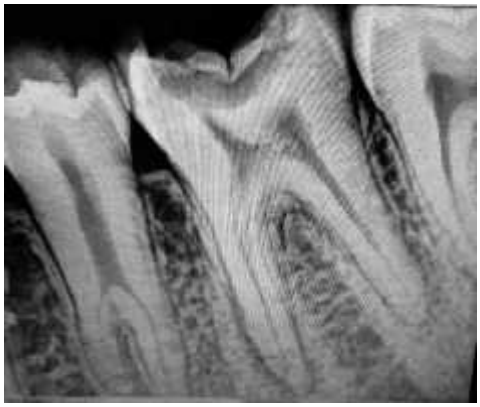


Figure 6: Periapical Condensing and Rarefying Osteitis

Discussion

It is significant to understand pulpal innervation characteristics in order to appreciate the mechanisms involved in the tests of pulpal vitality. Within the coronal pulp, nerve bundles diverge and branch out towards the pulpo dentin border, and emerge from their myelin sheaths (4),(5). Nerve divergence continues until each bundle looses its integrity and smaller fibre groups travel towards the dentine. This course is relatively straight until the nerve fibres form a loop and a resultant mesh termed the nerve plexus of Rashkow. Terminal axons exit from the Schwann cell investiture and pass between the odontoblasts as free nerve endings (6). This nerve plexus is well developed in the peripheral pulp along the lateral wall of coronal and cervical dentin, and along the occlusal aspect of the pulp chamber. Two types of sensory fibres are present in the pulp: the myelinated A fibres and unmyelinated C fibres. The A fibres predominantly innervate the dentine and are subgrouped according to their diameter and conduction velocities into Aβ and Aδfibres. The Aβ fibres may be more sensitive to stimulation than the Aδfibres, but functionally these fibres are grouped together. Approximately 90% of A fibres are Aδfibres. The C fibres innervate the body of the pulp. The Aδfibres have lower electrical thresholds than the C fibres, and respond to a number of stimuli which do not activate C fibres (7). Aδfibres mediate acute, sharp pain and are excited by hydromechanical events in dentinal tubules such as drilling or air-drying (8). The C fibres mediate a dull, burning, and poorly located pain, and are activated only by stimuli reaching the pulp proper (9),(10). C fibres have a high threshold and can be activated by intense heating or cooling of the tooth crown. Once activated, the pain initiated by C fibres can radiate throughout the face and jaws. C fibre pain is associated with tissue injury and is modulated by inflammatory mediators, vascular changes in blood volume and flow, and increases in pressure (11). As the intensity of the stimulus increases, more sensory nerves are activated which results in a progressive increase in the sensory response. The response to a given stimulus will be to the maximum where neural density is the highest. Important factors known to affect the response to pulp testing are the thickness of the enamel and dentin, and the number of nerve fibres in the underlying pulp.

Thermal tests include the application of cold and heat stimuli by various methods to the affected tooth, to determine sensitivity to thermal changes. Although cold and heat tests are based on sensitivity, they are actually conducted for different diagnostic purposes. A response

to cold stimuli usually indicates a vital pulp, regardless of whether that pulp is normal or affected.Contrasty, an increased response to heat stimuli is suggestive of pulpal or periapical pathology that may require endodontic intervention.

Cold Test

This test causes contraction of thedentinal fluid present within the dentinal tubules,resulting in a rapid outward flow of dentinal fluidwithin the patent dentinal tubules (12). This rapid movementof dentinal fluid results in 'hydrodynamicforces' acting on the Aδnerve fibres within thepulp–dentine complex, leading to a sharp pain, lastingduring the thermaltest (13). A large number of cold tests areemployed, the major difference between them is the degreeof cold that is applied to the affected tooth.Ideally, cold testsshould be done in conjunction with an electric pulp tester so that the results from one test will verify the findings of the other test. If a non-traumatized tooth does not respond either to electric pulp test or cold test, then the tooth is considered non-vital (14). The cold test is used to differentiatebetween reversible and irreversible pulpitis. Itshould be noted, whether stimulusapplication produces a lingering effect or if thepain subsides immediately on removal of the coldstimulus from the tooth. If the patient feels alingering pain, even after the cold stimulusis removed, a diagnosis of irreversible pulpitis given. Contrastly, if the pain subsidesimmediately after cold stimulus removal, adiagnosis of reversible pulpitis is given. The clinician should also take into considerationof other factors, such as a history of pain onlying down and the duration of pain. The diagnosisof reversible or irreversible pulpitis is onlya clinical diagnosis and may not correlate witha histological diagnosis.A simple method of applying a cold stimulusto a tooth is to wrap a sliver of ice in wetgauze and place it against the buccal surface,comparing the reaction between the test toothand a control tooth. Pencils of ice can be madeby filling a plastic straw or disposable syringe with water and freezing it in an upright position in a refrigerator.Ethyl chloride (boiling point – 41°C) may besprayed onto a cotton pellet, resulting in theformation of ice crystals, prior to applicationto the tooth. Dichlorodifluoromethane (DDM) (boiling point – 0°C) is a compressed refrigerantspray, which can similarly be sprayedonto a cotton pellet for cold testing (15). Another effective cold stimulus is frozen carbon dioxide (CO_2), also known as 'dry ice' or 'carbon dioxide snow' (boiling point – 72°C). This investigation is particularly effective when trying to assess teeth that have been restored with full-coverage metal restorations (16). When a cold test is

being done, one must begin with the most posterior tooth and advance towards the anterior teeth. This sequence will prevent any melted ice water dripping in a backward direction which may cause stimulation ofother posterior teeth, hence giving a false response.

Heat Test

This test can be undertaken using a heated gutta-percha stick or hot water. A guttaperchastick is heated with a flame or an electric heater until it becomes soft and glistens. It is then applied to the vaseline coated, buccal surface of the affected or test tooth. This test is difficult to use on posterior teeth because of limited access and a further disadvantage is that excessive heating may result in pulpal damage. Prolonged heat application will result in stimulation of Aδfibres initially, followed by the C fibres. Activation of C fibres may result in a lingering pain, therefore heat tests should not be applied for more than 5 seconds. However, inadequate heating of the guttapercha stick could result in the stimulus being too weak to elicit a pulpal response. The hot water can be administered through an irrigating syringe under rubber dam isolation, is also described as a means of heat testing (17). Frictional heat may be generated by using a rubber cup used for oral prophylaxis, without paste, against the buccal surface of a tooth (18).

Electric Pulp Test (EPT)

The objective of electric pulp test is to stimulate the intact Aδnerves in the pulpal dentin complex by applyingan electric current on the affected tooth surface. Apositive result is due to an ionic shift in thedentinal fluid within the dentinal tubules causing localdepolarization and subsequent generation ofan action potential from intact Aδ nerves. The electric pulp tester is a battery operatedinstrument, which is connected to a probe. Itfunctions by producing a pulsating electricalstimulus. The initial electric stimulus shouldbe at a very low value to prevent excessivestimulation and discomfort. The intensity ofthe electric stimulus is then increased steadily, and a note is made of the read-out on the digital display when thepatient acknowledges a sharp, tinglingsensation. The read out is not a quantitativemeasurement of pulpal health, but simplyimplies that the Aδfibres are sufficientlyhealthy to function of the pulp(19**)**.The electric pulp testis technique sensitiveand has a number of limitations. The requirementsof an electric pulp test are an adequate stimulus, an appropriate application method, and carefulinterpretation of results. Tooth isolation duringelectric pulp

testissignificant. Adequate drying of the enamel, placementof an interproximal plastic strip, and use ofrubber dam can prevent the spread of electricalimpulses to adjacent teeth or gingival tissue(20).Electric current can also be transferred toadjacent teeth through contacting metallic restorations (21). A conducting medium should also beused to ensure that maximum current passesfrom the electrode to the tooth surface (22).

The majority of radiographic periapical changes were seen in chronic pulpitis and chronic hyperplastic pulpitis. This might be due to the long standing inflammatory response, which has reached the periapical region through apical foramen of the infected tooth (23).Depending on the duration of infection, virulence of the microbes involved and the bacterial activity, the radiographic changes seen in periapical region can be widening of periodontal ligament, discontinuity in lamina dura, thickening of lamina dura, periapical rarefying osteitis, periapical condensing osteitis or periapical condensing and rarefying osteitis.

Similarly, Marmary et al (24), suggested a possible relationship between pulpitis and periapical changes. Olsson et al (25) and Sagne et al (26), also mentioned that the periapical changes may be due to the infection from the root canal of the infected tooth. In chronic, irreversible pulpitis, the partially necrosed pulp has functional sensory response and responds to vitality testing, but the radiographic picture may vary. In the present study, chronic pulpitis and chronic hyperplastic pulpits showed a predominance of widening of periodontal ligament space (22.6%) and periapical condensing osteitis (26.1%), respectively. In acute pulpitis, 58.6% cases showed periapical changes, in which, a predominance of widening of periodontal ligament space (29.3%) and remaining 41.4% cases showed no periapical changes. Dayal PK et al (27) has also shown similar results in his study with predominance of periodontal ligament space widening in chronic pulpitis and periapical condensing osteitis in chronic hyperplasticpulpitis.

Conclusion

Periapical changes are most commonly seen in chronic pulpitis and chronic hyperplastic pulpitis with predominance of periodontal ligament space widening and periapical condensing osteitis, respectively. Rapid advances in knowledge and advanced diagnostic aids in pulpal assessment should be used for more objective, accurate and proper diagnosis.

Corresponding Author

PavithraPriyadarshoni S.,

Tutor, Department of Public Health Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email ID: drpavithraphd88@gmail.com

Contact Number: 9176222104

References

1. Neelakantan P, Rao CV, Indramohan J. Bacteriology of deep carious lesions underneath amalgam restorations with different pulp-capping materials-an in vivo analysis. Journal of Applied Oral Science. 2012 Apr;20(2):139-45.
2. VelayuthamGopikrishna; Assessment of pulp vitality: a review; International journal of paediatric dentistry 2009;19: 3-15.
3. Thirumalai V, Neelakantan P. Pulp vitality tests. International Journal of Clinical Dentistry. 2014 Jan 1;7(1).
4. Dahl E, Mjör IA. The structure and distribution of nerves in the pulp–dentin organ. ActaOdontolScand1973; 31: 349–356.
5. Gunji T. Morphological research on the sensitivity of dentin. Arch HistolJpn1982; 45: 45–67.
6. Byers MR, Narhi MO. Nerve supply of the pulpodentin complex and responses to injury. In: Hargreaves KM, Goodis HE (eds). Seltzer and Bender's Dental Pulp. Chicago, IL: Quintessence Publishing, 2002: 155–157.
7. Olgart L. Excitation of intradental sensory units by pharmacological agents. ActaPhysiolScand1974; 92: 48–55.
8. Byers MR. Dental sensory receptors. Int Rev Neurobiol1984; 25: 39–94.
9. Narhi MVO. The characteristics of intradental sensory units and their responses to stimulation. J Dent Res 1985; 64: 564–571.

10. Markowitz K, Kim S. Hypersensitive teeth. Experimental studies of dentinal desensitizing agents. Dent Clin North Am 1990; 34: 491–501.
11. Narhi MVO. The neurophysiology of the teeth.Dent Clin North Am 1990; 34: 439–448.
12. Brännström M. A hydrodynamic mechanism in the transmission of pain-producing stimuli in dentine. In: Anderson DJ (ed.). Sensory Mechanisms in Dentine. Oxford: Pergamon Press, 1963: 73–77.
13. Brännström M. The hydrodynamic theory of dentinal pain: sensation in preparations, caries and dentinal crack syndrome. J Endod1986; 12: 453–457.
14. Trowbridge HO, Franks M, Korostoff E, Emling R. Sensory response to thermal stimulation in human teeth. J Endod1980; 6: 405–412.
15. Peters DD, Baumgartner JC, Lorton L. Adult pulpal diagnosis. Evaluation of the positive and negative responses to cold and electric pulp tests. J Endod1994; 20: 506–515.
16. Cohen S, Burns RC. Pathways of the Pulp, 8th edn. St Louis, MI: Mosby, 2002: 521.
17. Trope M, Sigurdsson A. Clinical manifestations and diagnosis. In: Ørstavik D, Pitt Ford TR (eds). Essential Endodontology: Prevention and Treatment of Apical Periodontitis. Oxford: Blackwell Science, 1998: 157–178.
18. Pitt Ford TR, Rhodes JS, Pitt Ford H. Endodontics. Problem-solving in Clinical Practice. London: Martin- Dunitz 2002: 12.
19. Walton RE, Torabinejad M. Diagnosis and treatment planning. In: Walton RE, Torabinejad M (eds). Principles and Practice of Endodontics, 3rd edn. Philadelphia,PA: W.B. Saunders, 2001: 49–70.
20. Dummer PMH, Tanner M, Mccarthy JP. A laboratory study of four electric pulp testers. IntEndod J1986; 19: 161–171.
21. Mumford JM. Relationship between the electrical resistance of human teeth and the presence and extent of dental caries. Br Dent J 1956; 100: 239– 244.
22. Myers JW. Demonstration of a possible source of error with an electric pulp tester. J Endod1998; 24: 199–200.
23. Michaelson RE, Seidberg BH, Guttuso J. An in vivo evaluation of interface media used with the electricpulp tester. J Am Dent Assoc1975; 91: 118–121.

24. Byer RM, Patrick GK, Charles LK, Effect of injury and inflammation on pulpal and periapical nerves, Journal of Endodontology. 1990; 16:78-83.
25. Marmary Y, Kutiner GA: Radiographic survey of periapical jaw bone lesions, oral surg, 1986;61:405-408.
26. Olsson G, Sagne S. Studies of Periapical Status in a Medieval Population. Dentomaxillofacial Radiology. 1976 Jan;5(1-2):19-23.
27. Dayal PK; Pulpoperiapical periodontitis: A radiographic study; Endodontology. 1999(11); 60-64.

Correlation of Salivary Flow Rate and Oral Manifestations in Diabetes and Health Patients

Yendodu Varshitha[1], Arvind. M[2], Gheena.S[3]

Graduate Student[1],Professor[2],Department of Oral Medicine and Radiology,Reader[3], Department of Oral Pathology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Science,Saveetha University, Chennai, India

Abstract

Introduction: Salivary diagnosis is increasingly becoming an important field in dentistry. Saliva is adapted to protect the tooth against dental caries and it has an immense ability to wash the tooth surfaces, it alsocontrolsmineralization and demineralization. Saliva is the principal defensive factor in the mouth, and a reduction in its flow rate affects orodental health. A decreased salivary flow rate may cause a variety of unspecific symptoms and so establishment of patients saliva flow is of primary importance in oral pathology and dentistry. The purpose of undertaking this study is to assess the salivary flow rate in the study groups and correlate with the observed oral manifestation so as to explore the treatment modality of improving the saliva flow and there by oral health.

Materials and Methods: In this study about 30 individuals were taken of which 15 are diabetic and 15 are non-diabetic. The age group of the sample population varied between 30 to 83 years. Saliva collection from the patient was done after the breakfast. Unstimulated saliva was collected using draining method. Saliva was collected for a period of five minutes measured individually for each minute.The study was conducted in city medical college, Chennai, Tamil Nadu.

Results: This study showed significant variation in salivary flow rates between diabetes and health emphasizing that most of the oral conditions are closely correlated to salivary secretion of

the individual. Of the oral manifestations screened, we found 100% positivity for dental caries followed by periodontitis.

Conclusion: This research was undertaken to increase the awareness of oral complications among diabetes and health.

Key Words: *Diabetes mellitus, Saliva, Periodontal diseases. Dental caries, Hyposalivation.*

Introduction

Diabetes mellitus is a metabolic diseasewhich is caused by the total or partial absence of insulin secretion.as group of metabolic diseases which is characterized by increased glucose levels due to problem of secretion of insulin, by insulin action, or both. The other name of diabetes mellitus is iceberg disease[1]. Diabetes mellitus has affected about 285 million people of world population; in 2014 it has reached 387 patients whichis 8.7%.

Diabetes has become universally experienced growing public health concern and common chronic metabolic disease [2-5]. Diabetes mellitus stands as sixth leading disorder that leads to death. The factors that are responsible of inducing salivary disorders in diabetic patients is ageing, head and neck radiotherapy, systemic disorders, and several drugs.It is associated with many oral complications like periodontitis, dental caries, halitosis, mucosal ulcers and certain fungal infections. Saliva plays an important role in controlling the above mentioned complications. Salivary secretions are important for maintaining oral health, assisting in the mechanical cleaning and also serving protective functions through the physiological and biochemical mechanisms. Hypo salivation contributes to the development of many health problems that can have serious negative effects on the quality life of the patient, affecting their eating habits, nutritional state, palate, speech, and tolerance of dental prostheses. These effects may increase the risk of oral infection, including candidiasis, and the patient's susceptibility towards dental caries, periodontal disease, and tooth loss.Italso acts as a defensive mechanism for orodental problems which requires early diagnosis and intervenation.Measurement of

salivary flow rate is an invaluable diagnostic tool in determining the prognosis of alternative treatment plan (Stahl et al., 1990)[6].

Materials and Methods

The participants in this study were 30 individuals of which 15 are diabetic and 15 are non-diabetic. The age group of the sample population varied between 30 to 83 years. The study was conducted in private medical college, Thandalam, Chennail, Tamilnadu.

Collection of Saliva

Saliva collection from the patient was collected between 9 a.m to 12 p.m after the breakfast.The patient was asked to rinse his or her mouth with mouthwash prior to collection of saliva Unstimulated saliva was collected using draining method by asking the patient to sit quietlywith head bent down and mouth open to allow the saliva to drip from lower lip in sampling tube.saliva was collected for a period of five minutes;measured individually for each minute.

The Normality tests Kolmogorov-Smirnov and Shapiro-Wilks tests results reveal that the variables except age do not follow Normal distribution. Therefore to analyse the data Parametric & Non-Parametric methods are applied. To compare mean age between groups independent samples t-test is used. To compare the salivary flow rate between groups Mann Whitney test is applied. To compare proportions between groups Chi-Square test is applied, if any expected cell frequency is less than five then Fisher's Exact test is applied. SPSS version 22.0 is used to analyse the data. Significance level is fixed as 5% ($\alpha = 0.05$).

Results

Descriptive Statistics

Table 1: Age distribution of individuals in groups

Age	Group			P- value
	Diabetic	Non Diabetic	Total	
N	15	15	30	0.001
Mean	57	40	49	
Std. Dev	15	10	15	
Minimum	31	30	30	
1st Quartile	45	33	33	
Median	54	36	47	
3rd Quartile	73	49	57	
Maximum	83	60	83	

In the present study, 30 subjects 15 diabetic and 15 non diabetic controls who were in the age range between 30 to 83 years were enrolled. Mean age of the diabetic and non diabetic subjects is 56.87 years and 40.27 years with statistical significance (p=0.001)(Table 1).

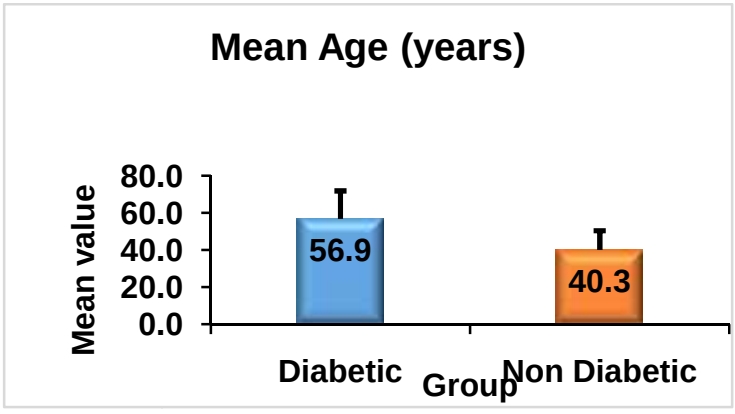


Figure 1: Mean age of the sample subjects in diabetic and non diabetic

Table 2: Saliva collected in the first minute

Saliva / 1st Min	Group			P-value
	Diabetic	Non Diabetic	Total	0.038
N	15	15	30	
Mean	0.8	0.4	.6	
Std. Dev	0.6	0.1	.5	
Minimum	0.0	0.2	.0	
1st Quartile	0.5	0.4	.5	
Median	0.5	0.5	.5	
3rd Quartile	1.5	0.5	.5	
Maximum	1.5	0.5	1.5	

Table 3: Saliva collected in the second minute

Saliva / 2nd Min	Group			P value
	Diabetic	Non Diabetic	Total	

N	15	15	30	0.050
Mean	0.4	0.5	0.5	
Std. Dev	0.6	0.3	0.5	
Minimum	0.0	0.1	0.0	
1st Quartile	0.0	0.5	0.0	
Median	0.0	0.5	0.5	
3rd Quartile	0.5	0.5	0.5	
Maximum	1.5	1.0	1.5	

Table 4: Saliva collected in the third minute

Saliva /3rd Min	Group			P value
	Diabetic	Non Diabetic	Total	
N	15	15	30	0.090
Mean	0.4	0.4	0.4	
Std. Dev	0.6	0.1	0.4	
Minimum	0.0	0.2	0.0	
1st Quartile	0.0	0.4	0.0	
Median	0.0	0.5	0.5	

3rd Quartile	0.5	0.5	0.5	
Maximum	1.5	0.6	1.5	

Table 5: Saliva collected in the fourth minute

Saliva / 4th Min	Group			P value
	Diabetic	Non Diabetic	Total	
N	15	15	30	0.004
Mean	0.3	0.5	0.4	
Std. Dev	0.5	0.2	0.4	
Minimum	0.0	0.2	0.0	
1st Quartile	0.0	.5	0.0	
Median	0.0	.5	0.5	
3rd Quartile	0.5	.5	0.5	
Maximum	1.5	1.0	1.5	

Table 6: Saliva collected in the fifth minute

Saliva / 5th Min	Group			P value
	Diabetic	Non Diabetic	Total	
N	15	15	30	0.011
Mean	0.3	0.5	0.4	
Std. Dev	0.5	0.2	0.4	
Minimum	0.0	0.2	0.0	
1st Quartile	0.0	0.5	0.0	
Median	0.0	0.5	0.5	
3rd Quartile	0.5	0.5	0.5	
Maximum	1.5	1.0	1.5	

Table 7: Mean value of salivary flow rate in diabetic and non diabetic

Saliva flow rate (Average)	Group			P value
	Diabetic	Non Diabetic	Total	
N	15	15	30	0.080
Mean	0.45	0.47	0.46	
Std. Dev	0.49	0.11	0.35	

Minimum	0.00	0.30	0.00	
1st Quartile	0.10	0.40	0.24	
Median	0.24	0.50	0.42	
3rd Quartile	0.70	0.50	0.50	
Maximum	1.50	0.68	1.50	

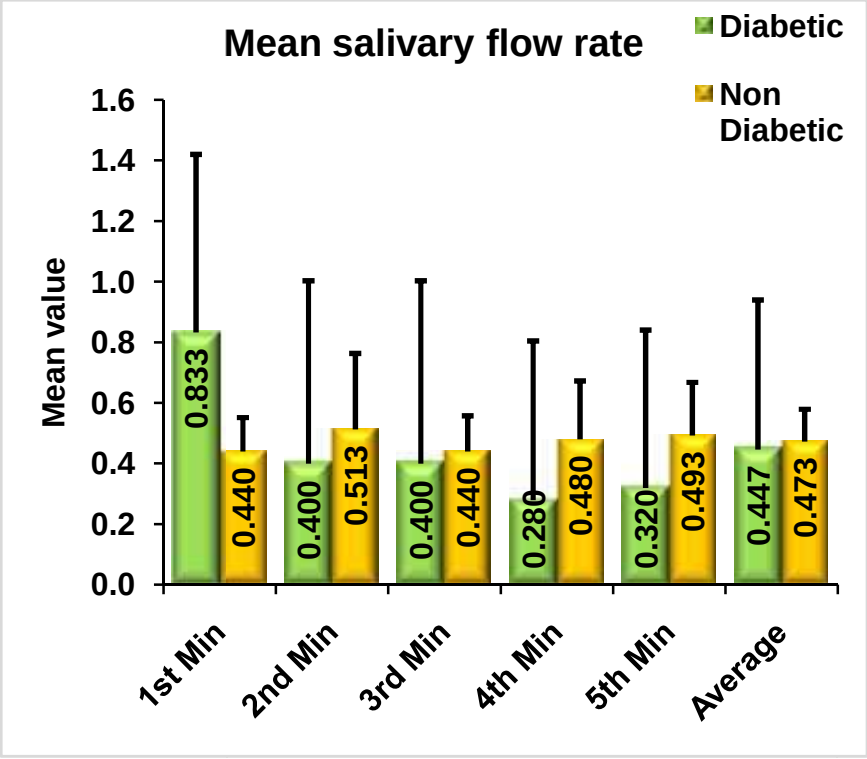


Figure 2: Salivary flow rate of first, second, third, forth. Fifth and the average

In this study the saliva is collected for 5 minutes, at each 1 minute the amount of saliva should be measured.In the 1st minute the mean salivary flow rate is significantly higher in diabetic than non diabetic subjects i.e. 18.43 on diabetic and 12.57 in non diabetic. Where as in second, third, fourth and fifth minute the mean flow rates has completely decreased compared to that of the non diabetic patients showing hyposalivation (Table 2-7), (Figure 2).

Table 8: Chi-Square test to compare gender proportions between groups

Gender	Group						P value
	Diabetic		Non Diabetic		Total		
	N	%	N	%	N	%	0.121
Male	12	80.0	8	53.3	20	66.7	
Female	3	20.0	7	46.7	10	33.3	

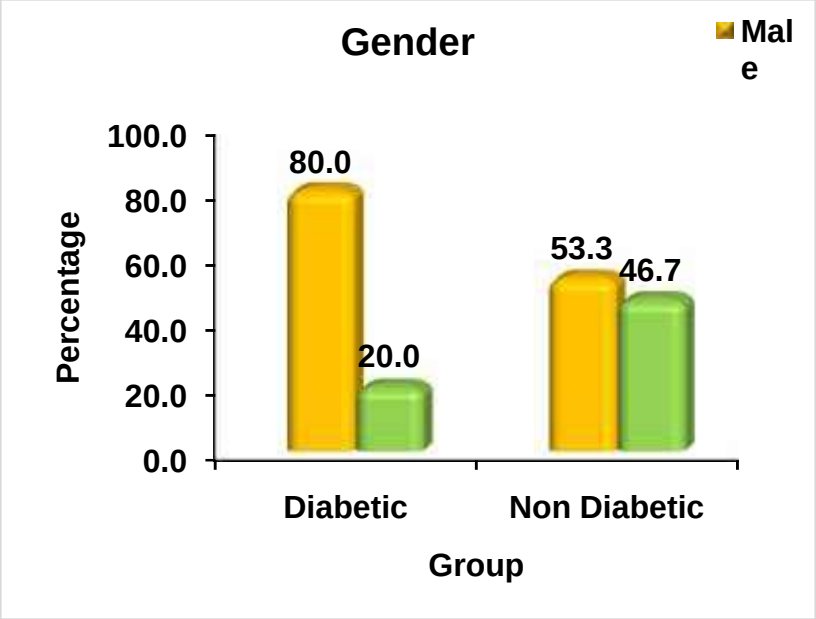


Figure 3: Comparison of gender proportions between groups

Among the diabetics there were 12 male and 3 female with mean age of 57 years. Among non diabetic patients there were 8 males and 7 females with mean age of 40 years with statistical significance of 0.121(table 8, Figure 3)

Table 9: Periodontitis in diabetic and non-diabetic

Periodontitis	Group						P value
	Diabetic		Non Diabetic		Total		
	N	%	N	%	N	%	
Yes	11	73.3	3	20.0	14	46.7	0.003
No	4	26.7	12	80.0	16	53.3	
Total	15	100.0	15	100.0	30	100.0	

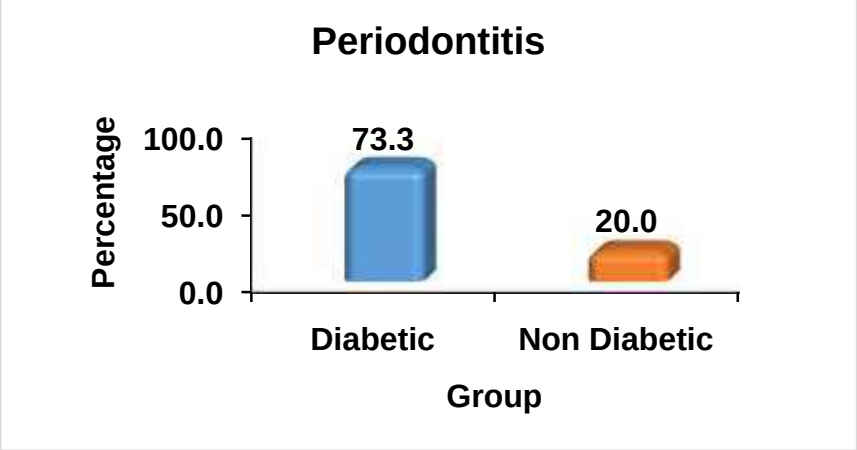


Figure 4: Periodontitis in diabetic and non-diabetic

According to the examination done on the patients regarding oral manifestation, about 73.3% of diabetic subjects have periodontitis where as only 20% periodontal problem seen in non diabetic with statistical significance (p=0.003)(Table 9, Figure 4)

Table 10: Oral candidiasis in diabetic and non-diabetic

Oral Candidiasis	Group						P value
	Diabetic		Non Diabetic		Total		
	N	%	N	%	N	%	
Yes	2	13.3	0	.0	2	6.7	0.483
No	13	86.7	15	100.0	28	93.3	
Total	15	100.0	15	100.0	30	100.0	

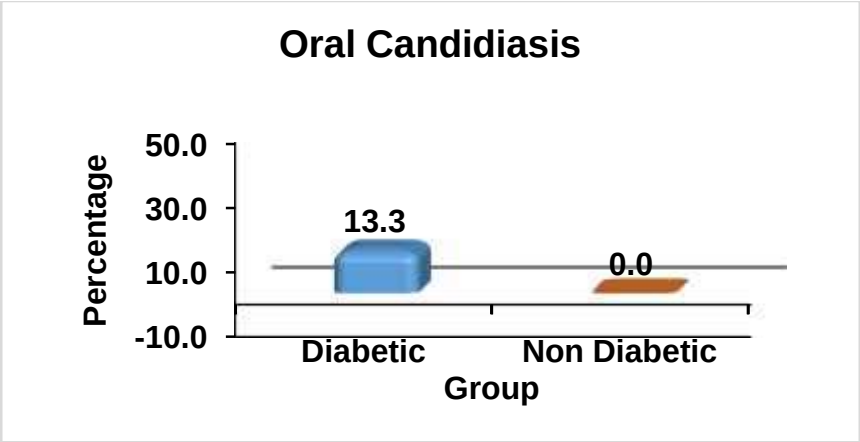


Figure 5: Oral candidiasis in diabetic and non-diabetic

13.3% of diabetic patients and 0% non diabetic subjects seems to have oral candidiasis with a statistical significance (p=0.483) (Table 10, Figure 5).

Table 11: Tooth loss in diabetic and non-diabetic

Tooth Loss	Group						P value
	Diabetic		Non Diabetic		Total		
	N	%	N	%	N	%	
Yes	8	53.3	5	33.3	13	43.3	0.269
No	7	46.7	10	66.7	17	56.7	
Total	15	100.0	15	100.0	30	100.0	

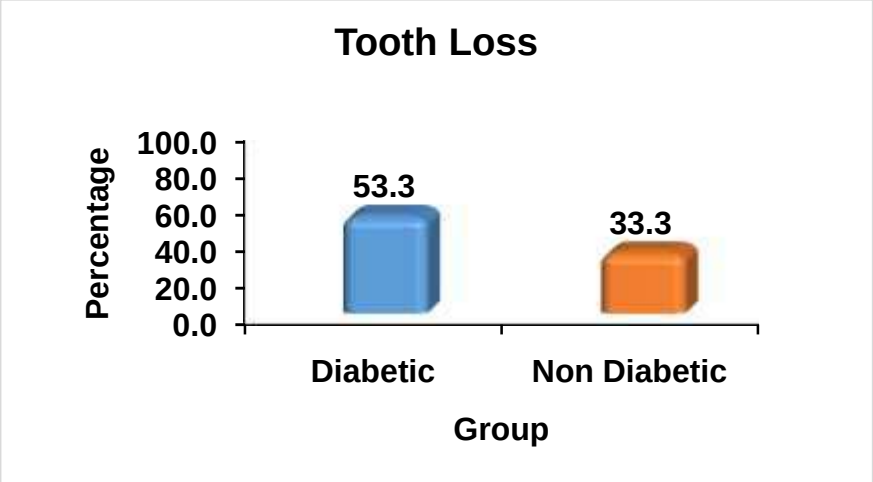


Figure 6: Tooth loss in diabetic and non-diabetic

53.3% of diabetic experienced tooth loss and 33.3% of healthy subjects shows tooth loss.(Table 11, Figure 6)

Table 12: Halitosis in diabetic and non-diabetic

Halitosis	Group						P value
	Diabetic		Non Diabetic		Total		
	N	%	N	%	N	%	
Yes	7	46.7	2	13.3	9	30.0	0.109
No	8	53.3	13	86.7	21	70.0	
Total	15	100.0	15	100.0	30	100.0	

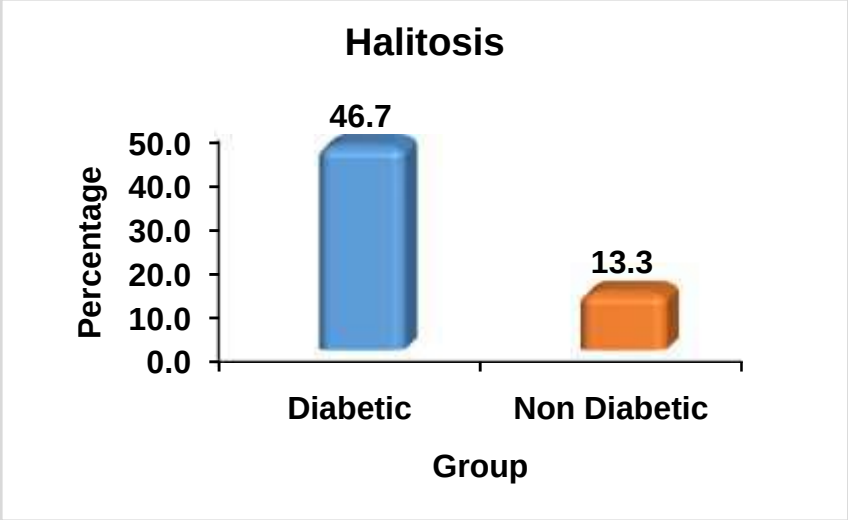


Figure 7: Halitosis in diabetic and non-diabetic

46.7% and 33.3% with statistical significance (p=0.269). halitosis is present highly in diabetic than healthy (Table 12, Figure 7)

Table 13: Dental caries in diabetic and non-diabetic

Dental Caries	Group						P value
	Diabetic		Non Diabetic		Total		
	N	%	N	%	N	%	
Yes	15	100.0	8	53.3	23	76.7	0.006
No	0	0.0	7	46.7	7	23.3	
Total	15	100.0	15	100.0	30	100.0	

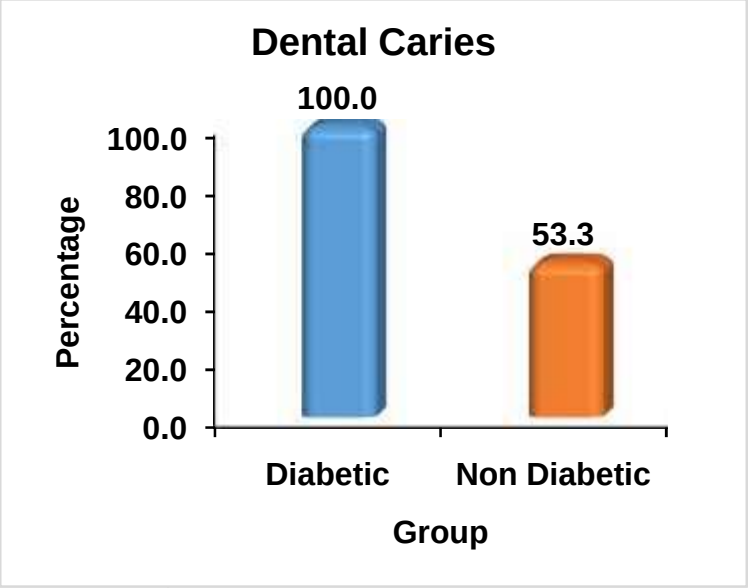


Figure 8: Dental caries in diabetic and non-diabetic

It is reported that all the diabetic subjects are subjected to dental caries where as in healthy the percentage came to half that is 53.3% (Table 13, Figure 8)

Table 14: Glossitis in diabetic and non diabetic

Glossitis	Group						P value
	Diabetic		Non Diabetic		Total		
	N	%	N	%	N	%	
Yes	5	33.3	0	0	5	16.7	0.042
No	10	66.7	15	100.0	25	83.3	
Total	15	100.0	15	100.0	30	100.0	

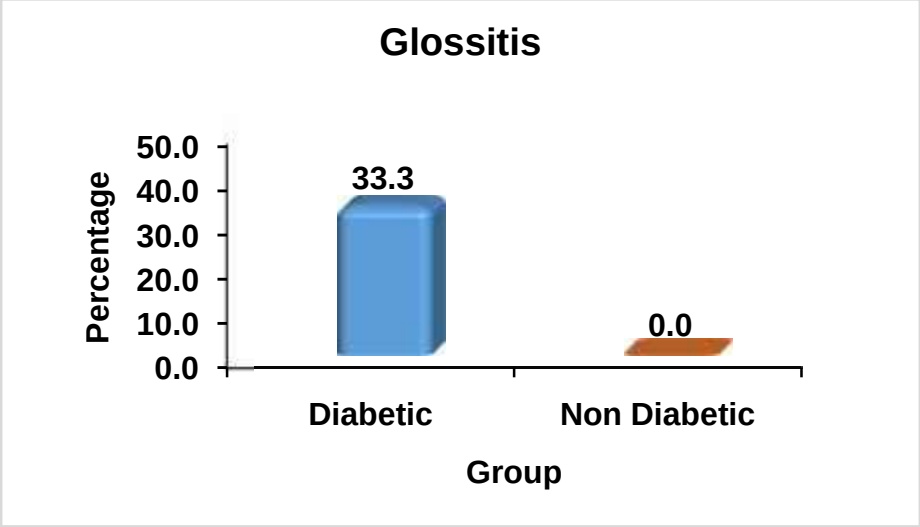


Figure 9: Glossitis in diabetic and non diabetic

33.3% of diabetic population reported to have glossitis (Table 14, Figure 9).

Table 15: Taste impairment in diabetic and non diabetic

Taste Impairment	Group						P value
	Diabetic		Non Diabetic		Total		
	N	%	N	%	N	%	
Yes	1	6.7	0	0	1	3.3	0.999
No	14	93.3	15	100.0	29	96.7	
Total	15	100.0	15	100.0	30	100.0	

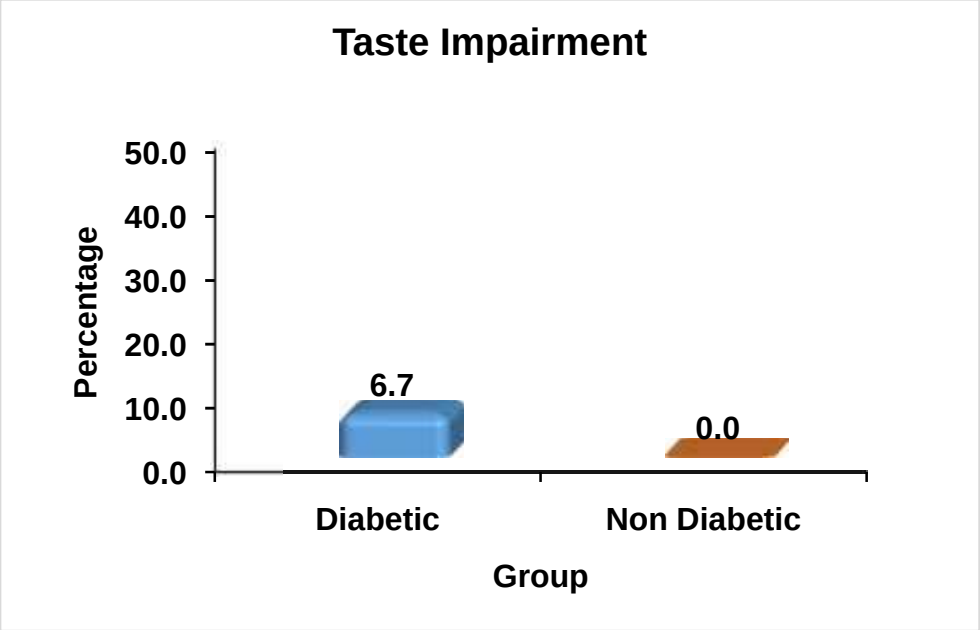


Figure 10: Taste impairment in diabetic and non-diabetic

Only 6.7% of diabetic patients experienced taste impairment and none in healthy(Table 15, Figure 10).

Table 16: Oral mucosal ulcer in diabetic and non-diabetic

Oral Mucosal Ulcer	Group					
	Diabetic		Non Diabetic		Total	
	N	%	N	%	N	%
Yes	1	6.7	2	13.3	3	10.0
No	14	93.3	13	86.7	27	90.0
Total	15	100.0	15	100.0	30	100.0

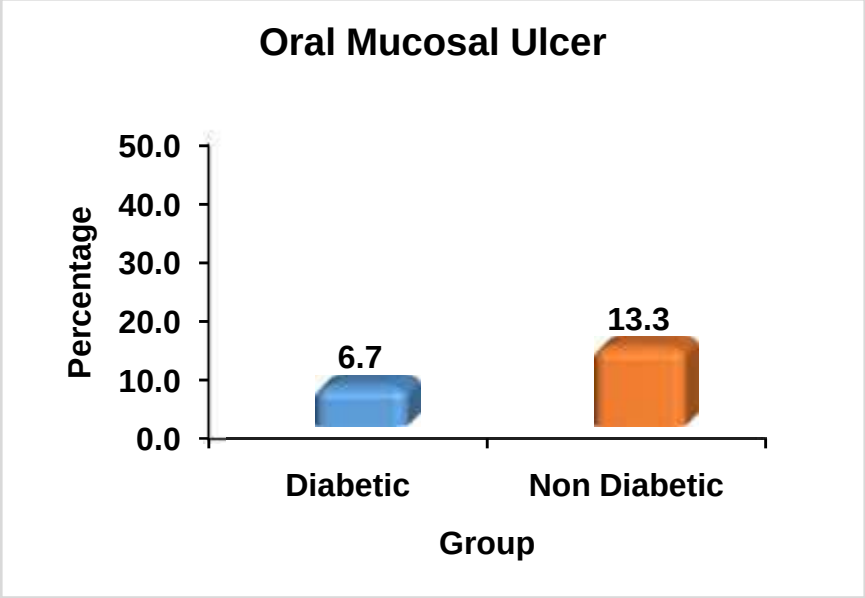


Figure 11: Oral mucosal ulcer in diabetic and non-diabetic

Surprisingly oral mucosal ulcers are more in non diabetic than diabetic that is 13.3% in non diabetic and 6.7% in diabetic (Table 16, Figure 11).

Discussion

Salivais a heterogeneous fluid consisting of proteins, glycoproteins, electrolytes, organic molecules, and also the compounds which are transported from the blood. Saliva constantly bathes the teeth and oral mucosa. saliva represents a mixture of all the secretions of both major i.e., parotid gland, submandibular gland and sublingual gland and minor salivary glands along with the gingival fluid. There are many biological factors in saliva which protects the enamel, dentin, and cementum from caries development and facilitate the remineralization.The ability of saliva to affect caries development is dependent on the quantity and composition of the secretion

In the present study it is reported that more than half of the diabetic patients are subjected to xerostomia. In the first minute the mean flow rate was surprisingly more in diabetic subjects than

that of the healthy subjects while other study outcomes were in contradiction to this finding, where as in the next four minutes the mean salivary flow was significantly more in healthy subjects than in diabetic subjects. It is observed that average mean salivary flow rate in healthy subjects are comparatively way higher than that of diabetic subjects which was consistent with other study findings(HakimehAhadian et al)[7]. The significant reduced salivary flow rate of diabetes is confirmed with the results of previous studies subjects (Kjellman 1970a: Conner et al. 1970)[8].

Diminished salivary flow rate can have deleterious effects on oral and systemic health(Navazesh et al., 1992a[9]; Atkinson and Wu, 1994[10]). Several studies reported that the unstimulated salivary flow rates in healthy individuals have found the average value which is about 0.3 ml/min. Value below 0.1ml/min are considered as hyposalivation and also values between 0.1-0.25ml/min(Tenovuo and Lagerlof, 1994)[11].It has been studied that there would not be any uniformly accepted reference values . The amount of saliva in the mouth changes within a person over time and individual (Ship et al., 1991)[12].

The affected salivary flow rate is mainly due to autonomic nerve dysfunction or microvascular changes which diminishes the ability of salivary gland to respond to hormonal stimuli[13]. Other causes are dehydration, side effects of drugs used by diabetic patients like diuretics, antihypertensive and antidepressents[14]. Lack of adequate salivary secretion leads to fungal infections like oral candidiasis and inadequate oral hygiene, discomfort in talking and swallowing food[15].

It was evident that all the diabetic subjects in this study developed dental caries which is closely associated with the hyposalivationwhich was also perceived in the study of(SadiaIqbal et al.,vol.31, no.1,june 2011)[16]. In addition to this periodontal disease was found as the mostcommon manifestation in the diabetic subjects followed by tooth loss, halitosis, glossitis, taste impairment, oral candidiasis and oral mucosal ulcer. this was in concordance with earlier literature(Sura A. A et al., Vol 3, No.2a,2015)[17] and also in other two studies(E Nandakumar et al[18]), (VaishnavisivakaliSubramanian et al[19]).

There are harmful effects of having a dry mouth, one of the study shows long term usage of biguanides drugs may decrease salivary flow rate[20].it is of extreme importance to keep saliva flowing as efficiently as possible. There are several ways to make this happen, and the most important is to increase the intake of liquids throughout the day. A dry mouth can sometimes be the first sign of dehydration, so drinking more water is the best way to fight the root cause, which increases the production of saliva, and a more provides balanced level of moisture in the mouth. Additionally, increasing the amount of fluids in the body can also help prevent ulcers and sores in the mouth and chapped lips. Severalstudies show that diabetics can fight dry mouth by drinking more liquids, but it is not a long term solution as once the body processed these fluids, the dry mouth again returns. When this happens, a more direct approach to the problem is needed. Fortunately, there are varioustreatmentto improve stimulate salivation, such as sugar-free gum, mouthwash, and mints. Some of therecent studies hasshown that drugs like Pilocarpine and Cevimeline has an effect of decreasing the sensation of oral dryness.

Conclusion

This study showed significant variation in salivary flow rates between diabetes and health emphasizing that most of the oral conditions are closely correlated to salivary secretion of the individual. This research was undertaken to increase the awareness of oral complications among diabetes and health. Of the oral manifestations screened, we found 100% positivity for dental caries followed by periodontitis. The data elicited from this study was restricted to the outpatient population visiting a medical hospital; a large scale study with screening for oral manifestations undertaken at various prominent hospitals in the metropolitan city would yield relevant epidemiological data.This study is a small step in the aforementioned direction.

Corresponding Author

Arvind. M,

Professor, Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

Email: arvindmuthukrishnan@yahoo.com,

Mobile Number: 9444303303

References

1. Vinayak V. Study of cutaneous manifestations of diabetes mellitus. Indian Journal of Dermatology. 2004 Jan 1;49(2):73-5.

2. Bellamy L, Casas JP, Hingorani AD, Williams D. Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. The Lancet. 2009 May 23;373(9677):1773-9.

3. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes care. 2004 May 1;27(5):1047-53.

4. Mokdad AH, Ford ES, Bowman BA, Nelson DE, Engelgau MM, Vinicor F, Marks JS. Diabetes trends in the US: 1990-1998. Diabetes care. 2000 Sep 1;23(9):1278-83.

5. Abegunde DO, Mathers CD, Adam T, Ortegon M, Strong K. The burden and costs of chronic diseases in low-income and middle-income countries.The Lancet. 2007 Dec 8;370(9603):1929-38.

6.Strahl RC, MAS SW, Charles F, Streckfus DD. Salivary Flow Rates: A Diagnostic Aid in Treatment Planning. Clinical preventive dentistry. 1990 Oct;12(4).

7. Ahadian H, Karbassi MH, Afkhami-Ardekani M, Haydaripoor Z, Sadrabad MJ, Kheirollahi K, Davodi A. Comparison of the unstimulated whole salivary flow rate in Diabetic Type II patients with healthy individuals. Iran J Diabetes Obesity. 2014 Jun 1;6:93-7.

8.Kjellman O. Secretion rate and buffering action of whole mixed saliva in subjects with insulin-treated diabetes mellitus. Odontologiskrevy. 1970;21(2):159-68.

9. Navazesh M, Christensen C, Brightman V. Clinical criteria for the diagnosis of salivary gland hypofunction. Journal of dental research. 1992 Jul;71(7):1363-9.

10. Atkinson JC, Wu AJ. Salivary gland dysfunction: causes, symptoms, treatment. Journal of the American Dental Association (1939). 1994 Apr;125(4):409-16.

11. Lagerlöf F. Caries-protective factors in saliva. Advances in dental research. 1994 Jul;8(2):229-38.

12. Ship JA, Fox PC, Baum BJ. Normal salivary gland function: how much saliva is enough. J Am Dent Assoc. 1991;122:63-9.

13. Anderson L. Journal of the American Dental Association, Frontal Oral Biology;10:1200–221. 1998.

14. Moore PA, Guggenheimer J, Etzel KR, Weyant RJ, Orchard T. Type 1 diabetes mellitus, xerostomia, and salivary flow rates. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics. 2001 Sep 1;92(3):281-91.

15.Sreebny LM, Vissink A, editors. Dry mouth, the malevolent symptom: a clinical guide. John Wiley & Sons; 2010 Feb 25.

16.SadiaIqbal, FarhatKazmi, SaadAsad, Muhammad Mumtaz, Ayyaz Ali Khan. Dental caries and diabetes mellitus. Pakistan Oral and Dental Journal Vol 31, No.1 (June 2011).

17. Sura AA, Ibrahim SA, Prathibhaprasad JM. Frequency and Pattern of Oral Manifestations and Oral Hygiene Practices among Cases with Diabetes Mellitus in Ajman, United Arab Emirates.International Journal. 2015;3(2A):4-9.

18.ENandakumar. Dental Manifestations in Diabetc and Non Diabetic patients: a review, Journal of pharmaceutical sciences and research. Vol. 7(7), 2015,482-484

19. VaishnavisivakaliSubramanian,Preetha,. Impact of diabetes mellitus type 2 on oral health among out patient department of saveetha dental college and hospital. International journal of science and research. Vol 5 issue, August 2016.

20. S SubashreeRathi, V Suresh, Evaluation of salivary flow rate in diabetic patients on sulfonylurea drugs and biguanidedrugs.Journal of Pharmtrch Research. Vol.8, no 9, pp 118-124,2015.

Demonstration of Spirochetes in Patients with Orthodontic Appliances - A Preliminary Study

K.Hemani[1], N. P. Muralidharan[2], Dhanalakshmi Ravikumar[3],
Graduate Student[1],Reader[2],Department of Microbiology,Senior Lecturer[3], Department of Pedodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: Orthodontic appliances are meant for the correction of malocclusions. Placement of such appliances will necessitate a protracted period of 2 - 3 years wear. Neglect in maintenance of good oral hygiene may result in compromised periodontal health. Though many organisms in the mouth are found increased in their proportion during orthodontics, organisms like spirochetes, that grow well with simple source of nutrients, will flourishtoa larger extent.This preliminary study was undertaken to evaluate the prevalence of spirochete population in patients wearing orthodontic appliances by assessing the spirochetes load at different time intervals of treatment.

Reason: Spirochete is present in very small proportions, even in normal health mouth. In patients with poor oral hygiene and halitosis,thespirochetes has been demonstratedand found increased in its proportion, this is capable of deteriorating the oral health. Elimination of these organisms is a challenge.Studies have also revealed increased spirochete proportions in plaque samples from Orthodontic patients with poor oral hygiene.This study was conducted to analyse the time dependant changes in population of spirochetes in patients wearing orthodontic appliances. This will help to bring about awareness to the society about the importance of oral hygiene in orthodontic patients at all ages.

Materials and Methods: Inclusion criteria included a total of 10 participantsin the age of 20 - 35 years, who wererandomly selected,on the basis of willingness to participate in the study. Patients irregular for treatment and those with other systemic complications and periodontally compromised prior to commencement of Orthodontics were excluded from the study. Plaque

samples were collected from the participants at the 3rd, 6th or 9th month of treatment as T1, T2 and T3, respectively.

Result:The mean percentage value of spirochetes of the group with patients having the intra oral appliances for 3 months[T1] was 35.50%±25.544, T2 with appliance duration 6 months was 37.00%± 18.439and T3for 9 months was 57.50 % ± 22.267.

Conclusion: The study confirmedthat there is a statistically significant difference between spirochetes count found between 6 months and 9 months duration.[p =.023] On average, spirochetes count in 9 months was about 20.5% higher than spirochetes count in 6 months. It is concluded that, the increase in the duration of wear of the orthodontic appliance from 6 to 9 months proportionately increased the spirochete population from 37.00% to
57 .50% respectively.

Key Words: *appliances, plaque, microorganisms, orthodontics, spirochetes*

Introduction

Orthodontics is the science of dentistry that deals with the correction of malocclusion. The desired tooth movement is achieved by means of an orthodontic appliance that generates a force to bring about tooth movement for the correctionofmalocclusion.The treatment spans a duration of two to three years.The various mechanical components that constitute an orthodontic applianceincludesbrackets,buccaltubes,archwires, ligations made of stainless steel, nickel titanium, brackets, wires, springs and elastomeric components.This complex appliancesetup, predisposes to entrapment of food debris, and poses difficulty in cleaning,jeopardising the oral hygiene.Neglect of adequate oral health measures, can result in accumulation of plaque and colonisation with periodontopathicbacteriaresulting in gingival inflammation and bleeding [1] among orthodontic patients [2,3,4].

Compromised oral hygiene predisposes to white spot lesions,caries and periodontal diseases [5], and exposes to systemic complications.[6-10].Orthodontics was earlier done mostly for young children is now needed for adults too. The long duration of orthodontic appliance wear therefore, is likely to compromise on host response predisposing to periodontal diseaseamongorthodonticpatients.Studies using DNA probes have proven a close relationship betweenperiodontal disease and invasion of streptococci, lactobacilli and spirochetes in the subgingival plaque [11,12]

This increase in bacterial count in the plaque and tongue surface sometimes results in halitosis. [13,14,15]. These factors additionally can result in gingivitis, white spot lesion and caries when hygiene is neglected , contributing to complication of this treatment[16].

There are various organisms that colonise subgingivally in oral cavity like spirochaetes and fuseiform bacilli [17] .The streptococcus mutans and lactobacillus [18] are more responsible for caries and treponema are responsible for periodontal breakdown and halitosis during orthodontics. These microbes grow exclusively in conditions of poor hygiene. An orthodontic appliance affects the subgingival microbial composition, thereby increasing the prevalence of periopathogens

A preliminary study was thereforeundertaken to assess anaerobicspirochete levels in orthodontic patients presenting with normal oral health at different time intervals .The timedassessment of the spirochete count was conducted, on the third , sixthand ninth month of treatment .

Materials and Methods

This study was conducted to evaluate, the prevalence of oral treponema in patients having orthodontic appliances and presenting with normal oral health. The study design, a prospective longitudinal study, approved by the Institutional Ethical Committee, was conducted in patients attending the outpatient clinic of the Department of Orthodontics, Saveetha Dental College. The participants were under Orthodontic treatment with MBT .022 slot prescription, Abzil 3M conventional appliance.

Inclusion criteria included a total of 10 participantsin the age of 20 - 35 years, who wererandomly selected,on the basis of willingness to participate in the study. Patients irregular for treatment and those with other systemic complications andperiodontally compromised prior to commencement of Orthodontics were excluded from the study. Plaque samples were collected from the participants at the 3rd, 6th or 9th month of treatment as T1, T2 and T3, respectively

Sample collection

Participants were detailed about the study and the reason of this study. An informed consent was obtained. Soft plaque samples were collected under strict aseptic procedurewith sterile tooth pick from the tooth surfaces at 25, 26, 27, 35, 36 37 region. The plaque samples collected were immediately smeared on a clean glass slide and dried. These slides with smear were labelled and were rolled in a tissue paper and transported for microbial examination.

Microscopic Method

The smears were stained with simple staining method[19,20,21] with dilute carbolfuchsin for two minutes and washed in clean drinking water. The smears were then dried and examined under oil immersion objective of the microscope. In each smear more than 100 fields were examined.Each field was divided into a quarter and the investigator quantified the organisms based on their shape.

The study examined the total microbial elements present on the smear per field, and, the relative proportion of spirochetes were taken into consideration. A cumulative index of approximately 100 fields was figured out as percentage in relation to the other bacterial structures.Thepercentage of proportion of spirochetes was tabulated among each group separately.

Statistical Analysis

The relative proportion of spirochetes in most of the fields examined were noted and tabulated. Statistical analyses were performed including means and standard errors of the mean for each group. A value of $P<0.05$ was considered significant.

Result

Table 1:Year percentage of spirochaete count:

T1 3 months	T2 6 months	T3 9 months
10%	75%	75%
50%	20%	20%
25%	25%	75%
30%	30%	20%
20%	50%	50%
50%	30%	75%
75%	20%	50%
75%	50%	60%
10%	20%	75%
10%	50%	75%
Mean-35.50%	Mean- 37.00%	Mean- 57.50%

Table 2: Paired Samples Correlations of spirochaete count

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	T1	35.50	10	25.544	8.078
	T2	37.00	10	18.439	5.831
Pair 2	T2	37.00	10	18.439	5.831
	T3	57.50	10	22.267	7.042
Pair 3	T1	35.50	10	25.544	8.078
	T3	57.50	10	22.267	7.042

		N	Correlation	Sig.
Pair 1	T1 & T2	10	-.333	.348
Pair 2	T2 & T3	10	.332	.349
Pair 3	T1 & T3	10	-.330	.352

Table 3:Paired Samples Test of spirochaete count

		Paired Differences					t	Df	Sig. [2-tailed]
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference Lower	Upper			
Pair 1	T1 & T2	-1.500	36.136	11.427	-27.350	24.350	-.131	9	.898
Pair 2	T2 & T3	-20.500	23.740	7.507	-37.483	-3.517	-2.731	9	.023*
Pair 3	T1 & T3	-22.000	39.030	12.342	-49.920	5.920	-1.782	9	.108

T2 and T3 difference is significant and the t value is 2.731 and p value is 0.023

T2 & T3 – insignificant

T1 & T3 – insignificant

The mean percentage value of spirochetes of the group with patients having the intra oral appliances for 3 months[T1] was 35.50%±25.544, T2 with appliance duration 6 months was

37.00%± 18.439and T3for 9 months was 57.50 % ± 22.267. There is an increase in the proportion of the organism with increased duration of intra oral orthodontic appliance wear,more between 6 and 9 months than between 3 and 6 months.

From the paired samples t values it is inferred that there is a statistically significant difference between spirochetes count found between 6 months and 9 months duration.[p =.023] On average, spirochetes count in 9 months was about 20.5% higher than spirochetes count in 6 months. It is concluded that that, increase in the duration of wear of the orthodontic appliance from 6 to 9 months proportionately increased the spirochete population from 37.00% to
57.50%respectively.

Discussion

Severe crowding predisposes to poor oral health, attracting an increase in plaque deposition which can cause inflammation and gingivitis [22] that eventually leads to periodontitis and even bone loss.Orthodonticappliancespredisposes to periodontitis when hygiene is neglected.

The present study proved that increase in duration of treatment increases the population of spirochetes, in cases with neglected oral hygiene[Table 1]. The increase is statistically significant between 6 and 9 months. Statistical insignificance of the increase in spirochete population between T1 and T3 is probably because of the wide standard deviation, which is a reflection of the variation seen in the samples collected. The variations wherein, the spirochete count reduced in the 6th and 9th months in a few cases, was because some participants understood the implications of oral hygiene explained to them prior to their informed consent, and resorted to strict regimes of hygiene. The count was reduced in those participants after the initial sample.

Other studies have also recordedgradual increase in spirochetes population with increase in duration of the treatment, in patients who showed no improvement in oral hygiene[23]. This increase in spirochete population may be due to the fact that oral treponema are not obligate anaerobes and grow on simple source of nutrition.

BrekkEckley, 2012 [24]concludedthat plaque score and probing depths increased with successive orthodontic visits. This was attributed to an increase in the periodontopathogens, P gingivalis, T denticola, and T forsythia, particularly when hygiene was compromised . Additionally dark field microscopy in their study, confirmed an increase in the small and

large spirochetes population, non-motile rods, filaments and fusiforms, with a concomitant decrease inall coccoid forms and motile rods. The microbial profile contributes to the aggravated gingival inflammation, bleeding and increase in periodontal pocket depth in patients with compromised oral health.

The rough surface of the metallic component, the high surface tension of the various polymeric materials used and the complicated design of the appliance creates an environment for food stagnation and thereby biohostability of the appliance. Therefore an ideal appliance should be a poor biohost that will neither allow active growth of organism nor passively provide a media for multiplication of microorganisms which may be either chromogenic or that induces halitosis or interferes with the mechanical properties of the wires which could jeopardise orthodontic tooth movement [25].

The finding that the plaque score, probing depths returned to normalin thirty days post removal of the orthodontic appliance, reiterates the influence of these organisms during orthodontic therapy [24]. In spite of brushing twice a day patients under orthodontic treatment both adults and young children, are prone to poor oral hygiene [26], as mechanical restraints like bands, brackets and wires used in orthodontic appliances hinder cleaning of the debri and cause accumulation of plaque and microbial flora.

To prevent the plaque build up in orthodontic treatment the patient must be properly educated about the various techniques, and oral hygiene methods like brushing, flossing and rinsing [27,28,29], and the use of various prophylactic tooth pastes and mouth rinses.[30,31].

The present study therefore, highlights that maintenance of oral hygiene, is of paramount importance during orthodontic treatment, a very important message to be sent out to the society, particularly the aspirants of aesthetic correction of their malocclusion. Spirochetes classified as periodontopathogens are normal commensals found in small proportion in the oral cavity. The present study confirmed that with neglect in oral hygiene their proportions can increase, which is prone to pathogenecity.

Further studies, are required with greater sample size, to isolate the most prevalent pathogenic strains, to enable specific measures of their prevention in an Orthodontic patient.

Conclusion

There is an increase in the spirochete number,with increase in the duration of intra oral appliance.The mean percentage value of spirochetes at T1 at 3 months with orthodontic appliance was 35.50%, at T2 with appliance duration 6 months was 37.00%, and at T3 with

appliance for9months was 57.50%. Increase in spirochete count from 6 to 9 months showed a statistical significance.Neglect in oral hygiene during Orthodontics,can predispose to increase in spirochaete countin plaque samples, as the duration of treatment increases

Corresponding Author

Dhanalakshmi Ravikumar,

Senior Lecturer,Department of Pedodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Science,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai, 600077, Tamil Nadu, India.

Email Id: dhana9677@gmail.com

Telephone Number: 9597011442

References

1. Naranjo AA, Trivino ML, Jaramillo A, Betancourth M, Botero JE. Changes in the subgingival microbiota and periodontal parameters before and 3 months after bracket placement. Am J OrthodDentofacialOrthop. 2006;130:17–22.

2. Zachrisson, S. and B. Zachrisson. Gingival condition associated with orthodontic treatment. Angle Orthod 1972. 42:26–34.
3. Alexander, S. A. Effects of orthodontic attachments on the gingival health of permanent second molars. Am J OrthodDentofacialOrthop 1991. 100:337–340.
4. Zachrisson, B. U. Cause and prevention of injuries to teeth and supporting structures during orthodontic treatment. Am J Orthod 1976. 69:285–300.
5. Brusca MI, Chara O, Sterin-Borda L, Rosa AC. Influence of differentorthodontic brackets on adherence of microorganisms in vitro.Angle Orthod 2007;77:331-6.

6. Li X, Kollveit KM, Tronstad L, Olsen I. Systemic diseases caused byoral infection. ClinMicrobiol Rev 2000;13:547-58.
7. Marques da Silva R, Caugant DA, Lingaas PS, Geiran O, TronstadL,Olsen I.

Detection of Actinobacillusactinomycetemcomitansbutnot bacteria of the red complex in aortic aneurysms by multiplexpolymerase chain reaction. J Periodontol 2005;76:590-4.

8. Gurenlian JR. Inflammation: the relationship between oral healthand systemic disease. Dent Assist 2009;78:8-10,12-4,38-40.
9. Higashi Y, Goto C, Hidaka T, Oral infection-inflammatorypathway, periodontitis, is a risk factor for endothelial dysfunctionin patients with coronary artery disease. Atherosclerosis2009;206:604-10.
10. Lucas VS, Omar J, Vieira A, Roberts GJ. The relationship betweenodontogenicbacteremia and orthodontic treatment procedures.Eur J Orthod 2002;24:293-301.
11. Anhoury P, Nathanson D, Hughes CV, Socransky S, Feres M, ChouLL. Microbial profile on metallic and ceramic bracket materials.Angle Orthod 2002;72:338-43.
12. Hirsch DI, Kulbersh R, Kaczynski R. Assessment of pretreatmentorthodontic patients using the BANA test. N-benzoyl-DLarginine-naphthylamide. Am J OrthodDentofacialOrthop1997;112:154-8.
13. Lara-Carrillo E, Montiel-Bastida NM, Sánchez-Pérez L, Alanís-Tavira J. Effect of orthodontic treatment on saliva, plaque and the levels of Streptococcus mutans and Lactobacillus. Med Oral Patol Oral Cir Bucal. 2010;15:924–929.
14. Scheie AA, Arneberg P, Krosgstad AO. Effect of orthodontic treatment on prevalence of Streptococcus mutans in plaque and saliva. Scand J Dent Res. 1984;92:211–217.
15. Ashwin. K.S, Karthikeyan. M, Nirmalkumar. R, Rajanivetha, Priyadarshini. R. Halitosis: a Short Review. Research J. Pharm. and Tech. 7[12]: Dec. 2014; 1494-1497.
16. Ay ZY, Sayin MO, Ozat Y, Goster T, Atilla AO, Bozkurt FY. Appropriate oral hygiene motivation method for patients with fixed appliances. Angle Orthod. 2007;77[6]:1085-9.
17. Paolantonio M, Pedrazzoli V, diMurro C, diPlacido G, Picciani C, Catamo G, De Luca M, Piaccolomini R. Clinical significanceofActinobacillusactinomycetemcomitansin young individuals during orthodontic treatment. A 3-year longitudinal study. J ClinPeriodontol. 1997;24[9Pt1]:610-7.

18. Bloom RH, Brown LR Jr. A study of the effects of orthodontic appliances on oral microbial flora. Oral Surg Oral Med Oral Pathol. 1964; 17:658-67.
19. DeLamater, E. D., Haanes, M. and Wiggall, R. H. , Studies on the life cycles of spirochaetes. II. The development of a new stain. Amer. Jour. Syphilis, Gonorrhea and Venereal Dis. 34, 515, 1950.
20. Beamer, P. R. and Firmipger, H. I., Improved methods for demonstrating acidfast and spirochetal organisms in histologic sections. Lab. Invest. 4, 9, 1955.
21. Levine, B. S., Staining Treponemapallidum and other treponemata. Publ. Health Repts. 67, 253, 1952.
22. Löe H, Theilade E, Jensen SB. Experimental gingivitis in man. J Periodontol. 1965; 36: 177-87.
23. Chauhan P, Dua VS, Kainth N, Tosh A, Tomar A. The effect of various oral hygiene products on the microbial flora in patients undergoing orthodontic treatment. APOS Trends Orthod. 2015;5[2]:63-9.
24. Brett Eckley , John Thomas, Richard Crout, Peter Ngan, Periodontal and microbiological status of patients undergoing orthodontic therapy, Hong Kong Dent J 2012;9:11-20
25. Kusy, R. P. A review of contemporary archwires: their properties and characteristics. Angle Orthod 1997. 3:197–207.
26. Huser MC, Baehni PC, Lang R. Effects of orthodontic bands on microbiologic and clinical parameters. Am J OrthodDentofacialOrthop 1990;97:213-8.
27. Sinclair M, Berry CW, Bennet CL, Israelson H. Changes in gingival and gingival flora with bonding and banding. Angle Orthod. 1987;57[4]:271-8.
28. Rao, D., Arvanitidou, E., Du-Thumm, L., and Rickard, A.H. [2011] Efficacy of an alcohol-free CPC-containing mouthwash against oral multispecies biofilms. J Clin Dent 22: 187–194.
29. Shahana R.Y, Muralidharan .N.P. Efficacy of Mouth Rinse in Maintaining Oral Health of Patients Attending Orthodontic Clinics. Research J. Pharm. and Tech 2016; 9[11]: 1991-1993.
30. Alada A, Ige A. Efficacy of different brands of mouth rinses on oral bacterial load count in healthy adults. African Journal of Biomedical Research 2004;7:125-12.

31. Charles CH, Sharma NC, Galustians HJ, Qaqish J, McGuire JA, Vincent JW. Comparative efficacy of an antiseptic mouthrinse and an antiplaque/antigingivitis dentifrice. Asix-month clinicaltrial. J Am Dent Assoc2001;132:670–5.

Detection of EFFA (endocarditisassociated antigen) gene from Enterococcus Faecalis Isolated from Patients with Endodontic Infections

KalaivaniNatarajan[1], R.V.Geetha[2], S.Sangeetha [3],
Graduate Student[1], Reader[2], Department of Microbiology
Reader[3], Department of Prosthodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: Enterococci are Gram-positive facultative anaerobic bacteria which are normal commensals in the gastrointestinal tract, oral cavity, vagina etc [1].They are organisms of low virulence, but are known to cause various clinical infections that includes septicaemia, endocarditis, urinary tract infections, wound infections, and meningitis. Enterococci now rank among the top three-nosocomial bacterial pathogens, and strains resistant to currently available antibiotics, which pose real therapeutic difficulties [2] .Up to 90% of enterococcal infections in humans, are caused by Enterococcus faecalis

Aim and Objective: To detect the efaA (Endocarditis associated antigen) gene from Enterococcus faecalis isolated from patients with endodontic infections.

Materials and methods: A total of 20 isolates of Enterococcus faecalis were collected from patients undergoing endodontic infections. The samples were inoculated in Mac Conkey agar Brain infusion agar. Presumptive identification of Enterococcus was done by Gram's staining, Catalase test and heat tolerance test. DNA extraction was done. The amplificationof virulence genes was carried out. DNA analysis was done using PCR amplification.

Results: EfaA gene was detected in 3 out of 20 isolates.

Conclusion:E.faecalis though a commencal bacteria in Genito urinary tract it is gaining importance as in nosocomial pathogen and in endodontic infections. Within the limitations of this study it can be concluded that efaA, a potent *E. faecalis*virulence gene can be found in strains obtained endodontic infections.

Key Words: *Virulence factors, DNA extraction, Bacteraemia, Septicaemia, endocarditis*

Introduction

Enterococci are Gram-positive facultative anaerobic bacteria which are normal commensals in the gastrointestinal tract, oral cavity, vagina etc [1].They are organisms of low virulence, but are known to cause various clinical infections that includes septicaemia, endocarditis, urinary tract infections, wound infections, and meningitis. Enterococci now rank among the top three-nosocomial bacterial pathogens, and strains resistant to currently available antibiotics, which pose real therapeutic difficulties [2] .Up to 90% of enterococcal infections in humans, are caused by Enterococcus faecalis [3].

In the past few years, *Enterococcus faecalis* has been mentioned with increased frequency with regard to teeth with asymptomatic persistent endodontic infections, predominantly in therapy-resistant endodontic infection [4]. *E. faecalis* have been able to form biofilms in root canals, and this ability can be important for bacterial resistance and persistence after endodontic procedures [5]. There are studies that suggest that Enterococcus faecalis has the greatest potential to invade dentinal tubules and remain viable in themon comparison with S.mutans and S.gordonii which gives it the ability to cause chronic periapical disease and failure of endodontic treatment [5].

Besides this Enterococcus, faecalis have several virulence factors that promote a broad spectrum of events that allows disease development and progression. The infection was initiated as the bacteria binds to the ligands that are present at the site of infection after which the colonization can be accelerated through antibacterial attachment. They also modify the host's immune response. One important virulence factor is efaA [6].

E. faecalis endocarditis antigen (efaA) was first identified from the antiserum of a patient with *E. faecalis* endocarditis. The amino acid sequence of the associated protein efaA revealed 55–60% homology to a group of streptococci proteins known as adhesins. Hemolysin, gelatinase, ace, and ESP also function as putative virulence factors of endocarditis-causing *E. faecalis*. *E. faecalis* strains derived from different sources such as endocarditis, urinary tract infections, and even endodontic infections have been shown to possess distinct patterns of 'virulence factors' [7].Hence the aim of our study was to detect the presence of putative *E. faecalis* virulence factor efaA in root canals of therapy-resistant endodontic infections using the PCR method.

Materials and method

A total of 20 isolates of Enterococcus faecalis were collected from patients undergoing endodontic treatment. The samples were inoculated in Mac Conkey agar and Brain infusion agar. Presumptive identification of Enterococcus was done by Gram's stain, Catalase test and heat tolerance test. On Gram's staining Enterococcus are Gram positive cocci arranged in pairs, on catalyst test they are Catalase negative and in heat tolerance test that tolerate the temperature of 60 degrees C for 30 minutes. On mac Conkey agar they showed small lactose fermenting colonies.

DNA Extraction

Genomic DNA used as template for polymerase chain reaction (PCR) amplification was prepared using conventional phenol-chloroform DNA extraction method.

Amplification

The amplification of virulence genes was carried out as follows: Initial denaturation at 95°C for 15 min followed by denaturation at 94°C for 1 min, annealing at 56°C for 1 min, and extension at 72°C for 1 min.

The Polymerase Chain Reaction amplification of the efaA gene was carried out as follows: Predenaturation at 95°C for 4 min followed by denaturation at 95°C for 30 cycles of 30 s each; 1 min for annealing at 52°C and elongation at 72°C for 1 min. Both positive control and negative control, consisting solely of the PCR reaction mixture without DNA template were included to check the validity of the technique utilized.

DNA Analysis

Twenty-five microliters of respective amplified products were loaded into the wells and electrophoresed at a constant current of 50V for about 45 min using 1.5% agarose gel. A 100 bp DNA ladder marker was included as the standard molecular weight marker. The electrophoresed gel was later subjected to ethidium bromide staining and photographed under UV transillumination

Result

EfaA gene was detected in 3 out of 20 isolates.

Table 1: shows the primer sequence of efaA gene

Gene	Primer sequence	Product size
efaA	EFA 1 GACAGACCCTCACGAATA EFA 2 AGTTCATCATGCTGTAGTA	705

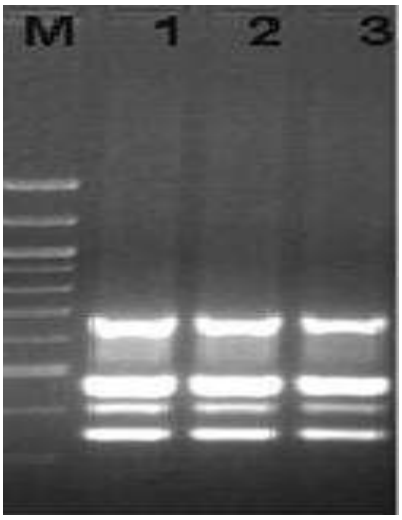


Figure 1: Shows the PCR amplification of efaA gene

Discussion

Enterococci are gram positive cocci that can occur singly, in pairs or as short chains. They are facultative anaerobes, possessing the ability to grow in the presence or absence of oxygen. Enterococci can withstand harsh environmental conditions [8]. Enterococci can grow at 10 degree Celsius and 45 degree Celsius, at pH 9.6, in 65% Nacl broth and survive at 60 degree Celsius for 30 minutes. E faecalis can adapt to adverse conditions [9].

There are currently 23 enterococci species and they are divided into five groups based on their interaction with mannitol, sorbose and arginine.*E. faecalis* can survive extreme

challenges. Its pathogenicity ranges from life-threatening diseases in compromised individuals such as bacteraemia,septicaemia, endocarditis, and urinary tract infections to less severe conditions, such as infection of obturated root canals with chronic apical periodontitis.The persistence of E.faecalis might be due to the virulence factors ace and esp.Virulence factors of *E. faecalis* enable adherence to host tissue, invasion and modulation of host inflammatory response, and secretion of various products which enhance biofilm formation [10].

Serum plays an important role in the invasion. The expression of virulence factor efaA is expressed when E.faecalis is grown in a medium that contains serum [11].The extra cellular matrix of our tissue consists of glycoproteins like collagen, laminin, fibronectin and proteoglycans.These can be exploited by micro-organisms for colonization and initiation.The ability of a microorganism to adhere to collagen play an important role in the pathogenesis of endocarditis [12]. The dentinal tissue and the heart tissues share common proteins, it is believed that efaA should be facilitated through bacterial adhesion to collagen and extracellular matrix relevant in endodontic infections [13].

About 3 out of 20 (almost 15%) isolates were detected with efaA gene. E faecalis have been able to form biofilms in root canals and this ability can be important for bacterial resistance and persistence after endodontic procedures.

A similar recent molecular-based study by Randa Salah indicated that virulence determinants endocarditis antigen (efaA) genes and ace genes has been found in *E. faecalis* isolates of oral rinse samples of patients suffering from dental diseases[14].

In a study conducted by Creti *et al*. showed that E.faecalis strains derived from different sources such as endocarditis, urinary tract infections possessed distinct patterns of virulence factors ace, efaA, and gel genes. These were found to be the most common virulence factors.

Thomas Preethee conducted a study in which 32 contaminated root canal samples were analysed in which 15 of them were positive for E.faecalis. Out of the 15 positive samples, efaA gene was identified in 11 samples (almost 73%) [15]. Bittencourt de Marques observed that the efaA gene is frequently associated with ESP or eep (enhanced expression of pheromone) and to a lesser extent with virulence markers aggA (aggregation substance gene) [16].

Although molecular based identification methods are designed to detect molecular DNA, a limitation of this method is that it cannot distinguish between DNA from viable and dead cells and it’s unclear whether the results from PCR methods represent the living flora or rather a record of an organism that have entered but not survived in the canal [17 -20].

Conclusion

E.faecalis though a commencal bacteria in Genito urinary tract it is gaining importance as in nosocomial pathogen and in endodontic infections. Within the limitations of this study it can be concludedthat efaA, a potent *E. faecalis*virulence gene can be found in strains obtained endodontic infections. Hence this virulence gene can be correlated with the pathogenicity of E.faecalis.Further studies should aim at obtaining blood samples from patients immediately after endodontic retreatment procedure to analyse the role of the efaA gene in causing bacteremia.

Corresponding Author

S.Sangeetha,

Reader, Department of Prosthodontics,

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences.

162 Poonamallee High Road, Chennai 600077Tamil Nadu, India

Email ID:dr.sangeethakiruba@gmail.com

Telephone Number: 9894067220

References

1. Jett BD, Huycke MM, Gilmore Ms (1994); Virulence of enterococci. Clinical Microbiological reviews.
2. Guven Kayaogolu, Dag Orstavik. Virulence factors of Enterococcus faecalis: a relationship to endodontic disease to endodontic disease. Crit.Rev.Oral.Biol.Med.2004;15(5):308-320
3. Suchitra U,Kundabala.M, Enterococcus faecalis : An endodontic pathogen
4. Love RM. Enterococcus faecalis--a mechanism for its role in endodontic failure. Int Endod J. 2001; 34:399–405.
5. Arias-Moliz MT, Ferrer-Luque CM, Espigares-García M, Baca P. Enterococcus faecalis biofilms eradication by root canal irrigants. J Endod. 2009; 35:711–four.
6. Virulence factors associated with enterococcus faecalis infective endocarditis: A mini review.

7. Sedgley CM, Molander A, Flannagan SE, Nagel AC, Appelbe OK, Clewell DB, et al. Virulence, phenotype and genotype characteristics of endodontic enterococcus spp. Oral Microbiol Immunol. 2005;20:10–9
8. Charles H. Stuart, DDS, Scott A. Schwartz, DDS, Thomas J. Beeson, DDS, and Christopher B. Owatz, DMD, Enterococcus faecalis: Its Role in Root Canal Treatment Failure and Current Concepts in Retreatment.
9. Flahaut S, HartkeA, Guard JC, Beachgoer A, Aboutbones P, Auffray Y (1996 a). Relationship between stress response towards ble salts, acids and heat treatment in Enterococcus faecalis. FEMS Microbiol Lett 138:49-54
10. Kayaoglu G, Ørstavik D. Virulence factors of enterococcus faecalis: Relationship to endodontic disease. Critical Rev Oral Biol Med. 2004; 15:308–20.
11. Lowe AM, Lambert PA, Smith AW. Cloning of an Enterococcus faecalis endocarditis antigen: Homology with adhesins from some oral streptococci. Infect Immun. 1995; 63: 703 - 6.
12. . Creti R, Imperi M, Bertuccini L, Fabretti F, Orefici G, Di Rosa R, et al. Survey for virulence determinants among enterococcus faecalis isolated from different sources. J Med Microbiol. 2004;53:13–20
13. Stuart CH, Schwartz SA, Beeson TJ, Owatz CB, Owatz Enterococcus faecalis: Its role in root canal treatment failure and current concepts in retreatment. J Endod. 2006;32:93 - 8
14. Randa Salah, Najla Dar-Odeh, Osama, Osam Abu Hammad, Shahabi Asem A. Prevalence of putative virulence factors and antimicrobial susceptibility of enterococcus faecalis isolates from patients with dental diseases. Biomedical Oral Health. 2008;8:1 - 7
15. Thomas Preethee, Deivanayagam Kandaswamy, and Rosaline Hannah, Molecular identification of an *Enterococcus faecalis* endocarditis antigen efaA in root canals of therapy-resistant endodontic infections, Journal of conservative dentistry.2012 Oct-Dec.
16. De Marques E Bittencourt, Suzart S, Occurence of virulence - associated genes in clinical Enterococcus faecalis isolated in Londrina, Brazil. F Medical Microbiology 2004; 53: 1069-73
17. Siqueria JF, Jr.Rocas IN. Exploiting molecular methods to explore endodontic infections. Part – 1 current molecular technologies for microbiological diagnosis. J Endod – 2005; 53; 13 – 20.

18. Sathorn C, Parashos P, Messer HH. How useful is root culture in predicting treatment outcome. J Endod. 2007; 33; 220 -5
19. RF Marickar, RV Geetha, P Neelakantan, Efficacy of Contemporary and Novel Intracanal Medicaments against Enterococcus faecalis. Journal of Paediatric Dentistry, Sept-2014
20. R.V. Geetha, Vishnu Priya Veeraraghavan,Evaluation of Anti-bacterial activity of Five root canal sealants against Enterococcus faecalis- An in vitro study, Int. J. Pharm. Sci. Rev. Res- 40(2), Sept – Oct 2016; Article No: 41.

Detection of PvlGene for the Presence of Leukocidin among Clinical Isolates of Staphylococcus Aureus

K. Pavithra[1], Gopinath[2], M S Nivedhitha[3],

Graduate Student[1],Assistant Professor[2], Department of Microbiology,Professor[3], Department of Conservative and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction:PVL and γ -haemolysin are considered to be members of a toxin family known as synergohymenotropic toxins, as they act on cell membranes by the synergy of two proteins that form a pore. Only 2% of S. aureus isolates produce PVL, while γ -haemolysin is produced by more than 99% of S. aureus isolates. PVL is the most leukocytolytic toxin in the family, however it does not exhibits no haemolytic activity on human erythrocytes. A sum of 20 clinical isolates of S. aureus were subjected to antibiotic sensitivity pattern followed by the detection of pvl gene by PCR. We have observed increased resistance to most of the routinely used antibiotics and 10% of our isolates found to have pvl gene. As this gene is directly associated with skin and soft tissue infections by S. aureus, our two isolates may even cause such infections, although none of these strains were not obtained from cutaneous lesions. Panton–Valentine leukocidin (PVL) is one of many toxins associated with S. aureus infection. Because it can be found in virtually all CA-MRSA strains that cause soft-tissue infections, it was long described as a key virulence factor, allowing the bacteria to target and kill specific white blood cells known as neutrophils. This view was challenged, however, when it was shown that removal of PVL from the two major epidemic CA-MRSA strains resulted in no loss of infectivity or destruction of neutrophils in a mouse model.

Materials and Method: These antibiotics were procured from Himedia, Mumbai. This was performed by Kirby-bauer disc diffusion method as per CLSI guidelines.

Result: We have observed a varied pattern of sensitivity among one *S.aureus*isolates. There was complete resistance observed for penicillin(100%), 9/20(45%)isolates were shown to the resistant to erythromycin,6/20(30%) were to cotrimoxazole,4/20(20%)were to linezolid followed by 3/20(15%) were resistant to ciprofloxacin and clindamycin

Conclusion: The pathogenicity of *Staphylococcus aureus* depends on various bacterial surface components and extracellular proteins.uggests that the Panton-Valentine leukocidin (PVL) is 1 such virulence factor that has a major role in pathogenicity.

Key Words: *Staphylococcus aureus, Pvl gene, PCR, Leukocidin ,Haemolysin.*

Introduction

Staphylococcus aureus is an important human pathogen that causes a wide range of diseases from mild superficial skin infection to life-threatening bacteremia and infective endocarditis, as well as toxin-mediated conditions such as toxic shock syndrome.[1] They produce more than 30 different extracellular products.[2]Nearly all strains exhibit a group of enzymes and cytotoxinsthat includes haemolysins (α, β, γ and δ), nucleases, proteases, lipases, hyaluronidase and collagenase. Some strains produce one or more additional exoproteins, which include toxic shock syndrome toxin-1 (TSST-1), the staphylococcal enterotoxins (SEA-E, G-I), the exfoliative toxins (ETA and ETB) and Panton–Valentine leukocidin (PVL) [3].

Two major mechanisms of eukaryotic cell death have been identified, namely necrosis and apoptosis. Necrotic cell death has classically been considered as a passive process resulting from physical or chemical injury. In contrast, cells undergoing apoptosis play an active role in their own demise: extracellular stimuli or genetic programming activates a cascade of intracellular

events resulting in morphological and biochemical alterations. Apoptosis has already been described in monocytes and PMNs exposed to S. aureus α-toxin and Fusobacterium necrophorum leukotoxin, respectively (15, 16). PVL-sensitive target cells, including PMNs, macrophages, and the promyelocytic cell line HL-60 (8, 17, 18) are thought to undergo osmotic lysis secondary to pore formation in the cell membrane. This process would be consistent with necrosis.

Although PVL has potent membrane-disturbing and cytolytic activities in vitro, the mechanisms by which it exerts its lethal effects on target cells, particularly on the first line of host defense cells such as neutrophils, and the sequence of events in the overall cytotoxicity are not known. Furthermore, in other clinical situations such as fulminant hepatitis (19), the apparent predominance of necrosis has been shown to be actually secondary to apoptosis. Hence, the mechanisms by which PVL causes injury to host tissues and potentially to peripheral blood cells during the course of infection are not clearly established, and the role of necrosis versus apoptosis in the pathogenesis of PVL-associated infections remains to be defined. The focus of the present study was to characterize the biological effects of S. aureus PVL on human peripheral PMNs in relation to the possible involvement of this toxin in necrotizing pneumonia pathogenesis.

Apoptosis was only triggered at low PVL concentrations, whereas higher concentrations induced necrotic alterations. These different actions may be related to the molecular properties of PVL. At low concentrations, PVL, which binds to specific but unidentified cell surface receptors (17), could produce small numbers of octameric pores (9). These pores could favor the entry of other PVL molecules into the cell, allowing the toxin to act on mitochondria and induce apoptosis. However, at high concentrations (200 nM), PVL may nonspecifically adsorb to the lipid bilayer, forming larger pores that are Ca2+ permissive, forming more numerous octameric pores, or leading to Ca2+ channel opening (36), all of which can result in necrosis. Moreover, larger pores would also lead to a loss of ATP, which is required for most apoptotic processes (37, 38). Thus, the modes of cell death in vivo (necrosis versus apoptosis) may critically depend of the PVL concentration. In staphylococcal infection, cells of the host tissue surrounding the bacteria are

likely to be exposed to a PVL concentration gradient: necrosis would occur locally, while apoptosis would occur in the periphery and even in circulating blood cells.

In previous study, the population structure of S. aureus, isolated from the nares of healthy persons in the Rotterdam area, the Netherlands, was elucidated (8). Strains were obtained from healthy children (<19 years) and elderly persons (>55 years). Invasive strains (blood culture, skin and soft tissue infections, and impetigo isolates) were included in this study (Table). All carriage and clinical isolates (n = 1,033) were mecA negative. We used the same strain collection to study the PVL prevalence in carriage and invasive isolates of S. aureus from a single geographic region.

Detection of PVL gene in Staphylococcus aureus

Staphylococcus aureus isolates were detected for the presence of *pvl*gene by PCR analysis. Detection of the gene was carried out using primer as depicted in table 1. Bacterial DNA was extracted by boiling lysis method. 1 µL of DNA extract was used as template for PCR reaction. The reaction mixture contained 2mM of $Mgcl_2$ 0.2mM dNTP mix and 0.8µM of *pvl*gene with IU of Taq polymerase (New England Biolabs) in a 1x PCR buffered reaction. A positive control of S. aureus with *pvl* gene was also included in this study. PCR amplification was carried out using thermal cycler (Eppendorf) with the following cycling condition. Initial denaturation at 96^{o}C for 2 min and 30 cycles for 30s, 52^{o}C for 60s and 73^{o} C for 30s, followed by a final extension of 6 min at 72^{o}C. PCR products were resolved in 1.5% agarose gel. A 100bp ladder was including in all the gel analysis.[8]

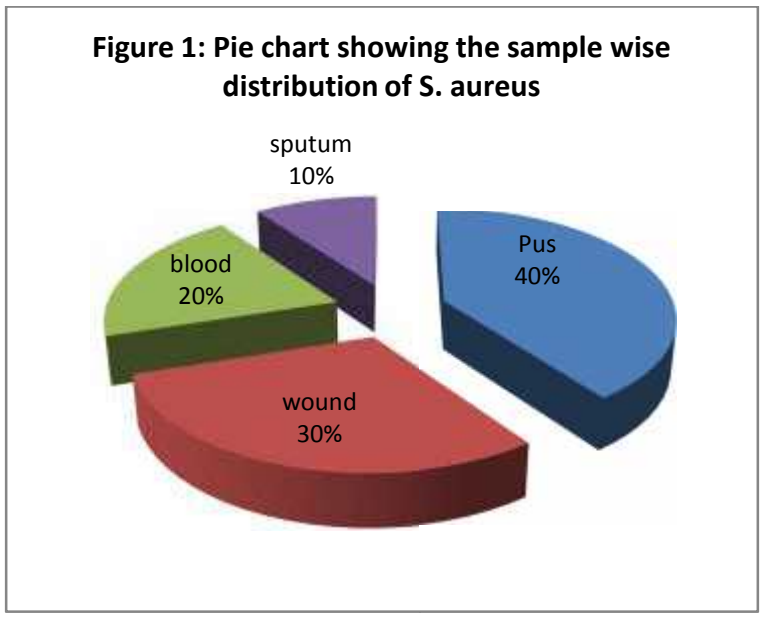
Table 1: Primer detail of *pvl*gene

Primer	Primer sequence	Product size
Pvl	ATC ATT AGG TAA AAT GTC TGG ACA TGA TCC A GCA TCA AST GTA TTG GAT AGC AAA AGC	433bp

Results

Sample wise distribution of clinical isolates of S. aureus

Of 20 clinical isolates of S. aureus, 8/20 (40%) were obtained from pus, 6/20 (30%) were from wound, 4/20 (20%) and 2/20 (10%) were from blood and sputum respectively (Figure 1).



Antibiotic susceptibility pattern

We have observed a varied pattern of sensitivity among one *S.aureus*isolates. There was complete resistance observed for penicillin(100%), 9/20(45%)isolates were shown to the resistant to erythromycin,6/20(30%) were to cotrimoxazole,4/20(20%)were to linezolid followed by 3/20(15%) were resistant to ciprofloxacin and clindamycin respectively (Table 1).

Table 2: Results of antibiotic susceptibility pattern of S.aureus

Antibiotics	Sensitive(%)	Intermediate(%)	Resistant(%)
Penicillin	0	0	20(100)
Erythromycin	14(70)	4(20)	2(10)
Clindamycin	15(75)	2(10)	3(15)
Ciprofloxacin	9(45)	8(40)	3(15)

Tetracyclin	14(70)	4(20)	2(10)
Cotrimoxazole	10(50)	4(20)	6(30)
Linezolid	10(50)	6(30)	4(20)

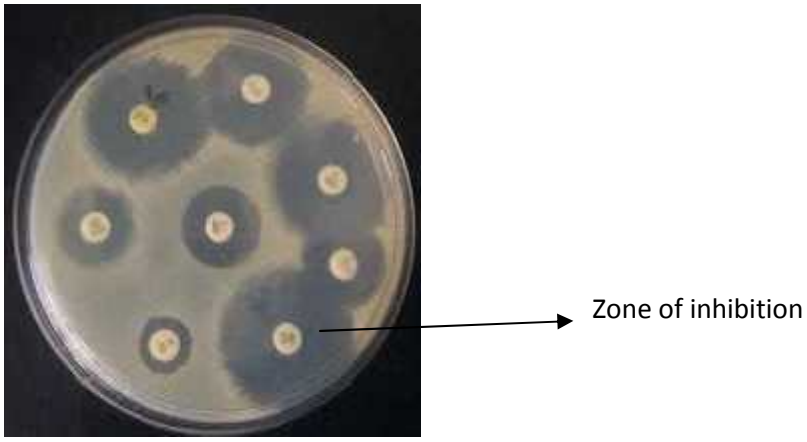


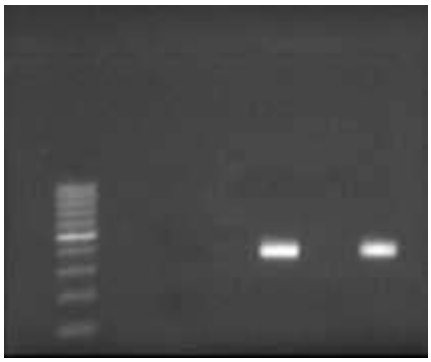
Figure 2: Representative picture showing antibiotic sensitivity pattern of *S. aureus*

Result of *pvl* gene in Staphylococcus aureus:

2/20 (10%) clinical isolate of S. aureus were found to possess *pvl*gene.

Figure 3: Representative gel picture showing pvl gene

L1 L2 L3 L4 L5 L6 L7 L8



-100bp; L6,L8-pvl gene positive

Discussion:

S. aureus is one of the most common Gram-positive bacterial pathogens in humans. It is an opportunistic pathogen that colonizes the skin of approximately 20% of the population without causing clinical symptoms (22, 23). However, breached mucocutaneous membranes or impaired host immunity facilitate tissue invasion and bloodstream dissemination of S. aureus (24). This bacterium can cause serious infections often associated with abscess formation such as osteomyelitis, endocarditis, and pneumonia that often require a prolonged and aggressive antibiotic treatment. Among the most serious complications of S. aureus infections are manifestations of septic and toxic shock syndromes that may lead to multiple organ failure (25). We recently described a novel clinical entity, S. aureus necrotizing pneumonia, which is associated with a massive cell lysis of host pulmonary tissue and is highly lethal in young immunocompetent patients (1). We also found that these cases were strongly associated with S. aureus strains producing the otherwise very infrequent exotoxin PVL (1). In the present report, we demonstrated that PVL is a remarkable bacterial virulence factor because this toxin is localized in the pulmonary lesions of patients who died from necrotizing pneumonia and induces PMN apoptosis by directly targeting mitochondria.

Staphylococcal infections are typically associated with tissue death. Induction of apoptosis by S. aureus may cause tissue damage, compromise the antimicrobial immune response, and thereby

facilitate bacterial spread. S. aureus can induce apoptosis of epithelial cells (26–28), endothelial cells (29–31), keratinocytes (32), osteoblasts (33), lymphocytes, and macrophages (34). In epithelial cells, keratinocytes, or osteoblasts, S. aureus–induced apoptosis may require internalization of the bacteria (26, 27, 30, 32, 33). Conversely, Bantel et al. (16) showed that S. aureus α-toxin does not require bacterial internalization to induce apoptosis. Similarly, our data indicate that bacterial invasion of PMNs is not required for PVL to induce apoptosis. Hence, soluble PVL can be regarded as a mediator of S. aureus–induced neutrophil apoptosis, depleting neutrophils available for phagocytosis. Neutrophils transit through the human circulation en route to tissues, where they form the first line of cellular defense against invading bacterial pathogens. Phagocytosis of complement-opsonized targets is a primary function of neutrophils at sites of inflammation, and clearance, by apoptosis, of PMNs that have phagocytosed microbes is important for the resolution of inflammation (35). By inhibiting neutrophil phagocytosis, PVL could enhance the pathogenicity of S. aureus. Neutropenia is significantly more frequent in patients with necrotizing pneumonia caused by PVL-positive S. aureus than in patients with pneumonia caused by PVL-negative S. aureus (1) and could be due to the presence of PVL in pulmonary endothelial cells. Circulating PMNs could encounter PVL during their passage into the pulmonary vessels and therefore enter the cascade that leads to their apoptosis. As the cellular systems used in this study may not accurately reflect host-pathogen interactions in vivo, we cannot exclude the possibility that other staphylococcal exoproteins might contribute to human cell death.

In the present study, we have detected pvl gene in 2/20 (10%) of our S. aureus isolates. Similar kind of study conducted by Johnsson and colleagues in 2004 reported PVL gene was detected in one out of 65 S. aureus isolates collected prospectively from septicaemic patients.[9] This finding was in agreement with our study and other previous studies showing prevalences of 0–2% in positive blood cultures.[10] In addition, none of the S. aureus isolates causing infective endocarditis was PVL-positive.[11]Thus, PVL does not seem to represent an important virulence factor in invasive bloodstream infections. Our isolates were not collected from skin and soft tissue related infection, even though, two strains were showed positive for this gene. In cutaneous infections, PVL has been associated more frequently with direct invasion and tissue

destruction (e.g., necrotising primary skin infections such as furunculosis) than with secondary infections after skin injury. he Staphylococcus aureus Panton-Valentine leukocidin (PVL) is a pore-forming toxin secreted by strains epidemiologically associated with the current outbreak of community-associated methicillin-resistant Staphylococcus aureus (CA-MRSA) and with the often-lethal necrotizing pneumonia. To investigate the role of PVL in pulmonary disease, we tested the pathogenicity of clinical isolates, isogenic PVL-negative and PVL-positive S. aureus strains, as well as purified PVL, in a mouse acute pneumonia model.

Isolates categorized by type of staphylococcal infection revealed that PVL positive isolates were strongly associated with superficial abscesses and other skin and soft tissue infections, whereas the association of the PVL positive isolates with bone and joint infections was low. These results confirm reports from previous studies where it was detected that 93% of PVL positive S. aureus isolates were associated with furunculosis and other skin and soft tissue infections [6]. The current study and recent reports from Europe demonstrate that PVL positive methicilin susceptible S. aureus has emerged as a significant cause of skin and soft tissue infections and invasive infections such as necrotising pneumonia, soft tissues necrosis [24-26]. Alhough PVL positive S. aureus are often associated with fatal necrotising pneumonia cases in the present study there were only two PVL positive MSSA caused pneumonia cases with positve outcome.

PVLpositive S. aureus is isolated rarely from cases of folliculitis or impetigo. PVL and γ -haemolysin are considered to be members of a toxin family known as synergohymenotropic toxins, as they act on cell membranes by the synergy of two proteins that form a pore. Only 2% of S. aureus isolates produce PVL, while γ -haemolysin is produced by more than 99% of S. aureus isolates. [4]Pvlis the most leukocytolytic toxin in the family, however it does not exhibits no haemolytic activity on human erythrocytes.[5] It also dermo-necrotic, as observed after .intradermal injection of rabbit skin. [6] At sub-lytic concentrations, PVL has been demonstrated to induce granule secretion and release of leukotriene B4 and interleukin-8 from human polymorphonuclear leukocytes.[6] Based on this background, we have undertaken this study to detect the presence of pvl gene among S. aureus by PCR. In 1932, Panton and Valentine described PVL as a virulence factor belonging to the family of synergohymenotropic toxins (4).

These toxins form pores in the membrane of host defense cells by synergistic action of 2 secretory proteins, designated LukS-PV and LukF-PV, which are encoded by 2 cotranscribed genes of a prophage integrated in the S. aureus chromosome (5). PVL is mostly associated with community-acquired methicillin-resistant S. aureus (MRSA) infections and distinguishable from nosocomial MRSA by nonmultidrug resistance and carriage of the type IV staphylococcal chromosome cassette element .

Conclusion:

In our study, we have seen only 10% of them were found to harbor pvl gene. As this gene is directly associated with skin and soft tissue infections by S. aureus, our two isolates may even cause such infections, although none of these strains were not obtained from cutaneous lesions. In order to prove its virulence, it is necessary to include more samples especially from cutaneous lesions.

Corresponding Author

M S Nivedhitha,

Professor,

Department of Conservative and Endodontics,

Saveetha Dental College Hospitals, Saveetha University,

Saveetha Institute of Medical and Technical Sciences.

162,PoonamelleeHigh Road,

Chennai-600095.

India,

Mobile number: 9840912367.

Email id: nivea16@hotmail.com

References:

1. Lowy FD. Staphylococcus aureus infections. N Engl J Med 1998: 339; 520–532.

2. Rogolsky M. Nonenteric toxins of Staphylococcus aurues. Microbiol Rev 1979: 43;320–360.

3. Dinges M, Orwin P, Schilievert P. Exotoxins of Staphylococcus aureus. Clin Microbiol Rev 2000: 13; 16–34.

4. Pre´vost G, Criebier B, Couppie´ P et al. Panton–Valentine leucocidin and gamma-hemolysin from Staphylococcus aureus ATCC 49775 are encoded by distinct genetic loci and have different biological activities. Infect Immun 1995:63; 4121–4129.

5. Kuroda M, Ohta T, Uchiyama I et al. Whole genome sequencing of methicillin-resistant Staphylococcus aureus. Lancet 2001:357;1225–1240.

6. Criebier B, Pre´vost G, Couppie´ P, Finck-Barbancon V, Grosshans E, Pie´mont Y. Staphylococcus aureus leukocidin: a new virulence factor in cutaneous infections? An epidemiological and experimental study. Dermatology 1992:185; 175–180.

7. Clinical and Laboratory Standards Institute. Performance Standards for Antimicrobial Disk Tests; Approved Standards; Doucement M2-A9, 9th ed., Vol 26. Wayne, PA: CLSI; 2015

8. Terry Alli OA, Ogbolu DO, Mustapha JO, Akinbami R, AjayiAO.The non-association of *Panton-Valentine leukocidin*and*mecA*genes in the genome of *Staphylococcus aureus* from hospitals in South Western Nigeria.*Indian Journal of Medical Microbiology, (2012): 30(2);159-64.*

9. Johnsson D, Molling P, Stralin K. Detection of Panton–Valentine leukocidin gene in Staphylococcus aureus by LightCycler PCR: clinical and epidemiological aspects. European Society of Clinical Microbiology and Infectious Diseases. 2004:10(10);884-889.

10. Finck-Barbancon V, Pre´vost G, Pie´mont Y. Improved purification of leukocidin from Staphylococcus aureus and toxin distribution among hospital strains. Res Microbiol 1991: 142; 75–85.

11. Lina G, Piemont Y, Godail-Gamot F et al. Involvement of Panton–Valentine leukocidin-producing Staphylococcus aureus in primary skin infections and pneumonia. Clin Infect Dis 1999: 29;1128–1132.

12. Couppie´ P, Criebier B, Pre´vost G, Grosshans E, Pie´mont Y. Leukocidin from Staphylococcus aureus and cutaneous infections: an epidemiologic study. Arch Dermatol 1994:130;1208–1209.

13. Geetha RV, Roy A, Senthilkumar S, Bhaskar AS, Vijayaraghavan R. A concept of a Probable Autoinjector for Bio-threat Agents. Defence Science Journal. 2016 Sep 1;66(5):464

14.Lina G, Piémont Y, Godail-Gamot F, Bes M, Peter MO, Gauduchon V, Vandenesch F, Etienne J. Involvement of Panton-Valentine leukocidin—producing Staphylococcus aureus in primary skin infections and pneumonia. Clinical Infectious Diseases. 1999 Nov 1;29(5):1128-32.

15.Diep BA, Chan L, Tattevin P, Kajikawa O, Martin TR, Basuino L, Mai TT, Marbach H, Braughton KR, Whitney AR, Gardner DJ. Polymorphonuclear leukocytes mediate Staphylococcus aureus Panton-Valentine leukocidin-induced lung inflammation and injury. Proceedings of the National Academy of Sciences. 2010 Mar 23;107(12):5587-92.

16.Genestier AL, Michallet MC, Prévost G, Bellot G, Chalabreysse L, Peyrol S, Thivolet F, Etienne J, Lina G, Vallette FM, Vandenesch F. Staphylococcus aureus Panton-Valentine

leukocidin directly targets mitochondria and induces Bax-independent apoptosis of human neutrophils. The Journal of clinical investigation. 2005 Nov 1;115(11):3117-27.

17. Jayakumar S, Meerabai M, Shameem Banu AS, Renu Mathew KM, BineshLal Y. Prevalence of high and low level mupirocin resistance among staphylococcal isolates from skin infection in a tertiary care hospital. Journal of clinical and diagnostic research: JCDR. 2013 Feb;7(2):238.

18.Kadiyala SV, Gopinath P. Detection of icaA gene for intracellular adhesion among clinical isolates of Staphylococcus aureus. Research Journal of Pharmacy and Technology. 2016 Sep 1;9(9):1451

19. .Pre´vost G, Couppie´ P, Pre´vost P et al. Epidemiological data on Staphylococcus aureus strains producing synergohymenotropic toxins. J Med Microbiol 1995: 42; 237–245.

20.Shah KK, Gopinath P. Detection of FnbA gene encoding for fibronectin binding protein among clinical isolates of Staphylococcus aureus. Research Journal of Pharmacy and Technology. 2017 Feb 1;10(2):378-80.

Determination Of Sex By Measuring Mastoid Process

Madhumathi .D [1], Thenmozhi.M.S [2], Manoranjitha.R[3]
Graduate Student [1],Professor [2] ,Department of Anatomy
Tutor [3],Department of Public Health Dentistry
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Background: Determination of sex is an essential criteria, for the identification of an individual.In number of civil and criminal matters identification of the individual, either living or dead, is needed. The anatomical and medical features are the two major characters to constitute the identity. The establishment of identity is required from fresh intact corpses, decomposed corpses, mutilated and dismembered corpses or skeletalised material

Aim: The aim of the research was to measure the height and width of mastoid process and distance between the asterion to tip of the mastoid process for the determination of sex.

Objective: In this research the measuring of height,width of mastoid process and distance between the asterion to tip of the mastoid process in the dry skulls of South Indian adults was focused.The measurement of mastoid process will differ in male and female.since it is different,the measurement is useful in anthropology and also in forensic studies.

Materials and Method: The collection of 30 human dry skulls and measuring the height,length and width of mastoid process for the sex determination.Determining the sex of human skeletal remains using the skull is important to the disciplines of human osteology, forensic anthropology, paleopathology,paleodemography for examination and diagnosis.

Result: 30 skulls are used for this study.In that 14 Skulls are identified as female skull and remaining 16 skulls are identified as male skull by measuring the area of a mastoid triangle by using heron's formula.The mastoid process is lengthier in males than in females.

Conclusion: Sex could be determined very well from the cranium using Anthropometry.Parameters like mastoid process length can be used as a good index for the determination of sex.From the present study conducted on 30 dry skulls it is concluded that mastoid length is a reliable indicator for sexual dimorphism in mastoid process of skulls. The result of this study offering a good opportunity to identify the sex using the mastoid process. The length of the mastoid process is higher in males than females.

KeyWords*:Mastoidprocess,Mastoidtriangle,asterion,porion,mastoidale,Heron'sformula,sexdifferentiation,Forsenic studies.*

Introduction

Determination of sex is an essential criteria, for the identification of an individual.In number of civil and criminal matters identification of the individual, either living or dead, is needed. The anatomical and medical features are the two major characters to constitute the identity. The establishment of identity is required from fresh intact corpses, decomposed corpses, mutilated and dismembered corpses or skeletalised material [2]. Sex determination of human or human skeletal remains is considered vital step in identification and is crucial for further analysis.Skull and pelvis assume great importance in establishing sex of an individual.The sex is best assessed from pelvis but it is very often damaged in case of decomposition,fire accidents,etc.,The second best area for the identification of sex is the skull.Krogman [21] states that skull is the most dimorphic and easily sexed portion of skeleton after pelvis, providing up to 92% reliability. Bass [22] states that the skull probably is the second best area of the skeleton to use for determining sex. Bass [22], Byers [23] and Pickering and Bachman [24] presented the idea that skull is the second best choice to estimate the sex of the dead body.Thefeatures in the skull, that helps in the sex determination includes general appearance,supra orbital ridges,glabella,zygomaticarches,mastoidprocesses,external occipital protuberance,mandible and palate.

The mastoid process characteristics has a greater utility in the determination of sex.It is a pyramidal shaped projection in the temporal bone situated behind the ear on each side(1). If the pelvis is absent in accidental cases,it is difficult to identify the gender of a human .In such cases,the mastoid process is used to determine the sex by measuring the mastoid triangle because the length varies in both the male and female.It is situated behind the external auditory meatus and lateral to styloid process [1].The mastoid process is sexually dimorphism has been affirmed non- metrically by Hoshi [18],Larnach and Macintosh [19] and metrically by Saavedradepaiva and sagre [1].Mastoid region is one of the regions with most dimorphic traits. The tip of mastoid process is sexually dimorphic. It tend to be vertical in male and pointed inwards in females [25].

Surface

A sex differentiation in the mastoid process is made by calculating the area of mastoid triangle which is a technique reported by Paiva and Segre [20].This technique involves the measurement of the distance between the three triangular points(Asterion,Porion and Mastoidale).

Asterion- Meeting point of lambdoid,occipitomastoid and parietomastoid sutures.
Mastoidale-Tip of the mastoid process.
Porion -The uppermost lateral point of external auditory meatus.

Calculating the area of the triangle and then adding the right and left areas of the triangle gives a value which is used to identify the sex which is calculated by using heron's formula.The value is higher in males when compared to females.This method of sex differentiation is useful in forensic studies.

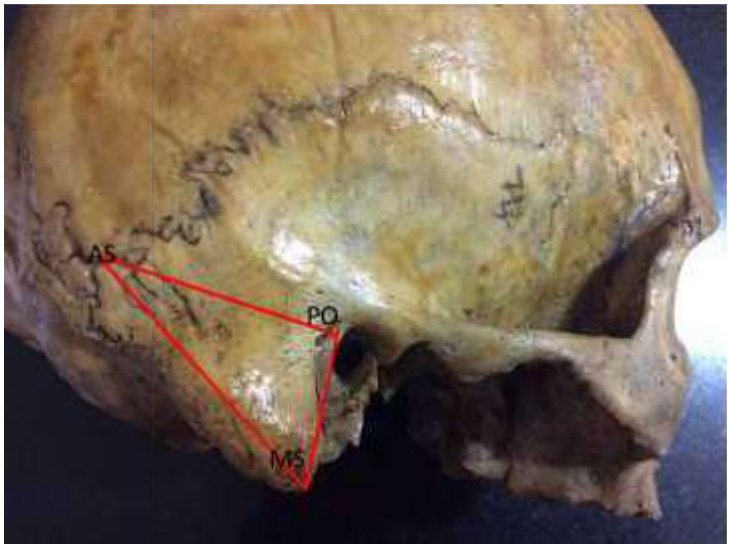
Materials and Methods

The study sample was taken from the Department of Anatomy,Saveetha dental college,SaveethaUniversity,Chennai,TamilNadu,India which represents the south Indian population.A total of 30 skulls were selected for the study.Three points (Asterion (As),Porion (Po) and Mastoidale(Ms)which forms the mastoid triangle were marked.The measurements are made by using the verniercalliper (0.01mm) between asterion to mastoidale,mastoidale to Porion and asterion to porion.The area of the triangle was calculated by the Heron's formula with the three sides a,b&c.

A=square root of s(s-a)(s-b)(s-c)

If the value of the total area is greater than or equal to1447.60mm2 ,it denotes the male skull or if the value is less than or equal to 1260.70mm2.

Figure 1: Shows Mastoid Triangle



Result

30 skulls are used for this study.In that 14 Skulls are identified as female skull and remaining 16 skulls are identified as male skull by measuring the area of a mastoid triangle by using heron's formula.The mastoid process is lengthier in males than in females.

Table-1: Shows values after measuring mastoid angle using Herons formula

	N	Range	Minimum	Maximum	Mean	Std. Deviation
LENGTH BETWEEN ASTERION AND MASTOIDALE .Right	30	21.0	35.0	56.0	45.630	5.2261
LENGTH BETWEEN ASTERION AND MASTOIDALE .Left	30	21.1	34.8	55.9	44.497	5.1854
LENGTH BETWEEN ASTERION AND PORION. Right	30	23.7	27.8	51.4	40.938	5.2963
LENGTH BETWEEN ASTERION AND PORION. Left	30	24.9	25.7	50.5	40.453	5.7737
LENGTH BETWEEN PORION AND MASTOIDALE. Right	30	17.5	13.9	31.4	23.289	4.5257
LENGTH BETWEEN PORION AND MASTOIDALE. Left	30	17.2	14.7	31.9	23.017	4.1514

Discussion

The determination of mastoid triangleis important in sexing the individuals.This is the most damaging part during the skull damage.This study involves the measurement of mastoid

process by using heron's formula.The area for mastoid triangle is calculated for each skull.According to the study by Paiva and Segre, male triangle area was significantly greater than female triangle area. Values calculated according to their study mean values was : in female : 570.9 mm2 and 575.1 mm2 in right and left sides. In male : 723.9 mm2 and 731.2 mm2 in right and left sides(1).One of the previous studies results shows male right :663.66mm2 ,male left : 662.54 mm2, female right : 595.92 mm2, female left : 589.29mm2 was found [20].

In this study,the value of the total area is greater than or equal to 1447.60mm2 ,it denotes the male skull or if the value is less than or equal to 1260.70mm2, denotes a female skull.In this study 14 skulls have a value less than 1260.70mm2 and 16 skulls have a value above 1447.60mm2.One of the defect of this technique is its use in sexing individual skulls and also due to asterion point localization, which position changes during the course of life [1].Other studies are done by using the known skulls(i.e.,by knowing whether skull is male or female).But this present study was done by using the unknown skulls (i.e.,without knowing the sex of the skull).

One of the previous studies suggests that, when skulls were placed on at surface, the male skulls rest on the mastoid processes while female skulls on occipital condyles or other portions of the skull. This observation indirectly indicates that males skulls have more mastoid length as compared to female skulls, that is why, male skulls rest on mastoid processes but not female skulls [3].

Area values are compared between present study and reference study which are depicted in the Table-2.

Table-2 depicts the difference in total area of males and females between present study and one of the reference studies

SEX	TOTAL AREA	
	Presentstudy	Reference study
Male	1492.62mm2	1461.06mm2
Female	1243.81mm2	1222.79mm2

Conclusion

Sex could be determined very well from the cranium using Anthropometry.Parameters like mastoid process length can be used as a good index for the determination of sex.From the present study conducted on 30 dry skulls it is concluded that mastoid length is a reliable indicator for sexual dimorphism in mastoid process of skulls. The result of this study offering a good opportunity to identify the sex using the mastoid process. The length of the mastoid process is higher in males than females.

Corresponding Author

Manoranjitha

Tutor,Department of Public Health Dentistry

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University

162,Poonamalle High road,Chennai 600077,Tamilnadu, India.

E-Mail: manomahi96@gmail.com

Telephone Number: 8248393335

Reference

1. Saavedra de Paiva LA, Serge M. Sexing the human skulls through the mastoid process. Rev HospClinFac Med Sao Paulo. 2003; 58(1): 15-20.
2. PekkaSaukko, Bernard Knight, Knight's Forensic pathology, 3rd edition, the establishment of identity of human remains: 98- 99.
3. JigyasaPassey, Suniti R Mishra, Rahul Singh, K Sushobhana, Shailendra Singh, Priyanka Sinha. Sex determination using mastoid processes. Asian Journal of Medical Sciences. 2015; 6(6):93-95
4. Dr. Sunil Poonia, Dr Seema Gupta, Dr.PoojaChoudhary and Dr. Manoj Verma. Sex determination using mastoid process measurements of dryskull bone: A Descriptive Analysis,International Multispecialty Journal of Health (IMJH) ISSN: 2016; 2(10):2395-6291
5. ShobhaVerma1, C. S. Ramesh Babu2,Sex determination by using mastoid process in western UP population,Journal of Research in Human Anatomy and Embryology. 2015; 1(1):1-5.

6. Sumathi (assistant professor),VVGpatnaik,Determination of Sex from Mastoid Process By Discriminant Function Analysis,Janatsoc. India. 2010; 59(2):222-228
7. Manoonpol,C,plakornkulV.Sex determination using mastoid process measurement in Thais,J Med Assoc Thai. 2012;95(3):423-9.
8. T.Stroicā, Laura; Badiu, Ileana Mirella; State, D.; Munteanu, O.; Baciu, Adina; Tartţa, E.; Cristea, The dimensions of mastoid process-indicators of sexual dimorphism**.** Romanian Journal of Functional & Clinical, Macro- &Microscopical Anatomy &Anthropology. 2013; 12(2):101-105.
9. ShobhaVerma, Ramesh Babu. Sex determination by mastoid process in western UP populations. J of research in human anatomy ansd embryology.2015;1(1):1-5.
10. SarawutSujarittham, M.D., KarndaVichairat , M.D.,SukonPrasitwattanaseree, Ph.D.andPasukMahakkanukrauh, M.D.,Thai Human Skeleton Sex Identification by Mastoid ProcessMeasurement,Chiang Mai Med. Bull. 2016; 50:43-50.
11. DeepaliJainM.Sc(Research Scholar in Forensic Science) PhD(Professor)aSurinderNathM.Sc, PhD(Formerly Professor of Anthropology)b".Sex determination of human crania using Mastoid triangle and Opisthion–Bimastoid triangle,J Forensic Leg Med. 2013;20(4):255.
12. N.R.Jaja,Christina O Auja,Mastoid Triangle for Sex Determination in Adult Nigerian Population: A Validation Study,J Forensic Sci. 2013;58(6):1.
13. TomohitoNagaoka,AkioShizushima,Sex determination using mastoid process measurements: standards for Japanese human skeletons of the medieval and early modern periods,Anthropological Science - anthropol sci. 2013; 116:105-113.
14. M.P. Sarangi, M.P. Saragi, S. Das, G.K. Sharma and J.J. Roy Burman",Mastoid module—A clue to sex determinationIndian Anthropologist 1992; 22(2):72-76
15. Anjapetros,sabrina B sholtsEvaluating sexual dimorphism in the human mastoid process: A viewpoint on the methodology,Anat. 2015;28(5):593-601.
16. Geethika. B, Thenmozhi. Sex determination using the mastoid process using South Indian skulls. Research J. Pharm. and Tech 2016; 9(11): 1869-1871.
17. Thejeswar EP, MS Thenmozhi. Role of mastoid process in determination of sex. Research Journal of Pharmacology and Pharmacodynamics. 2015; 7(2): 76-81.
18. Hoshi H. Sex difference in the shape of mastoid process in normaoccipitalis and its importance to the sex determination of the human skull. Okajima's Folia Anatomica Japonica, 1962; 38: 309–317.
19. .Larnach SL, Mactonish NWG, The craniology of the aborigines of Coastal New

South Wales. The Oceania Monographs. 1966; 13:43-4.

20. Swati Shah, Pratik Patel. Sexing the human skull using the mastoid process. NHL Journal of Medical Sciences. 2013; 6(2); 120-5.
21. W.M.Krogman, Mehmet YasarIscan, The human skeleton in Forensic medicine, 2nd edition, 191-194.
22. Bass WM : Human osteology : A Laboratory and field manual, 5th edn Columbia , MO : 2005
23. Byers S.N.Introduction to forensic anthropology , a textbook .Boston, A : Alyn and Bacon publishers,2002.
24. PickeringRB,BachmanDC.Theuseofforensicanthropology. Boca Raton , FL : CRC Press,1997.
25. De Moulin F. Importance de certaines measures craniensedans la determination sexualle des cranes. Bull at Mem de la socD'Anthropal de Paris 1972; 259- 64.

Dysmenorrhea in Women

Harini. N.S[1], Gowri Sethu[2], Sankari.M[3]

Graduate student[1], Professor [2],Department of Physiology,

Professor[3], Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Dysmenorrhea is a common menstrual complaint with a major impact on women's quality of life, work productivity, and health-care utilization. The prevalence of dysmenorrhea varies between 16% and 91% in women of reproductive age, with severe pain in 2%–29% of the women studied. Women's age, parity, and use of oral contraceptives were inversely associated with dysmenorrhea, and high stress increased the risk of dysmenorrhea. Inconclusive evidence was found for modifiable factors such as cigarette smoking, diet, obesity, depression, and abuse. Dysmenorrhea is a significant symptom for a large proportion of women of reproductive age; however, severe pain limiting daily activities is less common.

Aim: The main aim of this review article is to know in detail about Dysmenorrhea in Women.

Objective: The objective of this article is to know about dysmenorrhea in women, what is the cause, what are the consequences and why is it happening and also to know about dysmenorrhea in all aspects and to know the opinions from different authors.

Materials and Methods: The present study was to know about Dysmenorrhea in women. Various articles from different authors were collected and summed up as a review article so that Dysmenorrhea in women is known elaborately in all aspects.

Conclusion: In conclusion, this review shows that dysmenorrhea is a significant symptom for a large proportion of women throughout the reproductive years. Severe dysmenorrhea limiting daily activities is much less common. Improvement of the symptom over time has been observed, although many women also experience unchanged or worse symptoms. Dysmenorrhea is inversely related to age, parity or number of live births and oral contraception us. Dysmenorrhea is positively associated with stress related to both work and general life, as well as with family history of dysmenorrhea.

Keywords: *Dysmenorrhea, women, menstruation, painful periods, abdomen.*

Introduction

Dysmenorrhea or painful menstruation is defined as a severe, painful, cramping sensation in the lower abdomen that is often accompanied by other symptoms, such as sweating, headaches, nausea, vomiting, diarrhoea, and tremulousness, all occurring just before or during the menses [1]. There are 2 types of dysmenorrhea: Primary dysmenorrhea refers to pain with no obvious pathological pelvic disease and almost always first occurs in women 20 years or younger after their ovulatory cycles become established [1]. Secondary dysmenorrhea is caused by underlying pelvic conditions or pathology and is more common in women older than 20 years [1, 2].

Dysmenorrhea is considered the most common symptom of all menstrual complaints and poses a greater burden of disease than any other gynecological complaint in developing countries [3]. Among women of reproductive age worldwide, dysmenorrhea is more prevalent than the other 2 common types of chronic pelvic pain, namely, dyspareunia and noncyclical chronic pelvic pain [4]. Being a debilitating condition for many women, it has a major impact on health-related quality of life, work productivity, and health-care utilization [5–9]. As a result, dysmenorrhea is responsible for considerable economic losses due to the costs of medications, medical care, and decreased productivity [1]. The prevalence of dysmenorrhea reported in the literature varies substantially. A greater prevalence was generally observed in young women, with estimates ranging from 67% to 90% for those aged 17–24 years [10, 11].

A recent large Australian study of senior high school girls found that a higher proportion, 93%, of teenagers reported menstrual pain [12]. The studies in adult women are less consistent in reporting prevalence of dysmenorrhea and often focus on a specific group, with rates varying from 15% to 75% [10]. Severe pain sufficient to limit daily activities is considerably less common, affecting approximately 7%–15% of women [10], although a study of adolescents and young adults aged 26 years or less reported that 41% of the participants had limitations in their daily activities due to dysmenorrhea [7].

A range of risk factors for dysmenorrhea have been identified in the literature, although mixed results have been observed for many of these factors. In general, increased severity of dysmenorrhea has been suggested to relate to age [13–15], smoking [14, 16, 17], higher body mass index [18], earlier age at menarche [15, 18], nulliparity [15, 19], longer and heavier menstrual flow [15, 18], and family history of dysmenorrhea [20]. Women using oral contraceptives generally report less severe dysmenorrhea [14, 15, and 21]. Depression and stress have also been shown to increase the risk of dysmenorrhea [9, 22]. Other common factors, such as education [23], marital status [14], employment [19], alcohol consumption [17, 18], and physical activity [15, 18], show largely negative or inconclusive results.

Material and Methods

The present study was to know about Dysmenorrhea in detail. Various articles from different authors were collected and summed up as a review article so that dental crowding is known elaborately in all aspects. The articles were collected by manual searching of the references of the relevant retrieved articles, peer-reviewed physiologic journals, and gray literature. Search terms included dysmenorrhea, menstruation, women, painful periods. Non-English articles were excluded from the review in the study-selection stage. Data extraction and evaluation of primary studies were performed independently by 2 reviewers.

Discussion

Knowledge of physiology of reproduction is important for teenage girls to practice menstrual hygiene [47]. Dysmenorrhoea is a common distressing disorder in women that manifests during menstrual phase especially in young females [51]. Primary dysmenorrhea is a medical condition

of pain which is worst during first day of menstruation that interferes with daily activities. It is not associated with any pathological changes.[48]. Dysmenorrhea is defined as painful menstrual cramps of uterine origin. It is the common gynecological condition that can affect as many as 50% of women. [49].

Dysmenorrhea is one of the most common gynecological problems among female students, which may be the leading cause of absenteeism from college. Dysmenorrhea is of two types. The primary is a menstrual pain without any organic pathology. When the pelvic pain is associated with identifiable pathological conditions, it is called secondary. Dysmenorrhea also may be due to anxiety, emotional instability, a faulty outlook on sex and menstruation, or imitation of the mother's feelings about menstruation. Researchers reported that excessive production of prostaglandins, which causes severe uterine contractions, which cause pain and decrease blood flow and oxygen to the uterus. Similar to labour pains, these contractions can cause significant pain and discomfort. Prostaglandins may also contribute to nausea and diarrhoea. [49].

Most studies show that dysmenorrhea is a common problem affecting the majority of women in the community. Severe pain or pain limiting women's daily activities, however, occurred only in 2%–28% of adult women. Self-medication is a worldwide problem and it is common in developing countries like India every day, everywhere, consumers reach for self-care products to help them through their common health problems.[50] The lowest rate of 2% reported in a longitudinal study was possibly due to potential underreporting as 74% of the included women were employed and thus less likely to stay in bed and miss work, which is used to define severe pain in the study [37]. The prevalence of severe pain reported in this review appears to be higher than the 12%–14% reported in community-based studies in the World Health Organization review [4] but comparable with 5%–20% reported in another review of the condition in developing countries [38].

Weissman [37] found that dysmenorrhea persisted over the 6-year follow-up among the majority of women reporting it at baseline, and improvement or worsening of the symptom was equally likely. In their multivariable analysis, the presence of dysmenorrhea at baseline [excluding

women with severe dysmenorrhea] was a strong predictor of reporting moderate or severe dysmenorrhea at follow-up [odds ratio = 7.48, 95% CI: 3.09, 18.15]. More studies are needed to explore the natural history of the symptom. Despite some disagreement, the majority of the previous literature generally demonstrates an inverse association between both age and parity and dysmenorrhea [10, 13–15, 41].

This association was confirmed by the vast majority of studies included in this review, consistent across different types of study, although 3 studies failed to adjust for parity in their analysis on the association between age and dysmenorrhea [30, 32, 34]. Interestingly, Burnett et al. [26] found that the effect of age remained in the adjusted model including nulliparity, whereas the association between nulliparity and primary dysmenorrhea was no longer significant when controlled for age and smoking [$\beta = 0.93$; $P = 0.582$]. It is unclear though what the proportion of nulliparous women was in the study.

Furthermore, the study did find that the women most debilitated by pain were significantly more likely to be nulliparous. Nevertheless, the longitudinal study by Weissman et al. [37] provides stronger support for the inverse association between both age [odds ratio = 0.92, 95% CI: 0.86, 0.98] and livebirth [odds ratio = 0.20, 95% CI: 0.08, 0.53] and the severity of dysmenorrhea after controlling for each other; however, parity clearly had a much stronger effect in their analysis. In addition, the study also found that gravidity was less influential than livebirth, consistent with other studies showing no effect from pregnancies ending in miscarriage or abortion [15, 41].

Different mechanisms have been proposed for the relation between livebirth and dysmenorrhea. One is related to the pathogenesis of primary dysmenorrhea of the close association with elevated prostaglandin levels in the secretory endometrium that triggers pain [1, 42]. After a term delivery, the endometrium may release a lower level of prostaglandins, resulting in decreased pain [41]. Another hypothesis is that neuronal degeneration in the uterus following term pregnancy, due to disappearance of uterine adrenergic nerves and a decrease in uterine noradrenalin in the third trimester of pregnancy, may explain the disappearance or reduction of menstrual pain after childbirth [15].

A strong effect of family history of dysmenorrhea and risk of dysmenorrhea was shown in 2 studies, which is in line with some previous studies reporting a similar association, suggesting genetic susceptibility to dysmenorrhea among women with variant genotypes in a number of metabolic gene polymorphisms [20, 43]. However, other possible explanations are that the association could be related to conditioned behavior that is learned from mother or sisters for the possibility of societal reward or that control for pain exists [1]. Alternatively, it could be simply due to similar living patterns and lifestyles in the families [34]. Among the range of lifestyle and other demographic factors studied such as smoking, body mass index, and socioeconomic status, conflicting results were shown.

Previous studies on the association between smoking and dysmenorrhea are mixed. Although most cross-sectional studies show an increased risk among smokers [13, 14, 16], a negative effect was also seen [44]. Inconsistent results have also been observed for smoking and the incidence or the severity of dysmenorrhea in longitudinal studies [15, 18]. Sundell [15] found that the prevalence and severity of dysmenorrhea were increased in smokers and that the severity increased with the number of cigarettes smoked per day.

On the other hand, Harlow and Park [18] found that smoking was not associated with the probability of having pain or severe cramps but, among those with pain, smokers were more likely to have pain lasting longer than 2 days. Similarly, being overweight was found to be an important risk factor for the probability of experiencing pain and for increasing duration of pain in 1 longitudinal study [18], and severity of dysmenorrhea was not associated with either height or weight in another [15]. The systematic review also failed to detect a significant association between obesity and dysmenorrhea in the pooled analysis of 5 studies [40]. No association was shown among women's education, marital status, alcohol use, and the risk of dysmenorrhea in the current review, which is supported by the recent systematic review identified [40] and previous studies [14, 17, 18].

However, caution should apply when interpreting the systematic review results, as heterogeneity was present in all analyses with multiple studies, and the review is based largely on case-control studies that are subject to recall bias. A protective effect of OCPs or other forms of hormonal

contraceptive for dysmenorrhea is evident in the majority of previous studies [10, 13–15, 21], consistent across different study types.

This is largely confirmed by the current review, although 1 of the studies on a large sample of Indian women did not show any significant association between different methods of contraception, including OCPs, and moderate to severe dysmenorrhea [3].

The reason for this discrepancy may be due to the small number of women using OCPs [n = 43] in the study. There is evidence suggesting an association between early age at menarche and increased risk of dysmenorrhea [15, 18, 19], which is supported by the systematic review [40]. However, most of the studies reporting it in this review failed to show an association [26, 31, and 35] with the exception of the Indian study [3].

The reason for the discrepancy is not readily apparent, and the association may be confounded or mediated by other factors. Furthermore, although there is suggestion of some associations of other reproductive factors such as age at first birth, caesarean section, and heavy and irregular menses [14, 18, 41], this review is unable to reach any firm conclusion because of the limited number of studies reporting these effects. Among the psychological factors studied, a positive association between perceived stress, related to work or general life events, and the risk of dysmenorrhea was shown in most included studies.

The biological mechanism for association between work stress and dysmenorrhea is not well understood, although potentially through a cascade of neuroendocrine responses [36]. Stress inhibits the release of follicle-stimulating hormone and luteinizing hormone, leading to impaired follicular development. This can alter progesterone synthesis and release, which may influence the activity of prostaglandin.

Besides progesterone, stress-related hormones, including adrenaline and cortisol, also appear to influence prostaglandin synthesis and/or binding in the myometrium [36].

Furthermore, mental health may act as a mediator in the relationship between stress and dysmenorrhea, with high job stress increasing the risk of mental health morbidity, which in turn

is positively related to painful menses [28]. Of note is that these studies were generally conducted on groups of employed women, whereas another included cross sectional study of a random sample of Iranian women did not observe a significant association between stress level and the severity of pain in the adjusted analysis [34].

Conclusion

In conclusion, this review shows that dysmenorrhea is a significant symptom for a large proportion of women throughout the reproductive years. Severe dysmenorrhea limiting daily activities is much less common. Improvement of the symptom over time has been observed, although many women also experience unchanged or worse symptoms. From longitudinal or population data, this review has confirmed the following:

• Dysmenorrhea is inversely related to age, parity or number of livebirths, and oral contraception use;

• Dysmenorrhea is positively associated with stress related to both work and general life, as well as with family history of dysmenorrhea.

However, uncertainty still remains for a number of lifestyle factors, such as smoking, obesity and diet, psychological factors, and environmental factors. Furthermore, there is a lack of longitudinal data to study the natural history of dysmenorrhea and the effects of a range of modifiable risk factors over time. More research on these from population-based, prospective, longitudinal studies to generate robust evidence will help to support targeted preventive interventions.

Corresponding Author

M.Sankari

Professor,Department of Periodontics

Saveetha Dental College,Saveetha Institute of Medical And Technical Sciences,

Saveetha University, Chennai, India

162, Poonamalle high Road,Chennai -600077, Tamil Nadu, India.

Email.msankari@gmail.com

References:

1. Lentz G, Lobo R, Gershenson D, et al; Comprehensive Gynecology. Philadelphia, PA: Mosby Elsevier; 2012; 53[11]:837–846.

2. Impey L, Child T; Chicester, United Kingdom: Wiley-Blackwell; Obstetrics and Gynaecology; 2012; 20[3]:234–241

3. Patel V, Tanksale V, Sahasrabhojanee M, et al; The burden and determinants of dysmenorrhoea: a population-based survey of 2262 women in Goa, India. BJOG. 2006;113[4]:453–463.

4. Latthe P, Latthe M, Say L, et al; WHO systematic review of prevalence of chronic pelvic pain: a neglected reproductive health morbidity. BMC Public Health. 2006;6:177.

5. Ballagh SA, Heyl A; Communicating with women about menstrual cycle symptoms; J Reprod Med. 2008;53[11]:837–846.

6. Barnard K, Frayne SM, Skinner KM, et al; Health status among women with menstrual symptoms; J Womens Health [Larchmt]. 2003; 12[9]:911–919.

7. Rodrigues AC, Gala S, Neves A, et al; Dysmenorrhea in adolescents and young adults: prevalence, related factors and limitations in daily living; Acta Med Port. 2011;24[suppl 2]: 383–392.

8. Sharma A, Taneja DK, Sharma P, et al; Problems related to menstruation and their effect on daily routine of students of a medical college in Delhi, India; Asia Pac J Public Health. 2008; 20[3]:234–241.

9. Titilayo A, Agunbiade OM, Banjo O, et al; Menstrual discomfort and its influence on daily academic activities and psychosocial relationship among undergraduate female students in Nigeria; Tanzan J Health Res. 2009;11[4]:181–188.

10. Harlow SD, Ephross SA; Epidemiology of menstruation and its relevance to women's health; Epidemiol Rev. 1995;17[2]: 265–286.

11. Kennedy S; Primary dysmenorrhoea; Lancet. 1997; 349[9059]: 1116.

12. Parker MA, Sneddon AE, Arbon P; The menstrual disorder of teenagers [MDOT] study: determining typical menstrual patterns and menstrual disturbance in a large population based study of Australian teenagers; BJOG. 2010; 117[2]: 185–192.

13. Pullon S, Reinken J, Sparrow M; Prevalence of dysmenorrhoea in Wellington women; N Z Med J. 1988; 101[839]:52–54.

14. Messing K, Saurel-Cubizolles MJ, Bourgine M, et al; Factors associated with dysmenorrhea among workers in French poultry slaughterhouses and canneries; J Occup Med. 1993; 35[5]:493–500.

15. Sundell G, Milsom I, Andersch B; Factors influencing the prevalence and severity of dysmenorrhoea in young women; Br J Obstet Gynaecol. 1990; 97[7]:588–594.

16. Mishra GD, Dobson AJ, Schofield MJ; Cigarette smoking, menstrual symptoms and miscarriage among young women; Aust N Z J Public Health. 2000; 24[4]:413–420.

17. Parazzini F, Tozzi L, Mezzopane R; Cigarette smoking, alcohol consumption, and risk of primary dysmenorrhea; Epidemiology. 1994;5[4]:469–472.

18. Harlow SD, Park M; A longitudinal study of risk factors for the occurrence, duration and severity of menstrual cramps in a cohort of college women; Br J Obstet Gynaecol. 1996; 103[11]: 1134–1142.

19. Ng TP, Tan NC, Wansaicheong GK; A prevalence study of dysmenorrhoea in female residents aged 15–54 years in Clementi Town, Singapore; Ann Acad Med Singapore. 1992; 21[3]:323–327.

20. Parveen N, Majeed R, Rajar UDM; Familial predisposition of dysmenorrhea among the medical students; Pak J Med Sci. 2009;25[5]:857–860.

21. Juang CM, Yen MS, Horng HC; Natural progression of menstrual pain in nulliparous women at reproductive age: an observational study; J Chin Med Assoc. 2006;69[10]:484–488.

22. Gordley LB, Lemasters G, Simpson SR, et al; Menstrual disorders and occupational, stress, and racial factors among military personnel; J Occup Environ Med. 2000;42[9]:871–881.

23. Chen C, Cho SI, Damokosh AI, et al; Prospective study of exposure to environmental tobacco smoke and dysmenorrhea; Environ Health Perspect. 2000; 108[11]:1019–1022.

24. Fowkes FG, Fulton PM; Critical appraisal of published research: introductory guidelines; BMJ. 1991; 302[6785]:1136–1140.

25. Abenhaim HA, Harlow BL; Live births, cesarean sections and the development of menstrual abnormalities; Int J Gynaecol Obstet. 2006; 92[2]:111–116.

26. Burnett MA, Antao V, Black A, et al; Prevalence of primary dysmenorrhea in Canada; J Obstet Gynaecol Can. 2005;27[8]: 765–770.

27. Laszlo KD, Gyorffy Z, Adam S, et al; Work-related stress factors and menstrual pain: a nation-wide representative survey; J Psychosom Obstet Gynaecol. 2008;29[2]: 133–138.

28. Laszlo KD, Kopp MS; Effort-reward imbalance and over commitment at work are associated with painful menstruation; results from the Hungaro study Epidemiological Panel 2006. J Occup Environ Med. 2009; 51[2]:157–163.

29. Nohara M, Momoeda M, Kubota T, et al; Menstrual cycle and menstrual pain problems and related risk factors among Japanese female workers; Ind Health. 2011; 49[2]:228–234.

30. Ohde S, Tokuda Y, Takahashi O, et al; Dysmenorrhea among Japanese women; Int J Gynaecol Obstet. 2008;100[1]: 13–17.

31. Pawlowski B; Prevalence of menstrual pain in relation to the reproductive life history of women from the Mayan rural community; Ann Hum Biol. 2004; 31[1]:1–8.

32. Pitts MK, Ferris JA, Smith AM; Prevalence and correlates of three types of pelvic pain in a nationally representative sample of Australian women; Med J Aust. 2008;189[3]: 138–143.

33. Santer M, Warner P, Wyke S; A Scottish postal survey suggested that the prevailing clinical preoccupation with heavy periods does not reflect the epidemiology of reported symptoms and problems; J Clin Epidemiol. 2005;58[11]: 1206–1210.

34. Tavallaee M, Joffres MR, Corber SJ, et al; The prevalence of menstrual pain and associated risk factors among Iranian women; J Obstet Gynaecol Res. 2011;37[5]:442–451.

35. Unsal A, Tozun M, Aslan G; Evaluation of dysmenorrhea among women and its impact on quality of life in a region of western Turkey; Pak J Med Sci. 2010;26[1]:142–147.

36. Wang L, Wang X, Wang W, et al; Stress and dysmenorrhoea: a population based prospective study; Occup Environ Med. 2004; 61[12]:1021–1026.

37. Weissman AM, Hartz AJ, Hansen MD; The natural history of primary dysmenorrhoea: a longitudinal study; BJOG. 2004; 111[4]:345–352.

38. Harlow SD, Campbell OM; Epidemiology of menstrual disorders in developing countries: a systematic review. BJOG. 2004; 111[1]:6–16.

39. Harlow BL, Missmer SA, Cramer DW, et al; Does tubal sterilization influence the subsequent risk of menorrhagia or dysmenorrhea? Fertil Steril. 2002; 77[4]:754–760.

40. Latthe P, Mignini L, Gray R; Factors predisposing women to chronic pelvic pain: systematic review; BMJ. 2006; 332[7544]:749–755.

41. Juang CM, Yen MS, Twu NF; Impact of pregnancy on primary dysmenorrhea; Int J Gynaecol Obstet. 2006; 92[3]: 221–227.

42. Hong Ju, Mark Jones, and Gita Mishra; The Prevalence and Risk Factors of Dysmenorrhea; Epidemiol Rev 2014;36:104–113

43. Wu D, Wang X, Chen D, et al; Metabolic gene polymorphisms and risk of dysmenorrhea; Epidemiology. 2000;11[6]:648–653.

44. Andersch B, Milsom I; An epidemiologic study of young women with dysmenorrhea; Am J Obstet Gynecol. 1982; 144[6]:655–660.

45. Christiani DC, Niu T, Xu X; Occupational stress and dysmenorrhea in women working in cotton textile mills; Int J Occup Environ Health. 1995;1[1]:9–15.

46. Goldstein-Ferber S, Granot M; The association between somatization and perceived ability: roles in dysmenorrhea among Israeli Arab adolescents; Psychosom Med. 2006;68[1]: 136–142.

47. Nidhi Sharma , M. Benjamin Sagayaraj, Balamma Sujatha , Abdul Rahman A. M, Abirami M, Adhil Khan M. H, Aditya. K, Ajantha P; Prevalence of dysmenorrhoea in College Students; International Journal of Scientific and Research Publications; October 2014; Volume 4, Issue 10, p:1-6.

48. G.V.S Nivashini; Prevalence of Primary Dysmenorrhea along With irregular Periods; Research Journal of Pharmacy and Technology; August 2015; 8[8]:1099.

49. R Archana, Kumar Sai Sailesh , Deepthy Purushothaman , B Udaya Kumar Reddy, J K Mukkadan; Traditional Vestibular Stimulation: A Physiological Intervention for Dysmenorrhea; Journal of Medical Sciences and Health/Sep-Dec 2015/Volume 1/Issue 3; p:31-32.

50. Ms. I. Sigloria and Dr. P.Mangala gowri; Assess the prevalence of self-medication among people in a selected area; International Journal of Pharma and Bio Sciences; 2017 Jan ; 8[1]: [B] 276 – 280.

51. R. Archana, V. Philominal, Shyamala Thirumeni; Effect of acupressure and changes in heart rate variability in dysmenorrhoea; Recent Research in Science and Technology 2011, 3[10]: 01-06.

Effect of Green Tea and Ginger on Cariogenic Bacteria

Rebecca Rachel George[1], Geetha R. V[2], Ravindra Kumar Jain[3]
Graduate Student[1], Assistant Professor[2], Department of Microbiology
Reader[3], Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Dental caries (tooth decay) is one of the common dental problem in most countries, affecting 60-90% of school children and the majority of adults. Reports have brought out the evidence that green tea and ginger have a synergistic impact to limit the growth of bacteria such as E.coli owing to their anti-microbial effects. So the aim of the present study was to evaluate the anti-bacterial activity green tea and ginger alone and also their synergistic activity on Streptococcus mutans, the potent initiator and leading cause of dental caries worldwide. Antibacterial activity of green tea along with ginger was screened against Streptococcus mutans, using agar well diffusion technique. The results of this study showed that both green tea and ginger has got good antibacterial activity. But they didn't exhibit synergistic activity on the organism tested.

Materials and methods: Bacterial strain used was Streptococcus mutans. The organism was isolated. Isolation was done using selective media Mutans-Sanguis agar [Hi media M977], and maintained in nutrient agar slope at 4 in Department of Microbiology, Saveetha Dental College.

Results: Cinnamon and ginger have excellent antimicrobial activity on the growth of Mutans streptococci and lactobacilli, but their effects are more on lactobacilli than mutant streptococci. Cinnamon should be prescribed prior to ginger because it has more antibacterial activity on the growth on both bacteria types even in small concentration.

Conclusion: Green tea contains chemical ingredients such as polyphenols which possess antimicrobial, anti-inflammatory and anti-cariogenic effects. Oral rinsing with green tea reduces the quantity of cariogenic bacteria significantly and also decreases acidity of plaque and saliva as well. It can be used as a cost-effective caries preventive measure in developing countries.

Key Words: *Dental caries, agar well diffusion, synergistic effect, green tea, ginger.*

Introduction

Dental caries is the demineralization of the tooth surface caused by bacteria. [1, 2] The bacterial aspect of dental caries can be explained such that the bacteria produce acids that cause the irreversible solubilization of tooth structure. Streptococcus mutans is the main cause of dental decay while various lactobacilli are associated with progression of the lesion. Reports have brought out the evidence that green tea and ginger have a synergistic impact to limit the growth of bacteria such as E.coli owing to their anti-microbial effects. [3, 4]

The ability of this bacterium to produce (acid genic) and tolerate (acid uric) acids coupled with its property of synthesizing extracellular glucans allows its effective colonization in the oral cavity leading to the establishment of highly cariogenic dental biofilms [5]. Extracellular glucans which are synthesized from sucrose by glucosyltransferases (GTFs) play a critical role in the adhesive interactions of S. mutans and contributes to the structural integrity of dental plaque [6]. S. mutans expresses three different GTFs viz., GTF B, GTF C and GTF D. The insoluble and soluble glucan is mostly synthesized by GTF B and GTC C respectively. However, GTF C is known to synthesize a mixture of soluble and insoluble glucans [7]. These virulence properties thus provide a unique microenvironment for unobstructed survival of S. mutans in the oral cavity [8]. Therefore, approaches to inhibit various factors governing the virulence properties of S. mutans, could be an alternative to prevent dental caries.

There are numerous anti-plaque agents that are known to reduce dental biofilm formation amongst which fluoride is a well-known cariostatic agent [9]. However, its excessive use results in side effects like fluorosis and hence its use is limited [10]. Moreover, chlorhexidine which is considered as a standard anti-plaque agent have also been reported as genotoxic [11]. Therefore, despite of the presence of a variety of anti- plaque agents, the quest for an effective agent still continues. Therefore, the development of alternative therapeutic agents with anticariogenic properties and minimal side effects is a promising approach.

Zingiber officinale (or ginger), is one of the most extensively used herbs in the Indian system of traditional medicine. Many studies have revealed its numerous pharmacological activities, such as, antioxidant, antibacterial, anti-inflammatory, antinociceptive [12], antimutagenic [13] and

hepatoprotective [14]. Furthermore, there are several other studies showing the antibacterial effect of Z. officinale against a number of micro-organisms including S. mutans [15-17]. However, there are hardly any reports evaluating its anticariogenic potential. Hence, in view of the current need of alternative therapeutic approach, we initiated our study to provide an innovative and comprehensive insight of the effect of Z. officinale to inhibit various virulence traits of S. mutans.

Ginger has been known for medicinal purposes since antiquity. Ginger is from the botanical family Zingiberaceae. Its botanical name is Zinger officinale. The parts of the ginger plants that are of medicinal value are the rhizomes (underground stems). Ginger has long been an alternative therapeutic to prevent motion sickness i.e, nausea and vomiting. Its synergistic and individual impact with green tea is found to affect some bacterial populations.

Tea- the most preferred beverage of the century. The beverage tea has its roots in Southwest China, where green tea is not just a beverage but a medicinal drink too. The source of tea isCamellia sinensis[18].Thisaromatic beverage is commonly prepared by pouring hot or boiling water over the cured leaves of this plant. There are many different types of tea, such as white tea, green tea, yellow tea, black tea, oolong tea and pureh tea. The fact is that all these tea types have a common source, the Camellia sinensis. The processing method of the leaves after harvesting decides the type of tea produced.The different processing methods impart characteristic colour and flavour to the tea leaves. For our study, we chose three tea types, namely green tea, red tea and black tea respectively.

Green tea, an aqueous infusion of dried leaves of the plant Camellia sinesis, is the most popular beverage consumed worldwide [19]. It has received considered attention because of its scientifically proved beneficial effects on human health. The effect has been largely attributed to the poly phenol content namely epigallocatechin gallate which is known to induce apoptosis of tumor cells and has little or nil effect in normal cells [20, 21]. It is consumed throughout the ages in India, China, Japan and Thailand. Several epidemiological studies have proven that green tea also has some general health beneficial properties like anti- hypertensive, anti-bacterial, anti-viral and anti- fungal activity. Traditional Chinese medicine has recommended this plant for headache, body ache, and as an energy drink. It has cognitive function and positive impact on bone density, dental caries and periodontal health [22]. Green tea is usually available in the form

of beverage, mouth wash containing extract of green tea, and as a chewing gum. Green tea catching can be affected by various factors like type of green tea leaves, type of processing before drying, growing conditions and the geographic conditions. This present article reviews the effectiveness of green tea on oral health and periodontium.

Green tea is the most ancient and popular beverage consumed around the world it is made up of leaves of the plant camellia sinensis. It is loaded with polyphenols like flavonoids and catechins, which function as powerful antioxidants. These substances can reduce the formation of free radicals in the body, protecting cells and molecules from damage. These free radicals are known to play a role in aging and all sorts of diseases. One of the more powerful compounds in green tea is the antioxidant Epigallocatechin Gallate (EGCG), which has been studied to treat various diseases and may be one of the main reasons green tea has such powerful medicinal properties. Green tea also has small amounts of minerals that are important for health. Green tea can boost the metabolic rate in the short term, it makes sense that it could help you lose weight. Green tea contains many bioactive ingredients mainly polyphenols which play a key role in the prevention and treatment of many diseases the catechins in green tea may inhibit the growth of bacteria this can lower the risk of infections and lead to improvements in dental health, a lower risk of caries and reduced bad breath (halitosis) [23]

Green tea (Camellia assamica) is cultivated in many countries of the countries of the world, Japan being the largest producer in the world. Green tea has been chosen as a raw material for extracts used in different beverages, dietary supplements and cosmetic items.Green tea is derived from the dried leaves of the plant Camellia sinensis. Green tea is usually available in the form of beverage, mouth wash containing extract of green tea, and as a chewing gum. Green tea catching can be altered by different factors like type of green tea leaves, type of processing before drying, growing conditions and the geographic conditions. [24]Significant antibacterial activity has been reported for extracts of green tea. Green tea is observed to inhibit the growth of bacteria such as Streptococcus mutans and Lactobacillus acidophilus.

Materials and Methods

Test Microorganisms

Bacterial strain used was Streptococcus mutans. The organism was isolated. Isolation was done using selective media Mutans-Sanguis agar [Hi media M977], and maintained in nutrient agar slope at 4 in Department of Microbiology, Saveetha Dental College.

Methodology

The green tea and ginger were dissolved in distilled water in following concentrations 2.5mg/ml, 5mg/ml and 10mg/ml so that 100µl delivers 250µg/ml, 500µg/ml and 1000 µg/ml respectively.

Screening of antibacterial activity [Agar well diffusion technique]

Broth culture of the specified bacterial strain according to Mac Farmland's standard [24-26] 0.5 was prepared. On Muller Hinton agar [MHA-Hi media M1084] plate's lawn culture of the test organisms was made using sterile cotton swab and plates were dried for 15 minutes. Using a sterile well cutter wells measuring 4mm depth were made on the agar plates. 100 µl of different concentration of the extract were then filled in the wells. To determine the synergistic activity 50µl of green tea and 50µl of zinger extract from different concentrations were loaded on to the wells. 0.2% chlorhexidine was used as the positive control. The plates were incubated at 37°C overnight and the zone of inhibition of growth was measured in millimeters. All the tests were done in triplicate to minimize the test error.

Results and Discussion

The antibacterial activity of green tea and ginger at different concentrations was screened by agar well diffusion technique and zone of inhibition was measured in mm diameter. The results are given in Table 1. The green tea alone was more effective against Streptococcus mutans with a zone of inhibition of 22mm; ginger showed a zone of 19mm diameter and with both green tea and ginger the zone diameter was 20 mm.

The present study was to analyze the antibacterial activity of green tea and ginger alone and together on caries causing organisms. The results obtained from our study show that green tea and ginger have good antibacterial activity but do not have much of a synergistic impact against Streptococcus mutans.

There is convincing evidence that the different components of catechins have their action on different species of microorganisms responsible for caries production.

Taking two cups of green tea as a daily routine is good for the health as the results show that intake of green tea which contains catechins like epigallocatechin-3-gallate and polyphenols which show negative effect to the growth of bacteria and inhibit the growth of bacteria in the oral cavity which reduces the oral bacterial count and improves the oral health care so green tea plays a key role in prevention and treatment of many diseases.

Roopal V Patel demonstrated that combined extracts of ginger and honey showed maximum inhibitory concentration against S.mutans and S.aureus and were least effective against L.acidophillus in comparison with gentamycin, showing, there is a significant synergistic effect of antimicrobial activity from the combination of ginger and honey, against isolates from carious teeth [27].

Cinnamon and ginger have excellent antimicrobial activity on the growth of Mutans streptococci and lactobacilli, but their effects are more on lactobacilli than mutant streptococci [28]. Cinnamon should be prescribed prior to ginger because it has more antibacterial activity on the growth on both bacteria types even in small concentration (50mg/ml) [29].

Table 1:Anti-bacterial activity of Green Tea and Ginger

Organism	Conc. ml	E1 mm	E2 mm	E3 mm	Chlorhexidine mm
Streptococcus mutans	250µg/ml	14	08	10	24
	500µg/ml	17	12	14	27
	1000µg/ml	22	19	20	34

E1 – Green Tea, E2 – Ginger, E3 – Green Tea + Ginger

Conclusion

Green tea contains chemical ingredients such as polyphenols which possess antimicrobial, anti-inflammatory and anti-cariogenic effects. Oral rinsing with green tea reduces the quantity of cariogenic bacteria significantly and also decreases acidity of plaque and saliva as well. It can be used as a cost-effective caries preventive measure in developing countries. Ginger has been used as an herbal ingredient in dental products as well [30]. Together these two do not seem to significant synergistic effect.

Corresponding Author

Ravindra Kumar Jain

Reader, Department of Orthodontics

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Telephone Number: +91 9884729660

E-mail id.: orthoravi2@gmail.com

References

1. Hardier JM (May 1982). "The microbiology of dental caries". Dent Update 9 (4): 199–200, 202–4, 206–8
2. Enzo A. Palombo., Traditional Medicinal Plant Extracts and Natural Products with Activity against Oral Bacteria: Potential Application in the Prevention and Treatment of Oral Diseases. Review article. Evidence – Based complementary and Alternative Medicine Vol 2011,Article ID680354,15 pages
3. Chaudhari LK, Jawale BA, Sharma S, Sharma H, Kumar CD, Kulkarni PA., Antimicrobial activity of commercially available essential oils against Streptococcus mutans. J Contemp Dent Pract. 2012 Jan 1; 13(1):71-4.

4. Jeena K, Liju VB, Kuttan R: Antioxidant, anti-inflammatory and antinociceptive activities of essential oil from ginger. Indian J Physiol Pharmacol 2013, 57:51–62.

5. Marsh PD, Bradshaw DJ: Dental plaque as a biofilm. J Ind Microbiol1995, 15:169–75.

6. Kuramitsu H: Virulence factors of mutans streptococci: role of molecular genetics. Crit Rev Oral Biol Med 1993, 4:159–176.

7. Koo H, Hayacibara MF, Schobel BD, Cury JA, Rosalen PL, Park YK, Vacca-Smith AM, Bowen WH: Inhibition of Streptococcus mutans biofilm accumulation and polysaccharide production by apigenin and tt-farnesol. J Antimicrob Chemother 2003, 52:782–789.

8. Bowden GH, Hamilton IR: Survival of oral bacteria. Crit Rev Oral Biol Med 1998, 9:54–85.

9. Featherstone JDB: Remineralization, the natural caries repair process-the need for new approaches. Adv Dent Res 2009, 21:4–7.

10. Featherstone JDB: Prevention and reversal of dental caries: role of low level fluoride. Community Dent Oral Epidemiol 1999, 27:31–40.

11. Ribeiro DA, Bazo AP, da Silva Franchi CA, Marques ME, and Salvadori DM: Chlorohexidine induces DNA damage in rat peripheral leukocytes and oral mucosal cells. J Periodontal Res 2004, 39:358–61.

12. Jeena K, Liju VB, Kuttan R: Antioxidant, anti-inflammatory and antinociceptive activities of essential oil from ginger. Indian J Physiol Pharmacol 2013, 57:51–62.

13. Jeena K, Liju VB, Viswanathan R, Kuttan R: Antimutagenic Potential and Modulation of Carcinogen-Metabolizing Enzymes by Ginger Essential Oil. Phytother Res 2013. doi:10.1002/ptr.5064.

14. Abdel-Azeem AS, Hegazy AM, Ibrahim KS, Farrag AR, El-Sayed EM: Hepatoprotective, Antioxidant, and Ameliorative Effects of Ginger (Zingiber officinale Roscoe) and Vitamin E in Acetaminophen Treated Rats. J Diet Suppl 2013, 10:195–209.

15. Khan R, Zakir M, Afaq SH, Latif A, and Khan AU: Activity of solvent extracts of Prosopis spicigera, Zingiber officinale and Trachyspermum ammi against multidrug resistant bacterial and fungal strains. J Infect Dev Ctries 2010, 4:292–300.

16. Fatima S, Farooqi AHA, Kumar R, Kumar TRS, Khanuja SPS: Antibacterial activity possessed by medicinal plants used in tooth powders. J Med Arom Pl Sci 2000, 22:187–189.

17. Islam TH, Azad AHB, Akter S, Datta S: Antimicrobial activity of medicinal plants on Streptococcus mutans , A Causing Agent Of Dental Caries. J Eng Res Tech 2012, 1:121–126.

18. Effect of different types of tea on Streptococcus mutans: An in vitro study Priya Subramaniam, Uma Eswara, Maheshwar Reddy KR, Indian Journal of Dental Research, 23(1), 2012.

19. Graham, H. N. 1992. Green tea composition, consumption, and polyphenol chemistry. Prev. Med. 21:334-350.

20. Ahmad N, Gupta S, Mukhtar H. Green tea polyphenol epigallocatechin-3-gallate differentially modulates nuclear factor KappaB in cancer cells versus normal cells. Arch Biochem Biophysics. 2000; 376:338–46.

21. Chen ZP, Schell JB, Ho CT, Chen KY. Green tea epigallocatechin gallate shows a pronounced growth inhibitory effect on cancerous cells but not on their normal counterparts. Cancer Lett. 1998; 129:173– 9.

22. McKay DL, Blumberg JB. The role of tea in human health: an update. J Am Coll Nutr. 2002; 21:1–13.

23. Abhishek N. Study of Correlation of Green Tea and its Antioxidant Effect in Reducing Oral Bacterial Count. J. Pharm. Sci. & Res. Vol. 8(6), 2016, 517-519

24. Collins,CH and Lyne, P.M 1976.Microbiological methods, London, Butterworths and co.288p

25. Betty A.Forbes., Daniel F.Sahm., Alice S.Weissfeld. Bailey & Scott's Diagnostic Microbiology 11th edition Mosby page 229 – 257

26. Connie R.Mahon., George Manuselis., Saunder's Diagnostic Microbiology 2nd Ed

27. Jyotsna Srinath, Lakshmi. T. application of spices in dentistry- a literature review.Int. J. Drug Dev. & Res.; April - June 2014; Vol. 6; Issue 2; ISSN 0975-9344.

28. Patel, Roopal V., Vidhi T. Thaker, and V. K. Patel. "Antimicrobial activity of ginger and honey on isolates of extracted carious teeth during orthodontic treatment."Asian Pacific Journal ofTropical Biomedicine 1.1 (2011): S58-S61.

29. Ghada Al-Duboni, Muhamed T Osman, Redhwan Al-Naggar Antimicrobial Activity of Aqueous Extracts of Cinnamon and Ginger on Two Oral Pathogens Causing Dental CariesResearch Journal of Pharmaceutical, Biological and Chemical Sciences 07/2013; 4(3):957-965.

30. Omar OM (2013) Alternative Medicine: Implications on Dentistry. Altern Integ Med 1:e103. doi:10.4172/2327-5162.1000e103

Effect of Herbal and Fluoride Toothpaste on Dental Caries

Priyadharshni S[1], Geetha[2], Roja[3]
Graduate Student[1], Senior Lecturer[2], Department of Microbiology,
Tutor[3], Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University,Chennai, India

Abstract

Introduction: The aim of this present study is to compare the effect of herbal and fluoride toothpaste on the bacteria causing dental caries.The effect of the herbal toothpaste and fluoride toothpaste are compared and hence an idea on how to prevent, control and treat the dental caries is formulated.Lactobacilli acidophilus have been associated with the presence and progression of dental caries.Production of organic acids by lactobacilli is considered to be important in causing decalcification of the dentinal matrix.

Material and Methods: One herbal toothpaste and fluoride based toothpaste are used for the study. The effect of these two toothpastes on bacteria causing dental caries like lactobacilli will be studied by agar will diffusion technique. The findings are compared between the groups.

Results: The results suggested that Herbal toothpaste had maximum antimicrobial activity at all concentrations when compared to Fluoride toothpaste.The higher antimicrobial activity of herbal toothpaste could be attributed to the presence of different components like Lavang and Elaichi.

Conclusion: The study showed dentifrice with combination of herbs had better effect in inhibiting cariogenic bacteria compared to the ones with fewer herbal ingredients.

Key Words:*Dental caries, fluoride, herbal, toothpaste, lactobacillus.*

Introduction

Toothpastes are recognised as the best source of fluoride, which most effectively protects both deciduous and permanent teeth from caries. The cleaning abilities of toothpaste provided by abrasives, the antibacterial qualities, which, in turn, are provided by a variety of substances with different abilities to inhibit the growth of germs in the oral cavity.The purpose of oral hygiene using toothpaste is to reduce oral bacterial flora [1].Toothpaste is by far the most commonly used mode of delivery of fluoride and the decline in the prevalence of dental caries in developed countries is mainly attributed to its increased use [2,3].The advantages of fluoridated products is well documented in the literature. It is the most spoken and debated topic all over the world with researchers having diverse views about its use and safety.The intensive promotion of fluoridated toothpastes by the oral health care industry is a major factor in their increased use.The other alternative toothpaste on the market for use by infants, do not contain fluoride in their composition, but have herbal products or enzymes to produce antiseptic or antimicrobial effects against cariogenic and opportunistic micro-organisms in the oral environment [4]. The components of natural toothpastes should be as safe as those of conventional toothpaste provides safe as those of conventional toothpastes, provide efficient,protection against carcinogenic microorganisms [5].This study is thus aimed at comparing the antimicrobial efficacy of triclosan containing fluoridated toothpaste and Herbal tooth paste.

WHO declares that deprived oral health and its related diseases may have dreadful effect on common health as well as eminence of life. Dental caries is a common and major public health oral disease which hampers the attainment and protection of oral health in different age groups. The prevalence pattern and severity of dental caries varies with age, sex, race, socio-demographic characteristics, economic status, geographical location, food practice and oral hygiene habits within the same country or region in various parts of the world.

During the last few decades, the incidence of microbial diseases has amplified drastically. Microorganisms are the super bug agent responsible for causing dental caries. Many facultatively and obligately anaerobic bacteria dominate the microbial community of dental caries. But, the most important etiological agent of dental caries is Streptococcus mutans. Tooth decay takes place when a vulnerable tooth surface is colonized with cariogenic microbes and dietary source of sucrose or re ned sugar. Fermentation of carbohydrate leads to production of lactic acid by the action of bacteria which melts the hydroxyapatite crystal

structure of the tooth which grounds caries [6].

The experience of pain, problem with eating, chewing, smiling and communication due to missing, discoloured or spoiled teeth have a foremost impact on people's everyday life. Low self-esteem, adverse pregnancy outcomes, increased risk of myocardial infarction, cardiovascular disease, respiratory, erectile, diabetes complications, cavernous sinus thrombosis and Ludwig angina are associated with dental caries which can be crucial to human beings. Moreover, oral diseases hamper activities at school, at occupation and at residence causing millions of school and employment hours to be vanished each year throughout the globe. As a consequence, the treatment need is enhanced nowadays.

Globally, there is a great interest in the use of antimicrobial agents for prevention and treatment of dental diseases. The emergence of antibiotic resistance has been identified due to inappropriate prescription and practice of antibiotic use. In the recent years, dentists and dental professionals has shifted their interest from narrow spectrum antibiotic prescriptions to broad-spectrum aminopenicillinsdue to enhancement of bacterial isolates resistant to the former antibiotics in prescription practices. Antibiotic resistance increases the morbidity associated with dental caries and contributes substantially to rising costs of care and the need for more expensive drugs [7].

Among the plethora of diseases manifesting in the oral cavity dental caries remains one of the most prevalent diseases of this site. Even with improved means to maintain oral hygiene dental caries occurrence is still on the rise. This is predominantly due to changing dietary habits and increased consumption of sugar [8]. Also factors like geographic, climate, cultural, ethnic and socioeconomic conditions also play an important role. The global distribution of dental caries has shown distinctive variations [9]. The scenario in India also shows similarities with other developing countries. Prevalence studies on dental caries in India have shown a results ranging from 31.5% to 89% [10].

Periodontal diseases and dental caries are the most prevalent oral infections affecting mankind worldwide. Endogenous oral bacterial species such as A.actinomycetemcomitans, Porphyromonas gingivalis, Streptococcus mutans, Streptococcus sorbicus, Bacteroides sp.,Prevotellamsp.,Fusobacterium sp., and their metabolites play major role in the initiation and progression of these infections. Effective prevention of these infections can be achieved

by mechanical removal of Dental plaque by proper tooth brushing and flossing. However, the majority of the population, particularly aged individuals, may not perform mechanical plaque removal sufficiently, and thus antimicrobial mouth rinses such as triclosan and chlorhexidine may be used to limit these two plaque-related oral infections. These chemical agents used in the form of either dentrifrices or mouth rinses may have undesirable side effects such as tooth staining, taste alteration and development of hypersensitivity reactions. Although antibiotics are used routinely to prevent systemic infections originating from the oral cavity, they are not recommended for regular prevention of dental plaque formation because of the risk.

It is well established that most infectious oral disease such as dental caries and periodontal disease are linked to the microbial flora of the oral cavity. Dental caries is caused by acidogenic species of bacteria, mainly S.mutans, Lactobacillus and Actinomyces. These oral bacterial species metabolise sucrose to lactic and other organic acids in dental plaque produced on the surface of the tooth and dissolve calcium phosphate in the enamel, consequently giving rise to dental caries. Periodontal disease is mostly associated with anaerobic gram-negative rods such as A.actinomycetemcomitans, P.gingivalis, Tannerellaforsythus, Bacteroides, prevotella and Fusobacterium species. These periodontopathogens are frequently isolated from periodontal pockets of patients with periodontitis.

Materials and Methods

Toothpastes

Three toothpastes namely fluoride toothpaste A , a Herbal Toothpaste B and a herbal Toothpaste C were selected for this study. The products were collected from local market, Chennai.

Tests for Microorganisms

Bacterial strain was lactobacilli which is a fermentativebacteria and considered as a probiotic. Lactobacillus acidophilus was obtained from Microbiology Lab, Saveetha Dental College,Chennai.

Methodology

The antimicrobial activity of different concentrations of the dentifrices was determined by modified agar well diffusion method. Lawn culture of the test organisms were made on the Muller Hinton agar [MHA-Hi media M1084] plates using sterile cotton swab and the plates were dried for 15 minutes. Dentifrice dilutions at different concentrations were introduced into each of the three wells. The uninoculated plates were maintained as a control. The plates were incubated at 37o C for 24 hours for isolation of bacteria.The antimicrobial activity was evaluated by measuring the diameter of zones of inhibition (mm).

Result

The antibacterial activity of the three toothpastes at different concentrations was screened by modified well diffusion technique and the zone of inhibition was measured in mm diameter. The herbal toothpaste B was more effective against lactobacilli with a zone of inhibition of 29mm diameter, fluoride toothpaste A showed a zone of 25mm diameter and with the herbal toothpaste C the zone diameter was 23mm.Thus, One herbal tooth paste with multiple herbal ingredients had greater zone of inhibition compared to the fluoride containing tooth paste and other herbal toothpaste. The results are given in Table1.

Table 1: Zone of inhibition of three toothpastes(mm)

Organism	Fluoride toothpaste A	Herbal toothpaste B	Herbal toothpaste C	Control chlorhexidine
Lactobacillus acidophilus	25mm	29mm	23mm	29mm

Discussion

Good oral hygiene is necessary for the healthy teeth, gum and fresh breath. Fluoride and plant extracts incorporated in toothpastes help to protect teeth by binding with enamel to make it stronger [11]. Most of the people use toothpaste without knowing their potential efficacy, bacteria form an important group of micro-organisms found in both healthy and diseased mouths. It is of importance to know the role of plants play in

oral hygiene as a number of them have medicinal properties.Natural toothpastes are those without triclosan or fluoride (herbal toothpaste). They usually contain natural ingredients such as special mineral salts like Sodium fluoride and sodium chloride, and plant extracts like lemon, eucalyptus, rosemary, chamomile, sage and myrrh. There are toothpastes and other remedies that help kill and prevent bacteria in people's mouths[12]. Dental caries is a multifactorial disease in which, streptococci mutans and lactobacilli in dental plaque, play an important role. Lactobacillus species and other non-mutans acidogenic and acid-tolerant bacteria are also known as significant etiologic agents [13]. Furthermore, few research efforts have been directed toward addressing the potency or quality of herbal ingredients used in these dental products. It is generally agreed that, with the exclusion of fluoride from most natural herbal dentifrices, these dentifrices usually forfeit caries-preventive benefits [14]. While many herbal dentifrices claim to have antimicrobial properties and have caries preventive action, very little research has been conducted to investigate these claims.Results indicate that both herbal toothpaste and fluoride containing toothpaste were effective against both the cariogenic bacteria. The zone of inhibition when compared, the herbal dentifrices were comparatively better than the fluoride containing tooth pastes. Chemicals, mainly triclosan and chlorhexidine, have been added in mouth rinses and dentifrices to prevent plaque and gingivitis. But some of these substances show undesirable side effects such as tooth staining and altered taste. This had led to paying increased attention on using natural ingredients in herbal dentifrices. Herbal ingredients have several benefits; chamomile has anti-inflammatory effect, echinacea has immune stimulatory property, sage and rhatany have anti-hemorrhagic properties, myrrh is a natural antiseptic, and peppermint oil has analgesic, antiseptic, and anti-inflammatory properties. There are limited studies available regarding the efficacy of herbal dentifrices; hence, the present study was undertaken to assess their plaque and gingival preventive action.

Our study mainly concentrates in comparing efficacy of herbal dentifrices with conventional ones in inhibiting cariogenic bacteria, suggesting that efficacy of herbal dentifrices is similar and some have superior antibacterial efficacy [15]. Herbal toothpaste is available in most rural areas of the poor countries. It does not need expertise or any extra resources to manufacture it. Thus it appears to be a potent antimicrobial agent that could be considered as a medicinal plant. Thus provides evidence to the claim of anti-cariogenecity of herbal dentifrices[16]. The study also

showed dentifrice with combination of herbs had better effect in inhibiting cariogenic bacteria compared to the ones with fewer herbal ingredients[17].

Dental plaque deposit on teeth is a concern for both cosmetic and its pathogenic nature. Presence of plaque may be the culprit for dental caries, gingivitis, periodontal problems, and halitosis. Many mechanical aids are used worldwide to remove or control plaque, including tooth brushes, dental floss, mouth rinses, and dentifrices.Mechanical plaque removal is one of the most accepted methods of controlling plaque and gingivitis. But it is expected that less than one-third of the population can effectively perform mechanical plaque removal. Several chemical preventive agents have beneficial effects in the control of plaque and to reduce or prevent oral disease. Hence, various chemical formulations were tried in dentifrices[18]. Chemicals, mainly triclosan and chlorhexidine, have been added in mouth rinses and dentifrices to prevent plaque and gingivitis. But some of these substances show undesirable side effects such as tooth staining and altered taste[14]. This had led to paying increased attention on using natural ingredients in herbal dentifrices. Herbal ingredients have several benefits; chamomile has anti-inflammatory effect, echinacea has immune stimulatory property, sage and rhatany have anti-hemorrhagic properties, myrrh is a natural antiseptic, and peppermint oil has analgesic, antiseptic, and anti-inflammatory properties[19].This study has proved that herbal dentifrices do not cause any adverse effects on the oral cavity and are effective in reduction of plaque and gingivitis, as that of fluoridated non-herbal dentifrice. Several studies have proven the medicinal values of herbal products. Hence, medicated herbal toothpastes can be safely used to control plaque and gingivitis. Further long-term studies are required to prove their effectiveness[20].

During recent years, many researchers throughout the world have studied Miswak as a helpful plant in oral hygiene. Clinical trials have shown that regular use of chewing stick of Salvadora Persica reduces plaque. It has been reported that incidence of caries among users of chewing sticks is low despite the intake of a carbohydrate rich diet and a lack of modern dental prophylactic measures[21].The Arabian researchers concluded from a comprehensive survey of several thousands of Saudi school children that the low incidence of gingival inflammation was attributable to the practice of using Miswak for teeth cleaning (Gazi et al 1992).

In vitro studies indicate that, of a variety of common oral bacteria, members of the genus streptococcus (including the mutans streptococci) are especially sensitive to the

antimicrobial activities of S. Persica (Al-lafi&Ababneh 1995).

In a study the efficacy of Miswak in the prevention of dental caries has been investigated and compared with the efficacy of ordinary toothbrush and toothpaste[18]. The data collected at the end of the study showed that the risk of dental caries for each tooth in the control group was 9.35 times more than the case group (Aldini&Ardakani 2007).

It has been told rinsing with Miswak extract stimulated parotid gland secretion and raised the plaque PH, suggesting a potential role in caries prevention (Sofrata et al 2007).

It has been observed that miswak was as effective as a toothbrush for reducing plaque on buccal surfaces of teeth both experimentally and clinically (Mohammed et al 2006).

Another study compared the oral health efficacy of persica mouthwash with that of a placebo. The results showed that use of persica mouthwash improves gingival health and lower carriage rate of cariogenic bacteria when compared with the pretreatment values (Khalessi et al 2004).

Scientific evaluation of use of miswak revealed that it is at least as effective as toothbrushing for reducing plaque and gingivitis and that the antimicrobial effect of S. persica is beneficial for prevention/treatment of periodontal diseases (Al- Otaibi 2004).

A clinical study was conducted using patients` saliva and measuring the effect of miswak (chewing stick), miswak extract, toothbrush, and normal saline on mutans and lactobacilli[19]. The results showed that there was a marked reduction in Strep. Mutans among all groups.When the groups were compared, the reduction in Strep. Mutans was significantly greater using miswak in comparison to toothbrushing and there was no significant differences for lactobacilli reduction[21]. The investigators concluded that miswak has an immediate antimicrobial effect. Strep. Mutans were more susceptible to miswak antimicrobial activity than lactobacilli (Almas & Al-Zeid 2004).

Conclusion

Lactobacilli acidophilus were quantitatively assessed by the presence or absence of inhibition zone and their zones diameter. Among this, herbal toothpastes showed the highest resistance capacity. The herbal tooth pastes have similar and slightly better antibacterial activity compared to the conventional tooth pastes. The herbal tooth pastes which have combination of herbs have better antimicrobial activity compared to those with single ingredient against certain organisms. Thus turning to nature can be as good as the latest

advances. These traditional methods have numerous other claims also which are of great benefit for health. Exploring and harvesting these herbal products and blending the same with the advances may make oral and health care better. Thus the present article also provides insight to the dentists that the herbal toothpastes are equally effective as the conventional toothpastes and can be advised to those who prefer natural products.

Corresponding Author

E. Roja,

Reader, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai – 600 077, Tamil Nadu, India

Email Id: itsrojaa@gmail.com

Contact Number: 91 99941 74480.

References

1. Yihong Li1, Silvia Argimón, Catherine N. Schön,PrakaimukSaraithong, Page W. Caufield.Characterising Diversity of Lactobacilli Associated with Severe Early Childhood Caries: A Study ProtocolAdvances in Microbiology, 2015, 5, 9-20 Published Online January 2015 in SciRes.
2. Sarmad Ghazi Mohammed.Comparative study of invitro antibacterial activity of miswak extracts and different toothpastes.American Journal of Agricultural and Biological Sciences, 2013, 8 (1), 82-88 ISSN: 1557-4989:2013 Science Publication.
3. Mohan Kumar K P1, Priya N K2, Madhushankari G S.Anti Cariogenic Efficacy of Herbal and Conventional Tooth Pastes - A Comparative In-Vitro Study.Journal of International Oral Health. Mar-Apr 2013; 5(2):8-13.
4. Maria Kurian, Geetha.R.V Effect Of Herbal And Fluoride Toothpaste On Streptococcus Mutans - A Comparative Study /J. Pharm. Sci. & Res. Vol. 7(10), 2015, 864-865.

5. Sanchana, V.B.Awareness of fluoride in toothpaste among patients.International Journal of Recent Advances in Multidisciplinary Research Vol. 03, Issue 08, pp.1683-1686, August, 2016 .
6. AnushaRajagopalan,Herbal Products in Oral Hygiene Maintenance –A Review.IOSR Journal Of Pharmacy Volume 5, Issue 1 (January 2015), PP. 48-51.
7. GemimaaHemagaran, PrasannaNeelakantan,Remineralization of the Tooth Structure - The Future of Dentistry. International Journal of PharmTech Research CODEN (USA): IJPRIF ISSN : 0974-4304 Vol.6, No.2, pp 487-493, April-June 2014.
8. Shourie KL. Dental caries in Indian children. Ind J Medical Res, 1941; 29: 709-21.
9. WafaK.M.Talha, ManalElsaid, Ola M. Omar and Somaiya A. Eissa. The Effect ofMiswak and Fluoride Toothpastes on Dental Plaque, A Comparative Clinical and Microbiological Study.Nature and Science 2013;11(9).
10. DhruwChandrabhan, RajmaniHemlata, Bhatt Renu, Verma Pradeepisolation of Dental Caries Bacteria from Dental Plaque and Effect of Tooth Pastes on Acidogenic Bacteria.Open Journal of Medical Microbiology, 2012, 2, 65-69.
11. C. S. Toi, R. Mogodiri and P. E. Cleaton-Jones.Mutans streptococci and lactobacilli on healthy and carious teeth in the same mouth of children with and without dental caries.Microbial Ecology in Health and Disease 2000; 12: 35±41.
12. Rahul R.Deshpande,priyankakachare,gautamisharangpani,VivianK.varghesesneha S bahulkar. Comparative evaluation of antimicrobial efficacy of two commercially available dentrifices(fluoridated and herbal) against salivary microflora.International Journal of Pharmacy and Pharmaceutical Sciences ISSN- 0975-1491Vol 6, Issue 6, 2014.
13. Dr. TorwaneNileshArjun, Dr. AbhishekGouraha. Ashish Maheshwari, Dr. KalpeshChavan, Efficacy of herbal dentrifice in the reduction of dental caries compared against commercially available fluoride containing dentrifice:world journal of pharmacy and pharmaceutical sciences.vol4issue4.800-807.2015.
14. Barnes VM, Richter R, DeVizio W. Comparision of the short-term antiplaque/antibacterial efficacy of two commercial dentifrices. J Clin Dent. 2010;21:101–4.

15. George J, Hegde S, Rajesh KS, Kumar A. The efficacy of a herbal-based toothpaste in the control of plaque and gingivitis: A clinic-biochemical study. Indian J Dent Res. 2009;20:480–2.
16. de Oliveira SM, Torres TC, Pereira SL, Mota OM, Carlos MX. Effect of dentifrice containing Aloe vera on plaque and gingivitis control. A double-blind clinical study in humans. J Appl Oral Sci. 2008;16:293–6.
17. Radafshar G, Mahboob F, Kazemnejad E. A study to assess the plaque inhibitory action of herbal-based toothpaste: A double blind controlled clinical trial. J Med Plants Res. 2010;4:1182–6.
18. Sorna KH, Rajasekar T, Anandhi A, Shahanas B. A Comparative study of in vitro antibacterial activity of Neem and miswak extracts against isolated cariogens from dental caries patients. J Chem Pharm Res, 2011; 3: 638-45.
19. Wu-Yuan CD, Green L, Birch WX. In vitro screening of Chinese medicinal toothpastes: their effects on growth and plaque formation of mutans streptococci. Caries Res 1990;24:198-02.
20. Prasanth M. Antimicrobial Efficacy of Different Toothpastes and Mouth rinses: An in Vitro Study. Dent Res J 2011 Spring; 8(2): 85-94.
21. TheanH,WongML,Koh H(2007) The dental awareness of nursing home staff in Singapore- a pilot study. Gerodontology 24:58-63.
22. Yadav K,Prakash S(2017) Dental caries: A review. Asian J of Biomed and pharmaceutical sci 6:1-7.

Estimation of Copper and Zinc Levels in Oral Cancer Patients

LeyaMeriamMathew [1], Savitha.G [2], ShanthaSundari [3]

Graduate Student [1],Lecturer [2],Department of Biochemistry

Professor and Head 3,Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Background: Prevalence of oral cancer is high in India, other Asian countries, Brazil and France. Micro nutrients like copper and zinc are increasingly recognized for their role during the onset and progression of pre malignant and malignant orallesions. The aim of this study is to determine serum levels of copper and zinc among oral cancer patients**.** The objective is to estimate and compare the serum levels of copper and zinc in healthy and cancer patients and to derive Cu/Zn ratio, to establish a possible relationship between micro nutrients and oral cancer.

Materials and Methods:25 patients clinically diagnosed with oral cancer (study group)and 25 healthy individuals (control group)in the age group of 30-50 years were included in the study. Blood samples were collected and serum copper and zinc valuesfor both cancer and control group wereestimated using ERBA CHEM 5 semi auto analyzer. Mean and standard deviation of serum copper and zinc values for both groups weredetermined and compared. .

Results: Significantly higher values ($p < 0.04$) were noted for serum copper level (mean value249.39± 58.3µg/dl) for study group compared to control group (123.95 ± 16.2µg/dl).However there was no significant difference in serum zinc values between the groups.

Cu /Zn ratio of 1.43 and 2.86 for control and study group respectively was derived from the estimated value

Conclusion: Mean serum copper value in study group was significantly higher than in the control groupand may be considered as a biological marker for oral cancers.

Keywords*: Serum micronutrients, serum zinc, serum copper, premalignant, malignant oral lesion*

Introduction

Oral cancer is one of the most prevalent and 6th most common type of cancer in the world [1]. Prevalence of oral cancer is higher in, India, Asia Brazil and France compared to other countries globally.Incidence and distribution varies by age, ethnic group, culture and lifestyle associated factors [2]. Alcohol, viruses, genetic mechanisms, Candida and chronic irritation have modifying effects on the etiology of oral cancer .Tobacco can cause genetic damage and can lead to development of oral cancer. Smoking and alcoholconsumption are major risk factors [3]. It mostly affects the tongue,buccalmucosa, gingiva, lips, and palate [4].It was also found that higher intake of fruits and vegetables had lower risk of oral cancer but an unbalanced diet has been related to an elevated risk [5]. Clinical and histopathologicalexamination of oral lesions plays a major role in the diagnosis of oral cancer. In addition, it has been reported that levels of trace elements or micro nutrients such as copper, zinc, iron and selenium are altered in serum, plasma and tissues of pre malignant oral lesions [2] [6].

Copper, zinc, iron are chemical elements required in minute quantities for vital biochemical reactions such as free radical formation and cellular homeostasis[2].The enzymes of trace elements are an important part of certain biological and chemical reactions [7].Zinc is also an important constituent ofbiologicalbio membranes and contributes to membrane stability.It modulates activities of ribonucleic acid and deoxyribonucleic acid polymerase enzymes [8, 9].Theimportance of zinc for a healthy immune system, cell division, skin,hair, and muscle growth needs no mention.

Copper is involved in the release of energy during cell formation of red blood cells, collagen production, and iron absorption.

However, it was found that there is a potential link between trace elements and carcinogenesis. The ratio of copper to zinc is also believed to be a reliable biomarker in the development and progression towards malignancy [10].Copper and zinc are often recognized for their possible role in the prevention and modulation of diseases but recent studies have proved increased level of copper and decreased level of zinc in cancer [11].

The present study was undertaken to evaluate and compare the levels of copper and zinc in the blood serum ofnormaland patients with oral cancer.

Materials and Methods

The sample for thepresent studyconsistedof 50 individuals.25 healthy individuals from the outpatient clinic accepted to participate in the study as controls. 25 patients diagnosed with oralcancerwereincludedastheobservationalstudy group from the outpatient department, Oral Cancer Institute of Saveetha dental college. The study wasapproved by the Institutional Ethical Committee and informed consent was obtained from the patients prior to the blood sample collection. The participants were chosen based on thefollowing inclusion andexclusion criteria. Inclusion criteria included patients diagnosed with carcinoma, in the age group 30 – 50 years, and for control group, healthy individuals with no history of systemic disorders were chosen.Exclusioncriteria included immunocompromised adults, patients diagnosed with endocrine disorder, coronary artery disease andinfectious diseases like tuberculosis orsyphilis.Methodology included collection of 5ml of blood sample under absolute asepsis and transferred to acid washed test tubes .The blood collected was centrifuged at 2500rpm for 15 minutes and preserved in a frozen state untilanalysis. The samples were then analyzed using ERBA CHEM 5 semi auto analyzer. Serum levels of zinc and copper were assessed with the help of reagentsprovided.

The data obtained, was statistically analyzed using the SPSS software. Range of values of all the samples for serum copper and zinc were evaluated and grouped(Tables, II). Mean and standard deviation for serum copper and zincof both the groups were determined and compared for statistical significance $p<0.05$ (Table III). Cu/Zn ratio was then derived from the above estimated value.

Results

Analysis of the data revealedthat serum zinc values ranged from 60µg/dl to 119µg/dl in both the groups. (Table I).However a maximum of 8 samples was in the range of 100- 109µg/dl in controls, and a maximum of 7 samples were in the range of 90 - 100µg/dl in the study group. The mean value for serum zinc in control group was 86.64±17.4 µg/dl, whiletheserumzinclevel in the study group was found to be 85.17±14.98µ/dl (Table

III).There was no statistically significant difference in the mean serum zinc values betweenstudy and control groups.

Zinc serum level observed in the study	No. of samples in cancer study group (n=25)	No. of samples in control group (n=25)
60-69µg/dl	3	3
70-79µg/dl	2	4
80-89µg/dl	4	5
90-100µg/dl	7	5
100-109µg/dl	3	8
110-119µg/dl	6	0

On the contrary,the serum copper level ranged from 180-340ug/dl in study group whilein the control group it varied from 110-160ug/dl. (Table II). A maximum of 8 samples were in the range of120-129µg/dl in the control and a maximum of 8 were in the range of 300-339µg/dl in the study group. The mean serum copper values werefound to be higher instudygroup 249.39±58.3µg/dl, compared to that of healthy control group123.95±16.2µg/dl.(TableIII). Mean serum zinc levels showed no changes.

Cu /Zn ratio was1.43 and 2.86 for control and study group respectively.

Table II a	
Serum copper level in control group	No of samples (n=25)
110-119µg/dl	4
120-129µg/dl	8
130-139 µg/dl	5
140-149 µg/dl	4

Table II b	
Serum copper level in study group	No of samples (n=25)
180-219µg/dl	6
220-259µg/dl	5
260-299µg/dl	6
300-339µg/dl	8

. 150-159 µ

Table II: Frequency table and range of serum copper values in control (Table IIa) and study groups(Table II b)

Table III: Mean values and standard deviation of serum copper and

Trace metals	Mean Value ± SD in Control Group	Mean Value ± SD in Study Group	P value
Zinc	86.64±17.4µg/dl	85.17±14.98µg/dl	0.67
Copper	123.95±16.2µg/dl	249.39±58.3µg/dl	0.04*
Cu/ Zn Ratio	1.43	2.86	

p<0.05value

Discussion

Trace elementsCopper (Cu), zinc (Zn), selenium (Se) and molybdenum (Mo) are required in small concentration and have important role in many biochemical reactions. They are essential for proper functioning of life supporting processes and are an essential component of biologically active constituents [13, 14]. Copper is found in plasma (90-95%) as part of oxidative enzyme cerulo plasmin [15]. It is also a part of various enzymes like tyrosinase, cytochrome oxidase etc., and participates in oxidative process in cell metabolism. Zinc another micro nutrient is essential for gene transcription and cell multiplication and critical for activation of

RNA and DNA polymerase activity. Decreased zinc levels are associated with increased oxidative stress at cellular level [16].

The normal serum copper and zinc levels are 0.6 - 1.6µg/ml and 0.6 -1.5µg/ml respectively where atomic absorptionspectrometry has been used for analysis [17]. Others [10] have reported 114.20±38.69mg/dl, 64.57±31.54 mg/dl as copper and zinc values respectively in their control sample by means of calorimetric method. The values of these trace elements estimated in the present study were performed with ERBA CHEM 5 semi auto analyzer.

Concentrations of copper when exceeds the optimal level turn's toxic. It breaks down the DNA strands or modifies the bases and deoxyribose[12].Sliwinski et al observed that zinc did not induce DNA damage in normal cells but exerted a protective effect against DNA damaging agents but increased cytotoxic effect on cancer cells[11]. A number of studies have estimated copper and zinc levels in serum, blood and saliva in various carcinomas especially oral squamous carcinoma and have established a positive correlation with the incidence of malignancy. Significant alteration in serum levels of the trace elements have been reported in head and neck cancer, lung and breast cancer [2, 18]. The increased levels of copper andzinc in cancer reported earlier have been 209.85± 160.28mg/dl, 113.51± 52.30mg/dl respectively [10]. In the present study, copper showed a significant increase [249.39±58.3µg/dl] in oral cancer group compared to controlgroup [123.95± 16.23µg/dl].Similar findings have also been reported by Khanna et al,Amitkumar et al, Yunus et al, AyinampudiBK,Jayadeep A et al and Shetty SR et al. [2, 6, 10, 17, 19, 20,21and 22]. They observed increased copper levels in sera of patients in oral pre malignant and malignant patients.

The increased level of serum copper in OSMF patient was attributed by Khanna et al to high copper content in areca nut, the major physiological factor in the pathogenesis of OSMF [17] it initiates fibrin genesis and inhibit collagen degradation. It has also been explained that increase in serum copper levels in cancer patients is a consequence of increased production of copper containing cerulo plasmin an oxidative enzyme which is precipitated by an inflammatory response to cancer or decrease in catabolism by serum cerulo plasmin [16]. Jayadeep et al also suggested that rise in copper levels might be due to increased turnover of cerulo plasmin, a copper carrying globulin with oxidative activity [19].

Review of literature indicates low serum zinc level in several forms of cancer like cancer of breast, gallbladder, lung, colon and oral cancer [23, 24]. Serum zinc level in a present study did not reveal a similar finding. The serum zinc level among cancer patient in the present study was marginally lower or similar to

control group. Contrary to this finding statistically significant decrease in mean serum zinc levels have been reported by various other studies [18, 20, and 25].

Amit kumar et al [6] suggestedthat zinc deficiency impair protective mechanism designed to protect against DNA damage, enhance susceptibility to DNA damaging agents and ultimately increased risk of cancer

Increased copper zincratioseenin oral sub mucousfibrosis (OSMF) and oral squamous cell carcinoma (OSCC) was also noticed in patients with pancreatic cancer, breast cancer, lymphoma etc. Yunus et al concluded that alterations in serum copper, zinc, copper zinc ratio can be used as potential biomarkers in early detection of oral pre cancerous lesion and their malignant transformation to frank cancer at early stages [2].In the present study the ratio was 1.43 in control,and increased to 2.86 in the study group, probably due to increased copper values. Other reports have shown that Cu/Zn ratio was 0.97 in health and 1.10 in cancer patients, but the difference was not statistically significant [17,26].

This study confirms the earlier reports on the possible relation between serum copper level and oral cancer and can be considered as a biochemical marker for oral cancer. Further evaluation onpatients with oral premalignant lesions need to be performed which will be a major contribution to cancer screening programs .

Conclusion

Trace element copper level in oral cancer patients did show asignificantincrease compared to control group. Serum zinc level however did not show any difference between study and control groups.Alterations in serum copper level are suggestive of its important role in oral carcinogenesis, contributing to changes in Cu/Zn ratio.

Corresponding author

ShanthaSundari

Professor and Head,Department of Orthodontics,

Saveetha Dental College,

Saveetha Institute of Medial and Technical Sciences, Saveetha University,

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Email: shanthakkss@gmail.com

Telephone Number: +91 9444443640

References

1. KrishnaswamyK, Prasad MP, Krishna TP, Annapurna VV, Reddy GA. A case study of nutrient intervention of oral precancerous lesions in India. Eur J Cancer B Oral Oncol 1995; 31: 41-8
2. ShaikhMohdYunus ,PrakashGadodia,RitishWadwani,Namratha N Patil,Vinod Kumar Patil.,VanitaMurgod, Abhishek Singh Nayyar. Estimation and comparison of serum levels of copper,Zinc, Cu/Zn ratio as markers of disease activity in oral submucousfibrosis (OSMF) and oral squamous cell carcinoma (OSCC) patients.Clinical cancer investigation Journal. 2017; 6(1), 51-55
3. Bouquot H, WhitakerSB.Oral leukoplakia rationale for diagnosis and prognosis of its clinical subtypes or phases. Quintessence Int.1994;25:133-40
4. Paul RR ,Chaterjee J , Das AK , Cerevera ML, de la Guardia M , Chaudhari k . Altered elemental profile as indicator of homeostatic imbalance in pathogenesis of oral sub mucous fibrosis. Biol Trace elem Res 2002 ; 87:45 -56
5. Winn DM .Diet and Nutrition in the etiology of oral cancer. Am J ClinNutr. 1995; 61:437 -445 ,
6. Amit kumar, SuchethaKumar,DharnappaPoojary, HirenDarji,Rashmi, K.S.Estimation of serum Micronutrient Levels and The Possible Risk of Oral Cancer And Premalignancy. International Journal of Research in Science Engineering and Technology 2014; 13(1):8360-4
7. World HeathOrganisation.Trace elements in human nutrition and health 1996.
8. Cho GS, Han MW, LeeB, RohJL, Choi SH, ChoKJ. Zinc deficiency may be a cause of burning mouth syndrome, zinc replacement therapyhas therapeutic effects.JOralPathol med 2010; 39:722-7
9. ShettyR ,Babu S , Kumar S , Shetty P, Vijay R, Karikal A.. Role of trace elements in oral pre cancer and oral cancer – A biochemical study. J Cancer Res Treat 2013; 1:1-3
10. Maryam Baharvand, SoheliaManifar. Reihaneh, HamedMortaZavi, SiamakSabour., Serum levels of ferritin, copper and zinc in patients with oral cancer. Biomedical Journal. 2014;37 :331-336
11. Sliwinski T, Czechowska A, Kolodziejczak M, Jajte J, Wisniewska-JarosinskaZinc salts differentially modulate DNA damage in normal and cancer cells.CellBiol Int. 2009 Apr; 33(4):542-7.

12. TheophandesT, AnastassopoulouJ.Copper and carcinogenesis. Critt Rev OncolHematol,2002 ;42:57-64

13. Schwartz, M.Rok of trace elements in cancer. Cancer Res.1975; 35:3481-7.

14. Braverman. ER, Pfeiffercc. Essential trace elements and cancer orthomolPsychiatry 1982 ;11(1):28-41

15. Sham BergerRJ.Antioxidantsand cancer Se-sex and age adjusted human cancer mortality. AnhEnviron Health1976;31:231-3

16. Andrews GS. Studies of plasma zinc, copper, ceruloplasmin and growth hormone with special reference to carcinoma of the bronchus JaclynPathol 1979;32:(4) 325-33

17. SunaliKhanna, A.C.Udas, G.KiranKumar, S.Suvarna, F.R.Karjodkar, Traceelements (copper,zinc,selenium and molybdenum) as markerin oral submucous fibrosis and oral squamous cell carcinoma.J Trace Elem Med Biol 2013; 27:307-311.

18. VashisthaA,Singh.M, Chaudhary.M, Kaur.G, Alterations in serum level of copper,iron,zinc,vitamin c and lipid profile in patients with oral cancer and oral submucous fibrosis J.Chem.Pharm.Res 2015;7:261-265

19. Jaya Deep.A,Raveendran.P.K,Kannan.S,Naline Kumar K.R,MathewB, Krishnan NM, Serum levels of copper,zinc,iron,ceruloplasmin in oral leukoplakia and squamous cell carcinoma.J.Exp.Clin.Can.Res 1997;16:295-300

20. JhaIN,Singh HB, Prasad V,Serum Copper/Zinc ratio in oral Carcinoma. Indian J.Med.Res.1985;81;602-6

21. AyinampudiBK,NarasimhanSalivary Copper and Zinc levels in premalignant and malignant lesions J.oral maxilla Pathol 2012;16:175-802

22. More C. B., Patel H., Trace elements in Potentially Oral Malignant Disorders and Oral Malignant lesion-A Biochemical study. Int J Ora Max Dis.2016; 1(2):1-7

23. EJ, Schenker JG, Chevion M, Copper and Zinc levels in normal and malignant tissues. Cancer 1983;52:8672

24. Keerthika, Vishnu Priya V, Gayathri. Estimation of Serum Copper, Zinc and Iron in Patients with Oral Cancer. Int. J. Pharm. Sci. Rev. Res. 2016; 39(1):251-25

25. Varghese I, Suganathan CK, Balasubramaniyan G, VijaykumarV, Serum Copper and Zinc levels in pre malignant & malignant lesions of oral cavity. Oncology1987;44:224-7
26. SrilekhaCopper and Zinc Level in Oral Sub mucosal Fibrosis (OSMF) Patients. M Journal of Pharmaceutical Sciences and Research 2015; 7(8):573-574

Estimation of Fluoride Concentration Level in Coimbatore (TN)

Vidhya.T.K[1],Pradeep Kumar[2], Saravana Pandian[3],

Graduate Student[1],Reader[2], Department of Public Health Dentistry, Senior Lecturer[3], Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The aim and objective of this study is to evaluate the fluoride concentration of drinking water that is available in Coimbatore.

Material and Method: The fluoride concentration of drinking water collected from Pollachi is analysed. The F ion selective electrode method was used to measure the F concentration in the samples. The measurements were obtained for every sample to ensure reproducibility and appropriate statistical analyses were employed.

Results: The fluoride content is compared with EPA and WHO guidelines of drinking water.

Conclusion: Fluoride in water is usually of geologic origin. Waters with high levels of halide content square measure principally found at the foot of high mountains and in areas wherever the ocean has created geologic deposits. Intake of excess halide, most ordinarily in drinking-water, will cause pathology that affects the teeth. The management of drinking-water quality is thus crucial in preventing pathology.

Key Words: *Fluoride, water, estimation, concentration.*

Introduction

Water may be a chemical substance with the statement binary compound. A water molecule contains one element and 2 atomic number 1 atoms connected by valency bonds. Water may be a liquid at close conditions, however it typically co-exists on Earth with its solid state, ice, and vaporised state (water vapor or steam). Water conjointly exists in a very liquid state close to deliquescent surfaces . below word wont to name chemical compounds, alter oxide is that the scientific name for water, tho' it's nearly ne'er used .Water covers seventy.9% of the layer, and is important for all legendary kinds of life on Earth, 96.5% of the planet's water is found in oceans, 1.7% in groundwater, 1.7% in glaciers and also the ice caps of Antarctica and Greenland, a-little fraction in alternative giant water bodies, and 0.001% within the air as vapor, clouds (formed of solid and liquid water particles suspended in air), and precipitation. Only 2.5% of the Earth's water is fresh, and 98.8% of that water is in ice and groundwater. Fluoride is naturally occurring substance which is present in water [1]. Fluoride when present in optimal quantities is known to prevent caries by various mechanisms but more predominantly by deposition of calcium fluoride crystals which is more resistant to demineralisation [2].Community water fluoridation began as early as 1945 and is considered as an effective way of preventing caries in children as well as adults [3]. The World Health Organisation has identified dental caries as a worldwide epidemic and also recommended to add fluoride to drinking water in naturally occurring water which has less than optimal fluoride levels. The optimal fluoride level in drinking water is 0.7 to 1.2 ppm [4]. Fluorine is the most abundant element in nature, and about 96% of fluoride in the human body is found in bones and teeth. Fluorine is essential for the normal mineralisation of bones and formation of dental enamel Fluorine is often called as two-edged sword. Fluoride is the major component in water which prevents dental caries. If the level of fluoride in bottled water is too low, optimal caries prevention may not be achieved whilst if levels are too high, developing teeth may be at risk of enamel fluorosis. Logaswamy et al. assessed the standard of groundwater in Kavundampalayam region in Coimbatore district and finished that groundwater quality varied drastically. Sundar et al. studied the groundwater quality on Noyyal watercourse in Coimbatore district and finished that the studied parameters were on top of the standards. a shot has been created to work out the suitableness of groundwater in Coimbatore district for various functions. The groundwater quality of the Singanallur sub-basin are analysed by Priya et al. to envision its suitableness for drinking, irrigation further as

domestic usage. it absolutely was finished that the Singanallur tank water was of dangerous quality thanks to the discharge of domestic sewerage. The seasonal differences within the groundwater quality of Coimbatore town throughout the year 2011 are analysed throughout pre-monsoon and post-monsoon periods victimization Geographic data system (GIS) by Jebastina et al .supported the water quality index, the samples were categorised as glorious, good,poor, terribly poor and unsuitable. Variable applied math analysis, cluster analysis and Principal part analysis were performed by Jebastina et al. on water quality information of twenty seven samples collected among Coimbatore district. The attainable factors that cause contamination are identified. Water quality index has been calculated by Priya et al. for the Singanallur sub-basin and also the entire sub-basin was zoned to check the suitableness of water for drinking functions victimization the thus ware ArcGIS and also the results showed that the groundwater quality was international organisation t for drinking in a number of the areas marking a water quality index bigger than one hundred.

Knowledge of the fluoride content of the drinking water is essential to all oral care professionals to plan of preventive dental programs and prescribes of fluoride supplements. Evidence of fluoride content in drinking water around Coimbatore district, India is largely anecdotal. Hence this study was designed to estimate the fluoride concentration in drinking water in Coimbatore district.

Material and Method

Coimbatore also known as Kovai, is a major city in the Indian state of Tamil Nadu. Located on the banks of the Noyyal River surrounded by the Western Ghats, it is the second largest city in the state after Chennai and 16th largest urban agglomeration in India. It is the largest city in the Kongunadu region. It is administered by the Coimbatore Municipal Corporation and is the administrative capital of Coimbatore district. It is one of the fastest growing tier-II cities in India and a major hub for textiles, industries, commerce, education, information technology, healthcare and manufacturing in Tamil Nadu. It is often referred to as the "Manchester of South India" due to its cotton production and textile industries. Coimbatore is also referred to as the "Pump City" and it supplies nearly half of India's requirements of motors and pumps. The city is one of the largest exporters of jewellery, wet grinders, poultry and auto components with "Coimbatore Wet Grinder" and "Kovai Cora Cotton" recognised as Geographical Indications by the Government of India.

Coimbatore has an area of city 246.75 km2 (95.27 sq mi) and Metro 642.12 km2 (247.92 sq mi). Coimbatore has a population of 1,601,438. As per the 2011 census based on pre-expansion city limits, Coimbatore had a population of 1,050,721 with a sex ratio of 997 females for every 1,000 males, much above the national average of 929. It is the second largest city in the state after capital Chennai[47] and the sixteenth largest urban agglomeration in India. A total of 102,069 were under the age of six, comprising 52,275 males and 49,794 females.The average literacy of the city was 82.43%, compared to the national average of 72.99%.

Collection of Water Sample

About 500ml of water was collected in a clean dry polythene container and labeled with information like date of collection, source and place. Fluoride levels were analyzed by ion exchange method by the chief water Analyst, state level water testing laboratory, Tamilnadu Water Supply and Drainage Board (TWAD), Government of Tamilnadu, Chennai (ISO 9001-2000- Certified).

Results

Source

Open well

Experimental Section

The fluoride concentration of drinking water collected from Coimbatore around 5 places Singanallore, Kaliyapuram, Karupapalayam,Puliyakulam, and Agilandapuram. Thereafter it was subjected to analysis for the estimation of fluoride at the Tamil Nadu Water supply and Drainage Board using the SPADNS colorimetric method.

Table 1: Fluoride content of drinking water in Coimbatore, TN.

S.NO	PLACE	FLUORIDE CONTENT IN WATER
1	Singanallore	0.130
2	Kaliyapuram	0.882
3	Karupapalayam	0.116

4	Puliyakulam	0.043
5	Agilandapuram	1.424

Table 1 shows the fluoride ion concentration in drinking water in Coimbatore. It was found that fluoride content ranging from 0.04 to 1.424.

Conversely, some drinking water contains insignificant amounts of fluoride which may deprive the child of the optimal daily fluoride requirement [5]. However, in view of increasing awareness of toxic effects of elevated F intake, particularly in children, it is important that the F level of drinking water be monitored closely to prevent adverse reactions to F from that source.

Discussion

In the present study, fluoride contains in all samples ranged from 0.04-1.42 mg/L, which is less than optimum range of 1.5 mg/L, as recommended by WHO. Hence, fluoride contents in all the samples exhibit their suitability for drinking. In a study to assess the groundwater fluoride concentration in Kanchipuram by Pradeep Kumar during 2014 it was found that the fluoride concentration ranged between 0.05-1.04 mg/l. (6). Similar studies done by Pradeep Kumar in ChennekothapalliMandal, Anatapur District, Andra Pradesh the fluoride concentration was 1.46 and 1.68mg/dl (7) and in Ennore, Chennai it ranged from 1.83 to 2.01 ppm (8,9). In general, fluoride is value-added to water as a removal agent to get rid of microorganism and alternative doable contaminants (Sharma and Soneja,2003).In numerous countries, particularly in developing countries, fluoridization is commonly embarked upon so as to enhance the facility. Water fluoridization may be a relatively cheaper and easier possibility for many components in Central Asia, whose would like for water removal is imperative.(10) On the opposite hand, developed countries have higher water removal choices therefore sanctionative them to disenlist the fluoridization of water in their water purification schemes. In Central Asia, fluoridization of water is living despite the substantive proof of high levels of water fluoridization in several regions of Asian country, Asian nation and alternative Central Asia countries.(11,12) However, in recent years, numerous health problems relative to the persistent introduction of halide within the water are raised.Based on the assessment conducted by British earth science Survey(2012), fluoride build-up has became considerably apparent within the ground waters of Central Asia and African countries

that are hardest hit embody China, India, Sri Lanka, Pakistan, and alternative African and South yankee territories (British earth science Survey, 2012).Fluorosis arose in Asian nation and have become major problems for its numerous territories, together with Andra Pradesh and province. pathology additionally emerged as a significant issue in Democratic Socialist Republic of Sri Lanka and Bangla Desh (Fawell et.al.,2006).In Asian country, fluorisis is additionally a significant health issue, significantly in metropolis, Kusur and Sargogha districts (Tariq et.al.,2012).In recent years, recommendations on the elimination or a minimum of the reduction of halide within the water are steered as a healthier possibility for the removal of water. To some extent, reduction in halide usage has been implemented; but, undeniably, the health effects of halide in water ar still persistent.(13)

The fluoride content within the well water of the Coimbatore District varied from 0.2 to 1.2 mg/L in 2007 with minimum worth in VadavalliPoosarpatti and most in P.N palayam. In 2008 the concentration of halide varies from 0.2 to 2.3 mg/L with minimum worth in Gopalapuram and most in Kavundampalayam. throughout 2009 the variation in fluoride from 0.1 to 2.7 mg/L with minimum worth in Dayanur, Mandripalayam, Natchipalayam, Senjeripudur and most worth in Andipalayam and in 2010 the fluoride concentration varies from zero.1 to 2.5 with minimum worth in Bogampatti, Kolarpatti, Marudur, Samathur, Valparai and most worth in Andipalayam and P.N palayam.It is possible that this deficiency in halide or its low concentration may need affected the dental health of the shoppers. From the Indian Standards (ISI, 1991), 1.0 mg/L is that the fascinating limit of halide in potable and low halide level could cause decay and high fluoride level ends up in pathology. The sources of halide in well water could also be because of weathering of igneous and matter rocks, phosphate fertilizers, that area unit extensively used, typically contain halides as impurity and these could increase levels of fluoride in soil. the buildup of fluoride in soil eventually ends up in its leach because of percolating water, therefore increase halide concentration in well water (Alexander, 2008).(14, 15)

Fluoride may be a chemical that happens naturally at intervals many sorts of rock. the common concentration of inorganic halide from natural sources in Asian nation normal drinkable is mostly lower than 0.050 milligrams per metric capacity unit (mg/L), however concentrations will get beyond one mg/L. the utmost Acceptable Concentration of halide in drinkable in Asian nation is one mg/L. fluoridization is that the addition of halide compounds into drinkable, to regulate concentrations to levels between 0.8 and 1.0 mg/L for the helpful

result of dental caries hindrance.(16) The general public also are exposed to halide in dentifrice or alternative dental merchandise. Most of the halide found in groundwater is of course occurring from the breakdown of rocks and soils or weathering and deposition of part volcanic particles. halide may come back from in runoff and infiltration of chemical fertilizers in agricultural areas, septic and waste matter treatment system discharges in communities with fluoridated water provides and liquid waste from industrial sources.(17)

At low concentrations fluoride will scale back the danger of dental cavities. Exposure to somewhat higher amounts of fluoride will cause dental pathology. In its mildest type this leads to discolouration of teeth, whereas severe dental pathology includes corrosion and alteration of enamel. Even higher intakes of halide confiscated an extended amount of your time may result in changes to bone, a condition referred to as skeletal pathology. this will cause joint pain, restriction of quality, and probably increase the danger of some bone fractures.The maximum level of halide that the frame could tolerate is 1mg/l. keeping in sight the assorted sources through that halide finds entry into the body, level of + 1.0 mg/l halide in water is taken into account because the optimum level to forestall each decay and numerous types of pathology.(18) The utmost limit prescribed by BIS (Bureau of Indian Standards) for halide in drinkable is additionally 1mg/l. The concentration of halide was determined as 0.2 mg/l, and also the most concentration values were determined as 1.4 mg/l in a recent study conducted in dharmapuri district. Fluoride happens in sellaite, fluorite, cryolite, apatite, apatite, fluormica, biotite, mineral alternative|and several other rocks. Weathering of those rocks and prolonged residence time results in high halide groundwater.(19) Low atomic number 20, high atomic number 11 and high hydrogen carbonate area unit typical of high halide groundwater. the opposite sources for halide area unit infiltration of agricultural run off containing chemical fertilisers, improper disposal of liquid waste from industries, alumina smelting, cement production and ceramic and brick firing. Some quantity of halide is essential for the physical structure for healthy teeth and bones.The popular technologies for the removal of halide from water embody natural action followed by precipitation, membrane processes, action and surface assimilation. In natural action, trace amounts of halide ions tend to stay in answer because of solubility restriction.Clinical dental pathology is characterised by staining and erosion of the teeth. in additional severe cases all the enamel is also broken. However, halide might not be the sole reason for dental enamel defects. Enamel opacities like dental pathology area unit related to different conditions, akin to deficiency disease with

deficiency of vitamins D and A or an occasional protein-energy diet. bodily function of halide once six years ancient won't cause dental pathology.The long-run bodily function of huge amounts will cause probably severe skeletal issues. The condition and its result on individuals pathology is caused by excessive intake of halide. The dental effects of pathology develop abundant previous the skeletal effects in individuals exposed to massive amounts of halide. Chronic high-level exposure to halide will cause skeletal pathology. In skeletal pathology, halide accumulates within the bone more and more over a few years. the first symptoms of skeletal pathology, embody stiffness and pain within the joints. In severe cases, the bone structure might amendment and ligaments might calcify, with ensuing impairment of muscles and pain. Acute high-level exposure to halide causes immediate effects of abdominal pain, excessive secretion, nausea and inborn reflex.

Conclusion

Extensive abstraction of groundwater for irrigation functions within the central a part of the study space has given rise to a groundwater trough. This space conjointly shows larger annual fluctuations in groundwater levels. (20) The persistence of groundwater trough even once the recharge season indicates over-exploitation condition therein half. Except for this, groundwater levels within the space typically follow the surface topography.

Fluoride in water is usually of geologic origin. Waters with high levels of halide content square measure principally found at the foot of high mountains and in areas wherever the ocean has created geologic deposits. illustrious halide belts toward land. Intake of excess halide, most ordinarily in drinking-water, will cause pathology that affects the teeth. Paradoxically, low levels of halide intake facilitate to stop caries. The management of drinking-water quality is thus crucial in preventing pathology.

The quality of groundwater is mostly quite contemporary within the hills and slope regions, whereas it's slightly salt within the plains and valleys. AN analysis of temporal changes in groundwater quality indicates that it's passionate about geophysics, rock-water interaction further anthropogenetic activities. in an exceedingly year having adequate rain, the groundwater within the cragged space, having slopes quite one %, typically shows a pointy increase in TDS once recharge, whereas majority of the wells set within the plains show a

decrease. Such clear grouping isn't determined in years of below adequate rain.The GIS assisted info system would facilitate to use spring water management practices admire, correct spring water resource management in terms of spring water quality & amount, integrated management of water, land use and also the surroundings, and to forestall spring water quality deterioration through correct observation and analysis.

Corresponding Author

Saravana Pandian,

Senior Lecturer, Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id-sporthoresearch@gmail.com

Telephone Number: +91 9003101627

Reference

1.Tiemann M. Fluoride in drinking water: A review of fluoridation and regulation issues. BiblioGov; 2013 Apr 5.

2.Fejerskov O, Thylstrup A, Larsen MJ. Rational use of fluorides in caries prevention: a concept based on possible cariostatic mechanisms. Acta OdontologicaScandinavica. 1981 Jan 1;39(4):241-9.

3.Lewis DW, Banting DW. Water fluoridation: current effectiveness and dental fluorosis. Community dentistry and oral epidemiology. 1994 Jun 1;22(3):153-8.

4.Ayoob S, Gupta AK. Fluoride in drinking water: a review on the status and stress effects. Critical Reviews in Environmental Science and Technology. 2006 Dec 1;36(6):433-87.

5. Dobaradaran S, Mahvi AH, Dehdashti S. Fluoride content of bottled drinking water available in Iran. Fluoride. 2008 Jan 1;41(1):93.

6. Kumar RP, Vijayalakshmi B. Assessment of Fluoride Concentration in Ground Water in Madurai District, Tamil Nadu, India. Research Journal of Pharmacy and Technology. 2017;10(1):309.

7.Kumar RP, Vijayalakshmi B. Assessment of Fluoride Concentration in Ground Water in Madurai District, Tamil Nadu, India. Research Journal of Pharmacy and Technology. 2017;10(1):309.

8. Kumar RP, Vijayalakshmi B. Assessment of Fluoride Concentration in Ground Water in Madurai District, Tamil Nadu, India. Research Journal of Pharmacy and Technology. 2017;10(1):309.

9. Kumar PR, John J. Assessment of periodontal status among dental fluorosis subjects using community periodontal index of treatment needs. Indian Journal of Dental Research. 2011 Mar 1;22(2):248.

10. National Research Council, Fluoride in Drinking Water, 2006, pp. 8-9.

11. Federal Register 11396, April 2, 1986. Note: In 1986, MCLGs were known as recommended MCLs (RMCLs) and EPA was required to issue RMCLs before setting MCLs. EPA promulgated the fluoride RMCL November 14, 1985

12. National Research Council, Health Effects of Ingested Fluoride, pp. 121-123.

13. National Research Council, Fluoride in Drinking Water: A Scientific Review of EPA’s Standards, Committee on Fluoride in Drinking Water, Board on Environmental Studies and Toxicology, Division on Earth and Life Sciences, National Academies, March 2006.

14. U.S. Environmental Protection Agency, “National Primary Drinking Water Regulations: EPA’s Review of Existing Drinking Water Standards and Request for Public Comment,” 67 Federal Register 19069, April 17, 2002.

15. National Research Council. Fluoride in drinking water: a scientific review of EPA's standards. National Academies Press; 2007 Jan 5.

16. Edelstein BL, Cottrel D, O'Sullivan D, Tinanoff N. Comparison of calorimeter and electrode analysis of water fluoride. Pediatr Dent. 1992 Jan;14(1):47-9.

17. Thippeswamy HM, Kumar N, Anand SR, Prashant GM, Chandu GN. Fluoride content in bottled drinking waters, carbonated soft drinks and fruit juices in Davangere city, India.Indian Journal of Dental Research. 2010 Oct 1;21(4):528.

18. Gupta P, Kumar A. Fluoride levels of bottled and tap water sources in Agra City, India. Fluoride. 2012 Jul 1;45(2):307-10.

19. Aldrees AM, Al-Manea SM. Fluoride content of bottled drinking waters available in Riyadh, Saudi Arabia. The Saudi dental journal. 2010 Oct 1;22(4):189-93.

20.Matloob MH. Fluoride concentration of drinking water in Babil-Iraq.Journal of Applied Sciences. 2011 Dec;11(18):3315-21.

21.Fard RF, Mahvi AH, Hosseini SS, Khazaeic M. Fluoride concentrations in bottled drinking water available in Najaf and Karbala, Iraq. Fluoride. 2014 Jul 1;47(3):249-52.

Estimation of Stature from Head Length and Head Breadth-A Cross Sectional Study

Palak Shah [1], YuvarajBabu [2], Asha Ramesh [3]

Graduate Student [1],Assistant Professor [2],Department of Anatomy,

Senior Lecturer [3],Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: Stature is an important biological parameter in medico-legal examination. This includes the determination of species of origin, age, sex and stature from the bones. It occurs when highly decomposed or mutilated fragments are brought to the medico-legal examination. Sometimes only the skull of the human is brought to the examination and helps in the estimation of stature. Among the common questioner of medico legal examination e.g. age, sex, race, etc, estimation of stature becomes equally important in such cases.The aim of this study was to estimate the stature of human from the head length and the head breadth.

Materials and Methods: This prospective study was done in department of Anatomy, Saveetha Dental College, Chennai located in Southern India. It was carried out in September 2015. A total of 100 young and healthy individuals comprising of 73 girls and 27 boys were selected for the present study. Anthropometric methodology:The stature and then head measurements of each subject were taken by using standard anthropometric instruments, in centimetres, to the nearest millimetre according to the technique described by Vallois.

Results: Descriptive statistics for height, head length, head breadth. (Male = 27; Female = 73).Head length: The regression equation generated showing the correlation between the height and the head length : Height = 2.306Head length + 119.324 (R2 - 0.042)Head breadth: Theregression equation generated showing the correlation between the height and the head breadth :Height = 1.701Head breadth + 136.385 (R2 - 0.010)

Conclusion: From this study, it can be concluded that the stature can be estimated from head length and the head breadth of the South Indian population.

***Key Words**: Estimation; Stature; Head Length; Head Breadth; South India*

Introduction

Stature or standing height is defined as the distance the head vertex and the standing surface.Because it provides one piece of information that may be an aid in individual identification, stature prediction occupies a central position in anthropological research and forensic identification analysis in the events of accidents, murders, genocide or natural disasters. It occurs many a times when highly decomposed or mutilated bodies or fragmentary remains of skull are brought for medico-legal examination. Sometime only skull is brought for examination and this is most common in our region wherevictims are attacked by wild animals in deep forests. In such situation it becomes difficult to identify deceased.

Since all these parts of the body and bones are not always available for forensic examination, it becomes necessary to make use of other parts of the body like head and face region. But only a few studies have been conducted on cephalo - facial region with respect to estimation of stature. In identification of human remains, forensic anthropologists help to interpret evidence pertaining to manner or cause of death. Marks on bones provide very important information's to how death occurred. With all evidence of skeleton trauma, it is imperatives recognized and distinguish among the ante mortem, perimortem (around the time of death) postmortem (after death).There are a few studies for stature estimation from skull alone. It is proved that each race requires its own formula for stature estimation. Racial and ethnic variations also exist in population of different geographical areas. Marks on the bone provide a very important information to know how the death occurred. Therefore, this study was undertaken from maxillo-facial anthropometry using head length and the head breadth and formulate regression models for this purpose.

Among the common questionnaire of medico legal examination e.g. age,sex, race, etc, estimation of stature becomes equally important in such cases. There is definitive biological correlation of stature with all the body parts such as extremities, head,trunk, vertebral column etc. There are variations in the length of limb bones relative to stature and according to race,sex, age,sideofthe body,climate, heredity and nutrition [1].Whenever the body is recovered in mutilated or fragmented state, the problem of identification of the person is difficult even by the most experienced forensic experts. The mutilation of dead body is done by a criminal who wants to destroy all the traces of identity and to facilitate the disposal of dead body. Height is fundamental to assess growth, nutrition, calculating body surface area and predicting pulmonary function in childhood.

There are a few studies for stature estimation from skull alone. It is proved beyond doubt that each race requires its own formula for stature estimation. The climate and dietary habits ofthe people of different regions of India are variable. Racial and ethnic variations also exist in population of different geographical regions. Hence opinions based on the result of studies done in one population cannot be entirely applicable to other population [2]. Many studies have been conducted on stature from percutaneous measurements of various body part including arm, leg, feet, etc [3-6]. Considering this scenario there is a need of systematic study for stature estimation from fragmented and dismembered skull remains.

Since there are few studies in the existing literature which assesses the stature of the individuals using head length and head breadth in a particular ethnicity, it warrants studies in the research focus. This can be utilized in the field of forensic odontology and it can aid in identification of individuals who have succummbed to catastrpohic incidents or genocide or natural disasters. Therefore the present study was undertaken to determine stature from maxillo-facial anthropometry using head length and head breadth in the South Indian population.

Materials and Methods

This prospective study was done in the Department of Anatomy, Saveetha Dental College and Hospital, Chennai located in Southern India. This study was approved by the Institutional Review Board of the University. It was carried out from September 2015 to December 2015. A

total of 100 young and healthy individuals comprising of 73 girls and 27 boys were selected for the present study.

Anthropometric Methodology

The stature and then head measurements of each subject were taken by using standard anthropometric instruments, in centimetres, to the nearest millimetre according to the technique described by Vallois.

1. Stature: The stature was measured in standing position to the vertex in Frankfurt plane by using anthropometric rod.

2. Head Length (HL): The distance between the glabella and farthest projecting point in the mid-sagittal plane, on back of the head (Occiput). The later is termed as the Opisthocranion. Glabella is the most forward projecting point in the midline of the forehead at the level of supraorbital ridges and above the nasofrontal suture.

3. Head Breath (HB): The greatest transverse diameter on the head is from euryon to euryon. Euryon is bilaterally paired point that forms the terminus of the line of greatest breadth of the skull. Not a fixed point but determined with calliper.

The height of the individual was measureed using a standard height measuring frame.

Results

The statistical analysis was performed using the SPSS software (Macros version). The descriptive statistics for height, head length and head breadth. (Male = 27; Female = 73) has been depicted in [Table 1].The mean measurement for height is 161.99. The maximum height of the population was found to be 185cm while the minimum height for the population was 142cm. The mean head length was seen to be 18.501, and the maximum and minimum head length was 20.9cm and 16.3cm respectively. Had length means was found to be 15.05. The maximum head breadth was 16.5cm and the minimum head breadth was 14.1cm.

Head Length

The regression equation generated showing the correlation between the height and the head length :

Height = 2.306Head length + 119.324 (R^2 - 0.042)

Head Breadth

The regression equation generated showing the correlation between the height and the head breadth :

Height = 1.701Head breadth + 136.385 (R^2 - 0.010)

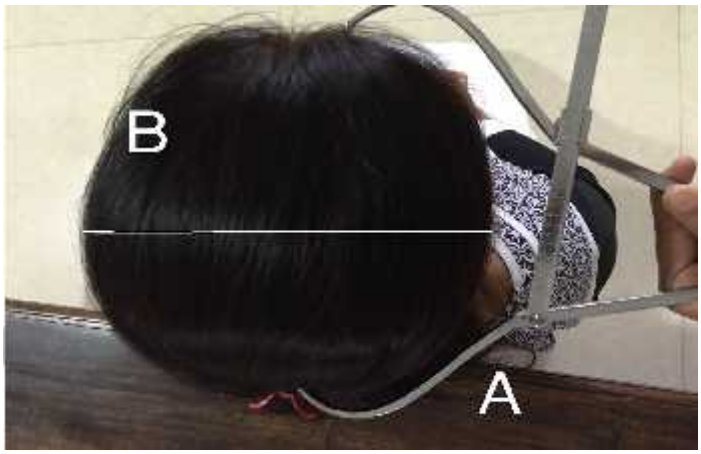


Figure 1: Measurements using a spreading calliper

A : Head Length

B: Head Breadth

Table 1 : Descriptive statistics for height, head length and head breadth

Measurement	Mean (cm)		Maximum (cm)	Minimum (cm)
Height	161.99		185	142

Head length	18.501		20.9	16.3
Head breadth	15.05		16.5	14.1

Discussion

An accurate estimation of stature for the purpose of identification has a significant forensic importance. The findings of our study. Several studies reported various statures for other ethnic groups and nations across the world. Mean stature of Ijaw males is 175.1 cm and that of Ijaw female is 166.3 cm, while the mean stature of Ikwerre male is 164.5 cm and that of Ikwerre female is 156.4 cm [7]. Adult Urhobo male has an average height of 172.5 cm and an adult Urhobo female has an average height of 161.7 cm [8]; while the mean stature of adult Ibo male is 167.6 cm and that of Ibo female is 163.2 cm [9]. The mean stature of Turkish adults is 174.4 cm (males) and 160.9 cm (females); that of Sri Lankans is 170.1 cm (males) and 157.6 cm (females) [10]; male Montenegrins are 183.2 cm tall while female Montenegrins are 168.4 cm tall making Montenegro the second tallest nation in the world after Netherlands (male 183.8, female 170.3) [11].

Mean stature for populations of adults, therefore, varies from minimum values for the Efe Pygmies of Africa at 144.9 cm for men and 136.1 cm for women to the maximum values for the Dutch of Europe at 184.0 cm for men and 170.6 cm for women [7].Though works concerning estimation of stature from long bones as well as from certain body dimensions have been put forward for some of the Indian population, works concerning estimation of stature from cranial dimensions are scanty. Hence present study is an attempt to estimate stature from head length and head breadth. And also tried to final out any correlation between stature and head length & head breadth. This is study is very unful when only skulls is bought for examination. The findings in the present study indicate that Head length and Head breadth are positively and significantly correlated with stature similar observations on stature have been reported in other races [12].

Stature Head length and head breadth were significantly greater in males when compared with females which is in occurrence with Bardale and Dixit [12] found correlation coefficient of head breadth with height as 0.26 which was not coincide with our study (0.053) and standard error of estimate for regression formula on head breadth was 6.40 which is similar to our study (6.80). For female the correlation coefficient of head breadth with height was 0.23 and standard error of estimate was 5.81. The values of SEE and r for females are very much similar to our results. Study onmaleGujjarsofIndia by Kewal Krishnan [13] showed that correlation coefficientfor head breadth in females was found to be 0.682 and SEE was 4.792. The regression equation given by themforstature estimation inmalesis 98.056 + 5.320 x Maximum head breadth. For highly correlated variates, regression equation can become practically useful for prediction and they are widely used in reconstruction formulae for stature, from the measurements of certain long bones [14].

Various workers have shown significant correlation between height and different parts of the body. Singh and Sohal, Jit and Singh have shown a significant correlation between height and length of clavicle. Charnalia, showed the significant correlation between height and foot-length, where correlation coefficient was 0.46. Athawale, derived a regression equation between total height and forearm bones. Shroff and Vare, have also derived the height from the length of superior extremity and its segments. It is stated that the racial characters are best defined in the skull. As a result cranial dimensions constitute one of the most important characters for determining the racial difference.

Variety of non-metric and metric parameters has been utilized in the assessment of ethnic and gender differences in cranio-facial morphology. The non-metric parameters are subjective as no quantitative techniques are devised. On the other hand, features that can be expressed as actual measurements, like cranial dimensions, provide more objective racial and gender diversity assessment of the crania. Correlation coefficients between the total height and head length among population of Rajasthan were found to be statistically significant and positive indicating a strong relationship between the two parameters. Previous studies have shown correlation coefficients of 0.627, 0.53, & 0.52 between head length and height, whereas in the present study it was 0.94 for males and 0.85 for females which is suggestive of strong positive correlation between these two parameters. Regression equations for their respective population were also derived by previous workers. Regression equations for stature prediction were formulated using head length and estimated statures in present study for study population and were compared with

the actual statures to check the accuracy. The results further confirmed that head length provides an accurate and reliable means in stature prediction. In forensic examinations and anthropological studies, prediction of stature from incomplete and decomposing cranial remains is vital in establishing the identity of an unknown individual. Therefore, formulae based on the head length provide an alternative stature predictor under such circumstances. The cranium has easily identifiable surface landmarks making the measurements possible even in compromised conditions.

Stature is one of the most commonly used criteria in studies on body structure [15]. Measurement of body height is important in many settings: it is an important measure of body size and gives an assessment of nutritional status as well as an important measure of determination of basic energy requirements, standardization of measures of physical capacity and adjusting drug dosage, and evaluation of children's growth, prediction and standardization of physiological variables such as lung volumes, muscle strength, glomerular filtration and metabolic rate [16]. It is also common knowledge that exact body height cannot always be determined through direct measurements because of various deformities of the extremities or in patients who have undergone amputations or similar injuries. Measuring stature can also be difficult in physically and mentally frail nursing home patients, e.g. patients that are wheelchair-bound or bedridden and those with osteoporosis, sequelae after hip fractures, or stroke. In such circumstances, an estimate of body height has to be derived from other reliable anthropometric indicators important and useful anthropometric parameters which is seen.

There is no data in literature to compare the correlation coefficients between age-height, age head length, age-head breadth and age- head height. The differences in relation to cranial dimensions observed in males and females may be used in determining sex and may be due to populations of different geographical areas.

Sexual dimorphism is an important component of the morphological variation in biological populations. The sex divergence in cranial morphometry observed in present study supports the previous observations. As rate of skeletal maturity in males and females vary during the course of growth and development, there is need for alternative formulae for genders.

In forensic examinations and anthropological studies, prediction of stature from incomplete and decomposed cranial remains is vital in establishing the identity of an unknown individual. Therefore formulae based on the cranial dimensions provide an alternative stature predictor

under such circumstances. Data can be used in conjunction with other anthropological techniques to sex and race determination of unknown individuals.Variety of factors such as age, race, sex and nutritional status affect development and growth and therefore differentnormograms are required for different populations. The present study has documented such norms for cranial dimensions and presented gender specific linear regression lines for stature and age prediction in adult Indian population. Thus population, sex and age specific regression lines will be of immense use in medico-legal, anthropological and archaeological studies.

Frutos conducted a study based on 118 complete humeri from Guatemalan forensic sample. He studied six anthropometric dimensions and concluded that the classification accuracies for the univariate functions range from 76.8% to 95.5% and for stepwise function procedure was 98.2%. Kemkes-Grottenthaler 60 evaluated the reliability of patella anthropometry in sex determination in a material from different archaeological samples [17]. He achieved almost 84% accuracy in sex determination. Patil and Mody conducted a lateral cephalometric study on central Indian population to devise a model for determination of sex. They took ten measurements on the radiographic cephalograms of 150 normal healthy individuals and determined sex by discriminant function analysis.

Conclusion

We conclude that the stature can be estimated from head length and the head breadth. Of the South Indian population. Besides this, age of the individual can also be estimated through regression lines using data on cranial dimensions. Thus data provided in the present study will of paramount importance to anthropologists to find racial differences when only parts of deceased or Indian anywhere in the world are available and may be key to sex determinatione can be estimated from the head length and the head breadth.

Corresponding author

Asha Ramesh,

Senior Lecturer, Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India

Mobile Number: +91-9841722113

E-mail Id: ash.periopg@gmail.com

References

1. Trotter M, Gleser GC. Estimation of stature from long bones of American Whites and Negroes. American journal of physical anthropology. 1952 Dec 1;10(4):463-514

2. Siddiqui MA, Shah MA. Estimation of stature from long bones of Punjabis. Indian J Med Res. 1944;31:105-8.

3. Joshi NB, Patel MP, Dongre AV. Regression equation of height on ulnar length. The Indian journal of medical research. 1964 Oct;52:1088.

4. MP P, AV D. Regression equation of height on tibial length. The Indian Journal of Medical Research. 1964 May;52:531-4

5. Mohanty NK. Prediction of height from percutaneous tibial length amongst Oriya population. Forensic science international. 1998 Dec 21;98(3):137-41

6. Özaslan A, İşcan MY, Özaslan I, Tuğcu H, Koç S. Estimation of stature from body parts. Forensic science international. 2003 Mar 12;132(1):40-5

7. İşcan MY. Forensic anthropology of sex and body size.Forensic Science International. 2005 Jan 29;147(2-3):107-12

8. Wankhede KP, Anjankar VP, Parchand MP, Kamdi NY, Patil ST. Estimation of Stature from Head Length & Head Breadth in Central Indian Population: An Anthropometric Study.

9. Chiba M, Terazawa K. Estimation of stature from somatometry of skull. Forensic science international. 1998 Nov 9;97(2-3):87-92

10. Krishna RN, Babu KY. Estimation of stature from physiognomic facial length and morphological facial length.Research Journal of Pharmacy and Technology. 2016 Nov 1;9(11):2071

11. Wankhede KP, Anjankar VP, Parchand MP, Kamdi NY, Patil ST. Estimation of Stature from Head Length & Head Breadth in Central Indian Population: An Anthropometric Study

12. Kalia S, Shetty SK, Patil K, Mahima VG. Stature estimation using odontometry and skull anthropometry. Indian Journal of dental research. 2008 Apr 1;19(2):150.

13. Krishan K. Estimation of stature from cephalo-facial anthropometry in north Indian population. Forensic Science International. 2008 Oct 25;181(1-3):52-e1.

14. Talbot PA, Mulhall H. The physical anthropology of Southern Nigeria: a biometric study in statistical method. Cambridge University Press; 2013 Mar 28

15. Kamdar RS, Babu YK. Estimation of stature from nasal height, breadth and depth.Research Journal of Pharmacy and Technology. 2016 Dec 1;9(12):2124

16. Agnihotri AK, Kachhwaha S, Jowaheer V, Singh AP. Estimating stature from percutaneous length of tibia and ulna in Indo-Mauritian population. Forensic science international. 2009 May 30;187(1-3):109-e1

17. Pelin C, Zağyapan R, Yazıcı C, Kürkçüoğlu A. Body height estimation from head and face dimensions: a different method. Journal of forensic sciences. 2010 Sep 1;55(5):1326-30.

18. Sagar S, Nath S. Estimation of stature from different head & face measurements among male and female Jatavs of Delhi. ISOR-JHSS. 2014;19(9):52

19. Akhter Z, Banu LA, Alam MM, Rahman MF. Stature estimation from craniofacial anthropometry in Bangladeshi Garo adult females. Mymensingh medical journal: MMJ. 2012 Jul;21(3):479-84.

20. Shah T, Patel MN, Nath S, Bhise RS, Menon SK. Estimation of stature from cephalo-facial dimensions by regression analysis in Gujarati population. Journal of Indian Academy of Forensic Medicine. 2015;37(3):253-7

Evaluating the Accuracy of the Lingual FlangeExtension in the Edentulous Impression StockTrays

Vidhya.T.K[1],Sangeetha[2],Saravana Pandian[3],

Graduate Student[1],Reader[2], Department of Prosthodontics,Senior Lecturer[3], Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: To investigate and compare the accuracy of edentulous stock trays and extension of alveololingual sulcus. In this study, 15 complete edentulous patients were evaluated to check accuracy of the impression trays to the extension of alveolingual sulcus in the mandibular arch in the patients. Using k-file, extension of alveololingual sulcus (anterior, middle and posterior) is measured in patient's mouth intraoral. The lingual extension of mandibular edentulous stock tray in anterior, middle and posterior was also measured and then compared to the patient's alveololingual sulcus extension.

Material and Method: In this pilot study 10 completely edentulous patients in both genders, who attended outpatient clinic in Saveetha dental college and hospitals were evaluated. K-file with a stopper and the metal scale is used to measure the extensions. Mouth mirror is used to retract the tongue from the area of interest. Patients were instructed to open their mouth and protrude their tongue so that it was ¼ inch ahead of the upper lip

Result: In this study, 15 complete edentulous patients were evaluated to check accuracy of the edentulous impression trays to the extension of alveololingual sulcus of the mandibular arch in the patients. Using k-file, extension of alveololingual sulcus (anterior, middle and posterior) is measured in patient's mouth intraoral. The lingual extension of mandibular edentulous stock tray in anterior, middle and posterior was also measured and then compared to the measured patient's alveololingual sulcus extension values

Conclusion: This study was taken to evaluate and compare the lingual extension of the mandibular edentulous stock trays to the extension of alveololingual sulcus in patients.

Key Words:*alveololingual, K-file, edentulous, impression trays, lingual extension*

Introduction

In the field of prosthodontics, retention and stability are two major concerns for complete denture. The objectives of impression making are to capture the entirepotential denture bearing surface and tissues to provide retention, support and stability for the denture under function. Impression making for complete dentures may be categorised as 1)Primary impressions and 2) Definitive impressions.(1)The basic function of primary impressions for complete dentures is to outline support. These also recommend the practice of using rigid stock trays, modified as necessary to 'fit the form of the denture-bearing area'. The tray is the foundation of an accurate impression.Over-large trays will causepatient discomfort and the likelihood of the incorporation of tissue folds. Under-sized trays will result in problems for the technician making the customised trays on undersized primary casts. The clinician will experience problems as undersized primary impressions commonly result in inadequate definitive impressions and these may result in support and stability problems in the processed denture.Recent studies indicate that flawed mandibular impressions account for the majority of denture problems in the mandible. In most of the case mandibular impressions tend to be 'short' of the retro molar pads and do not accurately record the functional forms of the floor of the mouth and the retromylohyoid fossae.(3) These deficiencies tend to result in an unstable denture.This study, evaluate and compare the lingual extension of the mandibular edentulous stock trays to the extension of alveololingual sulcus of the patients.(4) A **complete denture** is a removable appliance used when all teeth within a jaw have been lost and need to be prosthetically replaced. In contrast to a partial denture, a complete denture is constructed when there are no more teeth left in an arch, hence it is an exclusively tissue-supported prosthesis. A complete denture can be opposed by natural dentition, a partial or complete denture, fixed appliances or, sometimes, soft tissues.Retention in removable prosthodontics is defined as the resistance to vertical dislodgment that can arise from either muscular forces or physical forces. It can be gained from three different surfaces of the denture Occlusal surface, surface, Impression surface.(5)

Edentulous Stock Tray

The need to make accurate impression is fundamental to the practice o f Prosthodontics. This necessitates the dental clinicians to make careful assessment of the tissues to be recorded in the impression, type of impression trays, impression material and the techniques to be used. stock metal trays are the most commonly used impression trays The selected stock tray must cover all denture bearing area in over-extended manner. A primary cast with good coverage of proposed denture bearing area gives freedom of using any spacer design with tissue stops within the custom impression tray, thus dictating impression technique and allowing better final impressions.(6)The objectives of the primary impression are to record all the areas to be covered by the intaglio surface of the dentures and the adjacent landmarks with an impression material which is accurate and incorporates the minimum tissue displacement.(7)The use of alginate in an oversized perforated tray, where as others recommended use of impression compound by using a stock tray. Complete dentures certainly help in the control and breaking up of a bolus of food, but their chewing efficiency is considerably lower than that of natural teeth. This is due to natural teeth being firmly attached to the surrounding bone whereas dentures are merely sitting on the mucosa and thus must be actively controlled by the patient; the pain threshold of the denture-bearing mucosa is relatively easily exceeded so that the biting force, which is closely correlated with chewing efficiency of complete dentures, is reduced and may be only a sixth of that of dentate patients.(8,9)

Alveololingual Sulcus

The space in the floor of the mouth between the base of the tongue and the alveolar ridge, on each side extending from the frenum of the tongue back to the retro molar wall. It serves as a landmark for construction of denture. Achieving correct positioning of the lingual borders of the mandibular denture has taxed the abilities of generations of prosthodontists. (10) Entire border of the denture should be in contact with so displaceable tissues to complete the peripheral seal and achieve satisfactory stability and retention. Covering the maximum area of the denture foundation is one of the prime requisites to improve support and stability. Registering the exact tissue details under physiologic pressure relates to dentures being stable in function. Accurate border moulding within physiologic limits accomplishes the most desirable border seal. When deciding upon a course of treatment for an elderly patient, one

must always have the original complaint at the forefront of one's mind and plan a programme of care that can be achieved in the particular circumstances. For example, the request to see the patient may come from a relative who has become increasingly embarrassed that dentures are not being worn on social occasions. The health of the patient may have deteriorated to such an extent that successful control of a new lower denture is clearly out of the question.(11,12) It may be concluded that realistic treatment is the provision of an upper denture only, which will be worn for appearance's sake rather than for function. In such circumstances, it can be argued that the dentist is treating the relative as well as the patient, a course of action that surely is entirely justified. Although this particular illustration may be thought of as an extreme one, it is by no means uncommon in long-stay care homes and does serve to make the point that successful treatment is the 'art of the possible'.(13)

Complete denture

Most edentulous people over the age of 65 are wearing dentures that are more than 10 years old and, as a result, mucosal changes are present in between 44% and 63% of cases. The need for treatment, based on clinical judgement, suggests that 40% of 5-year-old dentures and 80% of 10-year-old dentures should be replaced. However, the picture is not that simple. Need can be measured in a variety of ways: 'Normative need' is the need defined by expert or professional opinion. 'Felt need' is the patient's subjective desire.(14) 'Expressed need' is recorded when the 'felt need' is activated through the patient seeking treatment. One estimate of 'normative need' has already been described. Others indicate that 70–85% of elderly people's dentures require attention and that such need far exceeds the 'expressed need'. Elderly people are likely to consider that treatment is required as a result of experiencing pain, difficulty in chewing, a deteriorating appearance, or because the existing dentures are broken or have been lost. However, the 'felt need' may not be activated for a variety of reasons, including the following: The dental problem is low on the list of priorities compared with other problems. (15,16)Inertia on the part of the patient.Ignorance of available services. Fear of treatment that may be required. It is important to remember that a large proportion of today's edentulous patients experienced dental treatment in less sophisticated times when pain was a frequent accompaniment. Inability to travel to a surgery because of ill health or

problems of transportation. A feeling that nothing can be done anyway and that the dental problem is just one of the inconveniences of old age, Finance.(17)

Material and Method

Patient

In this pilot study 10 completely edentulous patients in both genders, who attended outpatient clinic in Saveetha dental college and hospitals were evaluated.

Inclusion Criteria and Exclusion Criteria

The inclusion criteria patient should be completely edentulous with well-rounded residual ridge. The patients with congenital and acquired maxillary& mandibular defects, patients with severely resorbed mandibular edentulous ridge are excluded.

Methodology

K-file with a stopper and the metal scale is used to measure the extensions. Mouth mirror is used to retract the tongue from the area of interest. Patients were instructed to open their mouth and protrude their tongue so that it was ¼ inch ahead of the upper lip. Then the instrument is placed inside the patient's mouth so that extension of the floor of the mouth in anterior, middle & posterior region is measured under functional position. With the use of k-file and metal scaleanterior, middle & posterior region of the mandibular edentulous stock trays which is currently available is also measured.

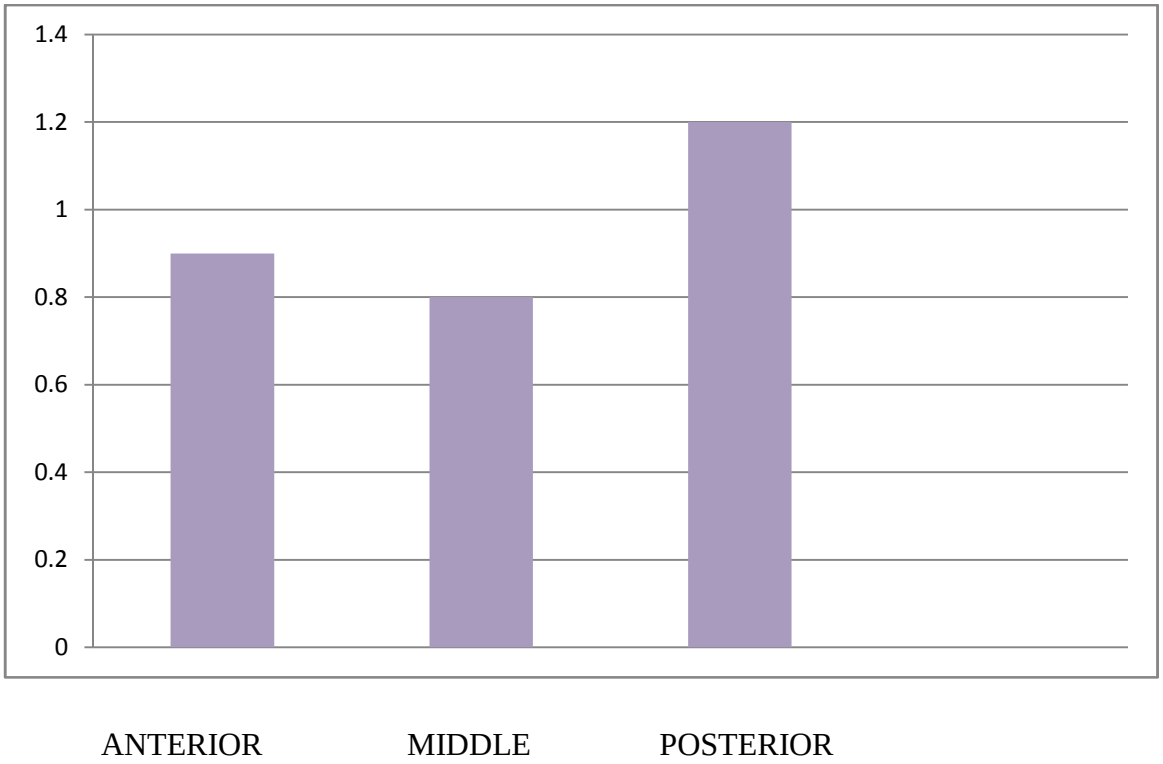
Results

In this study, 15 complete edentulous patients were evaluated to check accuracy of the edentulous impression trays to the extension of alveololingual sulcus of the mandibular arch in the patients. Using k-file, extension of alveololingual sulcus (anterior, middle and posterior) is measured in patient's mouth intraoral. The lingual extension of mandibular edentulous stock tray in anterior, middle and posterior was also measured and then compared to the measured patient's alveololingual sulcus extension values. In table1 shows

measurements of the alveololingual sulcus extension recorded in the patients intraorally.The entire patient showing posterior region of the alveololingual sulcus extension is higher than the middle and the anterior region comparatively. The extension of the middle region of alveololingual region is lower than the anterior and posterior region. Table 2 shows the measurement of the impression trays in size. Impression trays with the size L2 & L3 are used in the patients. In both the trays lingual flange extension, in the posterior region is higher than the middle and anterior region. The mean value of lingual extension of the all patient were evaluated and compared with impression tray size. Except the middle region of the impression tray, anterior and posterior are lesser or equal to the patients lingual extension. (18)The middle region of the impression trays shows higher than the patients average lingual sulcus extension.

Table1: Extension of Mandibular Alveololingual sulcus in Patients Intraorally

PATIENT NAME	AGE	SEX	ANTERIOR (CM)	MIDDLE (CM)	POSTERIOR (CM)
SHIVAGAMI AMMAL	80	F	1	0.7	1.2
PARIMALA	50	F	1.2	0.8	1.1
F.RAHMANBI	60	F	1	1.1	1.2
LALITHA	53	F	1.0	1.0	1.2
M.SARASWATHI	59	F	0.9	1	1.3
INDIRANI.G	67	F	1	0.9	1.2
MAARI	37	F	0.8	0.9	1.1
LAKSHMI AMMAL	58	F	0.8	0.7	1.1
MEENAKSHI AMMAL	60	F	1	0.8	1.1
KUMAR	45	M	0.9	0.8	1.1
MEAN			0.94	0.8	1.2



Graph 1: Extension of Mandibular Alveololingual sulcus in Patients Intraorally

Table 2: Impression trays with measurement

s.no	Impression tray size	Anterior(cm)	Middle(cm)	Posterior(cm)
1	L2	0.7	0.9	1.2
2	L3	0.9	0.9	1.2

Discussion

According to the patient's mandibular alveololingual sulcus extension, length of the posterior region is slightly greater than middle and anterior part. There are different types of tray sizes available, for lower it is L1, L2, L3 and L4. Mostly L2 and L3 are used. The key step to a successful final impression is the selection of proper tray. The distal end of alveololingual sulcus is lateral throat form. It has an great influence on fabrication of complete dentures. The extension of the denture in this area can resist horizontal forces and increase border sealing.

The length and the thickness of the flange in the space are different depending on the tonicity, activity and anatomic attachments of the adjacent structures. Except the middle region of the impression tray, anterior and posterior are lesser or equal to the patient's lingual extension. The middle region of the impression trays shows higher than the patients average lingual sulcus extension. It will result in over extended impression and sometimes cause discomfort to the patients.The excess length of the tray in the lingual region will produce an artificially stretched and thinned impression of the lingual sulcus which will tempt the dental technician to finish the denture short in that region thus losing valuable extension and bracing for the denture. (19,20)

In 1994 OGDEN.A. R, SIDDIQUE, BASKER .R M: study compared the sizes of patient's edentulous arches with the shape range of stock trays. It is concluded that even with tray modification. It is almost impossible to obtain an accurate impression of the shape of the all-important sulcus region.(21)

Conclusion

Primary impression in a stock tray should be regarded as a mean of producing a cast on which a more accurate working impression can be modified. Most of the impression trays are not sufficiently standardized. It is important to assess the size of the stock trays, not only the coverage of the retro molar pad and also lingual sulcus extension. (21)This enables the width of the tray to record the functional width of the sulcus. Recent guidelines from the British Society for the Study of Prosthetic Dentistry recommend to practice of using rigid stock trays, modified either by extending the tray or by trimming the tray extension as necessary to fit the form of the denture bearing area.

Corresponding Author

Saravana Pandian,

Senior Lecturer, Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id- sporthoresearch@gmail.com

Telephone Number: +91 9003101627

Reference

1. Hyde TP, McCord JF. Survey of prosthodontic impression procedures for complete dentures in general dental practice in the United Kingdom. Journal of Prosthetic Dentistry. 1999 Mar 1;81(3):295-9.
2. Jabeen B, ISRA S, Ilyas Y, Kazim M. IMPRESSION TECHNIQUES USED BY DENTAL SURGEONS IN DIFFERENT INSTITUTES-A STUDY. Pakistan Oral & Dental Journal. 2016 Mar 1;36(1).
3. Basker RM, Ogden AR, Ralph JP. Complete denture prescription--an audit of performance. British dental journal. 1993 Apr 24;174(8):278.
4. Zinner ID, Sherman H. An analysis of the development of complete denture impression techniques. Journal of Prosthetic Dentistry. 1981 Sep 1;46(3):242-9.
5. Boucher CO. A critical analysis of mid-century impression techniques for full dentures. Journal of Prosthetic Dentistry. 1951 Jul 1;1(4):472-91.
6. Zarb GA, Bolender CL, Carlsson GE. Boucher's prosthodontic treatment for edentulous patients. Chicago: Mosby; 1997 Jan.
7. Morrow RM, Rudd KD, Rhoads JE. Dental laboratory procedures: complete dentures. Vol 1. St Louis: Mosby. 1986.
8. Hyde TP, McCord JF. Survey of prosthodontic impression procedures for complete dentures in general dental practice in the United Kingdom. Journal of Prosthetic Dentistry. 1999 Mar 1;81(3):295-9.
9. Jain AR, Dhanraj M. A Clinical Review of Spacer Design for Conventional Complete Denture. Biology and Medicine. 2016 Jan 1;8(5):1.
10. Halpering AR, Graser GN, Rogoff GS. EJ. Mastering the art of complete denture. Chicago, IL: Quintessence Publ. Co.
11. Jain AR, Dhanraj M. A Clinical Review of Spacer Design for Conventional Complete Denture. Biology and Medicine. 2016 Jan 1;8(5):1.

12. Neill DJ, Nairn RI. Complete denture prosthetics. John Wright; 1990.
13. Al-Jabrah OA, Al-Shumailan YR. Association of complete denture wearing with the rate of reduction of mandibular residual ridge using digital panoramic radiography. International Journal of Dental Research. 2014 Feb 8;2(1):20-5.
14. Zmysłowska E, Ledzion S, Jędrzejewski K. Factors affecting mandibular residual ridge resorption in edentulous patients: a preliminary report. Folia morphologica. 2007;66(4):346-52
15. Kumar M, Kumavat V, Gupta R, Meena D. Residual ridge resorption: A Review. J Sci Tech. 2015;1(4):124-8.
16. Abirami G. Residual ridge resorption in complete denture wearers. Journal of Pharmaceutical Sciences and Research. 2016 Jun 1;8(6):565.
17. Lammie GA. Aging changes and the complete lower denture. Journal of Prosthetic Dentistry. 1956 Jul 1;6(4):450-64.
18. Abirami G. Residual ridge resorption in complete denture wearers. Journal of Pharmaceutical Sciences and Research. 2016 Jun 1;8(6):565.
19. Xie Q, Ainamo A, Tilvis R. Association of residual ridge resorption with systemic factors in home-living elderly subjects. Acta OdontologicaScandinavica. 1997 Jan 1;55(5):299-305.
20. Atwood DA. Reduction of residual ridges: a major oral disease entity. Journal of Prosthetic Dentistry. 1971 Sep 1;26(3):266-79.
21. Black DM, Delmas PD, Eastell R, Reid IR, Boonen S, Cauley JA, Cosman F, Lakatos P, Leung PC, Man Z, Mautalen C. Once-yearly zoledronic acid for treatment of postmenopausal osteoporosis. New England Journal of Medicine. 2007 May 3;356(18):1809-22.
22. Pluskiewicz W, Rogala E. Osteoporoza. Katowice: ŚląskaAkademiaMedyczna; 1995.

Evaluation of Internship Programme of Undergraduate Students in A Private Dental College

Sahana Kritivasan[1], Pradeep Kumar.R [2], Shabeenataj[3]
Graduate Student[1], Reader[2], Department of Public Health Dentistry,
Tutor[3], Department of Public Health Dentistry
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University,Chennai, India

Abstract

Introduction: Dental education is at a critical state due to advances in health care delivery system. In dentistry one can gain extensive amount of knowledge only when they are students.The aim of this study is to assess the Internship programme of undergraduate students of a private dental college in Chennai.

MaterialsandMethod: A cross-sectional study was conducted among undergraduates. A pretested questionnaire was used to assess the internship experience like critical thinking knowledge,problem solving skills .senseof responsibility,creativity,professionalattitude, appearances, quality of work and ethics. Descriptive were analysed.

Results: It shows that about 72.3% of interns were satisfied about their internship achievements.

Conclusions: The study concludes that the internship programme have built in the clinical competency of the interns.

Key Words*: internship, self evaluation, learning, communication, curriculum*

Introduction

Dental curriculum changes dynamically hence it needs to be monitored and reconstructed according to the needs of the student, community and profession, otherwise it becomes stagnant.According to Shetty V.B, a good curriculum should always restructures itself and should receive feedback from all stakeholders. [1] The dental education is at a critical stake due to the advances and changes in the health care delivery system. There is a wide belief that the dental students are not satisfied with their experience in the dental colleges which may be due to the stressful environment. [2]Psychological stress or distress has long been regarded as an important influence on the learning and performance of the students. It serves as a motivation to some portion of the student community and for the rest it has adverse negative effect on their studies, health and personal lives. [3]

Being a consumer of the dental programme, the students view must be considered by the curriculum evaluation committee while updating the dental curriculum [4]. A self assessment can be conceived as an ingrained habit or trait to reflect an individual. Students of dental profession are bright; goal oriented and is reflective, sightful and consistent in academic and in assessing their skills and ability. [5].Hence the study was designed to analyse the perception of the interns about the present scenario of dental education, clinicaltrials, teacher-student relationship, professional outlook and patient care.

Materials and Method

A pre-tested structured questionnaire were given to 65 students of Saveetha Dental College and Hospitals, Chennai who were undergoing the internship programme. The feedback of the interns regarding the questionnaire were gathered and the data collected were entered for further evaluation. Evaluation of the data was done and the output was obtained.

Result

The results showed an overall satisfaction in achieving the internship roles and responsibilities along with performance(table 1), organisation and working environment(table 2) and finally the academic portfolio and extra curricularsactivites(table3).

Table 1: Internship Roles and Responsibilities with Performance

Items	Strongly disagree %	Somewhat disagree %	Neither agree nor disagree %	Somewhat agree %	Strongly agree %
Achievement of internship objectives	4.6	6.2	16.9	53.8	18.5
Demonstration of communication skill	3.1	13.8	6.2	49.2	27.7
Application of classroom knowledge	4.6	7.7	20.0	40.0	27.7
Critical thinking and problem solving skill demonstration	7.7	7.7	15.4	49.2	20.0
Professional appearance	4.6	4.6	9.2	53.8	27.7
Response to supervision and criticism	6.2	9.2	20.0	44.6	20.0
Self sufficient, independent as well as a team worker capacity	1.5	1.5	15.4	40.0	41.5
Exhibition of responsibility and dependability	3.1	7.7	7.7	50.8	30.8
Exhibition of positive attitude towards work and co workers	3.1	4.6	7.7	36.9	47.7
Exhibition of professional attitude to behave	6.2	13.8	6.2	49.2	24.6

ethically					
Sensitive to diversity	6.2	12.3	23.1	40.0	18.5
Adaptation to changing circumstances	3.1	3.1	16.9	47.7	29.2
Sufficient quantity and quality of work production	6.2	6.2	18.5	40.0	29.2
Demonstrated awareness of strength/ weakness	1.5	9.2	7.7	52.3	29.2
Was comfortable handling all assigned responsibilities	6.2	0.0	20.0	49.2	24.6

Table 2: Organisation and working environment

Title	Strongly disagree %	Somewhat disagree %	Neither agree nor disagree %	Somewhat agree %	Strongly agree %
Providence of sufficient orientation to its mission, purpose, culture,policies and practices	10.8	1.5	13.8	56.9	16.9
The work environment was stimulating	7.7	12.3	13.8	46.2	20.0
The organisation invited me to provide feedback and input on the work in which I was engaged and to discuss	13.8	10.8	16.9	43.1	15.4

organisational matter					
The work was challenging	7.7	3.1	12.3	44.6	32.3
The internship tasks and experiences helped to meet the learning goals and objectives	4.6	1.5	16.9	49.2	27.7
Providence with access and insight into a variety of activities to contribute to the learning	4.6	3.1	15.4	50.8	26.2
Was conscious of my needs as an intern and made clear the task responsibilities	7.7	1.5	18.5	50.8	21.5
Assigned an appropriate amount of work	7.7	10.8	18.5	41.5	21.5
Discussed ways by which learning objectives could be achieved	7.7	4.6	27.7	43.1	16.9
Providence of regular assistance and willingness to answer the queries regarding work setting and specific tasks	4.6	7.7	20.0	50.8	16.9

Table 3: Academic Portfolio and extra curricular activities

Title	Strongly disagree %	Somewhat disagree %	Neither agree nor disagree	Somewhat agree %	Strongly agree %
Academic coursework provided preparations of	6.2	3.1	16.9	46.2	27.7

professional knowledge and skills					
Comparison helped in modulating the internship programme	7.7	10.8	18.5	44.6	18.5
Usage of CAD CAM, RVG and advances record management system was useful	7.7	10.8	10.8	41.5	29.2
Outreach program helped to explore more unknown problems	4.6	7.7	18.5	47.7	21.5
Participation in conferences, seminars and presenting scientific papers and posters provided insight	3.1	3.1	18.5	46.2	29.2
Research work helped in career building	9.2	7.7	24.6	35.4	23.1
Library provided adequate resources for various academic and research purposes	9.2	13.8	20.0	40.0	16.9
Usage of wifi and ipad was beneficial	9.2	10.8	10.8	41.5	27.7
No. Of clinical procedures provided adequate training and professional skills	10.8	9.2	13.8	43.1	23.1
UTSAV provided platform	6.2	6.2	6.2	24.6	56.9

to exhibit hidden talents					

Discussion

It is very important to consider the perspective of graduating dental students regarding their curriculu, motivation and support. Also it is necessary o assess their internship experience like critical thinking knowledge,problem solving skills.sense of responsibility,creativity,professional attitude, appearances, quality of work and ethics.

The study show the various perceptions of the interns of a private dental institution should that there was overall satisfaction in acheievong the internship roles and responsibilities along with performance. It also gives us an insight of the strengths and weakness of the current curriculum of that dental college which is monitored regularly.

Table 1 shows that about 49.2% of studnts have necessary communicaton skills. This is in accordance with the study conducted by Lanning S.K et al who quoted that, communication skills learned in the clinical setting, helps in providing a formal approach towards the patient and it is more efficient in enhancing the students confidence[3]. Also 49.2%of the interns have the ability of critical thinking and problem solving skills. About 50.8% of interns exhibit responsibility and defendabilty. About 49.2% were comfortable in handling all the assigned responsilities.

More than 40 percent of the students expressed that, they were able to apply their theoretical knowledge in their clinical practice which is higher than a similar study conducted by Ashri N in King Saud University,Riyadh.[4].

The internship tasks and responsibilities were handled comfortably by about 52.3 percent of the students which reflects that the students have gained adequate confidence to meet their goals and to understand their strength and weaknesses.

A study conducted by Kumar S in Rajasthan highlighted that students had stress due to the fear of their clinical in charge and the environment provided by the faculty to them during their clinical training [8].

The study by Chaudhary S mentioned that the way the practitioner carries himself influences the patients image of him or her[9]. Nearly 53.8 percent of the interns believed in maintaining a good professional appearance.

Nearly 40 percent of the students were elated about being a self sufficient worker as an individual as well as a team worker. A study in UK by Evans J showed the need for the development of a positive attitude towards inter professional education which will help in maintaining a good team work with the technicians and their colleagues which will lead to a better collaborative practice.[10]

This was furtherillustrated in a study done by Leisnert L in Sweden where the students responded that patient needs to be approached holistically which should be a permanent part of education.[11] About 47.7 percent of the students appeased positive attitude towards their co workers. This helps them to respect each other's role in providing care for the patient.

The Association of American Medical colleges considers ethical responsibility to self and others, reliability, adaptability and dependability as the crucial for effective professional behaviour.[12] Being an intern student who is going to be exposed to private practice soon needs to be aware and exhibit the same. About 50.8 percent of the students exhibited responsibility and dependability during the course work.

Nearly 50 percent of the students exhibited professional attitude to behave ethically and 40 percent of the students were sensitive to diversity at the working environment. Diversity during the clinical practice includes the difference in gender, socioeconomic status, religion of the co workers and patients.

About 47.7 percent of the students were able to adapt to the changing circumstances and 46.2 percent of the interns believed that the working environment was stimulating for them to perform from better to best.

Table 2 shows that about 56.9% agree that the working environment provides sufficient orientation to its mission , purpose , culture ,polices and practices . About 50.8% of the interns were stiulated by regular assistance and willingness of the faculty to clear their queries regarding specific tasks. Attitude of the faculty towards the intern helped in modulating their perspective of achieving their objectives.

Nearly 43.1 percent of the interns were satisfied in providing feedback and discussing organisational matters. Asimilar study was done by Curtis D.A in University of California which the organisation invited its students to a provide a self assessment feedback of preclinical examinations.[13]

The number of clinical procedures done by the interns provided them with adequate training and professional skills. This was strongly agreed by 66.1 percent of the students while in a similar approach done in Riyadh reported only 59.8 percent of its students were satisfied with the quantity and quality of work produced by them in different branches of dentistry that helped them in becoming a self sufficient clinician at the undergraduate level.[4]

Adding on to the work production, selection of proper techniques and giving proper care provides a good orientation to their mission as a clinician. This was also sighted in a study by Marchan S.[6]

A major portion of the study population agreed that the work was challenging and will help them in improving their performance and observational skills during their clinical practice.

Table 3 shows that about 41.4% of the iterns were highl elated with the usage of facilites like like CAD-CAM, RVG, TOUCH ON CLOUD, DENTAL COLLEGE RECORD MANAGEMENT SYSTEM USING IPAD.[15,16,17,18,19]Also it has newer methods of learning and opens out vast areas of resources. These revolutionary technologies enhance the dental practise and education.[18'19]The ciruculam also provides oppurtunites to add credentials to their profile by participating in conferences and seminars. This enourages the to tackle different obstacles that arise while engaged in research work.

A library is an important aspect of every institution but the usage and the importance of it is lacking in almost all the students.Only 40 percentage of the students realised the benefits of the adequate resources found in the library for various academic and research oriented purposes where as in a parallel study done by Shetty V highlighted its grief of only 35 percent of its students were satisfied with the library resources.[1]

Apart from the academic mission and orientation, the institution also provides an equal opportunity for its students to showcase their talents. By providing a platform for these talents to its students the institution will be able to explore ways to reduce stress among them and increase the efficiency of work produced by them.

Conclusion

This study has investigated the internship program of a private dental college in Chennai. Feedack of the interns highlights the success of the ciriculum. The enormous work produced by the interns helps to adapt to changing environment and overcome the stress. Patients satisfaction, time management and potent assessment becomes fruitful with efficient communication skills. The strength of the current program were a provision to critical thinking , problem solving skills , exhibiting responsibility , providing good working environment and revolutionary computer technologies.

Corresponding Author

Shabeenataj, Tutor, Department of Public Health Dentistry
Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences
162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India
Email: - shabeenataj2592@gmail.com, Telephone Number: +91 9843390930

References

1. Shetty V.B,Shirahatti R.V,PawarP,BDS.Student's Perceptions of Their Education on Graduation from a Dental School in India.Journal of Dental Education.2012;76(11).
2. Ganesh R,Sajida Sultana.N,Naz T.Crossroads of Dental Education:Perception among the Interns of the Dental Schools in Tamil Nadu,India.e-Journal of Dentistry.Jul-Sep 2013;3(3).
3. Lanning S.K,D.D.S;RansonS.L,WilletR.M.Communication Skills Instruction Utilizing Interdisciplinary Peer Teachers:Program Development and Student Perceptions.Journal of Dental Education.2007;72(2).
4. Ashri NA, AlhamadAR, Alnasser LU. Students and recent graduates perspective of dental curriculum at King Saud University, Riyadh-Saudi Arabia. Pakistan Oral & Dent. Jr. 2006;26(2):175-82.
5. Gordon M.J.A Review of the Validity and Accuracy of Self-assessments in Health Professions Training.AcademicMedicine.December 199; 6(12)66-70.
6. MarchanS.M,SmithW.A.J.Self Perceived Clinical Competencies and Proficiencies of Internationally Trianed Dentists at a Non-US Site Completing an Advanced Education in General Dentistry Residency.Open Access Scientific Reports.2012;1(6).13-14.
7. SpegmanA.M,GeisingerCenter of Health Research;Herrin S,DC,Western States Chiropractic College.Chiropractic Interns' Perceptions of Stress and Confidence.The Journal of Chiropractic Education.2007;2(2)21-24.
8. Kumar S,DagliR.J,MathurA,JainM,PrabuD,Kulkarni S.Perceived sources of stress amongst Indian dental students.European Journal of Dental Education 2009;13(4)39-45.

9. ChaudharyS,GowdaT.M,Kumar.T.A.B;Menta D.S .Journal of Dental Education.2013;7(10)77-84.
10. Evans J,Henderson A.The Future of Education and Training in Dental Technology:Designing a Dental Curriculum that Facilitates Teamwork across the Oral Health Professions. European Journal of Dental Education. 2012;2(6)25-29
11. Leisnert L, Karlsson M, Franklin I, Lindh L, Wretlind K. Improving Teamwork between Students from Two Professional Programmes in Dental Education.European Journal of Dental Education. 2012;3(1)17-26.
12. Middlemas S; Haftel H, MD, MHPE; Ross P.T, MA; Lypson M.L,. Assessing Interpersonal Communication Skills. The Use of Standardized Patients. 2009;5(8)54-58.
13. Curtis D.A, Lind S.L, Setia G, Finzen F.C, Dental Students Self Assessment of Preclinical Examinations. Journal of Dental Education. March 2008;7(3)72-75.
14. Barrak A. Al, Yami R Al. Assessment of Skills and Attitude of Dental Students and Interns towards e-Learning in KSU. International Journal of Excellence in Education. September 2013;6(1)65-67.
15. Murthykumar K, Veeraiyan DN, Prasad P. Impact of Video Based Learning on the Perfomance of Post Graduate Students in Biostatistics: A Retrospective Study. Journal of Clinical and Diagnostic Research : JCDR. 2015;9(12):ZC51-ZC53.
16. Renuka S, Gurunathan D. Usage of internet for academic purposes by undergraduate students. Journal of Advanced Pharmacy Education & Research| Jan-Mar. 2017;7(1).
17. Aruna S, Kavitha MS, Thenmozhi MP. EFFECTIVENESS OF SMALL GROUP LEARNING IN PROMOTING STUDENT LEARNING. Research Journal of Pharmacy and Technology. 2015;8:1076-78
18. KadambariSriram and Brundha M.P ' Effectiveness Of Papers 53 App In Teaching Methodology', International Journal of Current Advanced Research, 2017;06(03): 2886-2889.
19. P Thejeswar, E & Thenmozhi, MS. Educational Research-iPad System vs Textbook System. Research Journal of Pharmacy and Technology. 2015;8:1158- 59

Evaluation of Patterns of Third Molar Impaction in Chennai Population

Divyadharshini V[1], Nithya Jaganathan[2], Dinesh Prabu[3]

Graduate student[1,] Reader[2], Department of Oral Pathology,

Senior Lecturer[3], Department of Oral and maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: Tooth impaction is a pathological situation in which a tooth cannot, or will not; erupt into its normal functioning position, unless facilitated by treatment [1]. One of the important causes for third molar impaction is the inadequate space in the arches for the molars to erupt. Although many impacted teeth appear asymptomatic, they are considered as potential source of trouble. The purpose of the study was to investigate and analyse the prevalence and pattern of third molar impaction in adults between 18-35 years of age who attended a Private Dental College in Chennai, Tamil Nadu.

Materials and Methods:A total of 430 orthopantomograms (OPG) of patients ranging in age from 18 to 35 years were collected, of which 200 OPG's had third molar impaction. . The pattern of third molar impaction, their frequency, levels of eruption, and their angulations were evaluated. The position of impacted third molar was classified according to Pell & Gregory and Winters classification.

Result: Two hundred of the 430 OPG showed at least one impacted third molar. In males, theimpacted third molars were more likely to occur in the mandible (81.4%) than in the maxilla (30.1%). In females, the impacted third molars were more likely to occur in the mandible (78.2%) than in the maxilla (43.7%). Mesioangular (42.8%) impactions were more likely to occur in the mandible and Vertical (58.8%) impactions were more likely to occur in the maxilla. Level B impaction was themost common in both maxilla (77.1%) and mandible (84.3%).

Conclusion: Mandibular third molar has a high prevalence of impaction in the Chennai population with nosex predilection.

Key Word: *Chennai, Impaction, Population, Pattern,Third molar.*

Introduction

Impaction was derived from Latin word which is defined as the cessation of eruption of tooth caused by a clinically and radiographically detectable physical barrier in the eruption path or by an ectopic position of the tooth. Any tooth may be impacted, but certain ones are more commonly affected than others. Maxillary and mandibular third molar and the maxillary cuspids are more commonly impacted followed by premolars and supernumerary teeth. Generally, third molars have been found to erupt between the ages of 17 and 21 years [2, 3]. According to Elsey and Rock [4] impaction of the third molar is the most common and accounts to 98% of all impactions. The incidence of impaction of maxillary and mandibular third molar varies widely and is reported to be in a range of 22% and 18% respectively. Of the third molars, the mandibular teeth are more apt to exhibit severe impaction than the maxillary teeth. This can be attributed to the pattern and development of face and facial bones, tooth size and the eruption pattern.

Etiology of permanent teeth impaction includes several systemic and local factors. Cleidocranial dysplasia, Down syndrome, endocrine deficiencies (hypothyroidism and hypopituitarism), febrile diseases, and irradiation, are some of the systemic factors that may influence impaction of permanent teeth [5, 6]. Local factors include prolonged deciduous tooth retention, malposed tooth germs, arch-length deficiency, supernumerary teeth, odontogenic tumours abnormal eruption path, and cleft lip and palate [7-9].

Wisdom teeth become impacted when there is not enough room in the jaws to allow for all of the teeth to erupt into the mouth. Because the wisdom teeth are the last to erupt, due to insufficient room in the jaws to accommodate more teeth, the wisdom teeth become stuck in the jaws that are impacted. Genetics plays an important, albeit unpredictable role in dictating jaw and tooth size and tooth eruption potential of the teeth. Some also believe that there is a evolutionary decrease in jaw size due to softer modern diets. The formation of wisdom teeth differs by population, ranging from practically zero in Tasmanian Aborigines to nearly 100% in indigenous Mexicans. The difference is related to the PAX9 gene.

Wisdom teeth communicating with the mouth cause localized pain, swelling and bleeding of the tissue overlying the tooth. This tissue is called the operculum and the disorder called pericoronitis which means inflammation around the crown of the tooth. Low grade

chronic periodontitis commonly occurs on either the wisdom tooth or the second molar, causing less obvious symptoms such as bad breath and bleeding from the gums. As the teeth near the mouth during normal development, people sometimes report mild pressure of other symptoms similar to teething.

The aim of the present study was to evaluate the pattern of third molar eruption radiographically and correlate it with the gender of an individual. The study also aimed to evaluate the angulation and level of impaction among Chennai population.

Materials and Methods

The study was conducted on archived orthopantomogram radiographs available in the Oral Medicine and Radiology department of patients who were subjected to radiographic examination for other dental problems. The radiographs were exposed with Orthophos Sirona XG 3D hybrid machine. The exposures were taken at 60-64 kvp with 1-2 mA. Two hundred orthopantomograms (OPG) of patients aged 19years and older and their related data were selected from the records. Of which 113 belong to males and 87 belong to females. OPG of patients younger than 19 years; a history of any dental extraction; orthodontic treatment or dento alveolar trauma; incomplete root formation of the third molars; any pathological dento alveolar condition; any craniofacial anomaly or syndrome such as Down syndrome; cleidocranial dysostosis; and the presence of incomplete records or poor quality OPG were excluded from the study. Patient's OPG were evaluated to determine the third molar status.

Third molar was considered impacted if it was not in functional occlusion. The level of impaction was determined according to the relationship of the CEJ of the third molar relative to the alveolar bone level (Table 1). The angulations was assessed by measuring the angle formed between the long axis of third molar relative to the long axis of the second molar (Table 2).

Table 1: Pell and Gregory classification of levels of third molar impaction based on its position in bone

Level	Definition
Level A	Not buried in bone
Level B	Partially buried in bone
Level C	Completely buried in bone

Table 2: Winters classification of level of impaction based on angulation of third molar

Level of impaction	Angulation of third molar to second molar
Vertical impaction	10o to -10o
Mesioangular impaction	11o to 79o
Horizontal impaction	80o to 100o
Distoangular impaction	-11o to -79o

Results

The study evaluated 430 orthopantomograph. Out of these 230 were eliminated as it did not show at least one impacted teeth. A final set of 200 OPG's were taken for evaluation. Among the 200OPG's 113 (56.5%) were males and 87 (43.5%) were females. The number of impacted and non-impacted third molars in maxilla and mandible was evaluated in males and females. The impacted third molars were also evaluated based on angulation and level of impaction in maxilla and mandible.

Number of Impacted and Non-Impacted Third Molar in Males and Females

Maxilla

In maxilla, the number of impacted third molars in male was 68 (30.1%) and in female was 76 (43.7%). The total number of impacted third molars in maxilla was 144 (36%). The number of non-impacted third molars in male was 158 (69.9%) and in female were 98 (56.3%). The total number of non-impacted third molars was 256 (64%). There is significant difference between impacted and non-impacted third molars in males and females ($p<0.05$). (Table 3)

Table 3: Impacted teeth among males and females in Maxilla

	Male		Female		Total		P-value
	N	%	N	%	N	%	

Impacted teeth	68	30.1	76	43.7	144	36	0.005
Non impacted teeth	158	69.9	98	56.3	256	64	

Mandible

In mandible, the number of impacted third molars in male was 184 (81.4%) and in female were 136 (78.2%). The total number of impacted third molars in maxilla was 320 (80%). The number of non impacted third molars in male was 42 (18.6%) and in female was 38 (21.8%). The total number of non impacted third molars was 80 (20%). The results were not statistically significant ($p<0.05$). (Table 4)

Table 4: Impacted teeth among males and females in Mandible

	Male		**Female**		**Total**		**P-value**
	N	**%**	**N**	**%**	**N**	**%**	
Impacted teeth	184	81.4	136	78.2	320	80	0.420
Non impacted teeth	42	18.6	38	21.8	80	20	

Angulation of impaction

The angulation of impaction of right and left third molar in maxilla and mandible was determined using table 5 and table 6.

Maxilla

The most common angle of impaction in right maxillary third molar was vertical (53.8%). The most common angle of impaction in left maxillary third molar was vertical (63.8%). These data's suggest that the most common angle of impaction in maxilla is vertical (58.8%).

Table5: Angulation of Impaction in Maxilla

	HORIONTAL		VERTICAL		DISTOANGULAR		MESIOANGULAR	
	N	%	N	%	N	%	N	%
18	1	1.3	43	53.8	23	28.8	13	16.3
28	1	1.7	37	63.8	14	24.1	6	10.3

Mandible

The most common angle of impaction in right mandibular third molar was mesioangular (42.3%). The most common angle of impaction in left mandibular third molar was mesioangular (43.3%). These datas suggest that the most common angle of impaction in mandible is mesioangular (42.8%).

Table 6: Angulation of impaction in Mandible

	HORIONTAL		VERTICAL		DISTOANGULAR		MESIOANGULAR	
	N	%	N	%	N	%	N	%
38	46	29.3	43	27.4	0	0	68	43.3
48	61	37.4	33	20.2	0	0	69	42.3

Level of impaction

In maxilla and mandible level B impaction was more common 7.1% and 84.3% respectively (Table 7)

Table 7: Level of Impaction in Maxilla and Mandible

	MAXILLA		MANDIBLE	
	N	%	N	%
A	1	1	10	4.9
B	74	77.1	172	84.3
C	21	21.9	22	10.8

Discussion

Impacted third molar occurs frequently in patient and causes complications because of such natures it considered as a major problem in dentistry. Selection criteria included patients aged older than 19 years, as growth is essentiallycompleted by this age[10] and many third molars have their roots completed by this time.

In the present study it was revealed that the 36% of the maxillary third molar were impacted and 80% of the mandibular third molar teeth were impacted.

The mesioangular angle (42.8%) was the most common angle of impaction of third molar in the mandible which is in agreement with those of Quek et al [11], Kramer and Williams [12], Moris and Jerman [13] and Hemamalini Balaji et al [14].However, the findings are in contrastto those of Hugoson and Kugelberg [15], who found the verticalangulation to be the most common. The most common angulationregistered in the maxilla was the vertical angulation (58.8%) and this is inagreement with Quek et al [11]. However, it disagrees with Krugeret al[16] who found that mesioangular impaction was the mostfrequently observed pattern of impaction in the maxilla.

The B level was the most common level of impaction in both maxilla (77.1%) and mandible (84.3%) in this study. This agrees with the findings ofQuek et al [11] but not those of Hugoson and Kugelberg [15]. Many international studieshave been performed to identify the reason for third molar impaction. Several factors were reportedas possible causes for third molar impaction: including lack ofspace distal to the permanent second molar; retarded third molarmineralization; and early physical maturation[17-20].

It has been presented that a lower third molar may become impacted in three different ways: 1) It can follow the pattern of a normally developing third molar by decreasing its angulation to the mandibular plane and becoming more upright, but the up righting may not be sufficient to permit eruption; 2) its angular developmental position relative to the mandibular plane may remain unchanged; and 3) it can increase its angulation to the mandibular plane and become more mesially inclined.

The most common treatment for the impacted tooth was extraction and the reason for their extraction was because of impaction and its potential complications damaging the adjacent teeth or the involved tooth itself [21].A treatment controversy exists about the necessity and timing of the removal of asymptomatic, disease-free impacted wisdom teeth. Proponents of early extraction cite the cumulative risk for extraction over time and costs of monitoring to

retain wisdom teeth. Advocates for retaining wisdom teeth cite the risk and costs of unnecessary operations [22].

Corresponding Author

Dinesh Prabu M

Senior Lecturer,Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

E-Mail: rogerprabu@gmail.com

Telephone Number: +919790098719

Reference

1. Samir EB, George A. Third molars: A review. Am J. Orthod 1983; 83:131–137

2. Elsey MJ, Rock WP. Influence of orthodontic treatment on development of third molars. Br J Oral Maxillofac Surg. 2000Aug; 38(4):350-3.

3. Pahkala R, Pahkala A, Laine T. Eruption pattern of permanent teeth in a rural community in northeastern Finland. Acta Odontol Scand. 1991Dec; 49(6):341-9.

4. Khan NB, Cohan AN, AlMograbi B, AlDeyab S, Zahid T, AlMoutairi M. Eruption Time of Permanent First Molars and Incisors Among a Sample of Saudi Male Schoolchildren. Saudi Dent J. 2006 Jan-Apr; 18(1):18-24.

5. Bishara SE. Impacted maxillary canines: a review. Am J Orthod Dento- facial Orthop. 1992; 101(2):159–171.

6. Levi O, Regan D. Impaction of maxillary permanent second molars by the third molars. J Paediatr Dent. 1989; 5:31–34.

7. Raghoebar GM, Boering G, Vissink A, Stegenga B. Eruption disturbances of permanent molars: a review. J Oral Pathol Med. 1991; 20(4):159–166.

8. Jacoby H. The aetiology of maxillary canine impaction. Am J Orthod. 1983;84(2):125–132.

9. MoyresRE.HandbookofOrthodontics.4thed.Chicago, IL: YearBook Medical Publishers; 1988.

10. Fielding AF, Douglass AF, Whitley RD. Reasons for early removal of impacted third molars. Clin Prev Dent. 1981; 3(6):19–23.

11. Quek SL, Tay CK, Tay KH, Toh SL, Lim KC. Pattern of third molar impaction in a Singapore Chinese population: a retrospective radiographic survey. Int J Oral Maxillofac Surg. 2003; 32(5):548–552.

12. Kramer RM, Williams AC. The incidence of impacted teeth. Oral Surgical Med Oral Pathol. 1970; 29(2):237–241.

13. Morris CR, Jerman AC. Panoramic radiographic survey: a study of embedded third molars. J Oral Surg. 1971; 29(2):122–125.

14. Hemamalini Balaji. Evaluation of Impacted Mandibular Third Molar using Panoramic Radiographs. J. Pharm. Sci. & Res. 2015; 7(11):940-945.

15. Hugoson A, Kugelberg CF. The prevalence of third molars in a Swedishpopulation. An epidemiological study. Community Dental Health.1988; 5(2):121–138.

16. Kruger E, Thomson WM, Konthasinghe P. Third molar outcomes fromage 18 to 26: findings from a population-based New Zealand longitudinalstudy. Oral Surg Oral Med Oral Pathol Oral Radiol Endod.2001; 92(2):150–155.

17. Björk A, Jensen E, Palling M. Mandibular growth and third molar impaction. Acta Odont Scand. 1956; 14:231–272.

18. Ricketts RM. The principle of arcial growth of the mandible. Angle Orthod. 1972; 42(4):368–386.

19. Breik O, Grubor D. The incidence of mandibular third molar impactions in different skeletal face types. Aust Dent J. 2008; 53(4):320–324.

20. Richardson ME. The aetiology and prediction of mandibular third molar impaction. Angle Orthod. 1977;47(3):165–172.

21. Sonti SH. Incidence of Mandibular Third Molar Impaction in Patients Visiting a Private Dental College. IOSR-JDMS. 2014; 13(1):01-02.

22. Akshay S, Niha N. Third Molar Impaction- Review. Research J. Pharm. and Tech. 2014; 7(12): 1498-1500.

Evaluation of ph, Buffering Capacity and Flow Rate of Saliva in Caries Free People and in People with Dental Caries among Different Age Groups

Aparnaa.M[1], Jayalakshmi. S[2], Sreedevi .D[3]

Graduate Student[1],Senior Lecturer[2],Departmentof Conservative Dentistry and Endodontics

Senior Lecturer[3],Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: The saliva plays important roles in the maintenance of the oral health because it can prevent bacterial invasion, growth and metabolism through different mechanisms . Also, it can modulate the bacterial adhesion to teeth and attenuate the deleterious effects of the production of metabolites by oral microbiota due to its organic and inorganic components, contributing for oral health maintenance .The aim of this research is to evaluate the salivary pH,buffering capacity and flow rate levels of saliva in caries free people and in people with dental caries among different age groups .

Materials and Methods: The saliva samples of 120 children were collected with the aid of micropipette with disposable tips and plastic cylindersfor5 minutes. Salivary flow was determined by dividing the volume collected by the time of aspiration. All saliva produced was stored into a sterile plastic cylinder .The measurement of pH was performed with the aid of a digital pH meter and buffering capacity of saliva was measured.

Results: There was a significant decrease in the mean salivary flow rate, salivary pH and salivary buffer capacityinpeople with dental caries compared to caries free people among different age groups, P-value is statistically significant (p value - <0.05) in case of salivary pH and buffering capacity , whereas it is not significant in case of salivary flow rate (p value - >0.04).

Conclusion : The physicochemical properties of saliva, such as salivary flow rate, pH, buffering capacity and viscosity, has a relation with caries activity in people and act as markers of caries activity.

Keywords: *Buffering capacity,saliva,pH,flowrate,samples.*

Introduction

Saliva, a heterogeneous fluid comprising proteins, glycoproteins, electrolytes, small organic molecules and compounds transported from the blood, constantly bathes the teeth and oral mucosa. Whole saliva represents a mixture of the secretions of the major (submandibular, sublingual, parotid) and minor (accessory) salivary glands, together with the gingival fluid[1].Saliva is essential for the maintenance of oral health and it is an important diagnostic biofluid[1].It plays a pivotal role in protection and lubrication of oral mucosal tissues, remineralization of teeth, and alimentation[2,3].The composition of saliva gives it many important physical and biochemical properties[3].It is useful for diagnosis, prognosis, and management of patients with oral and systemic diseases[3].There is increasing inclination toward using saliva samples for the diagnosis of oral and systemic diseases[4].

Among the oral diseases, dental caries is the most common chronic disease of mankind[1,2].It is the main oral health problem in industrialized countries[3].Saliva definitely promotes oral health and hence lack of its secretion contributes to the disease process. The saliva which constantly moisturise the teeth and oral mucosa, functions as a cleansing solution, a lubricant, a buffer and an ion reservoir of calcium and phosphate, which are essential for the remineralization of initial carious lesions[1,2].A basal unstimulated secretion is produced continuously to moisturize and lubricate the oral tissues for more than 90 percent of the day[2,4].The normal resting salivary flow rate ranges from 0.25 to 0.35 milliliter per minute. Mechanical, gustatory, olfactory or pharmacological stimuli increase the production and secretion of saliva. Stimulated saliva represents 80 to 90 percent of daily salivary production, and the stimulated flow rate varies from 1 to 3 mL/minute[3].The salivary pH and the salivary buffering capacity are determined by the hydrogen bicarbonate balance in saliva. Salivary pH is approximately neutral, and buffering agents, such as inorganic phosphate in resting saliva and carbonic acid-bicarbonate system in stimulated saliva, help maintain neutrality[4].

Among the various protective functions of saliva, including diluting and cleaning the oral cavity, serving as a host defense, and buffering and enabling ion exchange, certain salivary characteristics outside the normal range of values may contribute to the caries process[4].Dental caries results from the dissolution of minerals from the tooth surface by organic acids formed from the bacterial fermentation of sugars.

Saliva maintains the integrity of oral hard and soft tissues and protects against immunologic bacterial, fungal and viral infections[5].Saliva controls the equilibrium between demineralization and remineralization in a cariogenic environment. Salivary buffers can reverse the low pH in plaque

and allow for oral clearance thus preventing demineralization of enamel. The flow rate and viscosity of saliva may also influence the development of caries[5].

The purpose of this study was to evaluate the relationship between the physiochemical properties of saliva such as flow rate, pH, buffering capacity in caries-free people and in people with dental caries among different age groups .

Materials andMethods

The study population consisted of 120 healthy children aged 7–15 years that was further divided into two groups:Group 1(7–10)years caries free and active children ,Group 2 (11–15) years caries free and active children. Unstimulated saliva samples were collected from all groups. Flow rates were determined, and samples analyzed for pH, buffer capacity.

Inclusion criteria :(1) children should be free from systemic or local diseases that affect salivary gland secretions (such as submandibular duct canaliculi, asthma and diabetes) and (2) children should be consuming only municipal water (those consuming hard water were not included as hard water consumption predisposes to dental fluorosis).

Exclusion criteria were on any medication for current and past illness and oral status other than dental caries like ulcers, oral tumors, herpetic lesions. Prior consent was obtained from the respective school authorities and from the parents through the school to conduct the study. The examinations were carried out in the subjects own surroundings that is, the school. The examination for dental caries was made according to the dentition status, and treatment.

Salivary Analysis

Collection of Saliva and Estimationof Salivary Flow Rate

Twelve milliliters of stimulatedwhole saliva was collected for the study.Salivawas stimulated by making the children to chew the paraffin wax for about 1 minute before spitting the saliva and the sample collection was carried out in the day time between 10 am and 12 pm, 2 h after breakfast. Then, the children were made to sit comfortably in a ventilated and well-illuminated room, and were instructed to spit the saliva, which was collected for exactly 5 min in a pre-weighed graduated cylinder. A note was made of this value. Saliva [redacted] collection was then continued till [redacted] [redacted] 12 mL of saliva was accumulated in the cylinder.Salivaryflow rate was calculated in gms/ml which is almost equivalent to ml/min.

Figure1: Collectionof Salivary Samples from the Subjects

Figure 2: Collection of Salivary Samplesin Plastic Cylinders

Estimation of pH and Buffering Capacity

• pH of saliva was measured by using digital pH me-ter.

• Buffering capacity of saliva {by Ericsson method 1959}:-0.5 ml of saliva was added to 1.5 ml of 5mmol/l HCl. The mixture was vigorously shaken and then centrifuged for one minute and allowed to stand for 10 min when the final pH of supernatant was measured by using manual pH meter (6)

Figure 3: Estimation of Salivary pHFig4:BufferingCapacity Analysis

Statistical Analysis

Statistical analysis of the data was performed with Student's t-test. Differences with P-values were considered to benot statistically significant.

Results

Salivary Flow Rate

Group 1

The mean level of flow rate in Group 1 Caries Free children is (3.61 +/- 0.58) , the mean level of flowrate in Group 1 Caries Active children is (3.22+/-0.23)therefore , p- value for group 1 is 0.4433 , which is not Statistically significant (p value > 0.04) by the rule of one – tailed hypothesis.

Group 2

The mean level of flow rate in Group 2Caries free children is (3.45+/- 0.2) and the mean level flow rate in Group 2 Caries active children is (3.25+/0.3)The mean level of flow rate is decreased in caries active children when compared to caries free children ,therefore the p – value is 0.467, it is not statisticallysignificant (p value >0.04) by the rule of one – tailed hypothesis. (Table-1)

Salivary pH

Group 1

The mean level of pH in Group 1 Caries Free children is (7.6+/0.94) , the mean level of pH in Group 1 Caries active children is (6.24+/-0.13),asthe p value is 0.000112 , therefore it is statistically significant (p value - <0.05) by the rule of one tailed hypothesis.

Group 2

The mean level of pH in Group 2 Caries free children is(7.13+/-0.13)and the pH in group 2 caries active children is (5.4+/-0.32)The mean level of pH is decreased in caries active children when compared to caries free children as the p value is 0.000306, and it is statistically significant(p value <0.05)by one tailed hypothesis .(Table-2)

Salivary Buffering Capacity

Group 1

The mean level of Buffering capacity inGroup 1 Caries Free children is (5.9+/0.53), and the mean level of buffering capacity in Group 1 Caries active children is (2.7+/-0.33), as the p - value is 0.006491, p value is statistically significant (p value <0.05)by one tailed hypothesis.

Group 2

The mean level of Buffering capacity in Group 2 Caries free children is (5.13+/-0.15) and the buffering capacity in Group 2 individuals is (3.5+/-0.21). Therefore the mean level of buffering

capacity is decreased in caries active children when com-pared to caries free children , p value is 0.004283,and it is statistically significant (p value <0.05) by one tailed hypothesis . (Table-3)

On observation from the below graphs : Series 1 represents the caries active children and series 2 represents caries free children .Graph 1 indicates salivary pHdecreased in caries active children when compared with caries free children which represents Table -3 , Graph 2 indicates salivary flow rate slightly decreased in caries active children when compared with caries free children which represents Table -1, Graph -3 indicates salivary buffering capacity decreased in caries active children when compared with caries free children which represents Table -2 .

Table 1: Shows the Salivary Flow Rate of Group 1 and Group 2 Caries Free and Caries Active Children

Groups				
Salivary flow rate	Mean	SD	T -value	P value
Dental caries free individuals				
Group 1 - age group (7-10 years)	3.61	0.58	1.432	(> 0.04)
Group 2 -age group (11- 15 years)	3.45	0.2	1.35	(>0.04)
Dental caries active children				
Group 1 - age group (7-10 years)	3.22	0.23	1.23	(>0.04)
Group 2 -age group (11- 15 years)	3.25	0.3	1.25	(>0.04

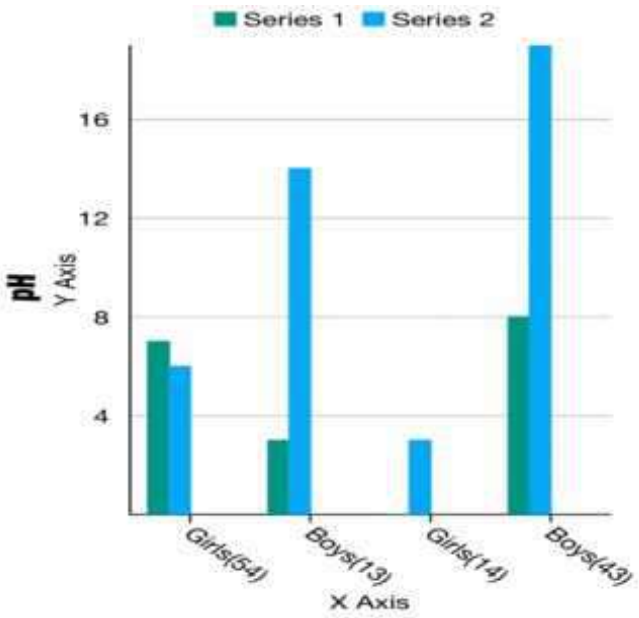
Table 2: Shows the Salivary pH of Group 1 and Group 2 Caries Free and Caries

Groups				
Salivary Buffering capacity	Mean	SD	T -value	P value
Dental caries free individuals				
Group 1 - age group (7-10 years)	5.9	0.53	2.44	(<0.05)
Group 2 -age group (11- 15 years)	5.13	0.15	2.3	(<0.05)
Dental caries active children				
Group 1 - age group (7-10 years)	2.7	0.33	0.8	(<0.05)
Group 2 -age group (11- 15 years)	3.5	0.21	1.43	(<0.05)

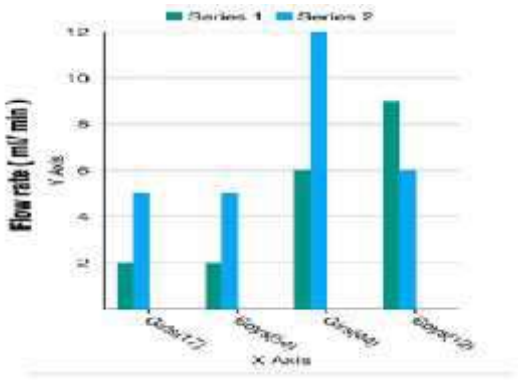
ActiveChildren

Table 3: Shows the Salivary Buffering Capacity of Group 1 and Group 2 Caries Free and Caries Active Children

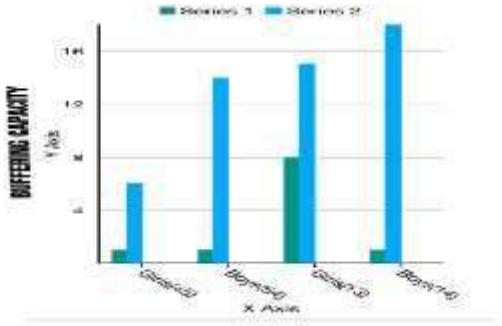
Groups				
Salivary pH	**Mean**	**SD**	**T -value**	**P value**
Dental caries free individuals				
Group 1 - age group (7-10 years)	7.6	0.94	3.55	(<0.05)
Group 2 -age group (11- 15 years)	7.13	0.13	3.45	(<0.05)
Dental caries active children				
Group 1 - age group (7-10 years)	6.24	0.13	2.54	(<0.05)
Group 2 -age group (11- 15 years)	5.4	0.32	1.97	(<0.05)



Graph 1: Shows the comparison of salivary pH between group 1 and group 2 caries active and caries Free Individuals



Graph 2: Shows the Comparison of SalivaryFlow Rate Between group 1 and group 2 Caries Active and Caries Free Individuals



Graph 3: Shows the comparison of salivary buffering capacity between group 1 and group 2 Caries Active and Caries Free Individuals

Discussion

Human saliva is an oral fluid that has several functions involved in oral health and homeostasis, with an active protective role in maintaining oral health[15].Saliva helps bolus formation by moistening food, protects the oral mucosa against mechanical damage and plays a role in the preliminary digestion of food through the presence of α-amylase and other enzymes. It also facilitates taste perception and also has a role in maintaining teeth enamel mineralization[14].

Dental caries has been thought of as a multifactorial disease as it is not only influenced by dietary factors but host factors as well documented[13].These defense systems include clearance, buffering, antimicrobial agents and calcium and phosphate delivery for remineralization, to name a few[12].The interaction of protective and pathologic factors in saliva and plaque biofilm, as well as the balance between the cariogenic and non-cariogenic microbial populations that reside in saliva,

decides the caries process[10].The factors in saliva most frequently related to dental caries are: (a) aciduric/acidogenic bacteria and (b) rate of acid production in the presence of glucose. Other factors that have been suggested as being related to dental caries include (a) amount of saliva secreted in a given time and (b) acid-neutralizing ability (buffering capacity) of saliva[14].

SalivarypH

In the present study , there was a significant difference in the mean salivary pH among the study groups Group 1 and Group 2 in both the caries free and caries active children and it is Statistically significant (p value < 0.05) . The results obtained are in accordance with the studies performed by Prabhakar in 2009[5] and Preethiin 2010[1].However, the results obtained in their studies were not significant. The salivary pH was only slightly reduced in caries-active children compared with caries-free children. Another study by Zhou in 2007[9]showed that the pH of saliva from early childhood caries children was statistically higher than that in caries-free children. In contrast, a study carried out by Thaweboonin 2008[8].revealed that the mean values for salivary pH were similar in caries-free and rampant-caries children. Swerdlove in 2013[3]andMalekipourin 2008[8].reported no relationship between the incidence of dental caries and the pH of normal resting saliva. Lambertsin 2005[12]observed no relationship of salivary pH rise activity and caries experience in caries-free and caries-active subjects[13].

Salivary Buffering Capacity

In the present study , there was a significant difference in the mean salivary buffering capacity among the study groups Group 1 and Group 2 in both the caries free and caries active children (p value <0.05) and it is Statistically significant (p value <0.05).The studies performed by Prabhakar in 2009[5]and Preethi in 2010[1]; Group I had a significantly higher mean salivary buffering capacity than that of Groups II and III. However, no significant difference was seen between the mean salivary buffering capacities of Group II and Group III. However, the results obtained in their studies were not significant[5].The salivary buffering capacity was only slightly reduced in caries-active children compared with caries-free children. Another study by Zhou in 2007[9]showed that the buffering capacity of saliva from early childhood caries children was statistically higher than that in caries-free children. A study performed by Malekipourin 2008[4]showed similar results, although the difference was not statistically significant[15].

Salivary Flow Rate

In the present study , there was only a small amount of significant difference in the mean salivary flow rate among the study groups Group 1 and Group 2 in both the caries free and caries active children (p value > 0.04) and it is not Statistically significant . Whereas, in the similar study of Lopez in 2003[2]therewas a significant difference in the mean salivary flow rate among the study groups ($P < 0.0001$). Group I had a significantly higher mean salivary flow rate than that of Groups II and III. Similarly, Group II had a significantly higher mean salivary flow rate than Group III[5].reported a salivary flow rate of 0.27 ± 0.14 in a group of children aged 5-12 years. The results obtained are in accordance with the studies carried out by Preethi in 2010[1]andPrabhakar in 2009[5]results obtained in their studies were not statistically significant. The salivary flow rate was only slightly reduced in caries-active children compared with caries-free people[18]. In contrast, a study performed by Thaweboon in 2008[8] revealed that the mean values for salivary flow rate were similar in caries-free and caries active people. The salivary flow rate did not influence the presence of caries[20].

Conclusion

Dental caries is a complex and dynamic process where a multitude of factors influence and initiate the progression of disease. One of the most important factor which influences the development of dental caries is saliva. Alterations in the physicochemical properties of saliva such as decreased salivary pH, buffering capacity play a major role in the development of caries, whereas salivary flow rate contribute only a small role in caries development. These results re-emphasize that there is a relationship of the various physiochemical properties of saliva, such as salivary flow rate, pH and buffering capacity along with the caries activity in the oral cavity.

Corresponding Author

Sreedevi. D,

Senior Lecturer ,Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University

162,Poonamallee High Road , Chennai 600077, Tamil Nadu, India

Email ID: jayaceram@gmail.com,

Telephone Number :9600586858

References

1. Preethi. Saliva: Its secretion, composition and functions and its relation with tooth structures. Br Dent J 2010; 172: 305-310
2. Lopez AVN, Bolscher JGM, VeermanECI.Sali-vary proteins: Protective and diagnostic value in cari-ology. Caries Res 2003; 38: 247-253.
3. Swerdlove ,Loimaranta,V. Saliva and Dental Caries. Adv Dent Res 2013: 14: 40-47.
4. Malekipour ID. The functions of saliva. J Dent Res 2008; 66: 623-662.
5. Prabhakar ,Aaltonen AS, ViljaPandTuohimma P. Antimicrobial factors in whole saliva of human infants. Infection and Immunity and its association with caries activity. 2010; 51: 49-53
6. PriorRL,CoaG.In vivo total antioxidant capacity: Comparission of different analyticalmethods. Free Radical Biology and Medicine 1999; 27:1173-1181.
7. Moore S, Calder KA,Miller NJ and Rice Evans CA. Antioxidant activity of saliva and periondontal disease. Free Radical Research 1997; 26:209-234.
8. ThaweboonO,DemirtasS,TulunogluI.Total salivary parameter levels of saliva in children related to caries, age and gender. Int J Paed Dent 2008; 16:186-191.
9. Zhou, Avery DR, Stookey GK. Dental caries in the child and adolescents and its relation with saliva In: Mc-Donald RE, Avery DR, Dean JA, editor. Dentistry for the child and ado-lescent. 8th ed. New Delhi: Elsevier; 2007:203-235.
10. Shafer W.G, Hine M.K and Levy B.M. ED. A text book of oral pathology 5th Ed .Philadelphia W. B. Saunders company.1993; 567-658.
11. Lavelle. L.B. Christophe Applied oral physiology, 2nd edition. Butterworths and Co (publishers) LTD 1988:128-141.
12. Lamberts ,McDevitt. Saliva as the next best diagnostic tool.Journal of Biochem.2005; 45(2):23-25.
13. Battino M, Ferreior MS, Gallardo I, Newman HN, Bul-lon P. The antioxidant capacity of saliva. J ClinPerio-dontol 2002; 29:189-194
14. Gopinath VK, Arzreanne. Saliva as a diagnostic tool for assessment of dental caries. Archives of Orofacial Sciences 2006; 1:57-59.
15. Thaweboon S, Thaweboon B, Nakornchai S, Jitmaitree S. Salivary secretory IgA, pH, flow rates, mutans streptococci and Candida in children with rampant caries. Southeast Asian J Trop Med Public Health. 2008;39:893–9.
16. Shafer WG, Hine MK, Levy BM. Dental caries. In: Sivapathasundharam B, Raghu AR, editors. A Textbook of Oral Pathology. 5th ed. Philadelphia: WB Saunders Company; 1993:567–8.

17. Nikiforuk G. Clinical features and classification of dental caries. In: Nikiforuk G, editor. Understanding Dental Caries Etiology and Mechanisms: Basics and Clinical Aspects.New York: Karger; 1985:10
18. AmanthiGanapathi ,Study Of Enzymatic Biomarkers To Evaluate Periodontal Severity , Pharm. Sci. & Res. 2016; 8(7):696-699.
19. Gokul, Vishnupriya, gayathri,A study on Variations of salivary pH with Intake of food,Asian journal of pharmaceutical science and clinical research 2016; 9(1):98-107
20. Nithinmanchery,Joseph John j ,Salivary cariogenic microflora,buffercapacity,secretion rate and its relation to caries experience, contempt dent 2015; 5(1):12-16

Evaluation of Surface Erosion of Flowable Resin Composite byAcidic Juices- An Invitro Study

Abiraamasri BL[1], Jayalakshmi Somasundaram[2], DhanalakshmiRavikumar[3]
Graduate Student[1], Senior Lecturer[2,] Department of Conservative and Endodontics,
Senior Lecturer[3], Department of Pedodontics and Preventive Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The restoration of lost teeth is usually desired for two primary reasons – esthetics and to restore the function. The material used to restore the function must also be esthetically appealing. In recent years, flowable resins with lower filler content have been developed. Flowable resins have a composition similar to conventional resins with decreased viscosity due to lower filler content. Oral cavity is a challenging environment, which is exposed to fluids of varying pH and thus exposing the composite restoration to the challenging atmosphere.The aim of the study is to evaluate the surface morphological changes of flowable composite resins when exposed to acidic juices.

Materials and Method: Forty discs (7mmX2mm) of 2 different brands of flowable composites (NanoceramBrightFlow and Tetric N-ceram) of uniform shade A2 were prepared. Discs were immersed in distilled water for 24 hours for hydration. The specimens from each category were randomly divided into 4groups – group 1 lime juice, group 2 orange juice, group 3 coke and group 4 distilled water. The baseline values of the specimens were recorded using a colorimeter and then immersed in the respective experimental fluids. Values were recorded after 7 days and 14 days after immersion.

Result: Out of the two composites used, Tetric N-ceram showed more resistance to surface erosion by acidic juices. Of the three experimental acidic fluids, lime showed higher surface erosion in both the categories. The minimum surface erosion of composite among the different experimental fluids was seen in distilled water followed by coke.

Conclusion: The present study concludes that, frequent exposure to acidic juices should be avoided to reduce the risk of surface erosion of the flowable composites.

Key Words: *surface erosion, acidic juices, and resin composite.*

Introduction

Composite resin restorations came into existence into dentistry since 1990s and have evolved to a greater extent till now. Based on consistency, composite resins can be broadly classified into packable and flowable composite resins. Flowable composite resins have a similar composition to conventional resins except for the filler load which has been reduced to 37%-53%[1]. This filler load modifies the viscosity of the flowable resin composite[2]. Most of the manufacturers pack the flowable resin composite in syringes for easy dispensing with small gauge needles. This also makes them ideal for use in small preparations [3,4]. Acid erosion, also known as dental erosion, is a type of tooth wear and it is defined as the irreversible loss of tooth structure due to chemical dissolution by acids not of bacterial origin. Dental erosion is one of the most common chronic disease of children between the age group of 5-17yrs[5] though it is onlyrecently been recognized as a dental health problem [6]. There is a widespread ignorance of the damaging effects of acid erosion generally; this is specific in the case with erosion due to fruit juices, because they are tend to be seen as healthy[7]There has been considerable attention in recent times focused on the problems of tooth surface loss in both adults and children[8]. Dental erosion is one of the common conditions, which seems to be trending higher in recent decades[9]. In certain studies which have reported the prevalence of dental erosion in different age groups,states that there is a clear trend of increasing prevalence with age in children[10]. Acidic foods and drinks are the most common substance which causes dental erosion. In general, the pH level of food and drinks are below 5.0– 5.7 which have been known to trigger dental erosion effects[11]. Numerous

clinical and laboratory reports shows a link between erosion to excessive consumption of drinks. Additionally, wine has been shown to erode teeth, with the pH of wine as low as 3.0–3.8[12].

Indications of Flowable Resins

1) Class 1 restoration.
2) Cavity base or a liner for class II cavity.
3) Pit and fissure sealant.
4) Class V abfraction.
5) Bonding of orthodontic brackets.
6) Bonding of lingual orthodontic retainers.
7) Reattachment of fractured restorations.
8) Splinting fractured and mobile teeth.
9) Minimally invasive class III.
10Lutting porcelain and composite resin veneers[13].

Materials and Methods

Sample Preparation

40 small discs were prepared (7mmx2mm dimensions). Two different types of Nano hybrid flowable resin composites of A2 shade were used (group 1 – Nano ceram Bright Flow) (group 2 – Tetric N-ceram). The flowable resin composite was packed in a Teflon-mould and finger pressure was given with Mylar strip placed over it and polymerized with Light curing unit for 40seconds. The discs were finished with finishing bur of yellow grade and polished with shofu composite polishing kit in two sequences. Then the discs were immersed in distilled water for 24 hours to maintain hydration.

Experimental Fluids

Experimental fluids used in the study were lime juice, orange juice and coke. Lime juice and orange juice were diluted to a palatable state and their pH values were recorded using a pH meter (Table.1).

Experimental Groups

A baseline value was first recorded for all groups using a colorimeter, and then group 1 (Nano-ceram) and Group 2 (Tetric N-ceram) were divided into 4 subgroups and immersed into the distilled water, orange juice, lime juice and coke. After 7 days, the discs were taken out of the solution using tweezers and washed in distilled water; dried and the values were recorded. Disc were again immersed in to the respective solutions and recorded after 14 days and the values were measured.

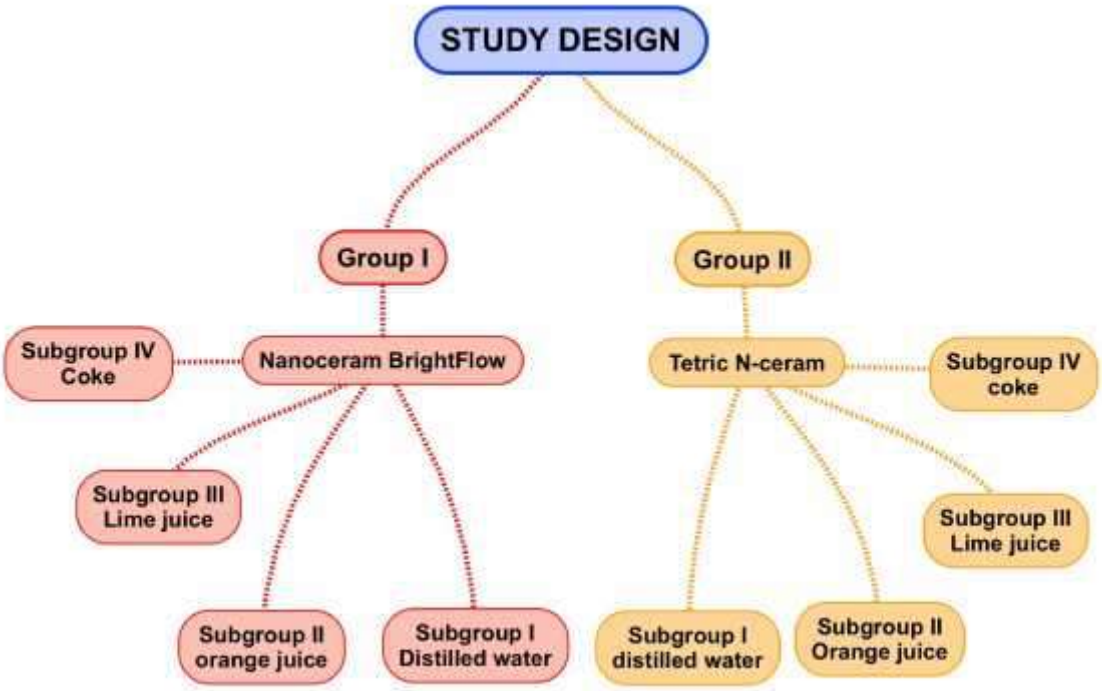


Figure 1: Flow chart depicting the study design

Result

The data were used to determine mean and standard deviation. The data was used to analyze with one-way ANOVA. From the statistical data, it was found that, there was no significant change in the values of day 7 when compared to control values, indicating that surface erosion has not taken place within the first 7 days. Specimens showed no significant change to coke in all the groups at all the days. But there were some significant changes in values, when compared with distilled water, which showed the least change.

The group that showed the highest susceptibility to surface erosion was group 2 Tetric N-flow. It showed surface erosion to lime juice on all days, which is high when compared to orange juice and coke. In group 1 Nano-ceram, orange juice and lime juice showed slightly more erosion when compared to coke. The experimental fluid that showed least surface erosion in both the group is coke. When comparing Tetric en flow and Nano Ceram for both after one week and after two weeks there was no significant p value.

Table.1: pH of the experimental fluids

EXPERIMENTAL FLUIDS	pH
Subgroup 1 (water)	7
Subgroup 2	3.4
Subgroup 3	3.6
Subgroup 4	2.53

Table-2: Mean and standard deviation of surface erosion of Tetric en flow and Nano Ceram for after one week and after two weeks observation.

TETRIC EN FLOW	SUBGROUPS	NO. OF SAMPLE (N)	MEAN	STD.DEVIATION
AFTER ONE WEEK	Subgroup 1	5	0.4000	0.5477
	Subgroup 2	5	0.0100	0.0071
	Subgroup 3	5	0.0080	0.0045
	Subgroup 4	5	0.0100	0.0071

AFTER TWO WEEKS	Subgroup 1	5	0.00	0.00
	Subgroup 2	5	0.98	2.02
	Subgroup 3	5	4.95	80.43
	Subgroup 4	5	0.99	2.02

NANO CERAM	SUBGROUPS	NO. OF SAMPLE (N)	MEAN	STD.DEVIATION
AFTER ONE WEEK	Subgroup 1	5	1.0000	0.0000
	Subgroup 2	5	0.0080	0.0084
	Subgroup 3	5	0.0040	0.0055
	Subgroup 4	5	0.0080	0.0084
AFTER TWO WEEKS	Subgroup 1	5	0.00	0.00
	Subgroup 2	5	1.19	0.81
	Subgroup 3	5	1.19	0.80
	Subgroup 4	5	0.78	0.81

Table -3: One way Anova to compare the surface erosion of Tetric en flow and Nano Ceram between one and two weeks (within the groups).

MATERIALS	**COMPARISON BETWEEN AFTER ONE AND TWO WEEKS (p value)**
TETRIC EN FLOW	0.97
NANO CERAM	0.97

Table – 4 One way Anova to compare the surface erosion of Tetric en flow and Nano ceram at different time interval (between the groups).

DURATION	**COMPARISON BETWEEN TETRIC EN FLOW AND NANO CERAM**

AFTER ONE WEEK	0.22
AFTER TWO WEEKS	0.21

Discussion

Oral cavity is the challenging environment, which is exposed to fluids of varying pH. Many soft drinks are acidic and the pH is 3.0 or lower. This means that drinking acidic drinks over a long period and with continuous sipping can erode the tooth enamel and the resin material as well[15]. Flowable composite resins have similar composition to conventional resins except in filler content. When flowable composites are exposed to fluids of varying pH for a prolonged period, there is a chance of surface erosion of flowable composites. Surface erosion can lead to discoloration, secondary caries, leakageand plaqueaccumulation[16].

The studyconducted by Lee (1994) shows that fillers tend to fall out from resin materials, and the matrix component decomposes when exposed to low pH environments.[17,18]

Wongkhantee (2005) conducted a study to observe the behavior of varies composite restoration material when exposed to many foods and drinks (e.g., Cola soft drink, drinking yogurt, orange juice, sports drink, Tom-yum soup) that affects the behavior of restorative materials. The result showed that Cola soft drink significantly reduced surface hardness of enamel, dentine and composite surface. Orange juice and sports drink significantly reduced surface hardness of enamel and drinking yogurt and Tom-yum soup did not reduce surface hardness. This invitro study confirms the erosive potential of certain acidic food and drinks that public should be aware of. [19] When comparing the present study with this study, cola showed no drastic change in the surface erosion of composite even after two weeks in the present study.

Linlin HAN (2008) performed a study to evaluate the morphological changes of surfaces of flowable resins eroded by acidic juices like orange juice, wine and whisky. The result

showed similar surface roughness in the specimens eroded with orange juice and wine. Among all the experimental fluids- orange juice, wine and whisky, orange juice and wine showed higher surface roughness. The author concluded that one cause was the fall out of fillers which is due to insufficient surface treatment of fillers with silane[20].

Campbell reported a linear relationship between the optical scattering coefficient and the filler concentration. Chemical differences among the resin components, such as purity of the oligomers and monomers and concentration/type of activators, initiators, inhibitors, oxidation of unreacted carbon – carbon double bonds and fillers may affect the color stability as well[21].

Majewski (2001), had stated regarding the site of erosion, the prevalence on the buccal surfaces was higher – the reason for this is unclear. The Soft drinks have many potential health problems, including dental caries and enamel erosion [22].

Bowen and Lawrence (2005),had stated that the most frequent source of the acids is soft drinks like cola. It is also indicated that the cariogenicity of cola is higher than that of milk andsucrose [23].

Edwards (1999); Owens (2007), compared caries and dental erosion. He stated that dental erosion seems to have much stronger relationship with soft drinks than dental caries. The erosive potential of drinks is mainly represented by their pH and the buffering capacity. Carbonated drinks had lower pH than fruit juices. The buffering capacities are in the following order: fruit juices>fruit-based carbonated drinks>non-fruit-based carbonated drinks [24]

Lussi (1995); Lippert(2004); Jensdottir(2005), stated that even the sports drinks have a stronger softening effect than fruit juices [25,26,27].

Hara and Zero (2006),had concluded that some of the supplements in the drink, such as calcium, could reduce the progress of enamel demineralization [28].

Conclusion

In the era of esthetic dentistry, flowable composites form an in-homogenous group of materials. They exhibit a wide variety in their composition and consequently a variety of mechanical and physical properties. According to this research study, frequent exposure to acidic juices should be avoided to reduce the risk of surface erosion of the flowable composites. They are used in a wide variety of clinical situations and are subjected to various challenges in the oral cavity.

Corresponding Author

Dhanalakshmi Ravikumar,

Senior Lecturer,Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email Id: dhana9677@gmail.com

Phone Number: 9597011442.

Reference

1. H. Linlin, O. Akira, F. Masayoshi andO.Takashi, Evaluation of Flowable Resin Composite Surfaces Eroded by Acidic and Alcoholic Drinks, Division of Cariology, Operative Dentistry and Endodontics, Department of Oral Health Science, Niigata University Graduate School of Medical and Dental Sciences, 26(6):906-14 · December 2007.
2. Baroudi K, Saleh AM, Silikas N, Watts DC. Shrinkage behavior of flowable resin-composites related to conversion and filler-fraction. J Dent 2007; 26: 651-655.
3. MahamuthaAffshana, Acid Erosion of Teeth in a Pediatric Population, J. Pharm. Sci. and Res. Vol. 7(7), 2015, 435-438.
4. Wickens J L. Dealing with failures. Br Dent J 1999; 186: 443-446.

5. Syed NaziaHussainy, Dentinal Hypersensitivity: A Detailed Review, J clin dent sci2016;1(2).
6. Gayathri R Menon, Karthikeyan, G. and James D Raj , Recent advancement in restorative composite materials, Ijras,Vol. 03, Issue 06, pp.1528-1530, June, 2016
7. IffatNasim, PrasannaNeelakantan,R.Sujeer,C.V.Subbarao, Color stability of microfilled, microhybrid and nanocomposite resins—An in vitro study, Journal of dentistry, Volume 38, supplement 2,2010.
8. Y Madhukar Rao, An evaluation of the color stability of tooth-colored restorative materials after bleaching using CIELAB color technique, Ijdr- Year : 2009 | Volume : 20 | Issue : 1 | Page : 60-64.
9. Milosevic A. Eating disorders and dentist. Br Dent J 1999; 186: 109-113. Shaw L, Smith, A J. Dental erosion — the problem and some practical solutions. Br Dent J. 1999; 186: 115-118.
10. Capp N J. Occlusion and splint therapy. Br Dent J 1999; 186: 217-222. Faigenblum, M. Removable prostheses. Br Dent J 1999; 186: 273- 276.
11. King P A. Adhesive techniques. Br Dent J 1999; 186: 321-326. Wickens J L. Prevention and maintenance. Br Dent J 1999; 186: 371-376.
12. Harley K. Tooth wear in the child and the youth. Br Dent J 1999; 186: 492-496.
13. García AH, Lozano MAM, Vila JC, Escribano AB, FosGalve P. Composite resins. A review of the materials and clinical indications. Medicina Oral Patologia Oral Y CirugiaBucal. 2006;11:E215–20
14. Neamat AB, Han L, Okamoto A, Iwaku M. Effect of alcoholic and low pH soft drinks on fluoride release from compomer. J Esthet Dent 2000; 12: 97-104.
15. Kitchens M, Owens BM. Effect of carbonated beverages, coffee, sports and high energy drinks, and bottled water on the in vitro erosion characteristics of dental enamel. J ClinPediatr Dent 2007; 31: 153- 159.
16. Unterbrink GL, Lienbenberg WH. Flowable resin composites as “filled adhesives”: Literature review and clinical recommendations. Quintessence International. 1999;30:249–57]

17. Lee SY, Greener EH, Mueller HJ, Chiu CH. Effect of food and oral simulating fluids on dentine bond and composite strength. J Dent 1994; 22: 352-359.
18. Bayne SC, Thompson JY, Swift EJ Jr, Stamatiades P, Wilkerson M. A characterization of first- generation flowable composites. J Am Dent Assoc 1998; 129: 567-577.
19. Wongkhantee S, Patanapiradej V, Maneenut C, Tantbirojn D. Effect of acidic food and drinks on surface hardness of enamel, dentine, and tooth- colored filling materials. J Dent 2005; 34: 214-220.
20. Linlin Han, Akira Okamoto, Masayoshi Fukushima And Takashi Okiji. Evaluation of Flowable Resin Composite Surfaces Eroded by Acidic and Alcoholic Drinks. Dent Mat J 2008; 27(3): 455－465
21. Campbell PM, Johnston WM, O'Brien WJ. Light scattering and gloss of an experimental quartz-filled composite. J Dent Res 1986;65:892 – 4.
22. Bowen WH, Lawrence RA. Comparison of the cariogenicity of cola, honey, cow milk, human milk, and sucrose. Pediatrics. 2005;116(4):921–926. doi: 10.1542/peds.2004-2462.
23. Majewski RF. Adolescent caries: a discussion on diet and other factors, including soft drink consumption. J Mich Dent Assoc. 2001;83(2):32–34.
24. Edwards M, Creanor SL, Foye RH, Gilmour WH. Buffering capacities of soft drinks: the potential influence on dental erosion. J Oral Rehabil. 1999;26(12):923–927. doi: 10.1046/j.1365-2842.1999.00494.x.
25. Lussi A, Jaeggi T, Scharer S. Prediction of the erosive potential of some beverages. Caries Res. 1995;29(5):349–954.
26. Lippert F, Parker DM, Jandt KD. Susceptibility of deciduous and permanent enamel to dietary acid-induced erosion studied with atomic force microscopy nanoindentation. Eur J Oral Sci. 2004;112(1):61–66. doi: 10.1111/j.0909-8836.2004.00095.x.
27. Jensdottir T, Bardow A, Holbrook P. Properties and modification of soft drinks in relation to their erosive potential in vitro. J Dent. 2005;33(7):569–575. doi: 10.1016/j.jdent.2004.12.002.

28. Hara AT, Zero DT. Analysis of the erosive potential of calcium-containing acidic beverages. Am J Dent. 2006;19(6):319–325.

Habit-Associated Salivary pH Changes in Premalignant and Malignant Conditions

D. Shruthi[1], Abilasha[2], Antonette Rhea[3],
Graduate Student[1], Senior Lecturer[2], Department of Oral Pathology, Senior Lecturer[3], Department of Prosthodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Saliva, a multi constituent oral fluid, has high potential for surveillance of general health and disease. Approximately of 700ml of saliva secreted daily is contributed by submandibular gland accounts for about 60%, parotid gland for about 30%, sublingual gland for about 5% or less and 7% from minor salivary glands. Saliva has a normal pH range of 6.5 - 7.6, the average pH is 6.7. Saliva acts as buffering agent and maintains the pH of oral cavity and prevents from environmental factors that may disturb the oral pH that enhances growth of microorganisms. The aim of this study is to analyze the salivary pH and determine its correlation in habit associated malignant and premalignant conditions.

Material and Methods: The study is conducted to assess the salivary pH among habit associated premalignant and malignant patients with apparently healthy control group. Saliva samples will be collected from the subjects and pH will be analyzed using pH strips.

Result: There was pH changes in certain cases on study but on statistical analysis there was no significance between pH of any conditions

Conclusion: Sample sized result showed there was no significance between pH of any conditions considered.

Key Words: *Salivary pH, habit, oral submucous fibrosis, leukoplakia, cancer*.

Introduction

Cancers are the most common cause of death in adults. Oral cancer is a heterogeneous group of cancer arising from different parts of the oral cavity, with predisposing factors, prevalence, and treatment outcomes. According to statistics, in 2012 incidence of oral cancer in India is 53842 in males and 231631 in females [1]. In India and Southeast Asia, the chronic use of betel quid has been strongly associated an increased risk for oral cancer [2]. Oral cancers can be diagnosed with early manifestations. The premalignant conditions are very important to be diagnosed as it can prevent a patient to become a cancer patient. The most common premalignant conditions of oral cancers are oral submucous fibrosis, leukoplakia, erythroplakia, oral lichen planus, and etc. Leukoplakia is described as a white patch or plaque that cannot be characterized clinically or pathologically as any other disease [3]. Several studies have shown leukoplakia has more malignant transformation rates ranging from 8.9 to 17.5 percent [4].Oral submucous fibrosis is a chronic fibrosing disease with progressive and unrelenting clinical course that leads to mucosal stiffness and reduced mouth opening. The initial presentation of OSF is inflammation. Inflammation is followed by hypovascularity and fibrosis visible as blanching of the oral mucosa with a marble-like appearance. Blanching may be localized, diffuse, or reticular. In some cases, small vesicles may develop that rupture and form erosions [5]. The predisposing factors of these conditions are smoking, alcohol intake, arecanut chewing, smokeless tobacco chewing, and etc. The most common is arecanut in combination with tobacco. Major constituents of betel quid are arecoline from betel nuts and copper, which are responsible for fibroblast dysfunction and fibrosis. Saliva, a multi constituent oral fluid, has high potential for surveillance of general health and disease. It has a normal pH range of 6.5 - 7.6, the average pH is 6.7. Alteration in salivary pH has a significant impact on orodental health. The aim of this study is analyze the salivary pH and determine its correlation in habit associated malignant and premalignant conditions [6].

Materials and Methods

The study was conducted to assess pH among habit associated malignant and premalignant patients with apparently healthy control group.

Selection of Subjects

A total of 30 subjects were included in this study of which 5 were oral cancer patients , 5 were oral submucous fibrosis patients , 5 were leukoplakia patients associated with adverse habits such as areca nut chewing, smoking, tobacco and 15 subjects of matching age with no addictive habits was taken as healthy control group. Volunteers suffering from systemic illness, pregnant women were not included in the study.

Saliva Collection Method

The subject was requested to not to drink or eat or chew or perform oral hygiene 15 minutes before and during the study. Subjects were made to sit in upright position and asked to spit on eppendorf container gradually. The subjects were instructed not to swallow during collection of saliva. After collection of saliva, the pH was measured. The salivary pH was measured immediately using pH strips. Manufacturer's instructions were followed while measuring salivary pH. The strip was dipped inside the collected saliva for approximately 10 seconds and color change was noted and compared with the standard chart given. The corresponding values was recorded and taken as the salivary pH.

Statistical Analysis

Data were entered and analyzed. The mean of the values of salivary pH was done with computerized software. The mean values of salivary pH of patients with carcinoma, oral submucous fibrosis, leukoplakia and normal control were compared.

Result

The results were used to determine mean and standard deviation. The data was used to analyze with one way ANOVA, t-test. From the statistical data, it is found, there is significant change in the values of pH in all conditions when compared to control group. Carcinoma group possessed highest pH changes when compared to OSMF and leukoplakia. Though Carcinoma is the condition in which highest pH change was noted there was no significance according to statistics whereas there was significant difference in case of OSMF and leukoplakia condition. On ANOVA one way test pH values of carcinoma, OSMF, leukoplakia conditions were compared and there was no significance

between any conditions. The insignificant differences may be as a result of limited sample size.

Table 1: T- Test

GROUP	*N*	*MEAN (pH)*	*SD (pH)*	*df*	*P-value*
CARCINOMA	5	7	1.41	8	0.7707
OSMF	5	8.2	0.45	8	0.0077
LEUKOPLAKIA	5	8.2	0.45	8	0.0353

Table 2: Anova One Way Test

GROUP	*Count*	*Sum*	*Average*	*Variance*
CARCINOMA	5	35	7	1.98
OSMF	5	41	7.2	0.2
LEUKOPLAKIA	5	41	7.2	0.2

Table 3: Source of Variation within and between groups

Source of Variation	*SS*	*df*	*MS*	*F*	*P-value*
Between Groups	4.8	2	2.4	3.0086	0.0873
Within Groups	9.5724	12	0.8		
Total	14.3724	14			

Discussion

This study was done to analyze the salivary pH and determine its correlation in habit associated malignant and premalignant conditions. The A total of 30 subjects were included in this study of which 5 were oral cancer patients , 5 were oral submucous fibrosis patients , 5 were leukoplakia patients associated with adverse habits such as areca nut chewing, smoking, tobacco and 15 subjects of matching age with no addictive habits was taken as healthy control group. Saliva samples were collected in eppendorf container and pH was checked using pH paper strips. The results were noted and statistically analyzed. The data was used to analyze with one way ANOVA, t-test. From the statistical data, it is found, there is significant change in the values of pH in all conditions when compared to control group. Carcinoma group possessed highest pH changes when compared to OSMF and leukoplakia. Though Carcinoma is the condition in which highest pH change was noted there was no significance according to statistics whereas there was significant difference in case of OSMF and leukoplakia condition. On ANOVA one way test pH values of carcinoma, OSMF, leukoplakia conditions were compared and there was no significance between any conditions. The insignificant differences may be as a result of limited sample size. Saliva is an excellent diagnostic mark for precancerous and cancerous conditions, this has been proved in several studies done. The saliva contains several biomarkers that are used for diagnostic purposes. Similar study done based on pH changes in patients with oral submucous fibrosis showed that there was significant change in pH in patients with oral submucous fibrosis who were associated with arecanut chewing. In that study the salivary pH recovery time was statistically significant with average of 15.37 min longer than that of controls[6]. Various studies has been done for detection of precancerous and cancerous conditions using salivary contents. A study was done to based on salivary sialic acid, total protein and total sugar in oral cancer condition. The study showed that there was significant rise in sialic acid, total protein and total sugar in oral cancer condition particularly in well differentiated squamous cell carcinoma[7]. A study done by shen et al in detection of oral squamous cell carcinoma using salivary proteomics showed that the several salivary proteins were revealed on Subtractive proteomics differential levels between the oral squamous cell carcinoma patients. The combination of these biomarkers yielded an operating characteristic value of 93%, specificity of 83% and sensitivity of 90%

in detecting oral squamous cell carcinoma[8]. The early detection of precancerous lesions or conditions helps in prevention of oral cancer and its associated morbidity and mortality[9]. The gold standard method for early detection of oral precancerous and cancerous condition is biopsy[10]. But biopsy is a invasive procedure thus many research has been made to bring out best non invasive method that provides results that are obtained from biopsy. Such non invasive methods that has been found and yet to be found. Such methods are salivary diagnostics, toluidine blue dye or tolonium chloride, oral CDx brush biopsy kits, and optical imaging systems[11]. In salivary diagnostics, human salivary transcriptome is used in detection of cancerous condition, in which seven RNA complex were elevated in the saliva[12]. In a study, the serum and salivary TSA/TP ratios and α-l-fucosidase activity were measured in oral precancerous condition and oral cancer patients compared to the controls. The levels were significantly higher when compared to controls. The α-l-fucosidase activity and the Salivary TSA/TP ratio were elevated with higher magnitude than the serum levels[13]. In a study, salivary transferrin were used as biomarker for the detection of oral cancer[14]. In another study the presence of salivary interleukin 6, 8 and TNF alpha in oral cancer was studied. The study showed that the interleukin 6 were elevated and was statistically significant[15]. Similar study that was done for detecting salivary TNF alpha, interleukin 1,6,8 in malignant transformation of oral lichen planus, but the study showed there was no significance of the biomarkers[16]. Rhodus et al in their study showed that the TNF-α, IL-1α, IL-6, and IL-8 were found to be elevated in the whole unstimulated saliva of patients with oral squamous cell carcinoma which was compared patients with oral premalignant lesions and controls[17]. In a study, the analysis indicates that the five candidate markers M2BP, MRP14, CD59, catalase, and profilin collectively, provide a sensitivity of 90% and a specificity of 83% for OSCC detection[18]. Profilin 1 may be secreted into tumor microenvironments during the early progressive stage of tumor formation[19]. There are several studies for detection of oral premalignant and malignant conditions but very few are effective[20].

Conclusion

Saliva can be considered as first line diagnostic aid with advanced technology. Salivary pH does not remain constant and it differs for every individual. As per the study there was

significant change in salivary pH changes in all conditions but statistically it is not significant, it may be as a result of limited samples. Further studies on salivary pH changes in premalignant and malignant conditions with more sample size may show significance. Thus salivary pH changes then can be considered as a noninvasive diagnostic method of premalignant and malignant conditions.

Corresponding Author

Antonette Rhea,

Senior Lecturer, Department of Prosthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email Id: arhea.a@gmail.com

Phone Number: +91 91766 45343

Reference

1. Varshitha. A. Prevalence of oral cancer in India. A/J. Pharm. Sci. & Res. 2015;7(10): 845-848.
2. Pindborg JJ, Murti PR, Bhonsle RB, Oral submucous fibrosis as a precancerous conditions. Scand J Dent Res 1984; 92:224-229.
3. Kramer IR, Lucas RB, Pinborg, jj. WHO Collaborating Centre for oral Precancerous Lesions. Definition of leukoplakia and related lesions: An aid to studies on oral precancer. Oral surg Oral Med Oral pathol 1978;46:518-539.
4. Bouquot JE, Whitaker SB. Oral leukoplakia- Rationale for diagnosis and prognosis of its clinical subtypes or “phases”. Quintessence Int 1994;25:133-140.
5. Uwe Wollina, Shyam B Verma, Fareedi Mukram Ali, and Kishor Patil. Oral submucous fibrosis: an update. Clin Cosmet Investig Dermatol. 2015; 8: 193–204.
6. Mandana Donghue, Praveen S Basandi et al, Habit associated salivary pH changes in oral submucous fibrosis- A controlled cross sectional study. J Oral Maxillofac Pathol. 2015 May- Aug;19(2):175-181.
7. Sanjay PR, Kaveri Hallikeri, Shivashankara AR. Evaluation of salivary sialic acid,

total protein, and total sugar in oral cancer: A preliminary report. Indian J Dent Res, 2008; 19(4): 288-291.

8. Shen Hu, Martha Arellano, Pinmanee Boontheung, Jianghua Wang, Hui Zhou, JiangJiang,David Elashoff, RogerWei,Joseph A. Loo and DavidT. Wong. Salivary Proteomics for Oral Cancer Biomarker Discovery. Clin Cancer Res 2008;14(19): 6246 - 6252.
9. Nair D, Pruthy R, Pawar U et al. Oral cancer: premalignant conditions and screening— an update. J Can Res Therapy, 2012; 8(1): s57–s66.
10. Fedele S. Diagnostic aids in the screening of oral cancer. Head Neck Oncol 2009;1(1): 5–10.
11. Epstein JB, Silverman S Jr, Epstein JD et al. Analysis of oral lesion biopsies identified and evaluated by visual examination, chemiluminescence and toluidine blue. Oral Oncol 2008; 44(6): 538–544.
12. Li Y, Zhou X, St John MA, Wong DT. RNA profiling of cell- free saliva using microarray technology. J Dent. Res. 2004; 83(3),199-203.
13. Bhairavi N. Vajaria, Kinjal R. Patel, Rasheedunnisa Begum, Franky D. Shah, Jayendra B. Patel, Shilin N. Shukla, Prabhudas S. Patel. Evaluation of serum and salivary total sialic acid and α-l-fucosidase in patients with oral precancerous conditions and oral cancer. OOOOjournal, 2013; 115(6): 764-771.
14. Yu-JenJou,abf,Chih-HoLaie,Chao-Hsien,ChenaJung-YieKaofShih-YinCheng,Ming-HsuiTsaicdSu-HuaHuangbCheng-WenLina. Proteomic identification of salivary transferrin as a biomarker for early detection of oral cancer. Analytica chimica Acta, 2010; 681(1): 41-48.
15. Mahnaz SahebJamee, Mohammad Eslami, Fazele AtarbashiMoghada, Abdolfattah Sarafnejad. Salivary concentration of TNFα, IL1α, IL6, and IL8 in oral squamous cell carcinoma. Med Oral Patol Oral Cir Bucal. 2008;13(5):292-5.
16. Nelson L. Rhodus Bin Cheng Sandra Myers Lindsay Miller Vu Ho Frank Ondrey The feasibility of monitoring NF- κB associated cytokines: TNF-α, IL-1α, IL-6, and IL-8 in whole saliva for the malignant transformation of oral lichen planus. Molecular carcinogenesis, 2005; 44(2):77-82.
17. Rhodus NL, Ho V, Miller CS, Myers S, Ondrey F. NF-kappab dependent cytokine

levels in saliva of patients with oral preneoplastic lesions and oral squamous cell carcinoma. Cancer Detect Prev. 2005;29(1):42-5.

18. Henson DE, Srivastava S, Kramer BS. Molecular and genetic targets in early detection. Curr Opin Oncol 1999;11:419 -25.
19. Huang CM, Ananthaswamy HN, Barnes S, et al. Mass spectrometric proteomics profiles of in vivo tumor secretomes: capillary ultrafiltration sampling of regressive tumor masses. Proteomics 2006;6:6107 -16.
20. Wong DT. Salivary diagnostics powered by nano- technologies, proteomics and genomics. J Am Dent Assoc 2006;137:313-21.

Hand Washing Techniques among Dental Students- An Educational Intervention

Nazia Zareen.I[1], Brundha M.P[2], L. Leelavathi[3]

Graduate Student[1],Senior Lecturer[2], Department of General Pathology,Senior Lecturer[3],Department of Public Health Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Good hand hygiene is one of the most important actions healthcare workers can take to reduce the spread of infection and protect the lives of their patients.The main medical purpose of washing hands is to cleanse the pathogens and chemicals which can cause personal harm or disease. Hand-washing reduces the number of transient organisms on the skin surface.The purpose of the study is to find the effectiveness of educational intervention about hand washing techniques among dental students.

Materials and Method: This study is anPre-Post teststudy devoted to improve skills about hand hygiene. It was carried out among 50 dental students within a dental college, Chennai. We observed the hand-washing technique done by the student before training and instructed them with a proper hand washing technique and they were observed. Wilcoxon signed test was used to assess the difference in the hand washing steps in pre and post training.

Results: The results of the study showed that only 10% of participants were aware about proper six steps wheareas 90% were not aware about the proper six steps of handwashing technique before training. After giving an hands on training, all were aware about the proper six steps of hand washing technique. Hence, hand washing skill training was effective and this difference was found to be statistically significant.

Conclusion: It was found from the study that there was a significant improvement in hand hygiene techniques after an educational intervention which helps to avoid the spread of nosocomial infection from the dental professionals to patients.

Key Word:*Hand washing ,Nosocomial infection, Attitude,Hygiene,Personal barriers.*

Introduction

Hand washing or hand hygiene is termed as a washing one's hands with or without the use of water or antiseptic liquid, or with the use of soap for the purpose of removing dirt, and/or microorganisms[1].Studies done by IgnazSemmelweis in the 19th century, first recognized the role of the practitioner's hand in the transmission of infections in his article on the etiology and concept of childbed fever which later became awareness on maintaining hand hygiene practices during clinical procedures or attending on every single patient for examination[2]. "My five moments for hand hygiene"wasdevelopedby World Health Organization in 2009. These five protocols that plea for the practice of hand hygiene which are incorporated as anotebefore touching a patient, before performing aseptic and clean procedures, after being at risk of exposure to body fluids, after touching a patient, and after touching patient surroundings. This concept of hand washing has been timely used to enhance better understanding, training, monitoring, and reporting hand hygiene among dental professionals [3].Selfprotectionbarriers have to be followed by every dental Health Care Professional as a step towards cross infection control. These measures include immunization, hand wash and personal barriers[4].Disease transmission between healthcare professionals and patients is generally considered to occur first, through which many exogenously acquired nosocomial infections can spread by inter- and intra-hospital facilities[5]. Dentist particularlyare at an increasingly prone to infections as they provide care working with sharp instruments at very high speeds and limited access in an environment that is bathed in saliva and, in many instances, blood[6]. Hands of dental students get progressively colonized with different microbial flora either commensal or potential pathogens during routine patient care. It has been learnt that bacterial count increases linearly over time[7]. Hand hygiene is an important healthcare controversy globally and is a single most cheap and efficiently measure to cut down the extent of nosocomialinfection and the spread of antimicrobial resistance across all hospitality surroundings right from advanced health care systems to primary healthcare centers[8,9] It is considered to be an simple , cheap and most effective means of controlling cross infections. Hand hygiene methods is designed to save lives and provide a safe treatment atmosphere for all patients and for health care professionals[10]. Hand washingtechnique is denotes in different terms are as hand antisepsis, disinfection, degerming, decontamination or sanitizing.Usage of various antimicrobial chemicals have been

incorporated into hand-washing agents such as chlorhexidine, iodophors, quaternary ammonium compounds, and alcohols[8]. As dental student become more involved in patient contact during their training, they are at risk of exposure to pathogens. It is the responsibility of academic institutions to facilitate appropriate preclinical immunization and provide training in infection control to protect patients and the health and careers of undergraduates, and to lay the foundation for safer work practices in health care[9].In this sense, hand hygiene is important preventive measure, working as the main action to reduce those infections and cross-transmission of said pathogens, especially the multi-resistant ones,including the oral healthcare setting[10].It is also to be a milestone of infectious disease control, and promotion of improved hand hygiene has been recognized as an important public health measure[11].

The reasons for poor hand-hygiene practices include lack of scientific knowledge, unawareness of risks, misconceptions (e.g., glove use obviates the need for hand hygiene), unavailability of hand-hygiene facilities (sinks or alcohol dispensers), lack of role models among colleagues or superiors, understaffing or patient overcrowding, andlack of institutional priority[12]. Similarfindingsreported that the main barriers to regular hand hygiene were lacked of adequate facilities, forgetful- ness and lack of time[13]. Major drawback for inappropriate hand washing among dental students which includes work overload, insufficient sinks, patients general condition, and inaccessibility of hand-washing agents[14]. The purpose of thestudy is to find the effectiveness of educational intervention about hand washing techniques among dental students.

Materials and Methods

A Pre-Post study design was used to find the effectiveness of educational intervention about hand washing techniques among 50 dental students in a dental college, Chennai. Students willing to participate were included in the study and those who did not attended health education intervention were excluded. Ethical clearance was obtained from the ethical committee of the institution. The intervention was done after observing their hand washing technique. Health education was given by demonstration and flip charts. It is included the proper six steps of hand washing and it’s necessity in maintaining personal hygiene and disease prevention. Six steps in hand washing technique are as follows

- Wet hands with water, apply enough soap to cover all hand surfaces. Rub hands palm to palm
- Right palm over left dorsum with interlaced fingers and vice versa.
- Palm to palm with fingers interlaced
- Fingers of one hand over the other with fingers interlocked
- Left thumb clasped over right thumb by rotational motion of rubbing and vice versa.
- Rotational rubbing backwards and forwards with clasped fingers of right hand in left palm and vice versa. Rinse hands with water and dry it.

Data was entered in SPSS software and statistical analysis was performed. The tests applied were : Wilcoxon signed tests and value is less than 0.01 were considered statistically significant.

Result

By observing the before and after hand washing technique, data was recorded and analyzed statistically. Majority of the dental students was not aware about all the steps in hand washing technique. Only 10% of participants were aware of six steps whereas 90% were not aware proper six steps of hand washing. Totally six steps were assessed before and after training of hand wash. It was found that there wasn't any difference before and after instructions in the step 1 hand washing technique among study participants. Before providing hands on training, it was found 88% of study participants were aware and 12% were not aware of step 2 in hand washing techniques. After hands on training, all the participants performed step 2 hand washing procedure correctly. This difference was found to be statistically not significant. In step 3, it was found 80% of study participants were aware whereas 20% were not aware of step 3 technique. After training all the participants performed step 3 procedure correctly, it shows statistically significant. Similarly before hand on training, it was found 58% and 70% of study participants were not aware of step 4 and step 5 respectively. After hand on training, all the participantsperformed step 4 and step 5 hand washing procedure correctly. This difference was found to be statistically significant.

Figure 1: Pre training

Figure 2: Post training

Table 1: Difference between post and pre training

	Step 1.post-step 1. Pre	Step 2. Post- step 2. Pre	Step 3. Post- step 3. Pre	Step 4. Post – Step 4. Pre	Step 5. Post- Step 5. Pre	Step 6. Post- step 6. Pre
Z	0	2	2	3.162	5.385	5.916
p value	1	.014	.046	.002	0	0

Discussion

Hand hygiene prevents cross infection in hospitals, however adherence to guidelines is commonly poor[15]. Proper hand washing and use of barriers such as gloves, gowns, and mask are the main components of standard precautions which can minimize muco-cutaneous exposures[16].While the methods incorporated in hand hygiene are simple, the complex linkage of elements that determine hand hygiene behavior which makes us to study the hand hygiene complex [17]. In this study, students before hands on training had less knowledge about the steps of hand washing techniques and 90% of study participants were not aware about hand hygiene, later they were given training on hand washing technique in which nearly all the participants performed all six steps in hand washing techniques correctly. Study done by Chatterjee et al. shown that moderate knowledge of hand hygiene was reported among 76% of the total study population. Only 8% of participants had good knowledge and 16% showed poor knowledge with respect to hand hygiene practices[18]. In another study by Nair et al,among health care workers of a tertiary health care centre at Raichur, Karnataka, it was reported that the study participants had moderate knowledge on hand hygiene[19].Study done by Van de Mortel et al. [20] have shown that 63% of medical students were aware of the proper indications of hand hygiene while Mann and Wood [21] reported awareness in only 56% of students. In a study done byKadi et.al, it was found that compliance with hand hygiene among medical students was only 17% as compared to the level of awareness (56%) among the same group of students[22]. Factors which are affecting the compliance of hand hygiene are lack of time, forgetfulness, lack of knowledge

of importance of hand hygiene in preventing cross infection, less access to hand washing resource, work overload and cause skin irritation to hand hygiene products. A study done by Abhinav Singh et.al, among dental students of central India, in which students had shown a poor response to knowledge based questions and the authors had stressed on the need for more education programs for infection control as mandatory along with Hepatitis B immunisation measures[23]. In contrast, study done by Glad Mohesh and AbinayaDandapani, the difference between knowledge and attitude had been much lesser compared to the actual practice of hand hygiene[24]. It depicts the real ground level situation where budding dental students have moderate understanding of good hygiene practices. This moderate information may turn out as poor practices during their professional course. The level of knowledge about the hand hygiene compliancewas poor among the students. Attributable reasons could be inadequate training for infection control measures, inadequate supply of personal protective equipment, and carelessness[6]. Ansari et al. had conducted a study to evaluate the effect of hand hygiene practices on patient's health and cost where before and after evaluation of hand hygiene practices among doctors and nurses from a tertiary health centre, was done. It was reported thatdrastic and significant improvement in the right technique and significance of hand hygiene (7.1% to 46.4% among doctors and 2.2% to 45.5% among nurses) knowledge was observed after a health education programme[25].

In the present study dental students were given hands on training to gain knowledge on how to wash handsand there was significant difference in each steps of hand washing procedure in before and after educational training. A study done by Mobashr KA and Ibrahim MA reported that most of the medical students performed hand hygiene and data showed that the overall average of medical student’s knowledge about nosocomial infection was 68.34%[26]. Ayub A et al. showed in their study, only 31.25% medical students always followed hand hygiene procedure and also complete knowledge about hygiene and infection control procedures was present among 77.5% of students[27].Standard techniques of hand washing were always found to be efficient in removing transient contamination on hands and the present study was designed to create an awareness about the knowledge on hand hygiene and its importance. The observed hand washing behaviors and the length of time washing hands relate differently to different factors and so we need to create awareness on handwashing to prevent from infections.

Conclusion

It was found from the study that there was a significant improvement in hand hygiene techniques after an educational intervention. We need to create awareness on hand hygiene and to be practiced in order to reduce nosocomial infections and other microbial infections.Compliance programs on hand washing technique should be developed early in training and more frequently in the graduation and professional courses for dental healthcare personnel. The review of guidelines on hand hygiene, however, should be improved both for practices and standardization to help the projection of successful strategies for the intervention.

Corresponding Author

L. Leelavathi,

Senior Lecturer, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Mobile Number: 9443702882,

Email id: karleela81@gmail.com

Reference

1. "The Hygiene Improvement Project (HIP) - Tippy-Tap: A simple low-cost technology for handwashing when water is scarce". USAID. Archived from the original on 25 October 2014. Retrieved 30 September 2015.
2. Semmelweis I. 1983. Etiology, Concept, andprophylaxis of childbed fever. Carter KC, ed. 1st Ed. Madison, WI: The University of Wisconsin Press.
3. H. Sax, B. Allegranzi, I. Uçkay, E. Larson, J. Boyce, and D. Pittet, "'My five moments for hand hygiene': a user-centred design approach to understand, train, monitor and report hand hygiene," Journal of Hospital Infection, vol. 67, no. 1, pp. 9–21, 2007.
4. VelmuruganRajan, and Mohammad Nabeel Nazar, " Cross Infection Control in Dentistry" RJPBCS , March - April 2014, 5(2) ;650-657.

5. Marcília Batista de Amorim-Finzi, Mauro Vieira Cezar Cury, Claudio Rodrigues R. Costa, Angelis Costa dos santos, Geraldo Batista de Melo, “Rate of Compliance with Hand Hygiene by Dental Healthcare Personnel (DHCP) within a Dentistry Healthcare First Aid Facility”Eur J Dent. 2010 Jul; 4(3): 233–237.
6. Preetha P. Kanjirath, Amy E. Coplen, Jody C. Chapman, Mathilde C. Peters and Marita Rohr Inglehart, “Effectiveness of Gloves and Infection Control in Dentistry: Student and Provider Perspectives” Journal of Dental Education May 2009, 73 (5) 571-580;
7. LavanyaJagdish, Trupti B. Naik, Ravi Kumar Gupta, AmruthKishanUpadhya, “ Prevalence of hand hygiene awareness among medical and dental students in a teaching hospital” Indian J Microbiol Res 2016;3(3):262-265.
8. Mani, A., Shubangi, A.M. & Saini, R. 2010. ‘Hand hygiene among healthcare workers”, Indian Jounal of Dental Research 21:1, 115-118.
9. Gillian M. McCarthy, Jonathan E. Britton,“A Survey of Final-Year Dental, Medical and Nursing Students: Occupational Injuries and Infection Control”J Can Dent Assoc 2000; 66:561
10. P. Mathur, “Hand hygiene: back to the basics of infection control,” Indian Journal of Medical Research, vol. 134, no. 11, pp. 611–620, 2011.
11. S. Kelc´ıkova, Z. Skodova, and S. Straka, “Effectiveness of hand hygiene education in a basic nursing school curricula,” Public Health Nursing, vol. 29, no. 2, pp. 152–159, 2012.
12. Marufa Sultana, RashidulAlamMahumud, [...], and Sarder Mahmud Hossain" Hand hygiene knowledge and practice among university students: evidence from Private Universities of Bangladesh"RiskManagHealthc Policy. 2016; 9: 13–20.
13. Omogbai JJ, Azodo CC, Ehizele AO, Umoh A. Hand hygiene amongst dental professionals in a tertiary dental clinic. African Journal of Clinical and Experimental Microbiology 2011; 12:9-14.
14. Barrett R, Randle J. Hand hygiene practices: nursing students' perceptions. J Clin Nurs 2008;17:1851-7.
15. Trampuz A1, Widmer AF. “Hand hygiene: a frequently missed lifesaving opportunity during patient care”. Mayo Clin Proc. 2004 Jan;79(1):109-16.

16. Chistiaens G, Barbier C, Mutsers J, Warnotte J, De Mol P, Bouffioux C. Hand hygiene: first measure to control nosoco- mial infection. Rev Med Liege 2006;61:31-6.
17. M.Monika Gayathri, Dr.M.P.Santhosh Kumar "Knowledge, Awareness and Attitude among Dental Students about Hepatitis B Infection"J. Pharm. Sci. & Res. Vol. 8(3), 2016, 168-170.
18. Chatterjee S, Mondal TK, Sarkar K, et al. Assessment of hand hygiene knowledge among medical students at a teaching hospital of India. Sch. J. Appl. Med. Sci. 2015;3(6C):2334-2339.
19. Nair SS, Hanumantappa R, Hiremath GS, Siraj MA, Raghunath P. Knowledge, attitude, and practice of hand hygiene among medical and nursing students at a tertiary health care centre in Raichur, India. ISRN Prev. Med. 2014:608927.
20. T. van de Mortel, “Development of a questionnaire to assess health care students’ hand hygiene knowledge, beliefs and practices,” Australian Journal of Advanced Nursing, vol. 26, no. 3, pp. 9–16, 2009.
21. C. M. Mann and A. Wood, “How much do medical students know about infection control?” Journal of Hospital Infection, vol. 64, no. 4, pp. 366–370, 2006.
22. Azzam al Kadi and Sajad Ahmad Salati"Hand Hygiene Practices among Medical Students"Interdisciplinary Perspectives on Infectious Diseases.Volume 2012 (2012).
23. Abhinav Singh, Bharathi M. Purohit, Ajay Bhamba et al.," Knowledge, Attitudes, and Practice Regarding Infection Control Measures Among Dental Students in Central India"Journal of Dental Education,Volume 75, Number 3 March 2011
24. Glad Mohesh and AbinayaDandapani. UJMDS 2014, 02 (03): Page 127-131.
25. Ansari SK, Gupta P, Jais M, Nangia S, Gogoi S, Satia S, et al. Assessment of the knowledge, attitude and practices regarding hand hygiene amongst the healthcare workers in a tertiary health care centre. Int. J. Pharma Res. Health Sci. 2015;3(3):720-726.
26. A.Mobashr, M. A. Ibrahim, W. A. Hussein, Perceptions of medical students towards nosocomial infections at college of
medicine- Babylon, Journal of Education and Practice,5(29), 2014, 73-84.

27. A.Ayub, A.Goyal, A.Kotwal, A. Kulkarni, A.Kotwal, A.Mahen, Infection control practices in health care : Teaching and learning
requirements of medical undergraduates, Medical Journal Armed Forces India, 69, 2013, 107-112.
28. Tvedt C, Bukholm G. Alcohol-based hand disin- fection: a more robust hand-hygiene method in an intensive care unit. J Hosp Infect 2005; 59: 229-34.

In Vitro Evaluation of Antibacterial Activity of Three Essential Oil against Streptoccocus Mutans

IndhulekhaVimalakshan[1], DeepaGurunathan[2], Geetha .R.V[3]
Graduate student[1], Professor[2], Department of Pedodontics,
Professor[3], Department of Microbiology
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: Dental caries is a major health problem. Many bacteria are involved in the process of developing dental caries whereby *Streptococcus Mutans are*one of the predominant bacteria that initiate the formation of dental caries. Indian medicinal plants are known to have many important secondary metabolites such as alkaloids, flavonoids, tannins, phenolic compounds which impart the antimicrobial properties. This study is aimed to determine the antimicrobial activity of Cinnamon oil, Rose Mary oil and Tea Tree oil against *Streptococcus Mutans.* To evaluate the antibacterial activity of the three essential oils against the selected bacterial pathogen

Material and Method: The antibacterial activity is carried out by *agar well* diffusion technique against the bacterial pathogen and the zone of inhibition is measured in mm diameter.

Result: In the present study, usage of these three oils was found to be equally effective against the microbe tested compared with the control.The maximum zone of inhibition was seen with Cinnamon oil measuring 26mm diameter followed by Tea tree oil and Rosemary oil

Conclusion: The result of this study reveals that essential oils exhibit antimicrobial propertyagainst *Streptococcus*Mutans. Hence it can be concluded that essential oils like cinnamon oil, tea tree oil and rosemary oil have effective antimicrobial property and can be utilized against various infections caused by oral microbes.

Key Words: *Cinnamon oil, Rosemary oil, Tea tree oil, Agar well diffusion technique, zone of inhibition*

Introduction

Dental caries is caused by the metabolic results of specific microorganisms which are the outcome that leads to disintegration of inorganic parts of enamel, dentin, cementum and degradation of their natural structure. Numerous microorganisms are associated with the process of developing dental caries in which Streptococcus Mutans are one of the prevalent microscopic organisms that starts the development of dental caries. Streptococcus Mutans are viewed as the most cariogenic in every single oral streptococcus[1]. Streptococcus Mutans can colonize the surface of the tooth and create a lot of extra-cellular and intra-cellular polysaccharides.[2]This microorganism in addition is very acidogenic and aciduric, and it uses a few salivary glycoproteins, subsequently overseeing the underlying phase of oral biofilm development and caries. Indian medicinal plants have been known to have numerous vital optional metabolites such as for an example, alkaloids, flavonoids, tannins, phenolic and antimicrobial properties. In the past, there has been an expanded enthusiasm for the helpful properties of some therapeutic plants and essential oils. [3]

Essential oils (EOs) are recognised for their identified antimicrobial action that goes against Streptococcus mutants. Their intricate unpredictable characteristic and mixed shape by fragrant plants are optional metabolites. They are known for their bactericidal, virucidal, fungicidal, calming, mitigating, pain relieving, spasmolytic, and local analgesic properties. The presence of complex chemical structures constitutes several groups such as terpenes and terpenoids, aromatic and aliphatic constituents, all characterized by low molecular weight, whereby explains their successful bacteriostatic and bactericidal action [4].

The aim of this studyis to assess the antibacterial activity of the three essential oils which is cinnamon oil, rosemary oil and tea tree oil against chosen bacterial pathogen

Cinnamon Oil

Cinnamon oil is the most volatile oil. Cinnamon is sweet zest savored over the world. It is additionally utilized as a home-grown tea. Cinnamon is also used to extricate an essential oil which has its correct sweet, pervading smell which is extremely mitigating. Cinnamon oil has numerous medical advantages [5].

The source of cinnamon oil is the sweet flavor from Cinnamomum verum. This is known as the true cinnamon. There is a less expensive assortment of cinnamon powder that we get from trees in the Cassia family. This modest cinnamon originates from China, while true cinnamon develops in numerous parts of the tropical world, notably in Sri Lanka and India. Although Cassia cinnamon is additionally used to extract the basic oil, the ones from the true cinnamon is the one which has great medical advantages. There are two sorts of essential oil obtain from this tree. First, is the Cinnamon leaf essential oil-the extract is steam refined from the leaves of cinnamon and it is yellowish in shade. Next is the, Cinnamon bark essential oil – the extract is steam refined from the bark of cinnamon and It is marginally rosy in shade with a trace of brown. [6].

Customary Uses of Cinnamon Oil

Cinnamon has been known as a standout among the most well-known flavors and seasoning. For an example, it has been utilized as a flavor in desserts and chewing gum due to the charming and invigorating impacts that it creates in the oral cavity. Therefore, it indicates useful impacts on oral wellbeing and is utilized for toothaches, oral diseases, and halitosis. [7] Cinnamon has additionally been utilized to treat skin inflammation and melisma. [8] In addition, cinnamon has also been utilized for the treatment of gastrointestinal and colonic diseases. [9] Cinnamon has coagulant impact and along these lines it can be utilized against discharging. [10] Cinnamon expands the blood stream in the uterus and enhances tissue recovery. [11] In addition, it has powerful antibacterial, antifungal, antiemetic, larvicidal, nematocidal and insecticidal properties. [12]Moreover , biological research has demonstrated that cinnamon has intense neuroprotective, hepatoprotective, cardioprotective and gastroprotective impacts due its powerful hostile to oxidant and calming properties. [13] Cinnamon oil can likewise be utilized as a part of fragrance-based treatment, which is helpful in utilization of plant base oil that can be consumed into the body by means of the skin or olfactory system.

Antibacterial Effects of Cinnamon Essential Oil and Cinnamon Extracts

One of the most well-established properties of cinnamon extricates, essential oil and their parts is the antibacterial movement against Gram Positive and Gram Negative microscopic organisms responsible for human irresistible maladies and debasement of sustenance or beauty care products. In the literature, there are number of studies demonstrating the

antibacterial action of cinnamon oil acquired from various herbal parts using extraction techniques.

In addition, cinnamon could be utilized to treat appealing malady. Nonetheless, there is an absence of clinical trials on the antibacterial impacts of cinnamon and its synthetic constituents, and in this manner its clinical adequacy isn't clear. Adding on, it is outstanding that cinnamon oil's poisonous quality is a standout among the vital issues, as some basic oils demonstrate the detailed negative impacts on human cells, for example, cytotoxicity and cell demise. Consequently, the use of common items in the treatment of appealing illnesses are possibly thought about in fascination rather than other options to engineer anti-toxins, particularly for the treatment of anti-infection safe bacterial diseases. [14]

Cinnamon oil has numerous restorative properties like Antibacterial whereby cinnamon oil is used in aromatherapy blends to get rid of airborne bacteria. It consequently avoids numerous respiratory tract contaminations. It has Antioxidant, Anthelminthic, Antiseptic, Immuno stimulant, Anti-incendiary, Analgesic, Expectorant, Antifungal, and Antidepressant and Hypoglycemic exercises. [15]

Rosemary Oil

Rosemary oil is derived from Rosmarinus officinalis (otherwise called Rosmarinus coronarium) hailing from the Labiatae family and is otherwise called incense. Rosemary oil has a unique, intense invigorating home grown smell; it is clear in shielding and watery in viscosity. The name was taken from the Latin word 'Rosmarinus' or 'ocean dew', as it is somehow attached to water. The Egyptians, Hebrews, Greeks and Romans thought about the herb as holy and even in the Middle Ages it was utilized to avoid indecencies spirits and also utilized as an assurance against torment. It was singed in French doctor's facilities amidst pestilences.

The principle compound segments of rosemary oil are a-pinene, borneol, b-pinene, camphor, boranyl acetic acid derivation, camphene, 1,8-cineole and limonene. [16]

Rosemary oil has been basically known for its relieving and calming impacts. While these have just been demonstrated as of late in a few studies, the true component by which rosemary applies this specific influence remains a secret. [17] Regardless, rosemary essential oil is known to have the additional wellbeing properties.

• Anti-provocative – decreases the diverse physiologic indications of incendiary procedures including expanded emission, swelling, agony, and delicacy

• Antimicrobial – can help execute certain microorganisms including gram negative and gram-positive microbes, oxygen consuming and anaerobic microscopic organisms, and certain types of growth

• Antioxidant – mitigates the impacts of oxidative weight on the body

• Antispasmodic – eases fitful conditions, for example, renal colic and dysmenorrhea

• Antitumorigenic – keeps the procedure and improvement of tumor cells and other neoplastic development

• Choleretic – invigorates the liver to deliver more bile in order to emulsify fat in the stomach related tract and enable it to be consumed and used appropriately

• CNS stimulant – fortifies the focal sensory system including incitement of the autonomic sensory system to make the individual feel more vivacious, have incredible state of mind, and better memory and other psychological procedures

• Hair producer – encourages the development of hair

• Hepato protective – secures the general propriety and physiologic work of the liver

• Mood enhancer – gives a superior feeling of prosperity

• Smooth muscle relaxant – enhances the general tonicity of the smooth muscles, especially those of the gastrointestinal tract and the respiratory tract. [18]

Uses

The principal use of rosemary today is in aromatherapy where it is often regarded for its ability to calm the senses while inducing an overall state of wellbeing. The following are the most common uses of rosemary oil.

• **Aromatherapy** – Either as a pure essential oil or as an addition to other essential oils, rosemary is extensively used in spas and health and fitness centers. It can induce a more

relaxed state, improve mood, and induce a state of wellness that can be an excellent way to manage one's stress.

• **Massage** – When used as an essential oil for massage therapy, rosemary can help relieve sore muscles while at the same time help facilitate the enhancement of blood flow. Consequently, this facilitates the more efficient elimination of toxic waste.

• **Management of Certain Infections** – Certain respiratory and digestive tract infections has been shown to be managed by the introduction of rosemary essential oil in the therapy. This is often used in combination with pragmatic antimicrobial therapies.

• **Alopecia** – There are plenty of rosemary products that are combined with other essential oils designed to help follicle-challenged individuals hair growth. When used in combination with aloe Vera extracts, rosemary oils can provide the benefit of an excellent hair cultivator and tonic.

• **Hepatotoxic Disorders** – Individuals who are addicted to drugs or have alcoholic problems or are taking hepato toxic medications, the consumption of rosemary oil supplements can help protect the liver by aiding in the different hepato metabolic processes.

• **Bronchial Asthma** – The anti-inflammatory property of rosemary oil can reduce the inflammation and the abnormal copious secretions in respiratory disorders. Rosemary is also known to reduce the spasms of the bronchial airways associated with asthma. This helps the individual to breathe a lot better.

• **Spasmogenic disorders** – Dysmenorrhea and renal colic are just two of the most common spasmogenic disorders. Rosemary can help reduce the spasms in these conditions to provide pain relief.

Other potential uses of rosemary oil include indigestion, gout, eczema, headache, and menstrual irregularities. [19]

Tea Tree Oil

Tea-tree oil, also known as melaleuca oil, is yellowish hued basic oil that is produced using the leaves of the plant Melaleuca alternifolia (local to Australia). Tea-tree oil and tea oil are totally unique items. Tea oil is the sweet flavorful cooking oil squeezed from Camellia sinensis (refreshment tea plant), or the tea oil plant Camellia oleifera. The topical use of tea-tree oil is known to have useful curative [20].

Tea Tree Oil Uses

Tea tree leaves were initially utilized for recuperating skin infirmities. Creepy crawly nibbles, skin spots, and cuts are treated by pounding the leaves and applying them to the territory required. Tea tree oil has likewise been utilized to regard conditions, such as, skin inflammation on athlete's foot, Dandruff, vaginitis, thrush, periodontal diseases, lice, dermatitis, Psoriasis, yeast contaminations, and as a general disinfectant. [21] It is frequently utilized as a part of creams, balms/cleansers, moisturizers, and shampoos. Not just Tea tree oil help with bad breath (halitosis), but since it has anti-microbial characteristics; it can likewise help mend gum diseases. It can treat serious gingivitis (gum malady/periodontal infection) and difficult gums. This is one of the fundamental reasons why a dental specialist would advise a patient to utilize toothpaste that contains Tea tree oil for treating awful breath. [22]

Material and Methods

Essential oils used are cinnamon oil, Rose Mary oil and tea tree oil.

Test organisms: The organism *Streptococcus Mutans* was isolated from saliva sample using special media [Mutants – sanguis agar] and maintained in Tryptose soya agar at 4-degree C in department of Microbiology, Saveetha dental college and hospitals.

Method

The Antimicrobial activity of the three essential oils was evaluated against *Streptococcus mutants* by agar well diffusion technique. [23] Both culture of the test organism was compared to Mac Farland's standard 0.5[24] which were set. Lawn culture of the test organism was made on the Muller Hinton agar [MHA-Hi media M-1084] plates using sterile cotton swab and the

plates were dried for 15 minutes. Wells measuring 4mm in depth was made on the agar using sterile cork borer. 100microlitres of the essential oils were added to the wells. 0.2% chlorhexidine was used as positive control. The plates were incubated at 37-degree Celsius overnight and the zone of inhibition of growth was measured in mm diameter. The entire test was done in triplicate to minimize the test error.

Results

In the experiment carried out, the antibacterial activity of Cinnamon oil, Rose Mary oil and tea tree oil were screened against Streptococcus Mutans using agar well diffusion test and the zone of inhibition was measured in mm diameter. The results are given in table 1. The area of inhibition where the growth of microorganisms was inhibited by the oils was observed to be significant against the pathogen used when compared with the control. The maximum zone of inhibition was seen with Cinnamon oil measuring 26mm diameter followed by Tree tea oil and Rosemary oil

Table 1: Antibacterial activity of three essential oils against *Streptococcus Mutans*

Essential oils	**Zone of inhibition (in mm diameter)**
Cinnamon oil	26
Rosemary oil	22
Tree tea oil	24
0.2% Chlorhexidine (control)	35

Discussion

Essential oils [EO] and different concentrates of plants have advance enthusiasm as sources of normal items with promising antimicrobial action. Essential oils are extracted from plant materials, for an example, bloomed buds, seeds, leaves, twig and so forth. EOs is a blend of

uneven constituents delivered by fragrant plants as auxiliary metabolites, as a defensive system against predators, microorganisms or climate misfortunes. The antimicrobial movement of essential oils is because of little Terpenoids and phenol compound present in it. Essential oils have been tried in vivo and in vitro antimicrobial action and a few has shown to be to having antimicrobial potential. Their system of activity gives an overwhelming impression on the cell layer by disturbing its structure in this manner causing cell spillage and cell passing, and optional activities perhaps hindering the film amalgamation and hindrance of cell breath. [23] They promptly enter the cell layer and apply their organic impact due to high instability and lipophilicity of essential oils. [25]

Usually ethanol is added to the essential oils as a solvent to enhance the volatility and aromatic properties. [26] To avoid the possible effect of solvent on the antimicrobial property, commercially available essential oils that were non-dilated and chemically not altered by any solvent or processing were used in this study. The Essential oil that was used in this study was the one commercially available in the local market. The three essential oils demonstrated in this study showed effective antibacterial activity against S.mutans, of which cinnamon oil was found to be the most effective. The antimicrobial activity of cinnamon oil is explained by its active substances which is cinnamon aldehyde and eugenol [27]. among the major component cinnamaldehyde exhibited the greater antibacterial activity against S.mutans. Therefore the essential oil of C.verum containing cinnamaldehyde showed the uppermost activity. Essential oils containing mainly aromatic phenols or aldehydes have been reported to exhibit considerable antimicrobial activity, whereas essential oil containing terpene ether, ketone or oxide has weaker activity. [28]

Antibacterial activity of cinnamon oil has been stated in studies conducted by Prabuseenivasan et al in Chennai in 2006,[8]Kalemba et al in 2006 in Poland[29] and Kamal RaiAneja et al in Haryana, India in 2009[30]. The essential oil of C.verum was not harmful when consumed in food additives and maybe used as an antibacterial agent. However, there are Occasional case reports of allergic contact dermatitis and stomatitis among consumers.

In the agar well diffusion examine, the size of effective inhibitory zone depends on the solubility and diffusion characteristics of the substance being tested. This makes the comparison of the different oils difficult. Therefor the results of this study may not directly reflect the extent of the antibacterial potential of these oils. However, as these effective zones were clearly visible, this is the proof of their antibacterial efficacy. Thus, as some these

essential oils have antimicrobial efficacy in contrast. In vitro, in vivo study containing this essential oil is recommended to allow these essential oils to be incorporated within formulation and marketed against oral infections.

Conclusion

Cinnamon oil, Rosemary oil and Tea tree oil exhibit antimicrobial property against S.mutans. Cinnamon oil showed highest activity against streptococcus mutants followed by Tea tree oil and Rosemary oil. Hence, this study concludes that the three essential oils has effective antimicrobial property and can be used to fight against various diseases, infection and other problems which are being caused by oral microbes.

Corresponding Author

Deepa Gurunathan

Professor, Department of Pedodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id: drgdeepa@yahoo.co.in

Telephone Number: +919994619386

References

1. Kleinberg I. A mixed-bacteria ecological approach to understanding the role of the oral bacteria in dental caries causation: an alternative to Streptococcus mutans and the specific-plaque hypothesis. Critical Reviews in Oral Biology & Medicine. 2002 Mar; 13(2):108-25.
2. Tayal DP. Photodynamic therapy as alternative treatment for disinfection of bacteria in oral biofilms. State University of New York at Buffalo; 2011.
3. Mukesh DS. Formulation and Evaluation of Polyherbal Dosage form exhibiting Anti acne activity.

4. Prabuseenivasn SJ. M and Ignacimuthu, S. BMC Complementary and Alternative Medicine. 2006; 30:6-39.
5. Chandrasekaran S, Geetha RV. Antibacterial Activity of the Three Essential Oils on Oral Pathogens-An In-vitro Study. Research Journal of Pharmacy and Technology. 2014 Oct 1; 7(10):1128.
6. Babu PS, Prabuseenivasan S, Ignacimuthu S. Cinnamaldehyde—a potential antidiabetic agent. Phytomedicine. 2007 Jan 10; 14(1):15-22.
7. Jakhetia V, Patel R, Khatri P, Pahuja N, Garg S, Pandey A, Sharma S. Cinnamon: a pharmacological review. Journal of advanced scientific research. 2010;1(2):19-23.
8. Nabavi SF, Di Lorenzo A, Izadi M, Sobarzo-Sánchez E, Daglia M, Nabavi SM. Antibacterial effects of cinnamon: From farm to food, cosmetic and pharmaceutical industries. Nutrients. 2015 Sep 11; 7(9):7729-48.
9. Anand P, Kunnumakara AB, Sundaram C, Harikumar KB, Tharakan ST, Lai OS, Sung B, Aggarwal BB. Cancer is a preventable disease that requires major lifestyle changes. Pharmaceutical research. 2008 Sep 1; 25(9):2097-116.
10. Hossein N, Abolfazl M, Mahdi S, Ali K. Effect of Cinnamon zeylanicum essence and distillate on the clotting time. Journal of Medicinal Plants Research. 2013 May 17; 7(19):1339-43.
11. Minich S, Msom L. Chinese Herbal Medicine in Women's Health. Women's Health. 2008 Sep 11.
12. Tung YT, Yen PL, Lin CY, Chang ST. Anti-inflammatory activities of essential oils and their constituents from different provenances of indigenous cinnamon (Cinnamomum osmophloeum) leaves. Pharmaceutical biology. 2010 Oct 1; 48(10):1130-6.
13. Verotta L, Macchi MP. 10 Healing Properties of Food. Connecting Indian Wisdom and Western Science: Plant Usage for Nutrition and Health. 2015 Apr 24:245.
14. Lair C. Feeding the Whole Family: Recipes for Babies, Young Children, and Their Parents. Sasquatch Books; 2009 Jun 2.
15. Altunkaya A, Yıldırım B, Ekici K, Terzioğlu Ö. Determining essential oil composition, antibacterial and antioxidant activity of water wormwood extracts. Gida. 2014 Feb 1; 39(1):17-24.
16. Tawfik AA, Read PE, Cuppett SL. Rosmarinus officinalis L.(Rosemary): In Vitro Culture, Regeneration of Plants, and the Level of Essential Oil and Monoterpenoid

Constituents. InMedicinal and Aromatic Plants X 1998 (pp. 349-365). Springer Berlin Heidelberg.

17. Balz R. The Healing Power of Essential Oils: Fragrance Secrets for Everyday Use. Lotus Press; 1996.
18. Hoffmann D. Healthy Digestion: A Natural Approach to Relieving Indigestion, Gas, Heartburn, Constipation, Colitis, and More. Storey Publishing; 2017 May 1.
19. Price S. Aromatherapy for Common Ailments: How to Use Essential Oils--Such as Rosemary, Chamomile, and Lavender--To Prevent and Treat More Than 40 Common Ailments. Simon and Schuster; 2003 Dec 23.
20. Olsen CB. Australian Tea Tree Oil Guide: First Aid Kit in a Bottle. Lotus Press; 1997.
21. Duke JA. Medicinal plants of the Bible. London: Trado-medic books; 1983.
22. Battaglia A, inventor; Jaleva Pharmaceuticals LLC, assignee. Film-forming resins as a carrier for topical application of pharmacologically active agents. United States patent US 8,709,439. 2014 Apr 29.
23. Kalemba DA, Kunicka A. Antibacterial and antifungal properties of essential oils. Current medicinal chemistry. 2003 May 1; 10(10):813-29.
24. Abubakar EM. Efficacy of crude extracts of garlic (Allium sativum Linn.) against nosocomial Escherichia coli, Staphylococcus aureus, Streptococcus pneumoniea and Pseudomonas aeruginosa. Journal of Medicinal Plants Research. 2009 Apr 30; 3(4):179-85.
25. Silva CD, Guterres SS, Weisheimer V, Schapoval EE. Antifungal activity of the lemongrass oil and citral against Candida spp. Brazilian Journal of Infectious Diseases. 2008 Feb; 12(1):63-6.
26. Inouye S. Laboratory evaluation of gaseous essential oils (Part 1). International Journal of Aromatherapy. 2003 Jan 1; 13(2-3):95-107.
27. Warnke, P.H., Lott, A.J., Sherry, E., Wiltfang, J. and Podschun, R., 2013. The ongoing battle against multi-resistant strains: in-vitro inhibition of hospital-acquired MRSA, VRE, Pseudomonas, ESBL E. coli and Klebsiella species in the presence of plant-derived antiseptic oils. *Journal of Cranio-Maxillo-Facial Surgery*,2013 Jun;41(4):321-6
28. Ağaoğlu SE, Dostbil N, Alemdar S. Antimicrobial activity of some spices used in the meat industry. Bull Vet InstPulawy. 2007; 51:53-7.

29. Vardar-Ünlü G, Candan F, Sökmen A, Daferera D, Polissiou M, Sökmen M, Dönmez E, Tepe B. Antimicrobial and antioxidant activity of the essential oil and methanol extracts of Thymus pectinatusFisch. etMey. Var. pectinatus (Lamiaceae). Journal of Agricultural and food chemistry. 2003 Jan 1; 51(1):63-7.
30. Aneja KR, Joshi R, Sharma C. Antimicrobial activity of Dalchini (Cinnamomum zeylanicum bark) extracts on some dental caries pathogens. J Pharm Res. 2009 Sep; 2(9):1387-90.

Influence of Mental Foramen In Age and Gender Determination Using Orthopantomograph (OPG)

Hansini B. Ajantha[1], Laliytha K.B [2], Pavithra Priyadarshoni S [3].

Graduate Student[1], Senior Lecturer [2], Department of Oral Medicine and Radiology,

Tutor [3],Department of Public Health Dentistry

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: The mental foramen is located in the apical region of the mandibular premolars but it has some variations regarding its distance to both premolars and the base border of mandible that may make a problem especially for the oral surgeon in his work. The aim of this study is to evaluate the variation of mental foramen in mandible according to sex and age using orthopantomogram (OPG).

Materials and Methods: The samples for this study are collected from 150 patients. The patients selected in this study ranges from 21 to 50 years old, divided into three groups, namely, 21 to 30, 31 to 40 and 41 to 50 and also divided according to gender. All radiographs are examined and then the position of the mental foramen for each patient is recorded.

Results: The results revealed that the distance between the mental foramen and the base border of the mandible changes with age and does not aid in determine the gender.

Conclusion: The position of mental foramen does not play a role determining the gender and has significance in determining the age.

Key Words: *Mental foramen, Inferior border of mandible, OPG, Age, Gender*

Introduction

Mental foramen is an important anatomical landmark to facilitate surgical, local anesthesia and other invasive procedures for dental surgeons performing peri apical surgery in the mental region of mandible. Its location and the possibility that an anterior loop of the mental nerve may be present mesial to the mental foramen, needs to be considered before any surgery in the foramina area in order to avoid any nerve damage. (1) The mental foramen is situated bilaterally on the antero lateral aspect of the mandible, down to the alveolar margin. The mental nerve and vessels emerges through the mental foramen and supply sensory innervation and blood supply to the soft tissues of the chin, lower lip and gingiva. (2) But the position of mental foramen vary among various age groups and gender. Mental foramen holds strategic importance in clinical dentistry and oral surgery. (3),(4) Its accurate identification determines the effectiveness of nerve block and prevention of post operative neurovascular complications in the mental region like neurosensory disturbances, paralysis, hemorrhage, altered sensation, orofacial pain, a typical neuralgia etc. Thus, the mental foramen is an important landmark in dentistry. The present radiographic study is aimed at evaluating how location of mental foramen varies according to age and gender in 150 patients.

Materials and Methods

Fifty patients with the age ranged 21 to 50 were selected for this study. The samples were divided into 3 equal groups regarding age as the following. First group: 21 to 30 years (45 patients), second group: 31 to 40 years (60 patients) and third group: 41 to 50 years (45 patients). Also the samples were divided into male and females following the age group. Fifty digital views were taken for the patients, using computerized digital panaromic X-ray machine. The orthopantomograms were examined for estimation of mental foramen for each patient. The distance between the mental foramen and inferior border of the base of mandible were measured. These measurements were done by using a digital ruler on the computer monitor. Readings were then taken and recorded and a conclusion was made.

Results

The results in table 1 show that the distance between the mental foramen and the inferior border of the mandible, for the first age group ranged between 2.87 and 0.27, with the mean value of 1.57. For the second age group ranged between 2.40 and 0.20, with the mean value of 1.3. Lastly, for the third age group ranged between 1.12 and 0.08, with the mean value of 0.6. The results in table 2 show that the distance between the mental foramen and the inferior border of mandible, for the male ranges from 1.67 to 1.82, and for the female it ranges from 1.65 to 1.79.

Table 1: Distance between the position of mental foramen and inferior border of base of mandible in OPG (in cm)

AGE GROUP	MAXIMUM	MINIMUM	MEAN VALUE
21-30	2.87	0.27	1.57
31-40	2.40	0.20	1.3
41-50	1.12	0.08	0.6

Table 2: Distance between the position of mental foramen and inferior border of base of mandible in OPG

GENDER	21 - 30		31 - 40		41 - 50	
MALE	MIN	1.48	MIN	1.47	MIN	1.45
	MAX	2.23	MAX	2.07	MAX	1.89
	AVERAGE	1.82	AVERAGE	1.79	AVERAGE	1.67
FEMALE	MIN	1.40	MIN	1.45	MIN	1.43
	MAX	2.18	MAX	2.09	MAX	1.87
	AVERAGE	1.79	AVERAGE	1.77	AVERAGE	1.65

Discussion

Age Changes in Mandible

After birth, the shape of the mandible is constantly modified. In neonates, the corpus of the mandible bilaterally holds the alveoli for deciduous incisors, the canine tooth and two molars. In this period the mandibular canal is relatively wide, because it holds the yet divided inferior alveolar nerve and vessels. It is placed near the lower edge of the mandibular corpus. This form of separate innervation of the canine tooth, premolar and molar areas exists only in the neonatal period. In the persistent form, this canal exists posteriorly to the mandibular foramen, as the canal of Serres, which holds the Serres vein. The mental foramen is located low and relatively far towards the posterior, below the first molar bud. The angle between the corpus and the ramus of the mandible is obtuse (150º–160º), and the condyloid process is small. However, the coronoid process in the mandible of a neonate is large and is located above the condyloid process. After birth, both halves of the jaw fuse. This process takes place in the mandibular symphysis, and moves from the bottom to the top. Because of the developing buds of permanent teeth, the corpus elongates posteriorly from the mental foramen. Together with the development of the alveolar part, the mandibular corpus height increases to make space for the elongating teeth roots. When the permanent teeth start to erupt, the mandibular canal is located just above the mylohyoid sulcus, and the mental foramen moves anteriorly, reaching its final place on the level of the second premolar tooth. The mandibular angle is less obtuse, and at the age of 4 has about 140º . When considering adults, the mandibular proportions are different, and the alveolar part of the mandible, as well as the base of the mandible are of similar height. The mandibular foramen moves cranially and positions itself in the centre of the corpus. The mandibular canal runs almost parallel to the mylohyoid line. The angle between the corpus and the ramus becomes more perpendicular, between 120º and 130º. In senile age, due to the loss of teeth, the size of the mandible decreases. The alveolar part undergoes atrophy. Due to those changes, the main part of the corpus is localised below the oblique line, and the mandibular canal together with the mental foramen relatively move upwards, running near the dental arch. The mandibular ramus leans posteriorly, thus the mandibular angle increases, and once again is about 140º. The neck of the condyloid process leans posteriorly (5),(6).

Mandible Innervation

Developmentally, both mandibular condyles are innervated by the third ramus of the trigeminal nerve , the mandibular nerve (7). This nerve is the one of the trigeminal branches and has a

mixed character. Its lower or posterior branch holds the sensory function and the upper or anterior one the motor function. The mandibular nerve innervates the mandible, the tempo-mandibular joint, the muscles, skin, mucosa, the periosteum, and all of the mandibular teeth. It exits the cranium through the oval foramen and goes posteriorly behind the lateral pterygoid muscle to the subtemporal fossa (8). The posterior group usually has three sensory branches namely, the lingual nerve, the inferior alveolar nerve and the auricotemporal nerve. The only sensory branch of the anterior group is the buccal nerve. It runs through the upper part of the subtemporal fossa and crosses the anterior edge of the mandibular ramus on the level of the upper molars (9). The inferior alveolar nerve is the only nerve from the posterior group that runs in the ossified mandibular canal. It branches off the posterior trunk, laterally from the lingual nerve, and runs between the medial and lateral pterygoid muscles (10). Next it goes caudally and anteriorly between the sphenomandibular ligament and the mandibular ramus, towards the mandibular foramen. The inferior alveolar nerve has both the sensory and motor components. Before entering the mandibular canal it gives off the mylohyoid nerve, which lies in the mylohyoid sulcus. Subsequently, after reaching the posterior edge of the mylohoid muscle, the mylohyoid nerve passes on its caudal surface, giving off motor branches to this muscle and to the anterior belly of the digastric muscle (11). The sensory branches reach the skin around the mental protuberance. This nerve sometimes can join the lingual nerve by a small branch piercing the mylohyoid muscle. Among the nerves in the immediate vicinity of the mandible that might be significant during standard anaesthesia, is the lingual nerve. Usually it passes between the heads of the lateral pterygoid muscle or between the lateral and medial pterygoid muscles. Then, it runs with the buccal artery on the lateral surface of the buccal muscle, until it reaches the angle of the mouth. They pierce the buccal muscle, but do not innervate him, and supply the buccal mucosa, and partially the gums. They also innervate the skin of the cheek, joining the branches of the facial nerve.

Knowing the site of the mental foramen allows for accurate delivery of local anesthesia for dental procedures and the avoidance of damage to the nerve in surgical procedures. It also aids in interpreting anatomical landmarks in forensic dentistry (12),(13). Panoramic radiographs were utilized in the present study to assess the mental foramen as it provides more wide and reliable field to view anatomy of the mandible as compared with periapical radiographs. Limitation of the present study was the use of panoramic radiographs for locating mental foramen instead of

commencing an anatomic study on skulls. In spite of clinical importance of mental foramen, it also offers importance in forensic application because mandibular characteristics are extremely useful in gender determination. Wical et al (14), Lindh et al (15), and Guler et al (16) commenced studies and reported that even with resorption of the alveolar bone above the mental foramen, the space from the foramen to the inferior border of the mandible remains relatively constant throughout life. Thus, stability of this part is not affected by resorption of alveolar process above the foramen. Chandra et al (17) concluded that the length from lower border of the mandible to the mental foramen exhibits sexual dimorphism, and panoramic radiography is an efficient tool for assembly of planned dimensions and can be measured as an added radiographic method to establish gender from the skeletal remains. Thakur et al (18), Mahima et al (19), and Naroor et al (20) reported the possibility that the height of the mandible and the distance from the mental foramen to the alveolar crest can be used to determine gender. In this research, the results shows that the distance between the mental foramen and the inferior border of the mandible is directly proportionate to age of the patient. It is seen that the distance is decreasing with ageing and this may be de to bone resorption that causes thinning of the inferior border of the mandible. Also, there is no significant difference of the distance between the mental foramen and base border of mandible seen in patients of selected age groups according to gender.

Conclusion

The results from the study revealed that the distance between the mental foramen and the inferior border of the mandible influences in determining the age and not the gender. Thus to conclude, the position of mental foramen does not play a role determining the gender and has significance in determining the age. Knowledge of both the position of the mental foramen and its distance from neighboring anatomical structures provides useful information for surgery and can also help to prevent complications, such as paresthesia. Further studies in populations of different races or ethnicities should be conducted to determine the position of the mental foramen, its role in determining age and gender and its distance from neighboring anatomical structures.

Corresponding Author

PavithraPriyadarshoni S.

Tutor, Department of Public Health Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id: drpavithraphd88@gmail.com

Contact Number: 9176222104

References

1. Ngeow WC, Yazawah Y. The location of the mental foramen in a selected Malay population. J Orals 2003 : 45(3) : 171 -5
2. Shankland 2nd WE. The position of the mental foramen in Asian Indians. The Journal of oral implantology. 1994;20(2):118.
3. Jennifer N. The position of the mental foramen according to age and sex in Singaporeans Malays and Indians. AnesthProg 1989: 36: 276-8.
4. Tebo HG, Telford IB. An analysis of the variations in position of the mental foramen. Anat REC 1950: 107(1): 61-6.
5. Monalisa R, Babu YK. Age Related Changes in Mandible. Research Journal of Pharmacy and Technology. 2015 Aug 1;8(8):1155.
6. Madhavan S. Mandibular Development and Its Age Change. Journal of Pharmaceutical Sciences and Research. 2014 Nov 1;6(11):360.
7. Ennes JP, Medeisros RM. Localization of mandibular foramen and clinical implication. Int J Morphol, 2009: 4: 1305–1311.
8. Skrzat J, Walocha J, Srodek R, Nizankowska A. An atypical position of the foramen ovale. Folia Morphol, 2006: 65(4): 396–399.

9. Mérida-Velasco JR, Rodríguez-Vázquez JF, de la Cuadra C, Mérida- -Velasco JA, Jiménez-Collado J. The course of the buccal nerve: relationships with the tempo-ralis muscle during the prenatal period. J Anat, 2001: 198(4): 423–429.
10. Lazarov N, Usunoff K. Descending projections from the trigeminal ganglion and mesencephalic trigeminal nucleus. Trakia J Scienc, 2003: 1: 5–14.
11. Skrzat J, Mróz I, Walocha J, Zawiliński J, Jaworek JK. Bilateral ossification of the stylohyoid ligament. Folia Morphol, 2007: 66(3): 203–206.
12. Shankland WE. The position of the mental foramen in Asians Indians . J Oral Implantol. 1994: 20(2) : 118 -123.
13. Al. Jasser, Nwoku AL. Radiographic study of the mental foramen in a selected Saudi population. Dentomaxillofacradiol. 1998: 27(6) : 341-343.
14. Wical KE, Swoope CC. Studies of residual ridge resorption. Part 1. Use of panoramic radiographs for evaluation and classification of mandibular resorption. J Prosthet Dent 1974 Jul;32(1):7-12.
15. Lindh C, Peterson A, Klinge B. Measurements of distance related to the mandibular canal in radiographs. Clin Oral Implant Res 1995 Jun;6(2):96-103.
16. Guler AU, Sumer M, Sumer P, Bicer I. The evaluation of vertical heights of maxillary and mandibular bones and the location of anatomic landmarks in panoramic radiographs of edentulous patients for implant dentistry. J Oral Rehabil 2005 Oct;32(10):741-746.
17. Chandra A, Singh A, Badni M, Jaiswal R, Agnihotri A. Determination of sex by radiographic analysis of mental foramen in North Indian population. J Forensic Dent Sci 2013 Jan; 5(1):52-55.
18. Thakur M, Reddy VK, Sivaranjani Y, Khaja S. Gender determination by mental foramen and height of the body of the mandible in dentulous patients: a radiographic study. J Indian Acad Forensic Med 2014 Jan-Mar; 36(1):13-19.
19. Mahima VG. Mental foramen for gender determination: a panoramic radiographic study. Medico-Legal Update 2009;9(2):33-35.
20. Naroor N, Shenai P, Chatra L, Veena KM, Rao PK, Shetty P. Gender determination using the mental foramen. J Cranio Max Dis 2015 Jul;4(2):144-147.

Interarch and Intraarch Relationship of Anterior Teeth with Periodontal Conditions- A Conventional Study

Boopana .K[1], Ravindra Kumar Jain[2], Geo Mani[3]

Graduate Student[1], Senior Lecturer[2], Department of Orthodontics, Senior Lecturer[3], Department of Pedodontics and Preventive Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The Aim of this research to study the interarch and the intraarch relationship of the anterior teeth with periodontal conditions.This study is done to know the relationship between the orthodontic anomalies which is assessed using the IOTN index and periodontal conditions assessed by the plaque index and gingival bleeding index.

Materials and Methods: The three parameters of the intraarch relationship on both dental arches (displacement of contact point, crowding, spacing) and the four interarch relationship (overjet , overbite, crossbite) are assessed with index of orthodontic treatment need (IOTN) and are correlated with the periodontal condition, plaque index and the gingival bleeding index.

Result: A significant correlation was found between parameters of intraarch and interarch relationship and the indices of periodontal conditions such as plaque index, gingival bleeding index.

Conclusion: This study provides valuable indication on the usefulness of making orthodontic treatment on the ground of preservation of periodontal condition.

***Key Words**: gingival bleeding index, plaque index, orthodontic anomalies , interarch, intraarch.*

Introduction

The past decades have witnessed a steady increase of the children and the adults undergoing orthodontic treatment in most of the countries [1, 2].Improvement of facial and dental aesthetics are routinely cited to justify the provision of orthodontic treatment and also form the cornerstones of some orthodontic anomalies[4, 5]. The index of orthodontic treatment need (IOTN) records the treatment need on the basis of two components, a dental health component (DHC) and an aesthetic component. The DHC records the indications for the treatment on the ground of potential deleterious effects of malocclusion on the health and functioning of the dentition[6, 7]. The etiology and the pathogenesis of the periodontal disease are known to be multifactorial, but dental plaque is considered to be an essential precursor. So any factor presumed to promote the plaque retention or to make its removal difficult might contribute to periodontal disease[8, 9,15]. For example, crowding of the teeth creates areas hardly accessible to tooth brushing and has been recognized to make a thorough cleaning of the teeth laborious. Various types of malocclusions have also been correlated to increase plaque indices[10, 11]. This study was undertaken to further investigate the correlation between the interarch and intraarch relationship of the anterior teeth which is assessed by the IOTN and some of the periodontal conditions, ie, hygiene , gingival bleeding index and plaque index[12,13].

Periodontitis is a chronic bacterial infection characterized by persistent inflammation, connective tissue breakdown and alveolar bone destruction mediated by pro-inflammatory mediators. Tumor necrosis factor-alpha (TNF-α) is an important pro-inflammatory mediator that produced causes destruction of periodontal tissues[20]. The etiology and pathogenesis of periodontal disease are known to be multifactorial, but dental plaque is recognized as an essential precursor. Hence, any factor presumed to promote plaque retention or to make difficult its removal might contribute to the local risk of periodontal disease. For instance, crowding of teeth creates areas hardly accessible to tooth brushing and has been recognized to make a thorough cleaning of the teeth laborious. Various types of malocclusions have also been correlated to increased plaque indices.[13] On the other hand, many investigations failed to

demonstrate any significant correlation between tooth irregularities and periodontal state. In addition, some authors stressed that the enhancement provided by orthodontic treatment in terms of periodontal health may be of minor importance compared with maintenance of good oral hygiene. Also, recently, Geiger wondered about the available findings on the correlation between malocclusion and periodontal disease and emphasized the need for additional

quantitative studies to validate the predictability of malocclusion as an etiologic factor of periodontal disease.

This study was undertaken to further investigate the correlation between the intraarch and interarch relationships of the anterior teeth as assessed by the IOTN and the ICON and some parameters of periodontal conditions, ie, hygiene, inflammation , disease severity, and treatment need.[17]

Materials and Methods

Subject

The study population consists of a sample of 100 young adults (44 females and 56 males), aged 20 to 35 years with no history of orthodontic treatment. They were local people from different areas of Chennai, India, who had come for a dental checkup to saveetha dental college and hospital. They participated in the study on a voluntary basis after receiving comprehensive information about the aims and design of the study.

Inclusion Criteria

To be included in the study subjects needed to fulfill the following criteria: 1)possession of the six natural teeth of the anterior sextant (incisors and canines) in each arch2) manual tooth brushing once or twice a day3) knowledge about oral hygiene methods.

Exclusion Criteria

All the subjects who had undergone professional plaque removal and scaling in the past five months were not included in the study.

Assessment of Periodontal Condition

The study subjects were examined for evaluation of the periodontal conditions in the dental office under the optimal conditions with a periodontal probe and a mouth mirror. Parameters related to the periodontal status were assessed directly in the upper and lower anterior sextant of both the maxillary and mandibular arches. These include the oral hygiene index by Greene and Vermillion 1960 and the gingival bleeding index by Ainamo and Bay 1975.

Oral Hygiene: The oral hygiene status was evaluated by measuring the amount of soft and mineralized deposit present on the tooth surface. The plaque was calculated on all the four surfaces labial , lingual , mesial and distal of both the incisors and the canines. . Cavities and restorations were not considered. The plaque was measured by the plaque index Silness and Loe in 1964 on all the four surfaces, and the mean score was obtained by dividing it by 4 for

each tooth. They are measured for all the 6 anterior teeth both in the upper jaw and the lower jaw and the mean score is obtained by dividing it by 6

Plaque Index : Plaque index by Silness and Loe certains the thickness of the plaque along the gingival margin; plaque plays an etiology of gingivitis13, 14.

Gingival Inflammation : the gingival inflammation was assessed by the gingival index (GBI). The gingival bleeding index is recorded after a gentle probing on the mesial, midline, and distal on the labial and the lingual surface of the each targeted teeth. Intraarch and Intraarch Relationship Parameters

The interarch and the intraarch relationship was assessed on the anterior sextant (teeth 13 to 23 and 33 to 43) by the IOTN index.

Result

The plaque index was calculated for all the samples and the mean value was taken separately for the male and female. The result shows that the female have lesser plaque when compared to the male. The oral hygiene of the female is better when compared to male. The mean value obtained for females in the upper anterior teeth is 0.83 ± 0.24 (MEAN + SD) and for the lower anterior teeth is 1 ±0.1(MEAN+SD). In case of males the plaque index is comparatively more, the upper anterior teeth have an average plaque index of 1 ± 0.34 and for the lower anterior teeth it is 2.12 ± 1.22 (MEAN + SD). The plaque index score is tabulated in table 1

Table 1: Comparison of Plaque Index and gingival bleeding index for Male and Female in the upper arch .

Periodontal Conditions	Male		Female	
Upper Arch	(Mean±SD)	Interpretation	(Mean±SD)	Interpretation
Gingival bleeding index	1.25 ± 0.353	moderate gingival inflammation	1.05 ± 0.212	Slight gingival inflammation
Plaque Index	1 ± 0.34	Average plaque control	0.83 ± 0.24	Very slight amount of plaque

The gingival bleeding index was taken for all the patients and they are recorded in the Performa. The mean of the gingival bleeding index was taken for the female upper arch and lower arch and male upper arch. The mean value of the gingival bleeding index for the female upper arch is 1.05 ± 0.212 and the gingival bleeding index for the female lower arch is 1 ± 0.12. The gingival bleeding index for the males is comparatively more than the females. The male upper arch has a mean value of 1.25 ± 0.353 and the lower arch has a mean value of 2 ± 1.414. The statistical analysis has shown the males have higher gingival bleeding index and plaque index when compared with females. The gingival bleeding index score is tabulated in table 2.

Table 2: Assessment of Gingival Bleeding Index and Plaque Index in the Lower Arch.

Lower Arch	(Mean ± SD)	Interpretation	(Mean ± SD)	Interpretatio
Plaque Index	2.12 ± 1.22	Severe plaque present	1 ± 0.1	Slight amount plaque
Gingival bleeding Index	2 ± 1.414	Severe gingival inflammation	1 ± 0.12	Moderate gingival inflammatio

The statistical analysis for the IOTN grading and the periodontal condition was done. Out of 100 patients 18 patients had minor malocclusion, including the contact point displacement less than 1mm. which belongs to grade 1. 20 patients had anterior and posterior open bite greater than 2mm but less than or equal to 4mm belongs to grade 2. 17 patients had increased overbite greater than 3mm with incompetent lips which belongs to grade 3. 5 patients had less extensive hypodontia requiring orthodontic space closure in upper anteriors which belongs to grade 4.

Rest of the 35 patients had impeded eruption of teeth due to crowding, displacement, presence of supernumerary teeth. The mean of the gingival bleeding index and the plaque index was calculated for each condition and is tabulated below

Table 3: Comparison of IOTN condition with Gingival bleeding Index and Plaque Index

IOTN Condition	PlaqueIndex (Mean±SD)	Condition	Gingival Bleeding Index (Mean±SD)	Condition
Grade 1 minor malocclusion, including the contact point displacement ≤1mm	0.5 ± 0.121	Very minimum plaque	1.1 ± 0.622	Less gingival inflammation
Grade 2 anterior and posterior open bite ≥ 2mm but ≤ 4mm	1.12 ±0.1	Moderate plaque	1.23 ± 0.322	Moderate gingival inflammation
Grade 3 increased overbite ≥ 3mm with incompetent lips	1.32 ± 0.32	Moderate plaque	2.1 ± 1.414	Moderate gingival inflammation
Grade 4 less extensive hypodontia requiring orthodontic space closure	0.93 ± 0.12	Very minimum plaque	1.32 ± 0.32	Less gingival inflammation
Grade 5 crowding , displacement , presence of supernumerary teeth	2.31 ± 1.11	High amount of plaque	3.22 ± 1.52	Severe gingival inflammation

Discussion

In case of males the plaque index is comparatively more, the upper anterior teeth have an average plaque index of 1 ± 0.34 and for the lower anterior teeth it is 2.12 ± 1.22 (MEAN + SD). The statistical analysis has shown the males have higher gingival bleeding index and plaque index when compared with females. The statistical analysis shows, for grade 1, minor malocclusion, including the contact point displacement less than or equal to 1mm there is very minimum plaque present and less gingival inflammation. In grade 2 , the anterior and posterior open bite less than or equal to 2mm but greater than 4mm there is moderate plaque and also moderate gingival inflammation. In grade3, increased overbite greater than 3mm with incompetent lips there is moderate amount of plaque and also gingival inflammation. In grade 4, less hypodontia requiring orthodontic space closure, there is very minimum amount of plaque and also very gingival inflammation. In grade 5, where there is crowding, displacement

, presence of supernumerary teeth there is high amount of plaque and also severe gingival inflammation. It shows that in crowding case the patients show high amount of plaque which is because the brushing cannot clear all the plaque due to the presence of tooth displacement and the presence of supernumerary teeth.

Varying degrees of correlation have been found between intraarch relationship and periodontal condition depending on the arch and the indices taken into account. Overall, it appears that, despite a normal hygiene regimen, the presence of malpositioned and crowded teeth in the mandibular arch is associated with difficulties in plaque removal and gingival inflammation. On the other hand, these parameters ranked lower in the maxillary teeth18,19 Comparisons with previous studies are complicated by virtue of the differences in methodological approaches and the indices used to assess both the periodontal and the occlusal conditions. Ingervall et al also studied the relationship between crowding of teeth and gingival condition in a smaller sample of dental students of almost the same age bracket, as in subjects of this study. They found that crowding of teeth (tooth displaced by two mm or rotated for at least 15) did not favor plaque accumulation on proximal tooth surfaces and influenced gingival inflammation only slightly20 Geiger et al1also found no significant correlation between crowding and periodontal condition21. On the other hand, Buckley in a study of 300 teenagers reported a low but significant correlation in the range of that found in this study between lower

incisor crowding as assessed by the Occlusal Feature Index, the PlI and the GI. The reason why crowding, although significantly correlated with hygiene and inflammation, is not correlated to disease severity is elusive. However, it is noteworthy that although gingivitis must precede periodontitis, not all gingivitis progresses to periodontitis22.

Conclusion

A significant correlation was found between certain parameters such as the gingival bleeding index, the plaque index with intra-arch and inter-arch relationship of the anterior teeth and periodontal conditions. This study provides valuable indication on the usefulness of making orthodontic treatment on the ground of preservation of periodontal condition.

Corresponding Author

Geo Mani

Senior Lecturer, Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077,

Tamil Nadu, India

Email Id: geosdch@gmail.com,
Telephone Number: 7358433246.

Reference

1. Shaw WC, O'brien KD, Richmond S. Quality control in orthodontics: factors influencing the receipt of orthodontic treatment. British Dental Journal. 1991 Jan 19;170(2):66.
2. Brook PH, Shaw WC. The development of an index of orthodontic treatment priority. The European Journal of Orthodontics. 1989 Aug 1;11(3):309-20.
3. ENG F. The development of the index of complexity, outcome and need (ICON). Journal of orthodontics. 2000;27:149-62.
4. Axelsson P, Lindhe J. The effect of a preventive programme on dental plaque, gingivitis and caries in schoolchildren. Results after one and two years. Journal of Clinical Periodontology. 1974 Jun 1;1(2):126-38.
5. Socransky SS. Relationship of bacteria to the etiology of periodontal disease. Journal of dental research. 1970 Feb;49(2):203-22.
6. Addy M, Griffiths GS, Dummer PM, Kingdon A, Hicks R, Hunter ML, Newcombe RG, Shaw WC. The association between tooth irregularity and plaque accumulation, gingivitis, and caries in 11–12-year-old children. The European Journal of Orthodontics. 1988 Feb 1;10(1):76-83.
7. Ashley FR, Usiskin LA, Wilson RF, Wagaiyu E. The relationship between irregularity of the incisor teeth, plaque, and gingivitis: a study in a group of schoolchildren aged 11-14 years. The European Journal of Orthodontics. 1998 Feb 1;20(1):65-72.
8. Buckley LA. The relationships between malocclusion, gingival inflammation, plaque and calculus. Journal of periodontology. 1981 Jan 1;52(1):35-40.
9. Al-Jasser N, Hashim H. Periodontal findings in cases of incisor cross-bite. The Journal of clinical pediatric dentistry. 1995;19(4):285-7.
10. Hashim HA, Al-Jasser NM. Periodontal findings in cases of posterior cross-bite. The Journal of clinical pediatric dentistry. 1996;20(4):317-20.

11. JAMES B, Jacobsson U, Nyman S. A clinical study of the relationship between crowding of teeth, periodontal and gingival condition. Journal of clinical periodontology. 1977 Sep 1;4(3):214-22.
12. Davies TM, Shaw WC, Worthington HV, Addy M, Dummer P, Kingdon A. The effect of orthodontic treatment on plaque and gingivitis. American Journal of Orthodontics and Dentofacial Orthopedics. 1991 Feb 1;99(2):155-61.
13. Geiger AM, Wasserman BH, Turgeon LR. Relationship of Occlusion and Periodontal Disease: Part VI.—Relation of Anterior Overjet and Overbite to Periodontal Destruction and Gingival Inflammation. Journal of periodontology. 1973 Mar;44(3):150-7.
14. Geiger AM, Wasserman BH, Turgeon LR. Relationship of Occlusion and Periodontal Disease: Part VI.—Relation of Anterior Overjet and Overbite to Periodontal Destruction and Gingival Inflammation. Journal of periodontology. 1973 Mar;44(3):150-7.
15. Vellayappan R. Shreeja.S.Varghese. Periodontal approach to improve Aesthetics: A Case Report. Journal of Medical Biomedical and Applied Sciences. 2017 Mar 6;5(3).
16. Geiger AM. Malocclusion as an etiologic factor in periodontal disease: a retrospective essay. American journal of orthodontics and dentofacial orthopedics. 2001 Aug 1;120(2):112-5
17. Ingervall B, Jacobsson U, Nyman S. A clinical study of the relationship between crowding of teeth, plaque and gingival condition. Journal of clinical periodontology. 1977 Sep 1;4(3):214-22.
18. Davies TM, Orth D, Shaw WC, Addy M, Dummer PM. The relationship of anterior overjet to plaque and gingivitis in children. American Journal of Orthodontics and Dentofacial Orthopedics. 1988 Apr 1;93(4):303-9.
19. Sangeetha S. Prosthodontic Space Management in Anterior Tooth Region Esthetically-A Case Study. Journal of Pharmaceutical Sciences and Research. 2016 Mar 1;8(3):173.
20. Lavezy LA. The relationships between malocclusion, gingival inflammation, plaque and calculus. Journal of periodontology. 1981 Jan 1;52(1):35-40.
21. David FR, Usiskin LA, Wilson RF, Wagaiyu E. The relationship between irregularity of the incisor teeth, plaque, and gingivitis: a study in a group of schoolchildren aged 11-14 years. The European Journal of Orthodontics. 1998 Feb 1;20(1):65-72.

22. Varghese SS, Thomas H, Jayakumar ND, Sankari M, Lakshmanan R. Estimation of salivary tumor necrosis factor-alpha in chronic and aggressive periodontitis patients. Contemporary clinical dentistry. 2015 Sep;6(Suppl 1):S152.

Invitro Antibacterial Activity of Acacia Catechu and RosmarinusOfficianalison Streptococcus Mutans.

S.Pooja[1], Geetha RV[2], Geo Mani[3],
Graduate Student[1], Reader[2],Department of Microbiology, Senior Lecturer[3], Department of Pedodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: Dental caries is steadily increasing in the underdeveloped and developing countries. S*treptococcus mutans* is a potent initiator of caries and various oral problems. The usage of these herbal extracts can be beneficial to the patients in maintaining the oral hygiene Herbal extracts have been found more useful in curing number of diseases including bacterial diseases leading to a rich source of antimicrobial agents. *Rosmarinus officinalis* belongs to *lamiaceae*family of herbs which is used as a food flavouring and medicinally known for its antibacterial properties. *Acacia catechu* which belongs to *Fabaceae*family of herbs is highly known for its powerful antibacterial and antioxidant activities.The aim of this study is to estimate the antibacterial activity of leaf extract Acacia catechu and Rosmarinus officinalis on streptococcus mutans.

Materials and Method: The antibacterial activity of leaf extract of Acacia catechu and Rosmarinus officinalis on Streptococcus mutans was screened using disc diffusion technique.

Results: The results of our present study shows the two extracts used have good antibacterial action on *Strepococcusmutants.*

Conclusion: Theuse and efficacy of medicinal plants contribute to their therapeutic properties, so that they are frequently prescribed.

Key Words*: Dental caries, anti bacterial, disc diffusion technique*

Introduction

The mouth cavity contains many microorganisms responsible for various infections and inflammatory diseases. The formation of dental caries is caused by the accumulation and colonization of oral microorganisms, especially *S. mutans* which adhere to and colonize the surface of teeth. Dental treatments in many countries are expensive and not easily accessible; therefore, people have turned to the use of medicinal plants in the form of composition of tooth paste, or simply chewing the plants directly to protect the teeth from diseases. Different plant species of medicinal importance have successfully been included

in mouthwashes and toothpastes in many developing countries.

Acacia catechu has a place with Family Fabaceae and subfamily Mimosoideae (1).The concentrates of Acacia catechu shows different pharmacological impacts like antipyretic, calming, antidiarrhoeal, hypoglycaemic, hepatoprotective, cell reinforcement and antimicrobial activities(2).Acacia catechu is exceptionally significant for its capable astringent and cancer prevention agent activity(3).Main substance constituents of Acacia catechu willd are catechin, (-)epicatechin, epigallocatechin, epicatechingallate, epigallocatechingallete,rocatechinphloroglucin,protocatechuic corrosive, quercetin, Porifera sterolglucosides, Poriferasterolacyglucosides, lupenone, lupeol, procynadinAC, kaempferol, dihydrokaemferol, taxi folin, (+)- afzelchin gum and mineral (4). Acacia catechu is useful as a topical agent for sore gums and mouth ulcers(5).oral bacteria like *Streptococcus mutans* is an potent initiator that causes dental caries/plaques(6).*Rosmarinus officinalis* (Rosemary) have been known to have antimicrobial, antioxidant, anti-diabetic, and anti-tumorigenic activities (7).In addition, it is known to affect the cell's adhesive properties, self-aggregation, and protein secretion; and this might help in the treatment of cardiovascular diseases andthrombosis .*Rosmarinus officinalis* have anticancer properties . Rosmarinus officinalis (Rosemary) was generally utilized for the treatment of a few diseases, for example, urinary tract contaminations, rheumatoid cholecystitis, loose bowels, and hypertension (8).Among the pharmacologically approved restorative employments of rosemary are antibacterial, anticancer ,antidiabetic, against inammatory and antinociceptive, cell reinforcement , antithrombotic , antiulcerogenic, enhancing subjective , antidiuretic , and hepatoprotective. other significant utilization of rosemary is in the perfumery business where the fundamental oils are utilized as normal elements of fragrances(9). Fundamental oils of rose-mary ruled by 1,8-cineole, α-pinene, camphene, α-terpineol, and borneol as key constituents are additionally in charge of different pharmacological impacts of the general cancer prevention agent and antimicrobial properties known for some, basic oils, and additionally other e ects including anticarcinogenic activities(10). *S. mutans* is common flora of human mouth that has polysaccharide capsule and has important rule to produce dental caries (11).Short chain carboxylic acid released from the enzymes of streptococcus mutans used as its fermentation by-products demineralize the enamel and lead to cavitation in the tooth. Hence considered to be the most cariogenic of all streptococci family(12). Hence the aim of this study is to estimate the antibacterial activity of *Acacia catechu* and *Rosmarinus officinalis* on *streptococcus mutans.*

Materials and Methods(13-15)

Test Microorganism

Bacterial strain used was Streptococcus mutans. The living being was secluded utilizing particular media Mutans - Sanguis agar [Hi media M977], and kept up in supplement agar slant at 4°C in branch of Microbiology, Saveetha Dental College.

Methodology

The leaf extracts of *Acacia catechu* and *Rosmarinus officinalis* were dissolved in distilled water in following concentrations 2.5mg/ml, 5mg/ml and 10mg/ml so that 100µl delivers

250µg/ml, 500µg/ml and 1000 µg/ml respectively .100µl of the extract were loaded on sterile filter paper discs measuring 6mm. The discs were dried and kept aseptically.

Screening of Antibacterial Activity [Disc diffusion technique]

Broth culture of the bacterial strain compared to Mac Farland's standard [4,5,6] 0.5 was prepared. culture of the test creatures were made on the Muller Hinton agar [MHA-Hi media M1084] plates utilizing sterile cotton swab and the plates were dried for 15 minutes.Filter paper discs loaded with different concentrations of the extract were placed on the respective plates. The plates were incubated at 37°C overnight and the zone of inhibition of growth was measured in millimeters. All the tests were done in triplicate to minimize the test error.

Result

Table 1: Anti-bacterial activity of *Acacia catechu* and *Rosmarinus officinalis*on Streptococcus mutans

Organism	Concµg/ml	E1 [Zone of inhibition in mm diameter]	E2 [Zone of inhibition in mm diameter]	Chlorhexidine [Zone of inhibition in mm diameter]
Streptococcus mutans	250	16	09	22
	500	19	14	30
	1000	26	22	38

E 1 – *Acacia catechu* E2 - *Rosmarinus officinalis* ,

The antibacterial movement of the fundamental oils at various fixations was screened by plate dispersion procedure and the zone of hindrance was measured in mm distance across.The results are given in the table 1. Acacia catechu leaf extract was more effective against *Streptococcus mutans* with a zone of inhibition of 26mm diameter (at conc1000 µg/ml.), *Rosmarinus officinalis* showed a zone of 22mm diameter . The use of natural antimicrobials may contribute to control the disordered growth of oral microbiota, thus overcoming problems caused by species resistant to conventional antimicrobials.

Discussion

Oral cavity is a complex ecosystem with higly divergent acid tolerant and acid-producing microbiota. Bacteria accumulate on both the hard andsoft oral tissues in biofilms(16). The primary acid tolerant bacteria associated with Dental plaqueincludes streptococcus mutans(17). The ethanolic bark extract of Acacia catechu has a antibacterial activity against streptococcus mutans(18).Acacia catechu Willd is widely used in Ayurveda for many diseases and mainly for skin diseases.Acacia catechu is very significant for its

effective astringent and cancer prevention agent exercises. It is ordinarily known as Katha which is a key element of Pan that is insect leaf readiness bit in India. It is helpful in Dental, oral, throat diseases and as an astringent for lessening overflowing from endless ulcers and wounds(19). In stomatitis, halitosis, dental caries and cavities, halitosis, dental caries and cavities, Acacia catechu is used with great benefit(20). Moreno et al. (2006) detailed that rosemary plants are rich wellsprings of phenolic mixes with high antimicrobial action against both Gram-positive and Gram-negative microscopic organisms. Plainly rosemary removes have bioactive properties, yet their antimicrobial exercises have not been profoundly described.Ouattara et al. (1997) researched the antibacterial movement of chose fundamental oils against some sustenance deterioration living beings. They reasoned that the fundamental oils of cinnamon, clove and rosemary were the most dynamic. Comparative outcomes were acquired by Valero and Salmeron (2003) for the antibacterial movement of rosemary basic oil against Bacillus cereus strains developed in carrot juices. Numerous mixes have been disengaged from rosemary, including flavones, diterpenes, steroids, and triterpenes. Of these, the cell reinforcement movement of rosemary removes has been basically identified with two phenolic diterpenes: carnosic corrosive and carnosol(24). Rosemary basic oil is an antimicrobial source and substance blend whose cancer prevention agent properties and antimicrobial impacts have been demonstrated in different inquires about. For instance, phenolic compound is found in rich. Basic oil is one of the principle mixes of this herb which exists in 1-2%(25). It comprises of borneole, limonene, camphene, camphor and different mixes like phenolic corrosive including rosmarinic corrosive, caffeic corrosive and chlorogenic corrosive. Antimicrobial impacts of rosemary fundamental oil and concentrate on different microorganisms have been examined. Jarrar et al. analyzed the impact of ethanol concentrate of rosemary alone and with cefuroxime on methicillin safe staphylococcus(26).

Natural substances have demonstrated antibacterial action mainly because most plants used in alternative medicine are composed of flavonoids, which act on bacterial cells disrupting the cytoplasmatic membrane and inhibiting the enzymatic activity. In our present study the two extracts used have goodanti microbial action on Strepococcus mutans. A study byEllon et.al showed that*A. catechu* leaves have still exerted some degrees of inhibition against Gram-negative organisms(27). Another study by Anitha Roy et.al showed that higher concentration of the extract showed good antifungal activity against all the fungal strains tested(28). A study by Morrissey et.al evaluated the antibacterial and antifungal potential of leaf extract of *A. catechu* so the use of this plant as an anti-infective agent in the ayurvedic medicine has been justified(29).

Conclusion

The use of plants for treating diseases is as old as the human species. Famous perceptions on the utilization and adequacy of restorative plants altogether add to the revelation of their remedial properties, with the goal that they are as often as possible endorsed, regardless of whether their compound constituents are not generally totally known. The consequences of our present examination demonstrates the two concentrates utilized have great hostile to bacterial activity on Streptococcus mutants. These plants have potential for improvement of antimicrobial operators against oral microorganisms, for use in tooth glue, mouth wash and so forth to prevent and treating oral contaminations.

Corresponding Author
Geo Mani,
Senior Lecturer,Department of Pedodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India
162, Poonamallee High Road, Chennai -600 077, Tamil Nadu, India.
Email Id: geosdch@gmail.com
Telephone number: 7358433246

Reference

1. Lakshmi T, Kumar A. Preliminary phytochemical analysis & Invitro Antibacterial activity of Acacia catechu willd Bark against Streptococcus mitis, Streptococcus sanguis& Lactobacillus acidophilus. *International journal of Phytomedicine.* 2011 Oct 1;3(4):579.

2. Kumar KS. Herbal Pharmacopoeias–an overview of international and Indian representation. *J Ayu Herb Med.* 2015;1:59-60.

3. Lakshmi T, Geetha RV, Roy A. In vitro evaluation of anti bacterial activity of Acacia catechu willd. heartwood extract. *Int J Pharm Biosci.* 2011;2(2):B188-92.

4. Pharmacopoeia IH, Indian Drug Manufacturers Association. Mumbai. India: *Indian Drug Manufacturing Association.* 2002:33-9.

5. Sham JS, Chiu KW, Pang PK. Hypotensive action of Acacia catechu. *Plantamedica.* 1984 Apr;50(02):177-80.

6. Ferraro MJ. Performance standards for antimicrobial susceptibility testing. *NCCLS*; 2001.543-559.

7. Gupta A, Gupta R, Lal B. Effect of Trigonellafoenum-graecum (fenugreek) seeds on glycaemic control and insulin resistance in type 2 diabetes mellitus: a double blind placebo controlled study. *Alternative Medicine Review.* 2002 Jun 1;7(3):255-6.

8. Huang MT, Ho CT, Wang ZY, Ferraro T, Lou YR, Stauber K, Ma W, Georgiadis C, Laskin JD, Conney AH. Inhibition of skin tumorigenesis by rosemary and its constituents carnosol and ursolic acid. *Cancer research.* 1994 Feb 1;54(3):701-8.

9. Bozin B, Mimica-Dukic N, Samojlik I, Jovin E. Antimicrobial and antioxidant properties of rosemary and sage (Rosmarinus officinalis L. and Salvia officinalis L., Lamiaceae) essential oils. *Journal of agricultural and food chemistry.* 2007 Aug 21;55(19):7879-85.

10. Fernandes CP, Mascarenhas MP, Zibetti FM, Lima BG, Oliveira RP, Rocha L, Falcão DQ. HLB value, an important parameter for the development of essential oil phytopharmaceuticals. RevistaBrasileira de Farmacognosia. 2013 Feb;23(1):108-14.

11. Hamada S, Koga T, Ooshima T. Virulence factors of Streptococcus mutans and dental caries prevention. *Journal of dental research.* 1984 Mar;63(3):407-11.

12. Kumar AS, Lakshmi T, Arun AV. Invitro Antibacterial Activity of Acacia catechu ethanolic leaf extract against selected acidogenic oral bacteria. Research Journal of Pharmacy and Technology. 2012 Mar 1;5(3):333.

13. Tille P. Bailey & Scott's Diagnostic Microbiology-E-Book. *Elsevier Health Sciences*; 2015 Dec 28. Mosby page 229 – 257.

14. Jain RA, Patni VI, Arora DK. Isolation and identification of flavonoid “quercetin” from Acacia catechu (LF) Willd-A katha yielding plant. *Journal of Phytological Research.* 2007;20(1).

15. Geetha RV, Roy A, Lakshmi T. In vitro evaluation of anti bacterial activity of heartwood extract of Acacia catechu on oral microbes. *Int J Curr Res Rev.* 2011 Jun;3:4-9.

16. Rogers AH, editor. Molecular oral microbiology. *Horizon Scientific Press*; 2008.

17. Anhoury P, Nathanson D, Hughes CV, Socransky S, Feres M, Chou LL. Microbial profile on metallic and ceramic bracket materials. *The Angle Orthodontist.* 2002 Aug;72(4):338-43.

18. Diamanti- Kipioti A, Gusberti FA, Lang NP. Clinical and microbiological effects of fixed orthodontic appliances. *Journal of Clinical Periodontology.* 1987 Jul 1;14(6):326-33.

19. Naik GH, Priyadarsini KI, Satav JG, Banavalikar MM, Sohoni DP, Biyani MK, Mohan H. Comparative antioxidant activity of individual herbal components used in Ayurvedic medicine. *Phytochemistry.* 2003 May 1;63(1):97-104.

20. Geetha RV, Roy A, Lakshmi T. In vitro evaluation of anti bacterial activity of heartwood extract of Acacia catechu on oral microbes. *Int J Curr Res Rev.* 2011 Jun;3:4-9.

21. Moreno S, Scheyer T, Romano CS, Vojnov AA. Antioxidant and antimicrobial activities of rosemary extracts linked to their polyphenol composition. *Free radical research.* 2006 Jan 1;40(2):223-31.

22. Ouattara B, Simard RE, Holley RA, Piette GJ, Bégin A. Antibacterial activity of

selected fatty acids and essential oils against six meat spoilage organisms. *International journal of food microbiology.* 1997 Jul 22;37(2-3):155-62.

23. Valero M, Salmeron MC. Antibacterial activity of 11 essential oils against Bacillus cereus in tyndallized carrot broth. *International journal of food microbiology.* 2003 Aug 15;85(1-2):73-81.

24. Frankel EN, Huang SW, Aeschbach R, Prior E. Antioxidant activity of a rosemary extract and its constituents, carnosic acid, carnosol, and rosmarinic acid, in bulk oil and oil-in-water emulsion. *Journal of Agricultural and Food Chemistry.* 1996 Jan 18;44(1):131-5.

25. Bakkali F, Averbeck S, Averbeck D, Idaomar M. Biological effects of essential oils–a review. *Food and chemical toxicology.* 2008 Feb 1;46(2):446-75.

26. Jarrar N, Abu-Hijleh A, Adwan K. Antibacterial activity of Rosmarinus officinalis L. alone and in combination with cefuroxime against methicillin–resistant Staphylococcus aureus. *Asian Pacific Journal of Tropical Medicine.* 2010 Feb 1;3(2):121-3.

27. Eloff JN. Which extractant should be used for the screening and isolation of antimicrobial components from plants?.*Journal of ethnopharmacology.* 1998 Feb 1;60(1):1-8.

28. Roy A, Geetha RV, Lakshmi T. In vitro evaluation of anti mycotic activity of heartwood extract of Acacia catechu Willd. *Journal of pharmacy research.* 2011;4(7).

29. Morrissey JP, Osbourn AE. Fungal resistance to plant antibiotics as a mechanism of pathogenesis. *Microbiology and Molecular Biology Reviews.* 1999 Sep 1;63(3):708-24.

Invitro Antimicrobial Effectiveness of Herbal Extracts against Selected Oral Microorganisms

A.Thilagavathy[1],Lakshmi.T[2],Geetha R.V[3],Aravind Sivakumar[4],

Graduate Student[1],Professor[2],Department of Pharmacology,Professor[3],Department of Microbiology,

Senior Lecturer[4], Department of Orthodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The objective of our study is to investigate the in vitro antibacterial activity of acetone leaf extract of Azadirachta Indica and Uva ursiagainst selected oral microbes that causes dental plaque in fixed orthodontic appliance patients.Herbal based compounds plays a vital role in dentistry. Orthodontic patients wearing fixed orthodontic appliances have dental plaques which may induce gingivitis too,Azadirachtaindica ,Uvaursi commonly known as neem possess significant anti-inflammatory and anti cariogenic activity, hence it can be recommended for these patients.

Materials and Methods: The inhibitory effect of acetone leaf extract of Azadirachta indica and Uva ursi were tested against streptococcus mutans,streptococcus salivarius,streptococcus mitis by using the Broth culture method.

Results: The acetone leaf extract of Azadirachta indica exhibited significant antibacterial activity against streptococcus mutans and streptococcus mitis with minimum bactericidal concentration of 62µg/ml whereas, Uva ursi acetone leaf extract showed antibacterial activity against streptococcus mutans streptococcus mitis ,streptococcus salivarius at concentration of 1mg/ml.

Conclusion: The acetone leaf extract ofAzadirachta indica and Uva-ursi were found to be bactericidal in action against tested bacterial strains.

Key Words:*Azadirachta indica, Uva ursi, anti bacterial activity, MBC, oral microbes ,orthodontic fixed appliance ,dental plaque*.

Introduction

Human dental plaque was one of the ecosystem. which microorganism was firstobserved. Dental plaque refers to the colonies of bacterial cell embedded in a protein matrix and polysaccharide which adheres to the surface of teeth by a characteristic bacterium.[1]Dental caries/plaque is a chronic disease of multifactor etiology and pathogens.[2] The early stage of dental caries is characterized by a destruction of superficial dental structures caused by acids which are by-products of carbohydrate metabolism by streptococcus mutans .[3] Teeth affected with dental caries are sources of infection, which can cause an inflammation of dental pulp, periodonteum and gums. If left untreated, this disease gradually leads to teeth loss, which causes chewing difficulties and aesthetic problems. [4]

The major role in prognosis and development of oral diseases is played by streptococci species from mutans group. These microorganisms shows their action of initiation of caries with the formation of the dental biofilm i.e. dental plaque.[5]acidogenic oral bacteria like streptococcus mutans, streptococcus salivarius, streptococcus mitis,streptococcus sanguis primarily causes dental caries/plaque (Lakshmi .T et al ., 2011)that surround orthodontic appliances in many patients undergoing orthodontic treatment [6] Such bacteria can lead to tooth enamel breakdown and potential discoloration of the tooth surface, and these aesthetic changes can persist for many years after orthodontic treatment[7,8]

It also has been reported that thepresence of fixed orthodontic appliance greatlyaffects oral hygiene and creates new retentive areas for plaque and debris [7] ,which in turn predisposes to increased carriage of microbes and subsequent infection .Therefore, prevention of bacterial attachment to orthodontic appliance is a critical concern for orthodontists[8,9]. The use of natural products has been one of the most successful strategies for the discovery of new drugs.

Azadirachta Indica commonly known as Neem which is used to treat various disease condition. Every part of the tree is used as traditional medicine for household remedy against various human ailments, from ancient period[10-15]. Nimbidin, a major crude bitter principle extracted from the oil of seed kernels of A. indica demonstrated several biological activities. Studies shows that Neem leaf extract is used to treat dental plaque and gingivitis.

Uva ursi (Arctostaphylos uva ursi), also known as bearberry because bears like eating the fruit, possess medicinal value. Native Americans used it as a remedy for urinary tract infections.[16] In fact, until the discovery of sulfa drugs and antibiotics, uva ursi was a common treatment for such bladder and related infections. The herb also contains tannins that have astringent effects, helping to shrink and tighten mucous membranes in the body. That, in turn, helps reduce inflammation and fight infection(Lakshmi .T et al .,2012).Uva ursi leaves are used for treatment of disease conditions not the berries.[17]

Several literature review proves that plants as intact crude organs and their products (e.g., powdered plants, extracts, etc) have been widely used by different cultures to promote oral hygiene since antiquity. Hence an attempt was taken in this study to evaluate the antibacterial efficacy of Azadirachtaindica and uvaursiinvitro against selected oral microbes inorder to control dental plaque that seems to be difficult to get removed by mechanical device in fixed appliance patients undergoing orthodontic treatment.

Materials and Method

Plant Material

Acetoneleafextract of Azadirachtaindica and Uvaursiwere collected from Hosur,

Tamil Nadu and were authenticated by Green Chem lab, Bengaluru ,India.

Preparation of Acetone Extract

Leaves were shade dried for a week. Dried leaves were milled to fine powder. Powder was passed through 100 mesh sieve and stored in a sealed polythene bag.2.5kg of powdered plant material were extracted with 10 liters of acetone at 65°C temperature, for 1 hour, in a 20 liter round bottom flask with Graham condenser attached. Condenser was cooled circulating with chilled water. After 1 hour of extraction, round bottom flask was cooled to room temp and the extract were filtered and collected. The Marc was extracted repeatedly with 10 liters of acetone, twice. The extracts were filtered and collected. The combined extracts was evaporated to dryness under reduced pressure in a Buchi Rotary Evaporator (Switzerland) at 65°C, to obtain 150g of powder extract. The w/w yield of the prepared extract was 6%. The extract were stored at 4 °C until used.

Test Microorganisms

Bacterial strains used were streptococcus mutans (ATCC 25175) ,streptococcus salivarius (ATCC 25975),streptococcus mitis (ATCC 9811) The organisms were obtained from Department of Microbiology , Saveetha Dental College & Hospitals, Chennai .

Methodology

The plant extract 200mg were weighed aseptically into a sterile tube and dissolved in 2ml of sterile Tryptic soy Broth (TSB).From the stock solution various concentrations were prepared,viz.,62µg,125 µg,250 µg,500 µg/100µl ,1mg,5mg,10mg/100µl respectively in to wells of micro plates.100µl of these concentration were taken and the plates were incubated at 37°C for 24hrs.

Screening of Antibactericial Activity

The tested organisms was grown in (TSB) Tryptic soy broth medium [Hi media ,Mumbai] for 24hrs at 37°C and concentration was adjusted to 0.5 Macfarland standard.[18-20]The above concentration of extracts were taken in 100µl quantities in a U bottom micro culture plates. 100µl of the bacterial suspension was added to each well.control well received plain broth without plant extract. The plates were kept in sealed covers and incubated at 37°C overnight and growth/no growth was detected. All the tests were done in duplicate to minimize the test error.

Minimum Inhibitory Concentration (MIC)

Minimum inhibitory concentration of herbal extracts against tested microorganism was determined by broth culture method .[21]A series of two- fold dilution of each extract
(62 µg/100µl to 10mg/100µl) was made in to which 100µl of the standardized bacterial suspension containing 10^6 organisms was made in Tryptic soy broth as specified by National Committee for Clinical Laboratory Standards (NCCLS, 1990)[22]The control well received plain broth without herbal extract .The plates were incubated at 37°C for 24 hours and observed for visible growth. As the extracts were colored, MIC could not be read directly by visual methods. Hence subcultures from all the wells were made and growth/no growth is detected.then the MBC were obtained.

Minimum Bactericidal Concentration (MBC)

The MBCs were determined by selecting wells that showed no growth. The least concentration, at which no growth was observed, were noted as the MBC.

Result and Discussion

Dental plaque, the biofilm that forms on the surface of teeth, can induce some of the most common diseases which include caries, gingivitis, and periodontitis. Acid-producing bacteria like streptococcus mutans, Lactobacillus acidophilus, streptococcus salivarius, Streptococcus mitis that surround orthodontic appliances are a common orthodontic problem in many patients undergoing Orthodontic treatment .[23,24] Such bacteria can lead to tooth enamel breakdown and potential discoloration of the tooth surface, and these aesthetic changes can persist for many years after orthodontic treatment. While the newer bonded orthodontic brackets have many advantages over the old metal bands that were fitted around each tooth, they do impede good oral hygiene, resulting in plaque accumulation and increased tooth enamel breakdown.

Prashanth et al conducted a study to evaluate the antimicrobial effects of the chewing sticks of Neem and mango against the micro organisms like Streptococcus mutans ,Streptococcus salivarius , Streptococcus mitis , Streptococcus sanguis which are involved in the development of dental caries. His data concluded that, Mango extract showed maximum zone of inhibition on Streptococcus mitis. Neem extract produced the maximum zone of inhibition on Streptococcus mutans. Even at low concentration neem extract showed some inhibition of growth for all the four species of organisms. Hence combination of neem and mango chewing sticks may provide the maximum benefit to mankind to prevent dental caries.[25]

Venka A et al conducted a study related to the antibacterial effect of Neem mouthwash against salivary levels of streptococcus mutans and Lactobacillus Acidophilus has been tested over a period of two months. Also its effect in reversing incipient carious lesions was assessed. He found that streptococcus mutans was inhibited by Neem mouthwashes, with or without alcohol as well as chlorhexidine, lactobacillus growth was inhibited by chlorhexidine alone. [26,27]

Beukes an orthodontist conducted an invitro study on the antimicrobial activity of phytomedicin against acidogenic oral bacteria on eight medicinal plants like Hydrastis Canadensis, CyclopiaIntermedia, Hypericum perforatum, Ginkgoaceae, Passiflora incarnata, Achillea Millefolium,Arciosiaphylos uva-ursi and Artemisia absinthium against S. mutans, S. sobrinus, L. casei and A.naeslundii involved in dental demineralisation., acetone, ethanolic, hexane form of extract wereUsed.The control treatments were chlorhexidine and fluoride.[28,29] His findings concludes that the acetone extracts of that includes Ginkgoaceae, Achillea Millefolium, Passiflora incarnata, Arciosiaphylos uva-ursi and Hypericum

perforatum showed significant inhibition zones against S.mutans, L.casei and S.sobrinus when compared to ethanolic and hexane extract.[30,31]

Hence our finding suggests the presence of No growth is an indication of high effectiveness of the extract whereas presence of Growth indicates the less effectiveness of the extract, which was represented in Table 1 .

Azadirachta indica leaf extract shows No growth (MBC) at a concentration of 62µg/ml against streptococcus mutans and streptococcus mitis. Uvaursi extract showed No growth at a concentration of 1mg/ml.whereas in table 2 Azadirachta indica shows No growth (MBC) at a concentration of 5mg/ml against streptococcus salivarius.Uvaursi leaf extract showed No growth at a concentration of 1mg/ml in conclusion,Azadirachtaindica leaf extract is highly effective against streptococcus mutans and streptococcus mitis when compared to Uva ursi leaf extract. On other hand Uva ursi leaf extract is found to efficient against streptococcus salivarius than Azadirachta indica leaf extract.

Table 1: Antibacterial Activity of Phytomedicine against Oral Microbes

Oral microbes	Herbal extract	62µg/ml	125 µg/ml	250 µg/ml	500 µg/ml	1mg/ml	5mg/ml	10mg/ml	Control	MIC/ MBC
S.mutans ATCC-25175	A.indica	--	--	--	--	--	--	--	++	62 µg/ml
	A.Uvaursi	++	++	++	++	--	--	--	++	1mg/ml

S.Salivarius ATCC-25975	**A.indica**	++	++	++	++	++	--	--	++	**5mg/ml**
	A.Uvaursi	++	++	++	++	--	--	--	++	**1mg/ml**
S.mitis ATCC-9811	**A.indica**	--	--	--	--	--	--	--	++	**62 µg/ml**
	A.Uvaursi	++	++	++	++	--	--	--	++	**1mg/ml**

++ = Growth -- = No Growth (Indicates the MIC/MBC)

Conclusion

Various literature reveals the antibacterial activity of Neem and Uva-ursi against oral bacteria. no work is done on the Neem leaf and Uvaursi acetone extract against acidogenic oral bacteria.hence our data proves the antibacterial efficacy of the acetone leaf extract of **Azadirachta indica**,and **Uva ursi** is an indicator of its broad spectrum antibacterial potential which may be helpful in eradicating streptococcus mutans and streptococcus mitis and provides an aid to control dental plaque . However, further studies are necessary to isolate and reveal the active compound(s) contained in the refined extract of Azadirachta indica and Uvaursiand to establish the mechanism of action.

Corresponding Author

Aravind Sivakumar,

Senior Lecturer, Department of Orthodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162,Poonamallee High Road,Chennai-600077, Tamil Nadu,India

Phone Number:+91 8220552400

Email Id:arvind.sivakumar@gmail.com

Reference

1.Gupta RK, Gupta D, Bhaskar DJ, Yadav A, Obaid K, Mishra S. Preliminary Antiplaque Efficacy of Aloe Vera Mouthwash on 4 Day Plaque Re-Growth Model: Randomized Control Trial. *Ethiopian Journal of Health Sciences*. 2014;24(2):139-144.

2.Hambire CU, Jawade R, Patil A, Wani VR, Kulkarni AA, Nehete PB.Comparing the antiplaque efficacy of 0.5% Camellia sinensis extract, 0.05% sodium fluoride, and 0.2% chlorhexidine gluconate mouthwash in children.*Journal of International Society of Preventive & Community Dentistry*.2015;5(3):218-226.

3.S. P. Lokegaonkar and b m. Nabarin vitro assessment of the antiplaque properties of crude m. Alba leaf extract journal of herbal medicine and toxicology 5 (1) 71-77 (2011).

4. Wings tyloo1 ,Evaluation of ellagic acid on the activities of oral bacteria with the use of adenosine triphosphate (atp) bioluminescence assay*african journal of biotechnology* vol. 9(25), pp. 3938-3943, 21 june, 2010.

5.Allen, P. F 2003. Assessment of oral health related quality of life, *Health Quality of Life Outcomes* 1: 40.

6.Chang HS, Walsh LJ, Freer Tj The effect of orthodontic treatment on salivary flow, pH, buffer capacity, and levels of mutans streptococci and lactobacilli. *Australian Orthodontic Journal 1999*;15:229-234.

7. Malgorzata Klukowska, Annike Bader, Christina Erbe, Philip Bellamy, Donald J. White, Mary Kay Anastasia, Heiner WehrbeinPlaque levels of patients with fixed orthodontic appliances measured by digital plaque image .*American journal of orthodontics and Dentofacial orthopedics*.2011 ,139 (5) e-463-470.

8.Anhoury P ,Microbial profile on metallic and ceramic bracket materials. Angle Orthodontist 2002;72:338-343.

9.Bos R, van der Mei HC, Busscher HJ Physico-chemistry of initial microbial adhesive interactions—its mechanisms and methods for study. FEMS Microbiology Reviews 1999;23:179-230.

10.Diamanti-Kipioti A, Gusberti FA, Lang NP Clinical and microbiological effects of fixed orthodontic appliances. *Journal of Clinical Periodontology* 1987;14:326-333.

11.Atack NE, Sandy jr ,Addy M Periodontal and microbiological changes associated with the placement of orthodontic appliances. A review.*Journal of Periodontology* 1996;67:78-85.

12. Chopra, R. N., Nayer, S. L. and Chopra, I. C., Glossary of Indian Medicinal Plants, CSIR, New Delhi, 1956.

13. Chopra, R. N., Chopra, I. C, Handa, K. L. and Kapur, L. D. (eds),Indigenous Drugs of India, U.N. Dhur and Sons, Kolkata, 1958, pp. 51–595.

14. Kirtikar, K. R. and Basu, B. D., in Medicinal Plants (eds Blatter, E., Cains, J. F., Mhaskar, K. S., Vivek Vihar, New Delhi, 1975,p. 536.

15. Thakur, R. S., Singh, S. B. and Goswami, A.,Curr. Res. Med. Aromat. Plants, 1981, 3, 135–140.

16. Lakshmi T, Krishnan V, Rajendran R, Madhusudhanan N.Azadirachta indica: A herbal panacea in dentistry – An update. *Pharmacognosy Reviews.* 2015;9.

17. Chatterjee, A. and Pakrashi, S. (eds), The Treatise on Indian Medicinal Plants, 1994, vol. 3, p. 76.

18. Beaux D, Fleurentin J, Mortier F. Effect of extracts of Orthosiphon stamineus Berth, Hieracium pilosella L., Sambucus nigra L. and Arctostaphylos uva-ursi (L.) Spreng.in rats.Phytother Res. 1999;13(3):222-225.

19. Blumenthal M, Goldberg A, Brinckmann J. Herbal Medicine: Expanded Commission E Monographs. Boston: *Integrative Medicine Communications*; 2000:389-393.

20.J.H Jorgenson & John turniegd Susceptibility test methods dilution and disc diffusion methods,Manual of Clinical microbiology vol 1 ,9th edition pg no.1153-1172.ASM Press Washington.

21.Betty A.Forbes., Daniel F.Sahm., Alice S.Weissfeld. Bailey & Scott's Diagnostic Microbiology 11th edition Mosby page no 229 – 257.

22. Ananthanarayan R and Paniker's: Textbook of Microbiology: 8th edition: *Publishers University Press: Hyderabad* 2009: 618.

23. Jennifer MA: Determination of Minimum Inhibitory Concentrations: *Journal of Antimicrobial Chemotherapy* 2001; 48, (SI): 5 -16.

24. National Committee for Clinical Laboratory Standards: Methods for Disc Susceptibility Tests for Bacteria that Grow Aerobically: NCCLS Document M2-A7: *National Committee for Clinical Laboratory Standards*: Wayne, USA: 2000; 7.

25.Demling Analysis of supra- and subgingival long-term biofilm formation on orthodontic bands *Eur J Orthod* (2009) 31 (2): 202-206.

26.Boyd RL, . Periodontal implications of orthodontic treatment in adults with reduced or normal periodontal tissues versus those of adolescents.*American Journal of Orthodontics and Dentofacial Orthopedics* 1989;96:191-198.

27.Prashanth GM et alThe effect of mango and neem extract on four organisms causing dental caries: Streptococcus mutans, Streptococcus salivavius, Streptococcus mitis, and Streptococcus sanguis: an in vitro study. *Indian J Dent Res.* 2007 Oct-Dec;18(4):148-51.

28.Vanka A.The effect of indigenous Neem Azadirachta indica [correction of (Adirachta indica)] mouth wash on Streptococcus mutans and lactobacilli growth.*Indian J Dent Res.* 2001 Jul-Sep;12(3):133-44.

29.Somu CA, Ravindra S, Ajith S, Ahamed MG. Efficacy of a herbal extract gel in the treatment of gingivitis: A clinical study. *Journal of Ayurveda and Integrative Medicine.*2012;3(2):85-90.

30. Lakshmi.T ,Aravind kumar.S ,Arun A.V Invitro Antibacterial Screening of Arctostaphylos Uva Ursi Leaf Extract on Selected Oral Plaque Forming Microorganisms .2012 ;4(5) 63-68.

31. Lakshmi.T ,Aravindkumar.SPreliminary phytochemical analysis & Invitro Antibacterial activity of Acacia catechu willd Bark against Streptococcus mitis, Streptococcus sanguis & Lactobacillus acidophilus. International Journal of Phytomedicine 3 (2011) 579-584

32. .Prabuseenivasan S, Jayakumar M, Ignacimuthu S. In vitro antibacterial activity of some plant essential oils.*BMC complementary and alternative medicine.*2006 Dec;6(1):39.

Is Periodontitis an Indicator for Diabetes Mellitus

N.S.Harini[1], Vivek Narayan[2], Sankari M[3]

Graduate Student[1], Reader[2], Department of Oral Medicine, Professor[3], Department of Periodontics.

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: Diabetes and periodontitis are multifactorial chronic inflammatory diseases affecting a very large population. Periodontitis can be used as a predictor for diabetes and pre-diabetes risk assessment.The manifestations of diabetes mellitus are widespread among which periodontitis is the most common oral manifestation and the two diseases appear to be interrelated. There is increased inflammation in diabetes which leads to increased prevalence of periodontitis in diabetes patients.The present study is aimed at finding out presence of diabetes mellitus in patients with periodontits, so that periodontitis could be used as an indicator for diabetes mellitus.

Materials and Methods: This study has a sample size of 50 and the patients were taken from Saveetha Dental College and Hospitals. This study involves among periodontally compromised patients to determine the presence of diabetes mellitus among them by using glucometer to assess their random blood sugar level (RBS LEVEL). Once the RBS was calculated, the patients' periodontal status was evaluated. Finally the periodontitis and diabetic level of the patients were correlated. The results were then statistically analysed and tabulated using bar graphs and the correlation was estimated.

Results: According to the survey, among 50 periodontally compromised patients, 22 patients were males, and 28 patients were females. Among these 50 periodontally compromised patients, under 20-30 years of age category 5 were males and 4 were females. 31-40 years of age group contained 4 males and 7 females. Under the age group of 41-50 years of age 4 were males and 3 were females. The age group of more than 50 years contained 10 males and 13 females.

Conclusion: Thus, the survey has shown positive results where periodontitis is an indicator for diabetes mellitus. The age distribution favourable for the condition is more than 50 years of age and the gender ratio is that females are more prone than males. For the diabetic status, awareness must be created among people to check their blood glucose level periodically.

Key Words*:Diabetes mellitus, Periodontitis, glucose level, periodontium, random blood sugar level.*

Introduction

Diabetes and periodontitis are multifactorial chronic inflammatory diseases affecting a very large population. Studies have shown that there is a significant relationship between the two diseases, as diabetes is associated with increased prevalence, severity, and progression of periodontitis. Periodontitis can be used as a predictor for diabetes and pre-diabetes risk assessment[1] Diabetes Mellitus is a metabolic disorder affecting carbohydrate, lipid, and protein metabolism. The manifestations of diabetes mellitus are widespread among which periodontitis is the most common oral manifestation and the two diseases appear to be interrelated. There is increased inflammation in diabetes which leads to increased prevalence of periodontitis in diabetes patients.[2].

Diabetes mellitus is a systemic disease with several major complications affecting both the quality and length of life. One of these complications is periodontal disease (periodontitis). Periodontitis is much more than a localized oral infection. Recent data indicate that periodontitis may cause changes in systemic physiology. The interrelationships between periodontitis and diabetes provide an example of systemic disease predisposing to oral infection, and once that infection is established, the oral infection exacerbates systemic disease[3]. In this case, it may also be possible for the oral infection to predispose to systemic disease.

In order to understand the cellular/molecular mechanisms responsible for such a cyclical association, one must identify common physiological changes associated with diabetes and periodontitis that produce a synergy when the conditions coexist. A potential mechanistic link involves the broad axis of inflammation, specifically immune cell phenotype, serum lipid levels, and tissue homeostasis. Diabetes-induced changes in immune cell function produce an inflammatory immune cell phenotype (upregulation of proinflammatory cytokines from monocytes/polymorphonuclear leukocytes and downregulation of growth factors from macrophages). This predisposes to chronic inflammation, progressive tissue breakdown, and diminished tissue repair capacity[4,5]

Periodontal tissues frequently manifest these changes because they are constantly wounded by substances emanating from bacterial biofilms. Diabetic patients are prone to elevated low density lipoprotein cholesterol and triglycerides (LDL/TRG) even when blood glucose levels

are well controlled. This is significant, as recent studies demonstrate that hyperlipidemia may be one of the factors associated with diabetes-induced immune cell alterations. Recent human studies have established a relationship between high serum lipid levels and periodontitis. Some evidence now suggests that periodontitis itself may lead to elevated LDL/TRG[6-8]

Periodontitis-induced bacteremia/endotoxemia has been shown to cause elevations of serum proinflammatory cytokines such as interleukin-1 beta (IL-1 beta) and tumor necrosis factor-alpha (TNF-alpha), which have been demonstrated to produce alterations in lipid metabolism leading to hyperlipidemia. Within this context, periodontitis may contribute to elevated proinflammatory cytokines/serum lipids and potentially to systemic disease arising from chronic hyperlipidemia and/or increased inflammatory mediators[10,11]

These cytokines can produce an insulin resistance syndrome similar to that observed in diabetes and initiate destruction of pancreatic beta cells leading to development of diabetes. Thus, there is potential for periodontitis to exacerbate diabetes-induced hyperlipidemia, immune cell alterations, and diminished tissue repair capacity. It may also be possible for chronic periodontitis to induce diabetes[12-15]

The present study is aimed at finding out presence of diabetes mellitus in patients with periodontits, so that periodontitis could be used as an indicator for diabetes mellitus.

Materials and Methods

This study has a sample size of 50 and the patients were taken from Saveetha Dental College and Hospitals. This study involves among periodontally compromised patients to determine the presence of diabetes mellitus among them by using glucometer to assess their random blood sugar level (RBS LEVEL).The glucometer is used by pricking the patient’s ring finger and utilising a drop of their blood in the test strip and the value is obtained immediately which helps to determine the RBS level. Once the RBS was calculated, the patients’ periodontal status was evaluated by estimating probing depth, bleeding, attachment loss, furcation and mobility. Finally the periodontitis and diabetic level of the patients were correlated. The results were then statistically analysed and tabulated using bar graphs and the correlation was estimated.

Results

Table 1: Total Sample Size

SEX	NUMBER
MALE	22
FEMALE	28
TOTAL	50

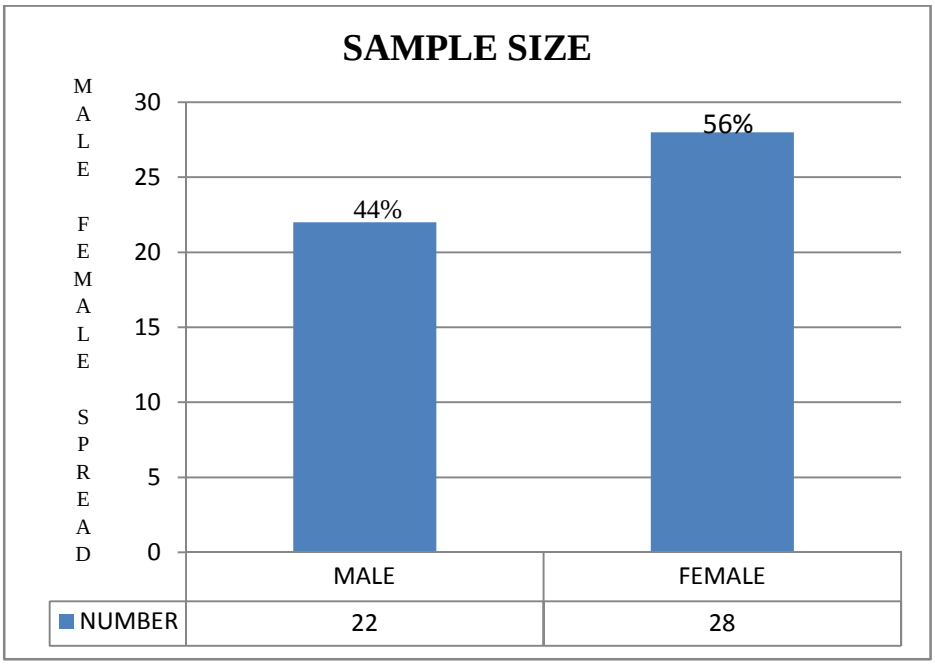


Figure 1: Bar Graph Showing the Total Sample Size of the Study

Table 2: Age Distribution

AGE	MALE	FEMALE
20-30	5	4
31-40	4	7
41-50	4	3
>50	10	13
TOTAL	23	27

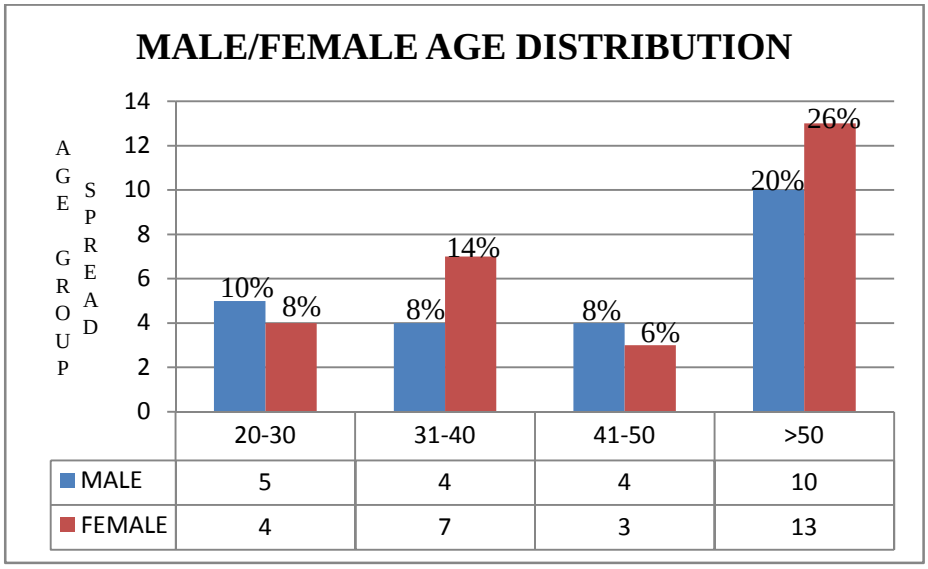


Figure 2: Bar Graph Showing Age Group Distribution among the Periodontitis Group

Table 3: Diabetic Status

SEX	KNOWN DIABETIC	UNKNOWN DIABETIC	TOTAL
MALE	**9**	**13**	**22**
FEMALE	**14**	**14**	**28**

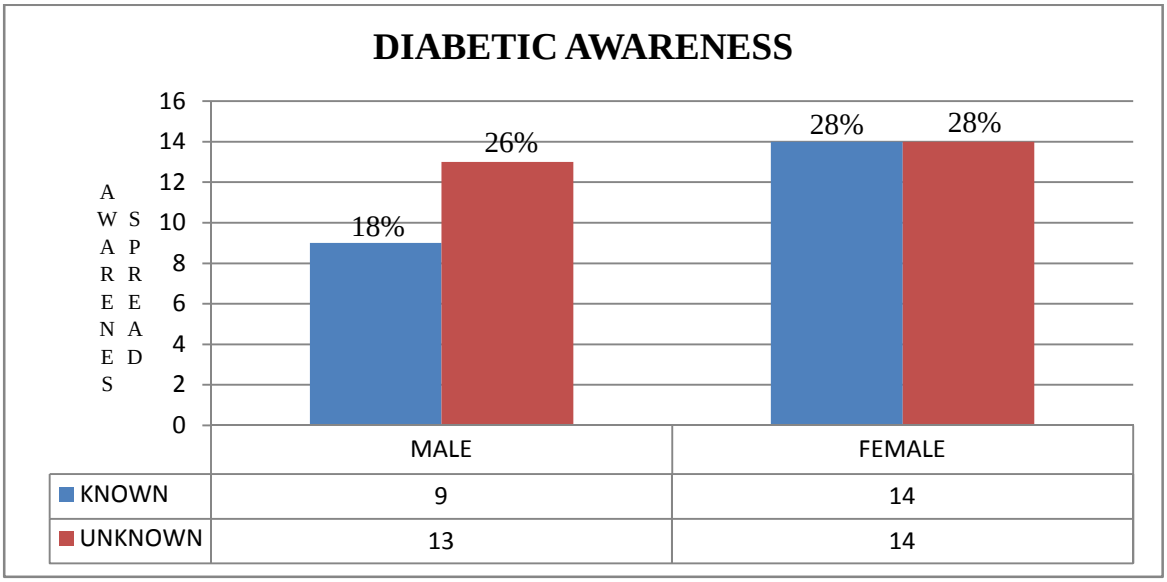


Figure 3: Bar Graph Showing the Diabetic Status among the Periodontitis Group

Discussion

Periodontal disease is the most prevalent disease of the oral cavity. The role of aging in periodontal disease is debatable, but the means of preventing periodontal disease are available[16-18].Periodontitis is a chronic inflammatory disease that results in the loss of connective tissue and alveolar bone support of the teeth [36]. Periodontitis is a chronic bacterial infection characterized by persistent inflammation, connective tissue breakdown and alveolar bone destruction mediated by pro-inflammatory mediators [37].Inflammation and tissue breakdown are led by an array of inflammatory destructive mediators associated with initiation and progression of inflammatory diseases like periodontitis [38].

Diabetic patients are more prone to periodontal diseases as they are more susceptible to infections. Studies have proved that periodontitis can be considered as a complication of diabetes[39].An increased concentration of MMP-8 and -9 in the gingival tissue of diabetic CP patients suggests that the expression of these MMPs contributes to the failure of the healing process in the diabetic condition [40]. In the present study, the survey has shown that females above the age of 50 are more prone for periodontal problems.

Sexual dimorphisms exist in the prevalence and severity of many human conditions and diseases. Risk assessment is a crucial component of personalized medicine in evidence-based clinical practice. Gender is known to be a modifier of the initiation & outcome of many conditions. [19]. Though studies show thatmales are prone to periodontitis our study reveals that females with periodontitis are more for diabetes mellitus.

The relationship between diabetes mellitus and periodontal disease is not clear, even though studied intensively. Gingival and periodontal diseases, in their various forms, have afflicted mankind since the dawn of history. Diseases of the periodontium occur in childhood, adolescence, and early adulthood but the prevalence of periodontal disease, tissue destruction and tooth loss increases with age[20].

Apart from age, the factors such as immediate environment of the periodontium and systemic factors, resulting from the general condition of the patient are responsible for the periodontal disease.[4] Diabetes mellitus deserves a special consideration in any comprehensive text of periodontal diseases. Diabetes mellitus affects many people, as does periodontitis, and is found with increasing frequency as people get older as is periodontitis.[21,22]

The prevalence of diabetes mellitus is more than twice as high in patients with periodontitis compared to healthy subjects. Periodontal disease may contribute to systemic inflammation, worsening insulin resistance and diabetes due to the generation of inflammatory cytokines [23-25]

A large body of information exists, showing that prevention, early diagnosis and consequent treatment of periodontal disease may have a major impact on the control of diabetes. Severe periodontal disease causes attachment loss, alveolar bone loss and tooth loss, extending beyond the local level to produce systemic effects. It exacerbates the inflammatory milieu, increases insulin resistance, and potentially worsens cardiovascular disease. Severe periodontitis is a risk factor for early death due to ischemic heart disease or renal dysfunction independent of diabetes, increasing the risk 3.2 fold as compared with those with none, mild, or moderate periodontitis [10].

Periodontitis and its Effects on Diabetes

Periodontal diseases are bacteria-induced infections affecting the periodontium and resulting in the loss of tooth attachment. Porphyromonas gingivalis plays a central role in periodontal disease. P. gingivalis can invade endothelial cells and is a potent signal for monocyte and macrophage activation. Thus, once established in the tissue, this chronic infection complicates diabetes control and increases the occurrence and severity of microvascular and macrovascular complications. The presence of the periodontal pathogen bacteria thus increases the risk of stroke, ischemic heart disease and atherosclerosis [26].

Other relevant periodontal pathogen bacteria are: actinobacillusactinomycetemcomitans, treponemadenticola, fusobacterium nucleatum, prevotellaintermedia, eikenellacorrodens und camphylobacter rectus. These periodontal pathogen bacteria cause a chronic local lowgrade inflammation and contribute to systemic inflammation. This is reflected by higher circulating levels of inflammatory markers such as C-reactive protein, IL-6 and TNFα, the latter being responsible for worsening insulin resistance and diabetes [8]. Exacerbating immune reactions lead to self-destruction of periodontal tissue.

The increasing pocket depth and root surface infection, bacterial impact and invasion of periodontal tissue, alveolar bone loss and local and systemic immune reactions all have negative effects on diabetes. The association between diabetes and periodontal disease may be due to numerous physiological phenomena seen in diabetes, such as impaired (immune)

resistance, vascular changes, altered microflora, and abnormal collagen metabolism. This tends to support the higher incidence and severity of periodontitis in diabetic patients. The association between gene sets (for example IL1-genotype) and periodontitis has been discussed with conflicting conclusions [4,7].

There is a direct causal or modifying relationship in which poor glycemic control results in more severe periodontitis [4]. The worsening effects of diabetes on periodontal inflammation are well known. Studies also indicate a correlation between periodontal inflammation on diabetic balance and insulin resistance syndrome [3,6,8].This evidence points to a vicious cycle in which diabetes and periodontitis exacerbate one another. Periodontal treatment may improve diabetes control measured as a reduction in glycated hemoglobin [9]. Because of this relationship, the diabetes specialist should put an emphasis on oral health and its control as an integral part of diabetes treatment.

Diagnosis of periodontal diseease Diabetes is a disease that frequently causes multiple comorbidities. Periodontal disease and other oral pathologies (gingivitis, candidiasis, oral malignancies etc.) are frequent complications of diabetes. The diagnosis and control of periodontal disease with its adverse effects on insulin resistance through the generation of inflammatory cytokines can have a major impact on diabetes control and diagnosis.[31-34]

1. Basic diagnostic indices for periodontitis are: the evaluation of gingival bleeding and swelling, recessions, attachment loss, pathological pockets (depth >4mm), loss of tooth insertion, pocket exsudation, foetor, bleeding on probing with a special ball-point probe and alveolar bone loss assessed from radiographs.[27]

2. A further diagnostic step is the determination of inflammation markers such as sCRP, TNFα, IL-6, IL-10.

3. For diabetic patients, the identification of subgingival bacteria, especially p. gingivalis, p. intermedia, actinobacillusactinomycetemscomitans and t. forsythensis, represents another important diagnostic tool [35]. Consequent follow-up examinations should include a bacterial test to monitor subgingival bacterial levels which are best measured using a multiplex polymerase chain reaction. Very useful and simple tools are informational flyers to help increase patient awareness of periodontal disease, its association with diabetes and its prevention and treatment[28]

Pathologic Mechanisms

Periodontal disease is a chronic gram-negative infection thought to increase insulin resistance. Through this mechanism, it contributes as known, to the development of metabolic imbalance [41]. As multiple studies, recognize, the pathogenesis of periodontal disease is complex. It shows a combination of 2 processes: the initiation and maintenance of a chronic inflammatory process, developed with the help of a diverse microbial flora with its diversity of bacterial products. The host response to this infectious mechanism mediates a "cascade" of tissue destructive pathways [42]. Some time ago, researchers linked the role of microorganisms as being the main etiologic factor in PD. Nowadays this is not sufficient. The major component of periodontal tissue destruction is believed to be the result of the host immune-inflammatory response to bacterial challenge. Plaque biofilm and the associated host response are involved in PD pathogenesis [43].

Current Concepts and Future Considerations

Society evolution is commonly associated with the increased spread of diseases such as diabetes mellitus (DM) and periodontal disease (PD). DM and PD are known to be among the most prevalent human disorders and they are frequently and concurrently present in many people[30]

World Health Organization states in 2000, that there are nearly 177 million diabetic people worldwide and approximately 1 million in Romania. The same organization estimates an increase in the diabetic population of over 370 million worldwide and of 1.8 million in Romania, until 2030. It has been known for a long period of time, that periodontitis is a common chronic gram-negative anaerobic infectious disease. Other authors consider periodontitis as a chronic inflammatory disease characterized by connective tissue attachment loss and alveolar bone resorption which leads to tooth loss, by periodontal pocket formation[32].

Prevention of Periodontitis

The following behavioural recommendations should be included in regular instructions for diabetic patients:

1) No smoking

2) Strictglycemic control

3) Professional counseling on the importance of oral health (self-response, self-questionnaire)

4) Qualified instruction in home oral care and the importance of soft tooth brushes

5) Thorough oral cleaning at least twice a day – after breakfast and before bedtime – for more than 3 minutes

6) Daily interdental cleaning (floss, tooth picks, interdental-brushes)

7) Tongue-brushing

8) Proper tooth brush handling - daily disinfection of the tooth brush, drying, alternate use of two brushes, no natural bristles.

9) Partner treatment - if only one suffers from periodontitis special care should be taken to avoid transfer of bacteria through shared dishes, cups, forks; no kissing allowed during acute phases

10) Antibacterial and anti-inflammatory ingredients in toothpaste and mouth rinses, for example o chlorhexidine (anti-inflammatory), triclosan and thymol (anti-inflammatory)[29]

Periodontal Treatment of Diabetic Patients

1. Measurement of pocket depth using a ball-point probe

2. Extraction and identification of subgingival bacterial samples (paper points and sterile tubes, available from distributors or laboratories)

3. Germ reduction through the use of antibacterial mouthwash, pocket solutions, subgingival application of metronidazol gel, chlorhexidine gel (applied with mouth guards several times a week). The elimination of periodontal infection with these antibacterial adjuncts results in a reduction of the periodontal pathogen bacteria to low or even nondetectable levels.

4. A follow-up extraction and identification of subgingival bacteria samples (same extraction sites)

5. Scaling and/or root planing accompanied by antibiotic treatment

6. Maintenance of intensive oral care and additional use of mouthwash, controlled with regular PCR analysis

7. Recall[30]

Conclusion

Thus, the survey has shown positive results where periodontitis is an indicator for diabetes mellitus. The age distribution favourable for the condition is more than 50 years of age and the gender ratio is that females are more prone than males. For the diabetic status, awareness must be created among people to check their blood glucose level periodically.

The potential mechanistic interrelationships Periodontal treatment to control colonization of microbial pathogens and simultaneously reduce the level of pro inflammatory cytokines is also essential to prevent a worsening of diabetic conditions. Secondly, while proinflammatory cytokines, such as TNF-a and IL-1b, play key roles in this bidirectional relationship, it is also of interest to develop immunomodulators that target these cytokines. However, one must be cautious, because modulating these cytokines may also affect the body's homeostasis. In summary, an in-depth understanding of the possible mechanisms linking periodontal disease and diabetes, in terms of periodontal destruction and periodontal healing, is essential to the future development of treatment strategies for patients with diabetes and periodontal disease.

Corresponding Author

M.Sankari,

Professor, Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Phone Number:9840285905

Email ID:msankari@gmail.com

References

1. Almas K, Lazzam S, Quadairi A;*The effect of oral hygiene instruction on diabetic type 2 male patients with periodontal disease;* J Contemp Dent Pract 2003; 15:24-35.
2. Cueto F, Rotundo R, Frazzingaro G, Muzzi L, Pini Prato GP; *Diabetes mellitus as a risk factor for periodontitis;* Minerva Stomatol 2001; 50: 321 – 330.
3. Grossi SG, Genco RJ;*Periodontal disease and diabetes mellitus: a two way relationship;* Ann Periodontol 1998; 3: 51- 61
4. Guzman S, Karima M, Wang HY, Van Dyke TE;*Association between interleukin-1 genotype and periodontal disease in a diabetic population;*J Periodontol 2003; 74: 1183-1190.
5. Iacopino AM; *Periodontitis and diabetes interrelationships: role of inflammation;* Ann Periodontotl 2001; 61: 125-137.
6. Loos BG, Craandijk J, Hoek FJ, Wertheim-van Dillem PM, van der Velden U;*Elevation of systemic markers related to cardiovascular diseases in the peripheral blood of periodontitis patients;* J Periodontol 2000; 71: 1528-1534 .
7. Nishimura F, Kono T, Fujimoto C, Iwamoto Y, Muramaya Y;*Negative effects of chronic inflammatory periodontal disease on diabetes mellitus;* J Int Acad Periodontol 2000; 2: 49-55.
8. Pucher J, Stewart J;*Periodontal disease and diabetes mellitus;*Curr Diab Rep 2004; 4: 46-50.
9. Saremi A, Nelson RG, Tulloch-Reid M, Hanson RL, Sievers ML, Taylor GW, Shlossman M, Bennett PH, Genco R, Knowler WC;*Periodontal disease and mortality in type 2 diabetes.* Diabetes Care 2005; 28: 27-32.
10. Mealey BL, Oates TW; *Diabetes mellitus and periodontal diseases;*J Periodontol 2006;77:1289.
11. Brem H, Tomic-Canic M;*Cellular and molecular basis of wound healing in diabetes;*J Clin Invest 2007;117:1219.

12. Cianciola LJ, Park BH, Bruck E, Mosovich L, Genco RJ;*Prevalence of periodontal disease in insulin-dependent diabetes mellitus (juvenile diabetes);* J Am Dent Assoc 1982;104: 653.
13. Nguyen NT, Nguyen XM, Lane J, Wang P; *Relationship between obesity and diabetes in a US adult population: findings from the National Health and Nutrition Examination Survey,* 1999e2006. Obes Surg 2011;21:351.
14. Christgau M, Palitzsch KD, Schmalz G, Kreiner U, Frenzel S; *Healing response to non-surgical periodontal therapy in patients with diabetes mellitus: clinical, microbiological, and immunologic results;* J Clin Periodontol 1998;25:112.
15. Czako L, Hegyi P, Rakonczay Jr Z, Wittmann T, Otsuki M;*Interactions between the endocrine and exocrine pancreas and their clinical relevance;*Pancreatology 2009;9:351.
16. Bano KA, Batool A; *Metabolic syndrome, cardiovascular disease and type-2 diabetes;* J Pak Med Assoc 2007;57:511.
17. Saruc M, Pour PM;*Diabetes and its relationship to pancreatic carcinoma*; Pancreas 2003;26:381.
18. Stauffer JA, Nguyen JH, Heckman MG, et al;*Patient outcomes after total pancreatectomy: a single centre contemporary experience.* HPB (Oxford) 2009;11:483.
19. Wofford MR, King DS, Harrell TK;*Drug-induced metabolic syndrome;*J Clin Hypertens (Greenwich) 2006;8:114.
20. Bascones-Martinez A, Matesanz-Perez P, EscribanoBermejo M, Gonzalez-Moles MA, Bascones-Ilundain J, Meurman JH; *Periodontal disease and diabetes e review of the literature;*Med Oral Patol Oral Cir Bucal 2011;16:e722.
21. Armitage GC; *Development of a classification system for periodontal diseases and conditions;* Ann Periodontol 1999;4: 1
22. Taylor GW;*Bidirectional interrelationships between diabetes and periodontal diseases: an epidemiologic perspective;* Ann Periodontol 2001;6:99.
23. Nelson RG,Shlossman M, Budding LM, et al;*Periodontal disease and NIDDM in Pima Indians;*Diabetes Care 1990;13: 836.
24. Soskolne WA, *Klinger A. The relationship between periodontal diseases and diabetes: an overview;* Ann Periodontol 2001;6: 91.

25. Saito T, Shimazaki Y, Kiyohara Y, et al; *The severity of periodontal disease is associated with the development of glucose intolerance in non-diabetics: the Hisayama study;*J Dent Res 2004;83:485.
26. Shimazaki Y, Saito T, Yonemoto K, Kiyohara Y, Iida M, Yamashita Y;*Relationship of metabolic syndrome to periodontal disease in Japanese women: the Hisayama Study;* J Dent Res 2007;86:271.
27. Mealey BL;*Diabetes and periodontal disease: two sides of a coin;* Compend Contin Educ Dent 2000;21:943.
28. Soskolne WA;*Epidemiological and clinical aspects of periodontal diseases in diabetics;*Ann Periodontol 1998;3:3.
29. Grossi SG, Genco RJ;*Periodontal disease and diabetes mellitus: a two-way relationship;*Ann Periodontol 1998;3:51.
30. Loe H;*Periodontal disease. The sixth complication of diabetes mellitus;*Diabetes Care 1993;16:329.
31. Acharya AB, Satyanarayan A, Thakur SL;*Status of association studies linking diabetes mellitus and periodontal disease in India;* Int J Diabetes Dev Ctries 2010;30:69
32. Papapanou PN;*Periodontal diseases: epidemiology;*Ann Periodontol 1996;1:1.
33. Arrieta-Blanco JJ, Bartolome-Villar B, Jimenez-Martinez E, Saavedra-Vallejo P, Arrieta-Blanco FJ;*Dental problems in patients with diabetes mellitus (II): gingival index and periodontal disease;* Med Oral 2003;8:233.
34. Campus G, Salem A, Uzzau S, Baldoni E, Tonolo G; *Diabetes and periodontal disease: a case-control study;* J Periodontol 2005;76:418.
35. Emrich LJ, Shlossman M, Genco RJ;*Periodontal disease in non-insulin-dependent diabetes mellitus;*J Periodontol 1991; 62:123.
36. Balaji Ramamoorthy, Sheeja S Varghese, Asha Ramesh; *Relationship between periodontal disease and abo blood group phenotypes-a cross sectional retrospective study;* 2015; Int J Pharm Pharm Sci, Vol 7, Issue 11, 386-388.
37. Sheeja S. Varghese, Hima Thomas, N. D. Jayakumar, M. Sankari, ReemaLakshmanan; *Estimation of salivary tumor necrosis factor-alpha in chronic and aggressive periodontitis patients;* Contemporary Clinical Dentistry; November 25, 2017, IP: 78.162.15.238.

38. ArchanaMootha, Sankari Malaiappan, N. D. Jayakumar, Sheeja S. Varghese, and Julie Toby Thomas; *The Effect of Periodontitis on Expression of Interleukin-21: A Systematic Review;* International Journal of Inflammation; Volume 2016, Article ID 3507503, 8 pages.
39. Shamitthaper, Tanvithaper, Vishnu priya v, Rajeev thaper, Reenathaper; *Prevalence of periodontitis in diabetic and non-diabetic patients;* Asian J Pharm Clin Res, Vol 9, Issue 1, 2016, 329-331.
40. Muthusamy,Senthil Kumar, Gelli Vamsi, Ramasamy Sripriya, and Praveen Kumar Sehga; *Expression of Matrix Metalloproteinases (MMP-8 and - in Chronic Periodontitis Patients With and Without Diabetes Mellitus;*Journal of Periodontology; November 2006, Vol. 77, No. 11, Pages 1803-1808.
41. Genco R., S.G. Grossi, A. Ho, F. Nishimura, J. Murayama; *A proposed model linking inflammation to obesity, diabetes and periodontal infections;* 2005; Periodontol 76: 2075- 84.
42. Page R; *The role of inflammatory mediators in the pathogenesis of periodontal disease;* J Perio Res;1991; 26: 230-42.
43. Card C., N. Mosquera-Lloreda, L. Salom, M.E. Gomez de Ferraris, A. Peydro; *Structural and functional salivary disorders in type 2 diabetic patients;* 2006; Med Oral Patol Oral Cir Bucal 11: E 309-14.

Isolation of Enterococcus Species in Post-Operative Local Sepsis in the Oral Cavity

Nivashini G.S.V[1], MuralidharanN.P[2], VinothKumar[3]

Graduate student [1],Professor[2], Department of Microbiology

Senior Lecturer[3], Department of Prosthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: Enterococci have emerged as an important cause of nosocomial infection. Successful antibiotic treatment against enterococcal infection usually depends on the synergistic bactericidal effect achieved by the combination of a cell wall-active agent, such as ampicillin or amoxicillin, and amoxyclav. However, the prevalence of enterococci resistant to one or more of these antibiotics is increasing day by day which has resulted in serious therapeutic difficulties. The isolation of enterococcus and sensitivity pattern of the drugs was discussed. The aim of this study is to assess the incidence of Enterococcus species in post-operative local sepsis in the oral cavity.

Materials and Method: In this study 75 patients who reported to the dental outpatient department over the period of 6 months.The samples were processed in the microbiology lab according to the standard protocol and organisms were isolated and identified. After identification of the organisms, the antibiotic profile for each organism isolated was found by performing AST(Antibiotic Susceptibility testing) by Kirbybauer's method.

Result: Totally 75 samples were collected and processed out of which 37 samples showed positive bacterial culture. From these positive cultures 9 different species are isolated and enterococcus found to be in 15 samples. Comparing the sensitivity pattern for the most commonly prescribed antibiotics against Enterococcus species, we state that they are highly sensitive to amoxyclav followed by ampicillin and amoxicillin.

Conclusion: These three drugs amoxyclav, ampicillin and amoxicillin are commonly used in dentistry. According to this study only less than 50 % of the enterococcus isolates are found to be susceptible. It is also reported that the enterococcus sp were isolated in nearly 40 percentage of the failed cases in oral lesions.

***Key Words**: Enterococcus, antibioticsamoxicillin, ampicillin and amoxyclav.*

Introduction

Oral cavity contains more than 700 species of microorganisms as commensals. Some of them are considered as opportunistic pathogens which colonises the traumatised tissue in the oral cavity, it includes aerobes, facultative anaerobes and obligate anaerobes. Eradicating such organisms from the septic pockets was normally done by treating them with the drugs that cover aerobic and anaerobic organisms. Success of such treatments depends on the choice of drug and susceptibility in the organism. Dentist and physicians have endeavoured for years to device some effective measure for preventing bacteraemia since it was demonstrated that organisms invade the blood stream with greatease than previously realised [1]. Very few organisms in the oral flora exhibits resistance to the conventionally used antimicrobial agents. Among the oral flora Enterococcus shows multiple drug resistance and persist even after antibiotic therapy. Enterococcus is normally ignored by most of the practitioners in clinical samples as it is normal flora. Antimicrobial resistance is a major concern in a clinical set-up. Newer pathogens are evolving day by day which is a major threat in treating hospitalised patients [2].Although enterococci are generally considered as transient constituents of the oral micro-biome with a low colonization density, surprisingly little is known about their oral prevalence and incidence particularly in different population groups. Most of the currently available data pertain to the oral carriage of E. faecalis mainly in patients undergoing endodontic therapy, and it is considered as one of 25 most abundant pathogens causing persistent endodontic infections. On the contrary, patients with periodontitis or gingivitis exhibit an enterococcal prevalence ranging from 3.7 to 35%. Interestingly, it is reported that 60% of diabetic patients yielded oral Enterococcus faecalis, and E. faecium, as opposed to only 6.6% in the controls [3].In the human oral cavity, E. faecalis has been frequently detected from patients with post-treatment apical periodontitis or refractory marginal periodontitis. Isolates from oral infections differ from group of hospital-derived isolates, as

they do not present many mobile genetic elements. However, they usually carry virulence factors related to adhesion and biofilm formation, which may account for the colonization of different oral sites.

Furthermore, oral strains may also carry few antibiotic resistance determinants that have the potential to be transferred to other pathogenic bacteria in biofilm communities [4].Enterococcal surface protein (ESP) was highly associated with infection-derived isolates of E. faecium and E. faecalis. Studies have shown that ESP gene was detected in most strains isolated from endodontic and periodontal. As ESP has been associated to higher biofilm production of the strains, the high prevalence of ESP within oral isolates suggests that this surface protein may be a potential virulence trait that participates in colonization of different niches of the oral cavity [5].Enterococci are common inhabitants of the human intestinal micro-flora and the genitourinary tract of men and women [6]. Enterococci are also frequently present in most animals and common contaminants in food or used as starters in meat and cheese processing. Enterococci are potential pathogens in many body sites and enterococcal infections are often opportunistic and more prevalent in hospitalized patients [7]. The general interest for enterococci and treatment of enterococcal infections has increased due to the appearance of antibiotic multi-resistant strains and especially to the occurrence of vancomycin-resistant strains (vancomycin-resistant enterococci VRE)[8].Furthermore bacterial organisms, oral microorganisms can include fungal, viral and protozoal infections. The bacteria include hundreds of types of organisms of which only predominant ones have been identified[8, 9]. A variety of organisms in the micro flora of the oral cavity adhere to the teeth, the buccal mucosa, the tongue, and the gingival sulcus. Each site has a unique way of allowing the organisms to initiate their residency. The normal flora in healthy individuals maintains similar patterns. When a local or systemic disease process or concomitant use of medications alters this overall pattern, atypical organisms begin to predominate and some normal organisms with a benign nature, such as Candida albicans, become pathogenic.

The micro flora of the oral cavity changes with the age, the eruption or loss of teeth, and the appearance of disease states (e.g., caries, periodontal disease). Systemic changes, such as pregnancy or drug intake, also alter the number and proportion of flora. These changes are due to alterations in the flow and composition of salivary fluid and in the levels and activity of defence components (e.g., immunoglobulin, cytokines) in the saliva [7, 8].Besides the

microbiological methods, the divergence of the findings regarding the prevalence of E. faecalis in root canal infections may be also dependent on the patient selection. Usually, studies that have included only restored teeth have shown lower prevalence of E. faecalis in root-filled canals than those that have also included non-restored teeth, considering the same detection method. Therefore, the presence of coronal leakage by defective coronal restorations, old temporary restorative materials, or non-restored teeth may have influenced the microbial findings of the latter studies. These findings support the hypothesis that E. faecalis may enter the root-filled canal via coronal leakage during or after root-canal treatment as secondary invaders. However, as many studies did not mention the quality of restoration, it is not possible to determine whether E. faecalis positive samples resulted from its persistence to prior root-canal treatment (persistent infections) or were originated after root-canal treatment from invading E. faecalis into the root-filling, via coronal leakage (secondary infections). This study is done to evaluate the association of Enterococcus species in postoperative cases in dentistry[3,8]. This study also includes the evaluation of the sensitivity pattern of the Enterococcus isolates against the conventionally used anti-microbial agents used for chemoprophylaxis and treatment.

Materials and Methods

Samples are collected from 75 patients who reported to the dental outpatient department over the period of 6 months. The patients selected for the study are those who have underwent extraction or surgical therapy and have developed local sepsis post operation. Two types of sample were collected depending upon the type of lesion. Swabs were collected from superficial lesions and sub mucosal areas. Aspirates were done for deeper lesions that forms abscess. The samples were collected aseptically using sterile techniques by moistening them with sterile normal saline and from deep seeded lesion, needle aspirations were done, and thenthe samples were immediately transported to the microbiology lab for processing. The Samples were collected before administration of antibiotics. The patients were informed about the study conducted and informed concern was obtained.

The samples were processed in the microbiology lab according to the standard protocol. In this study the aerobes and the facultative anaerobes were analysed by performing routine bacterial culture. Three media were used for isolation and identification of the organisms they

are,Nutrient agar, MacConkey agar and blood agar. The samples were inoculated in all the three media and incubated at 37°C for 24 hours aerobically. After incubation the plates were checked for the bacterial growth and the organisms were identifiedby biochemical reactions and other tests as per the standard protocols. After identification of the organisms, the antibiotic profile for each organism isolated was found by performing AST (Antibiotic Susceptibility Testing) by Kirbybauer's method.

Results

Total no of samples processed: 75

No of samples positive for bacterial culture: 37(Fig: 1)

Totally 9 different species of bacteria are isolated from the bacterial culture. (Table: I)

Enterococcus sp were isolated from 38% of the samples in aerobic bacterial culture.(Fig:2).

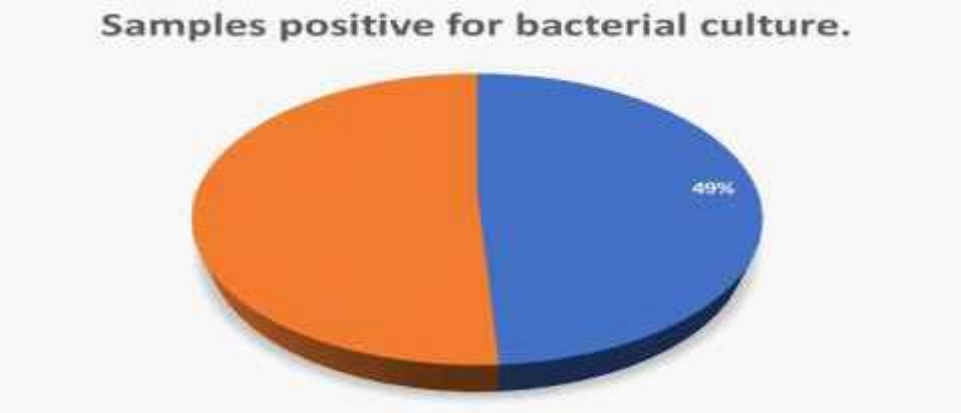
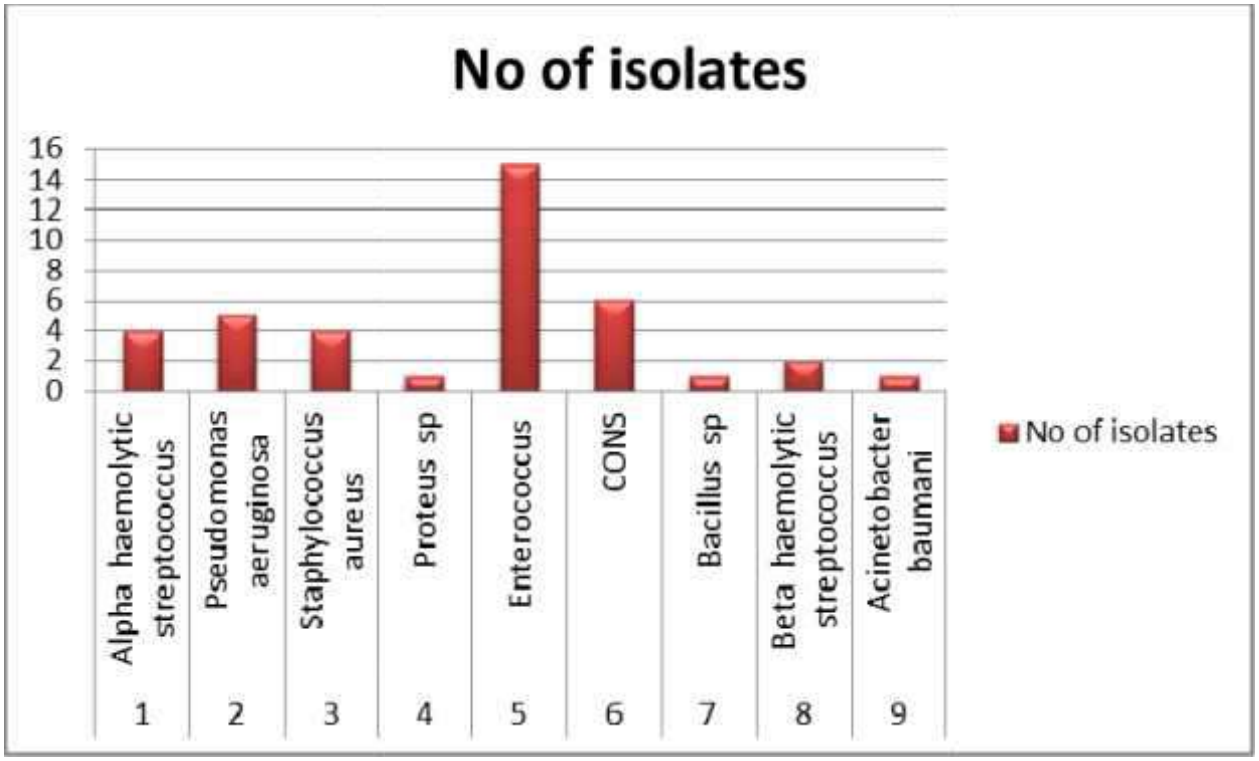


Figure 1: Pie chart showing the percentage of samples that are positive for bacterial culture out of the total no of samples processed.

Table-1: Table shows the different species of organisms isolated along with the number of isolate.

Sl.no	Organisms isolated	No of isolates
1	Alpha haemolytic streptococcus	4
2	Pseudomonas aeruginosa	5
3	Staphylococcus aureus	4
4	Proteus sp	1
5	Enterococcus	15
6	CONS	6
7	Bacillus sp	1
8	Beta haemolytic streptococcus	2
9	Acinetobacterbaumani	1



Graph 1: The bar diagram shows relative properties of bacteria isolated.

Discussion

In this study, 75 samples were collected out of which 37 samples showed positive for the microbial growth. Totally 9 different species were isolated from the samples out of which Enterococcus isolated in 38%of the samples. Enterococci are considered as resident component of the oral microbiome that may cause a variety of oral and systemic infections so we are concerned with the isolation and sensitivity pattern of enterococcus species from the post-operative complications in the oral cavity[9].

Bacteria in the mouth may trigger a rare, life-threatening heart infection called infective endocarditis. Enterococcus faecalis infective endocarditis was a disease of utmostimportance, with more infected patients, increasing frequency of health-care associated infections and increasing incidence of antimicrobial resistences3. Enterococcal meningitis is another deadly caused by Enterococcus faecalis and Enterococcus faecium disease with high mortality rate [10].

In the last few decades, the percentage of older adult Polish citizens with decreased masticatory function significantly increased. Relatively high prevalence of oral trichomonads and E. gingivalis in the present study, and, also the detection of Acanthamoeba spp. cysts in older adult patients, are in agreement with several previous findings. Various strains of Acanthamoeba sp. are worldwide amphizoic amoebae that may cause serious human health threats as etiological agents of granulomatous amoebic encephalitis and vision-threatening Acanthamoeba keratitis. The amoebae were isolated from the hospital environment, among others, as contaminants of surgical instruments and the dental irrigation system. It was considered that ontogenetic-depending factors influence the oral protozoans that are agents predisposing to local inflammations and a real threat of disseminated infections[8, 9, 11].

Enterococci are generally considered as transient oral bacteria. However, their prevalence in various population groups has not been fully evaluated, with only sparse data mainly from North American and European studies[11]. To our knowledge, this is the first study to report the prevalence of oral enterococci from a South American cohort belonging to a spectrum of age groups. As we intended to study a sample representative of the general population the

exclusion criteria included only the most frequently reported conditions that affect the oral microbiome such as, diabetes, pregnancy, wearing orthodontic appliances and dentures, use of mouth rinses, and medications that affected the salivary flow[12].

It is emphasized in literature that, in generally healthy persons, complex interrelations occur between multilayer components of biofilm, particular species of oral micro biota, and the host organism. This results in relatively constant composition of the oral cavity micro biome because the human immunological system influences the oral environment stability by some inhibition of multiplication activity of micro biota, which remains in labile homeostasis. Many biotic and abiotic factors that can alter this homeostasis may influence the multispecies microbial communities and increase the number of certain endogenous bacteria. This can also lead to a colonization of the oral cavity by exogenous species, including those that are potentially pathogenic.

Medicinal use of guava has been demonstrated in various scenarios. The important constituents of guava are vitamins, tannins, phenolic compounds, flavonoids, etc. The two important flavonoids quercetin are known for their antimicrobial and anti-oxidant effect. Guaijaverin inhibits the growth of streptococcus mutants as it increases hydrophobicity which an important factor for the oral pathogens to adhere to the tooth [13].This inhibits plaque deposit and inhibits the growth of microorganisms without disrupting the homeostatis of the oral cavity. Quercetin on the other hand disrupts the cell membrane, inactivates the extracellular proteins and form irreversible products. These prove to be bacteriostatic. It also contains vitamin c which improves healing property[14].

Deep enterococcal infections in the jaws (abscesses, bone sequestration and open surgical wounds) do occur and should be considered in the choice of antibiotics. All 18 cases were on antimicrobial treatment and 10 of them were on clindamycin, a drug that is not suitable for enterococcal infections. Unfortunately, clindamycin prescription by dentists is increasing, probably due to overuse or recommendations to use clindamycin in penicillin allergy cases[15]. The frequent occurrence of enterococcal infections in the oral cavity points to the importance for an appropriate microbiological diagnosis and susceptibility test in cases of need for antibiotic treatment [16].

At present, the guidelines published by the Infectious Diseases Society of America, the World Society of Emergency Surgery, and the Tokyo consensus meeting all recommend

treatment withsulbactam or amoxicillin plus clavulanic acid after cholecystectomy for non-complicated acute calculuscholecystitis. In the present series, we did not observe a benefit of postoperative antibiotic treatment on infections for patients with grade I or II acute calculuscholecystitis [14, 15, and 16].

Macrolides are also alternative regimens recommended for dental procedures when patients are allergic to penicillin. However, erythromycin seems to be of limited value against oral enterococci. Recently, Rams. Having shown that only 19% of periodontal E. faecalis clinical isolates were susceptible to erythromycin and most of the isolates (55%) showed an intermediate pattern. These findings are similar to previous evaluations of endodontic E. faecalis strains[17]. Moreover, the genetic determinant of macrolide resistance (ermB) has been detected in approximately 60% of endodontic E. faecalis isolates[18].

Tetracyclines are broad-spectrum antibiotics, but bacterial resistance has reduced their clinical usefulness in oral infections. This antibiotic has exerted poor in vitro activity against periodontal E. faecalis; tetracycline resistance was detected in over 50% of the E. faecalis periodontal isolates [19]. Moreover, a high prevalence of the genetic determinant of tetracycline resistance (tetM) has been recently detected in endodontic isolates [16]. Interestingly, in the latter study, approximately 50% isolates of endodontic origin carried both the ermB and tetM genes. The occurrence of multiple resistances to erythromycin and tetracycline is probably associated with the presence of conjugative transposons Tn916 family-Tn545, which carriesermB and tetM genes. It has been suggested that this mobile genetic element may have contributed to the dissemination of erythromycin and tetracycline resistance within the oral micro biota[20].

The drug of choice for most of the antibiotic prophylaxis in dentistry is amoxicillin, ampicillin and amoxyclav. Sensitivity pattern for the above mentioned drugs were tested and tabulated below.

Comparing the sensitivity pattern for the most commonly prescribed antibiotics against Enterococcus species, we state that the 3 antibiotics evaluated belong to beta-lactam group and are widely and commonly used by dentist in outpatient clinics. They show a similar susceptibility pattern 46.6 % with the enterococcus isolates. (Table: II)

Table- 2: Table showing the susceptibility pattern of Enterococcus species against the three drugs tested

Sl.no	ANTIBIOTIC	No. of isolates	No. susceptible	Percentage susceptible
1	Amoxyclav	15	7	46.6
2	Ampicillin	15	7	46.6
3	Amoxycillin	15	7	46.6

Conclusion

These three drugs amoxyclav, ampicillin and amoxicillin are commonly used in dentistry. According to this study only less than 50 % of the enterococcus isolates are found to be susceptible. It is also reported that the enterococcus sp were isolated in nearly 40 percentage of the failed cases in oral lesions. It is time to consider the other options for chemoprophylaxis in dental treatments. Isolation of enterococci resistant to multiple antibiotics has become increasingly common in the hospital setting5. This cannot be the drug of choice in elderly patients and patient having other underlying medical problems. Linezolid is also tested for all gram positive bacteria isolated from clinical samples which have shown high sensitivity, so this can be considered as an alternative for the patient underwent major surgery. The next drug which has a beneficial effect is ciproflaxin this can be given as an antibiotic cover for pre and post-surgical prophylaxis and for the outpatient procedures. According to National Nosocomial Infections Surveillance (NNIS) data from January 2003 through December 2003, more than 28% of Enterococcal isolates in ICUs of the more than 300 participating hospitals were vancomycin-resistant. Clonal spread is the dominant factor in the dissemination of multiple drug-resistant enterococci in North America and Europe. Virulence and pathogenicity factors have been described using molecular techniques. Several genes isolated from resistant enterococci encode virulence factors such as the production of gelatinase and haemolysin, adherence to caco-2 and hep-2 cells, and capacity for biofilm formation. The choice of antibiotics plays a very important role in fighting against micro-organisms; this can be achieved by checking the sensitivity pattern of drug against them.

Corresponding Author

VinothKumar

Senior Lecturer,Department of Prosthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University,

162, Poonamallee High Road,Chennai 600077, Tamil Nadu, India

E-Mail: doctor.vinoth.kumar@gmail.com

Telephone Number: +919790098719

Reference

1. I.B. Bender, D.D.S., R.S. Pressman, M.D., Sylvia G. Tashman, B.A.Comparative effects of local and systemic antibiotic therapy in the prevention of postextraction bacteraemia. The Journal of the American Dental Association. 1958; 57(1):54–66.
2. Ashwin K S, Muralidharan N P. Vancomycin-resistant enterococcus (VRE) vs Methicillin-resistant Staphylococcus Aureus (MRSA). Indian J Med Microbiol. 2015; 33(1):166-7.
3. Komiyama EY, Lepesqueur LSS, Yassuda CG, Samaranayake LP, Parahitiyawa NB, Balducci I. Enterococcus Species in the Oral Cavity: Prevalence, Virulence Factors and Antimicrobial Susceptibility. PLoS ONE 2016; 11(9): e0163001.
4. Pinheiro ET, Mayer MPA. Enterococcus faecalis in Oral Infections. J Interdiscipl Med Dent Sci 2015; 3:160.
5. Sun J, Sundsfjord A, Song X. Enterococcus faecalis from patients with chronic periodontitis: virulence and antimicrobial resistance traits and determinants. Eur J ClinMicrobiol Infect Dis 2012; 31: 267-272.
6. Anders Dahl and NielsEskeBruun. Enterococcus faecalis infective endocarditis: focus on clinical aspects. Expert Review of Cardiovascular Therapy. 2013; 11(9):1247-1257.
7. Razavi A, Gmür R, Imfeld T, Zehnder M. Recovery of Enterococcus faecalis from cheese in the oral cavity of healthy subjects. Oral Microbiol Immunol. 2007;22: 248–251.
8. Chiara Iaria, Giovanna Stassi, Gaetano Bruno Costa, Rita Di Leo, Antonio Toscano and Antonio Cascio.Enterococcal meningitis caused by Enterococcus casseliflavus. First case report. BMC Infectious Diseases2005; 5(1):3:1-3.

9. Courvalin P. Vancomycin resistance in gram-positive cocci. Clin Infect Dis. 2006; 42(1):25-34.
10. Deshpande LM, Fritsche TR, Moet GJ. Antimicrobial resistance and molecular epidemiology of vancomycin-resistant enterococci from North America and Europe: a report from the SENTRY antimicrobial surveillance program. Diagn Microbiol Infect Dis. 2007; 58(2):163-70.
11. Klibi N, Ben Slama K, Sáenz Y. Detection of virulence factors in high-level gentamicin-resistant Enterococcus faecalis and Enterococcus faecium isolates from a Tunisian hospital. Can J Microbiol. 2007; 53(3):372-9.
12. Kritivasan S and Muralidharan NP: Antimicrobial activity of fruit juices on oral bacteria. Int J Pharm Sci Res 2017; 8(1).289-93.
13. Yingling NM, Byrne BE, Hartwell GR. Antibiotic use by members of the American Association of Endontists in the year 2000: report on a national survey. J Endod. 2002; 28: 396–404.
14. Sun J, Song X, Kristiansen BE, Kjaereng A, Willems RJ. Occurrence, population structure, and antimicrobial resistance of enterococci in marginal and apical periodontitis. J ClinMicrobiol 2009; 47: 2218-2225.
15. Colombo AV, Barbosa GM, Higashi D, di Micheli G, Rodrigues PH,. Quantitative detection of Staphylococcus aureus, Enterococcus faecalis and Pseudomonas aeruginosa in human oral epithelial cells from subjects with periodontitis and periodontal health. J Med Microbiol 2013; 62(10):1592-1600.
16. Penas PP, Mayer MP, Gomes BP, Endo M, Pignatari AC, Bauab KC. Analysis of genetic lineages and their correlation with virulence genes in Enterococcus faecalis clinical isolates from root canal and systemic infections. J Endod. 2013; 39(7):858-864.
17. Sedgley CM, Molander A, Flannagan SE, Nagel AC, Appelbe OK, Clewell DB. Virulence, phenotype and genotype characteristics of endodontic Enterococcus spp. Oral Microbiol Immunol. 2005; 20(1):10–9.
18. Rams TE, Feik D, Young V, Hammond BF, Slots J. Enterococci in human periodontitis. Oral Microbiol Immunol. 1992; 7(4):249–52.
19. Souto R, Colombo AP. Prevalence of Enterococcus faecalis in subgingival biofilm and saliva of subjects with chronic periodontal infection. Archs Oral Biol. 2008; 53(2):155–60.

20. Sun J, Song X, Kristiansen B, Kjaereng A, Willems RJL, Eriksen HM. Occurrence, population structure and antimicrobial resistance of enterococci in marginal and apical periodontitis. J Clin Microbiol. 2009; 47(7):2218–25.

Lactobacilli Count In Halitosis

Nandhini.S[1], Muralidharan[2], Jayanth Kumar.V[3]

Graduate Student[1], Reader[2], Department of Microbiology, Reader[3], Department of Oral Medicine,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Halitosis has multiple etiology though physiological factors plays a major role.Presence of lactobacilli is considered as one of the reason for halitosis.When the salivary PH is altered,it facilitates the growth of lactobacilli.Halitosis is not a disease,it's a discomfort in the oral cavity.Dental caries also causes halitosis .If the number of this bacteria in the mouth is reduced,halitosis in the oral cavity can be prevented.This study is conducted to estimate the contribution and association of lactobacilli in halitosis.

Materials and Methods: Lactobacilli association with caries is already proved and its contribution in halitosis cases is estimated in this study. It is done by examining the lactobacilli load of patients with halitosis and dental caries.The saliva sample of bad breadth and widespread caries is collected in a sterile container.And 10 microlitre is transferred to the lactobacilli agar and its being incubated. Afterincubation, the number of colonies is demonstrated and counted.

Results: The lactobacilli count obtained ranges from 500 colonies/ml to 6800colonies/ml.65% of the samples had shown the colony count of more than 3000/ml and 35% of the samples had shown the colony count of less than 3000/ml.There is a considerable increase in many cases.This is definitely higher than the bacilli count in normal healthy oral cavity. In normal healthy oral cavity lactobacilli count is less than 1000/ml.

Conclusion: This indicates the Lactobacilli is not solely responsible for caries activity but it precipitates the problem. It is believed that extensive caries lesion in mouth is the reason for

bad breadth. This study shows there is a considerable increase in Lactobacilli count in carious individuals which contributes to halitosis and they are not responsible for initiation of caries.

***Key Words**: halitosis,odour,lactobacilli,breadth,discomfort*.

Introduction

Halitosis, also commonly known as “bad breath,” is a concern of many patients seeking help from health care professionals. The health care workers have neglected the subject of oral malodour but recently, along with the growing public and media interest in oral malodour; health care professionals are becoming more aware of their patient’s concern. A patient with halitosis is most likely to contact primary care practitioner for the diagnosis and management. Most physicians and dental practitioners are inadequately informed about the causes and treatments of halitosis. The present article succinctly focuses on the development of a systematic flow of events to come to the best management of the halitosis from the primary care practitioner’s point of view. The epidemiological research on halitosis is inadequate since it is still a considerable but underrated taboo.Oral malodour is a problem that afflicts large population.Because halitosis can connect or disconnect an individual from the social environment. It plays an important role in self-image and social interactions. Halitosis affects people with varying degrees of intensity. The most common oral malodour products are by products of metabolism of certain species of oral bacteria present in saliva, plaque and gingival crevicular fluid .Halitosis is mainly seen in diabetic patients and patients with caries.Lactobacilli,a gram positive bacteria is usually benign except in the mouth where they have been associated with cavities and tooth decay.[1]Studies have proven that lactobacillus count in saliva has been used as caries test for many years[2,3].Recent studies have also proven that lactobacilli is seen in caries and halitosis patients.[4]The reasons for the lack of scientific data are the difference in cultural and racial appreciation of odours for patients and investigators, and there is the absence of uniformity in evaluation methods, as for organoleptical as for mechanical measurements. Moreover, there are no universally accepted standard criteria, objective or subjective, that define a halitosis patient. There are few studies documenting the prevalence of halitosis in population wide or community based samples. In the general population, halitosis has a prevalence ranging from 50% in the USA to between 6% and 23% in china, and a recent study had revealed a prevalence ofself reported halitosis

among Indian dental students ranging from 21.7% in males to 35.3% in females.[7,8,12] Miyazaki concluded that there was increased correlation between older age and malodour with aging resulting in greater intensity the of odour. In above 60 years age group of the Turkish individuals, the incidence was around 28%.A thorough literature search reveals a lack of studies on halitosis in India, especially among the general population.

Etiology of Halitosis

It is imperative to understand the origin of halitosis as multidisciplinary therapy typically is required in halitosis with emphasis on the causative factor. Halitosis can be broadly classified on the basis of its origin as Genuine Halitosis and Delusional Halitosis .Physiological halitosis (foul morning breath, morning halitosis) is caused by stagnation of saliva and putrefaction of entrapped food particles and desquamated epithelial cells by the accumulation of bacteria on the dorsum of the tongue, recognized clinically as coated tongue and decrease in frequent liquid intake.[13,14,15]Intraoral conditions are the cause of 80–85% of halitosis cases.Periodontal infections are characterized by a tremendous increase in Gram-negative bacteria that produce volatile sulphur compounds (VSCs). The association between anaerobic bacteria that produces VSCs and halitosis has been well-documented.Most important VSCs are hydrogen sulphide (H2 S), methyl mercaptan and dimethyl sulfide.The dorsum of the tongue is the biggest reservoir of bacteria as a source of malodorous gases. Pericoronitis, oral ulcers, periodontal abscess, and herpetic gingivitis are some of the pathologies that result in increased VSCs. Diamines such as putrescine and cadaverine are also responsible for oral malodor as with the increase in periodontal pocket depth; oxygen tension decreases which results in low pH necessary for the activation of the decarboxylation of amino acids to malodorous diamines.[16,17]Odontogenic infections include retention of food debris in deep carious lesions and large interdental areas, maligned teeth, faulty restorations, exposed necrotic pulp, over wearing of acrylic dentures at night, wound infection at the extraction site and ill-fitting prosthesis. The absence of saliva or hypo function results in an increased Gram-negative microbial load, which increases VSCs, a known cause of malodour. Several mucosal lesions such as syphilis, tuberculosis, stomatitis, intraoral neoplasia and peri-implantitis allow colonization of microorganisms that releases a large amount of malodors compounds.[18]

Materials and Methods

Lactobacilli association with caries is already proved andits contribution in halitosis cases is estimated in this study.The saliva sample of bad breadth and widespread caries is collected in a sterile container.Transfer 10 microlitre to the lactobacilli agar and its being incubated.Afterincubation,the number of colonies is demonstrated and counted.Results are expressed as colony forming units per 100 millilitre in the table below.

Results

SL.NO	LACTOBACILLI COUNT/ ml
1	500
2	500
3	900
4	1000
5	1200
6	1900
7	1900
8	2100
9	2100
10	2300
11	2600
12	3000
13	3500
14	4100
15	5000
16	5000
17	5200
18	5500
19	6000
20	6800

In this study to know about the association between halitosis and amount of lactobacilli present in the saliva.The samples were inoculated into lactobacilli selective media.After incubation lactobacilli count was done.The lactobacilli count obtained ranges from 500 colonies/ml to 6800colonies/ml.65% of the samples had shown the colony count of more than 3000/ml and 35% of the samples had shown the colony count of less than 3000/ml.There is a considerable increase in many cases.This is definitely higher than the bacilli count in normal healthy oral cavity. In normal healthy oral cavity lactobacilli count is less than 1000/ml. In this study there is no direct correlation between Lactobacilli load ,saliva and caries activity.All the patients selected for the study have multiple carious lesion but Lactobacilli count in saliva ranges from 500 per ml to 6800 per ml.This indicates the Lactobacilli is not solely responsible for caries activity but it precipitates the problem.It is believed that extensive caries lesion in mouth is the reason for bad breadth.This study shows there is a considerable increase in Lactobacilli count in carious individuals which contributes to halitosis and they are not responsible for initiation of caries.

Discussion

Lactobacillus is associated with progression of caries which actively damage pulpal tissues of teeth.Increase in presence of Lactobacilli in saliva is directly associated with caries activity.Though Lactobacilli is not initiating caries formation initially,in a patient who has caries activity the presence of Lactobacilli is very important.Presence of Lactobacilli in mouth is associated with alteration in pH of the saliva.There are common findings seen in gastric and peptic ulcer.Eradication of halitosis must be considered first.Mechanical removal of biofilm and microorganisms is the first step in the control of halitosis. A systemic review by van derSleen et al. demonstrated that tongue brushing or tongue scraping have the potential to successfully reduce breath odour and tongue coating.Tongue scrapers are shaped according to the anatomy of the tongue and reduces 75% VSCs compared to only 45% using a toothbrush. Antibacterial mouth rinsing agents include chlorhexidine (CHX), cetylpyridinium chloride (CPC) and triclosan, which act on halitosis-producing bacteria. A systematic review, published by Cochrane, compared the effectiveness of mouth rinses in controlling halitosis.The researchers concluded that mouth rinses containing CHX and CPC

could inhibit production of VSCs while mouth rinses containing chlorine dioxide and zinc may neutralize the sulphur compounds producing halitosis. CHX is considered as the gold standard mouth rinse for halitosis treatment. CHX in combination with CPC produce greater fall in VSCs level, and both aerobic and anaerobic bacterial counts showed the lowest percentage of survival in a randomized, double–blind, cross–over study design. The usage of masking agents like rinsing products, sprays, toothpaste containing fluorides, mint tablets or chewing gum only have a short-term masking effect.[19,20]When treating patients with oral malodour, clinicians should relate not only to physiological odour and associated parameters but also to the nature of the subjective complaint. In halitosis management, a well-established understanding between a patient and a primary healthcare clinician can bring a successful result. A primary healthcare clinician must exhibit attitudes of acceptance, sympathy, support, and reassurance to reduce the patient's anxiety. Professionals can improve patient quality of life as a whole, improving their social interactions and relationships. A sustained encouragement and reassurance need to be given by the patient's primary healthcare clinician, family, and friends. Due to the multifactorial complexity of halitosis, patients should be treated individually, rather than be categorized.Diagnosis and treatment need to be a multidisciplinary approach involving the primary healthcare clinician, dentist, an ENT specialist, nutritionist, gastroenterologist and clinical psychologist.Future research is needed to test accessible methods of drawing a person's attention to his/her halitosis, being the first step of seeking treatment. [24,25,26]

Conclusion

Halitosis is an extremely unappealing characteristic of sociocultural interactions and may have long-term detrimental aftereffects on psychosocial relationships. With proper diagnosis, identification of the etiology, and timely referrals when needed, steps can be taken to create a successful individualized therapeutic approach for each patient seeking assistance. It is significant to highlight the necessity of an interdisciplinary method for the treatment of halitosis to prevent misdiagnosis or unnecessary treatment. The literature on halitosis, especially with randomized clinical trials, is scarce and additional studies are required. This indicates the Lactobacilli is not solely responsible for caries activity but it precipitates the problem.It is believed that extensive caries lesion in mouth is the reason for bad

breadth.[21,22]This study shows there is a considerable increase in Lactobacilli count in carious individuals which contributes to halitosis and they are not responsible for initiation of caries.[23]

Corresponding Author

V.Jayanth Kumar,

Reader, Department of Oral Medicine,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email Id:doctorjayanth@gmail.com

Mobile Number: +91 8056135807

References

1.Ljunh,Asa;wardstorm,Torkel,eds.Lactobacillus molecular biology;from Genomics to Probiotics.CaisterAcademic press,2009;140:7-9.

2.Twetman,S;Steckson-Blicks,C(.Probiotics and oral health effects in children .*International journal of pediatric dentistry*2008;18(1):3-10.

3.Meurman,J.H.;Stamatova,I().”Probiotics;Contributions to oral health”.Oral diseases.2007;13(5):443-51.

4.Makarova, k;slesarev,A.;wolf,Y,;Sorokin,A;Mirkin,;B;koonin,;E.Pavlova.N.et.al.oct 2006

“Comparitive genomics of lactic acid bacteria”.,2002;131:158-62.

5. Liu XN, Shinada K, Chen XC, Zhang BX, Yaegaki K, Kawaguchi Y. Oral malodor-related parameters in the Chinese general population. *J ClinPeriodontol* 2006;33:31-6.

6. Lee SS, ZhangW, LiY. Halitosis update: A review of causes, diagnoses, and treatments. *J Calif Dent Assoc* 2007;35:258-60.

7. Ashwath B, Vijayalakshmi R, Malini S. Self-perceived halitosis and oral hygiene habits among undergraduate dental students. *J Indian SocPeriodontol* 2014;18:357-60.

8. Miyazaki H, Sakao S, Katoh Y, Takehara T. Correlation between volatile sulphur compounds and certain oral health measurements in the general population. *J Periodontol* 1995;66:679-84.

9. Avcu N, Ozbek M, Kurtoglu D, Kurtoglu E, Kansu O, Kansu H. Oral findings and health status among hospitalized patients with physical disabilities, aged 60 or above. Arch GerontolGeriatr 2005;41:69-79.

10. Porter SR, Scully C. Oral malodour (halitosis). BMJ 2006;333:632-5.

11. Wilhelm D, Himmelmann A, Axmann EM, Wilhelm KP. Clinical efficacy of a new tooth and tongue gel applied with a tongue cleaner in reducing oral halitosis. *Quintessence Int* 2012;43:709-18.

12. Delanghe G, Ghyselen J, van Steenberghe D, Feenstra L. Multidisciplinary breath-odour clinic. Lancet 1997;350:187.

13. Aylikci BU, Colak H. Halitosis: From diagnosis to management. J Nat Sci Biol Med 2013;4:14-23. 14. Fletcher SM, Blair PA. Chronic halitosis from tonsilloliths: A common etiology. J La State Med Soc 1988;140:7-9.

15. Newman MG, Takei H, Klokkevold PR, Carranza FA. Carranza's Clinical Periodontology.. Missouri: Saunders and Elsevier Inc.; 2006;10: 330-42.

16. Rösing CK, Loesche W. Halitosis: An overview of epidemiology, etiology and clinical management. *Braz Oral Res* 2011;25:466-71.

17. Kinberg S, Stein M, Zion N, Shaoul R. The gastrointestinal aspects of halitosis.*Can J Gastroenterol* 2010;24:552-6.

18. Stoeckli SJ, Schmid S. Endoscopic stapler-a s s i s t e d diverticuloesophagostomy for Zenker's diverticulum: Patient satisfaction and subjective relief of symptoms. Surgery 2002;131:158-62.

19. Kim JG, Kim YJ, Yoo SH, Lee SJ, Chung JW, Kim MH. Halimeterppb levels as the predictor of erosive gastroesophageal reflux disease. Gut Liver 2010;4:320-5.

20. Lee H, Kho HS, Chung JW, Chung SC, Kim YK. Volatile sulfur compounds produced by Helicobacter pylori. *J ClinGastroenterol* 2006;40:421-6.

21. Messenger J, Clark S, Massick S, Bechtel M. A review of trimethylaminuria: (fish odor syndrome*). J ClinAesthetDermatol* 2013;6:45-8.

22. T. V. RajalakshmiRakshanaa, T. LakshmiAntibacterial efficacy of herbal mouthwash against oral microbes - in vitro assay, *J AdvPharmEdu Res* 2017;7(1):31-33.

23. Christina PrithikaSimon, Dr.Karthikeyan. Probiotics in Periodontology, *IOSR Journal of Dental and Medical Sciences* (IOSR-JDMS) e2279-0853, p-ISSN- 2279-0861. 13(4) :103-105.

24.Lakshmi T, Balusamy SR, Parameswari R. A review on green tea catechins in oral health management.*JAdvPharmEdu Res* 2017;7(3):178-181.

25. Nadar S, Saravana Dinesh SP. A questionnaire study about oral hygiene awareness among orthodontic patients. Int J OrthodRehabil 2016;7:97-100.

26.TrophimusGnanabagyanJayakaran , The Effect of Drugs in the Oral Cavity - A Review *J. Pharm. Sci. & Res.*2014;6(2):89-96.

Lipid Abnormalities in Thyroid Patients

Monalisa.R[1], Jagadeesan [2], Remmiya Mary Varghese[3]
Graduate Student [1],Reader ,[2]Department of General Medicine
Senior Lecturer[3], Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: Thyroid hormones influence all major metabolic pathways. SH stimulates the thyroid gland to make thyroid hormones and secrete them into the blood. The thyroid hormones control the body's metabolism and can affect cholesterol levels. When thyroid hormone levels are low and TSH levels are increased, cholesterol levels are increased.Their most obvious and well-known action is an increase in basal energy expenditure obtained acting on protein, carbohydrate and lipid metabolism. With specific regard to lipid metabolism, thyroid hormones affect synthesis, mobilisation and degradation of lipids, although degradation is influenced more than synthesis.The main and best-known effects on lipid metabolism include: (a) enhanced utilisation of lipid substrates; (b) increase in the synthesis and mobilisation of triglycerides stored in adipose tissue; (c) increase in the concentration of non-esterified fatty acids (NEFA); and (d) increase of lipoprotein-lipase activity. While severe hypothyroidism is usually associated with an increased serum concentration of total cholesterol and atherogenic lipoproteins, the occurrence of acute myocardial infarction (AMI) in hypothyroid patients is not frequent.The aim of the study is to assess the prevalence of lipid abnormalities in thyroid patients.To assess the prevalence of lipid abnormalities in thyroid patients. The Study of lipid profile was conducted in 50 thyroid patients.

Materials and Method: Cross sectional study demonstrating the lipid abnormalities in 50 thyroid patients in Saveetha medical college. Data was collected using a pro forma which consists of clinical profile,lipid profile and thyroid profile. Values were estimated and statistics was done.

Result: Total cholesterol and LDL cholesterol were found to maintain significant positive correlation with serum TSH. But TSH was found to show no correlation with serum HDL cholesterol and serum triglycerides level. Significant negative correlation of FT4 with

triglycerides level and no correlation of TSH with triglycerides appear to make a sense of functional disharmony between FT4 and TSH and, therefore, a large scale study is recommended to make it clear.

Conclusion: Results of our study suggest the findings of dyslipidemia in hypothyroid patients. Therefore, patients presenting with dyslipidemia are recommended to be investigated for hypothyroidism. As our sample size was small and duration of study was limited, another study with large sample size and longer duration is also recommended

Key Words *Thyroid,Lipid,Hypercholestremia, Hypercholestremia, Dyslipidaemia.*

Introduction

Hypothyroidism is a clinical syndrome resulting from a deficiency of thyroid hormones, which in turn results in a generalized slowing down of metabolic processes[1]. It is a common metabolic disorder in general population[2]. The thyroid dysfunction increases with age, especially in women[3]. Hypothyroidism is associated with many biochemical abnormalities. Levels of total cholesterol and low density lipoprotein cholesterol tend to increase as thyroid functiondeclines[2]. Thus hypothyroidism constitutes a significant cause of secondary dyslipidaemia [4]. In hypothyroid patients, despite the reduced activity of HMG CoA reductase, there is often an increase in the serum total cholesterol concentration, mainly due to raised levels of serum LDL cholesterol and intermediate density lipoprotein (IDL) cholesterol[5]. Decreased thyroid secretion greatly increases the plasma concentration of cholesterol because of decreased rate of cholesterol secretion in the bile and consequent diminished loss in the faeces due to decreased number of low density lipoprotein receptors on liver cells[6]. Decreased activity of LDL receptors resulting in decreased receptor-mediated catabolism of LDL and IDL is the main cause of the hypercholesterolemia observed in hypothyroidism[7]. Serum concentration of high density lipoprotein cholesterol was reported to be higher among newly diagnosed hypothyroid patients with the value more than 40 mg/dL in subclinical or clinical hypothyroidism whereas serum concentrations of HDL cholesterol were significantly lower among euthyroid and previously reported hypothyroid cases who were on thyroid replacement therapy[8]. Hypothyroid patients usually exhibit elevated levels of high density lipoprotein cholesterol (HDL-C) mainly due to increased concentration of HDL particles[9]. In some studies we find confronting results regarding high density lipoprotein cholesterol levels in hypothyroidism. In thyroidectomized rats there was 25.9%

decrease in HDL-C level, suggesting a defect in HDL metabolism [10]. HDL cholesterol level was found reduced in some other studies on hypothyroid patients[9]. Decreased thyroid secretion greatly increases the plasma concentration of triglycerides[6]. Nikkilia & Kekki11 have stated that hypertriglyceridemia in hypothyroidism is due to decreased activity of lipoprotein lipase (LPL), which results in decreased clearance of triglyceride-rich lipoproteins. Dyslipidemia is a well-known risk factor for cardiovascular diseases. The risk of coronary heart disease and other forms of atherosclerotic vascular disease rises with rising plasma cholesterol concentration and in particular with rising ratio of total cholesterol to high density lipoprotein (HDL) cholesterol. A weak positive correlation of coronary heart disease also exists with plasma triglyceride concentration[12]. Early diagnosis and proper management can significantly reduce the mortality and morbidity of dyslipidaemia cardiovascular diseases. Extensive large-scale randomised trials have shown that lowering total cholesterol and LDL cholesterol reduces the risk of cardiovascular events like angina, myocardial infarction and stroke, and also reduces the need for revascularization [12].

Many studies were done to assess the lipid profile status of hypothyroid patients. But controversies still prevail and that needs to attain consensus. So, we have designed this study in our population for evaluation of lipid profile in hypothyroid patients that might be helpful for clinical management of hypothyroid patients with dyslipidaemia.

The aim of the study is to assess the prevalence of lipid abnormalities in thyroid patients.

Materials and Methods

This cross sectional study was conducted in the department of General medicine of Saveetha Medical University, Chennault to evaluate the lipid profile of hypothyroid patients and to find out relationship of Dyslipidemia with severity of hypothyroidism. Clinically of both sexes.Hypothyroidism was diagnosed by clinical history, physical examinations and relevant laboratory investigations. Total 50 subjects were included in the study and out of them 11 overt hypothyroid patients were grouped as cases (Group I) and 9 euthyroid subjects were grouped as normal controls (Group II). Cases were further grouped on the basis of their serum TSH concentration into Group A (TSH level 5.01-50.00 mIU/L), Group B (TSH 50.00100.00 mIU/L) and Group C (TSH level>100.00 mIU/L).

Statistical Analysis

Statistical analyses were performed by using SPSS for Windows version 12.0. Mean values of the findings were compared among and between groups. Analysis of variance (ANOVA)

test and Unpaired 't' test were done to assess the significance among the groups and between groups respectively. Pearson correlation coefficient test was done to evaluate the correlation of biochemical parameters with the severity of hypothyroidism. 'p' values <0.05 were considered significant.

Results

Table I shows the comparison of the lipid profile parameters between the cases and the controls. Mean total cholesterol, HDL cholesterol, LDL cholesterol and triglycerides level in cases were higher than controls. HDL cholesterol was found significantly decreased in cases compared to controls. Table II shows lipid profile parameters in different groups of cases. There was no significant difference among the groups for serum total cholesterol, but a rising trend was found from Group A to Group C. For HDL cholesterol levels, significant differences were observed among the groups with maximum in Group B and minimum in Group C. Mean LDL cholesterol levels showed significant difference among the groups with maximum in Group C and minimum in Group A. Mean serum triglyceride levels showed no significant difference among groups.

Table 1: Lipid Profile of Study Subject

Parameters	Cases (n= 80)	Cases (n= 80)	Cases (n= 80)
Total cholesterol (mg/dL)	241.56 ± 60.05	146.94 ± 23.21	< 0.001
HDL cholesterol (mg/dL)	49.59 ± 11.69	55.89 ± 11.70	< 0.05
LDL cholesterol (mg/dL)	151.96 ± 59.60	71.43 ± 26.83	< 0.001
Triglycerides (mg/dL)	212.28 ± 100.73	98.87 ± 39.69	< 0.001

Table 2: Lipid Profile in Different Groups of Case

Parameters	Group A	Group B	Group C	'P'values
Total cholesterol (mg/dL)	215.93 ± 43.25	237.10 ± 64.49	254.76 ± 59.61	> 0.05

HDL cholesterol (mg/dL)	51.36 ± 9.84	53.34 ± 13.01	45.97 ± 10.35	< 0.05
LDL cholesterol (mg/dL)	119.64 ± 45.00	150.41 ± 61.28	165.41 ± 59.57	< 0.05
Triglycerides (mg/dL)	224.36 ± 88.83	221.16 ± 112.39	221.16 ± 112.39	> 0.05

Discussion

Hypothyroidism is a common metabolic disorder. The prevalence of primary hypothyroidism is 1:100, but it maybe 5:100 if patients with subclinical hypothyroidism (normal T4, raised TSH) are included [13]. According to a study done by Sawin [14], hypothyroidism is a common disorder with a prevalence rate up to 20%. In another cross-sectional study on twelve hundred and twelve subjects of both sexes and age 20-60 years, the incidence of subclinical hypothyroidism was 19.7% [15]. In our study, mean total cholesterol, LDL cholesterol and triglycerides were found significantly increased whereas HDL cholesterol was found significantly decreased in cases compared to controls. Jung [16] found mean plasma total cholesterol and LDL cholesterol levels elevated in hypothyroid cases than in normal controls. In another study, average serum total cholesterol level was found elevated in primary and secondary hypothyroidism [17]. Keyes &Heimberg18, Laker & Mayes [19] found triglyceride level elevated in hypothyroid patients. So, our study findings were consistent with the previous studies done by other investigators. Thompson [7] and Abrams & Grundy [20] have stated decreased activity of LDL receptors as the main cause of hypercholesterolemia in hypothyroidism. Serum concentrations of high density lipoprotein cholesterol was reported to be higher among newly diagnosed hypothyroid patients (subclinical or clinical) whereas serum concentrations of HDL cholesterol were significantly lower among euthyroid and previously reported hypothyroid cases who were on thyroxine replacement therapy [8]. Studies done by Michalopoulou [21], Diekman [22], Tsmihodimos [23] and Olukoga [24] showed average serum concentration of HDL higher in subclinical or clinical hypothyroidism. But, on the other hand, in a study on thyroidectomized rats, HDL-C showed a 25.9% decrease compared to controls [10]. In another study on thyroidectomized rats, there was 25% decrease in HDL cholesterol level compared to control rats [25]. Abrams & Grundy 20 have shown in their studies reduction of HDL cholesterol in hypothyroidism. So, our study is consistent with some of the studies and inconsistent with the others. A large

scale study on overt hypothyroid patients is recommended to come to a final conclusion. Increase in HDL cholesterol concentration is mainly due to increased concentration of HDL particles. Dullaart [26] have stated that decreased activity of CETP (cholesteryl ester transport protein) results in reduced transfer of cholesteryl esters from HDL to VLDL, thus increasing HDL cholesterol levels. Lam [27] have stated that in hypothyroid patients decreased activity of hepatic lipase leads to the decreased catabolism of HDL particles leading to increased HDL. So, decrease in HDL cholesterol level in our study might be due to increased activity of CETP and lipoprotein lipase in hypothyroid patients. There was no significant difference in serum total cholesterol concentrations among the groups, but a rising trend was found from Group A to Group C ($p = 0.0395$),making our results apparently consistent with the severity of hypothyroidism. This theme seems to be further consolidated by increasing LDL concentrations in line with the increasing severity of hypothyroidism from Group A to Group C as revealed in our study. This is further strengthened by our findings of negative correlation of FT4 and positive correlation of TSH with total cholesterol and LDL cholesterol levels. Significant difference in serum HDL cholesterol levels was observed among the groups with maximum in Group B and minimum in Group C. This finding partially indicates the lowest HDL with maximum degree of hypothyroidism. But the characteristic pattern of decreasing HDL with increasing severity of hypothyroidism could not be revealed in this study. This is further supported by our finding of no correlation of HDL with FT4 or TSH. This might be due to our small sample size. Regarding triglycerides levels, no significant difference was found among the groups, indicating no changing impact on triglycerides metabolism with severity of hypothyroidism. It is claimed that thyroid hormones facilitate the LPL activity11, but deficiency of thyroid hormones to inhibit LPL probably follows a ceiling point beyond which further reduction of thyroid hormones does not cause further inhibition of LPL. In our study FT4 has shown significant negative correlation with serum total cholesterol, LDL cholesterol and triglycerides level. No correlation was found between serum FT4 level and serum HDL cholesterol level. Total cholesterol and LDL cholesterol were found to maintain significant positive correlation with serum TSH. But TSH was found to show no correlation with serum HDL cholesterol and serum triglycerides level. Significant negative correlation of FT4 with triglycerides level and no correlation of TSH with triglycerides appear to make a sense of functional disharmony between FT4 and TSH and, therefore, a large scale study is recommended to make it clear. Results of our study suggest the findings of dyslipidemia in hypothyroid patients. Therefore, patients presenting with dyslipidemia are recommended to be investigated for

hypothyroidism. As our sample size was small and duration of study was limited, another study with large sample size and longer duration is also recommended.

Conclusion

Results of our study suggest the findings of dyslipidemia in hypothyroid patients. Therefore, patients representing with dyslipidemia are recommended to be investigated for hypothyroidism. As our sample size was small and duration of study was limited, another study with large sample size and longer duration is also recommended.

Corresponding Author

Remmiya Mary Varghese

Senior Lecturer, Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India

Mobile Number:+919840488439

Email id: remmiya@gmail.com

References

1.Greenspan FS. The thyroid gland. In: Greenspan FS & Gardner DG (eds). Basic & Clinical Endocrinology. 7th edi. New York: The McGraw-Hill Companies, 2004:215-294.

2. Khan MA, Majumder I, Hoque M, Fariduddin M, Mollah FH, Arslan MI. Lipid profile in hypothyroid patients: a cross sectional Study. Medicine today. 2013 Jul 28;25(1):21-4.

3. Duntas LH. Thyroid disease and lipids. Thyroid. 2002 Apr 1; 12(4):287-93.

4. Tsimihodimo v, Bairaktari e, Tzallas c, Miltiadus g, Liberopoulos E, Elisaf m. The incidence of thyroid function abnormalities in patients attending an outpatient lipid clinic. Thyroid. 1999 Apr; 9(4):365-8.

5. Stone NJ. Secondary causes of hyperlipidemia. Medical Clinics of North America. 1994 Jan 1; 78(1):117-41.

6. Guyton AC, Hall JE. The thyroid metabolic hormones. Textbook of medical physiology. 10th edi. New York: W B Saunders Company 1991; 11:931-42.

7. Thompson GR, Soutar AK, Spengel FA, Jadhav A, Gavigan SJ, Myant NB. Defects of receptor-mediated low density lipoprotein catabolism in homozygous

familialhypercholesterolemia and hypothyroidism in vivo. Proceedings of the National Academy of Sciences. 1981 Apr 1; 78(4):2591-5.

8. Castro AV, Bononi AP, Aragon F, Padovani CR, Nogueira CR, Mazeto GM, Pimenta WD. Clinical and laboratory evaluation of hyperlipemic and hypothyroid patients. Arquivos brasileiros de cardiologia. 2001 Feb; 76(2):123-6.

9. Heimberg M, Olubadewo JO, Wilcox HG. Plasma lipoproteins and regulation of hepatic metabolism of fatty acids in altered thyroid states. Endocrine Reviews. 1985 Oct 1; 6(4):590-607.

10. Huesca-G C, Franco M, Monta LF, Mass F, Posadas-Romero C. Chronic hypothyroidism induces abnormal structure of high-density lipoproteins and impaired kinetics of apolipoprotein AI in the rat. Metabolism. 2002 Apr 1; 51(4):443-50.

11. Nikkilä EA, Kekki M. Plasma triglyceride metabolism in thyroid disease. Journal of Clinical Investigation. 1972 Aug; 51(8):2103.

12. Haslett C. Diseases of cardiovascular system, Davidson's principles and practice of Medicine, Haslett C, Chilvers ER, Boon NA et al. Churchill Livingstone, Ediinburgh. P-381-388; 2002.

13. Haslett C, Chilvers ER, Corris PA. Respiratory disease. Davidson's Principles and Practice of Medicine, 19th edi. London: Churchill Livingstone. 2002:483-574.

14. Sawin CT, Castelli WP, Hershman JM, McNamara P, Bacharach P. The aging thyroid: thyroid deficiency in the Framingham study. Archives of Internal Medicine. 1985 Aug 1; 145(8):1386-8.

15. Kvetny J, Heldgaard PE, Bladbjerg EM, Gram J. Subclinical hypothyroidism is associated with a low-grade inflammation, increased triglyceride levels and predicts cardiovascular disease in males below 50 years. Clinical endocrinology. 2004 Aug 1; 61(2):232-8.

16. Jung CH, Sung KC, Shin HS, Rhee EJ, Lee WY, Kim BS, Kang JH, Kim H, Kim SW, Lee MH, Park JR. Thyroid dysfunction and their relation to cardiovascular risk factors such as lipid profile, hsCRP, and waist hip ratio in Korea. The Korean journal of internal medicine. 2003 Sep; 18(3):146.

17. Skanse B. On the difference in serum cholesterol between hypothyroidism of pituitary and of thyroid origin. In: Pitt-Rivers R, Green R, Tata JR, McCartney W, Taylor S, Pochin EE, Trotter WR (eds). The fourth international goitre conference. London: Pergaman Press, 1961:108-118.

18. Keyes WG, Heimberg M. Influence of thyroid status on lipid metabolism in the perfused rat liver. Journal of Clinical Investigation. 1979 Jul; 64(1):182.

19. Laker ME, Mayes PA. Effect of hyperthyroidism and hypothyroidism and carbohydrate metabolism of the perfused rat. Biochem J. 1981; 96:247-55.

20. Abrams JJ, Grundy SM. Cholesterol metabolism in hypothyroidism and hyperthyroidism in man. Journal of lipid research. 1981 Feb 1; 22(2):323-38.

21. Michalopoulou G, Alevizaki M, Piperingos G, Mitsibounas D, Mantzos E, Adamopoulos P, Koutras DA. High serum cholesterol levels in persons with high-normal TSH levels: should one extend the definition of subclinical hypothyroidism. European Journal of Endocrinology. 1998 Feb 1; 138(2):141-5.

22. Diekman T, Lansberg PJ, Kastelein JJ, Wiersinga WM. Prevalence and correction of hypothyroidism in a large cohort of patients referred for dyslipidemia. Archives of internal medicine. 1995 Jul 24; 155(14):1490-5.

23. Olukoga AO, Crowley VE, Lawal A, Weinkove C. Hyperlipidaemia and hypothyroidism Screen patients for hypothyroidism before treatment. BMJ. 1994 Apr 2; 308(6933):918.

24. Franco M, Castro G, Romero L, Regalado JC, Huesca-Gómez C, Ramírez S, Peréz-Méndez O, Medina A, Posadas-Romero C, Montaño LF. Decreased activity of lecithin: cholesterol acyltransferase and hepatic lipase in chronic hypothyroid rats: Implications for reverse cholesterol transport. InVascular Biochemistry 2003 (pp. 51-56). Springer, Boston, MA.

25. Dullaart RP, Hoogenberg K, Groener JE, Dikkescheo L, Erkelens DW, Doorenbos H. The activity of cholesteryl ester transfer protein is decreased in hypothyroidism: a possible contribution to alterations in high-density lipoproteins. European journal of clinical investigation. 1990 Dec 1; 20(6):581-7.

26. Lam KS, Chan MK, Yeung RT. High-density lipoprotein cholesterol, hepatic lipase and lipoprotein lipase activities in thyroid dysfunction—effects of treatment. QJM: An International Journal of Medicine. 1986 May 1; 59(2):513-21.

27.Perumal SS, Prasad S, Surapaneni KM, Joshi A. Health information-seeking behavior among hypothyroid patients at Saveetha Medical College and Hospital. Ethiopian journal of health sciences. 2015; 25(2):147-54.

28.Subramanium P1, Jagannathan N.Oral Manifestations of Hypothyroidism. Journal of lipid research. 1981 Feb 1; 22(2):323-38.

29.A.Trishala, Dr.V.Vishnupriyaand gayathiri r.Comparative assessment of lipid profile in pre and post-menopausal women in tuticorin district – a pilot. Journal of Clinical Investigation. 1979 Jul; 64(1):182.

30. Sundaravadanan BS, Sudarshan PB. Analysis of pattern of thyroid disorders in a tertiary care hospital and evaluating the accuracy of preoperative fine needle aspiration with postoperative histopathological examination. International Surgery Journal. 2017 Mar 25; 4(4):1267-71.

Measurement of Lateral Pterygoid Plates in Skulls

Nivedha Srinivasan[1], Saravana Kumar[2], Niveda S[3]
Graduate student[1,] Senior Lecturer[2], Department of Anatomy,
Tutor[3], Department of Public Health Dentistry
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Aim : The aim of the study is to evaluate a potential relationship between pterygoid plate symmetry and the occurrence of TMD by comparing LPP measurements.

Materials and Method : Fifty skulls were examined and the lateral pterygoid plate was measured with the help of a vernier caliper.

Result : The mean value of the left lateral pterygoid plate is 1.946 cm and the right lateral pterygoid plate is 1.969 cm. the difference between these two plates is +\-0.023cm.

Conclusion: The lateral pterygoid plate is thought to play an important in the control of jaw movements because of the presence of abnormal patterns of lateral pterygoid muscle activity in some TMD patients. The average value of the length of the LPP was measured in all instancesfrom the point of junction between lamina and the greater wing of sphenoid to the midpoint of the lower margin of the lamina. The lateral pterygoid muscle consists of a superior head and lower or inferior head. Classically, the inferior head is said to be active during jaw opening, jaw protrusion and contra lateral jaw movements and arises from the lateral surface of the LPP.

Keywords: *Lateral pterygoid plate, Skulls, Jaw movements, Muscle, Lamina*

Introduction

A broad thin plate that forms the lateral part of the pterygoid processes and gives attachment to the lateral pterygoid muscle on its lateral surface and to the medial pterygoid muscle on its medial surface, hence called as lateral pterygoid plate. It has two heads; superiorhead and inferior head.

Superior head is originated from the infra temporal crest of the greater wing of phenolic bone. The inferior head is originated from the lateral surface of lateral pterygoid plate. The two heads unite to form a strong tendon. They are inserted into the neck of the mandible, articulate disc of temporal mandibular joint (TMJ) and the capsule of the TMJ. They are supplied by the mandibular nerve where the nerve enters the deep surface of the muscle. Its action is to depress the mandible and draws the articulation disc forwards. It protracts the lower jaw. It is the opener of the jaw [1].

Its relations are; it is superficially related to the masseter muscle, maxillary artery and temporalismuscle. Its deep relations are sphenoid mandibular ligament, mandibular nerve, chorda tympani nerve and medial pterygoid muscle [2].Its upper border is related to deep temporal nerves, deep temporal vessels and nerve and artery to masseter. Its lower border is related to lingual nerve of inferior alveolar nerve, inferior alveolar artery, and sphenomandibular ligament.

Between the two heads the following structures are situated. Buccal branch of mandibular nerve, buccal branch of maxillary artery, pterygoid plexus of veins are found on the outer surface of the muscle, and the maxillary artery[3].

Temporomandibular disorder (TMD) is a general term that includes a series of clinical alterations involving the Temporomandibular joint (TMJ) and structures related to it, such as the masticatory muscles. These muscles are frequently related to TMD since they receive an overload, usually caused by parafunctional habits and occlusal disorders, so that clinical manifestation of this condition is translated into pain [4-7]. The lateral pterygoid muscle (LPM), one of the most important muscles in the physiology of mastication, is directly connected to the

TMJ and participates in the movements of opening, closing, laterality and mandibular protrusion. It is deeply localized to both the mandibular branch and temporal muscle, which makes its evaluation difficult [8]. Palpation of LPM is not possible owing to the topographic localization of its lower head. [9,10]. A small portion of the upper fibres of the LPM are inserted into both the articular capsule and articular disc of the TMJ, giving stability to the set and directly influencing articular disc position during mandibular movements [11,12]. MRI is a powerful tool for evaluation of changes in the articular structure and masticatory muscles,[13,14] but there are few MRI studies dedicated to the evaluation of changes in the LPM, such as hypertrophy, atrophy or muscle contracture associated, or not, with articular disc displacement[15]. MR images obtained in the oblique sagittal planes allow for a detailed observation of the LPM, articular disc, and mandibular head and fossa in the same image and beside a complete observation of, and the relationship between, the structures [16, 17].MR imaging in adult patients before and after mouth opening has suggested that volume changes occur in masticatory muscles between mouth opening and closing and that there is a difference between the changes in muscle volume. Although only small changes were observed in the masseter and medial pterygoid muscles, LPM volume significantly decreased during mouth opening. [18] Understanding the changes in the LPM images will help the clinician to understand better the clinical symptoms and allow for more accurate diagnosis of TMD, and therefore plan more effective treatment.

Materials and Method

Fifty skullswere examined and the lateral pterygoid plate was measured with the help of a vernier caliper.Fifty skulls from the anatomy department of The Saveetha Dental College and Hospital were taken to measure the right and left lateral pterygoid plates with the help of the Vernier caliper (Fig1).The measurement taken were evaluated. The mean value of the left and right pterygoid was calculated (Table 1)

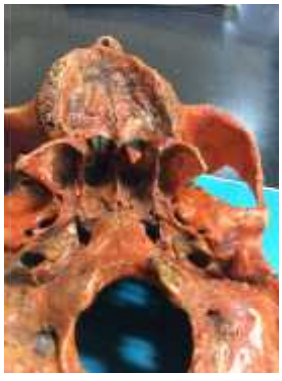


Figure 1: Shows Lateral pterygoid plate

Table 1: Shows the measurement of right and left Lateral pterygoid plate

S.No	MESUREMENT(Cms)	
	LEFT	***RIGHT***
1.	1.3	1.3
2.	1.9	2
3.	2.1	2.1
4.	1.9	1.8
5.	1.9	2
6.	1.8	1.8
7.	1.7	1.8
8.	2.3	2.2
9.	1.9	1.9
10.	2	2
11.	2.1	2.1
12.	2	1.9
13.	2.4	2
14.	2	2.1
15.	2.1	1.9
16.	1.5	2.4
17.	2.2	2.1
18.	2.2	2.1
19.	1.8	1.5
20.	2	2
21.	1.9	1.9
22.	2	1.7

23.	2	1.7
24.	2	2.1
25.	1.9	1.9
26.	2	1.7
27.	1.8	2
28.	2.1	2.2
29.	2	2.2
30.	1.9	1.9
31.	1.7	1.9
32.	2	2.1
33.	1.8	1.7
34.	1.9	1.7
35.	1.8	1.9
36.	2	2.1
37.	1.6	2.1
38.	2	2
39.	1.8	2.2
40.	2	2
41.	2.9	1.9
42.	2	2.5
43.	2	1.5
44.	2	2
45.	1.6	1.9
46.	2	2
47.	1.6	1.9
48.	2	2
49.	1.9	2.1
50.	2	2
OVERALL AVERAGE	1.946	1.96938

Result

The mean value of the left lateral pterygoid plate is 1.946 cm and the right lateral pterygoid plate is 1.969 cm. the difference between these two plates is +\-0.023cm. [Table-1]

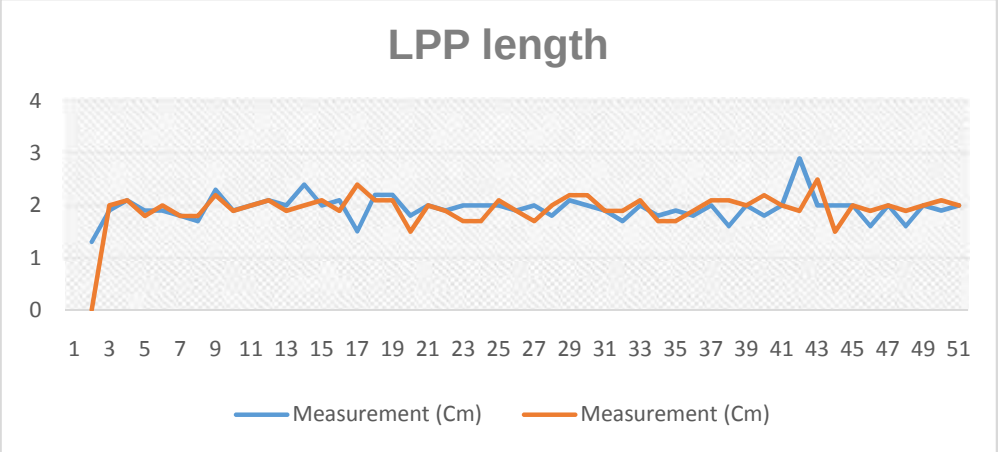


Figure 3: Graph showing measurement of left and right lateral pterygoid plate

Discussion

A broad thin plate that forms the lateral part of the pterygoid process, leads to an attachment to the lateral and medial surface known as the lateral pterygoid plate. According to L.Chris D Naidoo, in his article he had mentioned that the upper head of the lateral pterygoid muscle was approximately 33.3 mm in length and was significantly longer in males. There were no age-related differences. Approximately 29.5% of the fibers of this muscle were inserted into the meniscus of the temporomandibular joint [1].

One such study, conducted by Stratmann et al. in 2000, found that ". . . even after maximal impression of the buccal mucosa, a considerable residual distance remains between the ILP muscle and the buccinators fascia indented by the tip of the palpating forefinger."8 A systematic review carried out by Turp and Minagi (2001) also concluded that: "in the overwhelming majority of cases, palpation of the inferior lateral pterygoid muscle is impossible due to anatomical reasons."The study notes that these reasons include:

- Restricted space that does not allow access of the finger (a portion of this potential space is narrowed by the deep tendon of the temporal muscle)[3].

- Resistance by the tendinous origin of the buccinator muscle (pterygomandibular raphe)[4].
- Resilience of the buccal mucosa due to its close connection to the alveolar gingival that is firmly attached to the maxillary alveolar process [5].
- An obstructing deep origin of the medial pterygoid, which rests on the inferior face of the inferior head of the ILPM [6].

This may come as a surprise to practitioners dealing with TMDs in their practice, considering that epidemiological studies have shown that patients with these disorders frequently report the most tenderness to palpation of what was thought to be the “lateral pterygoid” muscles in this region [7-8]. TMJs had atrophy in both superior and inferior heads of the lateral pterygoid (SLPM &ILPM). However, atrophy was not present only in the ILPM. It has been concluded that since the SLPM only attached to the disc, the disc may displace interiorly very easily. Therefore, this situation will reduce the function of the SLPM. Reduced muscle function may cause muscle atrophy However, one must also consider that the same studies have shown that among individuals not suffering from or facial pain, the “lateral pterygoid” area is also considerably more tender than other masticatory muscles. If the LPM cannot be implicated for this tenderness, then, one must wonder what the actual cause of the pain on palpation is, if this muscle is, in fact, not accessible [9-12].

In another article where the relationship between the lateral pterygoid muscle and the meniscus was examinedand it was found out that 27.5% the upper head for the lateral pterygoid was attached to the condyle and 7.5% of other attachments of lateral pterygoid muscle to the meniscus. This study confirms that the lateral pterygoid muscle has variable attachments [13].
Macroscopically when the measurements of the upper and lower head of the lateral pterygoid plate were taken on 20 specimens, 13 specimens had both the heads of lateral pterygoid muscle (LPM) were present, whereas the rest7 had a separate fascia [14]. And according to another article, the LPM dystonia, a new technique is characterized by mandibular displacement towards the opposite side of the affected muscle. It may be associated with functional disorders affecting speech, swallowing, chewing and facial symmetry [15-18]. TMJs had atrophy in both superior and inferior heads of the lateral pterygoid (SLPM &ILPM). However, atrophy was not present only in the ILPM. It has been concluded that since the SLPM only attached to the disc, the disc

may displace interiorly very easily.

Therefore, this situation will reduce the function of the SLPM. Reduced muscle function may cause muscle atrophy [19, 20]. Statistical analysis, sensitivity, specificity, and negative and positive predictive values of the palpation technique were calculated and in 86 of 106 dissected specimens; a superficial fascicle of the medial pterygoid muscle was in direct proximity to the ILP muscle [21]. In these cases, a residual distance of 7.8 ± 3.2 mm remained between the ILP muscle and buccinators fascia indented by the tip of the examiner's finger. In 10 of 20 specimens with an absent superficial fascicle, the finger was able to reach the ILP muscle [22]. Hence it was concluded that it is recommended that the ILP muscle palpation technique should no longer be considered as a standard clinical procedure because it is nearly impossible to palpate the ILP muscle anatomically [23]. Since the morphometric analysis of the lateral pterygoid plate weren't mentioned in any of the articles hence this article has the measurement of the right and left lateral pterygoid plates.

Conclusion

The dimensions of lateral pterygoid plate differ on both sides and also from person to person (Table 1). By measuring its length, it allows us to understand the clinical significance like; difference in jaw movement and difference in the force applied since lateral pterygoid muscle is attached to it.

Corresponding Author

S.Niveda

Tutor,Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Email: drnivedasubash@gmail.com

Telephone Number - +91 9952979464

Referenceto be corrected according to Vancouver style

1. Naidoo LC, Juniper RP. Morphometric analysis of the insertion of the upper head of the lateral pterygoid muscle. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics. 1997 Apr 1;83(4):441-6.
2. Hernando MF, Cerezal L, Pérez-Carro L, Abascal F, Canga A. Deep gluteal syndrome: anatomy, imaging, and management of sciatic nerve entrapments in the subgluteal space. Skeletal radiology. 2015 Jul 1;44(7):919-34.
3. Taskaya-Yılmaz N, Ceylan G, Incesu L, Muglali M. A possible etiology of the internal derangement of the temporomandibular joint based on the MRI observations of the lateral pterygoid muscle. Surgical and Radiologic Anatomy. 2005 Mar 1;27(1):19-24.
4. Stratmann U, Mokrys K, Meyer U, Kleinheinz J, Joos U, Dirksen D, Bollmann F. Clinical anatomy and palpability of the inferior lateral pterygoid muscle. The Journal of prosthetic dentistry. 2000 May 1;83(5):548-54.
5. Davies JC, Charles M, Cantelmi D, Liebgott B, Ravichandiran M, Ravichandiran K, Agur AM. Lateral pterygoid muscle: A three-dimensional analysis of neuromuscular partitioning. Clinical Anatomy. 2012 Jul;25(5):576-83.
6. International Journal of Oral and Maxillofacial Surgery. MRI evidence of lateral pterygoid muscle palpation; P. BarriereJ.-C. LutzA. ZamanianA. WilkS. RhiemF. VeillonJ.-L. Kahn.October 2009; 38(10)
7. Journal of Oral and Maxillofacial Surgery. Insertions of the lateral pterygoid muscle: An anatomic study of the human Temporomandibular joint; Pierre Carpentier DEOJean-Pierre Yung DCDRichard Marguelles-Bonnet DSOMarc Meu. June 1988; 46(6):477–482.
8. Unnamalai and shonali International journal of Pharm and bio sciences, mandibular fractures and reconstruction, Saveetha dental college,august2006; 2(2):55 - 61
9. Kiruthika ptturajal, Karpagam Krishnamoorthy, treatment and management of temporal mandibular joint ankylosis, research and journal of pharmacy and technology2016; 9(10):1668-1670.
10. Keerthana Balaji Ms Thenmozhi, a study of occurrence and morphometric analysis of foramen of civinini in south indian dry skulls, Int Jr of Res | 2016; 2(4):34-37
11. Turp JC, Minagi S. Palpation of the lateral pterygoid region in TMD—where is the evidence? J Dent2001; 29:475–483.

12. Murray GM, Phanachet I, Ushida S, Whittle T. The human lateral pterygoid muscle: a review of some experimental aspects and possible clinical relevance. Aust Dent 2004; 49:2–8
13. Wang MQ, Yan CY, Yuan YP. Is the superior belly of the lateral pterygoid primarily a stabilizer? An EMG study. J Oral Rehabil 2001; 28:507–509
14. Taskaya-Yılmaz N, Ceylan G, Incesu L, Muglali M. A possible etiology of the internal derangement of the Temporomandibular joint based on the MRI observations of the lateral pterygoid muscle. Surg Radiol Anat 2005; 27:19–24
15. Westesson PL. Reliability and validity of imaging diagnosis of temporomandibular joint disorder. Adv Dent Res 1993; 17:331–335
16. Schellhas KP. MR imaging of muscles of mastication. AJR Am J Roentgenol 1989; 153:847–855
17. Yang X, Pernu H, Pyhtinen J, Tiilikainen PA, Oikarinen KS, Raustia AM. MR abnormalities of the lateral pterygoid muscle in patients with nonreducing disk displacement of the TMJ. Cranio 2002; 20:209–221
18. Musgrave MT, Westesson PL, Tallent RH, Manzione JV, Katzberg RW. Improved magnetic resonance imaging of the Temporomandibular joint by oblique scanning planes. Oral Surg Oral Med Oral Pathol. 1991; 71:525–528
19. Quemar JC, Ravalec X, Akoka S. Parasagittal magnetic resonance imaging of the lateral pterygoid muscle: a preliminary study. J Orofac Pain 1993; 7:169–174
20. Goto TK, Tokumori K, Nakamura Y, Yahagi M, Yuasak, Okamura K. Volume changes in human masticatory muscles between jaw closing and opening. J Dent Res 2002; 81:428–432

Mental Foramen: An Eagle Eye

S.Thaminee[1], Saravana Kumar[2], V.Ashok[3],

Graduate Student[1], Senior Lecturer[2], Department of Anatomy[2],Reader[3], Department of Prosthodontics[3],

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The mental foramen is a strategically important landmark during osteotomy procedures in implantology. Its location and the possibility that an anterior loop of the mental nerve may be present mesial to the mental foramen needs to be considered before implant surgery to avoid mental nerve injury. To provide anatomical information on the position, morphological variations and incidence of mental foramen and accessory mental foramen as they are important for dental surgeons, anaesthetists in nerve blocking and surgical procedures to avoid injury to neurovascular bundle in mental foramen area.

Material and Method: The present study was conducted using 120 dried human mandibles of both sexes. Size, shape and position of mental foramen and accessory mental foramen were determined using digital vernier calliper.

Results: Mental foramen was present in all observed mandibles and it was bilateral in position. Accessory mental foramen was present in 8 mandibles and was unilateral in position.

Conclusion: The knowledge about variation in size, shape and position of mental foramen and presence of accessory mental foramen may be helpful to the dental surgeons to achieve fullanesthesia and it's a guide for the implantologist to proceed with placing implant without damaging the nerve.

Key Words:*Mental foramen (MF), accessory mental foramen (AMF), mandible, premolar tooth.*

Introduction

The mental foramen is defined as the entire funnel-like opening in the lateral surface of the mandible at the terminus of the mental canal. Mental nerves and vessels passes through this foramen. This is contained entirely within the buccal cortical plate of bone.The mental foramen is the opening of mental canal, a branch of mandibular canal.Wall of the mental foramen is made up of cortical bone. The density of the foramen varies, as does the shape and definition of its border(1). Knowledge of the position of the mental foramen is important both when administering regional anesthesia, performing periapical surgery, dental implant surgery and endodontic treatment in mandible(2).

Foramen may occasionally be mis-diagnosed as a radiolucent lesion in the apical area of the mandibular premolar teeth.It also aids in interpreting anatomical landmarks in oral pathology andforensics(3).Mental foramen is a small foramen situated in anterolateral aspect of the body of the mandible. Normally, mental foramen is located below the interval between the premolars. It transmits mental nerve, artery and vein. Mental nerve is a branch of inferior alveolar nerve which supplies sensation to lower lip and the labial mucosa and lower canines and premolars. The most useful injection for anaesthetizing the mandibular teeth is the inferior alveolar nerve block(4).

To anaesthetise the anterior teeth, including the premolars and canines, it is possible to avoid giving inferior alveolar nerve block by injecting anaestheticsolution adjacent to the mental foramen. So,the study of position and morphological variation of mental foramen is very important because it will be helpful to localize the important neurovascular bundle passing through the mental foramen. Any foramen in addition to mental foramen in the body of the mandible is known as accessory mental foramen. Accessory mental foramen transmits the accessory branch of mental nerve. So, the knowledge of its position and incidence is helpful to dental surgeons to achieve complete anaesthesia because if this nerve is not blocked, anaesthesia will be incomplete(5).

Mental foramen is located in the face and in the body of the mandible at an equal distance from the superior and the inferior border and it is located below the interval between the premolars mental nerves and vessels passes through it. Accessory mental foramen is due to the branching of

mental nerve before passing through the mental foramen.Hence its shape,size and verification of its existence would prevent accessory nerve injury during periapical surgery. So the knowledge of its position and incidence is helpful to dental surgeons to achieve complete anesthesia because of this nerve is not blocked anesthesia will be incomplete. Mental nerve is the branch of the inferior alveolar nerve which supplies the sensation to the lower lip and the labial mucosa and the lower canines and the premolars. The most useful injection for anesthetizing the mandibular teeth is the inferior alveolar nerve block(4,5)

Mental foramen is also lie between the apices of the lower premolar,below the apex of the second premolar. Any foramen if addition to mental foramen is known as the accessary mental foramen in the body of the mandible (6). Variations like multiple or absent foramina are often encountered these variations in the position and the number of mental foramen have been reported in the pattern of their occurrence.Recent advancement in the clinical dentistry have increased the possibility of the procedures in the mental region and the detailed knowledge of the mental foramen and it may not only aid in the prevention of post-surgical neuromuscular complications and morbidity; but also hold the potential of contributing as the identification of maxillofacial anthropologic characteristic feature of different populations.

Human mandibles with over one mental foramen (MF) on one aspect do not seem to be such a rare discovery. Depending on the comparative size of the multiple MF traced on one side of a mandible, only one of them is considered as the main MF, while the others are regarded as accessory mental foramina (AMF). In order to distinguish an AMF from common buccal foramina without continuity with the mandibular canal, which are regarded as nutrient foramina, an AMF is defined as a foramen of an accessory bony canal originating from the mandibular canal (AMF). This foramen transmits the accessory branch of mental nerve. The knowledge of this foramen is essential to achieve complete anaesthesia(6,7).

The mental foramen (MF) is present on the anterolateral aspect on the body of the mandible. It transmits mental nerve and vessels. Mental nerve which is a branch of the inferior alveolar nerve supplies sensation to lower lip, labial mucosa, lower canines and premolars.Anaesthetising the anterior teeth together with premolars and avoiding inferior alveolar nerve block is feasible by

injecting anesthetic solution adjacent to the mental foramen (7).

Figure 1: Mental Foramen

Size of Mental Foramen

The average size of the foramen is 4.6 mm horizontally and 3.4 mm vertically on the lateral surface of the mandible.(6) The foramen is usually larger on the left side of the mandible. Based on its radiographic appearance, the mental foramen has been classified by Yosue and Brooks (2) into

four types:

Type I: mental canal is continuous with the mandibular canal

Type II: the foramen is distinctly separated from the mandibular canal

Type III: diffuse with a distinct border of the foramen

Type IV: unidentified group

Location

Anatomical variations occur concerning the mental foramen's location17,18. It is usually found more coronal than the mandibular canal.19,20Agthong et al.21 indicated, the foramen was 28 mm from the midline of the mandible and 14 to 15 mm from the inferior border of the mandible.(7) Similarly, Neiva et al.14 reported the foramen was 27.6 mm (range: 22 to 31 mm) from the midline and 12 mm (range: 9 to 15 mm) from the most apical portion of the lower

cortex of the mandible. Other
authors (8) commented the foramen was usually found halfway between the crest of bone and the inferior border of the mandible. However, this finding could be influenced by the amount of crestal bone loss.

Position

The positionof the mental foramen with respect to the teeth of the lower jaw
were classified according to the method ofTebo and Telford, 1950.

I. [Table/Fig-1]: Position of Mental Foramen in relation to teeth (Teboand Telford Classification)

1-Foramen lying on a longitudinal axis of passing between the canine and first premolar.
2-Foramen lying on the longitudinal axis of the first premolar.
3-Foramen lying on the longitudinal axis passing between first and second premolar
4-Foramen lying on the longitudinal axis of the second premolar
5-Foramen lying on the longitudinal axis passing between the secondpremolar and first molar.
6-Foramen lying on the longitudinal axis of first molar.The foramen which lay on a longitudinal axis which passed between the canine and the first premolar.

Detection of the Mental Foramen on Radiographs

Panoramic Films Radiographic assessment of the mental foramen must be interpreted cautiously. Jacobs et al.36 reported the foramen was detected on 94% (N = 545) of panoramic radiographs, but clear visibility was only attained 49% of the time. Similarly, Yosue and Brooks15 noticed the foramen on 87.5% (N = 297) of panoramic radiographs, and it was distinct 64% of the time. In another investigation in which four skulls were radiographed, (mean: 0.2 mm; range: 0.0 to 0.5 mm). It can be concluded that neither periapical nor panoramic films precisely portray the amount of bone coronal to the mental foramen.(9)

Ct Scans

CT scans are more accurate than conventional radiographs. Nevertheless, conventional radiographs can usually be used if potential radiographic distortions are taken into account(10). However, if it is difficult to locate the inferior alveolar canal or the mental foramen,

consideration should be given to obtaining a CT scan.

Conclusion

The anatomical variability of the position of the mental foramen should always be considered when performing periodontal or endodontic surgery in the area from canine to root of first molar tooth. The knowledge about variability in position of mental foramen and presence of accessory mental foramen is important in order to avoid nerve damage in connection with surgical procedure and to achieve complete effect ofanesthesia after mental nerve block.

Corresponding Address

V.Ashok,
Reader, Department of Prosthodontics,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India
162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India
Email:ashokprostho@gmail.com
Mobile Number: +91 962422299

References

1. Phillips JL, Weller RN, KulildJC.The mental foramen: Radiographic position in relation to the mandibular second premolar. *J Endod* 1992;18(2): 271-274.
2. Yosue T, Brooks SL. The appearance of mental foramina on panoramic radiographs. II. Experimental evaluation. *Oral Surg Oral Med Oral Pathol* 1989;68(57): 488-492.
3. Jassser NM, Nwoku AL. Radiographic study of the mental foramen in a selected Saudi population. *Dentomaxillofacial Radiol* 1995;27(7):341-343.
4. Fishel D, Buchner A, Hershkowith A, Kaffe.Roentgenologic study of the mental foramen. *Oral Surg Oral Med Oral Pathol*1976;41(5):682–686.
5. Agarwal DR, Gupta SB.Morphometric analysis of mental foramen in human mandibles of South Gujarat people .*J Sci Res*2011; 4(18): 15-18.
6. Sawyer DR, Kiely ML, Pyle MA. The frequency of accessory mental foramina in four ethnic groups. *Arch Oral Biol*1998; 43(4): 417-20.

7. Cag Irankaya LB, Kansu H. An accessory mental foramen: a case report. *Contemp Dent Pract*2008; 9(28): 98-104.
8. Naitoh M, Hiraiwa Y, Aimiya H, Gotoh K, ArijiE.Accessory mental foramen assessment using cone-beam com- puted. tomography. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2009;107(2):289–294.
9. Nandakumar,Dr.Thenmozhli.A Study of Incidence of Accessary Mental Foramen in South Indian Mandibles.Saveetha Dental college and hospital,Saveetha University.*JPSR*2015;7(7):445-447.
10. KetakiKamath.M, SharayuShanmathi.K.Studyof accessory mental foramen in dried mandibles.Saveetha Dental college and hospital,SaveethaUniversity.*IJSDR*2017;2(11): 2455-263.
11. Phillips JL, Weller RN, KulildJC.The mental foramen: Radiographic position in relation to the mandibular second premolar. *J Endod* 1992;18(2): 271-274.
12. Yosue T, Brooks SL. The appearance of mental foramina on panoramic radiographs. II. Experimental evaluation. *Oral Surg Oral Med Oral Pathol* 1989;68(57): 488-492.
13. Jassser NM, Nwoku AL. Radiographic study of the mental foramen in a selected Saudi population. *Dentomaxillofacial Radiol* 1995;27(7):341-343.
14. Fishel D, Buchner A, Hershkowith A, Kaffe.Roentgenologic study of the mental foramen. *Oral Surg Oral Med Oral Pathol*1976;41(5):682–686.
15. Agarwal DR, Gupta SB.Morphometric analysis of mental foramen in human mandibles of South Gujarat people .*J Sci Res*2011; 4(18): 15-18.
16. Sawyer DR, Kiely ML, Pyle MA. The frequency of accessory mental foramina in four ethnic groups. *Arch Oral Biol*1998; 43(4): 417-20.
17. Cag Irankaya LB, Kansu H. An accessory mental foramen: a case report. *Contemp Dent Pract*2008; 9(28): 98-104.
18. Naitoh M, Hiraiwa Y, Aimiya H, Gotoh K, ArijiE.Accessory mental foramen assessment using cone-beam com- puted. tomography. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 2009;107(2):289–294.
19. Nandakumar,Dr.Thenmozhli.A Study of Incidence of Accessary Mental Foramen in South Indian Mandibles.Saveetha Dental college and hospital,Saveetha University.*JPSR*2015;7(7):445-447.

20. KetakiKamath.M, SharayuShanmathi.K.Study of accessory mental foramen in dried mandibles.Saveetha Dental college and hospital,SaveethaUniversity.*IJSDR*2017;2(11): 2455-263.

Morphometric analysis of Foramen Magnum for Sex determination

Jayajanani.S [1], Jayashri.P [2]

Graduate Student [1],Senior Lecturer [2], Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Identification of human skeletal remains is the most important task for a forensic anthropologist during forensic examinations. The need for methods to estimate sex from cranial fragments becomes apparent when only a part of skull is brought for identification. In forensic or archaeological context estimation of sex is a very important step in the identification of any human skeletal remains discovered. Skull is one of the commonest parts of the skeleton used to determine the sex, and it is probably the second best region of the skeleton, next to the pelvis for this purpose. Foramen magnum is an important landmark of the skull base and is of particular interest for anthropology, anatomy, forensic medicine, and other medical field. The foramen magnum is an important anatomical opening in the base of the skull through which the posterior cranial fossa communicates with the vertebral canal. It is also related to a number of pathological conditions including Chiari malformations, various tumours, and occipital dysplasias. The aim of the review was to analyse the morphology of the foramen magnum in adult individuals in relation to sex.

Key Words: *-Foramen Magnum, Morphometric analysis, Anthropology, Sex determination, Population studies.*

Introduction

Identification of human skeletal remains is the most important task for a forensic anthropologist during forensic examinations. The need for methods to estimate sex from cranial fragments becomes apparent when only a part of skull is brought for identification.Sex determination from skull morphology is important in medico-legal cases. In most of the forensic studies the skeleton will be incomplete and makes gender identification difficult. Sex determination is one of the first steps in the identification of any human skeletal remains discovered in a forensic or archaeological context [1]. The most accurate results are obtained when the entire skeleton is available for study; however, skeletal material derived from forensic and archaeological contexts is rarely complete and undamaged. Therefore, it is important to establish methods for determining sex from skeletal elements likely to survive and be recovered [2].

Foramen Magnum

The foramen magnum is a large opening in the base of skull, it is oval, wider behind with greatest diameter being antero-posterior. It contains the lower end of the medulla oblongata, the vertebral arteries and spinal accessory nerves [3]. The dimensions of the foramen magnum are clinically important because the above mentioned vital structures passing through it may endure compression such as in cases of foramen magnum herniation, foramen magnum meningiomas and foramen magnum achondroplasia [4]. The knowledge of foramen magnum diameters is needed to determine some malformations such as Arnold Chiari syndrome, which shows expansion of transverse diameter [5]. The dimensions of the foramen magnum are important prior to the cutting off of the foramen magnum lesions or posterior cranial fossa lesions, because more the antero-posterior diameter, greater is the contra lateral exposure [4]. The diameters and area of the foramen magnum are greater in males than in females, hence its dimensions can be used to determine sex in the medicolegal conditions, especially in the following circumstances, such as explosions, aircraft accidents and war fare injuries [5, 6]. Foramen magnum is about 3cm wide by 3.5cm anteroposteriorly [7, 8]. It is located midway between and on a level with mastoid processes. The foramen magnum is surrounded by different parts of the occipital bone, squamous part lies behind and above, basilar part in front and a condylar part on either side [9, 10]. On each side its antero-lateral margin is encroached by occipital condyles, hence the foramen magnum is narrow anteriorly.

The anterior edge of the foramen magnum is slightly thickened and lies between the anterior ends of the condyles. The posterior half of the foramen magnum is thin and semicircular. Upper ends of anterior and posterior atlanto-occipital membranes are attached to the anterior and posterior margins of the foramen magnum respectively, and their lower ends are attached to the superior surface of anterior and posterior arches of the atlas respectively.[6] The foramen magnum is a wide communication between posterior cranial fossa and the vertebral canal. The narrow anterior part of the foramen magnum has apical ligament of dens, upper fasciculus of the cruciate ligament and membranatectoria, both are attached to the upper surface of basioccipital bone infront of the foramen magnum. Its wide posterior part contains the medulla oblongata and its meninges. In subarachnoid space spinal rami of the accessory nerve and vertebral arteries, with their sympathetic plexus, ascend into the cranium; the posterior spinal arteries descend posterolateral to the brain stem, where as anterior spinal artery descends anteromedian to the brain stem. The cerebellar tonsils may project into the foramen magnum [9].

Studies on Foramen Magnum for Sex Determination

The most conspicuous feature of the occipital bone is the large foramen magnum through which the cranial cavity communicates with the vertebral canal. The anterior border of the foramen magnum is formed by basilar process of the occipital bone, the lateral border by the left and right ex-occipitals and posterior border is formed by the supraorbital part of the occipital bone [10]. A pair of large smooth protuberances, the occipital condyles lies lateral to each side of the foramen magnum. Each condyle articulates with the corresponding superior articular facet on the atlas vertebra to form an atlanto–occipital joint [11]. A number of studies has investigated the utility of this anatomical region for sex assessment employing morphometric traits using discriminant function analysis.Teixeria (1982) published an initial study on estimation of sex based on the size of the foramen magnum. His findings based on a small sample of 40 adult (20 males, 20 females) Brazilian skulls indicated that if the area of the foramen magnum was 963 mm2 or larger, it was a male skull and if it was 805 mm2or less it was female skull. In another study, Routal et al. (1984) found the dimensions of foramen magnum in Indian sample to be sexually dimorphic and reported up to 100% accuracy of correctly identifying sex using simple demarking points .Many other studies have been conducted on different populations with respect to sexual dimorphism in foramen magnum and occipital condyles using different statistical considerations [12,13,14,15,16,17,18].Gapert et al., (2009) used discriminant function analysis and

regression analysis on an eighteenth and nineteenth century British sample. The discriminant functions developed in the study predicted correct sex in 70.3% of all cases. Gruber et al., (2009) did not find any sexual dimorphism in the diameters of the foramen magnum in central European dry specimen dating from Pleistocene to modern times. The foramen magnum had low discriminatory power in sexual dimorphism in UNIFESP sample and accurately classified 66.5% skulls (Suazo et al., 2009). In French sample, Macaluso Jr., (2011) found a low level of sexual dimorphism in the cranial base and in this study maximum length of left occipital condyle and minimum distance from occipital condyles gave the best results with accuracy rate of 67.7%. A study on sexual dimorphism by RaghavendraBabu et al which was based on antero-posterior diameter, transverse diameter and area of foramen magnum in population of coastal Karnataka region revealed predictability of foramen magnum measurements in sexing the crania as 64.5% for transverse diameter and 86.5% for the antero-posterior diameter.

Foramen Magnum in Chinese Population

A metrical study of 84 mature male Chinese skulls conducted at University of Edinburgh, which showed mean foramen magnum length for Chinese type I skull of 35.71mm and breadth was 28.24mm, whereas Chinese type 2 crania had mean foramen magnum length of 35.21mm and breadth was 28.00 mm. The CT scan of foramen magnum of 200 children of all ages and 100 adults, showed an average foramen magnum length of 35mm in adults and that of children reached the adult size by 3-4 years of age [19]. In a presentation of Brazilian skulls, the area of foramen magnum was 673- 1195 mm2 for males and 591-961mm2 for females [20]. A study conducted at Govt. Medical College, Surat for dimensions of foramen magnum, study sample was composed of 141 adult skulls (104 males, 37 females) it showed following foramen magnum measurements. In males skulls anteroposterior diameter varied from 3 - 4.2 cm with an average of 3.55±0.28cm, transverse diameter was varied from 2.5-3.5cm with an average of 2.46±0.19cm, and area of foramen magnum was varied from 5.8-10.29cm2 with an average of 8.19±0.94 cm2. In female skulls the anteroposterior diameter was varied from 2.8- 3.5cm with an average of 3.20±0.28cm, transverse diameter was varied from 2.4- 3.2 cm with an average of 2.71±0.16cm and area of foramen magnum varied from 5.49-8.79 cm2 with an average of 7.71±0.90 cm2. The study showed male skulls anteroposterior diameter, transverse diameter and area of the foramen magnum were significantly higher than female skulls [21].

In a study computerized tomographic dimensions of the foramen magnum of 63 achondroplastic individuals were compared to standards established for nonachondroplastic individuals. The size of the foramen magnum in patients with achondroplasia was small at all ages, particularly in those with serious neurological problems [22]. In an anatomic metric study of foramen magnum of 100 male and 100 female skulls of normal white people of Spain showed a mean sagittal diameter of 36.2±0.3mm and the area of foramen magnum of 888.4±13.9mm2 in male skulls. In female skulls mean sagittal diameter was 34.3±0.4mm, transverse diameter of 29.6±0.3mm and the area of foramen magnum of 801±17.1 mm2. Foramen magnum dimension of male skulls were significantly higher than female skulls [23]. In a study conducted at St.John Medical College, Bangalore on 350 skulls (175 males, 175 females) for foramen magnum dimensions showed. In male skulls antero-posterior diameter of foramen magnum was varied from 2.8-4.1 cm with a mean of 3.42±0.24cm, transverse diameter was varied from 2.3-3.6cm with a mean of 2.85±0.23cm and the area of foramen magnum was varied from 5.6-11cm2 with a mean of 7.69cm2. In female skulls antero-posterior diameter of foramen magnum was varied form 2.2-3.3cm with a mean of 2.8±0.22cm and area of the foramen magnum was varied from 5.1-10 cm2 with a mean of 7.8±0.98cm2[24]. The fitted nonachondroplastic foramen magnum growth curves demonstrate that the maximum growth occurs in the first 18 months and slows thereafter. Indeed, the sagittal dimension almost doubles within the first 2 years, while the transverse dimension enlarges by half the original dimension. Growth of this area is essentially complete by 5 years. The achondroplastic foramen magnum is small at birth, and during the first year it has a very severely impaired rate of growth essentially in the transverse dimension. This markedly diminished growth results not only from abnormal enchondral bone growth but also because of abnormal placement and fusion of the synchondroses.

In a measurement of area of foramen magnum in 219 skeletons (170 males, 39 females) of Turkey, it was observed that mean area of the foramen magnum was significantly different (909.91±126.02 mm2 in males, 819.01±117. 24mm2 in females). Correlation coefficient between the area of the foramen magnum and sex was 0.27. The results confirmed that mean area of foramen magnum is lower in females than in males [21].

The occipital bone is frequently used in procedures that seek to determine the sex in forensics or anthropology. From qualitative point of view, the roughness of the nuchal lines and the

prominence of the external occipital protuberance are good indicators for the diagnosis of sex.

On the other hand, from a quantitative point of view, the indexes have been built from the dimensions of the occipital condyles and the foramen magnum, and various authors have reported its usefulness in determining the sex, particularly with incomplete skeleton or cranial bones fractured [25, 26, 27].Catalina-Herrera (1987) [28] indicated that the sagittal and transverse dimensions of the foramen magnum were significantly higher in men's skulls. Zaidi& Dayal (1988) [29] classified a sample of Indian skulls according to the shape and dimensions of the foramen magnum, reporting differences between the skulls of male and female, which was similar to the findings of Henríquez-Pino et al. (1995) [30] in the Brazilian skulls.

In another study, Günay&Altinkök (2000) [31] examined the usefulness of determining the dimensions of the fora- men magnum in the diagnosis of sex, and noted that the diameters were of some use, while the total area was not a good indicator. Uysal et al. (2005) [32] reported sexual dimorphism by analyzing the dimensions of the foramen magnum in 3D computed tomography with 81% accuracy in determining the sex, a level that was higher than that obtained by Gapert et al. (2009) [33] in a sample of British skulls from the eighteenth and nineteenth centuries.

Foramen Magnum in Chiari Malformation

Most of the modern articles that explain the foramen magnum morphology are related to the Chiari malformation, which is defined as ectopia of the cerebellar tonsil at least 5 mm below the foramen (Chiari malformation type I: CM-I) [34]. Less frequently such herniations are complicated with myelodysplasia (CM-II) and cervical encephalocele (CM-III), as well as abnormalities of the paraxial mesoderm and maldevelopment of the contents of the posterior cranial fossa or other diseases, e.g. hydrocephalus, tumours of the central nervous system, or Paget's disease. Generally, the size, in particular the transversal diameter and area of the foramen, is smaller in non-complicated CM-I, but significantly higher values for all its measurements were found in the case of CM-I complicated with tethered cord syndrome and for CM-II. In all of these cases the volume of the posterior cranial fossa was smaller than in healthy individuals. The correlation between foramen morphology and size of the posterior cranial fossa was also highlighted by Acer et al. It is worth mentioning that cranial constriction, spinal cord tethering, cranial setting, intracranial hypertension, and intraspinal

hypotension increase the risk of cerebellar tonsil herniation and abnormality of the foramen magnum [34].

The shape of the evaluated structure results from developmental changes that influence the morphology of the 4 principal parts of the occipital bone and the intra-occipital synchondroses that connect them. Based on previous observations [35,36], Richards and Jabbour [38] divided the proper foramen magnum as well as the entire surrounded hard (atlas, dens of axis) and soft elements (ligaments, membranes) into ventral and dorsal parts that comprise 2 functional matrices. The ventral one includes skeletomotor-related structures — the ventrolateral border of the foramen (extending to a line between the bilaterally located anterior intraoccipitalsynchondroses), anterior longitudinal ligament, ventral arch of the atlas, anterior atlanto-occipital membrane, apical ligament and dens of the axis, cruciform and alar ligaments, as well as the tectorial membrane which separates it from the dorsal matrix. All the listed structures are important for the proper movement and stabilisation of the skull. Unlike the ventral matrix, the dorsal matrix is less involved in head motions but transmits important neurovascular elements. It contains the posterior edge of the foramen magnum, spinal cord, meninges and subarachnoid space, posterior atlanto-occipital membrane, the dorsal arch of the axis, as well as the accessory spinal nerve and various vascular structures, as listed in the introduction. As was stressed above, any abnormal development of the listed structure may significantly change the morphology of the foramen. The most common type is the previously presented Chiari malformations [34]. However, a number of brain and meningeal tumours, as well as various occipital dysplasias particularly assimilation of the atlas with the occipital bone and irregular formation of the occipital condyles may also cause encroachment into the foramen [34, 37]

Ford reported that the length of the fora- men increases more rapidly during the prenatal period when compared to its breadth. However, recently published data [38] shows that the in utero elongation of the foramen magnum from the 7th gestational month until birth is secondary mainly to the increasing length of its dorsal unit, while the length of the ventral unit does not differ significantly.

Such growth changes reverse in the early postnatal period (0–6 months). The dynamics of the process slow down after 1 year of age, when the dorsal unit reaches mature size but the length of the ventral unit continues to increase slowly until both anterior intraoccipitalsynchondroses become fully calcified (< 8.2 years). The postnatal period is also characterised by growth where changes in breadth dominate over changes in length. The breadth-dominated growth pattern is even longer and continues un- til about 9.5–10.0 years of age. Secondary to those

changes, the foramen index also changes from 62.7– –65.7% in the very early stages of the prenatal period to 69.8–71.0% at 0.75–2.5 years, and later be- comes more stable at 62.7–79.9%, with a mean value 78.88 ± 4.25% for adults (> 18 years). Un- like in the present results, in adult native South Africans, a high predilection of dolichotrematous and low frequency of meso- and brachytrematous foramina was found by De Villers. However, similar to our data, a much greater range of the index value (71.0–111.0%) was presented by other authors. All the authors note an oval or oval-to-round form of the foramen magnum as the dominant one for modern adult Homo sapiens. However, the shape may change during ontogeny. Richards and Jabbour [42] found 8 developmental variationsthat were noted in 470 examined skulls during various stage of human life from the 7th gestational month to 21 years of age. During the foetal period, the following forms were seen: a bi-rounded oval, a ventrally wide oval, a bi- pointed oval, and a dorsally convergent oval. According to the authors, a predominance of the oval variations was also visible during the postnatal period due to the growth of the anterior intraoccipitalsynchondroses. Similar to the current examined human population, in which the frequency of longitudinally oval (mostly bi-rounded and ventrally wide) and round types (two semicircle) were similar, the main tendency pointed by Richards and Jabbour [38] was an increase in the ventral breadth of the foramen making the foramen anteriorly wide, not fully circular. A low incidence of the proper circular variation was also found in the currently examined population. The differences in the frequencies of each variation may be secondary to various geographic and age ranges since most of the archaeological skulls (4500–100 years old) came from North America and their biological age was below 21 years of age. However, the main objection to such results is the fact that the study was based mostly on dried skulls, without the possibility of establishing the correct developmental follow-up changes that may occur in individuals.

Studies in South Indian Population

The average length of the foramen magnum (LFM) of South Indian male skulls was found to be 34.37 ± 2.38, which was lower than the Brazilian male skulls (35.7 ± 0.29) [39], the Turkish (37.2 ± 3.43) [40], Spanish (36.2 ± 0.3) [41], and the English populations (35.91 ± 2.41)11. Similarly LFM of the female skulls of the South Indian population was found to be 33.80 ± 2.56 which was lower than Brazilian population (35.1 ± 0.33) [39], Turkish (34.6 ± 3.16) [40], Spanish (34.30 ± 0) [41], and English populations (34.71 ± 1.91) [42]. Regarding the width of the foramen magnum (WFM), the values of the South Indian male skulls was found to be 28.98 ± 2.22 which was lower than Brazilianmale skulls (30.3 ± 0.20) [39], the

Turkish (31.6 ± 2.99) [40] , Spanish (31.1 ± 0.3) [41], and English populations (30.51 ± 1.77) [42]. The same measure for the female skulls of the South Indian population was found to be 27.60±2.67 which was lower than the Brazilian population (29.4±0.23) [39], Turkish populations (29.3 ± 2.19) [40], Spanish (29.6 ± 0.3) [41] and English populations (29.36 ± 1.96) [42] . The results demonstrated that sexual dimorphism is present in the foramen magnum and also racial difference is seen.

Conclusion

Generally speaking, the foramen magnum development is regulated by various biological (mostly genetic and hormonal) and non-biological factors during the pre- and postnatal periods. The non-biological group includes an asymmetrical foetal position in the uterine cavity, which may result in changes within muscle attachments. The majority of anatomical brain abnormalities also involve the asymmetrical deformity of cranial bones. Patients with achondroplasia were found to have an abnormally shaped foramen magnum, which was smaller in size, influencing the flow of blood and cerebrospinal fluid. In the postnatal period, the main role in abnormal, acquisitive bone formation is played by malnutrition and pathological states directly affecting bone mineralisation as well as prevalent customs.

Corresponding Author

P.Jayashri

Senior lecturer, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

Mobile:-+91-9841788803

Email id:-shrijaya2009@gmail.com

References

1. Krogman WM, IscanMY.The human skeleton in forensic medicine. Springfield, Illinois: Charles Thomas Pub Ltd. 1986.
2. Macaluso Jr PJ.Metric sex determination from basal region of the occipital bone in a documented French sample. Bull Mem.Soc.Anthropol.Paris 2011; 23:19-26.

3. Standarding S. Gray's anatomy. The anatomical basis of clinical practice. 39th ed. London : Elsevier Churchill Livingstone 2005:460
4. Murshed K A, Cicekeibasi A E, Tuncer I. Morphometric evaluation of the foramen magnum and variations in its shape. A study of computerized tomographic images of normal adults. Tur J Med Sci2003; 33:301-306.
5. Sgouros S, Goldin HJ, Hockely AD, Wake MJ, et al. Intracranial volume change in childhood. J Neurosurg1999; 91:610-616.
6. GunayY,AltinkokM.Thevalue of the size of Foramen magnum in sex determination. J Clin foirensic Med2000;7(3):147-149.
7. Romanes GJ. Cunmningham's text book of anatomy. 12th ed. Oxford : Oxford University Press. 1981:114.
8. Oliveira Ed, Rhoton AL, Peace D. Microsurgical anatomy of the region of the foramen magnum. Surg Neurol1985;24:293-352.
9. Rhoton AL. The foramen magnum. Neurosurgery Suppl2000;47(3):S155- S193.
10. Bannister LH, Berry MM, Collins P, Dyson M, et al. Gray's anatomy the anatomical basis of medicine and surgery. 38th ed. Edinburgh: Churchill Livingstone1995:567-568.
11. Singh KB. A material study of Chinese crania. J Anat soc India 1963:12-16.
12. Scheuer L, Black S.The juvenile Skeleton. The Elsevier; London.2004
13. Gunay Y, AltinkokM.The value of the size of foramen magnum in sex determination. J Clin Forensic Med2000;7:147-149.
14. Selma URM, Gokharman D, Kacar M, Tuncbielek I, KosarU.Estimation of sex by 3D CT measurements of the foramen magnum. J Foensic Sci2005; 50:1310-1314.
15. Deshmukh AG, DevershiDB.Comparison of cranial sex determination by univariate and multivariate analysis. J Anat Soc India2006; 55: 48-51.
16. Suazo GIC, Russo PP, Zavando MDA, Smith RL.Sexual dimorphism in foramen magnum dimensions. Int J. Morphol2009; 27(1):21-23.
17. Gruber P, Henneberg M, Böni T, RühliFJ.Variability of human foramen magnum size. Anat Rec (Hoboken) 2009; 292:1713-1719.
18. Babu YPR, KanchanTanuj, AttikuYamini, Dixit PN, KotianMS.Sex estimation from foramen magnum dimensions in an Indian population. Int J Legal Med 2012; 19:162-167.

19. Radhakrishna SK , Shivarama CH , Ramakrishna A , BhagyaB.Morphometric analysis of foramen magnum for sex determination in south Indian population. Nitte University Journal of Health Science 2012; 2(1):20-22
20. Schmetzer A. Measurement of the foramen magnunm in children. Neuroradiology 1971;2:162.
21. Tiexeira WRG. Sex identification utilizing the sizes of the foramen magnum. Am J Forensic Med & Path1982;3(3):203-206.
22. Routal RR, Pal GP, BhagwatSs, TamankarBP.Material studies with sexual dimorphism in foramen magnum of human crania. J Anat Soc India1984;33(2):85-89.
23. Catalina – Herrera CJ. Study of the anatomic metric values of the foramen magnum and its relation to sex. Acta Anat 1987;130:344-347.
24. Sayee R, Jankiram S, Thomas IM. Foramen magnum measurements of crania from Karnataka. J Anat Soc India 1987;36(2):87-89.
25. Hecht JT, Horton WA, Reid CS, Pyeritz RE, et al. Growth of the foramen magnum in achondroplasia. Am J Med Gen 1989;32:528-35.
26. Ferreira, F. V.; Rosenberg, B. & da Luz, H. P. The "Foramen Magnum" index in brazilians. Rev. Fac. Odontol. Sao Paulo 1967; 5(4):297-302.
27. Teixeira, W. R. Sex identification utilizing the size of the foramen magnum. Am. J. Forensic Med. Pathol., 1982; 3(3):203-6.
28. ZadvornovIu, N. Variations in the shape of the foramen magnum and the structure of its posterior border. Arkh. Anat. Gistol. Embriol.,1972; 63(7):42-50.
29. Catalina-Herrera, C. J. Study of the anatomic metric values of the foramen magnum and its relation to sex. Acta Anat. (Basel) 1987;130(4):344-7.
30. Zaidi, S. H. & Dayal, S. S. Variations in the shape of fora- men magnum in Indian skulls. Anat. Anz., 1988; 167(4):338- 40.
31. Henríquez-Pino, J.; Cricenti, S. V. &Didio, L. J. A. Morfometría del foramen magno y surelación con el tipo de cráneo en individuosbrasileños. Rev. Chil. Anat.,1995; 13(2):159-64.
32. Gapert, R.; Black, S. & Last, J. Sex determination from the foramen magnum: discriminant function analysis in an eighteenth and nineteenth century British sample. Int. J. Legal Med., 2009; 123(1):25-33.
33. Günay, Y. &Altinkök, M. The value of the size of foramen magnum in sex determination. J. Clin. Forensic. Med., 2000; 7(3):147-9.

34. Uysal, S.; Gokharman, D.; Kacar, M.; Tuncbilek, I. &Kosa, U. Estimation of sex by 3D CT measurements of the foramen magnum. J. Forensic Sci.,2005; 50(6):1310-4.
35. MilhoratTH,NishikawaM,KulaRW,DlugaczYD. Mechanisms of cerebellar tonsil herniation in patients with Chiari malformations as guide to clinical management. Acta Neurochir, 2010; 152: 1117–1127.
36. Moss ML. The functional matrix hypothesis re- visited. I. The role of mechanotransduction. Am J Orthod Dentofacial Orthop 1997; 112: 8–11.
37. Moss ML, Young R. A functional approach to craniology. Am J Phys Anthropol 1960; 18: 281–292.
38. Reynolds KK, Modaff P, Pauli RM (2001) Absence of correlation between infantile hypotonia and foramen magnum size in achondroplasia. Am J Med Genet 2001;101: 40–45.
39. Richards GD, Jabbour RS. Foramen magnum ontogeny in Homo sapiens: a functional matrix per- spective. Anat Rec 2011;294: 199–216.
40. Manoel C, Prado FB, Caria PHF, Groppo FC. Morphometric analysis of the foramen magnum in human skulls of brazilian individuals: its elation to gender. Braz J Morphol Sci 2009; 26(2): 104-108.
41. Murshed KA, Çiçekçibasi AE, Tuncer I. Morphometric Evaluation of the Foramen Magnum and Variations in its Shape: A Study on Computerized Tomographic Images of Normal Adults. Turkish J Med Sci. 2003; 33(1): 301- 306.
42. Herrera CJC. Study of the anatomic metric values of the foramen magnum and its relation to sex. Acta Anatomica. 1987; 130: 344-347.
43. Gapert R, Black S, Last J. Sex determination from the foramen magnum: discriminant function analysis in an eighteenth and nineteenth century British sample. Int J Legal Med. 2008; 123(1): 25-33

Morphometric Analysis of Jugular Foramen

Tandra das. T[1],
Graduate Student[1], Assistant Professor[2], Department of Anatomy,
Senior Lecturer[3], Department of Oral and Maxillofacial Surgery
Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

Abstract

Introduction: The jugular foramen is difficult to understand and to access surgically; It is difficult to conceptualise because it varies in size and shape in different crania, from side to side in the same cranium, from its intracranial to extra cranial end in the same foramen, because of its complex irregular shape, its curved course, its formation by two bones, and the numerous nerves and venous channels that pass through it. The jugular foramen is a large aperture in the base of the skull. It is located behind the carotid canal and is formed in front by the petrous portion of the temporal, and behind by the occipital; it is generally larger on the right than on the left side. A study is done to measure the jugular foramen in right and left side using 50 dry skulls of unknown sex in Department of Anatomy in Saveetha Dental college.

Materials and Methods:50 adult unsexed, dry human skulls were examined from the Department of Anatomy of Saveetha Dental College and hospital, Chennai. All the skulls were normal and were devoid of any malformation. The skulls were used for tutorial teaching for dental students to measure the anteroposterior and transverse dimensions.

Result: The mean length of jugular foramen is found to be 15.07 mm. The mean breadth is 9.02mm

Conclusion: The present study observed the right jugular foramen to be larger than left. Findings obtained in the present study supports previously reported morphometric variations of jugular foramen besides adding data. These findings will be of utmost usefulness to ENT surgeons, neurosurgeons, forensic experts and radiologists.

Key Words:*jugular foramen, dry skull, size, calliper, extracranial.*

Introduction

The jugular foramen is the most complicated to appreciate and to approach surgically; It is difficult to gestate because it differs in size and shape in every crania, from side to side in the same cranium, from its intracranial to extra cranial end in the same foramen, due to its intricate irregular shape, its curved course, its formation by two bones, and the several nerves and venous channels that pass through it.Jugular foramen is a large aperture in the base of the skull. It is situated behind the carotid canal and is formed by the petrous part of the temporal bone and behind by the occipital bone. The jugular foramen is the main route of venous outflow from the skull and is characterised by laterality based on the predominance of one of the sides. Sigmoid sinus persists as internal jugular vein in posterior part of jugular foramen. Itis of great surgicalsignificance , several essential structuresruns through it. It transports nerves and vessels out of base of skull[1]. Jugular foramen lies between the occipital bone and petrous part of the temporal bone, it is elongated and irregularly shaped[2] .The 9 Th, 10 Th , 11 Th cranial nerves exit the cranial cavity through this foramen [3] .

Hovelacque was the first to propose the subdivisions of jugular foramen. A fibrous or bony septum that joins the jugular spine of petrous bone to the jugular process of occipital bone, divides the foramen into anteromedial or pars nervosa and posterolateral or pars vascularis [4]. The ninth cranial nerve , inferior petrosal sinus and meningeal branch of ascending pharyngealartery pass through pars nervosa while others pass through the pars vascularis. A dome is bony roof present in the jugular foramen . The presence of dome indicates the presence of jugular bulb [5]. The Anomalies of the jugular bulb are correlated with jugular foramen, as glomic tumours, which are usually in immediate contact with the structures that cross it[6]. Pathological processes affecting jugular foramen include intracranial meningiomas, paragangliomas, schwannomas, metastatic lesions and infiltration inflammatory process from surrounding structures such as the middle ear [7]. The most preferred treatment for these cases includes surgical resection.Ligation of the internal jugular is sometimes performed during radical neck dissection with the risk of venous infarction, which some adduce to be due to ligation of the dominant internal jugular vein [8] It is generally said that although the Jugular foramen is larger on the right side compared to the left, its size as well as its height and volume vary in different racial groups and sexes. The foramen's complex shape, its formation by two bones, and the numerous nerves and venous channels that pass through it further compound its anatomy [9,10] Since 1500 A.D severalscientific investigators including

Versaliuswere fascinated by the difference in shape and for the jugular foramen. Versalius (1543) in his illustrations of base of skull has mentioned compartmentations of jugular foramen. Manyresearch have been done including osteological, radiological and microdissections were performed to understand the puzzle of compartmentation and variations in the anatomy of jugular foramen, which led to conflicting observations.[11]

Intracranial and extra cranial lesions may affect the jugular foramen in addition to intrinsic abnormalities. Surgical resection is the treatment of choice in the majority of these cases. Development in microsurgical procedures have made possible the removal of advanced Jugular foramen lesions, which were initially thought to be inoperable [12] . As neurosurgeons become bolder in approaching this region, so the need for familiarity with the detailed anatomy of this region becomes greater. In Vasalius[13] book De HumaniCorporisFabrica it is mentioned that there are only three illustrations of the base of the skull. Two are identical apart from the labelling, and the left jugular foramen is much larger than the right. The third illustration is that of a skull held by a skeleton and the details of the base are indistinct, but the right jugular foramen appears to be larger than the left.[14]The foramen shows variation in shape, size, laterallity for the same skull the difference may be related to sex and race [15] .The present study was conducted to study the morphometry of jugular foramen, significance between sizes of right and left foramen.

Materials and Methods

50 adult unsexed, dry human skulls were examined from the Department of Anatomy of Saveetha Dental College and hospital, Chennai. All the skulls were normal and were devoid of any malformation. The skulls were used for tutorial teaching for dental students to measure the anteroposterior and transverse dimensions. Study was done on 100jugular foramen in 50 dry skulls.The length and width of jugular foramen, on both right and left side were measured using the Digital Vernier Calliper.[16]

Result

Table 1: The mean length of jugular foramen is found to be 15.07 mm .The mean breadth is 9.02mm.

	Range in mm	Mean in mm

Length: left	9.96 to 18.11	13.96 mm (±) 2.1215
Breadth: left	4.91 to 14.01	8.22 mm. (±) 1.91
Length: right	9.63 to 18.62	16.18 mm (±) 4.184
Breadth: right	6.10 to 13.78	9.82 mm. (±) 1.899

Discussion

The jugular foramen is difficult to understand and to access surgically; the problems in exposing this foramen are due to its deep position and the adjacent structures such as carotid artery anteriorly, the facial nerve laterally, hypoglossal nerve medially and vertebral artery inferiorly.[18]

The size and shape of the jugular foramen is obviously related to the size of the internal jugular vein and the presence or absence of a prominent superior bulb. The right foramen is usually larger than the left. There is a very wide variation in the anatomy of the intra cranial venous sinuses which accounts for variation in size and shape of jugular foramen. The difference in size of the two internal jugular veins is already visible in the human embryo at the 23 mm stage (8 weeks post-conception) and probably results from differences in the pattern of development of the right and left brachiocephalic veins.[19]

In the current study, the length and breadth of jugular foramen in the right side is greater than that of the left side. From these observation it can be said that the area of the right foramen is greater than the left foramen.

Sturrock[17] examined the jugular foramina in 156 skulls and found out that, in 69% of cases the right was larger than the left and in 23% the left was larger

Osunwoke E.A et.al did morphometric analysis of the jugular foramen of 120 dry adult skulls in Southern Nigeria and found the mean length of the right and left jugular foramen was 15.76±0.22mm and 13.39±0.23mm respectively, while the mean width of the right and left jugular foramen was 9.34±0.18mm and 7.54±0.20mm respectively. Significant difference between the right and the left jugular foramen was observed. The right jugular foramen was found to be larger than the left in Southern Nigeria [5].

According to Wysocki, Reymond and Skarzyński[16] (2006), difference in the results can be explained by racial and separate factors. As to the separate factors, these authors mention the significant correlation between the size of the Jugular foramen and also the hypoglossal channel with skull volume, thus pointing to the significance of cranial capacity for brain venous drainage.[11]

Patel and Singel[6] studied 91 Indian skulls (Saurashtra region) and observed in 60.4% cases larger right foramen, in 15.4% larger left foramen and in 24.2% equal on both sides. The jugular fossa was observed in 38.5% cases on the right side, 14.3% cases on the left side, 21% cases bilaterally and absent in 25.3% of skulls.

In study of Roma Patel and C.D.Mehta the mean transverse diameter of jugular foramen on the right and left were 12.17mm (range: 4.516.5 mm and 11mm (range: 5-16mm) respectively, while their sagittal diameter measured 7.9mm (range:3–12.5 mm) and 6.2 mm (range: 3–12.5mm) on the right and left respectively. Both diameters are more on right side. In the study done by Idowu on Nigerian skull, he found mean transverse diameter of jugular foramen on the right and left were 13.90 mm (11.6–17.0 mm) and 14.11 mm (9.2–20.2 mm), while their sagittal diameter measured 10.22 mm (6.8–14.4 mm) and 9.57 mm (7.4–12.8 mm) on the right and left respectively . According to study done on turks skull by Ekinci and Unur, the sagittal and transverse diameters of the left jugular foramen were 7.6 and 15.5 mm, respectively, and on the right 8.4mm and 16 mm, respectively . Pereira, GAM. studied total 111 skulls (of southern Brazil) and it was noticed that mean transverse diameter was 15.82mm on right side and 15.86mm on left side; mean sagittal diameter was 9.21mm on right side and 8.65mm on left side . In study of Hussain Saheb et al [11] it was found that the mean length of jugular foramen on the right and left were 23.62mm and 22.86mm, while their widths measured 7.83mm and 6.83mm respectively. The mean area on the right was 584.36mm and on the left was 493.30mm. Predominance of one of the two foramina appeared in 89.6% of cases. Predominance on the right was 64.8% and 24.8% on the left. 10.4% cases were equal on both sides.

Hatiboglu and Anil [12] studied 300 Anatolian skulls from the 17th and 18th centuries and observed that in 61.6% the foramen was larger on the right side and in 26% it was larger on the left side and in the reminder of equal size.

Some studies have added other parameters for morphometric analysis like Tekdemir observed no partition in their studies while Ekinci et al.found bony bridges in 20% and tripartite jugular foramen in 0.7%. Rhoton and Buza noted 26% bony bridges; this was bilaterally represented in 8%. A bony bridge in 3 (7.5%) of the jugular foramen with 1 skull bilaterally was found in this series. Rhoton et al and DiChiro et al observed a separate bony canal anterior to the pars nervosa in 6% of skulls, while Patridgenoted a frequency of 25%. This bony canal was for the exit of the IX cranial nerve. The present study did not notified a separate bony canal anterior to the pars nervosa. The smaller pars nervosa is relatively consistent in size compared with the larger and more variable pars vascularis.[20]

Navsa and Kramernoted a significantly larger exocranial area on the right in blacks but one of normal size in whites in South Africa.

All these previous studies shows correlation to our study .This study can be further carried out using increased sample size by adding gender and age correlation for better ratios , comparison and complete morphometric analysis.

Conclusion

The clinical significance of jugular foramen is very important during surgeries. Therefore, the dimensions of jugular foramen is very much necessary. The jugular foramen exhibits complex anatomical relationships and contains significant vessel and neural structures. The present study observed the right jugular foramen to be larger than left. Findings obtained in the present study supports previously reported morphometric variations of jugular foramen besides adding data. These findings will be of utmost usefulness to ENT surgeons, neurosurgeons, forensic experts and radiologists.

Corresponding Author

R.N. Balakrishna, Senior Lecturer, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

162, Poonamallee High Road, Chennnai-600077, Tamil Nadu, India

E-mail: balakrishdr8394@gmail.com
Phone Number: +91 8939896729

References

1. Khanday S, Subramanian RK, Rajendran M, Hassan AU,Khan sh. morphological and morphometric study of jugular foramen in south indian population.
2. Pereira GA, Lopes PT, Santos AM. Morphometric aspects of the jugular foramen in dry skulls of adult individuals in Southern Brazil. J Morphol Sci. 2010;27:3–5.
3. Pereira GA, Lopes PT, Santos AM, Krebs WD. Morphometric aspects of the jugular foramen in dry skulls of adult individuals in Southern Brazil. J Morphol Sci. 2010;27(1):3-5.
4. Idowu OE. The jugular foramen-a morphometric study. Folia morphological. 2004;63(4):419-22.
5. Osunwoke EA, Oladipo GS, Gwunireama IU, Ngaokere JO. Morphometric analysis of the foramen magnum and jugular foramen in adult skulls in southern Nigerian population. Am J Sci Ind Res. 2012;3(6):446-8
6. Patel MM, Singel T. Variations in the structure of the jugular foramen of human skull in Saurashtrarejoin . J Anat Soc India. 2007;56(2):34-37.
7. Pereira GA, Lopes PT, Santos AM, Krebs WD. Morphometric aspects of the jugular foramen in dry skulls of adult individuals in Southern Brazil. J Morphol Sci. 2010;27(1):3-5.
8. Wysocki J, Chmielik LP, Gacek W. Variability of magnitude of the human jugular foramen in relation condition of the venous outflow after ligation of the internal jugular vein. J Otolaryngologia 1999;53:173–77. [2].
9. Navsa N, Kramer B. Aquantitative assessment of the jugular foramen. AnatomischerAnzeiger 1998;180:269–73. [3].
10. Hovelacque A. Osteologie. Paris. 1967;155-56. [4].
11. Kanemoto Y, Ochiai C, Yoshimoto Y, Nagai M. Primarily extracranial jugular foramen neurinoma manifesting with marked hemiatrophy of the tongue: case report. Surg Neurol. 1998 May; 49(5):534-37.

12. Woodhall B. Anatomy of the carnial blood sinuses with particular reference to the lateral. The laryngoscope. 1939 Oct 1;49(10):966-1009.
13. Vesalius, A. De HumaniCorporisFabrica. Basel: Oporinus 1543.
14. HussainSaheb S , Mavishetter G F, Thomas S T, Prasanna L C, Muralidhar P. A Morphometric study of the jugular foramen in human adult skulls of south India. J Biomed Sci and Res., 2010;2(4):240243.
15. Berge JK, Bergman RA. Variations in size and in symmetry of foramina of the human skull. Clinical Anatomy. 2001 Nov 1;14(6):406-13.
16. Wysocki J, Reymond J, Skarżyński H, Wróbel B. The size of selected human skull foramina in relation to skull capacity. Folia Morphologica. 2006;65(4):301-8.
17. SturrockR.R.Variations in the structure of the jugular foramen of the human skull. Journal of Anatomy. 1998;160:227-230.
18. Hatiboglu MT, Anil A. Structural variations in the jugular foramen of the human skull. J Anat 199;180:191–196.
19. JoshiniShanmugam, Dr. K. YuvrajBabu. Morphometric analysis of jugular foramen. J sciInd res 2016; 2319-7064
20. Chong VFH, Fan YF. Jugular foramen involvementinnaso-pharyngeal carcinoma. J LaryngOtology. 1996; 110: 897–900.

Morphometric Study of the Sellar Region of the Sphenoid Bone in South Indian Dry Skulls

Pavithra H Dave[1], M.S. Thenmozhi[2], ThummalaBhagyalakshmi[3]

Graduate Student[1] ,Professor and Head[2], Department of Anatomy,

.Senior Lecturer[3], Department of Pedodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Sella turcica is a saddle-shaped concavity in the body of sphenoid bone situated in the middle cranial fossa of skull, which is seen clearly on lateral cephalometric radiograph. Precise anatomical knowledge of the sellaturcica is important for neurologists and surgeons when operating in the region of cavernous sinus or the surrounding structures. The purpose of this study is to establish normative reference standards that could assist in evaluation and detection of pathological conditions. Precise knowledge of dimensions and anatomy of sellaturcica is required for effective and safe treatment of various pituitary disorders and in the procedure of pituitary ablation with radioactive implants. A Morphometric analysis of the Sella Turcica and structures in vicinity were done in thirty dry human skulls. The linear measurements in the sellaturcica were taken using a digital vernier caliper. Present study will provide the normal morphometric data for future reference and further studies.

Key Words:*sellaturcica, clinoid process, sphenoid bone, morphology, vernier caliper*

Introduction

Sella turcica is situated on the intracranial surface of the body of the sphenoid bone. 80% of the sella is occupied by the pituitary gland. Any abnormality or pathology in the gland could manifest from an altered size, shape and functions of the sellaturcica. Around 13% of brain tumour are found in the sellaturcica [1]. The anterior boundary of sellaturcica is completed by tuberculum sellae and laterally it is by middle clinoid processes. The posterior boundary represents a vertical plate of bone, the dorsum sellae on the superolateral margins, dorsum sella ends as small posterior clinoid process. In the middle of sella, the hypophyseal fossa is present which contains pituitary gland [2]. Any abnormality or pathology in the gland could manifest from an altered size and shape of the sellaturcica, to a disturbance in the regulation of secretion of glandular hormones; prolactin, growth hormones,etc [3].Sellaturcica is also usually used as a reference point with nasion to establish the base of the skull in cephalometric analysis. This is commonly done prior to orthodontic treatment.

All the anatomical details concerning the possible variants of the sellar region are taken into account by neurosurgeons in order to decide which approach is to be chosen. For this reason, neurosurgeons also perform anatomical studies on cadaveric specimens or dry skulls to obtain the additional information required[4].

Literature analysis showed that, the comprehensive craniometrical details of the sellar and suprasellar region of the sphenoid bone in the Indian skull is lacking though there is tremendous developments in neurosurgical techniques[4-5].

The enlarged sellaturcica on a radiograph has been found to be associated with adenomas, meningioma, primary hypothyroidism, prolactinoma, gigantism, acromegaly, empty Sella syndrome, and Nelson syndrome. A small size may lead to decreased pituitary function causing symptoms such as short stature and retarded skeletal growth.Research concerning the sella turcica has focused on both size and morphology [5-6].It is essential to study the morphology of sellaturcica to know the various anomalies or unusual appearance of sellar region. Therefore, the morphometric study was undertaken for present work involving the measurement of dimensions of sellaturcica[7].

Materials and Methods

A total of 30 dry human skulls of both sexes were examined from the Department of Anatomy at Saveetha Dental College and Hospitals, Chennai, Tamil Nadu. Keeping in view the aim of the study mentioned above, following observations were recorded in millimetres, using a Digital Vernier Calliper. The skulls with gross craniofacial malformations were excluded from the study. Figure 1 represents the distance between the lateral walls of the mid sellar region. Figure 2 represents the depth of the sella and Figure 3 represents the distance between tip of anterior clinoid process to posterior clinoid process.

Observations

The measurements of the sellar region are tabulated below in Table 1.

Table 1: Measurements of Sellar Region

S.NO	MEASUREMENTS	AVERAGE mm	RANGE-MALE mm	RANGE-FEMALE mm
1.	Distance between the lateral walls of the mid sellar region	11.8	11.5-18.5	10.5-14.5
2.	Depth of sella	8.59	5-11	5-9
3.	Distance between tip of anterior clinoid process to posterior clinoid process	11.01	9.5-11.5	5-11
4.	Inter distance between tip of anterior clinoid process	24.55	19.25-31.5	17-30

The anterior clinoid process is a constant landmark and showed pneumatization of one or both. The middle clinoid process was present in most of the skulls and absent in a few.

Results

In this current study, the distance between the lateral walls of the mid sellar region was found to be 11.8mm, the depth of the sella was found as 8.59mm, the distance between the anterior clinoid process to the posterior clinoid process was found to be 11.01mm and the inter distance between the tip of anterior clinoid process was 24.55mm.

Figure 1: Distance Between Lateral Walls of the Mid Sellar Region

Figure 2: Depth of Sella

Figure 3: Distance Between Anterior And Posterior Clinoid Process

Discussion

The importance of understanding the anatomy of sellar region and its variations is of utmost importance to neurologists and neurosurgeons dealingwith pathologies in this region. Also sellaturcica is of importance becausewithin its center lies the ‘sella point’ which helps in evaluation of craniofacial morphology[3].

The anterior and middle clinoid processes in the living are normally linked by a ligament which is not seen in the dry skull. If it does not ossify, it leaves behind a space between the anterior and middle clinoid process called the clinoid space. If the ligament ossifies it forms a foramen through which the internal carotid artery courses to supply the brain. This foramen is called “foramen clinoideocaroticum”, the carotico clinoid foramen [4]. The ossification of fibrous ligaments is considered a normal physiological process that occurs with age.

Sellar bridges may be sequelae of ossification in dura mater extending between the anterior and posterior clinoid process [5]. Ligamentous ossification occurring in the early age is possibly an extension of the normal ossifications of the anterior and posterior clinoid processes [6]. Formation of the sellar bridges may result directly from the pattern of sphenoid development or can be dictated by the physiological activities of chemical compounds that are involved in embryogenesis and buildup of the bones. Some proteins control the mechanism of hardening in ossifying structure or inhibit this process. For example, matrix Gla protein inhibits calcification in soft tissues while osteopontin slows down the ossification in hard tissues [7]. They reported a

total incidence of 22 % of various types of interclinoid bars. Increased prevalence has been reported in subjects with developmental craniofacial and tooth abnormalities [8].

Jones et al. [9] studied the incidence of sellaturcica bridges in 150 patients who had undergone combined surgical orthodontic corrections. They found sellar bridges in 16.7 % cases in the group treated by combined surgical and orthodontic means, whereas it was 7.3 % in the orthodontic-only group [10-11].

Erturk et al. [12] studied 119 adult dry skulls and 52 cadavers in which they found 8.18 % incidence of sellar bridges, higher on the right side (2.34 %) than on the left side (1.75 %) [13]. The absence of anatomic data on normal variation of the interclinoid ligament is a severe deficiency of modern anatomy textbooks. This study is very important for regional surgery; its anatomy has received less attention.

In the literature the anterior clinoid process has received more attention because it has been used by the surgeons, to gain entry into the clinoid space to explore the internal carotid artery and other structures in vicinity [14].The posterior clinoid process is less accessed for suchoperations. Any abnormality in the posterior clinoid process may compress the surroundingstructures especially the internal carotid artery and alter the attachment of the tentoriumcerebelli to the posterior clinoid process. The anterior clinoid process has been reported to be joined to the middle clinoid process by a fold of dura mater [15].

The size and shape of aperture in the centre of diaphragm sellae are variable. It is either round or elliptical in shape and is for the passage of stalk of pituitary gland. Its size is important in protecting the gland from transmitted pulsations of choroid plexus and for protecting the fibers of optic chiasma in cases of suprasellar extension of pituitary tumours. Variations in the morphometry of the sellar opening provides explanation for direction of growth of pituitary tumours towards cavernous sinus and supra sellar region [16]. The overall percentage incidence of round (60%) and elliptical (40%) shapes observed in the present study on Indian population of a wide age range could not be compared with the values reported in literature on adult Korean, Turkish British and Kenyan population [17].

The neural relationships of the sphenoid bone are among the most complex of any bone: the olfactory tracts, gyrus rectus, and posterior part of the frontal lobe rest against the smooth upper

surface of the lesser wing; the pons and mesencephalon lie posterior to the clival portion; the optic chiasm lies posterior to the chiasmatic sulcus; and the second through sixth cranial nerves are intimately related to the sphenoid bone. All exit the skull through the optic canal, superior orbital fissure, foramen rotundum, or foramen ovale, all foramina located in the sphenoid bone. The absence of such bony protection within the walls of the sinus may explain some of the cases of cranial nerve deficits and carotid artery injury aftertranssphenoidal operations [18]. The bone is often thinner over the carotid arteries than over the anterior margin of the pituitary gland.

The pterion and the “keyhole” are two important anatomical landmarks in the region of the greater wing in the lateral view .The pterion is located over the upper part of the greater wing. The “keyhole” is located just behind the junction of the temporal line and the zygomatic process of the frontal bone several centimeters anterior to the pterion. A burr hole placed over the pterion will be located at the lateral end of the sphenoid ridge. A burr hole placed at the keyhole will expose the orbit at its lower margin and dura over the frontal lobe at its upper margin.

In 1987 Quakinine& Hardy studied 250 sphenoid bones & found the average length was 8mm, average width was 12mm & depth was 6mm[19].Asad and Hamid showed that average width of sellaturcica was 14.9mm and depth was 9.9mm which is relatively higher than present study[20].

The surgical approach to sellar region by trans-sphenoidal, is already an established procedure in Neurosurgery and its complications are well described by some authors. Anatomical variations of this region are the causes of complications in this procedure. The most vulnerable structures along the sinus are: the internal carotid artery near the anterior wall of the sella, and optic nerves, located superolaterally[21]. The study had no aim to determine whether any of these structures were exposed within the sinus.

Most sellar region masses are pituitary adenomas and other neoplasms may occasionally be encountered in this location. The most common type of pneumatization of the sphenoid sinus was the sellar, and depending on the age group, sphenoid sinus was larger in males than females.The anatomical variations of structures of the sellar and parasellar regions varies widely and has been described in various forms [22].

Conclusion

Precise anatomical knowledge of the sellaturcica is important for neurologists and surgeons operating in the region of cavernous sinus or the surrounding structures.

All the linear measurements of the sellaturcica in the present study were within standard range. The results of the present study of sellar size may be used as reference guide for future studies about sellaturcica morphology with a larger population. A better understanding of these complex structures is essential in clinical diagnosis and treatment of disease.

Corresponding Author

ThummalaBhagyalakshmi

Senior Lecturer, Department of Pedodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

SaveethaUniversity,

162, Poonamallee High Road ,Chennai 600077 ,Tamil Nadu, India

Email: bhagya.thummala@yahoo.co.in

Telephone Number: +91 9642432345

References

1. Mehultandel, Daxakanjiya. Morphological study of sellaturcica in Gujarat state. Indian journal of research2015.4;(2):56-8.
2. DaxaKanjiya BJ. morphological study of sellaturcica in gujarat state. Medical Science. 2015 Feb 4;(2):78-84.
3. Pisaneschi M, Kapoor G. Imaging the sella and parasellar region. Neuroimaging Clinics. 2005 Feb 1;15(1):203-19.
4. Senthilkumar S, vijaykumar J, Nirmalpalit. Craniometrical study of the sellar and supra sellar region of the sphenoid bone in south Indian skull. Anatomical adjuncts. 2008; 5(2):1-4.

5. TejavathiNagaraj R, James L, Keerthi I, Balraj L, Goswami RD. The size and morphology of sellaturcica: A lateral cephalometric study. Journal of medicine, radiology, pathology& Surgery 2015,1;3:3-7
6. Andredaki M, Koumantanou A, Dorotheou D, Halazonetis DJ. A cephalometric morphometric study of the sellaturcica. The European Journal of Orthodontics. 2007 Oct 1;29(5):449-56.
7. Kolagi S, Herur A, Patil G, Rairam GB. Complete sellaturcica bridges prevalence and dimensions. J AnatSoc India. 2011 Jun 1;60(1):22-5.
8. Sakran Am, Khan Ma, AltafFm, Faragalla He, Mustafa Ay, Hijazi Mm, Niyazi Ra, TawakulAj, MalebariAz, Salem Amal A. A Morphometric Study of the Sella Turcica: Gender Effect. International journal of anatomy and research 2015,3;1:927-34.
9. Inoue T, Rhoton AL, Theele D, Barry ME. Surgical approaches to the cavernous sinus: a microsurgical study. Neurosurgery. 1990 Jun 1;26(6):903-32.
10. Hare HF, Silveus E, Smedal MI. Roentgenologic diagnosis of pituitary tumors. Radiology. 1949 Feb;52(2):193-8.
11. Pendergrass, E. P., Schaeffer, J. P., and Hodes, P. J. The Head and Neck in Roentgen Diagnosis, (Blackwell Scientific Publications, Oxford). 1956; 2:947.
12. Keyes JE. Observations on four thousand optic foramina in human skulls of known origin. Archives of Ophthalmology. 1935 Apr 1;13(4):538-68.
13. Joplin, G. F., Fraser. RCiba Foundation Colloquia on Endocrinology.1960; 13-14.
14. Kleinschmidt-DeMasters BK, Lopes MB, Prayson RA. An algorithmic approach to sellar region masses. Archives of Pathology and Laboratory Medicine. 2015 Mar;139(3):356-72.
15. Yamashita S, Resende LA, Trindade AP, Zanini MA. A radiologic morphometric study of sellar, infrassellar and parasellar regions by magnetic resonance in adults. SpringerPlus. 2014 Dec 1;3(1):291.
16. Saeger W, Lüdecke DK, Buchfelder M, Fahlbusch R, Quabbe HJ, Petersenn S. Pathohistological classification of pituitary tumors: 10 years of experience with the German Pituitary Tumor Registry. European Journal of Endocrinology. 2007 Feb 1;156(2):203-16.

17. Somerset HL, Kleinschmidt-DeMasters BK. Approach to the intraoperative consultation for neurosurgical specimens. Advances in anatomic pathology. 2011 Nov 1;18(6):446-9.
18. Prayson RA, Kleinschmidt-DeMasters BK. An algorithmic approach to the brain biopsy—part II. Archives of pathology & laboratory medicine. 2006 Nov;130(11):1639-48.
19. Kleinschmidt-DeMasters BK, Prayson RA. An algorithmic approach to the brain biopsy—part I. Archives of pathology & laboratory medicine. 2006 Nov;130(11):1630-8.
20. Lillehei KO, Kirschman DL, Kleinschmidt-DeMasters BK, Ridgway EC. Reassessment of the role of radiation therapy in the treatment of endocrine-inactive pituitary macroadenomas. Neurosurgery. 1998 Sep 1;43(3):432-8.
21. A Study On Anatomical Variations Of Anterior Clinoid Process And Caroticoclinoid Foramen. *Int. J. Of Adv. Res.* 4 (5). 1348-1355 (Issn 2320-5407)
22. Mugilan M.R., YuvarajBabuAnd Karthik Ganesh, Morphometric Analysis Of Location And Position Of Foramens Present In The Middle Base Of The Skull, International Journal Of Current Advanced Research, 2017; 6(3):2902-2903.

A Comprehensive Review On The Effects Of Olestra

Denesh Raj Elangovan [1], Dhanalakshmi Ravikumar [2]

Graduate Student [1], Senior lecturer [2],Department of Pedodontics and Preventive Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Olestra is known as fat substitute.It's also known as by its brand name Olean. It does not add on calories or cholesterol to the product. It's also used in food preparation such as potato chips.Originally it'sbeen approved by The Food and Drug Admiration (FDA) for the replacement of fats and oil. This review article highlights in detail about olestra.

Keyword: Olestra, fat, oil, Food, Drug

Introduction:

In 1996, the U.S. Food and Drug Administration (FDA) approved a new fat substitute, olestra (Olean), for use in “savory snacks” (potato, tortilla and corn chips; crackers; and extruded snacks) Federal Register 1996 [1]. Olestra has physical properties and taste and cooking characteristics similar to regular fats and oils [2].On the other hand, it does not contribute any energy to the diet because it is not hydrolyzed by gastric lipases and therefore it is not absorbed. Due to its unique properties, olestra can serve a zero-calories replacement for conventional fats and oils [2].

U.S Food and Drug Admistration History

In 1996, the U.S Food and drug Admistration (FDA) approved a new fat substitute called olestra or also known as Olean [1]. Despite its potential public health benefits as a nonenergy fat substitute, there is controversy concerning the effect of olestra. Experimental studies in both humans and animals find that olestra can sequester fat-soluble nutrient in the gut and thereby reduce their absorption [1]. This made the FDA in the addition of vitamin A, D, E and K [1, 2]. In approving olestra, the FDA made assumptions about the level of its consumption and its effect on serum micronutrients concentration. As a condition for approval, the FDA mandated a program of active post marketing [1]. The Olestra Post- Marketing Surveillance Study (OPMSS) was designed to investigate whether consumption of olestra containing food as part of self-selected diets[2].

Chemistry

Triglycerides, the energy yielding dietary fats consists of three fatty acids bonded to a glycerol [3]. Because olestra is synthesized from sucrose, it can bond with six, seven or eight fatty acids.

Effect on GI Tract

When olestra and a fat soluble nutrient comes in contact in the GI tract, a portion of the nutrient partitions into olestra [4]. The portion is then unavailable to the mixed intestinal micelles and is

removed from the body with the nonabsorbed olestra. Key factor controlling the partitioning mechanism include following:

1) The lipophilicity of the nutrient,

2) The relative amount of olestra and nutrient in the GI tract,

3) The time between consumption of olestra and nutrient [4]

Chemical CompositionOf Olestra

It is formerly called sucrose polyester, is the generic name of nonabsorbable, synthetic hexahepta and octaesters of sucrose and fatty acids, with the physical properties of conventional dietary fats [7]. The fatty acids are of chain length C12 and C24, derived from edible oils, such as soybean, corn or cottonseed. Olestra is not hydrolyzed by pancreatic lipase or colonic bacteria and it cannot be absorbed. Being nonabsorbable but with the physical properties of triglycerides olestras are potential; a caloric substitutes for dietary fat.

Vitamins Effects AndAbsorption

Clinical research shows a modest reduction in serum cholesterol and vitamin E levels. The effect on vitamin E absorbtion can be offset by supplementation of olestra with vitamin E [8]. The status of vitamin D and K and absorption of lipophilic drugs are not altered by daily consumption of 18g olestra. Although serum retinol levels are not reduced, additional research is focusing on effect of olestra on hepatic stores of vitamin A to assess the appropriateness of supplement [8]. Using olestra to reduce the amount of fat in high fat foods, without affecting other nutrients, should contribute to a diet lower in energy from fat and higher in energy from carbohydrate [9].

Drug Absorption

Studies were conducted in rats and humans which show that it did not effect absorption of lipophilic drug [8]. The drug tested included pranolol and the oral contraceptives norethindrone and ethynyl estradiol, which are the lipid soluble [10]. Diazepam was also tested due to its wide use. In humans, drug was tested in a milk shake made wither 16g olestra or 16g corn oil water [8]. It shows no different in amount absorbed [11].

Mineral Absorption

Olestra would not be expected to effect mineral absorption since minerals are not lipid soluble and it does not saponify in gastrointestinal tract [12].

Cholesterol Absorption

The absorption of cholesterol is decreased when olestra is included in the diet [8]. The effect has been observed when the source of intestinal cholesterol is either exogenous or endogenous [8].

Conclusion

It's been proven that olestra does not harm our human body unless it's been over consumed in a short period of time. 18g/day of olestra decreases the serum vitamin E levels to within normal range. The risk of over consumed can leads to cardiovascular disease, hypertension, obesity and diabetes.

Corresponding Author

Dhanalakshmi Ravikumar,

Senior Lecturer, Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences,

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

E-Mail : dhana9677@gmail.com,

Telephone number - 9597011442

References

1. Thornquist, M. D., Kristal, A. R., Patterson, R. E., Neuhouser, M. L., Rock, C. L., Neumark-Sztainer, D., &Cheskin, L. Olestra consumption does not predict serum

concentrations of carotenoids and fat-soluble vitamins in free-living humans: early results from the sentinel site of the Olestra Post-Marketing Surveillance Study. J Nutr 2000; 130(7): 1711-1718.

2. Schlagheck, T. G., Riccardi, K. A., Zorich, N. L., Torri, S. A., Dugan, L. D., & Peters, J. C. (1997). Olestra dose response on fat-soluble and water-soluble nutrients in humans. J Nutr 1997; 127(8):1646S-1665S.
3. Cooper, D. A., Berry, D. A., Spendel, V. A., King, D., Kiorpes, A. L., & Peters, J. C. Olestra dose response on fat-soluble and water-soluble nutrients in the pig. J Nutr 1997; 127(8):1573S-1588S.
4. Cooper, D. A., Webb, D. R., & Peters, J. C. Evaluation of the potential for olestra to affect the availability of dietary phytochemicals. J Nutr 1997; 127(8), 1699S-1709S.
5. Daher, G. C., Cooper, D. A., & Peters, J. C. (1997). Physical or temporal separation of olestra and vitamins A, E and D intake decreases the effect of olestra on the status of the vitamins in the pig. J Nutr 1997; 127(8): 1566S-1572S.
6. Peters, J. C., Lawson, K. D., Middleton, S. J., &Triebwasser, K. C. Assessment of the nutritional effects of olestra, a nonabsorbed fat replacement: introduction and overview. J Nutr 1997;127(8):1539S-1546S.
7. Aggarwal, A. M., Camilleri, M., Phillips, S. F., Schlagheck, T. G., Brown, M. L., &Thomfode, G. M. Olestra, a nondigestible, nonabsorbable fat. Dig Dis Sci1993; 38(6), 1009-1014.
8. Bergholz, C. M., Jandacek, R. J., & Thomson, A. B. Review of Laboraotory and Clinical Studies of Olestra, a Nonabsorbable Lipid. Can J GastroenterolHepatol 1991; 5(4), 137-146.
9. Eastwood, M. A., &All good, G. S. The effect of olestra on breath gas production and fecal microbial counts. EurJ ClinNutr 1995; 49(9):627-639.
10. Jones, D. Y., Koonsvitsky, B. P., Ebert, M. L., Jones, M. B., Lin, P. Y., Will, B. H., &Suttie, J. W. Vitamin K status of free-living subjects consuming olestra. AmJ ClinNutr 1991; 53(4): 943-946.
11. Kalyani P, Gayathri R, Vishnupriya V. Top food among adolescent. Int JPharmaSciRev Res 2016; 39(2): 20-24
12. Charan V.S.S. Leptin in obesity – A review. Int JPharmaSciRev Res 2016; 8(6): 428-430

A Comprehensive Review on Therapeutic Effects of Green Tea

Abiraamasri BL[1], DhanalakshmiRavikumar[2]

Graduate Student[1], Senior Lecturer[2,] Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Green tea is one of the most popular tea that is commonly cultivated in China and Japan where it gained its legendary origin about 4000 years ago. Green tea is extracted from the leaves of Camellia sinensis that undergoes small amout of oxidation during production. It spreads world wide because of it important beneficial effects which include weight loss, prevention of cancer, lowers cholesterol level, cardiac problems, lowers blood pressure, diabetes, anti-bacterial, anti-viral, anti-microbial effect. Types of green tea is mainly based on harvesting period. Best quality of leaves are got the first flush of harvest. Constituents of green tea are amino acids, lipids, carbohydrates , enzymes, carotenoids and dietary minerals. Chemical components of green tea include xanthine derivative, vitamins and catechine derivatives. Catechins have antibiotic and immunomodulatory effect. Green tea also have side effects due to presence small amount of caffeine which is the xanthine derivative. Side effects include nausea, vomiting, irritability, insomnia, high blood pressure and rapid increase in heart rate.

***Key Words*:** *Green tea, Camellia sinensis, prevention of cancer, catechins, xanthine derivative, nausea, vomiting*.

Introduction

Green tea is the most popular tea that is extracted from the leaves of Camellia sinensis which undergoes minimal oxidative effect on production[1]. Its legendary origin is from china which is about more than 4000 years ago. Green tea cultivation is also distributed in other countries including Japan, Korea, Thailand, Vietnam, Asia, Darjeeling, Assam and Ceylon[2].The first green tea was exported from India to Japan during the 17th century. It is estimated that about 2.5 million tons of tea leaves are produced each year throughout the world, with 20% produced as green tea, which is mainly consumed in Asia, some parts of North Africa, the United States, and Europe [3]. The association between tea consumption, especially green tea, and human health has long been appreciated [4]. Green tea and black tea are processed differently during manufacturing.Green tea gained its popularity in west where they commonly prefer black tea because of their production. Black tea is processed in way to allow fermentation whereas green tea processing avoids fermentation process. Green tea gains its popularity in west due to it high beneficial effects which include weight loss prevention of cancer, diabetes, heart disease, decreasing cholesterol level, lowering blood pressure and tooth decay. It is used as raw materials in beverage industries, dietary supplements and cosmetics[5]. There are various types of green tea which is based on harvesting period and growing conditions[6]. Based on growing condition, green tea is of two types- those grown under sun shine and those grown under shade. First harvesting period is between april to early may. Second harvest is between june through july. Third harvest is from late july to early august. Occasionally there might even fourth harvest. Only first flush brings good quality of leaves. Green tea contains variety of enzymes, amino acids, carbohydrates, lipids, sterols, polyphenols, carotenoids, tocophenols, vitamins, caffeine and related compounds, phytochemicals and dietary minerals[5]. There is a evidence that large volume consumption of green tea causes oxidative stress, liver toxicity and reduction in cancer such as breast, prostate, ovarian and endometrial cancer.

Chemical Constituents

Chemical contents of green tea are xanthine derivatives, vitamins and catechins. Xanthine derivatives includes theophylline, caffeine, theobromine, glutamide derivative. Nutritional components of green tea are vitamin A, vitamin C, fluoride and potassium[7]. All tannins in green tea are catechins. Tannin is a gallic acid ester of a carbohydrate or phenolic compound.

Tannins are acidic due to phenolic hydroxyl groups in gallic acid moiety. They also act as antioxidants and forms complexes with metals. Tannins in tea are hydroxylatedflavanoids. Catechins also have antibiotic and immunomodulatory effect.The preparation methods influence the catechins both quantitatively and qualitatively; the amount of catechins also varies in the original tea leaves due to differences in variety, origin, and growing conditions[8]. The preparation of fresh green tea cannot totally extract catechins from the leaves; therefore, the concentration found differs from the absolute values determined through the complete extraction of leaves [9]. Green tea does not appreciable amount of these catechin dimmers. They also have beneficial health effect such as neuroprotective activity, anti-inflammatory, anti-ulcer, anti-viral, anti-bacterial and anti-parasitic effect[10].Epigallocatechin 3-gallate (EGCG) which is a derivative of catechin constitutes 50%-75% of the total flavonoid content in green tea. EGCG is a potent antioxidant and act to inhibit a number of physiologically important enzymes. It might also have therapeutic application in the treatment of some types of cancer, chronic fatigue syndrome, neurodegeneration, periapical lesions and regulating HIV viral load.Tea components possess antioxidant, antimutagenic, and anticarcinogenic effects and could protect humans against the risk of cancer by environmental agents [11]. Sano et al. [14]reported the inhibitory effects of green tea leaves against tert-butyl hydroperoxide-induced lipid peroxidation, and a similar antioxidant effect on the kidney was observed after oral administration of the major tea polyphenol EGCG. The antioxidative potency of crude catechin powder and individual catechins was tested in experiments using the active oxygen method. Crude catechins reduced the formation of peroxides far more effectively than dl-α-tocopherol [12]. Shim et al. [16].studied the chemopreventive effect of green tea among cigarette smokers and found that it can block the cigarette-induced increase in sister chromatid exchange frequency.

Beneficial Effects

Green tea is known world wide only due to its high beneficial effects and that includes cavity prevention, cancer prevention, leukemia, brain functioning, diabetes, kidney failure prevention, weight loss, anti-viral and anti-bacterial effects[13].

Obesity

The effects of tea on obesity and diabetes have received increasing attention. Tea catechins, especially EGCG, appear to have antiobesity and antidiabetic effects [15]. African black tea extract has been shown to suppress the elevation of blood glucose during food intake and

reduce the body weight in KK-A(y)/TaJcl diabetic mice [17]. Although few epidemiological and clinical studies have shown the health benefits of EGCG on obesity and diabetes, the mechanisms of its actions are emerging based on various laboratory data. These mechanisms may be related to certain pathways, such as through the modulations of energy balance, endocrine systems, food intake, lipid and carbohydrate metabolism, and redox status [18].Recent data from human studies indicate that the consumption of green tea and green tea extracts may help reduce body weight, mainly body fat, by increasing postprandial thermogenesis and fat oxidation. In a randomized, double-blind, placebo-controlled, cross-over pilot study, six overweight men were given 300 mg EGCG per day for two days. Fasting and postprandial changes in energy expenditure and substrate oxidation were assessed. Resting energy expenditure did not differ significantly between EGCG and placebo treatments, although during the first postprandial monitoring phase, respiratory quotient values were significantly lower with EGCG treatment compared to the placebo. These findings suggest that EGCG alone has the potential to increase fat oxidation in men and may thereby contribute to the antiobesity effects of green tea. However, more studies with a greater sample size and a broader range of age and body mass index are needed to define the optimal dose [19].

Cavity Prevention

Green tea contains fluoride which helps to prevent cavities and strengthens teeth. Green tea is also said to kill oral bacteria that may bring bad breath.

Cancer Prevention

Breast cancer

In recent research it is found that premenopausal women who consumed more green tea had a lower number of lymph node metastases. In postmenopausal women greater consumption of green tea correlated increased expression of estrogen and progesterone receptor, which implies more differentiated tumor cells and prognosis better. Women with stage I or II breast cancer who consumed five or more cups of green tea a day had recovered more faster than those women who consumed less cups of green tea[20].

Leukemia

Green tea extract is a potent nucleoside transport inhibitor, which repair DNA after chemotherapy. EGCG which a green tea extract is used as an nontoxic adjuvant therapy for leukemia.

Brain Functioning

Green tea helps to protect brain from oxidative stress and lowers monoamine oxidase activity. Green tea in general, as well as phenolic components- catechins and epigallocatechingallate have been found to be effective at inhibiting MAO and lowering peroxide levels in glial cells in the brain[21].

Effect On Hair

Green tea polyphenols are only recently understood as positive factors in hair growth and follicle health. They possess some of the mechanisms of action as including inhibition of apoptosis (programmed cell death), radioprotection of follicle cells, profound antioxidant activity, and potential follicular inhibition of TGF-beta [22]. Green tea is an herbal dihydrotestosterone reliable contrarian. A high intake of green tea correlates to higher levels of sex hormone-binding protein globulin (SBGH) which carries hormones like testosterone around the body in a bound, unusable form so that tissues cannot use it directly. Testosterone is usually carried around the body by this binding protein, therefore, reducing levels of free testosterone, so that it cannot be converted to dihydrotestosterone (DHT) in the hair follicle, which is thought to shorten the hair cycle and cause hair loss in men. Green tea is thought to affect the 5α-reductase type I enzyme, which converts testosterone to DHT [23]. Although these findings are at preliminary stage these studies suggest that further analysis in this regards can prove to promising in future.

Effect on Cardiovascular Disease

Another gem associated with green tea is its ability to protect from cardiovascular diseases. Heart diseases are associated with a numberof risk factors and are most prevalent in the Western world, probably as a result of the lifestyle in this part of the world, which includes a diet high in saturated fats and low physical activity, and the large proportion of the population who smoke cigarettes have a high blood pressure. Green tea appears to be cardio-protective[24,25].Regular consumption of green tea also inhibits atherosclerosis. Ground green tea consumption decreases susceptibility of plasma and LDL to oxidation and also modulated cholesterol metabolism and might prevent initiation and progression of

atherosclerosis. Green tea has been shown to effectively lower LDL Cholesterol, triglycerides, lipid peroxides and fibrinogen while improving the ratio of bad / good cholesterol i.e. Ratio of LDL to HDL cholesterol. The potent antioxidant effect of green tea inhibits the oxidation of KDK cholesterol in the arteries which plays a major contributor role in the formation of atherosclerosis. People who drink at least three cups of green tea every day, a 2% lower risk of suffering a stroke is observed as compared with those who drink less than a cup a day [26]. Regular drinking of green tea seems to lower the chance of getting high blood pressure. The loss of arterial elasticity is one of the cause of high blood pressure. With age, this elasticity is lost and thromboxane is one cause of arterial constriction. Another cause of hypertension is an enzyme secreted by the kidneys called Angiotension converting enzymes (ACE). Green tea seems to block thromboxane as well as ACE production and appears to be their natural inhibitor which significantly reduces the blood pressure.

Effect on Renal Disease

Green tea has a protective effect on renal failure. Decreased kidney function due to aging and kidney failure are a frequent cause of death. A preliminary study in Mansoura University in Egypt has explored the possibility to protect kidney function from life threatening failure with the frequent use of green tea [27]. They found that animals with kidney failure when treated with 50mg/kg EGCG from green tea had recovered significantly with glomerular filtration rate in 7 days, reduced malondialdehyde and inflammatory cytokines and increased glutathione (antioxidant levels) as compared to resveratrol and quercetin. This study indicates that in streptozotocin (STZ)-induced diabetic nephropathy, kidney function appears to be improved with green tea (GT) consumption which also prevents glycogen accumulation in the renal tubules, probably by lowering blood levels of glucose. Therefore, GT could be beneficial additional therapy in the management of diabetic nephropathy[28].

Anti-Carcinogenic Activity

Abundant experimental and epidemiological evidences accumulated mainly in the past decade from several research analysis worldwide provides a convincing argument that green tea polyphenols reduces cancer risk in a variety of animal tumor bioassay systems[29].In this study, the extracts of green tea and green tea polyphenols have exhibited inhibitory effects against the formation and development of tumors at different organ sites in animals. These include animal models for skin, lung, oral cavity, oesophagus, stomach, intestine, colon, liver, pancreas, bladder, mammary gland, and prostate cancers [30]. EGCG can inhibit

tumorigenesis during the initiation, promotion and progression stages in animal models of carcinogenesis [31] . The majority of invitro cell culture, in vivo animal, and clinical intervention studies provided strong evidences supporting a chemopreventive effect of green tea extract or purifies EGCG, in preventing prostate cancer, but results from epidemiological studies of green tea consumption are mixed [32]. As per the analysis, role of green tea in breast cancer development in humans is still unclear[33]. There is an inverse association for green tea intake and risk of ovarian cancer [34]. Women who regularly drank green tea when the study began were 37% less likely to develop colorectal cancer compared to infrequent green tea drinkers. Women who continued to drink green tea regularly throughout the study shows a decreased risk of colorectal cancer by 57%. Green tea lowers risk of gallstones and biliary tract cancers. A human pilot study recently confirmed the protective effects of green tea against lung cancer seen in cell culture and animal studies. It has been shown that because of green tea consumption, DNA damage caused by smoking was decreased, cell growth was inhibited, and cellular triggers for apoptosis (cell suicide) in abnormal cells increased [35].

Effect on Rheumatoid Arthritis

Inflammation plays a key role in Osteoarthritis (OA) and Rheumatoid Arthritis. An approach that decreases inflammation may facilitates the development of effective strategies for its treatment and prevention. Green tea polyphenols offer a promising new option for the development of more effective strategies for the same. In a study it was found that EGCG, the major and most active component of green tea polyphenol (GTP), protects human chondrocytes from IL-Iβ induced inflammatory responses [36].Although the efficacy of EGCG or green tea extract in human RA or OA using the phase-controlled trials is yet to be tested, an extensive evaluation of the potential risks or benefits of using EGCG alone or together with anti-rheumatic drugs may open a new area of research wherein EGCG or its synthetic analog could be developed to enhance its clinical appeal[37].

Effect of Oral Health

Among oral diseases like dental caries and periodontal disease, dental caries is a multifactorial infectious disease in which nutrition, microbiological infection, and host response play important roles. Streptococcus mutans is mainly responsible for causing dental caries. Green tea has proved to have anti- Streptococcus mutans activity [38]. A study has uncovered yet another benefit of green tea consumption. It has been found that routine intake of green tea may also help in fighting against these oral diseases. It promotes healthy teeth

and gums. The study analyzed the periodontal health of 940 men, and found that those who regularly drink green tea had superior periodontal health [39]. Apart from their polyphenols content, green is a natural source of fluoride and an effective vehicle for fluoride delivery to the oral cavity. The mean fluoride concentration in green tea is ~ 2.1 ppm, which lies within the acceptable daily intake. According to a report, after cleansing the mouth with tea, approximately 34% of the fluoride is retained and shows a strong binding ability to interact with the oral tissues and their surface integuments [40]. This fluoride content may have a beneficial impact on caries and may carry out a wide range of biological activities including prevention of tooth loss and oral cancer. This trace fluoride mineral reacts with the enamel of the tooth and makes it 50-70% less susceptible to decay or gum damage and helps get beautiful white teeth [41]. A recent study suggests that there is an explicit association between the consumption of green tea and oral health. It is also evident that green tea products have been used for preventing and treating several oral and periodontal diseases[42]. Its frequent consumption greatly reduces bad breath (halitosis). Knowing the role of periodontopathic bacteria in producing volatile sulfur compounds, antimicrobial polyphenols in green tea can improve bad breath by suppressing these bacteria [43]. In consequence, green tea has been considered as functional food for oral health and is widely used in toothpaste formulation. Greater the concentration of catechins better the health benefits. So the consumption of green tea in comparison to other beverages may be widely recommended[44].

Kidney Health

Decreased kidney function due to aging and kidney failure are frequent cause of death. Epigallocatechinegallate induces antioxidant enzymes in the kidney, as well as reduce uremic toxins in the blood and improve kidney function[45]. Kidney problems are often associated with high blood sugar and consequent glycosylation of various proteins. Thus there is a strong link between kidney failure and diabetes. Since, green tea has the ability to lower serum glucose, there is also another way to help kidney failure.

Weight loss

Green tea increases the metabolism hence we gain lose in weight. The polyphenols found in green tea works to intensify levels of fat oxidation ant the rate at which the body turns food into calories.

Anti-Viral and Anti-Bacterial Effect

Tea catechins are strong anti-viral and anti-bacterial agent which make them effective for everything from influenza to cancer.

Side Effects

Green tea contains 2% to 4% caffeine, which affects thinking and alertness, increases urine output, and may improve the function of brain messengers important in Parkinson's disease. Polyphenols might be able to prevent inflammation and swelling, protect cartilage between the bones, and lessen jointdegeneration[46]. They also seem to be able to fight human papilloma virus (HPV) infections and reduce the growth of abnormal cells in the cervix (cervical dysplasia). The mild side effects can range from nausea, vomiting, irritability and insomnia usually associated with the caffeine content found in the tea. Some of the serious, but rare side effects are symptoms of high blood pressure, a rapid heart rate, seizures and allergic reactions like hives, rashes and itching. Harmful effects of tea overconsumption (black or green) are due to three main factors: (1) its caffeine content, (2) the presence of aluminum, and (3) the effects of tea polyphenols on iron bioavailability. Green tea should not be taken by patients suffering from heart conditions or major cardiovascular problems. Pregnant and breast-feeding women should drink no more than one or two cups per day, because caffeine can cause an increase in heart rhythm. It is also important to control the concomitant consumption of green tea and some drugs, due to caffeine's diuretic effects [47]. Some studies revealed the capacity of tea plants to accumulate high levels of aluminum. This aspect is important for patients with renal failure because aluminum can be accumulated by the body, resulting in neurological diseases; it is therefore necessary to control the intake of food with high amounts of this metal [20]. Likewise, green tea catechins may have an affinity for iron, and green tea infusions can cause a significant decrease of the iron bioavailability from the diet [45].Green tea can block absorption of certain vitamin, nutrients and proteins, so you should drink it moderately. If you are pregnant, you should not drink green tea or any tea, because of the caffeine. Drinking large amounts of green tea when you are pregnant can cause neural tube birth defects in babies[48]

Conclusion

Green tea is one of the most important tea which has more beneficial effect. One of the most important effect of green tea is its antioxidative effect. Even though it has many beneficial

effect, it also have side effects like nausea, vomiting, rashes due to presence of small quantity of caffeine.

Corresponding Author

DhanalakshmiRavikumar,

Senior Lecturer, Department of Pedodontics and Preventive Dentistry,

Saveeetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

Email id: dhana9677@gmail.com

Telephone Number : 959701142

Reference

1. McKay DL, Blumberg JB. The role of tea in human health: An update. J Am Coll Nutr. 2002;21:1–13.
2. Babu PV, Sabitha KE, Shyamaladevi CS. Therapeutic effect of green tea extract on oxidative stress in aorta and heart of streptozotocin diabetic rats. Chem Biol Interact. 2006;162:114–120.
3. Koo MWL, Cho CH. Pharmacological effects of green tea on the gastrointestinal system. Eur J Pharmacol. 2004;500:177–185.
4. Wu CH, Lu FH, Chang CS, Chang TC, Wang RH, Chang CJ. Relationship among habitual tea consumption, percent body fat, and body fat distribution. Obes Res. 2003;11:1088–1095.
5. Mukoyama A, Ushijima H, Nishimura S, Koike H, Toda M, Hara Y, Shimamura T. Inhibition of rotavirus and enterovirus infections by tea extracts. J Med Sci Biol. 1991;44:181–186.
6. Park H, Ko S, Kim J, Kim S. Effects of green tea extracts and polyphenols on the proliferation and activity of bone cells. J Bone Miner Res. 2003;18:342.
7. Johnson, R.; Bryant, S.; Huntley, A. L.. "Green tea and green tea catechin extracts: An overview of the clinical evidence", *Maturitas*,2012;73 (4): 280.

8. Jayakeerthana.S. Benefits of Green Tea: A Review. J. Pharm. Sci. & Res 2016;8(10):1184-1187.

9. Sheik R, Vishnu Priya V, Krishna Mohan S. Effect of Green Tea on Obesity – A Survey Int. J.Pharm.Sci.Rev.Res 2015;33(2):73-75.

10. Khokhar S, Magnusdottir SGM. Total phenol, catechin, and caffeine contents of teas commonly consumed in the United Kingdom. J Agric Food Chem. 2002; 50:565–570.

11. Fernandez PL, Martin MJ, Gonzalez AG, Pablos F. HPLC determination of catechins and caffeine in tea. Differentiation of green, black and instant teas. Analyst. 2000;125:421–425.

12. Chen ZY, Zhu QY, Wong YF, Zhang Z, Chung HY. Stabilizing effect of ascorbic acid on green tea catechins. J Agr Food Chem. 1998;46:2512–2516.

13. Amurdhavani B S Benefits of Green Tea in Dentistry- A Review,ResearchJPpharm and Tech. 2015:8(6): 772-774.

14. Sreevidhya T, GeethaRV, Antibacterial activity of the three essential oils on streptococcus mutans-an in-vitro study, Int. J. Drug Dev. & Res. 2014:6(4):9075-9344.

15. RoghithK,Karpagam K, Evaluation of lipid profile based on consumption of green tea- A systematic review,RJPT, 2016; 9(8):1277-1279.

16. Shim JS, Kang MH, Kim YH, Roh JK, Roberts C, Lee IP. Chemopreventive effect of green tea (Camellia sinensis) among cigarette smokers. Cancer Epidemiol Biomarkers Prev, 1995;4:387–391.

17. Kao YH, Chang HH, Lee MJ, Chen CL. Tea, obesity, and diabetes. Mol Nutr Food Res. 2006;50(2):188–210.

18. Kok E, Tascioglu S. Temperature dependace of copper and chromium transfer into various black and green tea infusions. J. Sci. Food Agric 1998; 76: 200-298.

19. Shoji Y, Nakashima H. Glucose-lowering effect of powder formulation of African black tea extract in KK-A(y)/TaJcl diabetic mouse. Arch Pharmacol Res. 2006;29(9):786–794.

20. Rudelle S, Ferruzzi MG, Cristiani I, Moulin J, Mace K, Acheson KJ, Tappy L. Effect of a thermogenic beverage on 24-hour energy metabolism in humans. Obesity. 2007;15(2):349–355.

21. Costa LM, Gouveia ST, Nobrega JA. Comparison of heating extraction procedures for Al, Ca, Mg and Mn in tea samples. Ann Sci. 2002;18:313–318.

22. Patil SM, Sapkale GN, Surwase US, Bhombe BT. Herbal medicines as an effective therapy in hair loss – A review. RJPBCS 2010; 1 (2): 773- 81.

23. Charles E. EpigallocatechinGallate Inactivates Clinical Isolates of Herpes Simplex Virus. AAC 2008; 962–970.

24. Thelle DS. Coffee, tea and coronary heart disease. Curr. Opin.Lipidol 1995; 6: 25-27.

25. Weisburger JH. Tea antioxidants and health. In: CadenasE,Packer L. Eds. Handbook of antioxidants. New York: Marcel Dekker 1996; 469– 486.

26. Lenore Arab, Weiqing Liu and David Elashoff .Green and Black Tea Consumption and Risk of Stroke: A Meta-Analysis. Stroke 2009; 40:1786-1792.

27. Mowafy AM, Salem HA, Gayyar MM, Mesery ME, Azab MF. Evaluation of renal protective effects of the green-tea (EGCG) and red grape resveratrol: role of oxidative stress and inflammatory cytokines. Nat Prod Res 2011; 25(8):850-856.

28. Renno WM, Abdeen S, Alkhalaf M, Asfar S. Effect of green tea on kidney tubules of diabetic rats. Br J Nutr 2008; 100(3):652-9.

29. Yang CS, Wang X. Green Tea and Cancer Prevention. Nutr. Cancer 2010; 62: 931-937.

30. Lambert JD, Elias RJ. The antioxidant and pro-oxidant activities of green tea polyphenols: A role in cancer prevention. Arch. Biochem. Biophys 2010; 501: 65-72.

31. Butler LM, Wu AH. Green and black tea in relation to gynecologic cancers. Mol Nutr Food Res 2011;55(6):931-40.

32. Green tea and prevention of esophageal and lung cancers. *Mol Nutr Food Res* 2011; 55(6):886-904.

33. Katiyar SK, Mukhtar H. Tea in chemoprevention of cancer: epidemiologic and experimental studies. Int. J. Oncol 1996; 8: 221- 238.

34. Dreaosti IE, Wargovich MJ, Yang CS. Inhibition of carcinogenesis by tea: the evidence from experimental studies. Crit. Rev. Food Sci. Nutr 1997; 37: 761-770.

35. Kohlmeier L, Weterings KGC, Steck S, Kok FJ. Tea and cancer prevention: An evaluation of the epidemiologic literature. Nutr. Cancer 1997; 27: 1-13.

36. Akhtar N, Haqqi TM. Epigallocatechin-3-gallate suppresses the global interleukin-1beta-induced inflammatory response in human chondrocytes. Arthritis Res Ther 2011; 13:93.

37. Ahmed S. Green tea polyphenol epigallocatechin 3-gallate in arthritis: progress and promise. Arthritis Res Ther 2010; 12:208.

38. Naderi N Jalayer, Niakan M, KharaziFard M J and ZardiS . Antibacterial Activity of Iranian Green and Black Tea on Streptococcus Mutans: An In Vitro Study. 2011; 8(2): 55–59.

39. Mitoshi K, Yoshihiro S,Masatoshi M and YoshihisaY.Relationship between Intake of Green Tea and Periodontal Disease. J Periodontol 2009; 80(3): 372-377.

40. Simpson A, Shaw L, Smith AJ. The bio-availability of fluoride from black tea. J Dent 2001; 29:15–21.

41. Okamoto M, Sugimoto A, Legun KP, Nakayama K, Kamaguchi A, Maeda N.Inhibitory effect of green tea catechins on cysteine proteinases in Porphyromonasgingivalis. Oral MicrobiolImmunol 2004; 19:118–120.

42. Hamidreza A, Ahmad M, Shayan, G, Hooman S, Keyvan S, Ali F. Review of The therapeutic effects of Camellia sinensis (green tea) on oral and periodontal health. Journal of Medicinal Plants Research 2011; 5(23): 5465-5469.

43. Liao S, Kao YH, Hiipakka RA. Green tea: biochemical and biological basis for health benefits. Vitam. Horm 2001; 62: 1-94.

44. VenkateswaraBabu, Sirisha K and Chava Vijay K. Green tea extract for periodontal health. J Indian Soc Periodontol 2011; 15(1): 18–22.

45. Hamdaoui MH, Chabchob S, Heidhili A. Iron bioavailability and weight gains to iron-deficient rats fed a commonly consumed Tunisian meal "bean seed ragout" with or without beef and with green or black tea decoction. J Trace Elem Med Biol. 2003;17:159–164.

46. Yang MH, Wang CH, Chen HL. Green, Oolong and black tea extracts modulate lipid metabolism in hyperlipidemia rats fed high-sucrose diet. J NutrBiochem. 2001;12:14–20.

47. Iso H, Date C, Wakai K, Fukui M, Tamakoshi A. The relationship between green tea and total caffeine intake and risk for self-reported type 2 diabetes among Japanese adults. Ann Intern Med. 2006;144:554–562.

48. Hara Y, Luo SJ, Wickremashinghe RL, Yamanishi T. Biochemistry of processing black tea. Food Rev. Int 1995; 11: 457- 471.

A Literature Review on the Effect of Psychoactive Drugs

Saarusree.E [1], Gayatri Devi. R [2], Asha Ramesh [3]

Graduate Student [1],Assistant Professor [2],Department of Physiology,

Senior Lecturer [3],Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

A psychoactive drug which is used either as pharmaceutical or psychotropic drug is a chemical substance which crosses the blood brain barrier and acts mostly in the central nervous system where it affects brain function resulting in alteration of cognition, consciousness, perception and behaviour. Psychoactive compound most commonly brings about changes in mood and consciousness that the person who is using it can find pleasant feeling for example euphorbia or advantageous for example increased alertness and thus reinforcing. Psychoactive drugs are chemical substances that affect the brain functioning, causing changes in behaviour, mood and consciousness. While these drugs can be used therapeutically to treat both physical and psychological disorders, they are also used recreationally to alter mood, perceptions and consciousness. Psychoactive substances are used by humans for a number of different purposes to achieve a specific end. These uses vary widely between cultures. Some substances in psychoactive drug may have illegal or controlled uses while othersdrug may have shamanic purposes but still others may have used medicinally. Other examples would be social drinking or sleep aids. Caffeine is the mostcommonly used psychoactive substance unless it is illegal to use.

Key Words: Caffeine; Psychotropic drugs; Blood-Brain Barrier; Addiction; perception

Introduction

A psychoactive drug which is commonly known as psychotropic substance and it is used as a chemical substance that acts onthe central nervous system where it can alter the function of brain, that can result in changes which are temporary in consciousness, attitude, behaviour and perception .These psychoactive drugs can be used either recreationally or purposefully to alter mankind consciousness such as consumption of coffee, alcohol and cannabis, as entheogens for the spiritual purposes such as the mescaline containing peyote cactus or psilocybin containing mushrooms and also used as medications such as the narcotics usage helps in controlling pain , stimulants used in psychoactive compound used to treat narcolepsy and attentional disorders and antidepressants or antipyretic used for treating neurological and psychiatric illnesses[1].These substances can be used either recreationallyor purposefullyto alter mankind consciousness whereas etheogens are used for ritual, spiritual, orshamanic purposes, as an important tool for studying or controlling the mind. Some multiple categories of psychoactive drugs, which are used as a prescription medicines, have various medicaland therapeutic uses in medical field. For example, some drugs are used as anesthetics, analgesics and hormonal preparations and further used as antiparkinsonian and anticonvulsant and for the treatment of neurological psychiatric related disorders , as hypnotic drugs, stimulant medication and anxiolytics used in the treatment of ADHD and sleep disorders. There are some of the psychoactive substances used for rehabilitation and detoxification program for the person who are using psychoactive drugs. This is especially true of the powerful dissociative used mainly are salvia divinornum and deliriants used mainly are jimson weed. Most frequently used pure form of psychedelic drugs to be non-addictive for example mescaline, psilocybin and LSD. Psychedelic drugs such as amphetamine or empathogen- entactogens such as MDMA and MDA that can produce an euphoriant effect or addiction stimulant and thus have an additional potential[2].

Basic Classification of Psychoactive Drugs

Caffeine is used in the worldwide populations. The most commonly used psychoactive substance is caffeine ,unless many other psychoactive drugs is legal to use and usually used in nearly places in south india. In northern populations , adults around 90% consumes caffeine daily[3]. Psychoactive drugs are classified into 5groups :

1]AnxiolyticsDrugs. Example areBenzodiazepine

2]Euphoriants drugs, Example are 3,4-methyl dioxymethamphetamine (Ecstasy), 3,4-methylene dioxyamphetamine, 6-amino propylbenzofuron, Indopan

3]Stimulants – Stimulants are used as a substance in waking up one person thoughts, stimulating the mind and some people consumes the usage of caffeine daily without having knowledge about its use. The examples are nicotine, cocaine, nicotine and amphetamine.

4]Depressants are used for the purpose as narcotics, hypnotics and sedatives. The depressants may include all of the following anxiety reducing, calmative, sleep-inducing, anesthetizing substances, which can sometimes induce perceptual changes, such as dream images, and also evoke feelings of euphoria.

Examples: alcoholic beverages (ethanol), opioids, barbiturates, benzodiazepines

5]Hallucinogens, including psychedelics, dissociatives and deliriants. This category encompasses all those substances that produce distinct alterations in perception, sensation of space and time, and emotional states

Examples: psilocybin, LSD and nitrous oxide [4].

Psychoactive drugs are often prescribed to manage pain. The experience which is characterised as pain is regulated primarily by endogenous opioid peptides. The pain can be primarily cured by usage of psychoactives drugs that can be operated on the neurotransmitter system, which is also commonly known as opioid receptor agonists. This class of drugs can be highly addictive, and includes opiate narcotics, like morphine andcodeine[5].

Action of Psychoactive Drugs in the Brain

Neurotransmitters which may include dopamine, acetylcholine, serotonin, norepinephrine, histamine and about other two dozen dosage psychoactive drugs. The influence of psychoactive drugs is systematically limits the synaptic changes in the brain.The exceptional case would occurs when the drugbring about changes by itself in its own way in other parts of the neuron, such as the cell membranes. For example, hallucinogens are used to be believed medication in causingsynesthesiafor example seeing, hearing- the words ends with ing by

mimicking norepinephrine, acetylcholine and serotonin[6]. Psychoactive drugs are used in operating byaffecting a single mankind neurochemistry, which in turn causes changes in a one person behaviour, perception, cognition, and mood. There are many other ways in which psychoactive drugs which may affect the brain cells. Each drug has a particular action on either one or more neurotransmitter or neuroreceptor present in the brain cells. Drugs that may increase their activity in particular neurotransmitter systems are commonly known as agonists. Agonists basically act by increasing the synthesis of one or more neurotransmitters, by playing an important role in reducing its reuptake from the synapses, which may act by mimicking the action by its functions like binding directly to the postsynaptic receptor. Drugs that may reduce their neurotransmitter activity are commonly known as antagonists, and functions by operating as interfering with synthesis or blocking the postsynaptic receptors so that the neurotransmitters cannot bind to them each other[7].

Adverse Effects of Psychoactive Drugs

There are a number of side-effects related while consuming psychoactive drugs which is associated with the psychotherapeutic agents. These side effects may includes and are not limited to drowsiness, delirium, dry mouth, tremor, irregular heartbeat, weight gain, agitation, headache, insomnia, gastrointestinal distress, nausea, menstrual irregularity, weight gain, weight loss, sex drive loss, skin rashes, and sweating. Patients should have informed priority the information about their healthcare provider if they had previous experience any of these side effects. In some other cases, a dosage which is given lethally has to be in the form of easily adjustable or change of the prescription can also used to alleviate any discomfortable which is caused by psychoactive products . Additional medications which has to be involved may also be used as prescribed medication in order to address the severe forms of side effects for example the anticholinergic medication which may be prescribed is used in the treatment of muscle spasms usually caused by antipsychotic medications [8]. Tarsive dyskinesia is a condition which is characterized by an involuntary movements of the mouth and other locations which is present in the body which has been reported in some psychoactive drugs consuming patients who oftenly used to take the antipsychotic medication on a long-term basis for more than several years. In some other cases, the condition involving psychoactive drugs is considered to be permanent, although discontinuing or involving the change of medication used may be considered as halt or reverse dose in most of the patients.Agranulocytosis is a potentially serious illness

commonly observed in the surrounding in which the white blood cells responsible that can typically fight against the infection in the body are commonly destroyed, it is a possible side effect of clozapine and psychoactive drug such as another antipsychotic. Patients who is taking clopaminemedication should undergo weekly blood tests analysis used to monitor their white blood cell counts[9].Psychotherapeutic agents which is used as psychoactive agents can be contraindicated which is not recommended for use in patients with certain medical conditions like diabetes mellitus, hypotension etc..,These psychoactive drugs may also interact with other prescription dosage for the purpose of beneficial effects and more the counter the medications is used, either used in the magnification or used in reducing the intended side effects effects of consuming one or both the drugs. In some psychoactive related circumstances, psychoactive substances are used to trigger serious and even life-threatening, and physical side effects [10]. For the some particular reasons , individuals who are undergoing prescribed psychoactive medication should inform priorly about their mental healthcare provider and any other psychoactive drug prescribing doctor undergoing all medications which they are used to take, and patient who have any medical conditions such as diabetes , hypotension which has not been yet to be disclosed. Many psychoactive drugs which has been used are contraindicated in pregnancy patients, particularly contra indicated in the first trimester during pregnancy .Patients should check their blood level with their doctorconsultation about the risks which is commonly associated with the psychotherapeutic medications and undergone possible treatment options when planning for a pregnancy[11].

Treatment Using Psychoactive Drugs

Drug addiction of psychoactive drugs is used as a treatment option with medications andmainly withbehavioural therapies management which is used as a kind of psychotherapy. A combination of the two drug addiction level of psychoactive drugs was found to be the most effective approach form among drug addiction level.

1]Medical management of psychoactive drugs

The main problem concerned while using medications is that drug dependent patients often visiting the ward are relaxed and not concerned about taking medication regularly and thereby making the treatment ineffective. It also remains to be ethically controversial whether the patient who are not cooperative during treatment phase and person who are under medication can be forced to follow the medication properly or whether treatments option with

others who are not undergone medication are made to be potentially irreversible, the following effects observed should be used [12].

2]Behavioural Therapies used while consuming psychoactive drugs

Behavioural therapies are tried to replace the motivation skill involved in usage of drugs with the motivation to engage in other behaviour management.These behaviour therapies most commonly rely on the same principles which has been followed for more than a year are learning and motivation that are primarily used to describes the development of dependence [13].

3] providing various substitute or maintaining the treatment in a proper way:

The person who is consuming drugs are forced to given the drug which acts as a drug in their own way without induction of any hazardous effects. This treatment option is usually involved to treat opioids dependence on methadone and other drug substance are usually substituted for heroin. This will help the person who is addicted to drugs in their own way to quit drugs and maintaining their life in least harmful way and avoiding to experiment the cause of death. In spite the use of substitution therapy in providing least harm to the patient it is proved to be against the ethical issues[14].

Various Behavioural Therapies in Treating Drug Addiction

There are usually many behaviour therapies are followed in management of dug addiction. Among that, most common behaviour therapy are listed out below:

1) Cognitive behavioural therapy: involved usually in finding out which factors is responsible for the triggers drug taking and involved in response to adapt to the new environment
2) Relapse prevention therapy: these prevention therapy normally involved in avoiding various circumstances to avoid relapse mechanisms, helps to find out environmental and emotional triggers for drug intake and other sort of cravings.
3) Contingency management therapy: usually provided with large number of punishments or either rewards to make the people quit the drug addiction habits
4) Motivational therapy: involved in the motivation of the individuals by obtaining consent according to their own specific levels of drug addiction and maintaining their goals by motivating them in their own way[15].

Legality of Psychoactive Drugs

The legality of the psychoactive drugs that has been found to be the most controversial factors that occurs throughout the most of therecent history; particularly involved the second opium war and the another cause which occurs is Prohibition , these are the two main historical examples that was used to be found out in legal controversy related to the surrounding psychoactive drugs. However, many factors has been followed in the recent years among the factors, the most dramatically document that used to be prescribe followed regarding the legality of psychoactive drugs is the only Single Convention based on the Narcotic Drugs, which is found to be an international treaty systems that used to be signed in the year 1961 found to be an Act provided by the united nations [16].The act was signed by the 73 nations including the India, United Nations and United Kingdom , the Single Convention on the use of Narcotic Drugs was established as Schedules form used for the legality of each drug delivery system and purposefully laying down an international agreement used to fight for the addiction to recreational drugs levels by sale combatting, use of the scheduled drugs and tackling the drugs .All the countries including United Nations, India and United Kingdom has to be signed for the purpose to pass the treaty laws used to implement these rules within their borders. However, some of the countries that has to be signed the Single Convention on the use of Narcotic Drugs, most commonly involved countries such as the Netherlands, are more lenient in their delivery system with their enforcement of these laws passed for usage of narcotic drug [17].In the United States particularly, the Food and Drug Administration (FDA) has proved to be an authority over all other drugs, which may include mainly those psychoactive drugs. The food and drug administration act used to be regulated in which psychoactive drugs are used more than the counter and which may be only available with a prescription base .[18].However, all other certain psychoactive drugs, such as drugs, alcohol and tobacco which have been listed in the Single Convention on the usage of the Narcotic Drugs are subjected to be under criminal laws. The Controlled Substances Act passed in the year 1970 also used to regulates the recreational drugs which is used to be outlined in the Single Convention act on the usage of Narcotic Drugs. Alcohol consumed which is regulated by the state governments, but the federal National Minimum Drinking Age Act used to penalises the states for not only following a national drinking age but also helps in its usage content. Tobacco which has been regulated by all fifty state governments, mostly people accepted suchas restrictions and prohibitions of certain drugs, especially the hard drugs used to get more seduced, which are considered to be illegal in most of the countries.In the medicalconcern , psychoactive drugs

used as a treatment for various illness is more widespread and generally accepted thing. Little controversy may exists while concerning over the counter psychoactive medications in antiemetics and antitussives[19].Psychoactive drugs are commonly prescribed to patients with psychiatric disorders. However, certain critics believe that certain prescription psychoactives, such as antidepressants and stimulants, are overprescribed and threaten patients' judgement and autonomy.These psychoactive drugs was considered to be the major concern in the diagnosis of narcotic drug usage in the majority of the countries with decreasing populations to the usage of psychoactive drugs[20].

Homeostatic Activity While Using Psychoactive Drugs

Exposure of the environment to a psychoactive substance causesa drastic changes in the functioning and structure of the neurons, as the nervous system after consumption of psychoactive drugs usually tries to re-establish the homeostasis disrupted in the nervous system by the presence of the drug also called as neurospasticity in technical terms . Exposure of psychoactive drugs to antagonists for a particular neurotransmitter shows varying increase in the number of receptors occurring for that neurotransmitter, and the receptors play an important role within themselves to become more sensitive. This is usually calledas sensitization[21]. Physical dependence of psychoactive substance on antidepressants or anxiolytics may result in worse depression or anxiety feeling, respectively, as these are withdrawal symptoms of using psychoactive drugs . Unfortunately, a correlation occurs because the clinical depression which is also called as major depressive disorder which is more often referred to as antidepressants causes depression, are usually requested by the patients and prescribed for the patients who are in the depressed state ,but it was not clinically depressed [22]. Psychoactive drugs shows increasing amounts of dopamine levels in the brain which may rewards the pathway and also acts mainly in the central nervous system for the purpose of altering ones behaviour ,mood changes , perceptions [23].

Addiction Level of Psychoactive Drugs

Psychoactive drugs are most commonly associated with the drug addiction. Addiction is basically divided into two types namely psychological addiction, which occurs due to

psychological problems in which a person who consumes the drug are forced by someone and not by their own willingness in spite a negative feedback may be obtained by the patient in a complete physical or by societal consequence, and physical addiction is the another way of addiction in which the person already consuming the drug by their own concerns to avoid physically uncomfortable by the society or even by medically harmful withdrawal symptomsthat will persist in the body[24].All the drugs which are not physically addictive by human beings , but any activity of the mankind will stimulate the brain dopaminergic reward system, in simple words if any pleasurable activityoccurs in the brain will lead to psychological drug addiction level[25]. Many professionals such as doctors, teachers, self helpful groups, and businesses markers will be well specialized in drug rehabilitation program with varying degrees of success rates and many parents made attempt to influence the actions and better choices of their children in drug addiction regarding psychoactive drugs [26]. Common forms of rehabilitationprogram may include psychotherapy, support groups and pharmacotherapy, which uses psychoactive substances in better amounts to reduce cravings and physiologically withdrawal symptoms when theperson is undergoing through any detoxification [27]. Methadone is itself used as an opioid drug and as a psychoactive substance, which is used in the common treatment for heroin addiction patients and as is another opioid marketed which is commonly available are buprenorphine [28].

Effect of Psychoactive Drugs on Children

The administration of psychoactive drugs in children starts at a particular period of 6-17years of age which is more common in the society and growing at an alarming rate in world .These psychoactive drugs commonly causes the opposite to the intended effect, which usually condemning in the children to a life of the misery and ill health[29]. As the childs emotional control breaks down due to medication effects of psychoactive drugs, mood stabilizers may be added to the considerable quantity. These childrens who end up on four or five psychiatric drugs at once a time and a diagnosis was found to be bipolar disorder that occurs by the age of eight or usually ten [30]. Children and adolescents who suffers under serious emotional disturbance are no longer treated and survive only in the hospitals, but more often in foster homes and community residences, facilities involved in this drug addiction program are learnt to treat children historically and adolescents undergoing psychoactive therapy face

with serious behavioural symptoms. Children and adolescents are usually hospitalized for shorter lengths of stay in the hospital ,resulting in pressure to use interventions that are likely to have a rapid effect, such as medication [31].Exposure to psychoactive drugs can produce changes to the brain that are used in counteracting their effects, which may lead to physical dependence and, some believe, make their effects temporary.For example, halothane is a GABA agonist,and ketamine is an NMDA receptor antagonist[32].

Discussion

According to Nutt et al and Morgan et al, there are lack of correlation among 6000 individuals in terms of harms related to psychoactive drugs. According to Di Forti et al ,there was minute difference between cannabis and skunk,which is other forms of psychoactive drugs considered to be increasing in harms of cannabidiol varieties . According to Morgan and curran, ketamine which is the most hazardous drug belongs to psychoactive compound, ranks eighth in overall harms of psychoactive drugs. According to Griffiths et al, cialis ,form of psychoactive drugs was considered as positive effect in curing hallucinations. According to Krupitsky et al, ketamine has improved effects in treatment of heroin addicts. According to krebs et al, psychoactive drugs play an important role in decreasing alcohol misuse. According to Johansen et al, 3,4- methylenedioxy- methamphetamine was considered to be most effective as an adjunct therapy for post-traumatic stress disorder (PTSD) and therapy-resistant anxiety disorders. According to Caulkins et al, alternative harm matrices would be taken into count which has the context of drug usage associated with harmful effects. According to Walton et al, cannabis, a form of psychoactive drugs used as sedative and anti-convulsants has been implicated for its organic form and its components is used as analgesics, stimulation of appetite and anti-emetics. Efforts which are renewed are made to demonstrate the efficacy of psychoactive drugs as adjunct therapy to psychoactives. Psychedelic drugs such as amphetamine or empathogen- entactogens such as MDMA and MDA that can produce an euphoriant effect or addiction stimulant and thus have an additional potential[33].

Conclusion

Drug usage and addiction level of psychoactive substance are used to superimpose a substantial healthy burden on the society. Recent advances in the brain cell researches are used to find various ways to reduce that healthy burden. General anaesthetics comes under the classes of psychoactive drug which are used onthe patientshelps to block the pain and other sensations. Most of the anaesthetics used helps to induce unconsciousness, which commonly allows the patients to undergo medical procedures like surgery without physical problems related pain or emotional problems related to trauma. To induce the unconsciousness which is caused by psychoactive drugs mainly anaesthetics are helpful in affecting most common GABA and NMDA systems. Thus, the judicious use of psychoactive drugs are warranted for de-addiction and rehabilitation programs as it bring about alterations in the mood and consciousness.

Corresponding author

Asha Ramesh

Senior Lecturer,Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University

Chennai-600077

Mobile: +91 9841722113.

Email: doc.ash4587@gmail.com.

References

1. Olivier B, van Dalen D. Social behaviour in rats and mice: an ethologically based model for differentiating psychoactive drugs. Aggressive Behavior. 1982, 1;8(2):163-168.
2. Zammit S, Moore TH, Lingford-Hughes A, Barnes TR, Jones PB, Burke M, Lewis G. Effects of cannabis use on outcomes of psychotic disorders: systematic review. The British Journal of Psychiatry. 2008, 1;193(5):357-63.
3. Kasimala BB. Caffeine in Chocolates a Hazardous Sign to Children’s Health. International Journal of Pharmaceutical & Biological Archive. 2011;2(6);37-42.

4. Bersani FS, Corazza O, Simonato P, Mylokosta A, Levari E, Lovaste R, Schifano F. Drops of madness? Recreational misuse of tropicamide collyrium; early warning alerts from Russia and Italy. General Hospital Psychiatry. 2013, 31;35(5):571-573.
5. Quiding H, Lundqvist G, Boreus LO, Bondesson U, Öhrvik J. Analgesic effect and plasma concentrations of codeine and morphine after two dose levels of codeine following oral surgery. European journal of clinical pharmacology. 1993, 1;44(4):319-23.
6. Avorn J, Dreyer P, Connelly K, Soumerai SB. Use of psychoactive medication and the quality of care in rest homes. New England Journal of Medicine. 1989,26;320(4):227-32.
7. Nicholi Jr AM. The nontherapeutic use of psychoactive drugs: A modern epidemic. New England Journal of Medicine. 1983, 21;308(16):925-33.
8. Barkley RA. A review of stimulant drug research with hyperactive children. Journal of Child Psychology and Psychiatry. 1977 , 1;18(2):137-65.
9. Sailesh KS, Archana R, Mukkadan JK. Impact of traditional vestibular stimulation on depression, anxiety and stress in college students. Biomedical Research. 2016;27(2):295-297.
10. Hindmarch I. Psychomotor function and psychoactive drugs. British journal of clinical pharmacology. 2004 ,1;58(7):720-740.
11. Lüscher C, Ungless MA. The mechanistic classification of addictive drugs. PLoS medicine. 2006 ,14;3(11):e437.
12. Gras A, Garnatje T, Bonet MÀ, Carrió E, Mayans M, Parada M, Rigat M, Vallès J. Beyond food and medicine, but necessary for life, too: other folk plant uses in several territories of Catalonia and the Balearic Islands. Journal of ethnobiology and ethnomedicine. 2016;12(1):23;1-53.
13. Azab A. Arum: a plant genus with great medicinal potential. European Chemical Bulletin. 2017 ,19;6(2):59-68.
14. Khan MN, Suresh J, Yadav KS, Ahuja J. Formulation and evaluation of antistress polyherbal capsule. Der Pharmacia Sinica. 2012;3(2):177-84.
15. Devi P, Meera R, Muthumani P, Kameswari B, Badmanaban R. Phyto-Physico chemical evaluation and Antioxidant activities of leaves of Naphelliumlappaceum. J. Pharm. Sci. & Res. 2009;1(3):117-22.

16. Olivier B, van Aken H, Jaarsma I, Van Oorschot R, Zethof T, Bradford D. Behavioural effects of psychoactive drugs on agonistic behaviour of male territorial rats (resident-intruder model). Progress in clinical and biological research. 1984;167:137-56.
17. Johnson B. Psychological addiction, physical addiction, addictive character, and addictive personality disorder: A nosology of addictive disorders. Canadian Journal of Psychoanalysis. 2003,1;11(1):135-160;279-280.
18. Zhang J, Xu M. Toward a molecular understanding of psychostimulant actions using genetically engineered dopamine receptor knockout mice as model systems. Journal of addictive diseases. 2001 ,10;20(3):7-18.
19. Hops H, Tildesley E, Lichtenstein E, Ary D, Sherman L. Parent-adolescent problem-solving interactions and drug use. The American journal of drug and alcohol abuse. 1990 ,1;16(3-4):239-58.
20. Mangini M. Treatment of alcoholism using psychedelic drugs: a review of the program of research. Journal of psychoactive drugs. 1998 ,1;30(4):381-418.
21. Cappell H, LeBlanc AE, Endrenyi L. Aversive conditioning by psychoactive drugs: Effects of morphine, alcohol and chlordiazepoxide. Psychopharmacology. 1973 ,1;29(3):239-46.
22. Hall W, Solowij N. Adverse effects of cannabis. The Lancet. 1998,14;352(9140):1611-1166.
23. Breggin PR. Psychiatric drug-induced Chronic Brain Impairment (CBI): implications for long-term treatment with psychiatric medication. International Journal of Risk & Safety in Medicine. 2011;23(4):193-200.
24. D'Aquila PS, Collu M, Gessa GL, Serra G. The role of dopamine in the mechanism of action of antidepressant drugs. European journal of pharmacology. 2000 ,29;405(1):365-73.
25. Hirschfeld RM. Clinical importance of long-term antidepressant treatment. The British Journal of Psychiatry. 2001;179(42):s4-8.
26. Harrison NL, Simmonds MA. Quantitative studies on some antagonists of N- methyl D- aspartate in slices of rat cerebral cortex. British journal of pharmacology. 1985; 84(2):381-91.
27. Leya Mathews AR. Management of pain using transdermal patches-a review. MANAGEMENT. 2016; 9(6):32-35.

28. Morales SS, Paramo OL, Cabural RC, Jarrell KK, Orendain SR, Parreñas CG. Analgesic Activity of Formulated Syrup of Cashew (Anacardiumoccidentale) Crude Leaf Extract on Albino Rats. Root Gatherers. 2016; 25(6):1-43.
29. Balaji O, Bairy K, Veena N. Management Of Depression In Terminally Ill Patients-A Critical Review. Management. 2017;10(5):31-36.
30. Berginc K. Pharmacokinetic interactions between drugs and dietary supplements: carbohydrate, protein, vitamin and mineral supplements. InDietary Supplements 2014 (pp. 85-126).
31. Sha HC, Ma QY, Jha RK, Ma ZH, Zhang M. Protective effect of resveratrol on intestinal mucosal barrier in rats with severe acute pancreatitis. Sichuan da xue xuebao. Yi xue ban= Journal of Sichuan University. Medical science edition. 2008;39(5):740-743.
32. Laskar JI, Chakravarty P, Dewan B. A study on incidence of adverse drug reactions with commonly prescribed drugs and causality assessment in Silchar Medical College and Hospital. International Journal of Basic & Clinical Pharmacology. 2017; 6(5):1175-83.
33. Ekanem PE, Ekanem R, Gaim K. Histological Patterns of Neurodegeneration of Frontal Cortex Neurons in Daturastramonium Treated Wistar Rats. Journal of Behavioral and Brain Science. 2016; 4;6(02):85-92.

A Review on Medicinal Values of Ginger

Yendodu Varshitha[1], Arvind. M[2]

Graduate Student[1],Professor[2],Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai India

Abstract

Ginger is a medicinal plant that is widely usedin traditional systems of medicine. The two most active constituents of ginger based preparations are gingerol and shagaol. It has antiemetic, antipyretic, analgesic, anti-arthritic and anti-inflammatory activities. Ginger (Zingiberofficinale Roscoe) has strong antioxidant effects helping in production of free radicals. Studies have proved the anti-tumour and hepatoprotective effects of ginger. This review discusses the pharmacological properties and clinical uses of ginger.

Key Words: *Ginger, Zingiberofficinale ,Antioxidant, anti-tumour ,Hepatoprotective.*

Introduction

Plants are being used by humans in many ways such as food and spices. Ginger or Zingiberofficinale, Roscoe belonging to the family Zingeberaceae which is a herbaceous rhizomatous plant. Ginger, the rhizomes of the plant Zingiberofficinale is arguably one of the most widely used culinary agent and spice in the world. In addition to its culinary use, ginger also possess medicinal properties and has been used since ancient times world wideto treat ailments like cold, headaches, nausea, stomach upset, diarrhoea, digestive, gastrointestinal disturbances, rheumatic complaints, asthma, parasitic infections, arthritis and muscular discomfort[1-7].

Pharmacognocy

Ginger belongs to family Zingiberaceae, genus Zingiber, species Zingiberofficinale. The ginger plant was first cultivated in China and then cultivated in India. It is a perennial reed like plant about a 3 to 4 feet tall. The root is gathered when the stalk withers, it is immediately scalded washed and scraped to kill it and prevent sprouting. The roots of ginger are used as a spice in cooking. The main constituents are sesquiterpenoids with zingiberene as main constituent

Figure 1: Ginger

Ginger and Antioxidant Action

Antioxidants are the substances that have the ability to counteract the damaging effects of free radicals in tissues and thus are believed to protect against cancer and heart disease[8]. About 40 antioxidants arepresentin ginger[9]. [6]-Dehydroshogaol, [6]-shogaol and 1-dehydro-[6]-gingerdione were shown to be potent inhibitors of nitrous oxide(NO) synthesis in activated macrophages[10]. The CO2 extract from ginger roots has been shown tohave a significant effect in inhibiting DPPH (2,2-Diphenyl-1-picril hydrazyl), up to 90.1%. Ginger also showed a higher chelating capacity. Increased generation of reactive oxygen species (ROS) and reactive nitrogen species (RNS) are implicated in various liver diseases, and in the toxic manifestations of various hepatotoxins[11,12]. Ginger extracts, oleoresins and the volatile oils possess free radical scavenging effects, and are effective in scavenging the superoxide, hydroxyl, nitric oxide in vitro[13]. The phytochemical zingerone is also reported to be an effective scavenger of the free radicals like superoxide[14-16], peroxy[17] and peroxynitrite[18], and to also inhibit the formation of14peroxynitrite-mediated tyrosine nitration. Another important phytochemical of the ginger.Gingerol is also shown to scavenge peroxyl radicals, to inhibit the production of nitric oxide and reduce generation of iNOS in LPS-stimulatedcells[19].

Ginger and Hepatoprotection

Phytochemical studies have shown that the unique culinary and medicinal properties of ginger are due to the presence of phytochemicals like zingerone, shogaols, gingerols, pardols, β-phellandrene, curcumene, cineole, geranyl acetate, terphineol, terpenes, borneol, geraniol, limonene, β-elemene, zingiberol, linalool, α-zingiberene, β-sesquiphellandrene, β-bisabolene, zingiberenol and α-farmesene. Scientific studies carried out in accordance to the principles of modern system of medicine have convincingly shown that ginger possesses numerous health benefits like antimicrobial, antiviral, gastroprotective, antidiabetic, anti-hypertensive, cardioprotective, anticancer, chemopreventive and immunomodulatory effects. In vitro laboratory studies on animals have also shown that ginger to possess hepatoprotective effects, and to protect the liver against the toxic effects of diverse class of

xenobiotic agents like alcohol[20-21], country liquor[22], acetaminophen[23], heavy metals[24,25], CCl[26], paraben[27] and bromobenzene[28].

Pharmacological Effects

Anti-Cancer Effects

The anticancer effects of ginger are thought to be attributed to various constituents including vallinoids, viz. (6)-gingerol and (6)-paradol, shogaols, zingerone, and Galanals A and B[29,30]

Anticoagulant Effects

Ginger has been shown to inhibit platelet aggregation[31,32,33] and to decrease platelet thromboxane production in vitro[33,34,35]. Gingerol, shogaol, paradol, and gingerol analogues exhibited antiplatelet activities.

Antiemetic Effects

Elements in ginger which are responsible for antiemetic effect are anticipated to be the gingerols, shogaols, and galanolactone, a diterpenoid of ginger[36,37,38].

Anti-Inflammatory Effects

Ginger has been found to inhibit prostaglandin biosynthesis[39] and interfere with the inflammatory cascade and the vanilloidnociceptor[40]. Ginger has been shown to share pharmacological properties with non-steroidal anti-inflammatory drugs (NSAIDs) because it suppresses prostaglandin synthesis through the inhibition of cyclooxygenase-1 and cyclooxygenase-2.

Gastrointestinal Effects

There is evidence that ginger rhizome (root) increases stomach acid production. If so, it may interfere with antacids, sucralfate (Carafate), H2 antagonists, or proton pump inhibitors. In contrast, other *in vitro* and animal studies have revealed gastro protective properties[41,42]. Shogaol, generally more potent than gingerol, has inhibited intestinal motility in intravenous preparations and facilitated gastrointestinal motility in oral preparations. Ginger extract has also been reported to inhibit the growth ofHelicobacter pylori in vitro.However, Desai et al. observed a significant increase in the exfoliation of gastric surface epithelial cells following the consumption of 6g or more of ginger[43].

Antimicrobial Activities

Ingenol and (6)–shogaol, isolated from ginger rhizome, demonstrated antiviral activity[44].

(10)-gingerol has been reported as active inhibitor of Mycobacterium aviumandMycobacterium tuberculosis in

vitro. Gingerol and related compounds have been investigated for antimicrobial activities.

(6)- gingerol and (12)-gingerol, isolated from ginger rhizome, demonstrated antibacterial activity against Porphyromonas gingivalis , Porphyromonas endodontalis and Prevotellaintermedia

Anti Arthritic Effect

Crude ginger extract,(6) gingerolto reduce joint swelling in animals induced with rheumatoid arthritis and exhibited potent anti inflammatory action.[46]

Ginger Uses in Dentistry

A study based on laboratory investigations to investigate the antifungal activity of zingiberoffcinale (Ginger) on Candida albicans. Candida albicans (PTCC 5027, ATCC10231) was obtained from Iranian microbial collection and was confirmed by Germ Tube formation test. Ethanol ginger extract was prepared. The results showed that the ethanol extract was effective on Candida albicans. This study indicates that ginger extract might have promising resultson oral candidiasis[a]

In other study Paradol, which was derived from ginger root and certain zingiberaceae plants, protected mouse skin from a tumor inducing agent, and showed dose-dependent cytotoxicity in an oral carcinoma cell line (KB), with specific features of caspase-3-mediated apoptosis. Viable KB cells were reduced in number to less than 50% of untreated control when incubated with 50 lM [6]-paradol for 48 h. In addition, an ethanol extract of ginger mediated anti-tumor promoting effects, decreased the number of tumors in a Sencar mice skin tumor model[20].

In a study Acyclovir-resistant clinical isolates of herpes simplex virus type 1 (HSV-1) were analyzed in vitro for their susceptibilities to essential oils of ginger, thyme, hyssop, and sandalwood. The active components of essential oils might consist of lipophilic carbohydrates that interact with the lipid membrane. These antibacterial active substances might exhibit similar activities against viral envelopes. Acyclovirresistant clinical isolates were significantly inhibited by the essential oils, and the titers of HSV were reduced by 95.9% to 99.9%. Essential oils act by inactivating HSV before it enters the cell. The effective dosage for a systemic application of essential oils is rather high and leads to cytotoxic effects. Furthermore, a short-term systemic bioavailability makes a systemic application unlikely [21] Aqueous ginger extracts which was used to check antimicrobial activity

found to be efficient[22]. For treatment of tooth ache [11] As a sialogogue, to promote salivation [18]

Conclusion

Various pharmacological properties and possible clinical use of ginger is discussed. Further human studies will have to be done to validate the use ginger as a therapeutic drug. With its numerous medical uses ginger promises to be an effective therapeutic agent for various disease in the future.

Corresponding Author

Arvind. M,

Professor, Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

Email: arvindmuthukrishnan@yahoo.com

Mobile Number: +91 9444303303

References

1. Baliga MS, Haniadka R, Pereira MM, D'Souza JJ, Pallaty PL, et al.Update on the chemopreventive effects of ginger and its phytochemicals. CritRev Food Sci Nutr.2011;51: 499-523.

2. Baliga MS, Haniadka R, Pereira MM, Thilakchand KR, Rao S, et al.Radioprotective effects of *Zingiberofficinale*Roscoe (ginger): Past, presentand future. Food Funct. 2012;3: 714-723.

3. Chrubasik S, Pittler MH, RoufogalisBD*Zingiberisrhizoma*: Acomprehensive review on the ginger effect and efficacy profiles. Phytomedicine. 2005;12: 684-701.

4. Ali BH, Blunden G, Tanira MO, NemmarASomephytochemical,pharmacological and toxicological properties of ginger (*Zingiberofficinale*Roscoe): A review of recent research. Food Chem Toxicol. 2008;46: 409-420.

5. Palatty PL, Haniadka R, Valder B, Arora R, BaligaMS Ginger in theprevention of nausea and vomiting: A review. Crit Rev Food Sci Nutr.2013;.53: 659-669.

6. Haniadka R, Rajeev AG, Palatty PL, Arora R, BaligaMS *Zingiberofficinale*(ginger) as an anti-emetic in cancer chemotherapy: A review. J AlternComplement Med. 2012;18: 440-444.

7. Haniadka R, Saldanha E, Sunita V, Palatty PL, Fayad R, et al. A reviewof the gastroprotective effects of ginger (*Zingiberofficinale*Roscoe). FoodFunct. 2013;4: 845-855.

8. Bordia A., Verma S.K., Srivastava K.C., "Effect of ginger (ZingiberofficinaleRosc.) and fenugreek (Trigonella foenumgraecum L.) on bloodlipids, blood sugar and platelet aggregation in patients with coronaryartery disease", Prostagtandins, Leukotrienes and Essential Fatty Acids. 1997;56 (5): 379-384,

9.Kikuzaki H., Nakatani N., "Cyclic diarylheptanoids from rhizomes ofzingiberoffcinale", Phytochemistry.1996;43 (1): 273-277,

10.Stoilova I., Krastanov A., Stoyanova A., Denev P., Gargova S., "Antioxidantactivity of a ginger extract (Zingiberofficinale)", FoodChemistry. 2007;102: 764-770,

11. Mallikarjuna K, SahityaChetan P, Sathyavelu Reddy K, RajendraWEthanol toxicity: rehabilitation of hepatic antioxidant defense system withdietary ginger. Fitoterapia. 2008;79: 174-178.

12. PoliG Liver damage due to free radicals. Br Med Bull. 1993;49: 604-620.

13. Baliga MS, Jagetia GC, Rao SK, BabuK Evaluation of nitric oxidescavenging activity of certain spices *in vitro*: A preliminary study. Nahrung.2003;47:261-264.

14. Jagetia G, Baliga M, VenkateshP Ginger (*Zingiberofficinale*Rosc.),A dietary supplement, protects mice against radiation-induced lethality:mechanism of action. Cancer BiotherRadiopharm. 2004;19: 422-435.

15. Al-Tahtawy RHM, El-Bastawesy AM, Monem MGA, Zekry ZK, Al-MehdarHA,et al. Antioxidant activity of the volatile oils of *Zingiberofficinale*(ginger).Spatula DD. 2011;1: 1-8.

16. Krishnakantha TP, Lokesh BR Scavenging of superoxide anions byspice principles. Indian J Biochem Biophys. 1993;30: 133-134.

17. Aeschbach R, Löliger J, Scott BC, Murcia A, Butler J, et al. Antioxidantactions of thymol, carvacrol, 6-gingerol, zingerone and hydroxytyrosol. FoodChem Toxicol.1994;32: 31-36.

18. Shin SG, Kim JY, Chung HY, JeongJC Zingerone as an antioxidantagainstperoxynitrite. J Agric Food Chem.2005;53: 7617-7622

.

19. Ippoushi K, Azuma K, Ito H, Horie H, HigashioH [6]-Gingerolinhibitsnitric oxide synthesis in activated J774.1 mouse macrophages and preventsperoxynitrite-induced oxidation and nitration reactions. Life Sci. 2003; 73: 3427-3437.

20. Shati AA, ElsaidFG Effects of water extracts of thyme *(Thymus vulgaris)*and ginger (*Zingiberofficinale*Roscoe) on alcohol abuse. Food Chem Toxicol. 2009;47: 1945-1949.

21. Bhandari U, Shamsher A, Pillai KK, Khan MSY Antihepatotoxicactivityof ginger ethanol extract in rats. Pharm Biol. 2003;41: 68-71.

22. Ajith TA, Hema U, Aswathy MS *Zingiberofficinale*Roscoe preventsacetaminophen-induced acute hepatotoxicity by enhancing hepatic antioxidantstatus. Food Chem Toxicol.2007; 45: 2267-2272.

23. Vitalis EC, Chukwemeka R, Philippe ME, ChinonsoNC Effectsof*Zingiberofficinale*on liver function of mercuric chloride-induced hepatotoxicityin adult Witar rats. Electron J Biomed.2007;3: 40-45

24. Khaki AA, Khaki A Antioxidant effect of ginger to prevents lead-inducedliver tissue apoptosis in rat. J Med Plants Res.2010;4: 1492-1495.

25. Yemitan OK, IzegbuMC Protective effects of *Zingiberofficinale*(Zingiberaceae) against carbon tetrachloride and acetaminophen-inducedhepatotoxicity in rats. Phytother Res 20: 997-1002.

26. Verma RJ, AsnaniV Ginger extract ameliorates parabeninducedbiochemical changes in liver and kidney of mice. Acta Pol Pharm . 2007;64: 217-220.

27. El-Sharaky AS, Newairy AA, Kamel MA, EwedaSM Protective effect ofginger extract against bromobenzene-induced hepatotoxicity in male rats. Food
Chem Toxicol. 2009;47: 1584-1590.

28. Aggarwal BB and ShishodiaS.Molecular targets of dietary agentsforprevention and therapy ofcancer. Biochem Pharmacol. 2009;71(10):1397-1421.

29. Miyoshi N, Nakamura Y, Ueda Y, Abe M, Ozawa Y, Uchida K and OsawaT.Dietaryginger constituents, galanals A and B, are potent apoptosis inducers
in Human T lymphoma Jurkatcells. Cancer Lett. 9-25-
2003;199(2):113-119.

30. Shukla, Y and Singh M. Cancer preventive properties of ginger: a brief review. Food ChemToxicol. 2007;45(5):683-690.

31. Wang CC, Chen LG, Lee LT and YangLL. Effects of 6-gingerol, an antioxidant from ginger, on inducingapoptosis in human leukemic HL-60 cells.In Vivo. 2003; 17(6):641-645.

32. Mahady,G B, Pendland SL, Yun GS,Lu ZZ and Stoia A. Ginger (Zingiberofficinale Roscoe) and the gingerolsinhibit the growth of Cag A+ strains of Helicobacter pylori. Anticancer Res.
2003;23(5A):3699-3702.

33. Nurtjahja-Tjendraputra E, Ammit AJ, Roufogalis BD, Tran VH and Duke CC. Effective anti-platelet and COX-1 enzyme inhibitors from pungent constituents of ginger. Thromb Res.2003;111(4-5):259-265.

34. Guh JH, KO FN, Jong TT and Teng CM. Antiplatelet effect of gingerolisolated from Zingiberofficinale. J Pharm. Pharmacol. 1995;47(4):329- 332.

35. Srivastava KC. Isolation and effects of some ginger components on platelet aggregation and eicosanoid biosynthesis. Prostaglandins LeukotMed. 1986;25(2-3):187-198.

36. Bhattarai S, Tran VH and Duke CC. The stability of gingerol and shogaol in aqueous solutions. J Pharm Sci. 2001;90(10):1658-1664.

37. Yamahara J, Rong HQ and Iwamoto M. Active components of ginger exhibiting anti-serotonergic action. PhytotherapyRes.1989;3(2):70-71. 38. Huang Q, Iwamoto M and Aoki S.Anti-5hydroxytryptamine3, effect of galanolactone, diterpenoid isolated from ginger. Chem Pharm
Bull. 1991;39(2):397-399

39. Srivastava KC and Mustafa T. Ginger(Zingiberofficinale) and rheumatic disorders. Med Hypotheses. 1989; 29(1):25-28.

40. Chrubasik S, Pittler MH and Roufogalis BD. Zingiberisrhizoma: acomprehensive review on the ginger effect and efficacy profiles. Phytomedicine. 2005;12(9):684-701.

41. Thomson M, Al Qattan, KK Al SawanSM,Alnaqeeb MA, Khan I and Ali M. The use of ginger (ZingiberofficinaleRosc.) as a potential anti-inflammatory and antithrombotic agent.Prostaglandins LeukotEssentFatty Acids. 2002;67(6):475-478.

42. Al Yahya, Rafatullah MA, MossaS,Ageel JS, Parmar AMNS, and Tariq M. Gastroprotective activity of ginger zingiberofficinalerosc., in albino rats. Am J Chin Med. 1989;17(1-2):51-56.

43. Desai HG, Kalro RH and Choksi AP. Effect of ginger & garlic on DNA content of gastric aspirate. Indian J Med Res. 1990;92:139-141.

44. Bordia A, Verma SK and SrivastavaKC. Effect of ginger (Zingiberofficinale Rosc.)and fenugreek (Trigonella foenumgraecum L.) on blood lipids, blood sugar and platelet aggregation in patients with coronary artery disease. Prostaglandins LeukotEssent Fatty Acids.1997;56(5):379-384.

45. Miri P, Bae J and Lee DS.Antibacterial activity of [10]-gingeroland [12]-gingerol isolated from ginger rhizome against periodontal bacteria. Phytothery Res. 2008; 22:1446-1449.

46. Funk JL, Frye JB, Oyarzo JN, Timmermann BN. Comparative Effects of Two Gingerol-Containing Zingiberofficinale Extracts on Experimental Rheumatoid Arthritis. J Nat Prod. 2009; 72:403-407.

A Review on Effect of Clonazepam in Burning Mouth Syndrome

S.Renuka[1],M.Sankari[2],
Graduate Student[1],Professor[2],Department of Periodontics,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Burning mouth syndrome (BMS) is a chronic or recurrent burning sensation in the mouth without an obvious cause. BMS is also named as stomatodynia, stomatopyrosis, glossopyrosis, glossodynia, sore mouth, sore tongue and oral dysesthesia.The prevalence of symptoms will be more in diabetic patients. This discomfort may affect the tongue, gums, lips, inside of your cheeks, roofof your mouth or widespread areas of your whole mouth. This review is to discuss the effect of clonazepam in burning mouth syndrome management.

Symptoms of BMS are relieved whenthe underlying medical condition, such as diabetes which is linked with cardiovascular, peripheral vascular, cerebrovascular, renal and psychological and ocular diseases [1] or yeast infection, is treated. Since BMS is a complex pain disorder, the treatment may not be constant for all patients. Drugs majorly used in treatment of BMS include Nortriptyline, Oral and topical clonazepam, Gabapentin, Pregabalin and alpha lipoic acid. Clonazepam, a benzodiazepine also widely used in the treating various chronic pain syndromes.This review elaborates the effect of clonazepam in the management of burning mouth syndrome.

Key Words*:clonazepam, pain, syndromes, sore mouth, sensation.*

Introduction

Burning mouth disorder (BMS) is an idiopathic condition described by a consistent burning, stinging or itching sensation in the tongue, with or without expansion to the lips, cheeks and oral mucosa.BMS is also called as glossodynia, glossopyrosis, glossalgia, stomatodynia, stomatopyrosis, sore tongue and mouth, burning tongue, oral or lingual parasthesia and oral dysesthesia[2], which occurs most commonly in peri and postmenopausal women.Theetiologyof BMS is unknown but it is thought to be multi-factorial, involving various local, systemic, and/or psychogenic causes [3].The risk factors for developing BMS includes thyroid disease, psychiatric illnesses, oral infections, drug use, dental treatment, vitamin/mineral deficiencies [4,5], Parkinson's disease, and chronic medical conditions including gastrointestinal and urogenital diseases[6,7-10]. Influenced patients regularly give different oral objections, including burning, xerostomia and taste modifications. BMS can appear suddenly or develops gradually over a period of time and the burning sensation become severe when you scald your mouth.BMS will not consistently associated with certain conditions such as chronic anxiety or depression, various nutritional deficiencies, type 2 diabetes (formerly known as non-insulin-dependent diabetes) and changes in salivary function but the treatment for these conditions has little impact on burning mouth symptoms. Recent studies have pointed to several cranial nerves dysfunction associated with taste sensation as a possible cause of burning mouth syndrome [11].BMS directly or indirectly affect the quality of the patient's life. Hence, management of this syndrome is very essential to prevent systemic dysfunctions. This review focuses on the effect of clonazepam in management of burning mouth syndrome.

Etiology

The cause of burning mouth syndrome can be classified as either primary or secondary.

Primary Burning Mouth Syndrome

Essential Burning mouth disorder When no clinical or lab variations from the norm can be recognized, the condition is called primary or idiopathic burning mouth disorder. Some examination proposes that idiopathic burning mouth disorder is identified with issues with taste and sensory nerves of the peripheral or central nervous system.

Secondary Burning Mouth Syndrome

Sometimes burning mouth syndrome is caused by an underlying medical condition. In these cases, it's called secondary burning mouth syndrome. It comprises ofdry mouth (xerostomia) , Nutritional deficiencies, such as a lack of iron, zinc, folate (vitamin B-9), thiamin (vitamin B-1), riboflavin (vitamin B-2), pyridoxine (vitamin B-6) and cobalamin (vitamin B-12), ill fitting dentures, allergic substances, GERD, Oral habiks like bruxism, diabetes, hypothyroidism, excessive mouth irritation, anxiety, certain medications, other oral conditions, such as a fungal infection of the mouth (oral thrush), an inflammatory condition called oral lichen planus or a condition called geographic tongue that gives the tongue a map-like appearance.

Classification

Lamey and Lewis (1989) classified BMS into three types depending on the intensity of pain [12].

1. Type 1 (35%) is characterized by patients having burning every day. The sensation of burning will be absent on waking and as the day continues, being maximal at night.This sort might be connected to systemic issue, for example, nutritional deficiencies and endocrine issue.
2. Type 2 (55%)Type 2 (55%) is characterized by burning sensation that happens each day, is present on arousing and regularly makes nodding off around evening time.This subgroup of patients frequently report mood changes, modifications in dietary patterns, and diminished yearning to mingle, which appear to be inferable from an adjusted rest design 3
3. Type 3 (10%)is described by intermittent sensation, show just on some days, with copying influencing irregular destinations, for example, the floor of the mouth, buccal mucosa, and throat. These patients frequently show tension and unfavourably susceptible responses, especially to nourishment added substances.

Pathophysiology

The pathophysiology of burning mouth syndrome (BMS) is complicated.It was originally considered a psychogenic illness; however, a neuropathic mechanism for BMS is currently

favoured. This is based on objectively measured abnormalities of physiologic responses of the trigeminal nerve in BMSpatients[13, 14].

The etiology is multifactorial with mounting evidence for a physiological basis. Axonal degenerative changes have been demonstrated in glossal terminal nerve fibres and sensory changes have been shown to be present in burning mouth patients, particularly perception of heat, cold, taste and nocioceptive stimuli [15-17]. Variations from the norm in trigeminal somatosensory evoked possibilities have been exhibited also [18]. This strongly suggests that there is a dysfunction of the small diameter afferent sensory fibres in burning mouth syndrome.

Imaging studies in patients have also suggested central nervous system changes [19, 20]. More recently, an enticing hypothesis has been proposed that burning mouth syndrome is associated with an alteration of gonadal, adrenal and neuroactive steroid levels. Woda et al. suggested that chronic anxiety or stress results in a dysregulation of adrenal steroids, a reduction in adrenal steroids will, in turn, lead to an altered production of neuroactive steroids in skin, mucosa and the nervous system [21].

The relationship with menopause is captured by the suggestion that the dramatic fall in gonadal steroids that occurs at that time further alters the production of neuroactive steroids. This “perfect storm” results in neurodegenerative alterations in small nerve fibres and areas of the brain related to somatic sensation. Thus explaining the peripheral and central changes already demonstrated in this condition [22-23]. There is aevidenceofhistopathological changes in nociceptive fibres in BMS patients [25]. However, a literature review by Galli et al demonstrated that both uneasiness and dejection may fundamentally add to the development of BMS [26].

A study by Mo et al indicated a neuropathic source for BMS, finding a loss of thermal sensitivity in individuals with this condition. Perception in different conditions has demonstrated that when a sensory circuit loses afferent signals that hyperactivity may bring about hallucinatory sensations. It would tend to account both for pain and for gustatory aggravations in burning mouth disorder (BMS).

Metallic or sharp tastes are viewed as symptomatic indications of an under animated gustatory circuit while under stimulated sensory circuitry shows burning sensations. The

cause of this proposed neuropathy is unknown.Studies focusing on trigeminal nerve alterations have found hypersensitivity and hyposensitivity as well as large and small fibres neuropathy [13, 14, 27, and 28]. Xerostomia in BMS is likely identified with neuropathy, as opposed to glandular dysfunction. In studies of burning mouth syndrome (BMS) patients in comparison to controls, no differences in salivary quantity or flow have been identified [29, 30].

BMS has been related with oral parafunctional propensities, for example, bruxism, holding, and tongue pushing. The bruxism and clenching are increased with anxiety, which is also associated with BMS. In spite of the fact that psychogenesis is no longer viewed as the essential cause, it mightexacerbate symptoms. Whether BMS has a neurologic abnormality in common with parafunctionalbehaviors has yet to be adequately investigated.

Burning mouth disorder (BMS) is additionally connected with a higher occurrence of gastrointestinal and urogenital illness, the noteworthiness of which is still unclear [31].

Clinical Features

A typical patient with BMS is postmenopausal woman of age 4th–6th decades with various medical comorbidities [32]. Clinical presentations may fluctuate as a few patient surgeries can be oligosymptomatic (pain and dysgeusia or xerostomia) or monosymptomatic (only pain) for the most part connected with dry mouth, sharp/metallic or modified taste [33]. The characteristics of BMS pain is chronic in nature with 4–6 months duration, burning or scalding type, sometimes itching sensation or numbness of the tongue, and other oral mucosal surfaces.[34]

The onset of pain can be either gradual and spontaneous or sudden and identified with a hastening occasion, for example, any dental procedure. Ordinarily, pain is confined to the tongue and some of the time including other mucosal surfaces likewise, for example, sense of taste, lip, buccal mucosa, and floor of the mouth. The pain will be continuous or intermittent, mild to moderate in intensity, restricted to the oral cavity and does not radiate to other regions of the face. The pain typically relieves on intake of food or liquids, which is in contrast to burning symptom in other diseases; in which the pain aggravates [35,36].

The pain is primarily two-sided and symmetrical on the front two-third of the tongue (71%–78%), followed by the dorsum and parallel outskirts of the tongue, the foremost piece of the

hard sense of taste, the labial mucosa, and gingiva, frequently showing up at a few areas [37]. A sensation of dry mouth with expanded thirst, taste changes, for example, an intense or metallic taste, loss of taste sensations.

Investigation

It is important to differentiate patients with primary BMS from those with symptoms caused by local or systemic factors (secondary BMS). The investigations for BMS are Haematological tests (CBC count, glucose levels, nutritional factors, autoimmune panel), oral cultures to confirm fungal, viral and fungal infections, salivary flow rates, patch tests for allergy testing, psychological or psychiatric assessment, salivary uptake scans, gastric reflux study, assessment of denture fit and function, Imaging techniques like MRI, CT scans and nuclear medicine[38].

Treatment

Studies on burning mouth syndrome (BMS) have focused more on cause than treatment. Currently, no definitive cure exists; many treatments have been tried with variable success. Before starting the treatment, it is important to know about nature of disease. Three treatment modalities are available. They are psychological therapy, topical medication, systemic medication. Psychological therapy includes cognitive behavioural therapy, group psychotherapy, electroconvulsive therapy, yoga, meditation and relaxation therapy. Topical prescription incorporates Clonazepam (1mg tablet to be broken down and hold in mouth), Lidocaine, Capsaicin, Benzydaminehydrochlorte, Doxepin cream, Lactoperoxidase oral flush, Sucralfate oral wash. Systemic medication includes Amitiptyline, Paroxetine, Amisulpride, Clonazepam,etc,.

The treatment options for BMS includes saliva replacement products, specific oral rinses or lidocaine, capsaicin, a pain reliever that comes from chili peppers, an anticonvulsant medication called clonazepam (Klonopin), certain antidepressants, medications that block nerve pain, cognitive behavioral therapy.

Clonazepam acts as an agonist of gamma-amino butyric acid (GABA) receptors. Systemic administration of clonazepam can result in inhibition of central nervous system functions producing an anticonvulsant action, light sedation, muscular relaxation, and tranquilizer effect. The topical use ofClonazepam can diminish the burning symptom without bringing

about the unfriendly impacts of its systemic use. Gremeau-Richard et al. [39] evaluated the efficacy of topical use ofClonazepam and found a greater decrease of pain score in Clonazepam-treated patients.

Ko et al and Amos et al. used a combined topical and systemic Clonazepam therapy. They found that mental status, beginning manifestation seriousness, and the nearness of xerostomia or potentially taste unsettling influence can fill in as result indicators ofClonazepam treatment for patients with BMS. They found that 80% of patients get more than a half diminishment in agony over the treatment time frame and 33% of the patients have finish torment determination, recommending that the consolidated topical and systemic Clonazepam organisation is a successful regimen for treatment of BMS [40,41].

Topical Clonazepam, a benzodiazepine, when applied as 0.5-1 mg 2-3 times day by day, acts by locally upsetting the neuropathologic component that underlies stomatodynia. Be that as it may, it diminishes the thickness or potentially ligand affinity towards benzodiazepine receptors. This, thusly, could cause unconstrained torment from the tissues concerned[42, 43]. Low measurements ofClonazepam dissolvable wafers accessible economically are better than tablets [44].

On systemic therapy, Clonazepam, a benzodiazepines, apply its impact by going about as a calming entrancing 0.25-2 mg measurements/day, 0.25 mg at sleep time, increment measurements by 0.25 each 4-7 days until oral burning is assuaged or reactions happen. As the measurements are expanded, pharmaceutical is taken in three isolated doses[42].

Mouthwash definitions have a few points of interest since the medication can be connected all the more consistently to the whole mouth, and a lovely flavour can be added to the mouthwash to improve consistence. Different medications as benzydamine hydrochloride [45] and capsaicin [46] have been tried as mouthwash details to treat BMS. Be that as it may, the outcomes did not turn out to be attractive as remedial choices. The utilization ofbenzydamine hydrochloride oral wash was strikingly not able to lessen the blazing sensation in BMS patients [45], while the capsaicin arrangement was successful however inadequately endured as 33% of the patients whined of extraordinary smoldering amid and for a couple of minutes after use of the capsaicin mouthwash [46].

Conclusion

Burning mouth syndrome remains a poorly understood common chronic oral-facial disorder which is difficult to diagnose and to treat. As BMS is associated with system symptoms, it is essential to manage the systemic cause. Medications are also used in the management of BMS but its effect is equal or less than the clonazepam. Most of the people are unaware about the burning mouth syndrome; many researches are needed to know about the effect of clonazepam in the management of BMS. This review study concluded that the use of the above topical, systemic and mouthwash aid in decreasing the amount of burning sensation associated with burning mouth syndrome produced in the oral cavity.

Corresponding Author
M.Sankari,
Professor,Department of Periodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162 Poonamallee High Road, Chennai-600077, Tamil Nadu, India.
Mobile Number: +91 9840285905
E-mail: msankari@gmail.com

References

1. Kesavamoorthy G, Singh AK, Sharma S, Kasav JB, Mohan SK, Joshi A. Burden of Diabetes Related Complications Among Hypertensive and Non Hypertensive Diabetics: A Comparative Study. Journal of clinical and diagnostic research: JCDR. 2015; 9(9):LC10.
2. Lisa A Drage, MD, Roy S Rogers III, MD. Burning mouth syndrome.Departmentof Dermatology, Mayo Clinic, 200 First Street SW, Rochester, MN 55905, USA.doi: http://dx.doi.org/10.1016/S0733-8635(02)00063-3.
3. Gurvits GE, Tan A. Burning mouth syndrome. World J Gastroenterol. 2013:19 (5):665-72.
4. Yilmaz Z, Renton T, Yiangou Y, Zakrzewska J, Chessell IP, Bountra C, Anand P. Burning mouth syndrome as a trigeminal small fibre neuropathy: Increased heat and capsaicin receptor TRPV1 in nerve fibres correlates with pain score. J ClinNeurosci. 2007;14:864–871.
5. Grushka M, Epstein JB, Gorsky M. Burning mouth syndrome: differential diagnosis. DermatolTher. 2002;15:287–291.

6. Netto FO, Diniz IM, Grossmann SM, de Abreu MH, do Carmo MA, Aguiar MC. Risk factors in burning mouth syndrome: a case-control study based on patient records. Clin Oral Investig. 2011;15:571–575.
7. Bergdahl M, Bergdahl J. Burning mouth syndrome: prevalence and associated factors. J Oral Pathol Med. 1999;28:350–354.
8. Grushka M. Clinical features of burning mouth syndrome. Oral Surg Oral Med Oral Pathol. 1987;63:30–36.
9. Scala A, Checchi L, Montevecchi M, Marini I, Giamberardino MA. Update on burning mouth syndrome: overview and patient management. Crit Rev Oral Biol Med. 2003;14:275–291.
10. Schiavone V, Adamo D, Ventrella G, Morlino M, De Notaris EB, Ravel MG, Kusmann F, Piantadosi M, Pollio A, Fortuna G, et al. Anxiety, depression, and pain in burning mouth syndrome: first chicken or egg? Headache. 2012;52:1019–1025.
11. Grushka, Miriam; Epstein, Joel B.; Gorsky, Meir. Burning Mouth Syndrome. American Family Physician .2002; 65(4): 615- 616.
12. Lamey PJ, Lewis MA. Oral medicine in practice: Burning mouth syndrome. Br Dent J. 1989;167:197–200.
13. Forssell H, Jaaskelainen S, Tenovuo O, et al. Sensory dysfunction in burning mouth syndrome. Pain. 2002; 99(1-2):41-47.
14. Grushka M, Sessle BJ, Howley TP. Psychophysical assessment of tactile, pain and thermal sensory functions in burning mouth syndrome. Pain. 1987; 28(2):169-84.
15. Formaker BK, Mott AE, Frank ME, The effects of topical anesthesia on oral burning in burning mouth syndrome. Ann NY Acad Sci,1998; 85(5): 776–780.
16. Grémeau-Richard C, Woda A, Navez ML, Attal N, Bouhassira D, Gagnieu MC, Topical clonazepam in stomatodynia: a randomizedplacebocontrolled study. Pain, 2004; 108(1/2): 51–57.
17. Yilmaz Z, Renton T, Yiangou Y, Zakrzewska J, Chessell IP, Bountra C, Burning mouth syndrome as a trigeminal small fibre neuropathy: increased heat and capsaicin receptor TRPV1 in nerve fibres correlates with pain score. J ClinNeurosci, 2007; 14(9): 864–871.
18. Gao S, Wang Y, Wang Z. Assessment of trigeminal somatosensory evoked potentials in burning mouth syndrome. Chin J Dent Res,2000; 3(1): 40–46.

19. Jääskeläinen SK, Rinne JO, Forssell H, TenovuaO, Kaasinen V, Sonninen P,. Role of the dopaminergic system in chronic pain – a fluordopaPET study. Pain,2001; 90(3): 257–260.
20. Hagelberg N, Fossell H, Aalto S, Luutonen S, Nagren K, Jääskeläinen S. Striatal D1 and D2 receptors in burning mouth syndrome. Pain,2003; 101(1/2): 149–154.
21. Woda A, Dao T, Grémeau-Richard C. Steroid dysregulation and stomatodynia (burning mouth syndrome). J Orofacial Pain,2009; 23(3): 202–210.
22. Zakrzewska JM, Critical commentary 1: steroid dysregulation and stomatodynia (burning mouth syndrome). J Orofacial Pain, 2009; 23(3): 211–213.
23. El-Etr M. Critical commentary 3: steroid dysregulation and stomatodynia (burning mouth syndrome). J Orofacial Pain,2009; 23(3): 216–218.
24. Eliav E, Nasri-Heir C. Critical commentary 2: steroid dysregulation and stomatodynia (burning mouth syndrome). J Orofacial Pain,2009; 23(3): 214–215.
25. Beneng K, Yilmaz Z, Yiangou Y, McParland H, Anand P, Renton T. Sensory purinergic receptor P2X(3) is elevated in burning mouth syndrome. Int J Oral MaxillofacSurg. 2010 Apr 24.
26. Galli F, Lodi G, Sardella A, Vegni E. Role of psychological factors in burning mouth syndrome: A systematic review and meta-analysis. Cephalalgia. 2016 Apr 27.
27. Lauria G, Majorana A, Borgna M, et al. Trigeminal small-fiber sensory neuropathy causes burning mouth syndrome. Pain. 2005; 115(3):332-7.
28. Yilmaz Z, Renton T, Yiangou Y, et al. Burning mouth syndrome as a trigeminal small fibre neuropathy: Increased heat and capsaicin receptor TRPV1 in nerve fibres correlates with pain score. J ClinNeurosci. 2007; 14(9):864-71.
29. Loeb LM, Naffah-Mazzacoratti MG, Porcionatto MA, et al. Chondroitin sulfate and kallikrein in saliva: Markers for glossodynia. International Immunopharmacology. 2008; 8:1056-1058.
30. deMoura SA, de Sousa JM, Lima DF, et al. Burning mouth syndrome (BMS): sialometric and sialochemical analysis and salivary protein profile. Gerodontology. 2007; 24(3):173-6.
31. Friedman DI. Topirimate-Induced Burning Mouth Syndrome. Headache. 2010 Jun 18.
32. Brailo V, Vuéiaeeviae-Boras V, Alajbeg IZ, Alajbeg I, Lukenda J, Aeurkoviae M. Oral burning symptoms and burning mouth syndrome-significance of different variables in 150 patients. Med Oral Patol Oral Cir Bucal. 2006;11:E252–255.

33. 21. Tatullo M, Marrelli M, Scacco S, Lorusso M, Doria S, Sabatini R, Relationship between oxidative stress and “burning mouth syndrome” in female patients: A scientific hypothesis. Eur Rev Med Pharmacol Sci. 2012;16:1218–21.
34. López-Jornet P, Camacho-Alonso F, Andujar-Mateos P, Sánchez-Siles M, Gómez-Garcia F. Burning mouth syndrome: An update. Med Oral Patol Oral Cir Bucal. 2010;15:562–568.
35. Gurvits GE, Tan A. Burning mouth syndrome. World J Gastroenterol. 2013;19:665–672.
36. Tatullo M, Marrelli M, Scacco S, Lorusso M, Doria S, Sabatini R, et al. Relationship between oxidative stress and “burning mouth syndrome” in female patients: A scientific hypothesis. Eur Rev Med Pharmacol Sci. 2012;16:1218–1221.
37. Tanaka M, Kitago H, Ogawa S, Tokunaga E, Ikeda M, Tomita H. Incidence and treatment ofdysgeusia in patients with glossodynia. ActaOtolaryngolSuppl. 2002;546:142–5.
38. Kamala, KA, “Burning Mouth Syndrome.” Indian Journal of Palliative Care. 2016;22(1): 74–79.
39. Gremeau-Richard C, Woda A, Navez ML, Topical clonazepam in stomatodynia: a randomized placebo-controlled study Pain, 2004; 108: 51–57.
40. Ko JY, Kim MJ, Lee SG, Kho HS. Outcome predictors affecting the efficacy of clonazepam therapy for the management of burning mouth syndrome (BMS). Arch GerontolGeriatr 2012; 55: 755–61.
41. Amos K, Yeoh SC, Farah CS. Combined topical and systemic clonazepam therapy for the management of burning mouth syndrome: a retrospective pilot study. J Orofac Pain 2011; 25: 125–30.
42. Woda A, Navez ML, Picard P, Gremeau C, Pichard-Leandri E. A possible therapeutic solution for stomatodynia (burning mouth syndrome). J Orofac Pain 998;12(4): 272-8.
43. Gorsky M, Silverman S Jr, Chinn H. Clinical characteristics and management outcome in the burning mouth syndrome. An open study of 130 patients. Oral Surg Oral Med Oral Pathol 1991;72(2):192-5.
44. Patton LL, Siegel MA, Benoliel R, De Laat A. Management of burning mouth syndrome: systematic review and management recommendations. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 2007;103(39):1-13.

45. Sardella, Andrea, "Benzydamine hydrochloride oral rinses in management of burning mouth syndrome: a clinical trial." Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology , 1999;88(6): 683-686.
46. Silvestre, Francisco-Javier,"Applicationof a capsaicin rinse in the treatment of burning mouth syndrome." Med Oral Patol Oral Cir Bucal, 2012; 17(1): e1-e4.

A Review on Emerging Treatment Modalities for Oral Submucous Fibrosis

R.Sharmila [1],Vivek Narayan[2],M. S. Nivedhitha[3],
Graduate Student[1], Senior Lecturer[2], Department of Oral Medicine and Radiology,
Professor and Head[3],Department of Conservative Dentistry and Endodontics,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Oral submucous fibrosis (OSMF) is a chronic insidious disease characterised by juxtaepithelial inflammation and fibroelastic changes in lamina propria leading to stiffness of the oral mucosa. OSMF is attributed to areca nut chewing which contains arecoline and arecaidine. Treatment of OSMF has always been challenging since the current treatment options do not provide a significant relief for the patient.

Oral submucous fibrosis (OSF) is a chronic, insidious disease that affects the lamina propria of the oral mucosa and, as the disease advances, it involves tissues deeper in the submucosa of the oral cavity with resulting loss of fibroelasticity. The disease manifests with blanching and stiffening of the oral mucosa leading to limitation in opening of the mouth.

Oral submucous fibrosis is a disease of the oral cavity and regarded as a potentially malignant disorder. Numerous treatment modalities ranging from various drugs to behavioral therapy have been tried with inconsistent results with varying degrees of success reflecting low predictability, requiring further evaluation and standardization. Novel treatment modality such as Hyperbaric oxygen therapy, Oxitard capsules , spirulina , Lycopene , pentoxyfilline, Turmeric, placental extracts advocated intralesionally along with oral drugs like carotenoids , hyaluronidase,betamethasone and dexamethasone.

A wide range of treatment including drug management, surgical therapy, and physiotherapy have been attempted till date, with varying degrees of benefit, but none have been able to cure this disease. This is mainly due to the fact that the etiology of the disease is not fully understood and the disease is progressive in nature.

Key Words:*Fibrosis, curcumin, dexamethasone, pentoxyfilline and placental extracts.*

Introduction

Oral submucous fibrosis (OSF) is a chronic, insidious disease that affects the lamina propria of the oral mucosa and, as the disease advances, it involves tissues deeper in the submucosa of the oral cavity with resulting loss of fibroelasticity. The disease manifests with blanching and stiffening of the oral mucosa leading to limitation in opening of the mouth. The presence of fibrous bands in lips, cheeks and soft palate is a hallmark of the disease. Oral submucous fibrosis predominantly affects South, South Asian and East Asian populations and is seen in India, Pakistan, Bangladesh, Nepal, Sri Lanka, southern parts of China, Taiwan, Melanesia and Micronesia and in the pacific islands.

Oral submucous fibrosis is a chronic disease of the oral cavity characterized by inflammation and progressive fibrosis of the submucosal tissues.

In 1952, Schwartz coined the term atrophicaidiopathica mucosa oris to describe an oral fibrosing disease he discovered in 5 Indian women from Kenya(1). Joshi subsequently coined the termed oral submucous fibrosis (OSMF) for the condition in 1953(2).

The pathogenesis of the disease is not well established, but the cause of oral submucous fibrosis is believed to be multifactoria. Factors include areca nut chewing, ingestion of chillies, genetic and immunologic processes, nutritional deficiencies, and other factors like areca nut component of betel quid plays a major role in the pathogenesis of oral submucous fibrosis(3,4,5).Commercially available products such as pan masala, guthka, and mawa have higher concentrations of areca nut per chew and appear to cause oral submucous fibrosis more rapidly than self-prepared conventional betel quid, which contains smaller amounts of areca nut.

Among the areca alkaloids such as arecoline, arecadine, guvacoline, guvacine, arecoline is the main agent responsible for fibroblast proliferation. Under the influence of slaked lime ($Ca(OH)_2$, arecoline get hydrolyzed to arecadine, which has pronounced effects on fibroblasts. Areca flavonoids tannins and catechins can cause increased fibrosis by forming a more stable and non soluble collagen structure by inhibiting collagenase enzyme activity. Studies have shown that there is 1.5 fold increase in collagen production by OSMF fibroblasts and with the progression of disease type 3 collagen is completely replaced by type 1 collagen which is more resistant to degradation. Also there has been an excess of alpha 1 (1) chains relative to alpha 2(1) chains, suggesting an alteration of collagen molecule during

the disease progression. Recently a study done on human buccal fibroblasts showed an increased expression of an insoluble cytoskeleton protein (57kDa) called vimentin in OSMF patients under arecoline influence. This protein vimentin is primarily expressed by mesenchymal cells, during cell growth, and tumorigenesis. Thus elevated vimentin expression stimulated by arecoline in OSMF may be suggestive of transformational changes in buccal fibroblasts of OSMF patients.

The commonly used treatment modalities for oral sub mucous fibrosis are placental extracts advocated intralesionally along with oral drugs like carotenoids.hyaluronidase,betamethasone and dexamethasone.

The emerging treatment modalities Are hyperbaric oxygen therapy. oxitard capsules, Lycopene, spirulina, pentoxyfilline and curcumin. Aim of the study is to provide an update on the various treatment modalities available for oral submucous fibrosis.

Hyperbaric Oxygen Therapy

Hyperbaric Medicine is the clinical specialty using pressure higher than local atmospheric pressure (>1atm) to treat diseases or injuries inside a hyperbaric chamber, to derive therapeutic benefit from breathing gases, usually oxygen. Hyperbaric Oxygen Therapy—"HYPER" means increased and "BARIC" means pressure.

Hyperbaric oxygen therapy is an well established therapy for decompression sickness ,a hazard of scuba diving ,other conditions include serious infections, bubbles of air in blood vessels and wounds that won't heal as a result of diabetes or radiation injury.

In OSMF increased fibrosis is due to imbalance between activation of fibroblasts and reduced degradation of collagen leading to increased fibrosis and trismus. Conconi et al., found that exposure to HBOT at 2.5 ATA for 120 min enhanced apoptosis of mouse fibroblast cell line (6).It also reduced cell proliferation when skin fibroblasts were cultured in a high-glucose medium at 2.5 ATA for 90 min on three consecutive days (7).

It may induce apoptosis of lymphocytes or/and decrease lymphocytic proliferation so as to keep fibroblasts from activating cytokines. HBOT at 1.0 or 2.5 ATA for 30 and 60 min enhanced 3T3/J2 fibroblast cell growth while at 2.5 ATA for 120 min, it exerted a pro apoptotic effect. HBOT may potentially contribute to the inhibition of fibroblasts by reducing IL-1b and TNF-α production. HBOT may be useful in the treatment for OSF by promoting fibroblast apoptosis and inhibiting fibroblast activation.

Indication of HBOT in Dentistry

1. Osteoradionecrosis
2. Post radiotherapy cases
3. Mandibular Osteomyelitis, Chronic Refractory Osteomyelitis
4. Periodontal disease
5. Infected Implants.

Contra Indications

1. Absolute

a. Untreated tension pneumothorax.

2. Relative

a. Upper respiratory tract infection.

b. Asymptomatic pulmonary lesions seen on chest x-ray.

c. History of thoracic or ear surgery.

d. Pregnancy.

e. Claustrophobia.

Adverse Effects

1. Middle ear barotrauma is the most common complication of HBOT therapy, with an incidence of about 2%. Inner ear barotrauma is a very rare occurrence.
2. Sinus squeeze is the second most common complication of HBOT therapy.
3. Tooth pain can occur during compression or decompression and this typically follows dental treatment that has created an air space under a dental filling.
4. Pulmonary oxygen toxicity can occur in patients if exposed for prolonged periods.
5. Fire is the most common fatal complication of hyperbaric oxygen therapy.

Oxitard Capsules and Lycopene

The formulation of the oxitard capsules contains the extracts of Mangiferaindica, Withania somnifera, Daucuscarota, Glycyrrhizaglabra, Vitisvinifera, powders of Emblica officinalis and Yashadabhasma; and oils of Triticum sativum. Mangiferaindica is shown to have antibiotic, anti-asthamatic, antiseptic, antiviral, hypotensive, anti-emetic properties. Withania somnifera provides overall health and wellness with its anti-stress, anti-anxiety, anti-

inflammatory, anti-convulsive and anti-arthritic properties. Daucuscarota acts as a good antiseptic as it is a rich source of vitamin A. Glycyrrhizaglabra normalizes the hoarseness in voice and has immunomodulatory and anti-inflammatory properties.
Vitis vinifera has anti-inflammatory, astringent and an effect to curb the burning sensation. Emblica officinalis is a rich source of vitamin C and is a potent antibiotic. Yashadabhasma contains zinc which plays a significant role in protein synthesis, cell division and wound healing. Triticum sativum is a rich source of minerals and has an antioxidant property .Oxitard has shown significant improvement in mouth opening, tongue protrusion, pain with the lesion, difficulty in swallowing and speech.

Lycopene is a powerful antioxidant obtained from tomatoes. It has been shown to inhibit various types of cancers and have potent benefits in oral pre malignant lesion where it has been shown to modulate dysplastic changes(8).The interest in the various mechanisms of action of this particular carotenoid is relatively recent and has the highest singlet oxygen quenching capacity with high capability of quenching other free radicals
Newer studies highlight the benefit of this oral nutritional supplement at a daily dose of 16 mg. Mouth opening in 2 treatment arms was statistically improved in patients with oral submucous fibrosis. This effect was slightly enhanced with the injection of intralesional betamethasone (two 1-mL ampules of 4 mg each) twice weekly, but the onset of effect was slightly delayed(9).

Lycopene is an potent antioxidant present in tomatoes and pink grapefruit. Like beta-carotene, Lycopene belongs to the family of carotenoids. As an antioxidant, it is about twice as powerful as beta-carotene. Review of the various safety studies has shown no adverse effects when administered in high doses even up to 3 g/kg/day of dietary or formulated lycopene. Presently, there is no consensus about the precise dosage of Lycopene when administered as a pure compound but various clinical trials suggest a dose ranging from 13 to 75 mg/kg/day. It warrants further studies in the future to help standardize an optimal dosage of the drug. In contrast to other management modalities for OSMF, Lycopene offers a noninvasive option that yields significant amelioration of the symptoms and signs of the disease as demonstrated by the improved maximal mouth (inter-incisal distance) opening up to 69.56%($P<0.05$). (10)The main focus of research has been the antioxidant property of lycopene.

However, it has also been shown to exert its effect via other mechanisms such as gene function regulation, gap junction communication, hormone and immune modulation, carcinogen metabolism, and modulation of metabolic pathways involving phase 2 drugmetabolizing enzymes. Lycopene is usually administered as a pill/ tablet per oral.This study was conducted to determine the frequency of positive and negative responses of lycopene in the treatment of oral submucous fibrosis.(11,12)

Lycopene is a carotenoid. They are natural pigments and antioxidant properties. Its profound benefits have been demonstrated with precancerous lesions such as leukoplakia.Mechanism of action lycopene shows abnormal fibroblasts inhibition in OSMF. It also regulates lymphocyte resistance to stress and suppresses infammatory response. This is the first line of therapy to OSMF. Lycopene exhibits the highest physical quenching rate constant with singlet oxygen.

Spirulina

Spirulina is blue green algae with rich natural source of proteins, carotenoids and other micronutrients (13). It has been primarily assessed in treating leukoplakia with promising results. The chemopreventive capacity to reverse precancerous lesions of spirulina is attributed to the antioxidant property with high amount of beta carotene and superoxide dismutase.Highly significant results were obtained with all three parameters namely burning sensation, mouth opening and tongue protrusion.

Recently, Shetty et al (14) have delineated the role of Spirulina as an adjuvant therapy in the management of OSMF. They conducted an interventional study on 40 cases of OSMF. The treatment groups were divided into two of twenty each, with the first group receiving Spirulina 500 mg BID and biweekly intralesional injections of steroid Betamethasone 4 mg/mL for a period of 3 months and the second group receiving placebo capsules BID and biweekly intralesional steroid injection of Betamethasone 4 mg/mL for 3 months. They found that Spirulina was effective in alleviating the burning sensation in patients probably due to its beta-carotene, phenolic acid, tocopherol, and micronutrient content. Additionally, they stated that use of beta-carotene systemically and topically helps in improving the epithelial integrity

and brings about redifferentiation of dysplastic epithelium. They suggested that Spirulina can be advocated as an adjuvant therapy in the early treatment of OSMF.

Similarly, Mulk et al (15) studied the effect of Spirulina (500 mg) and Pentoxifylline (400 mg). Each group (20 cases of OSMF per group) used the individual formulations twice daily for a period of 4 months. They obtained significant results in relation to burning sensation, mouth opening, and tongue protrusion. Efficacy wise, however, they were similar in respect to mouth opening and protrusion of the tongue. Although even with this study, the reduction in burning sensation was more in the Spirulina group. Further, they highlighted that no adverse effects were noted in the patients using Spirulina.

Another study, using two natural antioxidants was performed by Patil et al. (16) They studied 42 cases of OSMF by dividing them equally in two groups, first the Spirulina group (500 mg Spirulina in two divided doses for 3 months) and second the aloe vera group (5 mg gel for topical application thrice daily for 3 months) with an additional follow-up period of 2 months. In concordance with the Mulk study, they found no side effects in both the study groups. Both the study drugs brought about a similar reduction in burning sensation. Spirulina brought about significant clinical improvements in mouth opening and ulcers/erosion/vesicles. They put forth that Spirulina brings about better treatment outcomes as compared to aloe gel.

Similarly, Patil et al (17) also evaluated the efficacy of Spirulina and lycopene in 68 OSMF patients. They administered two divided doses of 500 mg of Spirulina in group I for a period of 3 months and two divided doses of lycopene 4 mg for the same time period. They observed that lycopene brought about improvements in the clinical symptoms, such as mouth opening, whereas Spirulina improved ulcers/erosion/vesicles in the OSMF patients. They also observed similar effects in terms of alleviation of pain and burning sensation and thus put forth that both the drugs can act as useful modality of treatment in cases of OSMF.

Pentoxyfilline

Trental (pentoxifylline) is a xanthine derivative. It belongs to a group of vasoactive drugs which improve peripheral blood flow and thus enhance peripheral tissue oxygenation. The mechanism by which Trental achieves this effect has not been determined, but it is likely that the following factors are involved:

- Trental, as with other xanthine derivatives, relaxes certain smooth muscles including those of the peripheral vessels, thus causing vasodilatation or preventing spasm. This action, however, may have a limited role in patients with chronic obstructive arterial disease when peripheral vessels are already maximally dilated.
- Trental improves flexibility of red blood cells. This increase in the flexibility of red blood cells probably contributes to the improvement of the ability of blood to flow through peripheral vessels (haemorheologic action). This property was seen during in vitro and in vivo experiments with Trental but the correlation between it and the clinical improvement of patients with peripheral vascular diseases has not been determined.
- Trental promotes platelet deaggregation.Improvement of red blood cell flexibility and platelet deaggregation contribute to the decrease in blood viscosity(18).

Pentoxifylline is a tri substituted methyl xanthine derivative, with numerous biologic activities. It is termed as a "rheologic modifier." It improves microcirculation and decreases aggregation of platelet as well as granulocyte adhesion. It increases leukocyte deformability as well as inhibits neutrophil adhesion and activation. It increases production of prostaglandin E2 and prostaglandin I2 by vascular epithelium and maintain cellular integrity and homeostasis after acute injury. In addition, it causes degranulation of neutrophils, promotes natural killer cell activity and inhibits T-cell and B-cell activation. Pentoxifylline has also shown a direct effect on inhibiting burn scar fibroblasts. Haddad *et al*. (19) treated 34 radiation-induced superficial fibrotic lesions of the skin with pentoxifylline and Vitamin E for 3 months and reported a significant effect in the improvement of radiation-induced fibrosis. It has been postulated that pentoxifylline may be a valuable drug for reducing burn scar contractures. It has been shown to be effective in various medical disorders such as stroke, aphthous stomatitis, peripheral arterial occlusion, cerebrovascular insufficiency and pre tibialmyxedema.

Vasodilators like pentoxifylline have vasodilating properties and hampered mucosal vascularity in OSMF could be increased by the use of pentoxifylline. Pentoxifylline sup-presses leucocyte function and alters broblast physiology and stimulates brinolysis. In one study the effect of pentoxyfilline was studied on the clinical and pathologic course of OSMF. This investigation was conducted as a randomized clinical trial incorporating a control group in comparison to pentoxifylline test cases The authors concluded that pentoxifylline can be

used as an adjunct therapy in the management of oral submucous fibrosis. In another study Pentoxifylline 400mg for a period of 7 months, showed an improvement in total signs and symptoms of OSMF. No significant side effects were observed.

The use of Trental (pentoxifylline) is contraindicated in:

- Patients who are hypersensitive to pentoxifylline or other xanthines such as caffeine, theophylline and theobromine or to any ingredient in the formulation or component of the container.
- Patients with acute myocardial infarction.
- Patients with severe coronary artery disease when, in the physician's judgement, myocardial stimulation might prove harmful;
- Patients with hemorrhage (e.g. extensive retinal bleeding) or at risk of increased bleeding;
- Patients with peptic ulcers.

In a pilot study, 14 test subjects with advanced oral submucous fibrosis given pentoxifylline at 400 mg 3 times daily were compared to 15 age- and sex-matched diseased control subjects. Statistical improvement was noted in all measures of objective (mouth opening, tongue protrusion, and relief from fibrotic bands) and subjective (intolerance to spices, burning sensation of mouth, tinnitus, difficulty in swallowing, and difficulty in speech) symptoms over a 7-month period(20).

In another study they showed a significant ($P < 0.05$) improvement in mouth opening, tongue protrusion, difficulty in speech and swallowing, pain associated with the lesion and burning sensation. Most side effects with pentoxifylline involve the central nervous system and the gastro-intestinal tract. The most common gastro-intestinal problems include dyspepsis, nausea and/or vomiting. A few may also report bloating, flatus and bleeding. The side effects related to the central nervous system include dizziness, headache, tremor, anxiety and confusion. 8 patients in the Group A experienced nausea, bloating of the stomach, dyspepsia and anxiety. The symptoms were mild in nature and resolved within a few days, without the need for cessation of the drug. This was similar to the findings of Rajendran *et al*. and Mehrotra *et al*. (21,22) The side effects are said to be dose related and can be minimized with reduction of dose.

Curcumin

Curcumin is a nonnutritive, nontoxic polyphenol natural product found in turmeric, a spice that has been used for centuries in India and elsewhere as an herbal medicinal treatment of wounds, jaundice, and rheumatoid arthritis. Sharma (1976), Ruby et al. (1995), and Sugiyama et al. (1996) studied the antioxidative properties of curcumin and its three derivatives (demethoxy curcumin, bisdemethoxy curcumin, and diacetyl curcumin). The authors demonstrated that these substances provide a protection of hemoglobin from oxidation at a concentration as low as 0.08 mM, except the diacetyl curcumin, which has little effect in the inhibition of nitrite-induced oxidation of hemoglobin. The effect of curcumin on lipid peroxide (LPO) has also been studied in various models by several authors. Curcumin is a good antioxidant and inhibits LPO in rat liver microsomes, erythrocyte membranes, and brain homogenates. The LPO has a main role in the inflammation, in heart diseases, and in cancer.

The use of curcumin in treating OSF in humans. Curcumin inhibits proliferation, disrupts the cell cycle, induces apoptosis, and decreases the expression levels of type I and III collagen; and it clearly demonstrates curcumin'santifibrotic potential in vitro. Administration of curcumin powder offers protection benzopyrene induced increase in micro nuclei in circulating lymphocytes and its excellent scavenger of free radical in vitro. (23,24) Curcumin oil and curcumin leoresin both act synergistically in vivo to offer protection against DNA damage. (25). Curcumin modulates the inflammatory response by.

a) Down-regulating the activity of cyclooxygenase-2 (COX-2), lipoxygenase, and inducible nitric oxide synthase (iNOS) enzymes.

b) Inhibits the production of the inflammatory cytokines, tumor necrosis factor-alpha (TNF-alpha), interleukin (IL) −1, −2, −6, −8, and −12, monocyte chemoattractant protein (MCP), and migration inhibitory protein.

Agarwal N et al. (26) conducted a study to check the efficacy of turmeric in 30 OSMF patients. An improvement in mouth opening and burning sensation was noticed. It was hypothesized that curcumin exerts anti-inflammatory activity by inhibiting a number of different molecules that participates in the process of inflammation. They also exhibit fibrinolytic property due to its ability to inhibit lipid peroxidation and check cellular proliferation, thereby reducing the rate of collagen synthesis.

Another study conducted by Deepa DA et al. (27) to evaluate the efficacy of curcumin and turmeric dispensed in two forms namely curcumin capsules and turmeric oil in 48 patients with OSMF. Statistically significant improvement was observed in the clinical signs and symptoms of patients treated with curcumin and turmeric oil. It showed anti-inflammatory action and fibrinolytic properties.

Yadav M et al. (28) conducted a study for comparison of curcumin with intralesional steroid injections in osmf patients. Improvement of burning sensation, interincisal distance and tongue protrusion was evaluated on a weekly basis and it was found that there was marked improvement in burning sensation, intreincisal distance and tongue protrusion.

BalwantRai(29) conducted a study to know the possible mechanism of action for curcumin in pre-cancerous lesions and condition based on serum and salivary markers of oxidative stress. It was found that curcumin mediates its anti pre-cancer activities by increasing levels of vitamins C and E and preventing lipid peroxidation and DNA damage. This could be due to curcumin-induced production of vitamins C and E and preventive DNA damage by decreasing the oxidation stress. This suggests that the anti-precancerous effects of curcumin are mediated through pro-oxidant and anti-oxidant pathways.

Zhang SS et al. (30) showed in their study that curcumin inhibits proliferation, disrupts the cell cycle, induces apoptosis, and decreases the expression levels of type I and III collagen; confirming its potential therapeutic value in OSMF patient.

Another study showed that use of curcumin in osmf significantly reduce connective tissue growth factor which is associated with the onset and progression of OSMF. (31)

Various studies have been conducted over a period of time showing the efficacy of curcumin in treating the osmf patients. Constant use of curcumin showed marked improvement in osmf patients due to its pharmacological activities. Further studies need to be carried out to prove the efficiency of curcumin in treating OSMF.

Conclusion

Our review of the literature for treatment of OSMF yielded a spectrum of treatment modalities to manage OSMF. Numerous treatment modalities that have been implicated to cure the disease are the use of corticosteroids, hyaluronidase, placentrix, hyperbaric oxygen

therapy, oxitardcapsules ,lycopene, spirulina, pentoxyfilline and curcumin. Though, till date, there is no single method which can be used as the definitive treatment modality for OSMF. Recent literature proves that the combination of drugs produce effective results in the management of this disease. A more extensive clinical trials involving a greater number of cases and including more parameters are necessary to come to a conclusion about a particular modality in the management of OSMF.

Corresponding Author

M. S. Nivedhitha,

Professor and Head,Department of Conservative Dentistry and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077,Tamil Nadu, India.

Telephone Number- +91 9840912367

E-mail- nivea16@hotmail.com

Reference

1. Schwartz J. AtrophiaIdiopathica Mucosae Oris. London: Demonstrated at the 11th Int Dent Congress; 1962;16:189-197.
2. Para, childcount:1Joshi SG. Fibrosis of the palate and pillars. Indian J Otolaryngol. 1953. 4(1):167-169.
3. Liao PH, Lee TL, Yang LC, Yang SH, Chen SL, Chou MY. Adenomatous polyposis coli gene mutation and decreased wild-type p53 protein expression in oral submucous fibrosis: a preliminary investigation. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2001;92(2):202-7.
4. Chang MC, Chen YJ, Chang HH, Chan CP, Yeh CY, Wang YL, Cheng RH, Hahn LJ, Jeng JH. Areca nut components affect COX-2, cyclin B1/cdc25C and keratin expression, PGE2 production in keratinocyte is related to reactive oxygen species, CYP1A1, Src, EGFR and Ras signaling. PloS one. 2014;22;9(7):101.
5. para, childcount:2Pant I, Rao SG, Kondaiah P. Role of areca nut induced JNK/ATF2/Jun axis in the activation of TGF-β pathway in precancerous Oral Submucous Fibrosis. Sci Rep. 2016;6(6):343-344.

6. Conconi MT, Baiguera S, Guidolin D, Furlan C, Menti AM, Vigolo S, Belloni AS, Parnigotto PP, Nussdorfer GG. Effects of hyperbaric oxygen on proliferative and apoptotic activities and reactive oxygen species generation in mouse fibroblast 3T3/J2 cell line. Journal of investigative medicine. 2003;51(4):227.
7. Lin HI, Chu SJ, Perng WC, Wu CP, Lin ZY, Huang KL. Hyperbaric oxygen attenuates cell growth in skin fibroblasts cultured in a high- glucose medium. Wound repair and regeneration. 2008;16(4):513-519.
8. Zainab L, Verma M, Tandon S. Treatment of Oral Sub mucous fibrosis with Er Cr: YSGG laser. Indian journal of dental research. 2011;22:472–4.
9. Kumar A, Bagewadi A, Keluskar V, Singh M. Efficacy of lycopene in the management of oral submucous fibrosis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2007; 103(2):207-213.
10. Canniff J P, Harvey W, and Harris M. "Oral submucous fibrosis: its pathogenesis and management." British dental journal 1998;160(12):429-435.
11. Heber D. Lu QY. Overview of mechanisms of action of lycopene ExpBiol Med (Maywood) 2002;227:920-923.
12. Heber D. Mechanisms of action of lycopene. In: Rao AV, editor. Tomatoes, Lycopene and human health. Scotland: Caledon Science Pres; 2006;24:65-72.
13. Babu M, Rengaswamy S, Padmanabhan P, Varghese C. Evaluation of chemoprevention of oral cancer with Spirulina Fusiformis Nutrition Cancer. 1995;24:197–202.
14. Shetty P, Shenai P, Chatra L, Rao PK. Efficacy of spirulina as an antioxidant adjuvant to corticosteroid injection in management of oral submucous fibrosis. Indian J Dent Res 2013; 24(3):347-350.
15. Mulk BS, Deshpande P, Velpula N, Chappidi V, Chintamaneni RL,Goyal S. Spirulina and pentoxyfilline - a novel approach for treatment of oral submucous fibrosis. J Clin Diagn Res 2013;7(12):3048-3050.
16. Patil S, Al-Zarea BK, Maheshwari S, Sahu R. Comparative evaluation of natural antioxidants spirulina and aloe vera for the treatment of oral submucous fibrosis. J Oral Biol Craniofac Res 2015; 5(1):11-15.
17. Patil S, Khandelwal S, Maheshwari S. Comparative efficacy of newer antioxidants spirulina and lycopene for the treatment of oral submucous fibrosis . Clin Cancer Investig J 2014; 3(6):482-486.

18. Haque MF, Meghji S, Nazir R, Harris M. Interferon gamma (IFN-gamma) may reverse oral submucous fibrosis. J Oral Pathol Med. 2001; 30(1):12-21.
19. Haddad P, Kalaghchi B, Amouzegar-Hashemi F. Pentoxifylline and vitamin E combination for superficial radiation-induced fibrosis: A phase II clinical trial. RadiotherOncol, 2005;77:324-6.
20. Rajendran R, Rani V, Shaikh S. Pentoxifylline therapy: a new adjunct in the treatment of oral submucous fibrosis. Indian J Dent Res. 2006; 17(4):190-8.
21. Rajendran R, Rani V, Shaikh S. Pentoxifylline therapy: A new adjunct in the treatment of oral submucous fibrosis. Indian J Dent Res 2006;17:190-8.
22. Mehrotra R, Singh HP, Gupta SC, Singh M, Jain S. Pentoxifylline therapy in the management of oral submucous fibrosis. Asian Pac J Cancer Prev 2011;12:971-4.
23. KaviA,T.Anjana B and Vaishali K. Haemoglobin levels in patients with oral submucous fibrosis: JIAOMR 2007;19(02), 329-333.
24. KeerththanaBalabaskaran, Dr. Arathy Srinivasan. L. Treatment Options For Oral Submucous Fibrosis: IOSR-JDMSvol, 2013;10(5): 33-35.
25. Agarwal N, Singh D, Sinha A, Srivastava S, Prasad RK, Singh G. Evaluation of efficacy of turmeric in management of oral submucous fibrosis. Journal of Indian Academy of Oral Medicine and Radiology. 2014;26(3):260.
26. Deepa DA, Balan A, Sreelatha KT. Comparative study of the Efficacy of curcumin and turmeric oil as chemopreventive agents in oral submucous fibrosis: a clinical and histopathological evaluation. J Indian AcadOral Med Radiol. 2010;22(2): 88-92.
27. Yadav M, Aravinda K, Saxena VS, Srinivas K, Ratnakar P, Gupta J, Sachdev AS, Shivhare P. Comparison of curcumin with intralesional steroid injections in Oral Submucous Fibrosis–A randomized, open-label interventional study. Journal of oral biology and craniofacial research. 2014;4(3):169-173.
28. Rai B, Kaur J, Jacobs R, Singh J. Possible action mechanism for curcumin in pre-cancerous lesions based on serum and salivary markers of oxidative stress. J Oral Sci. 2010;52(2):251–56.
29. Zhang SS, Gong ZJ, Li WH, Wang X, Ling TY.Antifibrotic effect of curcumin in TGF-β1-induced myofibroblasts from human oral mucosa. Asian Pacific Journal of Cancer Prevention. 2012;13(1):289-94.

30. El Khoury E, Abiad M, Kassaify ZG, Patra D. Green synthesis of curcumin conjugated nanosilver for the applications in nucleic acid sensing and anti-bacterial activity. Colloids Surf B Biointerfaces. 2015;7(127C):274–80.

A Review on Treatment and Management Diabetic Foot Ulcer

Pathmashri[1], Sathya seelan[2], Shantha Sundari[3]
Graduate Student[1], Asst Professor[2], Department of General Surgery,
Professor and Head[3], Department Of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Diabetes is a disorder of metabolism. It results in too much sugar in the blood, or high blood glucose. It involves problems with the hormone insulin. Normally pancreas is the organ responsible for releasing insulin to help our body store and use the sugar and fat from the food we consume. Insulin causes cells to take in sugar to use as energy or to store as fat. This causes blood sugar levels to go back down. Diabetes can occur when the pancreas produces very little or no insulin, or when the body does not respond appropriately to insulin. Thus it is important to treat diabetes on time to avoid consequences.

Key Words: *Diabetes, Hyperglycaemia, diabetes type 1, diabetes type 2*

Introduction

Diabetes is a disorder of metabolism. It results in too much sugar in the blood, or high blood glucose. It involves problems with the hormone insulin. Normally pancreas is the organ responsible for releasing insulin to help our body store and use the sugar and fat from the food we consume. Insulin is responsible for converting the sugar into energy or fat. Thus helping in the blood sugar to become normal. So this if there is any problem with the production of insulin or if the body does not respond to the insulin; it leads to Diabetes. There are 3 types of Diabetes: Type 1, Type 2 and Gestational diabetes [1].

Type 1 diabetes: It is when our immune system destroys the beta cells of pancreas which is responsible for the production of insulin, leading to no insulin production or less insulin production. This results in higher sugar level in blood since there is no insulin to convert them to energy.

Type 2 diabetes:It is when the production of insulin is normal but our body does not respond to the insulin, that is, the cell in our body becomes resistant to insulin. Insulin resistant happens primarily in fat, liver, and muscle cell.

Gestational Diabetes: It occurs in pregnant women, since the placenta produces hormones which increase the blood sugar level.

Signs and Symptoms of Diabetes

The most common diabetes symptoms include unusual weight gain, unusual weight loss, fatigue, non-healing wounds or cuts, sexual dysfunction in males, numbness and tingling sensation in hands and feet.

Classic symptoms of diabetes arepolyuria (increased urination),polydipsia (increased thirst), andpolyphagia (increased hunger) [1].

The major long-term complications of diabetes are that it causes damage to blood vessels. Diabetes increases the risk of cardiovascular disease [2].

Among patients with diabetes, 15% of the patients develop a foot ulcer, and 12-24% of individuals with a foot ulcer require amputation [3].

Diabetic Foot Ulcer

Diabetic foot ulcer is one of the major complications of diabetes mellitus. Diabetes mellitus is a metabolic disorder which slows down the wound healing process. Many studies show a

prolonged inflammatory phase in diabetic wounds, which causes a delay in the formation of granulation tissue and reduction in wound tensile strength [1]

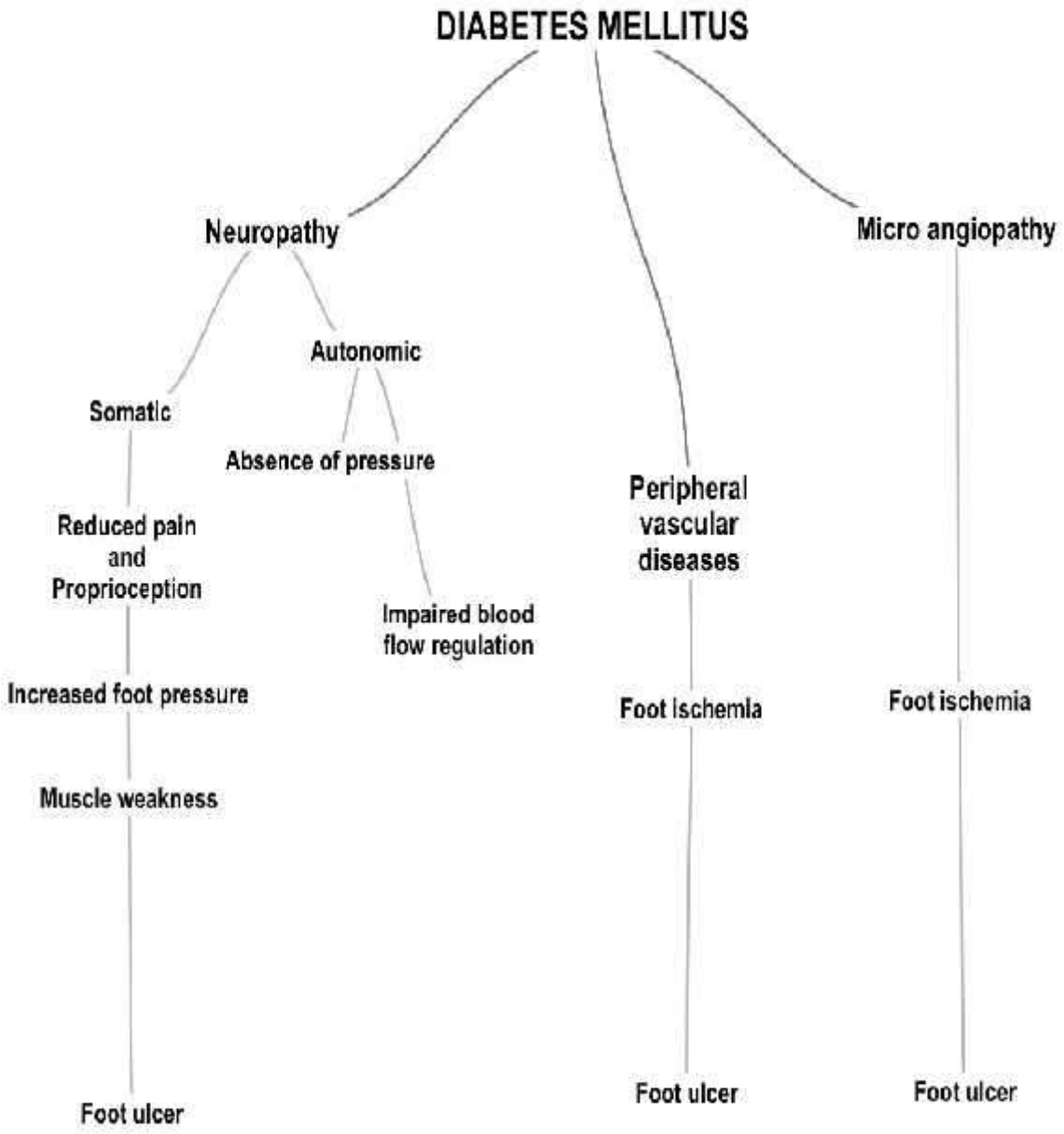
An ulcer is defined as a break in the skin surface that may to involve the subcutaneous tissues or even deeper to the level of muscle or bone[2]. Ulcers form due to a combination of factors, such as increased body weight, numbness of foot, poor circulation, irritation, and trauma, as well as duration of diabetes [3]. Ulcers often progress to infections of the surrounding tissue, osteomyelitis, and amputation [4, 5].

Patients who have diabetes for many years can develop neuropathy. Neuropathy is a reduced or complete lack of ability to feel pain in the feet caused because of never damage due to elevated blood glucose level for a long period of time. The nerve damage often can occur without pain, and one may not even be aware of the problem [6]. This canbe one of the causes leading to foot ulcer.If the foot ulcer gets infected then it cannot be cured by medication, in such cases Amputation is the only choice. Amputation refers to surgical removal of the ulcer.

Pathophysiology

Ulcer in diabetics can be because of three reasons Neuropathy, Peripheral vascular diseases and Micro angiopathy. Neuropathy can be of two types somatic and autonomic. In case of somatic the limbs loses its sense of pain and proprioception, which leads to increased foot pressure. Increase in foot pressure for a prolonged period of time causes muscle weakness which ultimately leads to foot ulcer. In case of autonomic the cause of foot ulcer will be because of impaired blood flow regulation.

Peripheral vascular diseases and micro angiopathy causes foot ischemia. Ischemic foot refers to a lack of normal arterial blood flow from the heart to the feet [7]. That is there is meaning there is not enough blood reaching the foot to provide the oxygen and nutrient needs required for the cells to continue to function.In case of peripheral vascular diseases the blood vessels are narrow and there is reduced blood supply to legs and feet. It also causes nerve damage this leads to lose of the feel of pain. In absence of pain the patient won’t realise the wound or ulcer in the foot and will continue putting pressure on the affected area, this makes the affected area worse and the presenting ulcer or infection will spread to the bone. Once the infection spreads to the bone, the damage cannot be reversed and it leaves us with one option which is amputation. The most common site of amputations are toes, feet and lower legs.



Flow chart 1: Pathophysiology of Foot Ulcer

:

Flow Chart 2: Pathophysiology of Foot Ulcer

Classification of Foot Ulcer

Foot ulcer in diabetic patients can be classified based on depth of ulcer penetration, the presence of wound infection, and the presence of clinical signs of lower-extremity ischemia [8,9].

Stages

Stage A: No infection or ischemia

Stage B: Infection present

Stage C: Ischemia present

Stage D: Infection and ischemia present

Grading

Grade 0: Epithelialized wound

Grade 1: Superficial wound

Grade 2: Wound penetrates to tendon or capsule

Grade 3: Wound penetrates to bone or joint

Management and Treatment

Healing is the primary goal to be achieved in the treatment of foot ulcer in diabetic patients[5].The faster the healing, the less chance for an infection. Management of foot ulcer also depends on the severity and grading of foot ulcer. Lesser the grade and stage faster and easier the management. The risk of ulceration and limb amputation in people with diabetes can be improved by routine preventive podiatric care, appropriate shoes, and patient education[10]

The initiating injury may be from acute mechanical or thermal trauma or from repetitively or continuously applied mechanical stress[11].

Treatment of diabetic foot ulcer includes

- Debridement
- Wound coverage
- Platelet-derived growth factors (PDGF)
- Enzymatic debridement
- Miscellaneous topical agents
- Hydrotherapy
- Treatment of Charcot foot
- Vacuum-assisted closure
- Surgical Care
- Revisional surgery
- Vascular reconstruction
- Hyperbaric Oxygen Treatment

Debridement

Debridement is defined as the removal of damaged, dead and /or infected tissues in order to improve healing process. Removal may be surgical, mechanical, chemical, and autolytic [12].

Wound coverage

Optimal wound coverage requires wet-to-damp dressings, which support autolytic debridement, absorb exudate, and protect surrounding healthy skin [13]. For wounds that are neither very dry nor highly exudative a polyvinyl film dressing which is semi permeable to oxygen and moisture and impermeable to bacteria can be used [14].

Platelet-Derived Growth Factors (PDGF)

PDGF when applied topically on the wound promoted the healing. Becaplermin gel 0.01% is a recombinant PDGF which is produced through genetic engineering is approved by the US Food and Drug Administration (FDA) to promote healing of diabetic foot ulcers [15]

Enzymatic Debridement

Collagen comprises a significant fraction of the necrotic soft tissues in chronic wounds; the enzyme collagenase, derived from fermentation of Clostridium histolyticum, helps remove nonviable tissue from the surface of wounds [16]. But is not a substitute for an initial surgical excision of a grossly necrotic wound.

Miscellaneous Topical Agents

Various other topical agents that have been used for wound management include sugar, antacids, and vitamin A and D ointment [12, 15].

Hydrotherapy

Intractable, infected, cavity wounds sometimes improve with hydrotherapy using saline pulse lavage under pressure [17]

Treatment of Charcot Foot

Charcot foot is a condition causing weakening of the bones in the foot that can occur in people who have significant nerve damage (neuropathy).

Charcot foot is treated initially with immobilisation using special shoes or braces but eventually may require podiatric surgery such as osteotomy and arthrodesis[18]

Vacuum-Assisted Closure

Clean but non-healing deep cavity wounds may respond to repeated treatments by application of negative pressure under an occlusive wound dressing (vacuum-assisted closure [VAC] [19].

Surgical Management

Debridement of the dead and infected tissues from the ulceration and curettage of the underlying osteomyelitis bone are under the surgical management [20].All patients harbouring diabetic foot ulcers should be evaluated by a qualified vascular surgeon or podiatric surgeon who will consider debridement, revisional surgery on bony architecture, vascular reconstruction, and options for soft tissue coverage before any surgical procedures [11, 20].

Revisional Surgery

Revisional surgery for bony architecture may be required to remove pressure points [21]. Such intervention includes resection of metatarsal heads or ostectomy.

Hyperbaric Oxygen Treatment

Hyperbaric oxygen therapy is used rarely and is certainly not a substitute for revascularization [22] .In the presence of an intractable wound and associated non-correctible ischemic arterial disease, hyperbaric oxygen therapy may be beneficial [23]

GlycaemiaControl

The Diabetes Control and Complications Trial, performed by the Diabetes Control and Complications Trial ResearchGroup,studiedthe effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus in the year 1993 [24]. In this it was found that uncontrolled hyperglycaemia is related to diabetic micro vascular complications and if it’s under control then the complications of diabetes including neuropathy and nephropathy can be prevented [24].

Measures for Prevention of Diabetic Ulcers

The risk of ulceration and limb amputation in people with diabetes can be improved by routine preventive podiatric care, appropriate shoes, and patient education [20].Diabetic clinics should screen all patients for altered sensation and peripheral vascular disease.

Cigarette smoking should be stopped, and hypertension and hyperlipidemia should be controlled [5].

Of diabetic foot ulcers, 85% are estimated to be preventable with appropriate preventive medicine, including the following [25]

- Daily foot inspection
- Gentle soap and water cleansing
- Application of skin moisturiser
- Inspection of the shoes to ensure good support and fit
- Minor wounds require prompt medical evaluation and treatment.
- Prophylactic podiatric surgery to correct high-risk foot deformities may be indicated.
- Avoid hot soaks, heating pads, and irritating topical agents.

Corresponding Author
Shantha Sundari
Professor and Head, Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162, Poonamallee High Road ,Chennai 600077,Tamil Nadu, India
Email:shanthakkss@gmail.com
Telephone number: +91 9444443640

References

1. Cooke DW, Plotnick L. Type 1 diabetes mellitus in pediatrics. Pediatr Rev. 2008 Nov 1;29(11):374-84.
2. O'meara SM, Cullum NA, Majid M, Sheldon TA. Systematic review of antimicrobial agents used for chronic wounds. British Journal of Surgery. 2001 Jan 1;88(1):4-21.
3. Amin N, Doupis J. Diabetic foot disease: from the evaluation of the “foot at risk” to the novel diabetic ulcer treatment modalities. World journal of diabetes. 2016 Apr 10;7(7):153.
4. McLennan S, Yue DK, Twigg SM. Molecular aspects of wound healing in diabetes. Primary Intention: The Australian Journal of Wound Management. 2006 Feb;14(1):8.

5. Naves CC. The diabetic foot: a historical overview and gaps in current treatment. Advances in wound care. 2016 May 1;5(5):191-7.
6. Reiber GE, Pecoraro RE, Koepsell TD. Risk factors for amputation in patients with diabetes mellitus: a case-control study. Annals of internal medicine. 1992 Jul 15;117(2):97-105.
7. Apelquist J, Balcker K, Van Houtum WQ, Nabuurs Fransen MH. Schaper NC: International consensus on the diabetic foot. In the International Working groups on the Diabetic foot.
8. Pecoraro RE, Reiber GE. Classification of wounds in diabetic amputees. Wounds. 1990;2(2):65-73.
9. Wagner FW. The diabetic foot. Orthopedics. 1987 Jan 1;10(1):163-72.
10. Cavanagh PR, Lipsky BA, Bradbury AW, Botek G. Treatment for diabetic foot ulcers. The Lancet. 2005 Nov 12;366(9498):1725-35.
11. Kranke P, Bennett M, Roeckl-Wiedmann I, Debus S. Hyperbaric oxygen therapy for chronic wounds. Cochrane Database Syst Rev. 2004 Jan 1;2(2).
12. Burden of Diabetes Gothamkesavamoorthy, awnish K singh, shruti sharma, jyotibala kasav4, surapanenikrishna Mohan5, ashish joshi6related Complications among Hypertensive and Non Hypertensive Diabetics: A Comparative Study. DOI: 10.7860/ JCDR / 2015 / 14461.6452.
13. Tomic-Canic M, Brem H. Gene array technology and pathogenesis of chronic wounds. The American journal of surgery. 2004 Jul 1;188(1):67-72.
14. Brem H, Sheehan P, Rosenberg HJ, Schneider JS, Boulton AJ. Evidence-based protocol for diabetic foot ulcers. Plastic and reconstructive surgery. 2006 Jun 1;117(7S):193S-209S.
15. Evans D, Land L. Topical negative pressure for treating chronic wounds: a systematic review. British journal of plastic surgery. 2001 May 1;54(3):238-42.
16. Lipsky BA, Berendt AR, Deery HG, Embil JM, Joseph WS, Karchmer AW, LeFrock JL, Lew DP, Mader JT, Norden C, Tan JS. Diagnosis and treatment of diabetic foot infections. Plastic and reconstructive surgery. 2006 Jun 1;117(7S):212S-38S.
17. Wieman TJ, Mercke YK, Cerrito PB, Taber SW. Resection of the metatarsal head for diabetic foot ulcers. The American journal of surgery. 1998 Nov 1;176(5):436-41.
18. Faries PL, Teodorescu VJ, Morrissey NJ, Hollier LH, Marin ML. The role of surgical revascularization in the management of diabetic foot wounds. The American journal of surgery. 2004 May 1;187(5):S34-7.

19. Marston WA, Davies SW, Armstrong B, Farber MA, Mendes RC, Fulton JJ, Keagy BA. Natural history of limbs with arterial insufficiency and chronic ulceration treated without revascularization. Journal of vascular surgery. 2006 Jul 1;44(1):108-14.
20. Ehrenreich M, Ruszczak Z. Update on tissue-engineered biological dressings. Tissue engineering. 2006 Sep 1;12(9):2407-24.
21. Streit M, Braathen LR. Apligraf–a living human skin equivalent for the treatment of chronic wounds. The International journal of artificial organs. 2000 Dec;23(12):831-3.
22. Demiri E, Foroglou P, Dionyssiou D, Antoniou A, Kakas P, Pavlidis L, Lazaridis L. Our experience with the lateral supramalleolar island flap for reconstruction of the distal leg and foot: a review of 20 cases. Scandinavian journal of plastic and reconstructive surgery and hand surgery. 2006 Jan 1;40(2):106-10.
23. Roeckl- Wiedmann I, Bennett M, Kranke P. Systematic review of hyperbaric oxygen in the management of chronic wounds. British journal of surgery. 2005 Jan 1;92(1):24-32.
24. Netten JJ, Price PE, Lavery LA, Monteiro- Soares M, Rasmussen A, Jubiz Y, Bus SA. Prevention of foot ulcers in the at- risk patient with diabetes: a systematic review. Diabetes/metabolism research and reviews. 2016 Jan 1;32(S1):84-98.

Adamts-13 in Myocardial Infarction

N.S.Harini[1], Brundha M.P[2], Sankari M[3]

Graduate Student[1], Reader[2], Department of General Pathology,

Professor[3], Department of Periodontics.

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

ADAMTS13, a disintegrin and metalloproteinase with a thrombospondin type 1 motif, member 13, is a metalloprotease that cleaves von Willebrand factor (VWF). There is considerable evidence that VWF levels increase and ADAMTS13 levels decrease in ST-elevation myocardial infarction (STEMI) patients. It is unclear whether this contributes to no re- flow, infarct size, and intramyocardial haemorrhage (IMH).The main aim of this review article is to know in detail about ADAMTS-13 in myocardial infarction.The objective of this article is to assess ADAMTS-13 and its role in myocardial infarction in all aspects and to know the opinions of different authors.The present study was to know about ADAMTS-13 in detail. Various articles from different authors were collected and summed up as a review article so that ADAMTS-13 and its key role in myocardial infarction is known elaborately in all aspects. We have demonstrated that the spacer domain particularly the exosite 3 which is the major target of anti-ADAMTS13 autoantibodies in patients with acquired TTP. Thus the autoantibody which binds to this region is expected to block the substrate binding and inhibit its proteolytic activity of ADAMTS13. Replacement of residues in exosite 3 with alanine is expected to completely abolish anti-ADAMTS13 IgG binding but this also significantly impairs the proteolytic activity of ADAMTS13, which render these mutants which are useless for therapy. Tremendous progress has been made in the past decade towards our understandings of the bio-synthesis, the structure-function relationship and co-factor – dependant regulation of ADAMTS13 function. These advances provide invaluable information which concerns the mechanisms of TTP and other atherothrombotic disorders as well as its inflammatory diseases. Hence novel diagnostic tools and therapeutics may be developed for managing these potentially fatal diseases.

Key Words:*ADAMTS-13, myocardial infarction, von willebrand factor, haemostasis, thrombosis.*

Introduction

In haemostasis and thrombosis, von willebrandfactor(VWF) plays an important role. It acts both as a cofactor in platelet adhesion and aggregation and also acts as a circulating carrier protein for coagulation factor VIII[1].It involves three major steps: (1) Vasoconstriction, (2) formation of a platelet plug, and (3) coagulation (secondary hemostasis). The first step is an immediate constriction of damaged blood vessels caused by vasoconstrictive paracrine released by the endothelium which results in a temporary decrease in blood flow within the injured vessel [41]. Meta analyses of prospective studies have suggested that increasing circulating vwf levels are associated with the risk of producing increased risk of coronary heart disease (CHD) [2]. The molecular weight or multimeric composition of VWF is an important determinant which plays the role of platelet – tethering. This is modulated by a disintegrin and metalloproteinase with a thrombospondin(type I) motif, and member 13 which is nothing but ADAMTS-13. ADAMTS-13 cleaves the VWF A2 domain which reduces its molecular weight and consequently also its platelet tethering function[3].Now the deficiency of ADAMTS-13 promotes VWF-induced platelet aggregation. This can result in thrombotic thrombocytopenic purpura [4]. It has therefore being suggested that circulating levels of ADAMTS-13 may influence circulating levels of VWF and/or its function. Therefore it influences the risk of thrombotic events such as myocardial infarction (MI) in the general population.Myocardial infarction (MI), occurs when blood flow stops to part of the heart causing damage to the heart muscle is commonly known as a heart attack. Coronary artery disease is the main cause of Most MIs. Risk factors include High blood pressure, smoking, diabetes, lack of exercise, obesity, high blood cholesterol, poor diet, and excessive alcohol, among others.

Adamts-13 with VWF Antigen

Both ADAMTS-13 and VWF antigen are risk factors for non-fatal coronary event which is nothing but myocardial infarction along with the effects that are in opposite directions which are clearly independent of each other. VWF has effects which are largely unaffected by other cardio vascular disease risk factors, unlike ADAMTS-13 which appears to have some sort of effects which are masked by co-existence of other risk factors [4]. After the largest set of cardio vascular risk factors have been adjusted, an additional 1 SD of VWF (about 60 IU $dL^{-}1$) is expected to raise the risk of myocardial infarction by about 35%. This is similar to

amount (27%) where the risk is lowered after increasing ADAMTS-13 by 1 SD. The association of VWF ANTIGEN with myocardial infarction is consistent with the recent meta-analysis of prospective studies of Coronary Heart Disease (CHD) [3].

Adamts-13 and Potential Human Diseases ADAMTS-13, first identified and cloned in 2001 is a member of the ADAMTS(A Disintegrin And Metalloprotease with ThromboSpondin type I repeats family) [1,2]. This ADAMTS-13 cleaves a large polymeric adhesion protein called as Von Willebrand Factor (VWF). This VWF is synthesised in vascular endothelial cells and megakaryocytes [3, 4]. This newly synthesised VWF is stored in intra cellular organelles. Weibel-Palade bodies in endothelial cells and alfa-granules in megakaryocytes and platelets [5].

VWF is released basically upon physiological or pathological stimulation and this forms an ultra-long or ultra-large (UL) “string-like” structure on the endothelial surface [6]. These string-like structures are hyper-active in recruiting circulating platelets to the site of endothelial activation and/or injury. Plasma ADAMTS-13 is primarily synthesised and released from the hepatic stellate cells and endothelial cells [7]. This binds and cleaves cell bound UL-VWF strings at the Tyr1605_Met1606 bond, thereby eliminating the UL-VWF strings from the endothelial surface, thus resulting in the fragmentation of VWF strings[6,7]. In addition to this ADAMTS-13 cleaves UL or large VWF in solution after being exposed to high fluid shear stress. This is already seen in micro circulation or at the site of narrow vessels and thrombus formation after injury [8].

Arterial shear stress induces conformational changes in soluble multimericVWF [9]. This becomes accessible by ADAMTS-13 for cleavage. This conformational changes can also be induced in vitro by an addition of denaturant such as urea or guanideine-HCl [10] is the molecular basis of various bio-chemical assays for plasma ADAMTS-13 activity.Due to severe deficiency of plasma ADAMTS-13 activity, there occurs an inability to cleave cell bound and soluble UL or large VWF in circulation. This results in the persistence of hyperactive UL-VWF on endothelial cells and in circulating blood, which leads to excessive platelet aggregation and disseminated VWF/platelet rich thrombus formation, which is the characteristic feature of thrombotic thrombocytopenic purpura [2].

Biosynthesis and Secretion of Adamts-13

Primarily ADAMTS-13, is synthesised in the liver of humans, mice and rats[8].The m-RNA, encoding the full length of ADAMTS-13, is detected only in the liver by Northern blotting analysis[3].But a truncated form of ADAMTS-13 m-RNA, is found in other tissues such as placenta and skeletal muscle by the same method[4].Fragments of ADAMTS-13 m-RNA, are amplified in many tissues including kidneys, spleen, thymus, prostate, pancreas, testis, ovary, small intestine, colon, and peripheral blood leukocytes[7]. ADAMTS-13 IS localised to cells stellate cells residing in the interstitial area of human, mouse, and rat livers. ADAMTS-13 m-RNA and protein have also been detected in vascular endothelial cells in vitro and in vivo[6].

The function of ADAMTS-13, which is synthesised in the endothelium, is not completely understood. When the endothelial cells produce trace amounts of ADAMTS-13 in culture, their massive surface coverage suggests a substantial contribution of endothelium derived ADAMTS-13 to plasma ADAMTS-13 [5].In addition to this ADAMTS-13 is released from endothelial cells may cleave the newly formed UL-VWF strings on the cell surface providing an additional mechanism to maintain a VWF-free surface[6].It has been demonstrated that ADAMTS-13 has either pro angiogenic or anti angiogenic effects depending on the cellular environment.

On one hand, when undergoing the treatment of HUVECs with recombinant ADAMTS-13 results in a dramatically increasedtube formation and cell migration which can be suggested with enhanced angiogenesis. While on the other hand, when vascular endothelial growth factor(VEGF) is present in the culture medium, the VEGF induced angiogenic activity is inhibited by ADAMTS-13 [8]. This anti-angiogenic effect is reversed by pre incubation of ADAMTS-13 with a polyclonal antibody against the C-terminal TSP1 5-7 repeats of ADAMTS-13 is undergone, which suggests a role of TSP1 repeats in mediating the pro and anti angiogenic effects [6].

Structure-Function Relationship of Adamts-13

ADAMTS-13 shares similar domain structure as compared with other ADAMTS family proteases, which comprises of a single peptide, a propeptide, a

metalloprotease,adisintegrinlike domain, first thrombospondin type 1 repeat(TSP1), Cys-rich and spacer domains. The more distal C-terminus contains seven additional TSP1 repeats and two CUB domains.

The function of each domain of ADAMTS-13 in its biosynthesis, secretion, and its proteolytic activity has been studied thoroughly in the recent years [4].When compared to propeptides of other ADAMTS or ADAM family proteases,the ADAMTS-13 propeptide is exceptionally short(only app.41 residues).[7]. While the propeptides of other ADAMTS proteases function as molecular chaperones to assist protein folding and maintain the latency of the proteases by a mechanism called “cysteine-switch”, the ADAMTS-13, propeptide is not required for its secretion and activation [9].Recombinant human ADAMTS-13 expressed in the cells with or without a propeptide secretes normally and is able to cleave VWF substrates with similar efficacy[8].

The metalloprotease domain of ADAMTS-13 has the expected hall marks of the reprolysin or adamalysin family of metalloprotease. This includes three histidine residues which co-ordinate the essential Zn2+ion in the sequence **H**E*XX***H***XX**G*XX***H**D. In addition, three putative Ca2+ -binding sites have been postulated based on modelling the metalloprotease domain of ADAMTS4 and ADAMTS5 [12]. The first putative Ca2+ bindingsite which comprises of amino acid residues Glu83, ASP173, Cys281 and Asp284 that are broadly conserved among ADAMTS and other metalloprotease and this appear to mediate low affinity of Ca2+ -binding.

The second putative Ca2+ -binding site consist of residues Glu164 and Asp166 in conjunction with oneor more of residues Asn162, Asp165 and Asp168[14]. Mutations at the second putative site have no effect on the Ca2+ -dependent ADAMTS13 activity. The third site is predicted to include residues Asp187 and Glu212 in conjunction with Asp182 or Glu184. Mutations at this site dramatically reduce Ca2+ -induced ADAMTS13 activity, suggesting that the residues at the third site play an important role in high-affinity Ca2+ binding and proteolytic activity[14].Replacement of the TCS domains of ADAMTS5 which is a closely related member of ADAMTS proteases, with those of ADAMTS13 alters with ADAMTS5 substrates specificity[8].

ADAMTS5 is not known to cleave VWF but a chimeric variant which consists of the metalloprotease and this integrin domain like of ADAMTS5 (MD5) and three non-catalytic TCS domains of ADAMTS13 (TCS13) (MD5/TCS13) is able to cleave the Glu1615-Ile1616 bond of VWF domain A2 in peptide substrates or VWF multimers that has been sheared [10]. However this cleavage site is no longer at the Tyr1605-Met1606 bond [11]. This further confirms the critical role of each of the non-catalytic domains of ADAMTS13 in substrates specificity[3].

Materials and Methods

The present study was to know about ADAMTS-13 and myocardial infarction in detail. Various articles from different authors were collected and summed up as a review article so that ADAMTS-13 is known elaborately in all aspects. The articles were collected by manual searching of the references of the relevant retrieved articles, peer-reviewed medicine journals, and gray literature. Search terms included ADAMTS-13, MYOCARDIAL INFARCTION, HAEMOSTASIS, THROMBOSIS.. Non-English articles were excluded from the review in the study-selection stage. Data extraction and evaluation of primary studies were performed independently by 2 reviewers.

Discussion

In up to 40% of ST-elevation myocardial infarction (STEMI) patients, imperfections in myocardial blood flow are observed despite reperfusion at the epicardiallevel[21]. This phenomenon, also referred to as no reflow, is occasionally observed angiographically directly after percutaneous coronary intervention (PCI). In a majority of cases, it is established several days later by cardiac magnetic resonance (CMR) as a hypoenhanced area within the hyperenhanced infarcted myocardium.[22,23] Both angiographic no reflow and CMR-defined no reflow are related to increased cardiac failure and death.[24-26]

Various mechanisms for this phenomenon have been proposed including tissue oedema, distal embolization of atherosclerotic debris, and also local microthrombi causing occlusion of capillaries leading to larger infarct size and worse outcome.[27] Recently, it was shown that CMR-defined no reflow actually contains intramyocardial haemorrhage (IMH) and

complete microvascular destruction.[28-30] von Willebrand factor (VWF) is an important factor in primary haemostasis. It attracts platelets to damaged endothelium, causing platelet adhesion; and it thereby initiates and stabilizes platelet aggregation.

VWF is released from endothelium during vessel injury in the form of ultra-large VWF multimers, which contain several platelet binding sites and are therefore more prothrombotic. ADAMTS13, a disintegrin and metalloprotease with a thrombospondin type 1 repeats-13, is a metalloprotease that cleaves VWF, reducing the size of VWF multimers and diminishing their prothrombotic features.[31] In STEMI patients, VWF levels are increased,[32,33] but it is not known whether this is related to infarct size and occurrence of no reflow and IMH. In parallel with the increase in VWF, ADAMTS13 decreases in STEMI patients.[32,33]

Recently, it was shown that ADAMTS13 knockout mice developed larger myocardial infarctions after coronary occlusion and showed decreased left ventricular function when compared with wild-type mice. Also, treatment with recombinant ADAMTS13 (rADAMTS13) reduced infarct size in wild-type mice.[34-36] However, the potential beneficial effects of rADAMTS13 on infarct size and infarct characteristics have never been tested in a large animal model of myocardial ischaemia-reperfusion. In the present study, the relationship between ADAMTS13 and VWF levels and CMR-derived infarct size as well as occurrence of IMH was determined prospectively in STEMI patients. Also, in a porcine model of myocardial ischaemia-reperfusion, closely resembling the clinical scenario of acute myocardial infarction (AMI) treated with primary PCI, intracoronary infusion of rADAMTS13 was tested for its effects on infarct size, formation of microthrombi, and IMH.

Regulation of Adamts13 Function

Cofactor – dependant regulation of coagulation enzymes have been well recognised. This increases the rate of an enzymatic reaction by several orders of magnitude [5]. Unlike other clotting factors that are being synthesised as inactive zymogens, ADAMTS13 is secreted as a constitutively active protease [4]. There has been no inhibitor identified till date. Plasma α2-macroglobulin inhibits many other matrix metalloprotease including ADAMTS-5,5,7 and 12[6]. But this does not seems to bind and affect ADAMTS13 activity towards VWF.

Therefore ADAMTS13 function must be regulated at its substrate level[7].Arterial shear stress can be simulated invitro using a cone plate viscometer, a bench-top-mini-vortex, and

micro fluidic system which generates the laminar flow [11]. Under mechanically induced shear stress, the proteolytic cleavage of multimeric VWF by recombinant ADAMTS13 increases as a function of increasing shear rate, incubation time and concentrations of ADAMTS13 enzyme[10]. This mechanical force-induced cleavage of an A1A2A3 tri-domain molecule or the A2 domain of VWF has already been demonstrated. These suggest that fluid shear stress place a major role in regulating the proteolytic cleavage of soluble VWF by ADAMTS13[12].

Structure-function analysis demonstrates that B-domain deleted FVIII variant (FVIII-SQ) exhibits a similar rate enhancing effect on proteolysis of VWF by ADAMTS13 as does full-length FVIII [15]. However FVIII variant lacking the acidic region which contains a major VWF binding site has no effect under the same conditions. There is an interesting fact that a light chain of FVIII (FVIII-LC), despite a 10-fold reduction in its binding affinity to VWF, is sufficient for accelerating the cleavage of VWF to a similar extent as are wild-type FVIII and FVIII-SQ[11], suggesting that binding of FVIII to VWF through its light chain mediates this cofactor activity.These biochemical findings are further corroborated with those obtained in vivo in a murine model. The hydro dynamic injection is a commonly used method to instantly transfect hepatocytes with plasmid of interest.

This manoeuvre also activates the endothelial cells which trigger the release of UL-VWF into plasma in mice. When this is injected with PBS alone, plasma ratios of high to low molecular weight VWF multimers in *fVIII*mice are higher *than* those in the *fVIII*mice reconstituted with a plasmid encoding FVIII-SQ or FVIII-LC[9], which suggest that the expression of a functional VWF-binding FVIII variant eliminates the accumulation of UL-VWF multimers under (patho)physiological conditions.

Ristocetin,which is an antibiotic that binds the A1 domain of VWF close to the site that GP1bα binds, also enhances cleavage of multimeric VWF by ADAMTS13[7,8]. These results suggest that the interaction between platelet GP1bα or (ristocetin) and the A1 domain plays a role where it affects the accessibility of the A2 domainn by ADAMTS13. There is an interesting fact thatristocetin alleviates the requirement of FVIII to enhance the cleavage of VWF by ADAMTS13 [9], whereas binding of platelet GP1bα to VWF enhances the effect of FVIII or vice versa as previously demonstrated.

Thus in the presence of physiological concentrations of platelets (150x103/µl), the C50 shifts to the left (from 5nM to 0.5nM)[7]. These results suggest that FVIII and platelet GO1b have

synergistic effects which enhances VWF proteolysis by ADAMTS13 under fluid shear stress. It has been postulated that the binding of FVIII to the D'D3 domain of VWF may result in large-scale conformational changes of the VWF multimers, which may include pulling away the D'D3 domain from its neighbouring A1 or A2 domain under shear stress.

Similarly, binding of platelets or soluble GP1bα to the VWF-A1 domain will expose the A2 domain for cleavage. Two or more platelets bound on each side of the scissile bond may dramatically increase the peak tensile force which is exerted on the central A2 domain[11], that enhances A2 domain unfolding and proteolysis by ADAMTS13, which are demonstrated by single molecule experiments[5,9,12].

Mechanism of Anti-Adamts13 Auto Anti Body Inhibition

Idiopathic TTP in adults is caused by a severe deficiency of plasma ADAMTS13 activity which is due to the immunoglobulin(IgG) type anti bodies[9]. The inhibitory anti bodies are detected in about 44-100% of acquired Idiopathic TTP patients where there is a severe deficiency of plasma ADAMTS13 activity[13]. Considering the sensitive assays such as enzyme-linked immunosorbent assay (ELISA) or flow cytometry-based technology, anti-ADAMTS13 IgGs can be deducted in all TTP patients who have severe deficiency of plasma ADAMTS13 activity.

The factors such as antibody mapping and profiling reveal that anti-ADAMTS13 IgG1 and IgG4 predominate in the plasma of acquired TTP patients. Also nearly all anti-ADAMTS13 IgGs bind the sys-rich and spacer domains particularly targeting the spacer domain [15]. Other ADAMTS13 domains including the propeptide, metalloprotease domain, disintegrin domain, first TSP1 repeat, more distil TSP1 repeats and CUB are less reactive when compared to the auto antibodies. Now a majority of TTP patients (approximately 90%) loose reactivity towards ADAMTS13 which follow the substitution of residues Arg568, Phe592, Arg660, Tyr661 and Tyr665 in exosite 3 of the spacer domain[14].

These residues has a major role in substrate recognition and proteolysis of VWF under certain conditions. Thus it is conceivable for the action of binding of anti-ADAMTS13 autoantibodies to this region because it place a role where it blocks the substrate binding and its proteolytic function[12].

Myocardial Infarction and Adamts-13 in Myocardial Infarction About 35% of the total population is being affected by myocardial infarction. This disease is most common. About 146000 people in the UK have an MI every year. It occursinpeople aged over 50 and this disease becomes more common with increase in age. Sometimes younger people are also affected. MI is three times more common in young men than young women. However after the menopause the female hormones present will no longer protect the heart and thus the risk is the same for both men and women.

MI may occur in people with diseases such as angina. It can also happen out of the blue in people with no previous symptoms of the heart disease. This is because atheroma often develops without leaving any symptoms at the first. Certain risk factors increase the risk of more atheroma forming. This can lead to ACS / MI.

Briefly, risk factors that can be modified and may help to prevent MI include:

- Smoking. If you smoke, you should make every effort to stop.
- High blood pressure. If your blood pressure is high it can be treated.
- If you are overweight, losing some weight is advised. Losing weight will reduce the amount of workload on your heart and also help to lower your blood pressure.
- A high cholesterol. This should usually be treated if it is high.
- Inactivity. You should aim to do some moderate physical activity on most days of the week for at least 30 minutes - for example, brisk walking, swimming, cycling, dancing, gardening, etc.
- Diet. You should aim to eat a healthy diet.
- Diabetes. People with diabetes have a higher risk of having ACS. This risk can be reduced by ensuring your blood pressure, cholesterol levels and glucose levels are well controlled.
- Family history. Your risk is increased if there is a family history of heart disease or a stroke that occurred in your father or brother aged below 55, or in your mother or sister aged below 65.
- Ethnic group. Certain ethnic groups - for example, British Asians - have a higher risk of developing cardiovascular disease.[2,9].

Treatment for Myocardial Infarction

Aspirin and Other Antiplatelet Medicines

As soon as possible after MI is suspected you will be given a dose of aspirin. Aspirin reduces the stickiness of platelets. Platelets are tiny particles in the blood that trigger the blood to clot. It is the platelets that become stuck on to a patch of atheroma inside an artery that go on to form the blood clot which leads to other complications such as thrombosis, etc[6]. Other antiplatelet medicines called clopidogrel or ticagrelor may be given. They work in a different way to aspirin and help reduce platelet stickiness. [4].

Injections of Heparin or Similar Medicine: These are usually given for a few days to help prevent further blood clots from forming.

Pain Relief:A strong painkiller such as morphine is given by injection into a vein to ease the pain.

Treatment to Restore Blood Flow in the Blocked Coronary Artery:

A coronary artery bypass graft (CABG) is a surgical procedure used to treat coronary heart disease. [40] The part of the heart muscle starved of blood does not die immediately. If blood flow is restored within a few hours, much of the heart muscle that would have been damaged and die will survive. This is why MI is a medical emergency, and treatment is given urgently. The quicker the blood flow is restored, the better the outlook.[5,1]. Anticoagulant drugs are commonly prescribed to patients with atrial fibrillation, pulmonary embolism, deep vein thrombosis, venous thromboembolism, congestive heart failure, stroke, myocardial infarction, genetic or acquired hypercoagulability, prosthetic heart valves and cardiac stents placement. [39]

There are two treatments that can restore blood flow back through the blocked artery:

- Emergency angioplasty. Ideally this is the best treatment if it is available and can be done within a few hours of symptoms starting. In this procedure a tiny wire with a balloon at the end is put into a large artery in the groin or arm. It is then passed up to the heart and into the blocked section of a coronary artery, using special X-ray guidance. The balloon is blown up inside the blocked part of the artery to open it wide again. A

stent may be left in the widened section of the artery. A stent is like a wire mesh tube which gives support to the artery and helps to keep the artery widened.[3].

- An injection of a clot-busting medicine is an alternative to emergency angioplasty. It can be given easily and quickly in most situations A beta-blocker medicine. Beta-blocker medicines block the action of certain hormones such as adrenaline (epinephrine). These hormones increase the rate and force of the heartbeat. Beta-blocker medicines have some protective effect on the heart muscle and they also help to prevent abnormal heart rhythms from developing. Beta-blocker medicines will also help to prevent having another MI.[8].

Insulin: Some people have a raised blood sugar level when they have an MI, even if they do not have diabetes. If this occurs, then your blood sugar levels may need to be controlled with insulin. If you have diabetes then it is also likely that you will need to be treated with insulin to control your blood glucose levels when you are in hospital.[13].

Oxygen: Oxygen is given which works to reduce the risk of damage to your heart muscle.

Conclusion

Cardiovascular disease is currently the major source of death in the developing countries,especially in India [37]. Employment of the serum cardiac markers rather than non-invasive or invasive procedures in the right time for accurate diagnosis of acute myocardial infarction (AMI) is a more important to restore the patient. [38]This aims in the improvement of nature and re-engineering ADAMTS13. We have demonstrated that the spacer domain particularly the exosite 3 which is the major target of anti-ADAMTS13 autoantibodies in patients with acquired TTP [2]. Thus the autoantibody which binds to this region is expected to block the substrate binding and inhibit its proteolytic activity of ADAMTS13. Replacement of residues in exosite 3 with alanine is expected to completely abolish anti-ADAMTS13 IgG binding but this also significantly impairs the proteolytic activity of ADAMTS13, which render this mutants which are useless for therapy [5].

There is an interesting fact that these variants were more resistant than WT and M1-M3 (with one to three residues mutated) to inhibition by anti-ADAMTS13 autoantibodies from acquired TTP patients. Thus these findings indicate that it is possible to re-engineer ADAMTS13 protease which improves its specific activity in the presence of autoantibodies

which offers therapeutic benefits to acquired TTP patients. Tremendous progress has been made in the past decade towards our understandings of the bio-synthesis, the structure-function relationship and co-factor – dependant regulation of ADAMTS13 function. These advances provides invaluable information which concerns the mechanisms of TTP and other atherothrombotic disorders as well as its inflammatory diseases. Hence novel diagnostic tools and therapeutics may be developed for managing these potentially fatal diseases[7].

Corresponding Author

M.Sankari,

Professor, Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai-600077,Tamil Nadu,India.

E-mail: msankari@gmail.com

Mobile Number: +91 9840285905s

References

1. Levy GG,NicholsWC,LianEC, Foroud T, McClintick JN, MeGeeBM,Yang AY, Siemieniak DR, Stark KR,Gruppo R, Sarode R, Shurin SB, Chandrasekaran V, Stabler SP, Sabio H, Bouhassira EE, Upshaw JD Jr, Ginsburg D, Tsai HM; Mutations in a member of the ADAMTS gene family cause thrombotic thrombocytopenic purpura, Nature. 2001; 413:488-494.
2. Zheng XL, Chung D, Takayama T, Majerus E, Sadler J, Fujikawa K, Structure of Von Willebrand factor-cleaving protease (ADAMTS13), a metalloprotease involved in thrombotic thrombocytopenic purpura, J Biol Chem. 2001; 276:41059-41063.
3. Sadler JE;VonWillebrand factor. Journal of Bio Chem.1991; 266:22777-22780.

4. Wagner DD,MarderVJ;Biosynthesis of Von Willebrand protein by human endothelial cells: processing steps and their intracellular localization. J Cell Biol. 1984; 99:2123-2130.
5. Dong JF, Moake JL, Nolasco L, Bernardo A, Arceneaux W, Shrimpton CN, Schade AJ, McIntire LV, Fujikawa K, Lopez JA; ADAMTS13 rapidly cleaves newly secreted ultralarge Von Willebrand factor multimers on the endothelial surface under flowing conditions. Blood. 2002; 100:4033-4039.
6. Niiya M, Uemura M, Zheng XW, Pollak E, Dockal M, Scheiflinger F, Wells RG, Zheng XL;Increased ADAMTS13 proteolytic activity in rat hepatic stellate cells upon activation in vitro and in vivo. J ThrombHaemost. 2006; 4:1063-1070.
7. Zhou W, Inada M. Lee TP, Benten D, Lyubsky S, Bouhassira EE, Gupta S, Tsai HM; ADAMTS13 is expressed in hepatic stellate cells. Lab Invest. 2005; 85:780-788.
8. Shang D, Zheng XW, Niiya M, Zheng XL;Apical sorting of ADAMTS13 in vascular endothelial cells and Madin-Darby canine kidney cells depends on the CUB domains and their association with lipid rafts. Blood, 2006; 99:2123-2130.
9. Dong JF;Cleavage of ultra-large von Willebrandfactorby ADAMTS13d underflow conditions.JthrombHaemost, 2006;203; 767-776.
10. Chauhan AK, Motto DG, Lamb CB, Bergmeier W, Dockal M, Plaimauer B, Scheiflinger F, Ginsburg D, Wagner DD; Systemic antithrombotic effects of ADAMTS13, J Exp Med. 2006;203; 767-776.
11. Chauhan AK, Motto DG, Lamb CB, Bergmeier W, Dockal M, Plaimauer B, Scheiflinger F, Ginsburg D, Wagner DD;Systemic antithrombotic effects of ADAMTS13; J Exp Med,2006; 1982; 307:1432-1435.
12. Dong JF;Structural and functional correlation of ADAMTS13; Curr Opin Hematol. 2007; 14:270-276.
13. Moake JL, Rudy CK, Troll JH, Weinstein MJ, Colannino NM, Azocar J, Seder RH, Hong SL, DeykinD;Unusually large plasma factor VIII; Von Willebrand factor multimers in chronic relapsing thrombotic thrombocytopenic purpura. N Engl J Med. 1982; 307:1432-1435.
14. Hosler GA, Cusumano AM, Hutchins GM;Thrombotic thrombocytopenic purpura and haemolytic uremic syndrome are distinct pathologic entities. A review of 56 autopsy cases. Arch Pathol Lab Med. 2003; 127:834-839.
15. Tsai HM;ADAMTS13 and microvascular thrombosis; Expert Rev Cardiovasc Ther. 2006; 4:813-825.

16. Majerus EM, Zheng X, Tuley EA, Sadler JE; Cleavage of the ADAMTS13 propeptide is not required for protease activity. J Biol Chem. 2003; 278:46643-46648.
17. Gao W, Zhu J, Westfield LA, Tuley EA, Anderson PJ, Sadler JE; Rearranging exosites in noncatalytic domains can redirect the substrate specificity of ADAMTS proteases. J Biol Chem. 2012; 287:26944-26952.
18. Gutierrez E, Petrich BG, Shattil SJ, Ginsberg MH, Groisman A, Kasirer-Friede A.; Microfluidic devices for studies of shear-dependent platelet adhesion. Lab Chip. 2008; 8:1486-1495.
19. Cao W, Sabatino DE, Altynova E, Lange AM,Casina VC, Camire RN, Zheng XL; Light Chain of Factor VIII is Sufficient for Accelerating Cleavage of Von Willebrand Factory by ADAMTS13 Metalloprotease. J Biol Chem. 2012; 287:32459-32466.
20. Jian C, Xiao J, Gong L, Skipwith CG, Jin SY, KwaanHc, Zheng XL;Gain-of-function ADAMTS13 variants that are resistant to autoantibodies against ADAMTS13 in patients with acquired thrombotic thrombocytopenic purpura. Blood 2012; 119:3836-3843.
21. Jaffe R, Charron T, PuleyG, Dick A, Strauss BH; Microvascular obstruction and the no-reflow phenomenon after percutaneous coronary intervention. Circulation 2008; 117:3152 –3156.
22. Wu KC;CMR of microvascular obstruction and hemorrhage in myocardial infarction. J CardiovascMagnReson 2012;14:68.
23. Harrison RW, Aggarwal A, Ou FS, Klein LW, Rumsfeld JS, Roe MT, Wang TY;Incidence and outcomes of no-reflow phenomenon during percutaneous coronary intervention among patients with acute myocardial infarction. Am J Cardiol 2013; 111: 178 –184.
24. Wu KC, Zerhouni EA, Judd RM, Lugo-Olivieri CH, Barouch LA, Schulman SP, Blumenthal RS, Lima JA;Prognostic significance of microvascular obstruction by magnetic resonance imaging in patients with acute myocardial infarction. Circulation 1998; 97:765 –772.
25. Morishima I, Sone T, Okumura K, Tsuboi H, Kondo J, Mukawa H, Matsui H, Toki Y, Ito T, Hayakawa T;Angiographic no-reflow phenomenon as a predictor of adverse long-term outcome in patients treated with percutaneous transluminal coronary angioplasty for first acute myocardial infarction. J Am Coll Cardiol 2000; 36:1202 –1209.
26. Kaul S, Ito H; Microvasculature in acute myocardial ischemia: part I: evolving concepts in pathophysiology, diagnosis, and treatment. Circulation 2004; 109:146 –149.
27. Ito H; No-reflow phenomenon and prognosis in patients with acute myocardial infarction. Nat Clin Pract Cardiovasc Med, 2006; 3:499–506.

28. Robbers LF, Eerenberg ES, Teunissen PF, Jansen MF, Hollander MR, Horrevoets AJ, Knaapen P, Nijveldt R, Heymans MW, Levi MM, van Rossum AC, Niessen HW, Marcu CB, Beek AM, van RN;Magnetic resonance imaging-defined areas of microvascular obstruction after acute myocardial infarction represent microvascular destruction and haemorrhage. Eur Heart J, 2013; 34:2346 –2353.
29. Betgem RP, de Waard GA, Nijveldt R, Beek AM, Escaned J, van RN;Intramyocardial haemorrhage after acute myocardial infarction. Nat Rev Cardiol, 2015; 12:156 –167.
30. Bekkers SC, Smulders MW, Passos VL, Leiner T, Waltenberger J, Gorgels AP, Schalla S; Clinical implications of microvascular obstruction and intramyocardial haemorrhage in acute myocardial infarction using cardiovascular magnetic resonance imaging. Eur Radiol, 2010; 20:2572 –2578.
31. Crawley JT, de GR, Xiang Y, Luken BM, Lane DA;Unraveling the scissile bond: how ADAMTS13 recognizes and cleaves von Willebrand factor. Blood, 2011; 118: 3212 –3221.
32. Rutten B, Maseri A, Cianflone D, Laricchia A, Cristell N, Durante A, Spartera M, Ancona F, Limite L, Hu D, Li H, Uren N, de GP, Mannucci P, Roest M; Plasma levels of active von Willebrand factor are increased in patients with first ST-segment elevation myocardial infarction: a multicenter and multiethnic study. Eur Heart J Acute Cardiovasc Care, 2015; 4:64 –74.
33. Matsukawa M, Kaikita K, Soejima K, Fuchigami S, Nakamura Y, Honda T, Tsujita K, Nagayoshi Y, Kojima S, Shimomura H, Sugiyama S, Fujimoto K, Yoshimura M, Nakagaki T, Ogawa H; Serial changes in von Willebrand factor-cleaving protease (ADAMTS13) and prognosis after acute myocardial infarction. Am J Cardiol, 2007; 100:758-763.
34. Doi M, Matsui H, Takeda Y, Saito Y, Takeda M, Matsunari Y, Nishio K, Shima M, Banno F, Akiyama M,Kokame K, Miyata T, Sugimoto M;ADAMTS13 safeguards the myocardium in a mouse model of acute myocardial infarction. ThrombHaemost, 2012;108: 1236– 1238.
35. Gandhi C, Motto DG, Jensen M, Lentz SR, Chauhan AK; ADAMTS13 deficiency exacerbates VWF-dependent acute myocardial ischemia/reperfusion injury in mice. Blood, 2012; 120:5224 –5230.
36. De Meyer SF, Savchenko AS, Haas MS, Schatzberg D, Carroll MC, Schiviz A, Dietrich B, Rottensteiner H, Scheiflinger F, Wagner DD;Protective anti-inflammatory effect of ADAMTS13 on myocardial ischemia/reperfusion injury in mice. Blood ,2012; 120: 5217–5223.
37. Deeksheetha. P;Awareness of the Patients about the Predisposing Causes of Myocardial Infarction; J. Pharm. Sci. & Res., 2015; 7(9):744-752.

38. Muntaanil kumar, Rajiswamy, Ramaswamy; Study on conventional and novel cardiac biomarkers in acute myocardial infarction;Asian J Pharm Clin Res, 2016;9(2): 327-331.
39. UmayalSwaminathan and Dr. Santhosh Kumar M.P;Peri-operative management of dental patients receiving oral anti coagulant therapy; International Journal of Current Research, 2017;9(12):63437-63440.
40. Ms.HenaLeni Grace D., Mrs.Aruna S. and Mrs.Mangala Gowri P; Effectiveness of structure teaching program on knowledge regarding cardiac rehabilitation among patients undergone coronary artery bypass grafting surgery;International Journal of Development Research, 2017;07(09):15154-15156.
41. Santhosh kumar MP; Local hemostatic agents in the management of bleeding in oral surgery; Asian J Pharm Clin Res, 2016; 9(3):35-41.

Adiponectin – A Review

T. Nandhini[1], Vishnu Priya[2], ManishRanjan[3],
Graduate Student[1],Professor[2],Department of Biochemistry,
Reader[3],Department of Conservative Dentistry,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

A circulating protein produced in adipocytes, called Acrp30 or adiponectin has insulin – mimetic and sensitizing actions in skeletal muscle and suppression of glucose production in liver. Adiponectin circulates as hexameric, oligomeric, to a lesser extent, trimetric forms. Adiponectin has attracted so many researchers because of its anti diabetic and anti atherogenic effects, which can be a therapeutic tool for diabetes and metabolic syndrome. Concentration of totaladiponectin in the blood is about 3-30µg/ml. High level of adiponectin reduce risk of coronary heart disease and myocardial infarction in healthy man. Human adiponectin consists of 244 amino acid residues and has a distinct domain structure. This review describes the pathophysiological role of adiponectin and its role in diseases.

Key Words: *Artherosclerosis,Hyperglycaemia,Hyperinsulinaemia,HMW(high molecular weight)*

Introduction

Adiponectin is an insulin-sharpening hormone with against diabetic, calming and hostile to atherogenic properties [1]. It is presently realized that fat tissue is a dynamic endocrine organ, emitting various organically dynamic proteins, otherwise called adipokines. Adiponectin is a protein formed and discharged solely by adiposities. Adipocytes are in charge of the formation of adiponectin, leptin, angiotensinogen and retinol-restricting protein. Some adipokines, including monocyte chemoattractant protein-1 and apelin, are created by the two parts [2].Adiponectin, likewise named Acrp30AdipoQapM1 or GBP28 was initially recognized autonomously by 4 bunches utilizing diverse methodologies. A littler type of adiponectin incorporates the globular space separates proteolytically from full-length adiponectin and exists in plasma, in spite of the fact that in little sums .Adiponectin associates adversely with serum triacylglycerol (triglyceride) and emphatically with serum HDL cholesterol in non-diabetic ladies or youthful solid man [3]. Plasma adiponectin level in human range from 0.5 to 30g/ml, which is around 1000-crease higher than the centralizations of most different hormones, for example, leptin, insulin and so on. In fact, adiponectin represents 0.01 % of aggregate human plasma proteins and is the most plentiful fat tissue protein [4]. The nearness of corpulence to a vast degree expands the danger of related comorbidities, for example, insulin obstruction, diabetes, dyslipidemia, hypertension and others. The blend of the previously mentioned pathologies is currently by and large alluded to as the metabolic disorder or disorder X [5]. Moreover, adiponectin has a potential part in the focal direction of vitality admission and consumption.

Structure

The literal structure of the HMWform of adiponectin is not yet known. Most likely several combined hexamers and/or trimers constitute high-molecular weight form of adiponectin. It is commonly supposed that disulfide bonds on top of some bonds with involvement of modified amino acid residues in collagen domain of adiponectin, take part in holding subunits of HMW form of adiponectin together .It is also believed that those oligomeric forms exist in the bloodstream as separate moieties and do not renovate into each other [6]. It has been revealed recently, that adiponectin oligomers are capable of binding Ca2+ ions which are thought to participate in maintenance of conformational steadiness of adiponectin. In plasma, its structural resemblance to complement factor C1q and the consistent finding of decreased

levels in obesity [7]. Full-length adiponectin motivated AMP-activated protein kinase (AMPK) phosphorylation and activation in the liver, while globular adiponectin do so in both skeletal muscle and the liver .

Unexpectedly, genes expressed in subcutaneous and visceral adipose tissue, 20% and 30% respectively, were bioactive secretory proteins, i.e. adipocytokines such as leptin and TNF-*α*. More surprisingly, 40%of the genes expressed in adipose tissue were novel and the most abundant among them, termed adiponectin, was novel as well.Adiponectin is the most abundant adipocytokine. Unlike leptin and other adipose tissue-derived hormones which circulateatpicograms or nanograms per milliliter, adiponectin circulates at very high levels (micrograms per milliliter). It circulates as several multimeric species, including a high molecular- weight (HMW) form thought to be the most clinically relevant.

Adiponectincontains 244 amino acids, a signal peptide, a collagen like domain at its N-terminus and a globular domain at its C-terminus, which shares sequence similarity with collagens X and VIII as well as complement factor C1q.In spite of the absence of primary sequence similarity, the crystal structure of the C-terminal globular domain resembles that of TNF-*α* [8]. Structurally, adiponectin belongs to the complement 1q family. Adiponectin monomer consists of an N-terminal collagenous domain and a C-terminal globular domain [9] that generates low-molecular-weight dimers or trimers, medium (hexamers) and high-molecular-weight complexes (HMW, dodecamers and 18 mers) in mammalian plasma[10]. The three multimeric forms are found in the circulation, associated with several serum proteins [11]. The globular fragment was also detected in the trimeric form in human and mouse plasmas and was shown to be generated by proteolytic cleavage [12].Collagen-like parts of three adiponectin molecules can work together forming triple coiled coil structure much alike to that in collagen.

Adiponectin Blood Test

Adiponectin is a hormone discharged by fat cells. It manages tissue aggravation and responsiveness to insulin is made by beta cells of the pancreas. It is discharged in blood after an ascent in blood glucose level, (for example, after a substantial feast) to help bring down it to typical levels. Insulin does this by fortifying cells to 'take in' glucose (and other nutrients).The most regular reason for lifted glucose is diabetes mellitus, or just "diabetes". There are 3 kinds of diabetes mellitus, all of which influence insulin work: Type 1 or insulin-

subordinate diabetes comes about because of the immune system obliteration of beta cells of the pancreas. Sort 2 or insulin-autonomous diabetes comes about because of the desensitization of cells to the impacts of insulin.Gestational diabetes may happen amid pregnancy and result in exorbitantly substantial children that display low glucose levels. Insulin affectability alludes to the degree to which cells react to insulin. Diabetes, heftiness, and other metabolic issue are related with low insulin sensitivity [13].Adiponectin raises insulin affectability. It builds the breakdown of unsaturated fats and abatements the generation of glucose by the liver. People with weight, type II diabetes, and other metabolic issue frequently have low adiponectin levels in blood. The Adiponectin Blood Test decides the levels of adiponectin in blood. It is utilized to analyze metabolic clutters, for example, type II diabetes.

Clinical Indications for Playing Out the Adiponectin Blood Test

Following are the clinical signs for playing out the Adiponectin Blood Test:

1.Frequent pee

2.Increased thirst

3.Fatigue

4.Blurry vision

5.Rapid weight reduction

6.Tingling and desensitizing in the furthest points

7.Obesity

8.History of cardiovascular illness

Example Accumulation ofAdiponectin Blood Test

Following is the example accumulation process for Adiponectin blood test

Test required- Blood

Procedure of acquiring blood test in grown-ups:

•A band is wrapped around the arm, 3-4 crawls over the accumulation site (shallow vein that exists in the elbow pit)

•The site is cleaned with 70% liquor in an outward winding, far from the zone of needle addition

•The needle top is evacuated and is held in accordance with the vein, pulling the skin tight

•With a little and snappy push, the vein is infiltrated utilizing the needle

•The required measure of blood test is gathered by pulling the plunger of the syringe out gradually

•The wrap band is evacuated, bandage is put on the accumulation site, and the needle is expelled

•The blood is quickly moved into the blood compartment, which has the proper additive/cluster activator/hostile to coagulant

•The syringe and the needle are arranged into the fitting "sharp compartment" for protected and sterile transfer

Arrangement required: No uncommon readiness is required before the test [14].

Adiponectinin Health and Disease

Diminished serum adiponectin fixation show insulin opposition and sort 2 diabetes. Additionally, hypoadiponectinemia was uncovered to be related with coronary conduit sickness . In people, adiponectin is an insulin-sharpening, vascular defensive, calming protein related with a more great lipoprotein subclass profile. Low coursing levels, especially of the HMW segment [15] are likewise a solid hazard marker for the advancement of the metabolic disorder and sort 2 diabetes . The fat cells in adipocytes may discharge cytokines as adiponectin and resistin. Adiponectin stays ordinary barring in the event of diabetes and weight it lessen [16].

A forthcoming investigation of patients with renal disappointment exhibited that patients who qualified new cardiovascular occasions had bring down plasma adiponectin levels. The fat tissue be an outsized resorvior of organically dynamic middle people for example Tumor putrefaction factor - α, and different adipokines, for example, leptin, resistin and adiponectin in provocative process [17]. Irritation describes atherosclerosis and large subjects, and people at high danger of atherosclerosis have elevated amounts of intense stage reactants, for example, CRP (C-responsive protein), an effective marker for foundational aggravation and an autonomous hazard factor for CAD. Myocardial hypertrophy and diastolic brokenness can go with diabetes and heftiness related conditions. Unsaturated fat oxidation was hoisted by the proteolytic section of adiponectin, most presumably because of expanded articulation of qualities engaged with beta oxidation and vitality dispersal in muscle.

The consumption of lipid stores from adipocytes amid interminable calorie confinement additionally prompts raised adiponectin levels in blood. Clinical examination utilizing CT

(PC tomography) checking demonstrates that stoutness with instinctive fat gathering (instinctive fat weight) relates intimately with diabetes mellitus, hyperlipidaemia, hypertension and atherosclerosis as of late, adiponectin was presented to be available in the cerebrospinal liquid (CSF) of rodents and people and to enter the CSF from the flow .

AdiponectinRole in Diabetes

Adiponectin levels appear to be decreased before the advancement of sort 2 diabetes, and organization of adiponectin has been joined by bring down plasma glucose levels and in addition expanded insulin affectability. Moreover, lessened articulation of adiponectin has been related with some level of insulin obstruction for hypoadiponectinaemia in connection to insulin opposition. The essential systems by which adiponectin improve insulin affectability seems, by all accounts, to be through expanded unsaturated fat oxidation and hindrance of hepatic glucose generation [18] .Adiponectin levels are expanded by thiazoledinedione treatment, and this impact may be critical for the upgraded insulin affectability prompted by thiazolidinediones. Interestingly, adiponectin levels are lessened by star incendiary cytokines particularly tumor corruption factor-alpha. In rundown, adiponectin notwithstanding conceivable mitigating and against atherogenic impacts seems, by all accounts, to be an insulin enhancer, with potential as another pharmacologic treatment methodology of the metabolic disorder and sort 2 diabetes.

It has been demonstrated that CSF contains bring down levels of adiponectin contrasted and serum in rodents [19], and this level builds resulting to intravenous infusion of adiponectin affirming its capacity to enter the cerebrum from the flow. Globular adiponectin shielded mice from diabetes and apolipoprotein E-inadequate mice from atherosclerosis [20] showing that globular adiponectin is likewise proficient as the full-length. Adiponectin has antidiabetic properties because of insulin-mimetic and insulin-sharpening activities, while mitigating and hostile to atherosclerotic impacts have likewise been continually detailed.

AdiponectinReceptors

Three adiponectin receptors, AdipoR1, AdipoR2 and T-cadherin, have been well-known. AdipoR1 is ubiquitous, but is most abundantly expressed in skeletal muscle is a receptor for hexameric and HMW adiponectin . Expression of AdipoR1 and AdipoR2 or suppression of AdipoR1 and AdipoR2 expression supports our conclusion that AdipoR1 and AdipoR2 serve as receptors for globular and full-length adiponectin and mediate increased AMPK, PPARα

ligand activities, fattyacid oxidation, and glucose uptake by adiponectin. A classical model of accelerated atherosclerosis apolipoproteinE(ApoE)-deficient transgenic mice, or the double transgenic ApoE/leptin-deficient mice led to amelioration of atherosclerosis in both models. This effect was accompanied by reduced expression of class A scavenger receptor and TNF [21].

In addition to sex, nutritional status and body weight, other factors affecting plasma adiponectin levels include menopausal status, and medications such as b-blockers and angiotensin II receptor blockers .However, the age of the study subjects was greater than 65 years, and therefore, all the female participants were menopausal [22]. Furthermore, the use of b-blockers and angiotensin II receptor blockers was not different between the three subgroups. Further analyses revealed that adiponectin depolarized PA neurons expressing both receptors Adip-R1 and Adip-R2, whereas AP neurons expressing only one receptor were insensitive indicating different roles of adiponectin and differentpathways of its receptors in controlling excitability of AP neurons.On the other hand, macrophage polarization phenotype regulates the expression of adiponectin receptors (AdipoRs) in ways that classical activation (M1) of macrophages suppresses [23] the expression of AdipoRs, and alternative activation (M2) preserves it .

AdiponectinSignalling and Function in Insulin and Target Tissue

APPL1 Protein

Adiponectin inspires various downstream flagging occasions. Be that as it may, no inborn protein kinase movement or phosphorylation because of adiponectin has ever been recognized in either AdipoR1 or AdipoR2. Moreover, substitution of the tyrosine deposits inside the intracellular N-end by site-coordinated mutagenesis has no effect on adiponectin flagging. Along these lines, the adiponectin receptors are likely transmembrane receptors that experience conformational change and couple the intracellular space with other flagging particles upon extracellular adiponectin official. Utilizing yeast two-crossover innovation recognized that APPL1 as the intracellular restricting accomplice of AdipoR1 and AdipoR2. The human Appl1 quality is situated in the 3p14.3-21.1 locale and encodes a 709-amino corrosive protein of 78 kDa.

Human hereditary examinations recommend that SNPs and point changes in the Appl1 coding district correspond with muscle versus fat conveyance and a high commonness of

diabetes.APPL1 is profoundly hydrophilic with no potential transmembrane locale and comprised of numerous auxiliary and useful spaces including BAR, PH, PTB, and looped curl (CC) .APPL1 straightforwardly ties to the intracellular areas of AdipoR1 and AdipoR2 by means of its C-terminal PTB and CC spaces, subsequently interceding the activities of adiponectin in the control of vitality digestion and insulin affectability .In refined skeletal muscle cells, concealment of APPL1 articulation decreased adiponectin-actuated glucose take-up and GLUT4 translocation.

On the other hand, overexpression of APPL1 upgraded the stimulatory activities of adiponectin in glucose digestion in muscle.Rab5, a GTPase downstream of APPL1, assumes an imperative part in APPL1-intervened adiponectin flagging .The significant activity of adiponectin on lipid digestion is to advance unsaturated fat oxidation, a procedure in which AMPK and acetyl CoA carboxylase (ACC) assume a basic part. Upon adiponectin incitement, APPL1 ties to protein phosphatase 2A (PP2A) and protein kinase, along these lines actuating PP2A and inactivating PKC by means of dephosphorylation. The inactivation of PKC brings about the dephosphorylation on liver kinase B1 (LKB1), enabling LKB1 to translocate from core to cytoplasm and initiate AMPK .notwithstanding the AMPK pathway,

APPL1 additionally intercedes adiponectin-prompted actuation of the p38 MAPK pathway and researched its effect on the mitigating activities of adiponectin . APPL1 fastened p38 MAPK together with its upstream initiating kinases including changing development factor-β actuated kinase 1 (TAK1) and mitogen-enacted protein kinase 3 (MKK3), accordingly assisting the phosphorylation of key compounds of this pathway [24].Interestingly, the activity of APPL1 on p38 MAPK pathway is particular to adiponectin, while its effect on TNF-α-incited p38 MAPK enactment is restricted . APPL1 in interceding the activities of adiponectin. Entire body knockout of APPL1 hinders adiponectin flagging and results in insulin opposition in significant insulin target tissue.

APPL2 Protein

APPL2 is an isoform of APPL1 and offers homology in amino corrosive succession with APPL1 protein .Similar to APPL1, APPL2 has a N-terminal BAR area, focal PH space, and C-terminal PTB space. APPL2 intervenes FSH flag transduction by authoritative to APPL1 through their individual BAR areas, which brings about the development of the FSH receptor– APPL1– AKT2 complex. Quite, APPL2 does not straightforwardly connect with

AKT2 [25]. APPL2 contrarily regulates adiponectin motioning in skeletal muscle cells. APPL2 specifically ties to AdipoR1 or AdipoR2 by means of its BAR space, in this way keeping the connection of APPL1 with AdipoRs. Therefore, APPL2 pieces adiponectin motioning through AdipoR1 and AdipoR2 by aggressive hindrance of APPL1. Moreover, APPL2 heterodimerizes with APPL1, in this way diminishing the authoritative of APPL1 to AdipoRs and debilitating the activities of adiponectin. Strangely, adiponectin balances the separation of the APPL1/APPL2 heterodimers, which can likewise be activated by insulin and metformin .Because APPL1 and APPL2 apply inverse activities in interceding adiponectin flagging, proposed the 'Yin and Yang' modulatory idea [26].

The Yin and Yang modulatory idea including APPL1/APPL2 offers a point by point atomic instrument by which adiponectin controls lipid and starch digestion. Predictable with the Yin-Yang hypothesis, with muscle-particular APPL2 removal or overexpression indicate individual upgraded or weakened insulin affectability, insulin-animated GLUT4 translocation, and glucose take-up in skeletal muscle .The restricting parts of APPL1 and APPL2 were additionally seen in the direction of the PI3K/AKT/NF-κB pathway in macrophages .

The AMPK Pathway

AMPK, a protein kinase managed by AMP, is a broadly perceived cell sensor for metabolic state. In skeletal muscle, both full-length adiponectin and the globular space have been appeared to trigger AMPK phosphorylation, prompting AMPK actuation .APPL1 assumes a key part in adiponectin-interceded AMPK phosphorylation .In essential culture of skeletal muscle detached from corpulent people or patients with type 2 diabetes, AMPK phosphorylation because of adiponectin is incredibly decreased, exhibiting that disabled flagging downstream of the adiponectin receptor may weaken the activities of adiponectin or cause adiponectin obstruction .Adiponectin has additionally been appeared to initiate AMPK phosphorylation in the liver[27]. Notably, constrained part of the AMPK pathway in the control of gluconeogenesis .At introduce, in any case, no different components, i.e. non-AMPK pathways, have been appeared to intervene the impacts of adiponectin on gluconeogenesis in the liver.

The PPAR pathway

Another key translation factor in metabolic direction is PPAR-α. In skeletal muscle, adiponectin definitely builds the articulation and action of PPAR-α, which thus upregulates acetyl CoA oxidase (ACO) and uncoupling proteins (UCPs), in this manner advancing unsaturated fat oxidation and vitality consumption .In the liver, adiponectinupregulates a few PPAR-α target qualities including CD36, which tweaks hepatic unsaturated fat take-up and digestion and ACO, which controls unsaturated fat oxidation .what's more, adiponectin has been appeared to expand hepatic glucose take-up through PPAR-α, in this manner enhancing hepatic insulin affectability [28].

Thiazolidinedione (TZD) class of PPAR-γ ligands upregulatesadiponectin articulation in adipocytes .The impact of TZDs on enhancing glucose resistance is debilitated in adiponectin-inadequate mice, showing that adiponectin intercedes, in any event to a limited extent, the insulin-sharpening activities of TZDs .The declaration of PPAR-γ is notably expanded in 3T3-L1 cells overexpressingadiponectin, which is related with improved adipocyte separation ,demonstrating that adiponectin advances the PPAR-γ pathway, in this way actuating a positive criticism circle that increments adiponectin articulation and adipocyte separation.

Other Adiponectin Flagging Pathways

Notwithstanding the pathways examined above, adiponectin has been appeared to instigate calcium discharge from sarcoplasmic reticulum in myocytes or advance calcium inundation, in this way actuating Ca2+/calmodulin-subordinate protein kinase (CaMKK-β) and AMPK, which brings about initiation of SIRT1 and PPAR-α and increment of mitochondria biogenesis .what's more, ceramide-interceded pathways likewise have been embroiled in intervening the activities of adiponectin revealed that both AdipoR1 and AdipoR2 are related with ceramidase exercises, which, upon adiponectin official, powerfully improves ceramides transformation to S1P [29]. A procedure that is autonomous of AMPK. The p38 MAPK pathway likewise assumes a part in adiponectinflagging .

Adipor1 Concealment Compounds Alzheimer's Infection like Pathologies

Pathogenic systems hidden the psychological weakness of spatial learning and memory and neurodegeneration. ADPN-AdipoR1 flagging pathway have a noteworthy part of insulin-sharpening impact through actuating protein kinase B (PKB or AKT) .AdipoR1 inadequacy

influences insulin-responsive phenotype, insulin in HT22 cells by dosage subordinate way. Curiously, AKD amass displayed lessened AKT phosphorylation contrasted and Scraggregate . Besides, AD-like neuropathologies, for example, proteinopathies and neuroinflammation .Preferentially, unusual protein collection, for example, Aβ creation and hyperphosphorylation. Aβ generation was significantly expanded in AKD .

Next, we explored whether glycogen synthase kinase 3β (GSK3β) shape is dynamic or not because of its part for tau hyperphosphorylation. Despite the fact that articulation level of GSK3β are exceedingly up managed in the two areas of AKD mice, phosphorylation of GSK3β at serine 9, thinking about an inhibitory shape, was diminished in the cortex of AKD mice with the exception of hippocampus . Successively, tau protein phosphorylation at serine 396/404 (combined helical fibers or PHF-1) in was profoundly controlled in the two districts of AKD mice, add up to tau (Tau5) level additionally expanded in AKD .neuroinflammatory phenotypes utilizing microglial marker and expert provocative cytokines. Ionized calcium-restricting connector particle 1 (Iba-1) and tumor-corruption factor alpha (TNF-α) level were up-controlled in AKD .

AdipoR1 knockdown in microglial cell line BV-2 showed M1 polarization, professional provocative phenotype, under the basal condition [29]. In synopsis, AdipoR1 knockdown have demonstrated neurodegeneration-interceded memory brokenness and in addition worldwide AD-like pathologies including insulin flagging brokenness, proteinopathies and neuroinflammation.

Adipor-Autonomous Activities of Adiponectin

Adiponectin receptor-intervened activities in different adiponectin target tissues, uncover that adiponectin applies some receptor-free exercises. adiponectin-responsive pre-autonomic (PA) neurons in the hypothalamus don't express AdipoR1 or AdipoR2 . Adiponectin could act in a roundabout way on those AdipoR− PA neurons through endocannabinoidsorneuromodulatory peptides discharged from the AdipoR+neurons because of adiponectin [30]. Adiponectin encourages the take-up of early apoptotic cells by macrophages and adjusts incendiary reactions through a receptor-subordinate pathway that includes calreticulin. RNAi-interceded thumping down of AdipoR1, AdipoR2, and T-cadherin does not influence adiponectin-animated expulsion of apoptotic bodies by macrophages by means of

calreticulindemonstrating that the procedure is free of the known AdipoRs engaged with the established metabolic direction.

Theoretically, receptor-subordinate intracellular flagging for the most part requires low circling ligand focuses, while receptor-free impacts are likely interceded by significantly higher ligand fixations. The physiological groupings of plasma adiponectin is ∼1000-crease higher than that of insulin and further investigations are expected to decide if adiponectin works through both receptor-subordinate mechanisms [31].

Future Difficulties and New Skylines

While adiponectin itself is certainly not an appropriate contender for insulin-sharpening drugs, segments of the adiponectin flagging pathway are promising druggable targets. little particle activator of the adiponectin receptor, AdipoRon, enhances glucose resilience and enhances insulin opposition encouraged a high-fat eating routine. Thus, little atoms that improve adiponectin flagging might be suitable alternatives for the treatment of stoutness connected metabolic maladies including type 2 diabetes. Nonetheless, the present test is that the Kd for little atoms, for example, AdipoRon authoritative to the AdipoR1 or AdipoR2 is considerably higher than that of the full-length or globular space of adiponectin, showing that high-partiality little particle AdipoR activators stay to be recognized [32].

Certain metabolic maladies, for example, type 1 diabetes and IR counter acting agent initiated type B insulin obstruction show high plasma groupings of adiponectin. Regardless of whether this wonder is a consequence of adiponectin obstruction, or compensatory increment to neutralize the inadequate or weakened insulin flagging, is yet to be determined [33]. Exhaustive comprehension of adiponectin and its downstream flagging pathways will give a manual for the advancement of novel medications in the treatment of corpulence related metabolic maladies.

Conclusion

Adiponectin has wide range of physiological and metabolical effects. It also appears to be cardioprotective and involved in modulating the inflammatory response. Its role in systemic inflammation and critical illness is not well-defined .More research is required to establish its role in inflammatory in metabolism.

Corresponding Author

ManishRanjan,

Reader,Department of Conservative Dentistry and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, PoonamalleeHigh Road, Chennai 600077,Tamil Nadu,India.

Phone Number: 9543445029

Email ID: **manish.mds30@gmail.com**

Reference

1. Wang, Y.Adiponectin inhibits cell proliferation by interacting with several growth factors in an oligomerization-dependent manner. J.B.C., 2005; 280(10): 18341-18347.
2. Maury E, BrichardSM.Adipokinedysregulation, adipose tissue inflammation and metabolic syndrome. Mol Cell Endocrinol,2010;314(2):1-16.
3. Matsubara M., Maruoka S. and Katayose S. Decreased plasma adiponectin concentrations in women with dyslipidemia. J. Clin. Endocrinol. Metab, 2002; 87(1): 2764–2769.
4. Stefan N, StumvollM.Adiponectin – its role in metabolism and beyond. HormMetabRes,2002;34(7):469-474.
5. ReavenGM.Syndrome X. Blood Press Suppl,1992;4(4):13-16.
6. WangY.Hydroxylation and Glycosylation of the Four Conserved Lysine Residues in the Collagenous Domain of Adiponectin. J.B.C., 2002; 277(2):19521-19529.
7. Arita Y, Kihara S, Ouchi N, Takahashi M.Paradoxical decrease of an adipose-specific protein, adiponectin, in obesity. Biochem Biophys Res Commun,1999; 257(6):79-83.
8. Shapiro, L. and Scherer, P. E. The crystal structure of a complement-1q family protein suggests an evolutionary link to tumor necrosis factor. Curr. Biol,1998;8(12): 335–338.
9. Scherer PE, Williams S, Fogliano M, Baldini G.A novel serum protein similar to C1Q, produced exclusively in adipocytes. J Biol Chem, 1995;270(3):9.
10. Waki H, Yamauchi T, Kamon J, Ito Y, Uchida S. Impairedmultimerization of human adiponectin mutants associated with diabetes. Molecular structure and multimer formation of adiponectin.J Biol Chem, 2003;278(15):40352–63.

11. Fruebis J, Tsao TS, Javorschi S, Ebbets-Reed D. Proteolytic cleavage product of 30-kDa adipocyte complement related protein increases fatty acid oxidation in muscle and causes weight loss in mice. Proc NatlAcad Sci U S A, 2001;98(2): 2005–10.
12. Waki H, Yamauchi T, Kamon J, Kita S. Generation of globular fragment of adiponectin by leukocyte elastase secreted by monocytic cell line THP-1. Endocrinology, 2005;146(4):790–6.
13. Lihn, A. S., Pedersen, S. B., andRichelsen, B.Adiponectin: action, regulation and association to insulin sensitivity. Obesity Reviews,2005; 8(3):13-21.
14. Maeda, K., Okubo, K.cDNA cloning and expression of a novel adipose specific collagen-like factor, apM1. Biochemical and Biophysical Research Communications,2002;221(2): 286-89.
15. Hara K, Horikoshi M, Yamauchi T, YagoH.Measurement of the high-molecular weight form of adiponectin in plasma is useful for the prediction of insulin resistance and metabolic syndrome. Diabetes Care,2006;29(8):1357-1362.
16. S.Mounika,G.Savitha.Association of Periodontal Diseases and Metabolic Syndrome.Research J. Pharm. and Tech,2015;8(4):994-996.
17. Priyalakshmi.S, Sankari.M.A Review on Metabolic Syndrome and Periodontal Disease.IOSR Journal of Dental and Medical Sciences , 2004;2279(11):01-05.
18. Rutter, M. K., Parise, H., Benjamin, E. J.Impact of glucose intolerance and insulin resistance on cardiac structure and function: sex-related differences in the Framingham Heart Study. Circulation , 2003;107(5): 448–454.
19. 19.Harshini.Adiponectin . International Journal of Pharma and Bio Sciences,2014;6299(14):71.
20. Kanai, H., Matsuzawa, Y., Kotani, K.Close correlation of intra-abdominal fat accumulation to hypertension in obese women. Hypertension,1996;16(6);484–490.
21. Caja S, Torrente M, Martinez I, AbelendaM.Adiponectin values are unchanged during pregnancy in rats. J Endocrinol Invest, 2005;28(2):609–15.
22. Yamauchi T, Kamon J, Waki H, Imai Y. Globular adiponectin protected ob/ob mice from diabetes and ApoEdeficient mice from atherosclerosis. J Biol Chem, 2003;278(10):2461–8.
23. Van Stijn C.M., Kim J., LusisA.J.Macrophage polarization phenotype regulates adiponectin receptor expression and adiponectin anti-inflammatory response. FASEB J,2005; 29(4):636–649.

24. Nechamen C.A., Thomas R.M., Dias J.A. APPL1, APPL2, Akt2 and FOXO1a interact with FSHR in a potential signaling complex. Mol. Cell. Endocrinol,2007; 260-262(7): 93–99.
25. Merl V., Peters A., OltmannsK.M.Serumadiponectin concentrations during a 72-hour fast in over- and normal-weight humans. Int. J. Obes. (Lond),2005;29(2):998–1001.
26. Cheng K.K., Lam K.S., Wang Y.Adiponectin-induced endothelial nitric oxide synthase activation and nitric oxide production are mediated by APPL1 in endothelial cells. Diabetes , 2007;56(1):1387–1394.
27. Tomas E., Tsao T.S., SahaA.K.Enhanced muscle fat oxidation and glucose transport by ACRP30 globular domain: acetyl-CoA carboxylase inhibition and AMP-activated protein kinase activation. Proc. Natl Acad. Sci. USA,2002;99(3):16309–16313.
28. Yamauchi T., Nio Y., Maki T.Targeted disruption of AdipoR1 and AdipoR2 causes abrogation of adiponectin binding and metabolic actions. Nat. Med,2007;13(5):332–339.
29. Holland W.L., Miller R.A., Wang Z.V. Receptor-mediated activation of ceramidase activity initiates the pleiotropic actions of adiponectin. Nat. Med,2001;17(3):55–63.
30. HoydaT.D., Samson W.K., Ferguson A.V. Adiponectin depolarizes parvocellularparaventricular nucleus neurons controlling neuroendocrine and autonomic function. Endocrinology ,2009;150(1): 832–840.
31. Takemura Y., Ouchi N., Shibata R.Adiponectin modulates inflammatory reactions via calreticulin receptor-dependent clearance of early apoptotic bodies. J. Clin. Invest,2007;117(6): 375–386.
32. Okada-Iwabu M., Yamauchi T., Iwabu M.A small-molecule AdipoR agonist for type 2 diabetes and short life in obesity. Nature ,2013;503(2):493–499.
33. Semple R.K., Halberg N.H., Burling K. Paradoxical elevation of high-molecular weight adiponectin in acquired extreme insulin resistance due to insulin receptor antibodies. Diabetes,2007;56(8):1712–1717.

Adverse effects of carbonated soft drinks on oral cavity – An update

S.Pooja[1], Anitha Roy[2], Geo Mani[3],
Graduate Student[1],Professor[2], Department of Pharmacology,
Senior Lecturer[3],Department of Pedodontics and Preventive Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

The aim of this study is to review that over consumption of carbonated soft drinks is a common cause of oral diseases and to understand the oral problems caused by the consumption of carbonated soft drinks.The consumption of soft drinks has increased dramatically over past several decades.The greatest increase in soft drink consumption has occurred among children and adolescents.Dentists are becoming more concerned about over consumption of soft drinks which results in dental disease.High soft drink consumption leads to excessive caloricintake,which causes childhood obesity,a growing problem among children.All soft drinks are acidic which corrodes the teeth by eroding its enamel.The high amount of sugar consumed through soft drinks lead to the development of bacteria that attack the teeth thus aggravating dental problems.From various studies it has been found that carbonated drinks are more aggressive towards dental erosion.hence this articlereviews the adverse effects of carbonated drinks and provides knowledge to the patients about the consequences of soft drink consumption to minimise the risk.

Key Words:*Carbonated drinks,decades,acidic,dental erosion.*

Introduction

Soft drink consumption has become a highly visible with increase energy intake and body weight(1).The soft drinks are popular beverages(2).Recent research has shown that carbonated drinks can have serious effects on teeth in terms of erosion(3).A soft drink is a beverage that is carbonated and it does not contain alcohol.Refreshments like colas, shimmering water , ice tea, lemonade , squash, and organic product punch are among the most widely recognized sorts of sodas, while hot chocolate, hot tea, espresso, drain, tap water, and milkshakes don't fall into this characterisation (4).Carbonation occurs when carbon dioxide is dissolved in water or an aqueous solution(5).

Generally soft drinks are composed of filtered water,artificial additives and refined sugar.They usually lack nutritional value and add up calories and it makes you gain weight. Soft drinks containing inherent acids and sugars have both acidogenic and cariogenic potential. It has been found that dental erosion is considered as a clinical problem in the oral health of school children and young adults and acidic drinks are thought to be an important cause(6).Now days children started consuming soft drinks at a young age, and consumption increases through young adulthood(7).High consumption of soft drinks and other sugary drinks are associated with a number of health problems, including overweight and obesity, type 2 diabetes, osteoporosis and dental caries(8).

High soda pop consumption is linked with widespread demineralization of enamel and extensive caries in pits and fissures and in the interproximal areas(9).It has been found that males of 13 to 18 years old drank two or more cans of soft drinks a day, while females of 13- to 18-year-old drank two cans per day. Adolescents prefer the taste of soft drinks while younger children prefer the taste of still fruit-flavored drinks (10).Those with the strongest taste preference for soft drinks consume more than those with lesser taste preferences.Parental influences such as parental consumption and parenting style are also important factors(11).Causative components for tooth disintegration are isolated into extraneous and inborn classes. Inborn disintegration happens from automatic gastrointestinal unsettling influences, for example, gastroesophageal reflux ailment (GERD) and from intentional disgorging of gastric acids showed by anorexic as well as bulimic people(12).Outward tooth disintegration causes incorporate natural elements, medicaments, way of life, and eating regimen. In present day social orders the outward

factor is ending up more critical, because of the expanded utilization of corrosive beverages as soda pops, don drinks, organic product squeezes and natural product teas(13). Thus this article educates the patient about the adverse effects of carbonated soft drink consumption.

pH of Beverages

Table 1: pH of Beverages

Beverages	PH Value
1. Coca-cola	2.48
2. Sprite	3.27
3. 7up	3.19
4. Pepsi	3.3
5. Mountain dew	3.14

Most soda pops contain maybe a couple regular sustenance acidulants - phosphoric corrosive and citrus extract. Periodically, different acidulants, for example, malic corrosive or tartaric corrosive are additionally used(14).The pH shows how acidic something is. A customary mouth has a pH of 6.3 to 7, which is close unprejudiced with no mischief done to teeth.The pH of the refreshments won't change notwithstanding when the drinks are left revealed at room temperature(15).Many basic drinks have pHs adequately low to dissolve and diminish the lacquer surface(16).All soda pops are exceptionally acidic, yet dim colas, for example, Coke and Pepsi are considerably more acidic. Creature examines have demonstrated that phosphoric corrosive is exceptionally erosive at pH 2.5 however significantly less so at pH 3.3. Citrus, malic and tartaric acids are thought to be particularly erosive as a result of their acidic nature and the capacity to chelate calcium at higher pH.5 Citric corrosive was more erosive than malic corrosive when planned to exploratory beverages at high pH(17).

Effects of Acidic Beverages on Tooth

It is well known that disease loves acid, and particularly true of dental caries. Dental caries, by definition, is tooth demineralization caused by acidic byproducts of the bacterial fermentation of dietary sugars.These acidic, or low pH, beverages can contribute to the demineralization of dental hard tissues(18).The soft drinks are thought to cause damage to the teeth because of two properties – first, the low pH and acidity of some drinks can cause erosion on the enamel surfaces and secondly the fermentablecarbohydrate in drinks is metabolized by plaque micro-organisms to generateorganicacids in the dental plaque and saliva, resulting in demineralization and leading to dental caries(19).Acidic drinks have recognized as a powerful extrinsic factor of dental erosion (20).Studies have discovered that a high commonness of dental disintegration in youngsters and youthful grown-ups identifies with their soda pop utilization or other dietary component. Dental pits are related with carbonated beverage.This is vital in light of the fact that the measure of sugars that are expended is imperative in shaping caries, which is the point at which a hole influences just the veneer, the external defensive layer of a tooth. Individuals who drink at least three glasses of circulated air through beverages (vaporous beverages) every day have substantially higher odds of dental rot, fillings and teeth misfortune.Therefore, soft drinks contain acid and sugar that corrode and destroy the teeth in one shot(21).

Role of Carbonated Drink Consumption on Dental Erosion and Tooth Decay

Tooth deterioration, or tooth wear, is the loss of tooth structure.Basically, tooth disintegration alludes to the wearing endlessly of the critical step of your teeth, which is called polish.The ideal (neutral) pH of the mouth ranges from 6.5-7.5. A pH of 5.5 is considered to be the threshold level for the development of dental decay. Both soft drinks and sports drinks have been shown to have a pH between 2.5 and 3.5. The normal reason for dental disintegration in youngsters is probably going to be acidic refreshments, for example, carbonated games beverages and natural product juices. These have been accounted for to be related with serious loss of dental veneer, especially when expended amid periods when there is little spit in the mouth, for example, instantly after substantial donning exercises (22).Dental erosion seems to have much stronger relationship with soft

drinks. The erosive potential of drinks is mainly represented by their pH and the buffering capacity.More studies showed that dental erosion was associated with the drinking methods. Holding the drink longer in the mouth leads to a more pronounced pH drop(23). Drinking with an increasing flow rate and with decreasing outlet diameter could increase the erosion depth (24). The effect is also strengthened when acid temperature grows higher(25).Few studies have reported that Animal studies have shown that phosphoric acid is very erosive at pH 2.5 but much less so at pH 3.3. Citrus, malic and tartaric acids are thought to be particularly erosive in view of their acidic nature and the capacity to chelate calcium at higher pH.5. Citrus extract was more erosive than malic corrosive when defined to test drinks at high pH.This acid, plus the extra acid already present in the drink, demineralizes (weakens) your tooth enamel. The enamel is the strong, outer coating of the teeth that guards against tooth decay and protects the teeth lifelong.When the acid makes it weak, tooth decay (cavities) can begin. Refined sugars play a pivotal role in the caries (tooth decay) process. Regular (non-diet) soft drinks contain large amounts of sucrose or high fructose corn syrup that are considered to be highly cariogenic (high decay potential). A recent study in the U.S. showed a positive relationship between cumulative caries (tooth decay) scores and the frequency of mealtime and between-meal use of carbonated beverages. Daily between-meal consumption of soft drinks three or more times per day has been shown to increase the risk of dental decay by 179%. Research has shown that bedtime is the worst time to consume sugar-sweetened drinks.

Signs of Soft Drink Erosion

There would be a change in morphology and surface characteristics of the teeth .Smooth surface enamel may develop broad shiny concavities. The teeth may even have a glazed appearance(26).

Wide buccal concavities in mandibular premolars and molar.Concavities with an enamel cuff at the free gingival margin. Deep shiny concavities occlusally in premolars and molars.Restorations that ‘rise’ above the occlusal surface.Sealants that ‘rise’ above the occlusal surface.Thin maxillary central incisors.Increased incisal translucency in maxillary central incisors.Surface characteristics missing.Loss of surface detail in the

primary dentition(27).

Dental examination found that crescent-shaped lesions were present on the cervical region of the buccal and labial surfaces of the teeth of thepatients(28). The individual manner of drinking acidic soft drinks has been said to affect how long the teeth are in contact with the erosive challenge and therefore influence the pattern of destruction caused by them. The drinking method strongly affects tooth-surface pH and thereby the risk for dental erosion. Six different methods of drinking were tested in a randomized order by Johansson Holding; short-sipping; long-sipping; gulping; nipping; and sucking(29).

Other Health Risks Associated with Softdrinks

It likewise is critical for general dental practitioners to comprehend the foundational wellbeing results of over the top utilization of sodas. Expanded vitality consumption identified with sodas has been related with an expanded level of corpulence among American kids and young people(30).Another result of expanded soda pop utilization has been a decrease in the utilization of dairy items. This has brought about the dislodging of many required supplements for the two youngsters and teenagers and in addition for grown-up females(31).Consuming more phosphate than calcium can have a deleterious effect on bone health(32). The Nurses' Health Study, which followed the soundness of about 90,000 ladies more than two decades, found that ladies who drank more than two servings of sugary drink every day had a 40 percent higher danger of heart assaults or demise from coronary illness than ladies who infrequently drank sugary refreshments(33).

Results from Other Studies

Al-Majed found an on a very basic level higher inescapability of dental crumbling in young women (29.9%) than in young fellows (25.7%), which is in simultaneousness with the results of past examination of 12-year-old Cuban children by Künzel,However, Dugmore besides, Truin. have found an in a general sense higher regularity in young fellows than in young women, while Correr, Peres likewise, Ping Wang et al. found no complexity between sexual introductions. Nazriya,found that Carbonated drinks,

particularly coke has most astounding pH and buffering limit took after by Fanta and Sprite. An examination by priyadharshini et al indicated 78.5% of the young people among chennai populace are dependent on carbonated refreshments. An examination by MahumuthaAffshana propose that disintegration is as predominant in chennai teenagers. Conceivable reason may incorporate Sampling variety or territorial varieties. Another examination by shreds.s,2015 was discovered that 73% of understudies favored carbonated beverages. An in vitro consider by Grenby ,recommendedtitratable corrosive was a superior guide than pH to decide erosive properties of refreshments. Be that as it may, add up to sharpness or pH estimations of refreshments couldn't be utilized with dependability to foresee their capability to cause molar disintegration. An investigation Hassan et al looked at corrosive freedom of noncarbonated and carbonated soda pops in the mouth, there was no measurable contrast in the leeway between the beverages. It was reasoned that carbonation may not specifically in charge of the erosive capability of various refreshments. Levitch,further recommended that a blend of disintegration and scraped spot may work synergistically to advance improvement of non-carious cervical injuries. The clinical ramifications of this is toothbrushing promptly after ingestion of acidic substances or refreshments may accomplish more damage than great by quickening tooth structure misfortune. A few investigations have uncovered lessened salivary cushion limit in patients with disintegration when contrasted and controls while others affirm low salivary stream rates as the significant determinants in a few patients with disintegration.

Reducing Tooth Decay and Dental Erosion

Drink fluoridated water and use fluoride toothpaste.

Swish out your mouth with water to dilute the sugar and acid.

Use a straw to keep sugars and acids away from your teeth.

Never consume soft drinks or juice at bedtime. (The liquid pools in your mouth and coats your tongue and teeth.)(46).

Discard the top to anticipate progressing tasting.

Read the names sweetened beverages are high in sugar.

Get regular dental cleanings to remove plaque (bacteria) build-up on your teeth(47).

Edwards and co-workers recommended that drinking through a straw positioned toward

the back of the mouth may reduce the erosive potential of soft drinks(48).

Patient should avoid toothbrushing enamel which has been softened by exposure to black cola. This may cause additional material loss due to tooth brush abrasion(49) Kids ought to be urged to drink acidic beverages rapidly and utilize a straw with the goal that the fluid is pushed to the back of the mouth.Also, avoid buying re-sealable bottles and discourage sipping over a long period of time. The liquid fruits can be used as a natural alternative to synthetic beverages(50).

Conclusion

Excessive intake of soft drinks could cause complex dental consequences including dental erosion and caries.It is necessary to educate patients about the harmful effects of excessive soft drink consumption.fluoride can be used to decrease the potential ravages of soda pop.Programmes could bedeveloped and administered in schools, though there is also clearly a role for parents in helping to reduce carbonated drink consumptionindicated by the increased consumption at weekends.Conversely, the negative effects of carbonated drink consumption should be emphasized to younger generations.

Corresponding Author

Geo Mani,

Senior Lecturer,Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai -600 077,Tamil Nadu, India.

Email Id: geosdch@gmail.com

Telephone number: 7358433246

References

1. Vartanian LR, Schwartz MB, Brownell KD. Effects of soft drink consumption on nutrition and health: a systematic review and meta-analysis. American journal of public health. 2007;97(4):667-75.

2. Erickson PR, Alevizos DL, Rindelaub DJ. Soft drinks: hard on teeth. Northwest dentistry. 2001;80(2):15-9.
3. Buchanan H, Coulson NS. Consumption of carbonated drinks in adolescents: a transtheoretical analysis. Child: Care, health and development. 2007;33(4):441-7
4. Kishore S, Aggarwal P, Muzammil K. KAP Study About Carbonated Drinks Among Medical and Nursing Students at Teaching Hospital.JK Science, 2009, 11 (4),196.
5. Amato D, García-Contreras F, Paniagua R. Carbonated beverage consumption and bone fractures. Archives of pediatrics & adolescent medicine. 2001 1;155(2):200-1.
6. Seow WK, Thong KM. Erosive effects of common beverages on extracted premolar teeth. Australian Dental Journal. 2005;50(3):173-8.
7. Erickson PR, Alevizos DL, Rindelaub DJ. Soft drinks: hard on teeth. Northwest dentistry. 2001;80(2):15-9.
8. Rangan A, Hector D, Louie JC, Flood VM, Gill T. Soft drinks, weight status and health: health professional update. NSW Cluster of Public Health Nutrition,2009:1-25.
9. Kaplowitz GJ. An update on the dangers of soda pop. Dental assistant.2011 ;80(4):14.
10. May J, Waterhouse PJ. Dental erosion and soft drinks: a qualitative assessment of knowledge, attitude and behaviour using focus groups of schoolchildren. A preliminary study. International journal of paediatric dentistry. 2003 1;13(6):425-33.
11. Grimm GC, Harnack L, Story M. Factors associated with soft drink consumption in school-aged children. Journal of the American Dietetic Association. 2004;104(8):1244-9
12. Gandara BK, MSD and Edmond L Truelove MS. Diagnosis and management of dental erosion. The Journal of Contemporary Dental Practice. 1999 25;1(1):1-7.
13. Shipley S, Taylor K, Mitchell W. Identifying causes of dental erosion. General dentistry. 2005;53(1):73-5.
14. Rugg-Gunn AJ, Hackett AF. Nutrition and dental health. Oxford University Press,USA; 1993. (2): 1471-2458.

15. Seow WK, Thong KM. Erosive effects of common beverages on extracted premolar teeth. Australian Dental Journal. 2005;50(3):173-8.
16. Seow WK, Thong KM. Erosive effects of common beverages on extracted premolar teeth. Australian Dental Journal. 2005;50(3):173-8.
17. Hughes JA, West NX, Parker DM, Van Den Braak MH, Addy M. Effects of pH and concentration of citric, malic and lactic acids on enamel, in vitro. Journal of dentistry. 2000;28(2):147-52.
18. Holloway PJ, Mellanby M, Stewart RJ. Fruit drinks and tooth erosion. Brit. dent. J.. 1958;104:305-309.
19. Grobler SR, Jenkins GN, Kotz D.The effect of consumption and method of drinking. Br Dent J. 1985 ;158(8):293-6.
20. Agostinelli G, Brown JM, Miller WR. Effects of normative feedback on consumption among heavy drinking college students. Journal of drug education.1995 ;25(1):31-40.
21. Kishore S, Aggarwal P, Muzammil K. KAP Study About Carbonated Drinks Among Medical and Nursing Students at Teaching Hospital.JK Science;2009;11 (4):196
22. Khan F, Young WG, Law V, Priest J, Daley TJ. Cupped lesions of early onset dental erosion in young southeast Queensland adults. Australian dental journal. 2001;46(2):100-7
23. Johansson AK, Lingström P, Imfeld T, Birkhed D. Influence of drinking method on tooth- surface pH in relation to dental erosion. European journal of oral sciences. 2004;112(6):484-9.
24. Shellis RP, Finke M, Eisenburger M, Parker DM, Addy M. Relationship between enamel erosion and liquid flow rate. European journal of oral sciences. 2005;113(3):232-8
25. Eisenburger M, Addy M. Influence of liquid temperature and flow rate on enamel erosion and surface softening. Journal of oral rehabilitation.2003 ;30(11):1076-80.
26. Lussi A, Jaeggi T, Zero D. The role of diet in the aetiology of dental erosion. Caries research. 2004;38(1):34-44.
27. Johansson AK, Lingström P, Imfeld T, Birkhed D. Influence of drinking method

on tooth- surface pH in relation to dental erosion. European journal of oral sciences. 2004;112(6):484-9.

28. Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB. High glycemic index foods, overeating, and obesity. Pediatrics,1999 ;103(3):26-28.
29. Cavadini C, Siega-Riz AM, Popkin BM. US adolescent food intake trends from 1965 to 1996. Archives of disease in childhood, 2000;83(1):18-24.
30. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review–. The American journal of clinical nutrition,2006 ;84(2):274-88.
31. Fung TT, Malik V, Rexrode KM, Manson JE, Willett WC, Hu FB. Sweetened beverage consumption and risk of coronary heart disease in women–. The American journal of clinical nutrition,2009;89(4):1037-1042.
32. Lussi A, Jaeggi T, Zero D. The role of diet in the aetiology of dental erosion. Caries research,2004;38(1):34-44.
33. Milosevic A. Sports drinks hazard to teeth. British journal of sports medicine, 1997 ;31(1):28-30.
34. Al- Majed I, Maguire A, Murray JJ. Risk factors for dental erosion in 5–6 year old and 12–14 year old boys in Saudi Arabia. Community Dentistry and Oral Epidemiology,2002 ;30(1):38-46.
35. Künzel W, Cruz MS, Fischer T. Dental erosion in Cuban children associated with excessive consumption of oranges. European Journal of Oral Sciences, 2000;108(2):104-9.
36. Truin GJ, Van Rijkom HM, Mulder JV, Van‘t Hof MA. Caries trends 1996–2002 among 6-and 12-year-old children and erosive wear prevalence among 12-year-old children in The Hague. Caries Research. 2005;39(1):2-8.
37. Correr GM, Alonso RC, Correa MA, Campos EA, Baratto-Filho F, Puppin-Rontani RM. Influence of diet and salivary characteristics on the prevalence of dental erosion among 12-year-old schoolchildren. Journal of Dentistry for Children. 2009; 76(3):181-187.

38. Edwards M, Creanor SL, Foye RH, Gilmour WH. Buffering capacities of soft drinks: the potential influence on dental erosion. Journal of oral rehabilitation. 1999;26(12):923-927.
39. Priyadharshini.T ,Gayathridevi.R. Caffeine consumption habits and perception of adolescents in chennaipopulations.Asian J Pharm Clin Res, 2016; 9(3):149-151.
40. Affshana M. Acid Erosion of Teeth in a Pediatric Population. Journal of Pharmaceutical Sciences and Research,2015 ;7(7):435.
41. Shreya S, Priya V. A study on the Effect of Aerated Drinks on Enamel Surface of the Tooth. Research Journal of Pharmacy and Technology,2015 ;8(8):1137.
42. Grenby TH, Mistry M, Desai T. Potential dental effects of infants' fruit drinks studied in vitro. British Journal of Nutrition,1990 ;64(1):273-283.
43. Hassan L, Wilson R, Bartlett D. Comparison of acid clearance of noncarbonated and carbonated soft drinks in the mouth. International Journal of Prosthodontics. 2007;20(2):114
44. Levitch LC, Bader JD, Shugars DA, Heymann HO. Non-carious cervical lesions. Journal of Dentistry. 1994;22(4):195-207.
45. Jarvinen VK, Rytomaa II, Heinonen OP. Risk factors in dental erosion. Journal of dental research. 1991 ;70(6):942-7.
46. Addy M. Dentine hypersensitivity: new perspectives on an old problem. International Dental Journal,2002;52(S5P2):367-75.
47. Cheng R, Yang H, Shao MY, Hu T, Zhou XD. Dental erosion and severe tooth decay related to soft drinks: a case report and literature review. Journal of Zhejiang University science B,2009 ;10(5):395-9.
48. Shellis RP, Finke M, Eisenburger M, Parker DM, Addy M. Relationship between enamel erosion and liquid flow rate. European journal of oral sciences, 2005 ;113(3):232-8.
49. Erickson PR, Alevizos DL, Rindelaub DJ. Soft drinks: hard on teeth. Northwestdentistry,2001;80(2):15-9.
50. Kim JW, Jang KT, Lee SH, Kim CC, Hahn SH, García-Godoy F. In vivo rehardening of enamel eroded by a cola drink. ASDC journal of dentistry for children,2001;68(2):122-4.

Albumin As A Biochemical Marker In Acute Myocardial Syndrome

Ashok Kumar.A[1], Anitha Roy[2], Pradeep.D[3]

Graduate Student[1],Professor and Head [2],Department of Pharmacology,

Reader [3],Department of Oral and maxillofacial surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract:

Aim and objective: To evaluate use of albumin as a biochemical marker for detection of acute myocardial syndrome.

Background: Myocardial Ischemia occurs when there is a lack of blood perfusion of the myocytes, leading to deficiency of oxygen and nutrients, thus affecting their vital functions. The diagnosis of cardiac ischemia always remains as a challenge in emergency medicine. There is a need for early and sensitive markers of cardiac ischemia as current diagnostic tests fail to identify many chest pain patients and who are at a high risk for adverse cardiac events. A number of candidate biomarkers have been released for the detection of cardiac ischemia however, ischemia modified albumin(IMA) is proven to be the most efficient and has been released for usage in clinics. Changes in IMA concentration have shown to occur during coronary angioplasty-induced ischemia. Clinical studies indicate that IMA appears to offer on admission an early test which can be combined with electrocardiographic findings and cardiac troponin measurements for the early exclusion of acute coronary syndrome. The mechanism of IMA formation and the precise entity being measured are not fully known. Nevertheless, IMA measurement remains the only current clinical biomarker which may be used for the diagnosis of patients suspected of cardiac ischemia.

Keywords - Ischemia, Albumin, Biomarker, Ischemia modified albumin, Albumin Cobalt Binding Assay, Acute myocardial syndrome.

Introduction:

Ischemia occurs due to deficiency in the cardiac blood flow, leading to deficiency of oxygen and nutrients, and so affecting their vital functions. If Ischemia is reversible, no myocardial damage occurs. Necrosis of the cell and myocardial ischemia results if the ischemia is irreversible. Manifestations of myocardial ischemia differs like chest pain, epigastric or arm discomfort , breathlessness , nausea and vomiting. However the ultimate challenge in medicine is to identify myocardial ischemia before necrosis has occurred. There is a need for early and sensitive markers of cardiac ischemia as. The current diagnostic tests fail to identify many chest pain patients and who are at a high risk for adverse cardiac diseases, hence there exists a need to identify certain markers of cardiac ischemia. Cardiac markers of cell necrosis such as myoglobin, CK-MB and troponins are highly sensitive and exhibit good specificity. However they do not detect myocardial ischemia in the absence of necrosis[1,2] .

Various biomarkers used in detection of myocardial infarction are as follows : Myeloperoxidase, MMP-9, sCD40L, Pregnancy associated plasma protein A, Choline, Ischemia modified albumin, unbound free fatty acids, Glycogen phosphorylase isoenzyme BB, Placental growth factor. Recently, a new biomarker has been developed Ischemia modified albumin(IMA). IMA is produced during an ischemic attack and present in blood in easily detectable concentrations. This review will highlight the role of albumin as a biochemical marker in acute myocardialsyndrome.

Mechanism of production of IMA:

The N-terminal portion of human serum albumin (HSA) is a binding site for transition metal ions such as cobalt, copper and nickel[2] . Many divalent metals bind HSA in the circulation but in concentrations far lower than that required to impact albumin directly. The N-terminal portion of HSA has higher chances of biochemical degradation and has less stability than the albumin of other species[3] . IMA is a form of HSA in which the N-terminal amino acids are unable to bind to transition metals. Myocardial ischemia generates free radicals [4,5] acidosis develops [6] and release of free iron and copper ions occurs [7,8]. HSA is a scavenger for divalent metal ions. It may thus be postulated that in ischemia, these processes may result in change in the ability of the N-terminus to bind to transition metal ions. The release of these ions

likely initiates one potential pathway for IMA generation and thus need not be considered interference to negatively affect IMA. The postulated mechanism is that localized ischemia results in acidosis and the release of copper II from weak binding sites on circulating proteins. Ascorbic acid which acts as a reducing agent converts free copper II to copper I and reacts with oxygen to form copper II which generates superoxide free radicals. Superoxide dismutase converts the superoxide free radicals to hydrogen peroxide which is then degraded by catalase. Copper II ions released are immediately scavenged by albumin but are tightly bound to the N terminus. Copper bound albumin is then damaged by hydroxyl free radicals, causing removal of the three N terminal amino acids and release of copper II ions to repeat the process in a chain reaction [9] . An increase in IMA was observed in patients minutes after transient occlusion and reperfusion during coronary angioplasty [10,11] .

Clinical interpretation of IMA:

The use of biomarkers for the identification of suspected acute coronary syndromes depends on the presence of myo necrosis as a surrogate indicator for myocardial ischemia. It has been reported that in the setting of myocardial ischemia, there are modifications to the amino acids of the N terminus of the human albumin molecule. These modifications alter the N-terminus in such a way that it can no longer bind transition metals, such as cobalt [12] From this observation, an assay measuring the ability of albumin to bind cobalt was developed, which has been suggested to be useful for the detection of myocardial ischemia. In experimental models and in clinical studies, IMA has been shown to rise within minutes after the onset of ischemia, stay elevated for 6 to 12 hours, and return to normal within 24 hours [13,14] . Furthermore, IMA has been shown to predict with high sensitivity subsequent elevation in the troponins in the clinical setting [15].

Aslan D et al determined reference values of IMA from a population of 283 healthy individuals and ranged from 52 to 116 kilounits/L, with a 95th percentile at 85 kilounits/L [16] . While the optimum cutoff for IMA for ruling out ACS is 85 kilounits/L, a higher value of 100 kilounits/L is suggested for risk stratification. IMA is normally distributed in a group of apparently healthy volunteers and is not correlated with smoking, age, race, gender, or Framingham risk score. IMA is not specific for cardiac ischemia. IMA has an elevated level in

most patients with cirrhosis of liver, advanced cancers and acute stage infections; all these conditions produce free radicals of considerable quantity. IMA seems to be produced in patients with brain ischemia (stroke), end-stage renal disease and intrauterine ischemia [17,18] . Data exist suggesting that very low albumin levels and the presence of co-existing lactic acidosis might affect assay performance [19,20] . Additional data are needed to clarify the impact of total albumin alterations on IMA interpretation. Moreover, the ACB test is an indirect measure of IMA and such albumin has not yet been isolated [21] . An immunochemical assay, which would improve the analytical sensitivity of the test but might not have an impact on the clinical specificity, has not yet been developed.

Clinical applications of IMA:

IMA as a marker of supply ischemia:

Ischemia detection is very difficult to detect prior to infarction and remains as a challenging concept. It would be very helpful to be able to identify quickly and accurately which patients really have myocardial ischemia and may be in need of either treatment or intervention to prevent subsequent events. In clinical trials use of IMA has performed reasonably well, focusing on its capability for early measurement to characterize acute coronary syndrome patients. Blood levels of IMA rise promptly during myocardial ischemia triggered by a primary reduction of blood flow (supply ischemia), as seen in patients undergoing percutaneous coronary intervention (PCI), stay elevated for about 6 hours and return to baseline within 12 hours [22,23,24] In a previous report, IMA production was decreased in patients with collateral vessels, which is likely to be a protective effect of collateral circulation against PCI-induced myocardial ischemia-reperfusion injury [23] . IMA levels and objective markers of ischemia such as lactate levels and isoprostane concentrations are believed to be in good correlation according to some studies in this setting [24] . Moreover, IMA was shown to increase significantly in patients undergoing intracoronary ergonovine spasm provocation test and may have a role as a biochemical marker for transient myocardial ischemia induced by coronary vasospasm [25].

However, as promising as the IMA assay appears some degree of caution is still suggested. The currently accepted strength of IMA lies in its negative predictive value for

excluding the presence of ischemia. Unfortunately, predictive values cannot necessarily be translated into clinical practice; in reality, predictive value is less a measure of test performance than it is a reflection of disease prevalence in the population being tested. It is a basic principle of diagnostic testing that adding tests together increases sensitivity. Thus, adding IMA to traditional markers will increase sensitivity, but so would adding other biomarkers. In this manner, specificity will be reduced, leading to an even higher false-positive testing rate. Nevertheless, IMA currently remains the only ischemia assay to have reached the clinical validation stage. For efficient provision of care in the Emergency Department, a high negative predictive value may be critical, because the correct exclusion of ACS preserves limited and expensive resources, such as stress tests, hospital beds and catheterization slots [26].

IMA as a marker of demand ischemia :

A number of studies have demonstrated that exercise may induce perturbations in circulating IMA levels [27-34] . In a study the IMA concentration has been shown to decrease immediately post race followed by an elevation 24-48 hours post exercise in 19 healthy marathon runners [28] . Similar findings were confirmed by additional reports assessing IMA levels in healthy individuals following completion of high workload exercise [29] . The IMA has also been evaluated in healthy subjects following hand grip and was found to decrease significantly after forearm ischemia and to return to baseline thereafter; similar changes were reported for the IMA:albumin ratio. However, Falkensammer et al reported a significant increase in circulating IMA concentrations in 12 healthy volunteers after exercise induced calf-muscle ischemia [30] . In the same study a significant negative correlation was found between IMA and albumin, but no association of IMA levels with lactate was observed. It is clear that there is no homogeneous response in exercise-induced changes of IMA. Exercise results in a number of potential ischemic sites, such as skeletal muscle or gastrointestinal tract. It seems that the hemoconcentration that occurs during physical exercise induces an increase in albumin plasma levels and, consequently, a decrease in the non-bound portion of a fixed amount of cobalt. Still one should also take into account that myocardial ischemia during a stress test may be different in either physical or pharmacological stress and may not be as severe as the ischemia that occurs during angioplasty or in the ACS settings. Lactic acidosis can also interfere with the analysis of IMA as there is a negative correlation between them. Hence, significant care must be taken in the interpretation of

post-exercise IMA data. Elucidation is required to understand the mechanism for detectable transient changes of IMA in response to demand ischemia. To date, there is no approval for use of this test in conjunction with a stress test. Standardized and rigid protocols are required for clinical assessment of IMA after ischemic exercise and sampling time points and similar study populations.

IMA as a marker of myocardial injury:

IMA has been evaluated as a potential indicator of transient myocardial ischemia in different clinical models of myocardial injury. Radiofrequency (RF) catheter ablation is a widely used treatment for cardiac arrhythmias but results in a detectable injury to the myocardium, which is unrelated to ischemia-reperfusion injury per se [35] . Moreover, any changes in IMA in association with RF ablation cannot be attributed to albumin variations because albumin levels are not expected to vary in relation to this procedure. Several researchers found that IMA plasma levels did not change immediately after, 2 and 20 hours after RF ablation compared to baseline values, whereas CK, CK-MB and troponin I increased significantly compared to baseline [36]. However, a previous report on IMA in RF ablation showed that IMA significantly increased 30 min after the procedure and returned to baseline values in 8 hours [37] . The different timing may explain the contradiction between the latter study's findings and ours, although the effects of ischemia on the N-terminal region of albumin can be detectable up to 6 hours after the ischemic event. There is one study reporting that IMA plasma levels increase 1 and 6 hours following cardioversion in patients with AF, along with an increase of CK and CK-MB; [38] it may be that cardioversion produces skeletal damage at the proximal site of shock applications, along with cardiac ischemia in the more distal cardiac tissues. Transvenous lead implantation of either permanent pacemakers or defibrillators induces minimal myocardial injury, expressed as a rise in cardiac troponins, which are very sensitive and specific markers of myocardial cell necrosis [35] . E. Sbarouni et al found that IMA and CK increased at 6 and 48 hours after permanent pacemaker/defibrillator implantation compared to baseline values, whereas CK-MB and troponin I increased at 6 hours and returned to baseline at 48 hours [39] . These data suggest that insertion of endocardial leads is accompanied in most patients by minimal necrosis. This seems to be preceded by transient ischemia, as shown by a significant increase in IMA levels, which could be attributed to myocardial ischemia. Despite the lack of evidence in the aforementioned clinical

situations, it could be speculated that the underlying mechanism may be the damage to the N-terminus by reactive oxygen species or disruption at the cellular level. Recently, IMA has been described as a useful discriminative marker to exclude pulmonary embolism, which is known to be associated with right ventricular dysfunction and myocardial ischemia [40] . The serum IMA levels of 30 patients with definitively diagnosed pulmonary embolism were significantly higher than those of 30 healthy volunteers[41].

Measuring IMA:

Albumin cobalt binding test:

IMA was measured by the albumin cobalt binding test (ACB Test) on the Roche Cobas MIRA PLUS instrument. The mechanism whereby IMA represents a marker of ischaemia is based upon the fact that human serum albumin (HSA) has the ability to bind certain transition metal ions, particularly cobalt and copper, at the N-terminus. The structure of HSA N-terminus changes after exposure to albumin such that it can no longer bind cobalt was previously reported by Bar-Or [12,41]. Blood was collected for the IMA test in serum separated tubes. Specimens were frozen at 220°C or colder within two hours. Frozen samples were gently vortexed after thawing. Specimens handled in this way showed no significant
difference in assay results from the fresh specimens. In the ACB Test, 95 ml of a patient sample and 5 ml of cobalt chloride (CoII), are incubated for five minutes. During incubation, the Co(II) binds to the N-terminus of unaltered albumin in the sample; albumin for which the N-terminus is altered as a result of ischaemic processes binds to the Co(II) to a far lesser extent. After incubation, 25 ml of dithiothreitol (DTT) is added to the mixture. DTT forms a coloured complex with Co(II) that is not bound at the N-terminus of albumin, and this complex is measured spectrophotometrically at 500 nm. The mean recorded as the result of the assay was used to obtain duplicate IMA values The assay is based on the premise that myocardial ischemia causes changes in human serum albumin (HSA) that are demonstrated by reduced exogenous

cobalt (II) binding. A known amount of cobalt (II) is added to a serum specimen and measurement of the unbound cobalt (II) by colorimetric assay using dithiothreitol (DTT) is used to determine the concentration of ischemia modified serum albumin. An inverse relationship thus exists between the level of albumin bound cobalt and the intensity of the color formation [42].

Prognostic value of IMA :

In the early hours of ACS, selection of patients who are at high risk for cardiac events is an important factor in determining the appropriate treatment strategy. The use of serum IMA concentrations for the early prediction of adverse clinical outcomes in patients with suspected ACS has only recently been the subject of investigation, but shows controversial results. Worster et al enrolled 189 patients who presented to an Emergency Department within 6 hours after chest pain, seeing an emergency physician who ordered a cTn-I test, and who had no serious cardiac outcome before the troponin result became available [43]. Of the 189 patients, 24 had a serious cardiac outcome within 72 hours after their arrival at the Emergency Department. The timely ratios for measurement of IMA within 6 hours after chest pain predicting a serious cardiac outcome within the next 72 hours were 1.35 (95% confidence interval [CI] 0.315-5.79) for IMA≤80 Units/mL and 0.98 (95% CI 0.86-1.11) for IMA>80 Units/ mL. Although these data suggest that IMA is a poor predictor of serious cardiac outcomes in the short term, they may be explained by taking into account the following:

Elevation of IMA is non-specific and hence may induce any prognostic significance if it is used for prognosis before first establishing the diagnosis. Post-PCI IMA elevation more than 130 kilounits/L was associated with higher target lesion revascularization at nearly 4-years follow-up (p=0.026)[44]. Interestingly, in patients with ST segment elevation myocardial infarction treated with PCI and who developed heart failure, IMA levels were significantly associated with a decreased left ventricular ejection fraction, representing an early marker of left ventricular dysfunction.[45]

IMA levels vary considerably, even among healthy individuals, and taking these variations into account may improve the predictive characteristics of IMA. Aparci et al have also investigated the prognostic significance of IMA in patients with ACS. IMA was found to be significantly related to 1-year mortality but with a cut-off value of 477 Units/mL. The prognostic

significance of IMA needs be evaluated in large populations and randomised study groups before it can be used for risk stratification in patients with ACS.

Conclusion :

The remarkable increase in the discovery of new biomarkers demands great attention to their assessment before clinical application. The value of IMA lies in its negative predictive value for excluding the presence of ischemia in a population with a low prevalence of coronary artery disease. Rather than providing evidence of ischemia, perhaps its greatest role will be reassurance. Although, this marker is not very specific, it is highly sensitive when negative. An elevated level of IMA also appears to be an outcome predictor which is an interesting thought. Before changing clinical practice, meaningful studies addressing utility, outcomes and cost-effectiveness need to be conducted. “Crossing the boundary from research to clinical application,will require replication in multiple settings, experimental evidence supporting a pathophysiologic role, and ideally, intervention trials demonstrating that modification improves outcomes” .

Corresponding author:
Pradeep.D,
Reader,Department of Oral and maxillofacial surgery,
Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences.
162 Poonamalle High Road ,Chennai 600077,Tamil Nadu, India.
Email : pradeep.dhasa@gmail.com
Telephone Number: 9789936383

References:

1) Kontos MC, Jesse RL. Evaluation of the emergency department chest pain patient. American Journal of Cardiology. 2000 Mar 9;85(5):32-9.

2) Farkouh ME, Smars PA, Reeder GS, Zinsmeister AR, Evans RW, Meloy TD, Kopecky SL, Allen M, Allison TG, Gibbons RJ, Gabriel SE. A clinical trial of a chest-pain observation unit

for patients with unstable angina. New England Journal of Medicine. 1998 Dec 24;339(26):1882-8.

3) Chan B, Dodsworth N, Woodrow J, Tucker A, Harris R. Site-specific N-terminal auto-degradation of human serum albumin. The FEBS Journal. 1995 Jan 1;227(1-2):524-8.

4) McCord JM. Oxygen-derived free radicals in postischemic tissue injury. New England Journal of Medicine. 1985 Jan 17;312(3):159-63.

5) Levine, R. L.: Ischemia: from acidosis to oxidation. FASEB J., 2013 Mar 7(13): 1242–1246.

6) Cobbe SM, Poole-Wilson PA. The time of onset and severity of acidosis in myocardial ischaemia. Journal of molecular and cellular cardiology. 1980 Aug 1;12(8):745-60.

7) Berenshtein, E., Mayer, B., Goldberg, C., Kitrossky, N. and Chevion, M.: Patterns of mobilization of copper and iron following myocardial ischemia: possible predictive criteria for tissue injury. J. Mol. Cell Cardiol., 29(11): 3025–3034 (1997).

8) Chevion, M., Jiang, Y., Har-El, R., Berenshtein, E., Uretzky, G. and Kitrossky, N.: Copper and iron are mobilized following myocardial ischemia: possible predictive criteria for tissue injury.Proc. Natl. Acad. Sci. U.S.A., 90(3): 1102–1106 (1993).

9) Marx, G. and Chevion, M.: Site-specific modification of albumin by free radicals. Reaction with copper(II) and ascorbate. Biochem. J., 236(2): 397–400 (1986).

10) Sinha M, Gaze D C, Tippins J R. *et al* Ischemia-modified albumin is a sensitive marker of myocardial ischemia after percutaneous coronary intervention. Circulation 20031072403–2405.2405

11) Bar-Or D, Winkler J V, Vanbenthuysen K. *et al* Reduced albumin-cobalt binding with transient myocardial ischemia after elective percutaneous transluminal coronary angioplasty: a preliminary comparison to creatine kinase-MB, myoglobin, and troponin-I. Am Heart J 2001141985–991.991

12) Bar-Or D, Curtis G, Rao N, et al. Characterization of the Co2+ and Ni2+ binding amino-acid residues of the N terminus of human albumin: an insight into the mechanism of a new assay for myocardial ischemia. *Eur J Biochem.* 2001;268:42-47.

13)Sinha M, Gaze D, Collinson P, et al. Ischemia modified albumin (IMA) is useful for discriminating acute coronary syndromes from nonischemic presentations in the emergency department. *J Am Coll Cardiol.* 2003;41:6(suppl A):402A.

14) deFilippi C, Yoon S, Ro A, et al. Early detection of myocardial ischemia by a novel blood based biomarker: the kinetics of ischemia modified albumin. *J Am Coll Cardiol*. 2003;41:6(suppl A):340A.

15) Christenson RL, Duh SH, Sanhai WR, et al. Characteristics of an Albumin Cobalt Binding Test for assessment of acute coronary syndrome patients: a multicenter study. *Clin Chem.*2001;47:464-470.

16) Aslan D, Apple FS. Ischemia modified albumin: clinical and analytical update. Lab Med. 2004; 35: 1-5. 18) Abboud H, Labreuche J, Meseguer E, et al. Ischemia-modified albumin in acute stroke. Cerebrovasc Dis. 2007; 23: 216-220.

17) Deepa T, Thirrunavukkarasu N. Saliva as a potential diagnostic tool. Indian J Med Sci 2010;64:293-306.

18) Gaze DC, Crompton L, Collinson P. Ischemia-modified albumin concentrations should be interpreted with caution in patients with low serum albumin concentrations. Med Princ Pract. 2006; 15: 322-324.

19) Zapico-Muñiz E, Santaló-Bel M, Mercé-Muntañola J, Montiel JA, Martínez-Rubio A, Ordóñez-Llanos J. Ischemia-modified albumin during skeletal muscle ischemia. Clin Chem. 2004; 50: 1063-1065.

20) Bhagavan NV, Lai EM, Rios PA, et al. Evaluation of human serum albumin cobalt binding assay for the assessment of myocardial ischemia and myocardial infarction. Clin Chem. 2003; 49: 581-585.

21) Nandhini.S. Essential Hypertension –A Review Article. J. Pharm. Sci. & Res. Vol. 6(9), 2014, 305-307.

22) Garrido IP, Roy D, Calvino R, et al. Comparison of ischemiamodified albumin levels in patients undergoing percutaneous coronary intervention for unstable angina pectoris with versus without collaterals. Am J Cardiol. 2004; 93: 88-90.

23) Sinha MK, Vazquez JM, Calvino R, Gaze DC, Collinson PO, Kaski JC. Effects of balloon occlusion during percutaneous coronary intervention on circulating ischemia modified albumin and transmyocardial lactate extraction. Heart. 2006; 92: 1852-1853.

24) Cho DK, Choi JO, Kim SH, et al. Ischemia-modified albumin is a highly sensitive serum marker of transient myocardial ischemia induced by coronary vasospasm. Coron Artery Dis. 2007; 18: 83-87.

25) Roy D, Kaski JC. High-risk acute coronary syndrome patients and cardiac biomarkers in the emergency department: any role for new biomarkers of myocardial ischaemia? Eur Heart J. 2007; 28: 2297.

26) Shave R, George K, Gaze D. The influence of exercise upon cardiac biomarkers: a practical guide for clinicians and scientists. Curr Med Chem. 2007; 14: 1427-1436.

27) Apple FS, Quist HE, Otto AP, Mathews WE, Murakami MM. Release characteristics of cardiac biomarkers and ischemiamodified albumin as measured by the albumin cobalt-binding test after a marathon race. Clin Chem. 2002; 48: 1097-1100.

28) Middleton N, Shave R, George K, et al. Novel application of flow propagation velocity and ischaemia-modified albumin in analysis of postexercise cardiac function in man. Exp Physiol. 2006;91:511-519.

29) Falkensammer J, Stojakovic T, Huber K, et al. Serum levels of ischemia-modified albumin in healthy volunteers after exercise-induced calf-muscle ischemia. Clin Chem Lab Med. 2007; 45: 535-540.

30) Roy D, Quiles J, Sharma R, et al. Ischemia-modified albumin concentrations in patients with peripheral vascular disease and exercise-induced skeletal muscle ischemia. Clinical Chemistry. 2004; 50: 1656-1660.

31) Van der Zee PM, Verberne HJ, Straalen JP, et al. Ischemiamodified albumin measurements in symptom-limited exercise myocardial perfusion scintigraphy reflect serum albumin concentrations but not myocardial ischemia. Clin Chem. 2005; 51: 1744-1746.

32) Sbarouni E, Georgiadou P, Theodorakis GN, Kremastinos DT. Ischemia-modified albumin in relation to exercise stress testing. J Am Coll Cardiol. 2006; 48: 2482-2484.

33)Kurz K, Voelker R, Zdunek D, et al. Effect of stress-induced reversible ischemia on serum concentrations of ischemia-modified albumin, natriuretic peptides and placental growth factor. Clin Res Cardiol. 2007; 96: 152-159.

34) Rao SP, Miller S, Rosenbaum R, Lakier JB. Cardiac troponin I and cardiac enzymes after electrophysiologic studies, ablations, and defibrillator implantations. Am J Cardiol. 1999; 84: 470.

35) Sbarouni E, Georgiadou P, Panagiotakos D, Livanis EG, Theodorakis GN, Kremastinos DT. Ischaemia modified albumin in radiofrequency catheter ablation. Europace. 2007; 9: 127- 129.

36) Roy D, Quiles J, Sinha M, et al. Effect of radiofrequency catheter ablation on the biochemical marker ischemia-modified albumin. Am J Cardiol. 2004; 94: 234-236.

37) Roy D, Quiles J, Sinha M, et al. Effect of direct-current cardioversion on ischemia-modified albumin in patients with atrial fibrillation. Am J Cardiol. 2004; 93: 366-368.

38) Sbarouni E, Georgiadou P, Panagiotakos D, Livanis E, Theodorakis GN, Kremastinos DT. The ischemia-modified albumin in relation to pacemaker and defibrillator implantation. Pacing Clin Electrophysiol. 2008; 31: 83-87.

39) Turedi S, Gunduz A, Mentese A, et al. Value of ischemiamodified albumin in the diagnosis of pulmonary embolism. Am J Emerg Med. 2007; 25: 770-773.

40)Bar-Or D, Lau E, Winkler JV. A novel assay for cobalt-albumin binding and its

potential as a marker for myocardial ischaemia—a preliminary report.
J Emerg Med 2000;19:311–15.

41)Chawla R., Navendu Goyal, *Rajneesh Calton and Shweta Goyal et al, Ischemia modified albumin : A Novel marker for Acute Coronary Syndrome. Indian Journal of Clinical Biochemistry, 2006, 21 (1) 77-82

42) Worster A, Devereaux PJ, Heels-Ansdell D, et al. Capability of ischemia-modified albumin to predict serious cardiac outcomes in the short term among patients with potential acute coronary syndrome. CMAJ. 2005; 172: 1685-1690.

43)Dusek J, Stásek J, Tichy´ M, et al. Prognostic significance of ischemia modified albumin after percutaneous coronary intervention. Clin Chim Acta. 2006; 367: 77-80.

44) Dominguez-Rodriguez A, Abreu-Gonzalez P, Garcia-Gonzalez MJ, Samimi-Fard S, Kaski JC. Relation of ischemia-modified albumin levels and left ventricular systolic function in patients with ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention. Clinica Chimica Acta. 2008 Feb 1;388(1-2):196-9.

45) Aparci M, Kardesoglu E, Ozmen N, Ozcan Ö, Cebeci BS, Cingozbay BY, Dincturk M. Prognostic significance of ischemia-modified albumin in patients with acute coronary syndrome. Coronary artery disease. 2007 Aug 1;18(5):367-73.

Alcholism And Its Ill Effects

Anandadeeban K B S[1], Balakrishna R.N[2]

Graduate Student [1], Senior Lecturer [2], Department of Oral and Maxillo Facial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Alcohol is the ingredient found in beer, wine and spirits which causes drunkenness. It is formed when yeast ferments the sugars in different foods. For example, wine is made from the sugar in grapes, beer from the sugar in malted barley, cider from the sugar in apples, and vodka from the sugar in potatoes, beets or other plants. This paper provides a brief overview of the health and body effects of alcohol. Various areas covered include the effects of alcohol on body parts, health effects of acute alcohol use, and health conditions related to chronic alcohol use. The aim of this review is to know about alcoholism and its ill effects.

Key Words : *alcoholism,withdrawal,cirrohosis of liver*

Introduction

Alcohol is classed as a sedative hypnotic drug [1, 2] which meansit acts to depress the central nervous system at high doses. At lower doses, alcohol can act as a stimulant inducing feelings of euphoria and talkativeness, but drinking too much alcohol at one session can lead to drowsiness, respiratory depression, coma or even death[3].If alcohol consumption is acute and potentially lethal sedative effect at larger doses, alcohol has influence on all organ in the body, and these effects depend on the alcohol blood concentration.[4]. In a medical context, alcoholism is said to exist when two or more of the following conditions is present: a person drinks large amounts over a long time period, find difficulty in controlling, acquiring and drinking alcohol will take up a more time.Alcohol is strongly desired, usage results in not fulfilling responsibilities, usage results in social problems, usage results in health problems, usage results in risky situations, withdrawal occurs when stopping, and alcohol tolerance has occurred with use.[5] Risky situations include drunken drive or having unprotected sex with multiple partners, among other things.After a drink is swallowed, the alcohol is rapidly absorbed into the blood: 20 % through thestomach and 80 % through the small intestine), with effects felt within 5 to 10 minutes after drinking. It usually peaks in the blood after 30 to 90 minutes,and thus is carried through all the organs of the body [6, 7].Liver performs 90 % of the metabolismor breakingdown, of alcohol from a toxic substance to water and carbon dioxide,and therest excreted through the lungs, through the kidneys and via sweat.Only a certain amount of alcohol can be breakdown per hour, in which for an average personis around one standard drink which raises the blood alcohol concentration about 15 to 20 mg/dL [8]. Alcohol use can affect all parts of the body, but it particularly affects the brain, heart, liver, pancreas, and immune system. This can result in mental illness, Wernicke–Korsakoff syndrome, an abnormal heartbeat, liver cirrhosis, and an increase in the risk of carcinoma, among other diseases.Drinking during pregnancy can cause damage to the baby resulting in fetal alcohol spectrum disorders. Women are generally more sensitive than men to the harmful physical and mental effects of alcohol.[9]

Reasons for alcoholism

Many factors can increase the risk of alcohol abuse. People may become alcoholic forseveralreason and gradually develop a dependency on drinking. For example, consuming

alcohol during difficult times like death in the family or job loss can potentially trigger long-term alcohol abuse.

Relieve stress

Relying on alcohol to reduce stress that occur everyday,may impact the likelihood of developing alcoholism. Since alcohol is a depressant and a sedative, drinking produces feelings of pleasure. However, frequent drinking builds tolerance, requiring you to consume more alcohol in order to achieve the same effects.

Feel good

Consuming alcohol can provide some people a break from reality. It offers a sense of relief from underlying issues your mind may be trying to escape from. However, continual alcohol use to get through the day or week can turn into a serious drinking problem [10].

Cope with loss

Losing a family member or friend can take a toll on you emotionally, physically and mentally. Alcohol can ease the feeling that people hav and is used to get through the tough times. Depending on alcohol, even temporarily, can spiral into a drinking problem.

Overcome anxiety

Fewindividuals are naturally anxious, causing them to worry. Drinking lowers an individual's inhibitions and makes them more comfortable in social situations. Over time though, this can lead to addictive behaviors [11].

Body effects of alcohol

Concentration of blood rises and the feeling of drunkenness occurs, when alcohol is drunk faster than the liver can break it down. The higher the blood alcohol concentration, the greater the effects on the body.

However, blood alcohol concentration won't relate exactly with symptoms of drunkenness and various people have different symptoms even after drinking the same amount of alcohol.

The blood alcohol concentration level, and every individual's reaction to alcohol, is influenced by [12]:

• The ability of the liver to metabolise alcohol

• The presence or absence of food in the stomach

• The concentration of alcohol in the beverage

• How quickly alcohol is drunk

•Age, sex, ethnicity, body type

•How frequently a person drinks alcohol

Overall body effects

Alcohol intake can affects all body parts which includes:

• Blood and immune system
• Bones and muscles
• Brain and nervous system
• Breasts in women
• Eyes
• Heart and blood pressure
• Intestines
• Kidneys and fluid balance
• Liver
• Lungs
• Mental health
• Mouth and throat
• Pancreas and digestion of sugar
• Sexual and reproductive system in men
• Sexual and reproductive system in women
• Skin and fat
• Stomach and food pipe

Health conditions related to chronic alcohol use

For some people, alcohol is a regular or occasional drink enjoyed at social occasions that causes no apparent harm. However, even moderate alcohol use carries some risks, as alcohol causes breast cancer even at low doses, can damage the developing fetus before a woman even knows she is pregnant and can lead to addiction and dependence in any individual. When drunk regularly over time and/or drunk in a pattern of heavy single drinking sessions, alcohol can cause a variety of health conditions. These include carcinoma and other disease which include as alcoholic liver disease, which can present from reversible to permanent liver damage due to alcohol. The risks of alcohol-related cancers and other health conditions caused by alcohol are greatest in those who are dependent on alcohol or drink heavily, and the risks increase with the average amount of alcohol drunk [12].

The Harmful Effects of Alcohol

Harms Associated With Alcohol

Alcohol abuse is a major factor in death, disease, accidents and crime in Australia. The problems associated with alcohol use generally fall into two areas:

- Short-term harm due to intoxication called as binge drinking
- Long-term harm due to alcohol dependence

Binge Drinking

Binge drinking occurs when a person drinks heavily over a short period of time resulting in immediate and severe intoxication. Binge drinking is sometimes defined as ‘drinking to get drunk’. The health risks associated with binge drinking include the potential to develop toxic damage to the small bowel which causes diarrhoea, depression of the central nervous system, hangovers, headaches, and stomach problems resulting in nausea, shakiness and vomiting. Importantly, because intoxication stops one thinking clearly and acting sensibly, binge drinking can also lead a person to put themselves and others at risk of harm from other things. For instance, injury due to falls, risky behaviour or assault. It is for this reason that alcohol is closely associated with road accidents, fights and violence, coercive sexual activity and unprotected sex.[13]

Alcohol Poisoning

Serious binge drinking can lead a person to suffer alcohol poisoning. This occurs when the blood alcohol level rises to a dangerous point. At very high blood alcohol levels, a person may fit, lose consciousness and slip into a coma. There have been cases when the person intoxicated dies. Death from alcohol poisoning usually occurs in one of three ways:

• The blood alcohol level reaches such a high level that the depressant effects of the drug slow down the parts of the brain and nervous system that control breathing and the heart. Usually the drinker dies because they have stopped breathing and their heart has stopped, usually while unconscious [14]

• While unconscious, the drinker has been sick and choked on their own vomit. There are also rare reports of an unconscious drinker choking on their own tongue.

• The alcohol reacts with another drug that the person has taken. This can be either a prescription drug, over the counter medication or an illicit substance. These deaths are even more unpredictable as they can happen at a relatively low blood alcohol level. It is also important to be aware that an intoxicated person can also die of exposure in comparatively warm temperatures. Alcohol affects the body's thermostat, as well as the drinker's perception as to what is hot or cold, therefore someone who has been drinking can feel quitewarm when in fact their body temperature is dropping sharply[15]

Alcohol Dependence

While drinking a small amount of alcohol is generally not harmful for most people, regular drinking of a lot of alcohol can cause health, personal and social problems for a person over the long-term.People who drink heavily may become dependent on alcohol.[16] There are degrees of dependence, from mild dependency to compulsive drinking often referred to as 'alcoholism'. Alcohol dependence can be a physical problem, or psychological, or both.

For instance, to some degree many of us are psychologically dependent on alcohol if we feel that we cannot socialise at a party without a drink. In the case of an alcoholic person who is both physically and psychologically dependent, alcohol becomes central to their life. Such a person would suffer withdrawal symptoms such as, tremor, nausea, anxiety, depression, sweating, headache and difficulty sleeping, if they were to try to stop drinking or to cut down the amount they drink.

Alcohol Effect on Saliva

Saliva is a aqueous fluid containing organic and inorganic products secreted by salivary gland.Saliva is helpful in mastication, speech, deglutition, lubrication and mucosal protection.Decreased salivary flow rate may lead to dental caries, oral pain and fungal infection.Alcohol diffuses rapidly into saliva during the drinking. Then salivary concentration becomes temporarily higher than in plasma. Within half an hour, salivary alcohol concentration equilibrates with the alcohol level in plasma. This suggests that alcohol can easily penetrate the whole body, including salivary glands. The alcohol metabolizes to aldehyde. After alcohol intake, acetaldehyde level in saliva exceeds the level in blood. Damage to the oral tissues is possibly due to the action of acetaldehyde [17].

Alcohol Effects on Local Anaesthetics

Alcohol affecting the efficacy of local anesthetic depends of the dosage and quantity. Alcohol ingestion use to relieve pain has proven to be relevant but as the body develop tolerance and addiction towards the alcohol, patient have to increase the dosage and quantity to achieve analgesic property. To achieve analgesic property, orally administered ethyl alcohol (1ml/kg of 100% ethyl alcohol + 1ml/kg tonic water) produced tolerance to pain comparable to 0.17mg/kg s.q. morphine. 10.[18]

Alcohol and Its Effects

Alcohol is absorbed directly into the bloodstream through the stomach and the small intestine. Food in the stomach slows down the rate at which alcohol is absorbed, but does not prevent intoxication or drunkenness. All alcohol consumed will reach the bloodstream, regardless of how much food is in the stomach. Alcohol is distributed throughout the water in the body, but not into fatty tissue .

The liver breaks down about 91 per cent of alcohol, and a small amount leaves the body in urine, sweat and the breath. The liver can only work at a fixed rate, getting rid of about three-quarters of a standard drink an hour. Sobering up takes time, and cold showers, exercise, black coffee, fresh air or vomiting will not speed up the process. Someone who drinks a lot at night may still have a high concentration of alcohol in their bloodstream the following day .

Immediate Effects

After a few drinks, the person may feel more relaxed, have reduced concentration and slower reflexes. After a few more drinks, they may have fewer inhibitions, more confidence, reduced coordination, slurred speech and intense moods.

If the person continues to drink, they may experience confusion, blurred vision and poor muscle control. Continuing to drink may result in nausea, vomiting and sleep. Consuming more alcohol could possibly result in coma or death.

Long Term Effects

Heavy consumption of alcohol over a long period of time can cause damage to many parts of the body. Impairment of brain and liver functions can be permanent. If the person's diet is also poor, this can further affect their health.

Emotional difficulties, such as depression and relationship problems, are also likely.Other possible long-term effects include:

- Cancer Of The Mouth, Throat, Oesophagus, Lips, Liver
- Brain Injury, Loss Of Memory, Confusion, Hallucinations
- High Blood Pressure Enlarged Heart And Changes In Rbc.
- Weakness And Loss Of Muscle Tissue
- Sweating, Flushing And Bruising Of The Skin
- Inflamed Stomach Lining, Bleeding And Stomach Ulcers
- Increased Risk Of Lung Infections
- Severe Swelling Of The Liver, Hepatitis And Cirrhosis
- Inflamed Pancreas
- Tingling And Loss Of Sensation In Hands And Feet
- For Men, Impotence, Shrinking Of Testicles And Reduced Sperm
- For Women, Greater Risk Of Gynaecological problems

Central Nervous System

One of the easiest ways to understand alcohol's impact on your body is by understanding how it affects your central nervous system. Slurred speech is one of the first signs you've had too much to drink. Alcohol can reduce communication between your brain and your body. This makes coordination more difficult.[18]. You may have a hard time balancing. You should never drive after drinking.As alcohol causes more damage to your central nervous system, you may experience numbness and tingling sensations in your feet and hands.Drinking also makes it difficult for your brain to create long-term memories. It also reduces your ability to think clearly and make rational choices. Over time, frontal lobe damage can occur. This area of the brain is responsible for emotional control, short-term memory, and judgement, in addition to other vital roles.Chronic and severe alcohol abuse can also cause permanent brain damage. This can lead to Wernicke-Korsakoff syndrome, a brain disorder that affects memory.

Sexual and Reproductive Health

You may think drinking alcohol can lower your inhibitions and help you have more fun in bed. But the reality is quite different. Men who drink too much are more likely to experience erectile dysfunction [19]. Heavy drinking can also prevent sex hormone production and lower your libido.

Women who drink too much may stop menstruating. That puts them at a greater risk for infertility. Women who drink heavily during pregnancy have a higher risk of premature delivery, miscarriage, or stillbirth.Women who drink alcohol while pregnant put their unborn child at risk. Fetal alcohol syndrome disorders (FASD) is a serious concern. Other conditions include:

- Learning Difficulties
- Long-Term Health Issues
- Increased Emotional Problems
- Physical Development Abnormalities

Skeletal And Muscle Systems

Long-term alcohol use may prevent your body from keeping your bones strong. This habit may cause thinner bones and increase your risk for fractures if you fall[19]. And factures may heal more slowly.Drinking alcohol may also lead to muscle weakness, cramping, and eventually atrophy.

Immune System

Drinking heavily reduces your body's natural immune system. This makes it more difficult for your body to fight off invading germs and viruses.People who drink heavily over a long period of time are also more likely to develop pneumonia or tuberculosis than the general population. About 10 percent of all tuberculosis cases worldwide can be tied to alcohol consumption. Drinking alcohol also increases your risk for several types of cancer, including mouth, breast, and colon[20]. Click here to learn the basics of alcoholism. You can also read about the stages of alcoholism and recognizing an addiction.

Tolerance and Dependence

People who drink heavily usually develop a tolerance to alcohol. This means that they need to drink more to experience the same effect. As a result, some people can drink large amounts of alcohol without appearing to be intoxicated. However, the amount of alcohol consumed can still damage their health.

People who regularly drink heavily may become dependent on alcohol. Dependence can be psychological or physical, or both [21]. People who are psychologically dependent on alcohol find that drinking becomes far more important than other activities in their life. People who are physically dependent upon alcohol find that their body is used to functioning with alcohol present.

Alcoholism and Family

The effects of alcohol abuse can have an impact on not only the individual who drinks, but also on the entire family unit. Someone who abuses alcohol has a higher risk of divorce and a

higher risk of being involved in domestic violence. Even when a couple stays together, alcoholism puts a strain on the marriage relationship [22]. The spouse of an alcoholic may become co-dependents and start to cover up for the alcoholic. He or she may make excuses for the alcoholic spouse's behaviour, clean up messes left by the alcoholic, lie for the alcoholic, or take on added responsibility at work or home. By covering for the other person, the co-dependent spouse enables the alcoholic to escape the consequences of his or her alcoholism and remain in denial.

When a parent is an alcoholic, it can affect the children as well. Parental alcoholism effects include a higher risk of child abuse or neglect, a higher risk of later drug or alcohol abuse by the child, and long-lasting emotional trauma. The children of alcoholics may also be more prone to developing mental illness later in life.

Withdrawal

If a person who is physically dependent on alcohol suddenly stops drinking, they will experience withdrawal symptoms because their body has to readjust to functioning without alcohol. Alcohol withdrawal symptoms include:

Loss of appetite

- Nausea
- Anxiety
- Insomnia
- Irritability
- Confusion
- Tremors
- Sweating

In severe cases, alcohol withdrawal may cause convulsions, cramps, vomiting, delusions, hallucinations and even death [21,22] A person considering withdrawing from alcohol should first consult a doctor or other health professional.

Conclusion

There are a number of drug treatment options available. While abstinence may be a suitable treatment aim for some people, many programs recognise that for others this may not be possible or realistic. Most programs adopt strategies that have an overall aim of reducing the harms and risks related to the person's alcohol use. Some treatment options include counselling, withdrawal, detoxification and medication, pharmacotherapy. Residential and "out-patient" programs are available[23]. Treatment is more effective if tailored to suit a person's circumstances, and usually involves a combination of methods.

Acknowledgement

The author expresses his thanks and gratitude to Saveetha Dental College, Chennai for granting permission to publish this review paper.

Corresponding author

Balakrishna R.N

Senior Lecturer, Department of Oral and Maxillo Facial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, PoonamalleHigh Road, Chennai 600077, Tamilnadu, India

Telephone number :8939896729

Email id- balakrishdr8394@gmail.com

References

1. Deepthi.K,Lailtha K.B.,A case control study on effects of alcoholism, Int J Pharm Bio Sci 2015 Oct; 6(4): (B) 188 – 192.

2. Looi Qiao Xin, BDS, Dr. Dinesh Kumar, Efficacy of Local Anesthetics on Alcoholics. International Journal of Advanced Research (2014), Volume 2, Issue 2, 44-47

3. Roehrs T, Roth T. Sleep, sleepiness, and alcohol use. Alcohol research and Health. 2001 Jan 1; 25(2):101-9.

4. Jellinek EM. The disease concept of alcoholism.

5. Vonghia L, Leggio L, Ferrulli A, Bertini M, Gasbarrini G, Addolorato G. Acute alcohol intoxication. European Journal of Internal Medicine. 2008 Dec 1;19(8):561-7.

6. Lohr R. Acute alcohol intoxication and alcohol withdrawal.Hospital medicine. 2005:1243-50.

7. Zakhari S. Overview: how is alcohol metabolized by the body?.Alcohol Research & Health. 2006 Dec 22;29(4):245-55.

8. Schuckit MA. Alcohol-related disorders.InSadockVA Comprehensive Textbook of Psychiatry. 2000 2005.

9. Rehm J, Baliunas D, Borges GL, Graham K, Irving H, Kehoe T, Parry CD, Patra J, Popova S, Poznyak V, Roerecke M. The relation between different dimensions of alcohol consumption and burden of disease: an overview. addiction. 2010 May 1;105(5):817-43.

10. Klatsky AL, Friedman GD, Siegelaub AB, Gérard MJ. Alcohol consumption and blood pressure: Kaiser-Permanente multiphasic health examination data. New England Journal of Medicine. 1977 May 26; 296(21):1194-200.

11 Callahan CM, Tierney WM. Health services use and mortality among older primary care patients with alcoholism. Journal of the American Geriatrics Society. 1995 Dec 1;43(12):1378-83.

12. Mallick J. Drugs and driving in Australia: A survey of community attitudes, experience and understanding. Australian Drug Foundation; 2007.

13. Room R, Babor T, Rehm J. Alcohol and public health. The lancet. 2005 Feb 5;365(9458):519-30.

14. Kuhn C, Swartzwelder S, Wilson W, Wilson LH, Foster J. Buzzed: The straight facts about the most used and abused drugs from alcohol to ecstasy. WW Norton & Company; 2008.

15. Degenhardt L, Chiu WT, Sampson N, Kessler RC, Anthony JC, Angermeyer M, Bruffaerts R, De Girolamo G, Gureje O, Huang Y, Karam A. Toward a global view of

alcohol, tobacco, cannabis, and cocaine use: findings from the WHO World Mental Health Surveys. PLoS medicine. 2008 Jul 1;5(7):e141.

16. Vaillant GE. The natural history of alcoholism. Cambridge, Mass.: Harvard University Press; 1983.

17. Holder HD. Alcoholism treatment and potential health care cost saving. Medical care.1987 Jan 1:52-71.

18. Beauchamp DE. Beyond alcoholism: Alcohol and public health policy.

19. Holder HD, Blose JO. Alcoholism treatment and total health care utilization and costs: A four-year longitudinal analysis of federal employees. Jama. 1986 Sep 19;256(11):1456-60.

20. Holder HD, Hallan JB. Impact of alcoholism treatment on total health care costs: A six-year study. Advances in alcohol & substance abuse. 1986 Sep 1;6(1):1-5.

21. Magruder-Habib K, Durand AM, Frey KA. Alcohol abuse and alcoholism in primary health care settings. Journal of Family Practice. 1991 Apr 1;32(4):406-14.

22. Magruder-Habib K, Durand AM, Frey KA. Alcohol abuse and alcoholism in primary health care settings. Journal of Family Practice. 1991 Apr 1;32(4):406-14.

All Ceramic Materials – An Update

Maria Kurian[1] , Dhanraj Ganapathy[2] , Sreedevi Dharman [3]

Graduate Student [1], Professor and Head [2],Department of Prosthodontics,

Senior Lecturer [3],Department of Oral Medicine

. Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Dental ceramics are a network of materials which helps in the easy understanding of their advances, composition build up and its various applications in dentistry. Metal ceramic restorations have been in use for about 3 decades now. This kind of restoration is being increasingly used due to its astonishing performance and decent esthetics. Due to the increased demand of better aesthetics and concern about the biocompatibility of metal, all ceramic material restorations were introduced.In the recent years, there have been immense developments in the properties and the manufacturing methods of the all ceramic materials. Steps are being taken to use more of all ceramic restorations when compared to the use of metal ceramic restorations. Research is being done in order to improve the properties of all ceramic materials which will provide better aesthetics and function of the restorations. There are various alloys of ceramics that have been developed. There have also been advances in the manufacturing methods of the ceramic materials. Various cements are used for specific types of ceramics which help in the longevity of the restorations. The aim of this review is to understand the various advances in the vast field of dental ceramic materials.

Key Words*: All Ceramics, Zirconium, Microstructure, Strength, Aesthetics.*

Introduction

Ceramic materials are best able to mimic the appearance of natural teeth. However, two obstacles have limited the use of ceramics in the fabrication of dental prostheses: 1) brittleness leading to a lack of mechanical reliability and 2) greater effort and time required for processing in comparison to metal alloys and dental composites. There have been developments in the processing methods of ceramic materials which has simplified the work of dental lab technicians and improved the quality and properties of the material. Due to this, there is an increase in the use of all-ceramic prostheses and restorations. Core materials are the basis of all-ceramic dental restorations, and a variety of them have been seen to be used for the same. Zirconia blocks are being provided by manufacturers which are then being converted into crowns and bridges using the CAD/CAM system. Zirconia is seen to have better mechanical properties when compared tometals;this is due to its microstructure.[1] The properties of conventional zirconia were further improved by the composite in nano-scale such as zirconia/alumina nanocomposite. There are various reactions of zirconia seen such as in low temperatures, it shows long-term stability, and it's bonding to porcelains and to cement, X-ray opacity and biocompatibility. Engineers generally develop the new material which is later put to practical use in dentistry. A material such developed is dental ceramics. In dentistry, ceramics are considered as nonmetallic, inorganic structures composed of oxygen with metallic or semi-metallic compounds. They are usually sodium, potassium, calcium, magnesium, aluminum, silicon, phosphorus, zirconium & titanium.[2] Dental ceramics are materials that are part of systems designed with the purpose of producing dental prostheses that in turn are used to replace missing or damaged dental structures.[3] Metal ceramic systems are those in which ceramic provides the aesthetics and metal provides the necessary strength due to its mechanical properties.[4] Some metals used as restorative materials in dentistry may constitute a problem for some patients. These problems may reveal themselves as allergies,[5] gum staining[6,7] and release of metallic ions into the gingival tissue[8] and the gingival fluid.[9] These drawbacks, as well as the search for more esthetic materials by patients and dentists, have stimulated research and development of metal-free ceramic systems. The aim of this review is to understand the various advances in the vast field of dental ceramic materials.

Classification of Dental Ceramics

Dental ceramics can be classified in many different ways according to their: [10]

(a) Indication or use.

(b) Composition.

(c) Processing method.

(d) Firing temperature.

(e) Microstructure.

(f) Translucency.

(g) Fracture resistance.

(h) Abrasiveness.

Microstructural Classification

At the microstructural level, we can define ceramics by thenature of their composition of glass-to-crystalline ratio.The microstructures ofmaterials can come in various variability, but they are grossly divided into four basiccompositional categories, each having a few subgroups under them:

• Composition category 1 – glass-based systems

• Composition category 2 – glass-based systems with fillers, usually crystalline

• Composition category 3 – crystalline- based systemswith glass fillers and

• Composition category 4 – polycrystalline solids

Classification Based on Processing Technique

In general, for dentistry, they can be classified as:

a. Powder/liquid, glass-based systems,

b. Machinable or press able blocks of glass-based systemsand

c. CAD/CAM or slurry, die-processed, mostly crystalline(alumina or zirconia) systems.

1. Powder Condensation

In this technique, moist porcelain is applied using a special brush. The porcelain is then compressed by removing the excess moisture. This is then placed in a vacuum which causes further compression of the porcelain. Powder condensation uses feldspathic porcelain.

Feldspathic porcelain: It is a mixture in which potassium and sodium feldspars occur naturally. These are composed of potash (K2O) and soda (Na2O) respectively. Alumina (Al2O) and soda (Na2O) are also seen in trace amounts. Leucite and a glass phase are formed when potassium feldspar is fired to high temperatures. This glass phase softens during firing allowing coalescence of the porcelain powder particles. This process is called liquid phase sintering. This process forms a dense solidwhen kept in a high temperature. Since leucite has a large coefficient of thermal expansion, it is added to some glasses to control their thermal expansion.[11]This method results in a large amount of residual porosity. The crystalline particles that strengthen the material on a microscopic scale are not connected to each other but are separated by glassy regions. The porosity and discontinuous nature of the crystalline phase lead to relatively low strength and a wide variation in strength. Ceramics fabricated by powder condensation have greater translucency than can be achieved using other methods [12], so these materials are usually applied as the aesthetic veneer layers on stronger cores and frameworks.

2. Slip Casting

A slip is a low viscosity slurry or mixture of ceramic powder particles suspended in a fluid which is generally water. Slip casting involves forming a mold or negative replica of the desired framework geometry and pouring a slip into the mould. The mold is made of a material generally gypsum that extracts some water from the slip into the walls of the mold through capillary action, and some of the powder particles in the slip become compacted against the walls of the mold forming a thin layer of green ceramic that is to become the framework. The remaining slip is discarded, and the framework can be removed from the mold after partial sintering to improve the strength to a point where the framework can support its own weight. The resulting ceramic is very porous and must be either infiltrated with molten glass or fully sintered before veneering porcelain can be applied. Ceramics which are fabricated by slip casting is seen to strengthen the crystalline particles which forms a network thereby creating a higher fracture resistance. Use of this method in dentistry has been limited to one series of three products for glass infiltration (In-Ceram, Vita Zahnfabrik). The limited application of slip casting in dentistry is probably because the method requires a complicated series of steps, which provide a challenge to achieving accurate fit and may result in internal defects that weaken the material from incomplete glass infiltration [13].

3. Hot Pressing

Molds for pressable dental ceramics are formed utilizing the lost wax technique. Glass-ceramic ingots are used as pressable ceramics. The composition of the ingots is similar to that of the powder porcelains, the only difference being that they show less porosity with more crystalline content. The ingots are heated to a high temperature where they become a highly viscous liquid, and then pressed slowly into the formed mold. The advantage of this technique is that it utilizes the experience that the lab technician already has in lost wax method with metal alloys. Pressable ceramics are available from manufacturers as prefabricated ingots made of crystalline particles distributed throughout a glassy material. The microstructure is similar that of powder porcelains, however, pressable ceramics do not contain much porosity and can have a higher crystalline content because the ingots are manufactured from non-porous glass ingots by applying a heat treatment that transforms some of the glass into crystals. This process can be expected to produce a well controlled and homogeneous material. In the dental laboratory, the pressable ingots are heated to a temperature at which they become a highly viscous liquid, and they are slowly pressed into the lost wax mold. The advantage of hot pressing is that dental technicians are already experienced at achieving good accuracy of fit using the lost wax method with metal alloys [14]. Contrary to intuition, the higher crystalline content and lack of porosity do not lead to increased fracture resistance or decreased variability of strength [15]. Pressable ceramics usually have application only as core and framework materials. Pressable veneering materials, such as IPS e.max ZirPress (Ivoclar-Vivadent) are available, but the depth of layered esthetics may be limited when using pressable ceramics for veneering materials.

4. Computer-Aided Design

Like pressable ceramics, CAD-CAM ceramics are available as prefabricated ingots. These ingots are milled or cut by computer-controlled tools. After the tooth preparation is completed, an optical impression is taken by the use of an optic scanner.[16] The image is then transferred to the system’s software. Then the software designs the restoration and sends the data to the computer controlled milling machine that grinds the ceramic block according to the desired shape. In the case of presintered ceramics, the ingots are porous, which enables fast milling

without bulk fracture of the ceramic. The disadvantage of presintered ingots is the need for subsequent sintering treatment to eliminate the porosity. The computer software must compensate for the shrinkage that occurs during sintering to achieve good accuracy of fit. Densely sintered ceramics are available in non-porous ingots, which are more difficult to mill, but they do not require any further sintering. Glass infiltrated CAD-CAM ingots have similar composition to slip cast ceramics, but starting with a porous ingot eliminates the complicated steps of slip casting. After milling, the porosity is eliminated by molten glass infiltration. Dental CAD-CAM systems have been available for 20 years. In recent years, the increasing use of polycrystalline alumina and zirconia as framework materials and the increasing popularity and variety of CAD-CAM systems seem to be mutually accelerating trends.[17]

Properties

Strength and Fracture Toughness

There are two interrelated properties that often are quotedregarding ceramics intended for structural purposes:

a. Strength and

b. Fracture toughness

Strength

Mechanical failure of ceramic materials is almost completely controlled by brittle fracture. Usually, this brittle behavior combined with surface flaws resulted in relatively low ceramic strengths. Increased crystalline-filler content within the glass matrix, with a more even distribution of particles and finer particle size, has yielded significant improvements in the flexural strength of ceramic materials.[18,19] However, strength improvements are still limited by the inherentweakness of the glass matrix. All ceramics fail because of crack propagation at a critical strain of 0.1%.[20] Applied stresses can cause a crack to grow throughout the matrix, causing the ultimate failure of that restoration.

Fracture Toughness

A more important physical property is fracture toughness, which has been reported to be between 8 MPa m1/2 and 10 MPa m1/2 for zirconia. This is significantly higher than any previously

reported ceramic, and roughly twice the amountreported for the alumina materials. Fracture toughness is a measure of a material's ability to resist crack growth (i.e., a measure of the amount of energy necessary to cause crack growth). Clinically, restorations are not loaded to failure as is done in a flexural strength test; instead, millions of subcritical loads (chewing) are applied. Materials ultimately fail because of this cyclic fatigue by crack propagation.Thus, materials with higher fracture toughness are more ideal clinically as it takes more energy to cause crack growth. Other factors such as stress corrosion (chemically assisted crack growth) and residual flaws in the materialgreatly affect the final strength of a finished material.[21,22]

Contraindications of All Ceramic Restorations

1. Heavy occlusal forces: Ceramic restorations should be avoided in patients with parafunctional habits such as bruxism because of the brittle nature of the material and also its abrasive potential.
2. Inability to maintain a dry field: ceramic restorations require good moisture control at the time of their cementation to ensure positive outcomes.
3. Deep subgingival preparations: this is not considered an absolute contraindication, although supragingival preparations are desirable to produce a more accurate recording during impression taking. [23]

Aesthetic Considerations

Achieving a lifelike match requires the clinician/technician to choose a base ceramic having an appropriatetranslucency for thepatient. For a single anterior tooth, all ceramics havesufficient longevity based on clinical trial data, so thechoice can be made on aesthetic capability alone. Ceramic restorations give the teeth a natural tooth appearance.[24]Ceramic materials show a range of optical behaviors which changes from system to system, thus care must be taken when selecting which system to use.[25]Ceramic systems are capable of adjusting the levels of translucencyand control the colour. These are best learned byclinical practice.

Factors Contributing To Restorative Success

Restorative success depends on various factors and not just on the type of ceramic used.It is known that the tooth preparation for an all ceramic restoration must have rounded line angles

along with the finish line being a deep chamfer or a 90° rounded shoulder. If the restoration is going to be bonded, supragingival margins are preferred. Subgingival restorations cannot be done as it would be impossible to isolate these preparations. In such cases, the conventional cementation method is preferred. Sufficient occlusal reduction is required to be done in order to provide enough bulk to the ceramic restoration. Metal-ceramic restorations are preferred in cases where there is restricted interocclusal distance. Metal of the metal ceramic is seen to be placed in the lingual or occlusal surface. In addition to ensuring sufficient room for the foundation, it is also necessary to provide adequate resistance and retention form.Aesthetics can be increased when there is either a dark underlying tooth or metallic core. This is generally done in case of using alumina or zirconium. A feldspathic jacket, leucite-reinforced glass ceramic, or glass-infiltrated magnesium aluminum can be used in cases where high translucency of the restoration is required.All ceramic restorations can be provided to patients with parafunctional habits only after careful examination. If a patient insists on a metal-free restoration, the higher-strength core should be considered and occlusal guards used to prevent possible fractures. Attrition of the opposing tooth structure due to an opposing all-ceramic crown causes problems. The higher leucite-containing ceramics have a tendency for high abrasion potential.It is noted that anterior and posterior teeth require different materials as the amount of force during mastication is not the same for the two. Since the development of using cement to lute all-ceramic restorations has made indirect metal-free restorations can be done faster and more efficiently. Often, however, compromises must be made when clinicians and patients are faced with choosing between the ultimate aesthetics and function.

Effect on the Wearing of Opposing Dentition

In many individuals, tooth wear is a natural unavoidable process that is usually a result of tooth to tooth and/or tooth and restoration contact. Contact usually occurs when sliding movements are taking place. The process of wear essentially becomes accelerated by the introduction of restorations inside the oral cavity, and the rate of tooth surface loss is greater in case of opposing ceramic restorations. Wear of the entire dentition is possible without the presence of any restoration, and it should always be aimed for the hardness of restorative materials and their wear behaviour remains similar to that of natural enamel; otherwise, tooth surface loss may lead to a variety of clinical problems including damage to the opposing enamel structure, loss of occlusal

vertical dimension, problems in mastication, temporomandibular joint problems,hypersensitivity, and esthetic impairment.For many decades, ceramics have been used for esthetic restorations because of their excellent esthetic qualities and superior biocompatibility. However, ceramics are brittle in nature, require careful polishing techniques and are abrasive to the opposite dentition. There have been suggestions by authors that placement of ceramics over the occlusal surfaces should be avoided so that opposing dentition wear could be minimized. For that reason, modified ceramics have been developed recently in an attempt to decrease their wear characteristics.The newest materials have vastly contributed toward the interest in esthetic dental restorations. The effects of newly introduced dental ceramics have been extensively studied in laboratories. However, laboratory studies which test the abrasion resistance could produce results that are completely different for the same materials tested in clinical studies. In spite of the various technological advancements, there is no evidence on the clinical wear caused due to ceramics when it is restored opposing restored teeth and natural dentition.[26]

Cementation of All Ceramic Restoration

Resin-modified glass ionomer cements (RMGIs) or composite resins can be used for definitive cementation of all-ceramic restorations. The cements used can either be self-setting or sets by dual curing, where a light helps to set the cement at the margin of the restoration. It is recommended to use the adhesive technique with the light curing composite resin. Composite resin cements used today have a chemistry based upon our current generation of composite resins and bonding adhesives.[27] Resin-based cements include composite resin cements that are self-adhesive and self-setting or those that require an additional bonding adhesives used before the cement is placed. These cements can be light curing only, self-curing, or dual cured. The challenge to the chairside assistant for all-ceramic restorations is what steps are necessary both for pretreatment of the restoration and in preparation for cementation. Cements are available in different forms such as capsules which need to be triturated or they have to be mixed on a paper pad with a cement spatula. Most composite resin cements and some RMGI cements can be mixed by extruding from a double-barrel system into auto mixing tips. All the cements used for cementation has a consistency, viscosity and film thickness which is apt for cementing the all ceramic restorations. It should be noted that there is variability in the handling characteristics of each class of cement, and even differences within the same class of cement. It is critical that the

dentist and their chairside assistant read the instructions as it relates to material dispensing and mixing before it is used for cementing the restoration.[28] Cements are recommended so that they match the physical properties of the restorative material being used. It is important to follow the manufacturer's recommendations to achieve a clinically successful result.

Silicate Ceramics

Silicate ceramics have the property that when it is etched with hydrofluoric acid and bonded, the physical properties or porcelain is seen to be improved.[29] Silicate porcelains are very brittle in nature thus in function, they can develop small cracks which are known as microcracks. The use of adhesive composite resin cement strengthens the etched porcelain by interlocking into the microscopically roughened surface, not allowing the microcracks to propagate through the porcelain, which would create a fracture.Porcelain veneers are manufactured using from silicate ceramics. Silicate ceramics are found to be thinner and more translucent than oxide ceramics which helps in the easy etching and bonding to the tooth surface. Porcelain veneers is seen to be cemented best using light-cured composite resin cement. The composite resin cements come in packs that contain an etch-and-rinse adhesive which must be used first, followed by a porcelain primer and finally the light-cured cement in the shades that matched the ceramic must be used.Crowns which are fabricated from leucite-reinforced ceramics can be cemented using composite resin cements which may be in the light-cured, self-cured, or dual-cured type. Self-adhesive resin cements are typically not used. Lithium disilicate ceramics was seen to behigh-strength durable porcelain which soon overtook the use of leucite-reinforced ceramics. Lithium disilicate ceramic restorations can be cemented with any of the composite resin cements. Many lithium disilicate restorations are more slightly more opaque than leucite reinforced ceramics. Although veneers fabricated with lithium disilicate can be cemented with light-cured composite resin, it is not recommended that light-cured resin cement be used for crowns because of their greater thickness. Dual-cured, self-cured and self-adhesive resin cements can be used.

Oxide Ceramics

Oxide ceramics are fabricated with metal oxides. These ceramics are very resistant to fracture. Oxide ceramics are seen to be resistant to etching. Using specialized air abrasive aluminum oxide/silicate coated particles, sand blasting can be effective in making the surface more adhesive.[30] Cementation of oxide ceramics can be done with the same cements that are used for PFM restorations. Oxide ceramics which are also called as zirconia restorations, can be cemented with glass ionomer cements or composite resins. In the experience of the authors, the easiest cements to use with zirconia restorations are RMGIs and self-adhesive composite resin cements. Both these cements do not require etching or the use of a special bonding agent for the luting of the ceramic restorations. Some manufacturers provide a separate surface primer for the zirconia before using composite resin cement.

Repair

Approaches to the repair of porcelains have beenreviewed relatively recently.[15] Repair often offers bothdentist and patient a cost-effective alternative toreplacement. Repair involves the bonding of resinbasedproducts to remaining porcelain. The porcelainresinbond is formed by both etching the surface tocreate micromechanical attachment features and by theapplication of silane coupling agents to provide somechemical interaction between the silicon-based ceramicand carbon-based resins. It is reported that porcelainrepair systems form durable bonds to fractured porcelainand exposed metal surfaces.[31]

Conclusion

Ceramics have the property of showing the similar optical characteristics of enamel and dentine and thus are widely used in dentistry. Ceramics also has good biocompatibility andchemical durability. The new generation of ceramic materials presents interesting options, both in terms of material selection and in terms of fabrication techniques. A better understandingof the ceramic materials with respect to the restoration design and efficient use can enable these restorations to perform productively.

Corresponding Author

Sreedevi Dharman

Senior Lecturer,Department of Oral Medicine

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences. Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

E-mail: sanjamrut@gmail.com

Telephone Number: +919841009003

References

1. M Madhu Priya, Geo Mani; Advancement of Materials in Dentistry; Research & Reviews: Journal of Dental Sciences; 2017; 5(2):46-50.
2. Dr. Arunachalam Lakshmanan, Sintering of Ceramics, New Emerging Techniques, 2012, 203-25.
3. Arvind Shenoy, Nina Shenoy1, Dental ceramics: An update. J Conserv Dent 2010;13:195-203.
4. Arango SS, Vargas AP, Escobar JS, Monteiro FJ, Restrepo LF. Ceramicsfor dental restorations -An Introduction. 2010; 77:2636.
5. Stejskal VD, Danersund A, Lindvall A, Hudecek R, Nordman V, YaqobA. Metal specific lymphocytes: Biomarkers of sensitivity in man,Neuro Endocrinol Lett 1999;20:289-98.
6. Arvidson K, Wroblewski R. Migration of metallic ions from screwpostsinto dentin and surrounding tissues. Scand J Dent Res 1978; 86:200-5.
7. Venclíkova Z, Benada O, Bártova J, Joska L, Mrklas L. Metallicpigmentation of human teeth and gingiva: Morphological andimmunological aspects. Dent Mater J 2007; 26:96-104.
8. Bumgardner JD, Lucas LC. Cellular response to metallic ions releasedfrom nickel chromium dental alloys. J Dent Res 1995; 74:1521-7.

9. Mehulic K, Prlic A, Komar D, Prskalo K. The release of metal ionsin the gingival fluid of prosthodontic patients Acta Stomatol Croat2005; 39:47-51.
10. Philips' Science of Dental Materials, ed 11. Saunders,2003
11. Antonson SA, Anusavice KJ. Contrast ratio of veneering and core ceramics as a function of thickness. Int J Prosthodont. 2001; 14(4):316–320.
12. Donovan TE. Factors essential for successful all-ceramic restorations. J Am Dent Assoc 2008; 139 Suppl: 14S-18S.
13. Craig's Restorative Dental Materials, ed 12. Mosby, 2006
14. Sulaiman F, Chai J, Jameson LM, Wozniak WT. A comparison of the marginal fit of In-Ceram, IPS Empress, and Procera crowns. Int J Prosthodont. 1997 Sep–Oct; 10(5):478–484.
15. Tinschert J, Zwez D, Marx R, Anusavice KJ. Structural reliability of alumina-, feldspar-, leucite-, mica- and zirconia-based ceramics. J Dent. 2000 Sep;28(7):529–535.
16. Hemalatha Ranganathan, Dhanraj M Ganapathy, Ashish R Jain, Cervical and incisal marginal discrepancy in ceramic laminate veneering materials: A SEM analysis, Contemp Clin Dent. 2017; 8;272-8.
17. Luthardt RG, Sandkuhl O, Herold V, Walter MH. Accuracy of mechanical digitizing with a CAD/CAM system for fixed restorations. Int J Prosthodont 2001; 14(2):146-51.
18. Seghi RR, Daher T, Caputo A. Relative flexural strength of dentalrestorative ceramics. Dent Mater 1990; 6:181-4.
19. Seghi RR, Sorensen JA. Relative flexural strength of six new ceramicmaterials. Int J Prosthodont 1995;8:239-46.
20. Jones DW. The strength and strengthening mechanisms of dentalceramics. In: Mclean JW, editor Dental Ceramics: Procedings of the FirstInternational Symposium on Ceramics. Chicago: Quintessance; 1983.
21. Conrad HJ, Seong WJ, Pesun IJ. Current ceramic materials and systemswith clinical recommendations: A systematic review. J Prosthet Dent2007; 98:389-404.
22. Manicone PF, Rossi Iommetti P, Raffaelli L. An overview of zirconia ceramics: Basic properties and clinical applications. J Dent 2007; 35:819-26.
23. Sturdevant's Art & Science of Operative Dentistry, ed 4. Mosby 2002

24. Griggs JA. Recent advances in materials for all-ceramic restorations. Dent Clin North Am 2007; 51(3):713-27, viii.
25. Raptis NV, Michalakis KX, Hirayama H. Optical behavior of current ceramic systems. Int J Periodontics Restorative Dent 2006; 26(1):31-41.
26. Clelland NL, Agarwala V, Knobloch LA, Seghi RR. Relative wear of enamel opposing low-fusing dental porcelains. J Prosthodont 2003; 13:168-75.
27. Platt JA, Duke ES. Resin-based luting cements. Compend Contin Dent Educ. 2000; 21(1):40-44.
28. Strassler H, Suh BI, Wang Y. Compatibility of oxalate desensitizer with glass ionomer cements. J Dent Res. 2006; 85(special issue A): abstract 1885.
29. Friedman MJ. A 15-year review of porcelain failure--a clinician's observations. Compend Contin Educ Dent. 1996; 19(6):625-638.
30. Baldissara P, Querzè M, Monaco C, et al. Efficacy of surface treatments on the bond strength of resin cements to two brands of zirconia ceramic. J Adhes Dent. 2013; 15(3):259-267.
31. Latta M, Barkmeier WW. Approaches for intraoral repair ofceramic restorations. Compend Contin Educ Dent 2000; 21:635–639.

All CeramicSystem- A Review

Affrin. J.H [1] , Preetham Prasad Nitla [2], Sreedevi Dharman[3]
Graduate Student [1],Senior Lecturer [2],Department of Prosthodontics,
Senior Lecturer [3],Saveetha Dental College
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

This article describes the ceramic system and processing techniques available today in dentistry. It aims to help the clinician to understand advantages and disadvantages of ceramics. The properties ,classification, products gives us more idea about various options. The desire for beautiful teeth is a good reason to pay attention to the selection and quality of the restoration. In search for the ultimate aesthetics restorative materials, all ceramic system is the best choice among all the restorative materials. tremendous progress has been made in terms of mechanical performance, with a ten-fold increase in flexural strength and fracture toughness.

Key Words: *Ceramic, properties, classification, aesthetics, strength.*

Introduction

Replacement of a missing natural teeth was considered very important by mankind over all ages [1]. The earliest forms of materials used for replacement of missing teeth included wood, ivory, shells, etc.Over time, more attention was diverted towards replacement of the missing tooth that resembled the natural teeth. The first porcelain tooth material was documented by a Frenchdentist names De Chemant in 1789.Typically, they demonstrate excellent strength and hardness properties; however, they are often brittle in nature. Some ceramics like superconductors, also display magnetic properties.Ceramics basically consists of glasses, porcelain, glass ceramics or high crystalline structures. Ceramics are more resistant to corrosion than Plastics. Ceramics have gained their importance during the stone age more than 10,000 years ago, especially in human society.

The two main categories of ceramics are traditional and advanced. Traditional ceramics include objects made of clay and cements that have been hardened by heating at high temperatures. Traditional ceramics are used in dishes, crockery, flowerpots, and roof and wall tiles. Advanced ceramics include carbides, such as silicon carbide, SiC; oxides, such as aluminium oxide,nitrides, such as silicon nitride,and many other materials, including the mixed oxide ceramics that can act as superconductors. Advanced ceramics require modern processing techniques, and the development of these techniques has led to advances in medicine and engineering.

The entire thickness of the crown was 100% ceramic.All ceramic systems in dentistry have been developed for many decades.It provides maximum aesthetics to the restoration.Despite its success, the demand for improved aesthetics and the concerns regarding the biocompatibility of the metal has lead to all-ceramic restorations[2]. Dental ceramics are otherwise called as dental porcelain. They are non-metallic,inorganic structures primarily containing compound of oxygen with one or more metallic or more metallic or semi metallic elements[3]. What highlights the all ceramic system is their aesthetics and High durability. Ceramic are considered the best in mimicking the natural tooth appearance[4].The optical behaviour of ceramic materials differ from system to system and this should be taken into consideration during the selection of which system to be used[5].

Historically there were three types of ceramic materials - earthen wear,stone wear and porcelain. Earthenware is pottery that has not been fired to vitrification and is thus permeable to water[6].Stoneware is a vitreous or semi-vitreous ceramic made primarily from stoneware clay or non-refractory fire clay[7]. Stoneware is fired at high temperatures[8].It may or may not be glazed.[9]. It has high value of aesthetics. The first application was porcelain dentures. Interestingly porcelain is still quite widely used in dentistry.

At the beginning of the nineteenth century Charles Henry Land developed the porcelain jacket crown, based on a feldspathic composition, which is still used today in a slightly modified form. It had a very good aesthetics. Due to the low flexural strength of porcelain. It resulted in high incidence of failure. Vita Zahnfabrik developed the first commercial porcelain in 1963. Fifty years later, reinforcement of the jacket crown with aluminium oxide was achieved as a result of the work of McLean and Hughes [10].Dental ceramic shows a rapid growing field of interests as far as dental materials research is concerned. There is always demand existing for tooth coloured restoration. So future of ceramics appears very bright.

All ceramic systems are generally contraindicatedin situations such as: i) When there is a para-functional habit; ii) Inadequate support for the dental preparation; iii) It is not advisable as abutment bridge, unless it is placed on the anterior area and iv) When there is a pronounced overbite in the anterior area.The use of all ceramic is increasing day by day. But all ceramics systems are not all the same. They differs in the aesthetic potential. Despite its aesthetic and biocompatibility, there are failures existing due to their limited properties which are common to all ceramic crowns. The majority of all ceramic crowns are produced using computer technology, e.g. CAD/CAM technology which is based upon 3D design.

Classification

There are many types of dental ceramics which are available in dental laboratory which include core ceramic, Linuxceramic, marginceramic, opaque dentin ceramic, dentin ceramic, enamel ceramic, stain ceramic, glaze ceramic ceramic, and addition ceramic.

They are further classified according to –

- Use /indication

- Composition
- Processing method
- Firing temperature
- Microstructure
- Translucency
- Fracture resistance
- Abrasiveness

Based On Microstructure

I. Glass Based System With Silica

II. Glass based systemwith silica and fillers

a) low to moderate leucite containing feldspathic glass

b) high leucite

c) lithium disilicate

III. Crystalline based system with glass fillers.

IV. Polycrystalline solids.

Based On Processing Technique

- Powder/liquid glass based system
- Pressable blocks of glass based systems
- CAD/CAM systems

Based On Composition

- Silicate
- Oxide ceramics
- Glass ceramics

Based On Types

- Feldspathic Porcelain
- Aluminous Porcelain
- Reinforced Porcelain
- Glass Infiltrated Porcelain
- Glass Infiltrated Zirconia

- Glass Ceramic

Based On Temperature

- Ultra low fusing <850C
- Low fusing 850-1110C
- Medium fusing 1101C - 1300C
- High fusing >1300C

Based On Structure Metal

- Cast metal, glassceramic, sintered coated ceramic, and CAD-CAM.

Based On Reinforcement Method

- Reinforced ceramic core system, resin -bonded ceramics, metal ceramics.

Composition

Dental ceramics are usually made up of minerals called feldspar with added silica and fluxingoxides. Feldspar - K2oAl2O3SiO2,Albite-Na2O.Al2O3.6SiO2,Which is roughly consisting of 80% feldspar,15% silica ,4% clay. The main component is SiO2. There are minor components like Al2O2,MgO,ZiO2 and oxides. Ceramics are prone to fracture when they are exposed to tensile stress. Zirconia is known for its high fracture toughness.

Properties

For centuries, ceramics were used by those who had little knowledge of their structure. Today, understanding of the structure and properties of ceramics is making it possible to design and engineer new kinds of ceramics. Advanced ceramics are also used in the medicine, electrical, and aerospace industries. All ceramics are ideal aesthetics. They are alterable in time.Dental ceramics is a crystal phase and and glass phase based on the silica structure. It is not closely packed. It has both ionic and covalent characteristics. They have a glassy appearance. Silicate cements are first dental cements to use glass as its component. Dental ceramics have the ability to mimic the natural tooth in colour. Ceramics cannot undergo metal or chain realignment and they are not capable of changing their shape without fracturing unless their are heated to high temperature. They withstand chemical erosion that occurs in other materials subjected to acidic or caustic environments. Ceramics generally can

withstand very high temperatures, such as temperatures that range from 1,000 °C to 1,600 °C (1,800 °F to 3,000 °F).

They either have high melting or softening temperatures. They are brittle therefore they do not have tendency to undergo plastic deformation. They are basically hard and wear resistance. They do not conduct electricity or heat readily. They are stronger in compression than tension. They are not fully chemically Inert; they are often resistant to chemicals that would damage metals or polymers.Since they are inert they are harmless to human tissues.
Some metals used as restorative materials in dentistry may constitute a problem for some patients. These problems may reveal themselves as allergies, [11] gum staining[12,13] release of metallic ions into the gingival tissue[14] and the gingival fluid[15].Other qualities like Inertness, increased translucency, bio-compatibility, low temperature and electrical conductivity.These crowns are ideally suited to people who have minimal space within their mouth for a crown or prefer something which has a natural appearance.

Although there are some advantages of these crowns it is only fair to highlight the disadvantages as well.The disadvantage are due to inability to withstand the functional forces that are present on the oral cavity,they are brittle,they have low impact toughnesss,they appear opaque and lifeless even it is a right colour.Due to shrinkage,It is difficult to manufacture dimensionally accurate products The properties of a particular ceramic depend not just on the materials but also, on its crystalline structure. The properties of advanced ceramics have made them important for some much more extraordinary applications. The refinement needed to produce these crowns makes them more difficult to fit. They require expertise on which increases their cost.

Classification Of Ceramic System

- Glass Ceramics/ Castable Ceramics
- Sintered Ceramics
- Slip Casting Ceramics
- Hot Pressed / injection ceramics
    - machinable ceramics

Castable Ceramic

It is further classified into

- Mica based

Eg: Dicor

- Hydroxyapatite basedEg: Cerapearl
- Lithia based

Glass Ceramic

Glass ceramics were first developed by Corning Glass Works in the late 1950s. According to McLean,[16] the first works on glass ceramics were performed by Mac Culloch, but his work did not receive much attention. Further investigations by Grossman and Adair[17,18] concluded with the development of a tetra silicic mica-containing ceramic system.Glass ceramic is basically composed of a glass matrix phase and a crystal phase. Dicor was the first commercially available castable ceramic material for dental use.This has a glassy matrix and a crystalline phase. DICOR is a castable containing tetrasillic mica glass ceramic material.Composition is SiO2-45-70%,K2O-20%,MgO-13-30, 55% volume of tetrasillic mica crystals. It has increased strength,toughness, resistance to abrasion, thermal shock resistance,chemical durability,decreased translucency.

It is supplied as DICOR castable ceramic cartridges,special DICOR casting crucibles,DICOR shading porcelain.The advantages of this material are chemical and physical uniformity,marginal accuracy,uncomplicated fabrication from wax-up to casting, ceramming, and colouring, ease of adjustment, excellent aesthetics resulting from natural translucency, light absorption, light refraction, and natural colour for the crown, and its inherent resistance to bacterial plaque[19]. Some the advantages of DICOR are uniformity and purity of the material,minimal processing shrinkage, good fit, low CTE equal to that of the tooth structure,minimal abrasiveness to the tooth structure, radio opacity like dentin,moderately highly flexural strength of 152MPa.The disadvantages of the DICOR are low tensile strength,inability to be coloured internally, labour intensive,high cost. The indications of DICOR are inlays, onlays,partial tooth coverage.

Hydroxyapatite Based Castable Glass Ceramic

The example of hydroxyapatite based castable glass ceramics is Cerapearl. The complication includes CaO-45%,P2O5-15%,Mg2O-5%,SiO2-35%. The casting shrinkage of Cerapearl is

0.53%. It melts at 1460 degree C and flows like a melting glass. The cast material has an amorphous microstructure when reheated at 870 degree Celsius forms crystalline hydroxyapatite. The crystalline structure is similar to enamel. The modulus of rupture is 150MPa.The orientation and size of the crystals can account for crack deflection and blunting, which, in turn, accounts for the increase in fracture toughness over the leucite-reinforced ceramics.[20].

Sintered All Ceramic Material

Examples are Leucite- reinforced feldspathic porcelain : Optec HSP, Aluminous based porcelain : Vitadur-N TM core, Alumina based porcelain : Hi ceram, Magnesia based feldspathic porcelain, Zirconia based porcelain: Mirage II, Hydrothermal low fusing ceramics: Dueceram LFC. It is supplied as powder which can be mixed with water to form a slurry. This slurry can be built up in a layer on a refractory die to form the restoration. The powders are available in different shades and translucency. Theyare stronger because they are reinforced by crystalline phases.Clinical results have demonstrated an excellent in vivo performance at 15 years[21].

Aluminous Core Ceramics

They advocated using aluminous porcelain which is composed of aluminous oxide (alumina)crystals dispersed in a glassy matrix. The technique is devised by McLean[22]used as an opaque inner core containing 50% by weight alumina for high strength. This core was veneered by a combination of aesthetic body and enamel porcelains with 15% and 5% crystalline alumina respectively and matched thermal expansions. The resulting restorations were approximately 40% stronger than those using traditional feldspathic porcelain. Alumina is used because good mechanical properties, interfacial region between alumina and porcelain are virtually stress free. Crystals are used than powdered alumina. It has high modulus of elasticity and high fracture toughness. Advantagesincludes increase flexural strength,increased elasticity and toughness. The disadvantage of aluminous porcelain are extensive reduction, dentin preparation, binding is limited and high failure rates.

Leucite Reinforced Feldspathic Porcelain

OPTEC HSP material (leneric/Pentron, Inc.) is a feldspathic porcelain containing up to 45 vol% tetragonal leucite[23].In this type, the leucite crystals are dispersed in a glass matrix

which are nothing but potassium aluminium silicate. The leucite and glass components are fused together during baking process at 1020degree Celsius. It has high modulus of rupture and compressive strength. It does it require core unlike aluminous PJC. Examples include Optec HSP. Advantages includes : lack of metal or opaque substructure, good translucency compared to alumina crowns, moderate flexural strength, ability to be used without special laboratory equipment, and can be etched. The disadvantages includes: marginal inaccuracy caused by sintering shrinkage, has potential to fracture in posterior teeth, has increased leucite content, requires a special die material. It is used in inlays,onlays,crowns for low stress areas and veneers.

Magnesia Based Core Ceramic

Magnesia core ceramic was developed as an experimental material in 1985[24].It has high expansion core material. Advantages includes high CTE , flexural strength of magnesia core is 131 Mpa,aesthetics are superior to PFM. The disadvantages is that it can't be used in fixed partial denture.The magnesia core material can be significantly strengthened by glazing, thereby placing the surface under residual compressive stresses that have to be overcome before fracture can occur[25].

Zirconia Based Feldspathic Porcelain

Example is Mirage II which are tetragonal zirconia fibres. The advantages includes fracture toughness and thermal shock resistance. The disadvantages are translucency and fusion temperature can be adversely affected.The modulus of rupture of commercially available zirconia-reinforced feldspathic dental porcelain(Mirage II) is not significantly different from that of conventional feldspathic porcelain[26].Zirconia undergoes a crystallographic transformation from monoclinic to tetragonal at 1173°C. Partial stabilisation can be obtained by using various oxides such as CaO, MgO, Y2O3, and CeO,which allows the high-temperature tetragonal phase to be retained at room temperature. The transformation of partiallystabilised tetragonal zirconia into the stable monoclinic form can also occur under stress and is associated with a slight particle volume increase. The result of this transformation is that compressive stresses are established on the crack surface, thereby arresting its growth. This mechanism is called transformation toughening[27].

Slip Casting Ceramic

Slip-cast ceramics for dental restorations were introduced in the 1990s.crystalline phases are available, namely alumina (Al2O3), spinel (MgAl2O4) and zirconia-alumina (12 Ce-TZP-Al2O3).Ceramics are very weak and porous and must be infiltrated with glass or fully sintered before application of the veneering porcelain[28]. Materials processed by this technique tend to have fewer defects from processing, and exhibit higher toughness than the conventional feldspathic porcelain[29].The use of this technique in dentistry has been limited to one of three products[30].This limitation might be due the complicated steps, which makes achieving an accurate fit difficult[31]. Alumina-based slip-cast ceramics contain 68 vol % alumina, 27 vol % glass and 5 vol % porosity[32].The presence of large alumina crystals with a high refractive index, and a non-negligible amount of porosity, account for some degree of opacity in this all-ceramic system. Spinel-based slip-cast ceramics offer better translucency [33] similar to that of lithium disilicate heat-pressed ceramics, at the expense of mechanical properties [34].The framework contains a mixture of magnesia and alumina (MgAl2O4) to improve the translucency of thematerial [35]. Zirconia-toughened alumina slip-cast ceramics comprise 34 vol % alumina and 33 vol % of 12 mol % ceria-stabilized zirconia (12Ce-TZP).

Infiltrated Ceramics

They are otherwise known as slip cast all ceramics. Examples of infiltrated ceramics are alumina based (In ceram), In- Ceram Spinell,In- Ceram Zirconia , In- Ceram 2000. The core of In- Ceram spiral is MgAl2O2. The core of IN- Ceram alumina is 70% alumina infiltrated with 30% sodium lanthanum glass. The core of in-Ceram zirconia is 70% weight if alumina and 30% zirconia. In-Ceram spiral is used in anterior single unit inlays,onlays, crowns and veneers. In- Ceram alumina is used in anterior and posterior crowns and anterior three unit FPD’s. In-Ceram Zirconia in posterior crowns and posterior FPD’s. The advantages of the glass infiltrated ceramics systems are high flexural strength and fracture toughness,aesthetics, biocompatibility and ability to be used with conventional luring cements. The disadvantages of glass infiltrated ceramics systems are high chemical solubility,technique sensitivity, high cost,long time period for fabrication. It is contraindicated if functionally appropriate design of the restoration is not ensured, inadequate preparation, bruxism,severe discolouration of prepared teeth.

Pressable Ceramic

Examples are leucite bases :IPS Empress, Spinel based : AL Ceram. Hot pressed ceramics are becoming popular in dentistry. The restoration are waxed, invested and pressed in a manner somewhat similar to gold casting. There is better marginal adaptation with hot pressing than the high strength alumina core material. Most hot pressed material contains leucite as a major crystalline phase,dispersed in a glassy matrix. The Crystal size varies from 3 to 10 mm leucite content is 35% and glass 65%. Leucite is used as a reinforcing phase due to the tangential stressed it creates within the porcelain.This temperature is held for 20 minutes in a specially designed automatic press furnace[36].They are produced by sintering. The flexural strength of IPSEmpress material was significantly improved after additional firings[37]. The advantages includes : excellent aesthetic,translucent ceramic core,moderately high flexural strength, excellent fit, minimal shrinkage. Some of the disadvantages are potential to fracture in posterior, needs expensive equipment's, needs to use resin cement to Bond the crown micro mechanically to the tooth structure.

Machinable Ceramic

Computer Aided Design/Computer Aided Design (CAD/CAM) technology was introduced in dentistry by Duret in the early 70's[38].The best example of machinable ceramics is CAD-CAM. This uses digital information about the tooth preparation or a pattern of the restoration to provide a computer aided design (CAD) on the video monitor for inspection and modification. Thestages of fabrications are computerised surface digitalisation,computer aided design computer assisted manufacturing, computer aided aesthetics,computer aided finishing. Commercially available CAD-CAM Systems are procera, celay,sophna,Cicero,Cercon,Dux,Denticard,the Japanese system,Cercon.

Cerecincludes Cerec 1, Cerec 2, Cerec 3, Cerec 3D. Cerec 1 is a simple inlays. It has a very sharp internal angles . The Occlusal surface cannot be fabricated with CEREC 1. Cerec 2 is improved version of Cerec 1 with an addition of cylindrical grinders. The Occlusal surface can be grounded with Cerec 2. Cerec 3 is radio controlled operating system due to its design and million chamber units can be delude separately. It simplifies Occlusal and functional registration. Cerec 3D is latest version which allows 3D view of the preparation.

Celay system uses a copy milling technique to manufacture ceramic inlays and onlays.Celay system is a mechanical device based on pantographic tracing of a resin inlay or onlay

fabricated directly onto the prepared tooth or onto the master die[39].This material is similar to vita mark II Ceramic used with the Cerec 2 system[40].It has a marginal accuracy better than Cerec 2 system. Procera Allceram system involves an industrial CAD/CAM process. In LAVA system , a CAD/CAM procedure is used for fabrication of zirconia frameworks all ceramic system.

Cementation of All ceramic system

The protocol used for cementation of all-ceramic restorations can be essential for success[41].Clinicians can effectively etch silica-based all-ceramics for adhesive bonding[42].The clinical life span of such all-ceramic restorations significantly increased when this protocol is used[43].

Conclusion

The desire for beautiful teeth is a good reason to pay attention to the selection and quality of the restoration. In search for the ultimate aesthetics restorative materials, all ceramic system is the best choice among all the restorative materials.tremendous progress has been made in terms of mechanical performance, with a ten-fold increase in flexural strength and fracture toughness. Technology has developed metal-free ceramic materials which are capable of reproducing a natural appearance, whichtraditional materials have been replaced by them.The future of ceramics for dentistry is clearly open to new technologies.All-ceramic materials and systems will continue to improve.

Corresponding Author

Sreedevi Dharman

Senior Lecturer, Department of Oral Medicine,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162,Poonamalle High Road,Chennai-600077,Tamil Nadu, India

E-Mail: sanjamrut@gmail.com

Telephone Number: 919841009003

Reference

1. McLean JW. Evolution of dental ceramics in the twentieth century. Journal of Prosthetic Dentistry. 2001 Jan 1;85(1):61-6.
2. Fischer H, Marx R. Fracture toughness of dental ceramics: comparison of bending and indentation method. Dental materials. 2002 Jan 1;18(1):12-9.
3. Shenoy A, Shenoy N. Dental ceramics: An update. Journal of conservative dentistry: JCD. 2010 Oct;13(4):195.
4. Griggs JA. Recent advances in materials for all-ceramic restorations. Dental Clinics. 2007 Jul 1;51(3):713-27.
5. Raptis NV, Michalakis KX, Hirayama H. Optical behaviour of current ceramic systems. International Journal of Periodontics & Restorative Dentistry. 2006 Jan 1;26(1).
6. Shenoy P, Aithal PS. A Study on History of Paper and possible Paper Free World.
7. Carty WM, Senapati U. Porcelain—raw materials, processing, phase evolution, and mechanical behaviour. Journal of the American Ceramic Society. 1998 Jan 1;81(1):3-20
8. Maniatis Y, Tite MS. Technological examination of Neolithic-Bronze Age pottery from central and southeast Europe and from the Near East. Journal of archaeological Science. 1981 Mar 1;8(1):59-76.
9. Ferracane JL. Materials in dentistry: principles and applications. Lippincott Williams & Wilkins; 2001.
10. Stejskal V, Hudecek R, Stejskal J, Sterzl I. Diagnosis and treatment of metal-induced side-effects. Neuro Endocrinol Lett. 2006 Dec;27(Suppl 1):7-16.
11. ARVIDSON K, WRÓBLEWSKI R. Migration of metallic ions from screwposts into dentin and surrounding tissues. European Journal of Oral Sciences. 1978 May 1;86(3):200-5.
12. Venclikova Z, Benada O, Bartova J, Joska L, Mrklas L. Metallic pigmentation of human teeth and gingiva: morphological and immunological aspects. Dental materials journal. 2007;26(1):96-104.
13. Bumgardner JD, Lucas LC. Cellular response to metallic ions released from nickel-chromium dental alloys. Journal of dental research. 1995 Aug;74(8):1521-7.

14. Mehulić K, Prlić A, Komar D, Prskalo K. The release of metal ions in the gingival fluid of prosthodontic patients. Acta stomatologica Croatica. 2005 Mar 15;39(1):47-51.
15. McLean JW. Evolution of dental ceramics in the twentieth century. Journal of Prosthetic Dentistry. 2001 Jan 1;85(1):61-6.
16. Adair PJ, inventor; Corning Inc, assignee. Glass-ceramic dental products. United States patent US 4,431,420. 1984 Feb 14.
17. Grossman D, inventor; Corning Inc, assignee. Tetrasilicic mica glass-ceramic method. United States patent US 3,732,087. 1973 May 8.
18. Malament KA, Grossman DG. The cast glass-ceramic restoration. The Journal of prosthetic dentistry. 1987 Jun 1;57(6):674-83.
19. Mansour YF, Al-Omiri MK, Khader YS, Al-Wahadni A. Clinical performance of IPS-Empress 2 ceramic crowns inserted by general dental practitioners. J Contemp Dent Pract. 2008 May 1;9(4):9-16.
20. Haag P, Andersson M, Vult von Steyern P, Odén A. 15 Years of Clinical Experience with Procera® Alumina.
21. Mclean Jw. The reinforcement of dental porcelain with ceramic oxides. Br. Dent. J.. 1965;119:251-67.
22. Schmid M, Fischer I, Salk M, Strub) (1992). Mikrogefugeleucit-verstarkter glaskeramiken. Schweiz MonatsschrZahnmed 102:1046-1053.
23. Denry IL. Recent advances in ceramics for dentistry. Critical Reviews in Oral Biology and Medicine. 1996 Apr;7(2):134-43.
24. Wagner WC, O'Brien WJ, Mora GP. Fracture-surface analysis of a glaze-strengthened magnesia core material. International Journal of Prosthodontics. 1992 Sep 1;5(5).
25. Seghi RR, Daher T, Caputo A. Relative flexural strength of dental restorative ceramics. Dental Materials. 1990 Jul 1;6(3):181-4.
26. Baccour H, Medhioub M, Jamoussi F, Mhiri T. Influence of firing temperature on the ceramic properties of Triassic clays from Tunisia. Journal of materials processing technology. 2009 Mar 19;209(6):2812-7.
27. Denry IL. Recent advances in ceramics for dentistry. Critical Reviews in Oral Biology & Medicine. 1996 Apr;7(2):134-43.
28. Griggs JA. Recent advances in materials for all-ceramic restorations. Dental Clinics. 2007

Jul 1;51(3):713-27.

29. Sakaguchi RL, Powers JM. Craig's Restorative DentalMaterials-E-Book. Elsevier Health Sciences; 2012 Jul 16.
30. Griggs JA. Recent advances in materials for all-ceramic restorations. Dental Clinics. 2007 Jul 1;51(3):713-27.
31. Sulaiman F, Chai J, Wozniak WT. A comparison of the marginal fit of In-Ceram, IPS Empress, and Procera crowns. International Journal of Prosthodontics. 1997 Sep 1;10(5).
32. Guazzato M, Albakry M, Ringer SP, Swain MV. Strength, fracture toughness and microstructure of a selection of all-ceramic materials. Part II. Zirconia-based dental ceramics. Dental materials. 2004 Jun 1;20(5):449-56.
33. Heffernan MJ, Aquilino SA, Diaz-Arnold AM, Haselton DR, Stanford CM, Vargas MA. Relative translucency of six all-ceramic systems. Part I: core materials. Journal of Prosthetic Dentistry. 2002 Jul 1;88(1):4-9.

34. Magne P, Belser U. Esthetic improvements and in vitro testing of In-Ceram Alumina and Spinell ceramic. International Journal of Prosthodontics. 1997 Sep 1;10(5).

35.Heffernan MJ, Aquilino SA, Diaz-Arnold AM, Haselton DR, Stanford CM, Vargas MA. Relative translucency of six all-ceramic systems. Part I: core materials. J Prosthet Dent 2002;88(1):4-9.

36.Dong JK, Luthy H, Wohlwend A, Schärer P. Heat-pressed ceramics: technology and strength. International Journal of Prosthodontics. 1992 Jan 1;5(1).

37.Duret F, Blouin JL, Duret B. CAD-CAM in dentistry. The Journal of the American Dental Association. 1988 Nov 1;117(6):715-20.

38.Höland W, Frank M, Rheinberger V. Surface crystallization of leucite in glasses. Journal of non-crystalline solids. 1995 Jan 1;180(2-3):292-307

39. Eidenbenz S, Lehner CR, Schärer P. Copy milling ceramic inlays from resin analogs: a practicable approach with the CELAY system. International Journal of Prosthodontics. 1994 Mar 1;7(2).

40. Donovan TE. Factors essential for successful all-ceramic restorations. The Journal of the American Dental Association. 2008 Sep 1;139: S14-8.

41.Malament KA, Socransky SS, Thompson V, Rekow D. Survival of glass-ceramic materials and involved clinical risk: variables affecting long-term survival. Practical procedures & aesthetic dentistry: PPAD. 2003:5-11.

42. Sorensen JA, Kang SK, Torres TJ, Knode H. In-Ceram fixed partial dentures: three-year clinical trial results. Journal of the California Dental Association. 1998 Mar;26(3):207-14.

43. Zeng L, Zhang Y, Liu Z, Wei B. Effects of repeated firing on the marginal accuracy of Co-Cr copings fabricated by selective laser melting. Journal of Prosthetic Dentistry. 2015 Feb 1;113(2):135-9.

Alteration in the Proportion of Bacterial Flora in the Oral Cavity after Consuming Probiotics

Nivedha Srinivasan[1], N P Muralidharan[2], Niveda S[3]

Graduate student[1,] Professor[2], Department of Microbiology,

Tutor[3], Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Products containing Probiotics had various health benefits. Traditionally Probiotics have been associated with treatment of gastrointestinal infections and disease. Various studies suggested the use ofProbiotics for oral health purposes. This study focuses on alteration in the proportion of bacterial flora in the oral cavity before and after consuming Probiotics

Keywords- *Probiotics, oral health, lactobacillus, mitis, S.Mutans*

Introduction

Probiotics can be defined as living microbes that beneficially influence the health of the host when used in adequate numbers. Probiotics are associated with gut health, and most clinically been focused on the prevention or treatment of gastrointestinal infections and diseases. Recently they have been suggested for oral health purposes too. Probiotics mouthwash can also be considered as an effective oral hygiene regimen [1]. It allows concomitant inhibition of pathogen adhesion and competitive exclusion of pathogenic microorganisms, thus allowing the production of anti-microorganism substances and modulation of the immune system. The most commonly used Probiotics bacterial strains belong to the genera Lactobacillus and Bifidobacterium.

Lactobacilli andBifidobacterium are generally regarded as secure, recently even more fermented food products have been associated for wellbeing. The species composition of both Lactobacillus and Bifidobacteriummicro biota is different between patients with periodontitis and those who are periodontally healthy. On the other hand, both Lactobacilli and Bifidobacterium are also associated with dental caries. The aim is to examine potential mechanism of Probiotics bacteria in the oral mucosa and to summarize its observed effects.

Materials and Methods

This study was carried out in nineteen healthy individuals within the age range of 18-48. The healthy volunteers were selected after the careful examination of the oral cavity for any lesions [2]. The subjects were given yakult, a Probiotics daily product made by fermenting a mixture of skimmed milk with a special strain of the bacterium Lactobacillus caseishirota, once daily for seven days. Saliva samples were collected before consuming Yakult on the first day as baseline. The collected saliva samples were inoculated into mutantssanguisagar(hi media: code m977),mitissanguis agar (hi media: code m259) and lactobacillus (hi media: code m641) which are known to be an indicator media for Streptococcus mutans, mitis and Lactobacillus bacteria [3]. The sample was diluted in 1:20 times with sterilenormal saline and 10µl of the sample is added into mutanssanguis agar, mitissanguis agar and lactobacillus which were incubated for 24 hours at 37^{o}c aerobically. After the incubation for 24hrs, the numberofbacterial colony forming unit (CFU) were counted. The colonies with white frosted glass appearance confirms Streptococcus mutans, the colonies with blue frosted glass appearance confirms Streptococcus mitis and yellow frosted glass appearance confirm Lactobacillus. After consuming Yakult for seven days, second sample was collected on the

seventhday and processed similarly. The number of mutans, mitis and Lactobacillus were counted and compared with the mutans, mitis and Lactobacillus count of samples before consuming Yakult.

Results

Table 1: Showing bacterial count before and after consuming Probiotics

	Before consuming Probiotics			**After consuming Probiotics**		
S.No	S.Mutans	Mitis	Lactobacillus	S.Mutans	Mitis	Lactobacillus
1	88	120	208	76	68	212
2	68	80	244	56	48	244
3	60	72	64	88	72	96
4	60	96	112	48	36	128
5	112	108	112	64	44	132
6	88	288	224	80	108	248
7	88	48	96	68	48	132
8	44	64	116	44	28	240
9	84	88	224	84	64	232
10	68	172	96	64	72	192
11	80	48	172	32	44	172
12	128	80	140	36	52	172
13	124	64	156	28	64	160
14	68	120	188	40	48	232
15	48	72	212	36	44	232
16	36	40	228	28	32	228
17	136	68	240	72	32	252
18	180	56	276	68	56	292
19	92	72	196	80	76	212
	1652	1756	3304	1092	1036	3808

It is evident that usage of probiotics showed significant reduction in the bacterial count of S. Mutans and Mitis and there is no reduction in the bacterial count of lactobacillus after the usage of probiotics.

Figure 1: Pie chart depicting the bacterial count

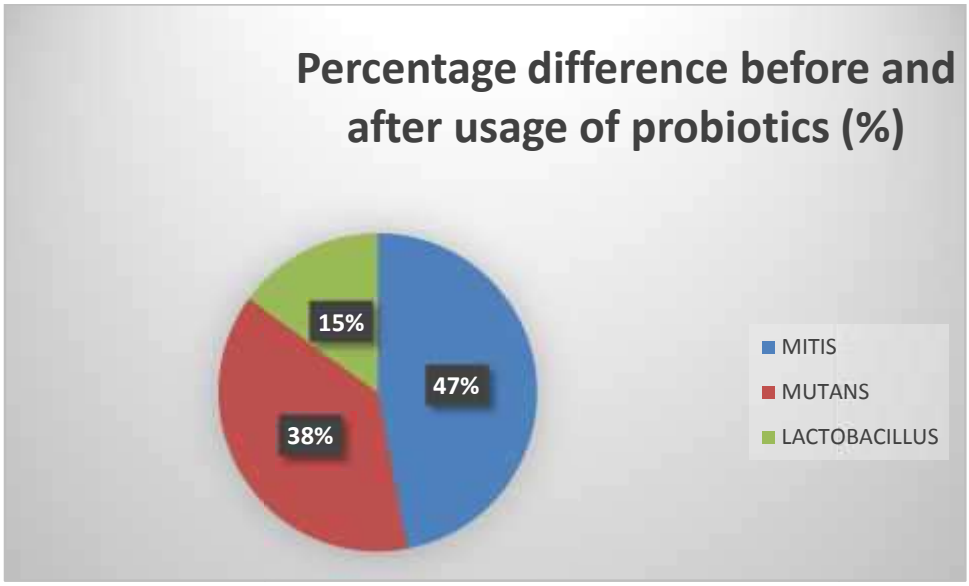


Table 2 shows percentage difference among bacterias before and after consuming probiotics

Organism	Percentage difference before and after usage of probiotics (%)
S. Mitis	**47%**
S.Mutans	**38%**
Lactobacillus	**15%**

Discussion

This study was conducted to find out the alteration in the proportion of bacterial flora in the oral cavity after consuming Probiotics [4-7]. Twenty healthy volunteers were selected after the careful examination ofthe oral cavity for any lesions. Among the twenty volunteers, one was removed for not complying with the protocol. Saliva samples were collected from the remaining nineteen volunteers. After seven days period of consuming Probiotics, the salivary

samples were subjected to microbiological examinations specific to the Streptococcus mutans, mitis and Lactobacillus load. Among the nineteen volunteers, all of them have shown a significant reduction in the mutans and mitis count. The mean average reduction of Mutans and mitis were found to be 33.89% and 41% respectively. But there was a significant increase in the Lactobacillus count with the mean average being 13.23%. Though there has been no consistent reduction in the mutans and mitis count, the mode of frequencies suggest that there is a definite influence of Yakult in reducing the bacterial pathogenic load.

Only a few studies are available on the prevalence and role and effects of Probiotics bacteria in the mouth. A study conducted among 130 volunteers at Thailand found 3790 lactobacillus strains from healthy oral cavities. Of these, five species expressed inhibitory effect against other microorganisms including oral Candida [8-10]. Also in Finland, showed an inhibitory effect on salivary counts of Streptococcus mutans and yeasts by using a combination of IgG andbifidobacteria in a short term intervention study in young adults who consumed specially manufactured cheese [11-13]. The results showed no significant difference between the groups in s. Mutans counts after the intervention, but during the post treatment period there was a significantly greater reduction in these counts in the intervention group compared with the control group [14]. More recently, Probiotics cheese was investigated in 294 self-acting elderly, aged 70-100 yr. The results showed that the Probiotics were reduced the prevalenceof oral Candida and also the risk for hypo salivation in the elderly [15]. Some studies states that the salivary pellicle influences the colonisation of bacteria on the tooth surfaces and saliva is also a potential medium for a person-to-person transmission of bacteria,including pathogenic species [16-18].

Probiotics improve colonization resistance to gut pathogens by reinforcing the mucosal barrier and restoring normal gut micro ecology after diarrhoea [18]. Probiotics have been shown to normalize an increased permeability of bacteria. Bindingis considered to be the first step of pathogenesis and binding of bacteria to the intestinal mucosa or mucus may allow the colonization [19].

Researchers state that lactic acid bacteria can produce different antimicrobial components such as organic acids, hydrogen peroxide, carbon peroxide, diacetyl, low molecular weight antimicrobial substances, bacteriocins and adhesion inhibitors. Probiotics can also activate and modulate the immune system [8], and they have shown to reinforce gut defence by immune exclusion, immune elimination and immune regulation [9]. Nevertheless, since

mouth represents the first part of the gastrointestinal tract, there is every reason to believe that the fermented milk containing Lactobacillus caseishirota has a beneficial role in the mouth [19,20].

Conclusion

Probiotics containing dairy products such as Yakult, curd, cheese are consumed on daily basis. These products reduce the pathogenic bacteria load in the oral cavity and also known to enhance the digestive system. This is known to bring direct benefit in reducing dental caries, dental plaque, xerostomia, halitosis etc. They are probably going to play an important role in dealing with the problems arising from overuse of antibiotics and antimicrobial resistance. Probiotics are nevertheless, a new and interesting field of research in oral microbiology and oral medicine. The concept casts new light on the connections between diet and health, including oral health.

Corresponding Author

S.Niveda

Tutor,Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Email: drnivedasubash@gmail.com

Telephone Number: +919952979464

References MINIMUM 20 REFERENCES

1. Aas, J. A., Griffen, A. L., Dardis, S. R., Lee, A. M., Olsen, I., Dewhirst, F. E., Leys, E. J. &Paster, B. J. Bacteria of Dental Caries In Primary And Permanent Teeth In Children And Young Adults. J ClinMicrobiol 2008; 46:1407-1417
2. SaxelinM. Lactobacillus Gg- A Human Probiotics Strain With Thorough Clinical Documentation. Food Rev Int 1997;13:293-313
3. Cohen Ps, Laux Dc. Bacterial Adhesion To and Penetration of Intestinal Mucus in Vitro. Methods Enzymol 1995; 253:309-314

4. Rojas M, Conway Pl. Colonization by Lactobacilli of Piglet Small Intestinal Mucus, J. ApplBacterial 1996; 81:474-480.
5. OuwehandAc, Isolauri E, Salminen S. The Role of Intestinal Microflora forthe Development ofthe Immune System in Early Childhood. EurJ Nutr 2002; 41:32-37
6. Silva M, JacobusNv, Deneke C, Gorback Sl. Antimicrobial Substance from A Human Lactobacillus Strain. Antimicrobial Agents Chemother 1987; 31:1231-1233.
7. OuwehandAc. Antimicrobial Components from Lab. In: Saliminen S, Wright A, Eds. Lactic Acid Bacteria. New York: Marcel Dekker Inc. 1998;139-159
8. Kato I, Yokura T, Mutai M, Macrophage Activation by Lactobacillus Casei in Mice. MicrImmunol 1983; 27:611-618.
9. Isolauri, KirjavainenPv, Saliminen S. Probiotics- Role inthe Treatment of Intestinal Infection and Inflammation? Gut.2002; 50(3):129-134.
10. SokheeS, Chulasiri M, Prachyabrued W. Lactic Acid Bacteria from Healthy Oral Cavity of Thai Volunteers: Inhibition of Oral Pathogens. J. ApplMicrobial 2001; 90: 172-179.
11. AholaAj, Yli-Knuuttila H, Suamalainen T, Poussa T, Ahlstrom A, MeurmanJh, Korpela R. Short-Term Consumption Of Probiotics- Containing Cheese And Its Effect On Dental Caries Risk Factors. Arch Oral Biol 2002;47:799-804
12. HatakkaK, Ahola A. Yli-Knuuttila H, Richardson M, Poussa T, Suamalainen T, MeurmanJh, and Korperla R. Cheese withProbiotics Bacteria Reduce Prevalence of Oral Candida inthe Elderly. Am J ClinNutr 2005; In Press.
13. Mohan Jothika, P. PranavVanajassun, Effectiveness Of Probiotic, Chlorhexidine And Fluoride Mouthwash Against Streptococcus Mutans – Randomized, Single-Blind, In Vivo Study,Saveetha Dental College,Jispcd, 2015 May; 5(1): S44–S48.
14. Horvath A, Dziechciarz P, Szajewska H. Meta-analysis: Lactobacillus rhamnosus GG for abdominal pain-related functional gastrointestinal disorders in childhood. Aliment Pharm Ther. 2011; 33:1302-10.
15. Kukkonen K, Savilahti E, Haahtela T, Juntunen-Backman K, Korpela R, Poussa T, et al. Long-term safety and impact on infection rates of postnatal Probiotic and Probiotic (symbiotic) treatment: Randomized, double-blind, placebo-controlled trial. Pediatrics. 2008; 122: 8-12.

16. Smerud HK, Kleiveland CR, Mosland AR, Grave G, Birkeland S-E. Effect of a Probiotic milk product on gastrointestinal and respiratory infections in children attending day-care. Microbial Ecology to Health and Disease. 2008; 20:80-5.

17. Weizman Z, Asli G, Alsheikh A. Effect of a Probiotic infant formula on infections in child care centers: Comparison of two Probiotic agents. Pediatrics. 2005; 115:5-9.

18. Spindler-Vesel A, Bengmark S, Vovk I, Cerovic O, Kompan L. Synbiotics, Probiotics, glutamine, or peptide in early enteral nutrition: A randomized study in trauma patients. Jpen-Parenter Enter. 2007; 31:119-26.

19. Rayes N, Hansen S, Seehofer D, Muller AR, Serke S, Bengmark S. Early enteral supply of fiber and lactobacilli versus conventional nutrition: A controlled trial in patients with major abdominal surgery. Nutrition. 2002; 18: 609-15

20. Alho OP, Koivu M, Sorri M, Rantakallio P. The occurrence of acute otitis media in infants. A life-table analysis. Int J Pediatr Otorhinol. 991; 21:7-14.

Alteration in the Taste Function after the Extraction of an Impacted Mandibular Third Molar- A Review.

Deepti Anna John[1], Jaiganesh Ramamurthy[2]

Graduate Student[1], Reader[2], Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Technical and Medical Sciences, Saveetha University, Chennai, India

Abstract

The third molar extraction is one of the most common procedures done in Oral and maxillofacial surgery. The common solution to an impacted third molar is to remove the tooth by surgical extraction. The third molar extraction is a frequently done dental procedure. The extraction of the third molar requires accurate planning and skill. The mandibular third molar lies closely to the branches of the mandibular nerve such as the lingual, inferior alveolar and buccal nerves and the chorda tympani branch of the facial nerve. The surgery endangers the Lingual nerve and the Inferior alveolar nerve, and if the lingual nerve gets damaged there are alterations in the taste function. The review is done to get a better understanding on how the taste function gets affected after the removal of a third molar and if it is a common occurrence and to determine the incidence and the occurrence of change in the taste function after the third molar is removed.

Key Words*: Third molar, taste change, anaesthesia, extraction, impaction*

Introduction

Impaction, according to Mead in 1945 is defined as a tooth that is prevented from erupting because of lack of space, malposition and other impediments. It was also defined by Peterson as those teeth that fails to erupt into the dental arch within the expected time. According to Farman in 2004, impacted teeth are those teeth that prevented from eruption due to a physical barrier within the path of eruption (1). The third molar is one of the three molars per quadrant of the human dentition. The third molar is commonly known as the wisdom tooth as it erupts between the ages of 17 and 25. The most common tooth to get impacted is the Third molar which occurs in about 73% (2) and hence affects the other teeth or causes problems in the oral cavityranging from infection, inflammatory to cystic lesions necessitating their surgical removal.

The common solution to an impacted third molar is to remove the tooth by surgical extraction. The third molar extraction is a frequently done dental procedure. In 1977, it was stated that 2.25 million third molars are removed annually but this procedure is of a difficult degree and can lead to multiple complications such as the iatrogenic damage to the trigeminal nerve.

Reason of Mandibular Third Molar Impaction

Many theories have been proposed for the impaction of the mandibular third molar such as insufficient retromolar space, the mandibular ramus growth depends on resorption at its anterior surface and deposition at its posterior surface, but due to defect in this process, the mandibular third molars don’t get enough space to erupt (3). The eruption also depends on the favourable path of eruption, like if the tooth bud is angulated towards the medial side during the early stages of calcification and root development the path of eruption will be deemed unfavourable (4). Angulation of the mandible can also affect the eruption pathway. Yamaoka et al found that the root angulation is related to impaction as angulated roots were more common in impacted mandibular third molars as compared to erupted mandibular third molars (5). A lack of sufficient eruption force for third molars and decreased mesial movement of the modern human dentition due to lack of interproximal attrition (6).

Impaction can be of different types such as mesio angular, vertical, distoangular and horizontal Impaction. Mesioangular is the most common type of Impaction seen, studies say that there is a strong genetic influence that plays a role in molar Impaction (7).

Relation of the Mandibular Third Molar with the Mandibular Nerve

The Trigeminal nerve is the Vth cranial nerve which is divided into three branches, the ophthalmic, maxillary and the mandibular nerves. The trigeminal nerve (V) is responsible for the general sensory innervation of the tongue.

The mandibular nerves are divided into many branches such as the inferior alveolar, lingual, buccal nerves. The mandibular third molar lies closely to the branches of the mandibular nerve such as the lingual, inferior alveolar and buccal nerves and the chorda tympani branch of the facial nerve.

Neurosensory disturbances related to the inferior alveolar nerve due to the close anatomic relationship between the roots of mandibular third molars and the inferior alveolar canalis one of the most grave complications of such a procedure. The prevalence of inferior alveolar nerve paraesthesia following third molar surgery ranges approximately from 0.4 to 8.4 % according to different studies(8).So during the surgical extraction there are chances that these nerves could get damaged. The nerve damage causes sensory disturbances such as loss of gustatory function (dysgeusia and aguesia) (9). The nerve damage may be due to the anaesthetic injection given that causes haemorrhage with the epineurium or the neurotoxic effect of the anaesthetic.

As the mandibular third molar is located at the distal end of the body of the mandible where the ramus is thin. This is the region of weakness and a fracture can easily occur if a greater amount force is applied during impacted wisdom tooth elevation without preliminary and adequate removing of surrounding bone. The buccal alveolar bone is thicker than the lingual. The external oblique ridge forms the buttress that reinforced the buccal plate. The lingual nerve is situated close to the cortical plate and there is an increased risk of lingual nerve. In a few cases third molar roots may penetrate into mandibular canal or close relationship of the canal with the roots can evoke inferior alveolar nerve damage during the

surgery (10).The taste sensation is responsible by the three cranial nerves: facial nerve (VII), glossopharyngeal (IX) and vagus(X).

The close anatomic proximity of the mandibular third molar to the chorda tympani or lingual nerve or both during surgical procedures could lead to gustatory deficit (11). In the anterior 2/3rd of the tongue, the taste impulses are initiated and are transmitted to the medulla oblongata through the taste fibres that arise from the lingual nerve. Then the fibres leave the nerve and form a part of the chorda tympani. Some of the gustatory fibres arise on the tongue also might reach the brain stem through the mandibular nerve. So, the presence of this pathway may be the reason of unilateral loss of taste (12). Hence, the aim of the review is to understand whether the removal of an impacted mandibular third molar can affect the gustatory function.

Extraction of Mandibular Third Molar and Its Significance to Gustatory Function

According to C.D Lehman (1995), the tongue is divided into two halves which are innervated by two different nerves- the anterior two thirds is innervated by the chorda tympani branch of the facial nerve and the posterior one third is innervated by the glossopharyngeal nerve. Anaesthesia of the chorda tympani nerve given during a dental procedure increases the taste sensation of the areas innervated by the glossopharyngeal nerve. This follows the principle of Halpern and Nelson's release of inhibition hypothesis (13). Yanagisawa et al (1998), reported that if the chorda tympani nerve was anaesthetized the taste of quinine was intensified and the taste of NaCl was diminished. In 40% of his subjects, tastes occurred in the absence of stimulation (taste phantoms); the phantoms may occur due to the anaesthesia that releases inhibition normally occurring between the central projections areas of different taste nerves (14).

Shafer DM et al (1999) observed the taste function of 17 patients before the third molar surgery and at 1 month and 6 months after the surgery, the taste intensity in certain areas of the tongue had diminished and the patients with the severely impacted molar gave the lowest values. Their study concluded that gustatory deficits occur after the third molar extraction and last as long as 6 months and also depends on the depth of the impaction (15). DGulicher et al (2001), their study involved the removal of 1106 impacted mandibular molars

from 687 patients, the common sensation was examined to assess sensory deficit. 3.6% of the operated sides showed impairment of labial sensation and 2.1% of lingual sensation. Persisting Sensory diminution was seen in the inferior alveolar nerve and lingual nerve. The factors concerned according to the study were general anaesthesia and the individual operator (16).

Anwar B.Bataineh (2001), the study was conducted with 741 patients whose 741 mandibular third molars were removed under local anaesthesia, the results showed that 19 patients had post-operative lingual nerve paraesthesia and they belonged in the under 20-year old age group (17).Steven B. Graff-Radford et al (2003) examined a case of a 24 year old woman, who underwent local anaesthetic blocks and removal of upper and lower molars by her dentist but started feeling numbness and tingling of the left side of her tongue and the floor of her mouth, she also felt a change in the taste of meat. On examination, it was seen that the lingual nerve was damaged (18).U.K.Akal et al (2004), the study was done to determine the drastic taste changes after all the four third molars were removed.27 patients were subjected to two gustatory test but the final result showed no significant changes in the gustatory function after the third molar surgery (19).Linda M.Bartoshuk et al(2005),reported that taste phantoms are localized to contra lateral rear of the tongue when anaesthesia is given to the chorda tympani (20).Ana Claudia Amorim Gomes et al (2005), their study had a total of fifty five patients referred for bilateral mandibular third molar removal, lingual flap retraction done during the surgical procedure damaged the lingual nerve (21).WaseemJerjes et al (2010), their study reported that permanent sensory dysfunction is depends on the surgical skills of the operator, type of impaction, radiographic proximity of the tooth to the inferior alveolar nerve (22).Lourdes Ridaura- Ruiz et al (2012),their studied reported that lower third molar removal under local anaesthesia may cause light lingual sensibility impairment, but this in most cases remains undetected to the patients. The injuries if present are seen for only a week after the surgery. The taste remains unaffected (12). W. P Smith (2013), observed 1000 patients and reported a preoperative warning that temporary neurosensory deficit is highest when the root apices are intimate with the inferior alveolar nerve and permanent damage is done when the nerve is intimate with the canal (23).

Investigation Done to Determine the Close Proximity of the Mandibular Nerve to the Impacted Third Molar

Pre-operative radiographic assessment of the proximity of the mandibular nerve to the impacted molar is a vital part and essential requirement before surgical removal of impacted mandibular third molars. Orthopantomograms are commonly used for the assessment.
Radiographs should be carefully studied and the following features should be noted prior to the extraction procedure.

a. Radiographic distance between the impacted third molar and the inferior alveolar canal. The mean distance is said to be 1.5011mm
b. Risk predictor signs –
    - Darkening of the root- indicates the impingement of root on the canal
    - Interruption of the white line –discontinuity in the opaque border that indicates the border of the inferior alveolar canal.
    - Diversion of the canal
    - Narrowing of the root
    - Dark and bifid root – indicates impingement by the canal

Presence of two or more of the risk factors seen in the radiograph indicates the close proximity of the nerve to the tooth and increases the risk of damage to the nerve (24)
Therefore in these cases, the oral surgeon should do the extraction using copious amounts of irrigation of the socket and direct visualisation of the inferior alveolar canal, to ensure least amount of complications.

Panoramic radiographs are said to be gold standard diagnostic tools in the preoperative assessment of mandibular third molars and their relationship with the inferior alveolar canal (25) and have been known as the radiographs of choice. As a protocol, the Finnish Student Health Service in Helsinki proposes use of advanced imaging techniques only when panoramic radiographs suggest a close relationship between the impacted mandibular third molar and the inferior alveolar canal.

Conclusion

The literary search was done to understand the connection of the mandibular third molar with the taste function. From the articles, it's concluded that the removal of the mandibular third molar does not cause drastic changes to the gustatory function or no change at all in most cases, but if the nerves get damaged there are chances for neurosensory dysfunction to be present but it may last for at least 6 months. It is possible that if the taste loss persists beyond 6 months together with other factors that affect taste and accumulate over a lifetime, contributes to the decrease in taste function seen with age. The major factors that are responsible for the damage of the nerves closely related to the mandibular third molar are application of anaesthesia and the skill of the operator.

Corresponding Author

Jaiganesh Ramamurthy,

Reader, Department of Periodontics

Saveetha Dental College, Saveetha Institute of Technical and Medical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077, Tamil Nadu, India.

Mobile Number: +91-98404 43463,

Email: dr.r.jaiganesh@gmail.com

References

1. Spiotto M, Juodzbalys G, Daugela P. Mandibular Third Molar Impaction: Review of Literature and a Proposal of a Classification. Journal of Oral and Maxillofacial Research. 2013;4(2):e1.

2. Khan NB, Chohan AN, AlMograbi B, AlDeyab S, Zahid T, AlMoutairi M. Eruption Time of Permanent First Molars and Incisors Among a Sample of Saudi Male Schoolchildren. Saudi Dent J 2006 Jan-Apr;18(1):18-24.
3. Björk A. Prediction of mandibular growth rotation. Am J Orthod 1969; 55:585–99.
4. Richardson M. Changes in lower third molar position in the young adult. Am J Orthod Dentofacial Orthop. 1992 Oct;102(4):320-7
5. Yamaoka M, Tambo A, Furusawa K. Incidence of inflammation in completely impacted lower third molars. Aust Dent J 1997 Jun;42(3):153-5
6. Bermúdez de Castro JM. Third molar agenesis in human prehistoric populations of the Canary Islands. Am J Phys Anthropol 1989 Jun;79(2):207-15.
7. Sagar.S. Variation in Anatomy and Position of Mandibular Third Molars in South Indian Population. J Pharm Sci Res 2015; Vol. 7(6):385-6
8. Valmaseda-Castellon E, Berini-Aytes, Gay-Escoda C. Inferior alveolar nerve damage after lower third molar surgical extraction: a prospective study of 1,117 surgical extractions. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001;92:377–83.
9. Loescher A, Smith K, Robinson P. Nerve Damage and Third Molar Removal. Dental Update. 2003;30(7):375-82.
10. Kay LW, Killey HC. The extraction of teeth. The impacted third molar. Dent Update 1976; Mar-Apr;3(2):49-52.
11. Klasser GD, Utsman ,EpsteinJB.Taste Change Associated with a Dental Procedure: Case Report and Review of the Literature.Todays FDA 2009;21:21-5.
12. Ridaura-Ruiz L, Figueiredo R, Valmaseda-Castellon E, Berini-Aytes L, Gay-Escoda C. Sensibility and taste alterations after impacted lower third molar extractions. A prospective cohort study. Medicina Oral Patología Oral y Cirugia Bucal. 2012:e759-e764.
13. C.D. Lehman, L.M Bartoshuk, F.C. Catalanotto, J.F.Kveton, R.A. Lowlicht. Effect of anaesthesia of the chorda tympani nerve on taste perception in humans. Physiol Behaviour57:943-51.
14. K.Yanagisawa, L.M Bartoshuk, F.C. Catalanotto, J.F.Kveton, T.A Karrer. Anaesthesia of the chorda tympani nerve and taste phantoms. Physiol Behaviour 1998;63(3):329-35.

15. Shafer DM, Frank ME, Gent JF, Fischer ME –Gustatory function after third molar extraction. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999;87(4):419-28.
16. D.Gulicher,K.L. Gerlach. Sensory impairment of the lingual and inferior alveolar nerves following removal of impacted mandibular third molars. Int J Oral Maxillofacial Surgery 2001;30(4):306-12.
17. Anwar B.Bataineh- Sensory nerve impairment following mandibular third molar surgery. J Oral Maxillofacial Surg 2001;59(9):1012-7.
18. Steven B, Graff-Radford, Randolph W. Evans. Lingual nerve injury. Headache 2003; 43:975-83.
19. UK Akal, Z Kucukyavuz, R.Nalcaci, T Yilmaz. Evaluation of gustatory function after third molar removal. Int J Oral Maxillofacial Surg 2004;33(6): 564-8.
20. Linda M. Bartoshuk, Derek J.Snyder, Miriam Grushka, Ann M.Berger, Valerie B.Duffy, John F.Kveton. Taste Damage; Previously Unsuspected Consequences. Chemical Sense 2005;30 supplement(1):218-9.
21. Ana Claudia Amorim Gomes, BelmiroCavalcanti do EgitoVasconcelos. Emanuel Dias de Oliveira e Silva, Luiz Carlos Ferreira da Silva-Lingual nerve damage after Mandibular Third Molar Surgery: A Randomized Clinical Trial. Amer Assoc Oral Maxillofacial Surgeons 2005;63:1443-6
22. Risk factors associated with injury to the inferior alveolar and lingual nerves following third molar surgery — revisited. Brit Dent J 2010;208(10):475-8.
23. W.P.Smith. The Relative risk of neurosensory deficit following removal of the mandibular third molar teeth: the influence of radiography and surgical technique. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2013;115(1):18-24.
24. Deshpande.P, Guledgud MV, Patil K. Proximity of Impacted Mandibular Third Molars to the Inferior Alveolar Canal and Its Radiographic Predictors: A Panoramic Radiographic Study. J Maxillofac Oral Surg 2013;Jun;12(2):145–51.
25. Ghaeminia H, Meijer GJ, Soehardi A, Borstlap WA, Mulder J, Bergé SJ. Position of the impacted third molar in relation to the mandibular canal. diagnostic accuracy of cone beam computed tomography compared with panoramic radiography. Int J Oral Maxillofac Surg 2009;38:964–71.

Amyloidosis

Benazir HussainM[1], Sivakumar M[2]

Graduate Student[1], Senior Lecturer[2], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

Abstract

The term amyloid describes the deposition in the extracellular space of certain proteins in a highly characteristic, insoluble fibrillar form. . Amyloidosis can occur any single organ or associated with other organs . It is formed in in the form of a tumor like lesion or an infiltrative process. In the proper clinical setting, that is, in a patient with chronic inflammatory disease and especially in a patient with multiple myeloma, amyloidosis should be considered as a possible cause of worsening or new symptoms or imaging findings. Occasionally, the radiologic findings may precede the clinical findings, thus providing the radiologist with the opportunity to contribute to the patient's care. However, to make a difference in patient care, the radiologist must be familiar with the diverse imaging findings of amyloidosis as well as the patient's clinical history, which could raise the suspicion of amyloidosis.Amyloidosis describes the various clinical syndromes that occur as a result of damage by amyloid deposits in tissues and organs throughout the body. The clinical significance of amyloid varies enormously, ranging from incidental asymptomatic deposits to localized disease through to rapidly fatal systemic forms that can affect multiple vital organs. Currently available therapy is focused on reducing the supply of the respective amyloid fibril precursor protein and supportive medical care, which together have greatly improved survival Quality of life and prognosis of some forms of hereditary systemic amyloidosis can be improved by liver and other organ transplants. Various new therapies, ranging from silencing RNA, protein stabilizers to monoclonal antibodies, aimed at inhibiting fibril precursor supply, fibril formation or the persistence of amyloid deposits, are in development; some are already in clinical phase.

Key Words*: amyloidosis, dialysis ,lysosome, systemic amyloidosis, nephrotic syndrome.*

Introduction

Amyloidosis is a disorder of protein folding in which various proteins are able to autoaggregate in a highly abnormal fibrillar conformation. Amyloid fibrils accumulate in the extracellular space, and the deposits progressively disrupt the structure and function of tissues and organs throughout the body.[1] Amyloidosis is a rare condition; approximately 500 new cases are referred to the UK National Amyloidosis Centre each year and it has been estimated that 0.5 – 1.0 deaths per 1000 in the UK are due to the most prevalent AL type. Amyloid type is classified according to the fibril protein, and some 25 different proteins are known to form amyloid fibrils in vivo.[2] Deposition of amyloid is diverse, ranging from localized deposits that can be incidental to progressive systemic disease which can be rapidly fatal. Amyloidosis can be inherited or acquired, and its anatomical distribution and natural history vary greatly between, and sometimes within, fibril types. Precise pathological diagnosis and comprehensive clinical evaluation are imperative for appropriate clinical management.The systemic amyloidoses are a group of complex diseases caused by tissue deposition of misfolded proteins that results in progressive organ damage. The most common type, immunoglobulin light chain amyloidosis (AL), is caused by clonal plasma cells that produce misfolded light chains.[3] The purpose of this review is to provide up-to-date information on diagnosis and treatment options for AL amyloidosis. Early, accurate diagnosis is the key to effective therapy, and unequivocal identification of the amyloidogenic protein may require advanced technologies and expertise. Prognosis is dominated by the extent of cardiac involvement, and cardiac staging directs the choice of therapy. Treatment for AL amyloidosis is highly individualized, determined on the basis of age, organ dysfunction, and regimen toxicities, and should be guided by biomarkers of hematologic and cardiac response. Alkylator-based chemotherapy is effective in almost two thirds of patients. Novel agents are also active, and trials are ongoing to establish their optimal use. Treatment algorithms will continue to be refined through controlled trials. Advances in basic research have led to the identification of new drug targets and therapeutic approaches, which will be integrated with chemotherapy in the future[4].

Fibril Formation and Amyloid Proteins

Amyloid fibrillogenesis remains poorly understood. Experiments have shown in vitro that nearly any polypeptide chain can be driven towards misfolding and aggregation given specific conditions,(4) but relatively few proteins are amyloidogenic in vivo. The

polypeptides involved in amyloidosis are structurally diverse in their normal conformation and may be notably rich in b-sheet, a-helix or b-helix.(5) During amyloidogenesis, multimeric proteins dissociate to their monomeric components, and may further be enzymatically cleaved before or during their conversion into amyloid fibrils.(6,7) There are essentially three circumstances in which amyloid deposition occurs. The first is when there is sustained abnormally high abundance of certain proteins that are normally present at low levels, such as serum amyloid A protein (SAA) in chronic inflammation, underlying susceptibility to AA amyloidosis. The second is when there is normal abundance of a normal, but to some extent inherently amyloidogenic protein over a very prolonged period, such as transthyretin in senile amyloidosis (ATTR). The third situation is the presence of an abnormal protein with markedly amyloidogenic structure, such as certain monoclonal immunoglobulin light chains in AL amyloidosis and genetic variants of transthyretin, apolipoprotein AI and fibrinogen Aa chain, etc. in hereditary amyloidosis. Despite the heterogeneity of the various precursor proteins, the morphological structure and histochemical properties of all amyloid fibrils are remarkably similar. The core structure comprises antiparallel b-strands of polypeptide chains lying perpendicular to the long axis of the fibril.(8) On electron microscopy, amyloid fibrils are characteristically straight, non-branching and 7 –10 nm in diameter.(9) Amyloid deposits also contain the non-fibrillar normal plasma protein, serum amyloid P component (SAP),(10) which is bound in a reversible calciumdependent manner to a ligand present on all amyloid fibrils.

Diagnosis of Amyloidosis and Imaging

Amyloidosis is extremely heterogeneous and clinical presentation varies widely depending on which organs are involved. Diagnosis is often made late in the course of the disease, and frequently as an unexpected histological finding when a failing organ is biopsied. Congo red staining of tissue yielding the characteristic apple green birefringence under crossed polarized light remains the gold standard for confirming the presence of amyloid, this pathognomonic optical effect being produced by alignment of the dye molecules along the fibrils. The protein composition of the amyloid fibril, i.e. the type of amyloidosis, must then be ascertained, and this is most accessibly achieved by immunohistochemistry(11-13). However, immunohistochemical staining of amyloid deposits can be confounded by many factors including background staining and loss of antigenic determinants in the fibrillar conformation, especially in the common AL type. Proteomic analyses comprising mass spectrometry on amyloid material cut out from tissue sections by laser capture microscopy

has lately proved to be effective in a large proportion of cases.(14-16) Whereas target organ biopsies are usually diagnostic, amyloid deposition can be patchy and random 'screening' biopsies, e.g. of the gut, are negative in a considerable proportion of cases. Various imaging techniques can make an important contribution to the diagnosis and evaluation of organ involvement in amyloidosis. Of these, only radiolabelled SAP scintigraphy is specific.(17) The normal plasma protein SAP binds reversibly to all types of amyloid fibril, and is present in all amyloid deposits. Intravenous injection of 123I-SAP rapidly equilibrates between the relatively small quantity of endogenous SAP within the circulation and SAP present within the extravascular amyloid deposits. SAP scintigraphy thus enables visceral amyloid to be imaged for diagnostic purposes, and since the method is quantitative, it enables the deposits to be monitored serially. SAP scintigraphy has characterized the dynamic nature of amyloid, and has shown that frequently the deposits gradually regress when the supply of the respective precursor protein is reduced. Limitations include restricted availability, and the inability to image amyloid deposits in small or moving structures such as nerves and the heart. Fortunately, several cardiac imaging modalities yield characteristic and clinically important information regarding myocardial involvement. Echocardiography has long been used to demonstrate thickening of the ventricular walls and valves, and to evaluate the predominant diastolic restrictive abnormality that occurs in cardiac amyloidosis . Cardiac involvement has been defined as a mean left ventricular wall thickness of .12 mm in the absence of hypertension or other causes of left ventricular hypertrophy,(18) although poor echocardiographic windows and interoperator variability are significant limitations. Cardiac magnetic resonance (CMR) imaging has lately fallen into widespread clinical practice, demonstrating very characteristic late gadolinium enhancement in subendocardium or more diffusely.(19,20) Although the role of CMR for monitoring progression or regression of amyloid has yet to be defined, the use of equilibrium CMR may prove to be a useful tool in quantification of amyloid, a technique which has been validated in fibrosis.(21) The biomarkers N-terminal pro brain natriuretic peptide (NT-proBNP) and cardiac troponins are also now widely used to provide information on cardiac involvement, prognosis and response to chemotherapy in AL amyloidosis.(22) Since 5 –10% of systemic amyloidosis is hereditary, genetic testing is often required, but the results must be interpreted in light of other findings, notably immunohistochemical or proteomic typing of the amyloid.(23-26) The clinical phenotype associated with particular mutations may be a classification of systemic amyloidosis by precursor protein Type Fibril protein precursor Clinical syndrome Acquired forms of systemic amyloidosis AA Serum amyloid A protein Reactive systemic amyloidosis

associated with chronic inflammatory diseases AL Monoclonal immunoglobulin light chains Systemic amyloidosis associated with monoclonal plasma cell dyscrasias AH Monoclonal immunoglobulin heavy chains Systemic amyloidosis associated with monoclonal plasma cell dyscrasias Ab2M (DRA) b2-microglobulin Periarticular and, occasionally, systemic amyloidosis associated with long-term dialysis ATTRwt Normal plasma transthyretin Prominent cardiac involvement with progressive heart failure

Hereditary systemic amyloidosis ATTRm Genetically variant transthyretin Autosomal dominant systemic amyloidosis Familial amyloid polyneuropathy AGel Genetically variant gelsolin Autosomal dominant systemic amyloidosis Predominant cranial nerve involvement with lattice corneal dystrophy AFib Genetically variant fibrinogen A alpha chain Autosomal dominant systemic amyloidosis Non-neuropathic with prominent renal involvement AApoAI Genetically variant apolipoprotein AI Autosomal dominant systemic amyloidosis Predominantly non-neuropathic with prominent visceral involvement and slowly progressive renal impairment AApoAII Genetically variant apolipoprotein AII Autosomal dominant systemic amyloidosis Non-neuropathic with prominent renal involvement ALys Genetically variant lysozyme Autosomal dominant systemic amyloidosis Non-neuropathic with prominent visceral involvement, very slowly declining renal function ACys Genetically variant cystatin C Hereditary cerebral haemorrhage with cerebral and systemic amyloidosis Others ALECT2 Leukocyte chemotactic factor 2 Slowly progressive renal amyloid with nephrotic syndrome and liver involvement DRA, dialysis-related amyloidosis . Cardiac involvement has been defined as a mean left ventricular wall thickness of .12 mm in the absence of hypertension or other causes of left ventricular hypertrophy,(18) although poor echocardiographic windows and interoperator variability are significant limitations. Cardiac magnetic resonance (CMR) imaging has lately fallen into widespread clinical practice, demonstrating very characteristic late gadolinium enhancement in subendocardium or more diffusely.(19,20) Although the role of CMR for monitoring progression or regression of amyloid has yet to be defined, the use of equilibrium CMR may prove to be a useful tool in quantification of amyloid, a technique which has been validated in fibrosis.(21) The biomarkers N-terminal pro brain natriuretic peptide (NT-proBNP) and cardiac troponins are also now widely used to provide information on cardiac involvement, prognosis and response to chemotherapy in AL amyloidosis.(22) Since 5 –10% of systemic amyloidosis is hereditary, genetic testing is often required, but the results must be interpreted in light of other findings, notably immunohistochemical or proteomic typing of the amyloid.(23-26).The clinical

phenotype associated with particular mutations may vary, even within a given kindred, and since penetrance is variable, patients with AL amyloidosis can occasionally have an incidental mutation.(27,28) Conversely, some patients with hereditary amyloidosis have a potentially misleading but coincidental monoclonal gammopathy.(28,29)Biochemical analyses are an integral part of the diagnostic process. A plasma cell dyscrasia can be identified in approximately 94% of patients with AL amyloidosis.(30) Monoclonal proteins can be detected by serum and urine electrophoresis and immunofixation, although the fully quantitative high sensitivity serum free light chain (FLC) assay is usually best for serially monitoring progress and response to chemotherapy in AL amyloidosis.(31) Bone marrow examination and skeletal X-ray surveys are required to exclude frank multiple myeloma. It is, however, important to note that incidence of monoclonal gammopathy of undetermined significance occurs in at least 3% among people over 50 years, and demonstration of a plasma cell dyscrasia therefore does not by itself confirm amyloidosis is of AL type. Diagnosis of amyloidosis is thus a multidisciplinary process, encompassing the clinical picture, various imaging modalities, histology, immunohistochemistry, proteomics, haematological and biochemical investigations and genetic analyses.

Localized Amyloid

Localized amyloid deposition results from the local production of fibril precursor proteins.(32)Most clinically significant deposits are AL type, associated with foci of low-grade monoclonal B-cells which secrete monoclonal immunoglobulin light chains in the immediate vicinity.(33,34) The most frequent sites of deposition are the respiratory tract, the urogenital tract, the skin and orbits, but all are rare.(35) There are case reports of amyloid affecting almost any site ranging from intracranial amyloidomas,(36)amyloidoma affecting the larynx and oropharynx(37) to localized amyloid of the vagina.(38) Local resection of the ‘amyloidoma’ can sometimes be curative,(39) but amyloid deposits can recur within the same site or elsewhere in the same tissue. Amyloid deposits that appear to be localized can sometimes be a manifestation of systemic disease. It is therefore important to fully investigate patients in order to exclude systemic amyloidosis.(40) Once established that the amyloid deposit is localized, the management is dictated by the area involved and the degree of symptoms. Due to the extremely rare nature of the disease, management strategies have been somewhat experimental, ranging from radiotherapy,(41) to carbon dioxide laser ablation.(42) If the lesions are not causing symptoms, then clinical surveillance may well be all that is needed. Localized masses of amyloid can be found at insulin injection sites when repeated

administration to the same area has occurred over many years.(43) This form of amyloid stains with antibodies to insulin and has been termed iatrogenic A-Ins type amyloid.

Systemic Amyloidosis

Four major acquired forms of systemic amyloidosis have so far been identified. These are systemic light chain amyloidosis (AL); systemic amyloid A amyloidosis (AA); dialysis-related amyloidosis (DRA); and senile systemic amyloidosis (ATTRwt).

Systemic AL Amyloidosis

AL amyloidosis is the commonest form of amyloid and is thought to be the cause of death in 1/1500 people in the UK. The fibrils are formed of fragments of monoclonal immunoglobulin light chains consisting of all or part of the variable domain (VL).(44) AL amyloidosis is a rare complication of monoclonal gammopathies and can occur in association with any form of monoclonal B-cell dyscrasia.30 The plasma cell proliferation fraction is usually similar to that of monoclonal gammopathy of undetermined signifi- cance (MGUS).(45) Approximately 15% of patients have multiple myeloma and within this group symptomatic myeloma is unusual.(30) Median age at presentation is 50– 60 years and both sexes are equally affected.30 Presentation is extremely variable as almost any organ can be affected except the brain. It is usually diagnosed incidentally on a biopsy. Clinical suspicion of amyloidosis should be raised in any patient with unexplained nephropathy, cardiac failure, peripheral or autonomic failure, hepatomegaly or splenomegaly or any unexplained multisystem disease. Approximately 50% of cases involve the kidneys presenting with proteinuria and frequently nephrotic syndrome. Kidney remains the commonest tissue from which the disease is identified. Patients with AL amyloidosis can develop acquired factor X deficiency, underlying some reluctance to perform biopsies in patients with AL amyloidosis. Factor X assays are important in patients with abnormal clotting, and replacement with fresh frozen plasma prior to biopsy in those who are deficient is recommended.(46) However, overall, the incidence of bleeding complications following renal biopsy in 138 patients with cast nephropathy or amyloid was no different from those without.(47) Cardiac involvement causing heart failure at presentation occurs in 15–30% of cases. Symptoms of cardiac involvement are those of congestive cardiac failure most commonly with progressive breathlessness .The prognosis in untreated AL amyloidosis is very poor with a median survival of only 6 –15 months and a 10-year survival rate of ,5%.30 The prognosis, however, is somewhat dependent on the organs involved with cardiac and autonomic nerve

involvement conferring a particularly poor prognosis. The current management of AL amyloidosis is to aim to suppress the production of the underlying B-cell clone with chemotherapy and in turn halt the production of amyloidotic light chains. Evidence suggests that remission of the underlying clonal disease is associated with preservation of organ function and in some cases remission of amyloid deposits. Early identification and treatment is associated with improved survival.All current therapies in AL amyloidosis have been derived from experience in multiple myeloma, but adverse effects are far more frequent and severe in AL due to the multisystem nature of the disease. It is vitally important that an individual assessment is made for each patient and treatment regimens are tailored accordingly. A variety of therapies have been employed ranging from high-dose autologous stem cell transplantation to oral-based regimens.

Systemic AA Amyloidosis

AA amyloidosis is a rare complication of chronic inflammatory conditions. The fibrils in AA amyloidosis are derived from the circulating acute phase reactant SAA, which is produced by hepatocytes under the transcriptional regulation of pro-inflammatory cytokines.(60) The median plasma concentration of SAA in health is ,3 mg/L, but it can increase more than a thousand-fold during the acute phase response. Longstanding elevation of SAA is a prerequisite to development of AA amyloidosis but it is rare and its incidence varies throughout the world, and for example seems less common in the USA compared with central Europe and Scandinavia.(61-63) The commonest predisposing conditions in the Western world are the chronic inflammatory arthropathies, which account for over 50% of cases. In the developing world, reported cases are mainly associated with underlying infection. Patients with hereditary periodic fever syndromes are especially susceptible, perhaps due to the lifelong nature of these inflammatory diseases, and this risk is substantially increased when there is a family history of AA amyloidosis(64). Other rare causes include Castleman's disease, vasculitis and neoplasias such as lymphoma and mesothelioma. Biopsy and postmortem studies have suggested a prevalence of up to 3 – 6% in rheumatoid arthritis (RA), and 11– 13% in familial Mediterranean fever despite availability of colchicine treatment for the latter.The factors that govern susceptibility to AA amyloidosis in the face of a high SAA concentration are yet to be determined. Polymorphisms in the gene encoding for SAA1 isotype may contribute in part.AA amyloidosis predominantly affects the kidneys, with more than 95% of patients presenting with proteinuria, and around 10% of patients having already reached end-stage renal failure at diagnosis(50-54). Splenic involvement is evident on SAP

scintigraphy almost without exception, and deposits commonly occur in the adrenal gland, liver and GI tract, although usually without associated organ dysfunction. Cardiac and neuropathic involvements are extremely rare.(62) Patients with persistent inflammation frequently develop progressive renal dysfunction and end-stage renal failure within 5– 10 years. Almost 60% have nephrotic syndrome at presentation, which confers a high risk of infection. Acute kidney injury is common and is often non-reversible, emphasizing the need for great care with regard to hypoperfusion, nephrotoxic drugs and surgery. Patient outcome has gradually improved, with a recently reported median survival of 133 months.(62-63) Outcome is poorer in association with older age, lower serum albumin and end-stage renal failure at presentation. Serum SAA concentration has a powerful and modifiable influence on outcome; complete suppression of inflammation in terms of SAA concentration persistently ,5 mg/L is frequently associated with regression of amyloid and preservation of renal function. Treatment will differ according to the nature of the underlying chronic inflammatory disorder, and there has lately been much progress with biological therapies for rheumatoid arthritis, etc., but progressive renal dysfunction remains common and end-stage renal failure occurs in up to a third of patients. Median survival on dialysis is in the order of 4– 5 years, which is similar to that among age-matched non-diabetic patients; renal transplantation has been performed in selected cases with reportedly excellent outcomes.

Dialysis-related Amyloidosis

Dialysis related amyloidosis is a complication of long-term dialysis following endstage renal failure. The underlying fibril is due to b2- -microglobulin (b2M). b2M is the light chain component of the major histocompatibility complex (MHC) class 1 molecule. It is synthesized in all cells that express MHC class 1 molecules. b2M is cleared from the body by the kidney. It is freely filtered by the glomerulus and reabsorbed by the proximal tubular cells. When patients develop end-stage renal failure, b2M accumulates and the circulating concentration rises from normal levels (1 – 2 mg/L) to 50 –70 mg/L(59).

Senile AA Systemic Amyloidosis

It is a disease of the elderly and usually affects men; the fibril is composed of normal wild-type transthyretin. Transthyretin is a transport protein that circulates in a tetrameric form. More than 95% of transthyretin is produced in the liver, with the remainder in the choroid plexus and in the retina.The most common transthyretin mutation involves the substitution of methionine for valine at position 30 (ATTRV30M). This usually presents with sensori-motor

peripheral neuropathy and, unlike most other mutations, cardiac involvement is rare. The disease typically develops by age 30– 40 years in the Portuguese focus, but about 20 years later in the Swedish one.(57) The most common aetiology of FAP in the UK and Ireland is the T60A variant. This usually presents after the age of 50 and often with autonomic symptoms, but cardiac amyloid is virtually always present at diagnosis. Three to four percent of black individuals have the V122I transthyretin variant, which is associated with a predominantly cardiac phenotype that is clinically indistinguishable from senile cardiac amyloidosis; it usually presents after age 60 and is not associated with neuropathy.(60)

TheHereditary Systemic Amyloidosis

Hereditary amyloidosis is a group of diseases due to mutations in different specific proteins. Hereditary systemic amyloidosis can be divided into neuropathic and non-neuropathic forms. The former comprise familial amyloid polyneuropathy (FAP), usually caused by mutations in the transthyretin gene (ATTR) and gelsolin amyloidosis (AGel)(61). Non-neuropathic forms include fibrinogen Aa-chain amyloidosis (AFib), apolipoprotein AI amyloidosis (AApoAI), apolipoprotein AII amyloidosis (A ApoAII) and lysozyme amyloidosis (ALys).

Lysozyme Amyloidosis

Lysozyme amyloidosis was first described by Pepys et al. in 1993. Lysozyme is a bacteriolytic enzyme found in high concentrations in the liver, articular surfaces, saliva and tears. It is highly expressed in granuloctes, monocytes and bone marrow precursor cells. To date, seven amyloidogenic mutations have been found: Ile56Thr, Phe57Ile, Trp64Arg, Asp67His, Trp112Arg, Tyr54Asn and Asp67Gly. Clinical presentation is with very slowly progressive renal failure, usually in the third and fourth decades. Involvement of the liver, lymph nodes, GI tract and spleen also occurs. Sicca syndrome due to salivary amyloid deposition is frequent in patients with Try64Arg and Asp67His variants, and lung and thyroid deposits have been reported in patients with Ile56Thr(54).

Conclusion

Amyloidosis is a diverse group of multisystem diseases in which much progress has been made in recent years. There have been major developments in diagnosis, ranging from scintigraphic imaging methods and cardiac MRI, to improved molecular characterization of amyloid deposits through mass spectrometry and genetic sequencing. Clinical outcomes have improved through advances in chemotherapy, biologic anti-inflammatory agents and organ

transplantation in AL, AA and hereditary amyloidosis, respectively. Assessment of underlying diseases and amyloidotic organ function with biochemical markers such as the serum free light chain assay, SAA measurements and the cardiac biomarker NT-proBNP, have greatly improved the monitoring of patients. A new era of clinical trials to test the first drugs specifically designed to prevent or remove amyloid deposits is upon us, promising a brighter future for patients with these very serious diseases.

Corresponding Author

Sivakumar M

Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Telephone Number- +919443602627

Email Id: drsivaomfs@gmail.com

References

1. Pepys MB. Amyloidosis. In: Frank MM, Austen KF, Claman HN, Unanue ER, eds. Samter's Immunologic Diseases. 5th edn. Boston: Little, Brown and Company, 1994:637 –65

2. Pepys MB. Pathogenesis, diagnosis and treatment of systemic amyloidosis. Philos Trans R Soc Lond B Biol Sci 2001;356:203 – 10

3.Westermark P, Araki S, Benson MD. Nomenclature of amyloid fibril proteins. Amyloid: Int J Exp Clin Invest 1999;6:63 –6

4.Bucciantini M, Giannoni E, Chiti F. Inherent toxicity of aggregates implies a common mechanism for protein misfolding diseases. Nature 2002;416:507 – 11

5. Uversky VN, Fink AL. Conformational constraints for amyloid fibrillation: the importance of being unfolded. Biochim Biophys Acta 2004;1698:131 –53

6. Nielsen L, Khurana R, Coats A. Effect of environmental factors on the kinetics of insulin fibril formation: elucidation of the molecular mechanism. Biochemistry 2001;40:6036 –46

7. Quintas A, Vaz DC, Cardoso I, Saraiva MJ, Brito RM. Tetramer dissociation and monomer partial unfolding precedes protofibril formation in amyloidogenic transthyretin variants. J Biol Chem 2001;276:27207 –13

8.Sunde M, Serpell LC, Bartlam M. Common core structure of amyloid fibrils by synchrotron X-ray diffraction. J Mol Biol 1997;273:729 – 39

9.Jaroniec CP, MacPhee CE, Bajaj VS. High-resolution molecular structure of a peptide in an amyloid fibril determined by magic angle spinning NMR spectroscopy. Proc Natl Acad Sci USA 2004;101:711 –16

10. Pepys MB, RademacherTW, Amatayakul-Chantler S. Human serum amyloid P component is an invariant constituent of amyloid deposits and has a uniquely homogeneous glycostructure. Proc Natl Acad Sci USA 1994;91:56 –106

11. Tennent GA, Lovat LB, Pepys MB. Serum amyloid P component prevents proteolysis of the amyloid fibrils of Alzheimer's disease and systemic amyloidosis. Proc Natl Acad Sci USA 1995;92:4299 –303

12.Kisilevsky R, Fraser P. Proteoglycans and amyloid fibrillogenesis. Ciba Found Symp 1996;199:58 –67

13. Brenner DA, Jain M, Pimentel DR. Human amyloidogenic light chains directly impair cardiomyocyte function through an increase in cellular oxidant stress.2004;94:1008 –10

14.Vrana JA, Gamez JD, Madden B. Classification of amyloidosis by laser microdissection and mass spectrometry-based proteomic analysis in clinical biopsy specimens. Blood 2009;114:4957 –9

15.Sethi S, Theis JD, Leung N. Mass spectrometry-based proteomic diagnosis of renal immunoglobulin heavy chain amyloidosis. Clin J Am Soc Nephrol 2010;5:2180 –7

16. Klein CJ, Vrana JA, Theis JD. Mass spectrometric-based proteomic analysis of amyloid neuropathy type in nerve tissue. J Pharm 2001:12;30-102

17. MB Hawkins PN, Lavender JP, Pepys. Evaluation of systemic amyloidosis by scintigraphy with 123I-labeled serum amyloid P component. N Engl J Med 1990;323:508-13

18.Gertz MA, Comenzo R, Falk RH. Definition of organ involvement and treatment response in immunoglobulin light chain amyloidosis (AL): a consensus opinion from the 10th International Symposium on Amyloid and Amyloidosis. Am J Hematol 2005;79:319 –28

19.Maceira AM, Joshi J, Prasad SK. Cardiovascular magnetic resonance in cardiac amyloidosis. Circulation 2005;111:186 –93

20.Perugini E, Rapezzi C, Piva T. Non-invasive evaluation of the myocardial substrate of cardiac amyloidosis by gadolinium cardiac magnetic resonance. Heart 2006;92:343 –9

21.Flett AS, Hayward MP, Ashworth MT. Equilibrium contrast cardiovascular magnetic resonance for the measurement of diffuse myocardial fibrosis: preliminary validation in humans. J Pharm. 2010;122:138 – 44

22.Tabbibizar R, Maisel A. The impact of B-type natriuretic peptide levels on the diagnoses and management of congestive heart failure. Curr Opin Cardiol 2002;17:340 –5

23.Lachmann HJ, Booth DR, Booth SE. Misdiagnosis of hereditary amyloidosis as AL (primary) amyloidosis. N Engl J Med 2002;346:1786 –91

24.Gillmore JD, Lachmann HJ, Rowczenio D, et al. Diagnosis, pathogenesis, treatment, and prognosis of hereditary fibrinogen A alpha-chain amyloidosis. J Am Soc Nephrol 2009;20:444 –51

25. S, Granel B, ValleixSerratrice J. Lysozyme amyloidosis: report of 4 cases and a review of the literature. Medicine (Baltimore) 2006;85: 66– 73

26.HaagsmaEB, Hawkins PN, Benson MD. Familial amyloidotic polyneuropathy with severe renal involvement in association with transthyretin Gly47Glu in Dutch, British and American-Finnish families. Amyloid 2004;11:44– 4

27. Landau HJ, Comenzo RL, Zhou P. AL amyloidosis in a patient with a T60A TTR mutation. XIth International Symposium on Amyloidosis. Florida: CRC Press, 2008:160 – 162

28.Comenzo RL, Zhou P, Fleisher M, Clark B, Teruya-Feldstein J. Seeking confidence in the diagnosis of systemic AL (Ig light-chain) amyloidosis: patients can have both monoclonal gammopathies and hereditary amyloid proteins. Blood 2006;107:3489 –91

29.Gregorini G, Izzi C, ObiciL.Renalapolipoprotein A-I amyloidosis: a rare and usually ignored cause of hereditary tubulointerstitial nephritis. J Am Soc Nephrol 2005;16:3680 – 6

30. Kyle RA, Gertz MA. Primary systemic amyloidosis: clinical and laboratory features in 474 cases. Semin Hematol 1995;32:45 –59

31.Lachmann HJ,Gallimore R, Gillmore JD. Outcome in systemic AL amyloidosis in relation to changes in concentration of circulating free immunoglobulin light chains following chemotherapy. Br J Haematol 2003;122:78 – 84

32. Pasternak S, White VA, Gascoyne RD. Monoclonal origin of localised orbital amyloidosis detected by molecular analysis. Br J Ophthalmol 1996;80:1013 –7

33.Parathan KK, Kumar M, Kannan R, Chitrambalam P, Volga H. Cardiac amyloidosis in a patient with multiple myeloma. Journal of evolution of medical and dental sciences-jemds. 2015 Oct 8;4(81):14248-52

34. Lewis JE, Olsen KD, Kurtin PJ, Kyle Laryngeal amyloidosis: a clinicopathologic and immunohistochemical review. Otolaryngol Head Neck Surg 1992;106:372 –7

35. Goodman HJB, Bridoux F, Lachmann HJ. Localized amyloidosis: clinical features and outcome in 235 cases. Hematologica 2005;90 (Suppl. 1):203 [abstract]

36. Gandhi D, Wee R, Goyal M. CT and MR imaging of intracerebral amyloidoma: case report and review of the literature. AJNR Am J Neuroradiol 2003;24:519 –22

37. Parmar H, Rath T, Castillo M, Gandhi D. Imaging of focal amyloid depositions in the head, neck, and spine: amyloidoma. AJNR Am J Neuroradiol 2010;31:1165 –70

38. Vhlivanov B, BelovegdovV, Ivanov G, Pea H. Primary localised amyloidosis of the vagina. Aust N Z J Obstet Gynaecol 2008;48:120 – 2

39.Tirzaman O, Wahner-Roedler DL, Malek RS. Primary localized amyloidosis of the urinary bladder: a case series of 31 patients. Mayo Clin Proc 2000;75:1264 –8

40. Shah PL, Gillmore JD, Copley SJ.The importance of complete screening for amyloid fibril type and systemic disease in patients with amyloidosis in the respiratory tract.Sarcoidosis Vasc Diffuse Lung Dis 2002;19:134 – 42

41.Kurrus JA, Hayes JK, Hoidal JR, Menendez MM, Elstad MR. Radiation therapy for tracheobronchial amyloidosis. Chest 1998;114:1489 – 92

42. Nugent AM, Elliott H, McGuigan JA, Varghese G. Pulmonary amyloidosis: treatment with laser therapy and systemic steroids. Respir Med 1996;90:433 –5

43.Shikama Y, Kitazawa J, Yagihashi N. Localized amyloidosis at the site of repeated insulin injection in a diabetic patient. Intern Med 2010;49:397 – 401

44.Comenzo RL, Zhang Y, Martinez C, Osman K, Herrera GA. The tropism of organ involvement in primary systemic amyloidosis: contributions of Ig VL germ line gene use and clonal plasma cell burden. Blood 2001;98:714 – 20

45.Witzig TE, Timm M, Larson D, Therneau T, Greipp PR. Measurement of apoptosis and proliferation of bone marrow plasma cells in patients with plasma cell proliferative disorders. Br J Haematol 1999;104:131 –7

46.Choufani EB, Sanchorawala V, Ernst T. Acquired factor X deficiency in patients with amyloid light-chain amyloidosis: incidence, bleeding manifestations, and response to high-dose chemotherapy. Blood 2001;97:1885 – 7

47. Fish R, Pinney J, Jain P. The incidence of major hemorrhagic complications after renal biopsies in patients with monoclonal gammopathies. Clin J Am Soc Nephrol 2010;5:1977 – 80

48.Dubrey SW, Cha K, Anderson J. The clinical features of immunoglobulin light-chain (AL) amyloidosis with heart involvement. QJ Med 1998;91:141 –57

49. Levine RA. Amyloid disease of the liver.Correlation of clinical, functional and morphologic features in forty-seven patients. Am J Med 1962;33:349 – 57

50. Kyle RA, Bayrd ED. Amyloidosis: review of 236 cases. Medicine 1975;54:271 – 99

51. Levy M, Fryd CH, Eliakim M. Intrahepatic obstructive jaundice due to amyloidosis of the liver. A case report and review of the literature. Gastroenterology 1971;61:234 –8

52.Pinney JH, Lachmann HJ, Bansi. Outcome in renal AL amyloidosis following chemotherapy. J Clin Oncol 2011;29:674 – 81

53. Klein AL, Hatle LK, Burstow DJ. Comprehensive Doppler assessment of right ventricular diastolic function in cardiac amyloidosis. J Am Coll Cardiol 1990;15:99– 108

54. Goodman HJ,Gillmore JD, Lachmann HJ. Outcome of autologous stem cell transplantation for AL amyloidosis in the UK. Br J Haematol 2006;134:417 – 25

55.Dispenzieri A, Lacy MQ, Katzmann JA. Absolute values of immunoglobulin free light chains are prognostic in patients with primary systemic amyloidosis undergoing peripheral blood stem cell transplantation. Blood 2006;107:3378 –83

56. Gertz MA, Lacy MQ, Dispenzieri A. Myeloablative chemotherapy with stem cell rescue for the treatment of primary systemic amyloidosis: a status report. Bone Marrow Transplant 2000;25:465 –70

57. Kumar SK, Gertz MA, Lacy MQ. Recent improvements in survival in primary systemic amyloidosis and the importance of an early mortality risk score.Jcdr. 2011;86:12 –18

58. Skinner M, Sanchorawala V, Seldin DC. High-dose melphalan and autologous stem-cell transplantation in patients with AL amyloidosis: an 8-year study. Ann Intern Med 2004;140:85 – 93

59.Sattianayagam PT. Solid organ transplantation in AL amyloidosis. Am J Transplant 2010;10:2124 – 31

60.Urieli-Shoval S, Linke RP, Matzner Y. Expression and function of serum amyloid A, a major acute-phase protein, in normal and disease states. Curr Opin Hematol 2000;7:64 –9

61.Parathan, Mahendra Kumar.Cardio Amyloidosis in patients with multiple myeloma.Ann Clin Biochem 1998;35:745 – 53

62.Lachmann HJ, Goodman HJB, Gilbertson JA.Natural history and outcome in systemic AA amyloidosis. N Engl J Med 2007;356:2361 –71

63.De Beer FC, Mallya RK, Fagan EA. Serum amyloid A protein (SAA) concentration in inflammatory diseases and its relationship to the incidence of reactive systemic amyloidosis. Lancet 1982;2:231 – 4

64. Bhavani.G , Systemic amyloidosis ,J Pharm.2015,12: 360-362

AnInsight ofIronDeficiencyAnaemiainAdolescents

Hansini B. Ajantha[1],S. Pavithra Priyadarshoni[2], Gayatri Devi M[3].

Graduate Student[1],Tutor[2], Department of Public Health Dentistry, Assistant Professor[3], Department of Physiology.

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Iron is crucial for human biologic functions, including respiration, energy production, DNA synthesis, and cell proliferation. The human body has evolved to conserve iron inways, including the recycling of iron after the breakdown of red cells and the retention of iron. Since excess levels of iron can be toxic, absorption of iron is limited to 1 to 2 mg daily, and most of the iron needed daily, about 25 mg is provided through recycling by macrophages that phagocytose senescent erythrocytes. The two mechanisms are controlled by the hormone hepcidin, which maintains total iron content within normal ranges, avoiding both iron deficiency and excess. Iron deficiency refers to the reduction of iron stores that precedes iron deficiency anemia or persists without progression. Iron-deficiency anemia is a more severe condition in which low levels of iron are associated with anemia and the presence of microcytic hypochromic red cells. This review considers iron deficiency and its anemia in light of advances in the understanding of systemic iron homeostasis and examines causes, pathophysiological featuresand treatment options in adolescents.

KeyWords: *Anaemia, Iron deficiency, Adolescence, Dietary requirements, Treatment.*

Introduction

World Health Organization defines adolescence as a period of human development characterized by the transition between their childhood and adult life with somatic, psychological and social changes. Teenagers present a higher demanding of nutrients reasoned by the enlargement of total blood volume and iron pool, in consequence of an increase of thin body mass quantity. In addition, girls have a mineral loss due to the

menstrual cycle. Thus, iron deficiency is nearly almost twice higher during puberty(1). A 30% prevalence of iron deficiency anaemia, at a minimum, has been noted among children, adolescents, and women in non-industrialized countries(2). Iron deficiency anaemia is considered to be one of the main public health issues throughout the world. Insufficient dietary intake and absorption of iron is the top responsible for malnutrition. Even mildly, it is harmful to the health since it is associated to damages in productive capability, cognitive development and immune competence of individuals(3). Adolescent pregnancies are also at increased risk of severe anaemia, preterm deliveries, still births, and neonatal deaths, due to competition between nutritional requirements of the developing foetus and the mother's requirement for continuing growth(4). Knowledge of the degree and main risk factors of anaemia in adolescence is of public health importance as this is a window of opportunity for school based interventions to improve adolescent health(5).

ImportanceofIron

Iron is a significant element for most life on Earth, including human beings. Iron is needed forcomplex processes that continuously take place on a molecular level, which are indispensable to human life. Iron is an essential component of haemoglobin, myoglobin, and many enzymes in cellular metabolism and DNA replication and repair. It also plays a crucial role in the development of the central neurological system(6),(7), autoimmune system (8),(9)endocrine system- thyroid regulation (10),(11)and cardiovascular system (12). In the development of the brain, iron accounted for the myelination of white matter and the development and functioning of the different neurotransmitter systems, including the dopamine, nor epinephrine, and serotonin systems (13),(14),(15).

IronDistribution in HumanBody

The human body contains approximately 3–5 g of iron, distributed as illustrated in Fig. 1. The majority of body iron (~60–70%) is utilized within haemoglobin in circulating red blood cells. Approximately 20–30% of body iron is stored in hepatocytes and in reticuloendothelial macrophages in the liver. The remaining body iron is primarily localized in myoglobin, cytochromes, and iron containing enzymes. A healthy individual absorbs daily 1–2 mg of iron from the diet, which compensates nonspecific iron losses by cell desquamation in the skin and the intestine. In addition, menstruating women physiologically lose iron from the

blood. Erythropoiesis requires approximately 30 mg iron/day, which is mainly provided by the recycling of iron via reticuloendothelial macrophages(16),(17),(18),(19),(20).

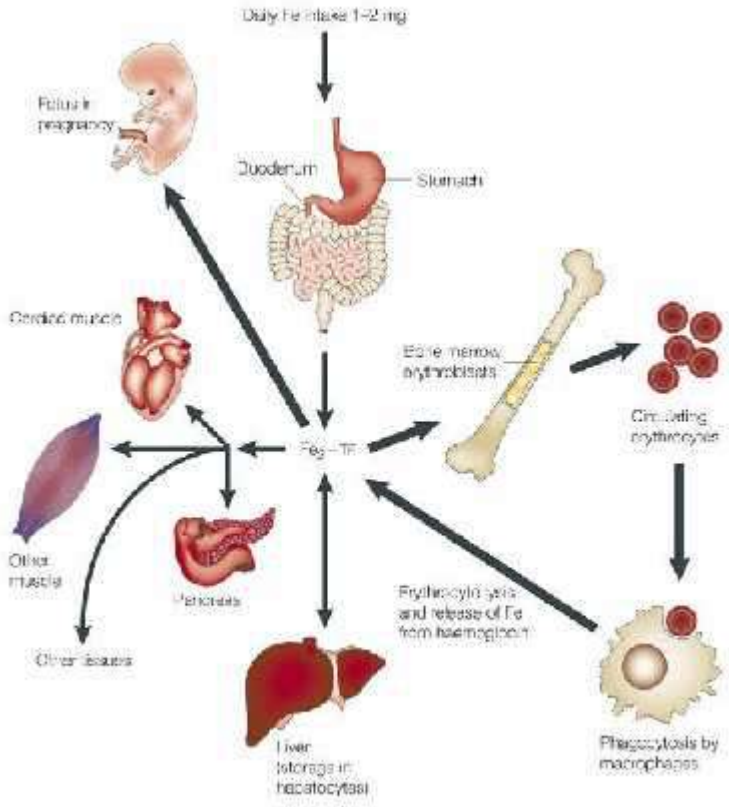


Figure 1: Iron distribution in human body

MechanismofAdaptationofIronDeficiency

The mechanisms of adaptation to iron deficiency are centered on the suppression of thehormone hepcidin and tissue hypoxia that develops consequent to anemia. The production of erythropoietinby the kidney increases in response to enhanced levels of hypoxia-inducible factor 2α (HIF-2α). As a consequence of the stimulation of erythropoietin, erythropoiesis is increased and hypochromic microcytic red cells are produced due to the low availability of iron. Senescent red cells are destroyed by macrophages and the iron found in those cells is recycled. The increase in erythropoiesis suppresses the production of hepcidin. HIF-2α increases the expression of the duodenal divalent metal transporter 1 (DMT1)(21) on the apical surface of enterocytes to increase the transfer of dietary iron from the lumen to enterocytes. Hepcidin levels are depressed in response to a reduction in the physiologic signals that maintain its production (e.g., increases in levels of iron-bound transferrin and in

the iron content of the liver),(22) to the increased activity of the inhibitor trans membrane protease, serine 6 (TMPRSS6),(23) to the reduction in levels of the activator bone morphogenetic protein 6 (BMP6), and to increased inhibition from erythropoietin-stimulated erythropoiesis. Ferroportin (FPN), which is no longer being degraded because of the low levels of hepcidin, exports the available iron across the enterocyte basal membrane and from macrophage stores to the circulation(24). Once stores are exhausted, levels of circulating iron decrease, even if absorption from the lumen is increased. Reduced levels of iron in the liver trigger increases in the synthesis of the iron carrier transferrin (referred to as apotransferrin when not bound to iron), further decreasing levels of iron-bound transferrin, the ligand of the transferrin receptor. Consequently, the uptake of iron from transferrin receptors by all cells and organs (eg. skeletal muscles and the heart) is reduced.

DietarySourcesof Iron

The most common dietary sources of iron in diets of adolescents included ready-to-eat cereal, bread, and beef. The availability of dietary iron for absorption and utilization by the body varies by its form. Heme iron, which is found in meat, fish, and poultry, is highly bio available while non heme iron, found predominantly in grains, is much less so. More than 80% of the iron consumed is in the form of non heme iron. Bioavailability of non heme iron can be enhanced by consuming it with heme sources of iron or vitamin C. Because the absorption of iron from plant foods is low, vegetarians need to consume twice as much iron to meet their daily requirement (25).

DietaryRequirementsforAdolescents

Iron is vital for transporting oxygen in the bloodstream and for preventing anaemia. For both male and female adolescents, the need for iron increases with rapid growth and the expansion of blood volume and muscle mass. Iron needs are highest during the adolescent growth spurt in males, onset of mensuration in girls and after menarche in women. The dailyrequireddietary allowance for iron is 8 mg/day for 9-13 year olds, 11 mg/day for males ages 14-18 and 15 mg/day for females ages 14-18 (26).

CausesofIronDeficiencyAnaemia

Poverty, malnutrition and famine are self-explanatory causes of anemia in the people living with iron deficiency in developing countries, especially children and pregnant women. In

addition, a cereal-based diet decreases iron bioavailability because phytates in grains sequester iron in a poorly absorbable complex. Other common causes include hookworm infections and schistosomiasis, which cause chronic blood loss(27). Strict vegan and vegetarian diets, mal absorption, and chronic blood loss resulting from heavy menstrual losses are well-known causes of iron-deficiency anemia in developed countries Chronic blood loss from the gastrointestinal tract, including occult blood may reveal the presence of benign lesions. Persons who donate blood regularly are also at risk for iron deficiency, and their iron levels should be monitored. Non-steroidalanti-inflammatory drugs and anticoagulants may contribute to blood loss, and proton-pump inhibitors are a frequently overlooked cause of impaired iron absorption(28). In developing countries, low iron intake combined with intestinal infections with nematodes may result in severe anemia, especially in young children and adolescents. In end-stage kidney disease, iron-deficiency anemia results from blood loss during dialysis, reduced hepcidin clearance, inflammation, and certain drugs (e.g. Proton-pump inhibitors and anticoagulants). In elderly persons, the prevalence of anemia correlates with advanced age and multiple related conditions, including iron deficiency, inflammatory disorders, decreased levels of erythropoietin, and cancer(29). Obesity is associated with mild iron deficiency because of subclinical inflammation, increased hepcidin levels, and decreased iron absorption(30).

ClinicalFindings

Iron-deficiency anemia is chronic and frequently asymptomatic and thus may often go undiagnosed. Weakness, fatigue, poor concentration, and poor work productivity are nonspecific symptoms associated with low delivery of oxygen to body tissues and decreased activity of iron-containing enzymes. The extent to which these non-hematologic effects of iron deficiency are manifested before anemia develops is unclear. Iron deficiency is also associatedwithpoor cognitive performance and delayedmental and motor development in children. Severe iron-deficiency anemia in pregnancy is associated with an increased risk of preterm labor, low neonatal weight, and increased newborn and maternal mortality. Iron deficiency may predispose a person to infections, precipitate heart failure, and cause restless leg syndrome. In patients with heart failure, iron deficiency has a negative effect on the quality of life, irrespective of the presence of anemia(31).

DiagnosticInvestigations

The diagnosis of anemia is made after confirmation of a reduced blood haemoglobin concentration as shown by a full blood count. Thresholds to define anemia depend on age, sex, pregnancy, altitude, and smoking. Diagnosis of iron deficiency is somewhat complex, and use of several iron status indicators in combination seems to provide the best assessment of iron insufficiency. Several laboratory markers of iron status are available. First, red cell indices on full blood counts might show a reduced mean cell haemoglobin, which corresponds to hypochromia, and a reduced mean cell volume, corresponding to microcytosis. Mean cell haemoglobin and volume are inexpensive, widely available, and sensitive measures, but become abnormal only in longstanding iron deficiency. These measures are also decreased in several chronic disease states, including haemoglobinopathies such as thalassaemia or sideroblastic anemia. In the absence of inflammation, serum ferritin measurement is the most specific test that correlates with total body iron stores. It is a universally available and standardized measurement(32). Iron deficiency is diagnosed below the cutoff of 15 μg/L in patients older than 5 years. Ferritin concentrations are increased independently of iron status in acute and chronic inflammatory disorders, malignant disease, and liver disease—a serious diagnostic limitation. In these situations, patients with a ferritin concentration of 50 μg/L or higher could still be iron deficient. In chronic kidney disease, cutoff of 100 μg/L have been suggested and 200 μg/L in case of haemodialysis. Other assays can be helpful in these disorders, such as serum iron and serum transferrin concentrations or total iron-binding capacity, which is needed to calculate the transferrin saturation. In cases of iron deficiency, serum iron is reduced and total iron-binding capacity is increased, resulting in a substantial reduction in transferrin saturation (i.e., the ratio of serum iron to total iron-binding capacity). The threshold of 16% is generally used to screen for iron deficiency, but a threshold of 20% is used in the presence of inflammation. Serum soluble transferrin receptors (sTfR) derive from proteolysis of the membrane transferrin receptor. This process shows tissue iron deficiency and inversely the amount of iron available for erythropoiesis. In case of iron deficiency, synthesis of transferrin receptors is increased, leading to a corresponding increase in sTfR. A substantial advantage of measurement of sTfR compared with other assays is that sTfR concentrations are not affected by inflammation(33). In routine practice, measurement of sTfR is not needed for a diagnosis of iron deficiency anaemia. The ratio between these receptors and the logarithm of serum ferritin (i.e., the sTfR–F index) seems to discriminate chronic disease better than either test individually(34). When the sTfR–F index

is low, anemia is probably caused by chronic disease. When it is high, iron deficiency is probably the major cause of anemia(35). Bone marrow aspiration is still thought of as the gold standard for diagnosis of iron deficiency. It is not affected by inflammation and is highly specific, but is invasive, uncomfortable for the patient, expensive, and affected by recombinant human erythropoietin. Thus, bone marrow aspiration is reserved for very specific cases, when other techniques are negative or conflicting. In cases of iron depletion, zinc transport across the intestine increases. Thus, an increased concentration of zinc protoporphyrin in erythrocytes (>80 μg/dL) is associated with iron deficiency anemia. Measurement of the proportion of circulating hypochromic red cells as a proportion of total red blood cells is the most sensitive marker of iron deficiency in patients with chronic kidney disease-6% is the cutoff(36). Unfortunately, a fresh blood sample is needed for this analysis, and automated analysers are not widely available. Reticulocyte haemoglobin content is a very early indicator of iron status, and shows available iron for erythropoiesis during the 3–4 days before measurement(37). A reticulocyte haemoglobin content of less than 27·2 pg is diagnostic. But access to this assay is poor, and false normal values can occur in patients with raised mean cell volumes or thalassaemia(38).

TreatmentforIronDeficiencyAnaemia

The treatment modalities for managing iron deficiency anaemia will depend on the underlying cause. Once the cause of iron deficiency anaemia has been ascertained, either oral or parenteral iron therapy is commonly prescribed to correct the deficiency. Oral iron therapy is usually adequate for most patients; it is an efficient, well tolerated and cost-effective way to replace iron stores. Historically, ferrous sulphate has been used to treat Iron Deficiency Anaemia because it is better absorbed by the gastrointestinal tract. Parenteral iron therapy is necessary for patients intolerant or unresponsive to oral iron supplementation(39). Historically, parenteral iron therapy has been used with caution because of its anaphylactic potential. Despite the introduction of newer intravenous iron preparations with improved safety profiles, practitioners seem hesitant to administer intravenous iron (40).

Oral Iron Therapy

The oral administration of iron is a convenient, inexpensive, and effective means of treatingpatients. Among the various iron preparations on the market, iron sulfate is the most commonly used. The recommended daily iron dosage for adults with iron deficiency is 100 to

200 mg of iron and that for children is 3 to 6 mg per kilogram of body weight for a liquid preparation. The supplement should be administered in divided doses without food. The addition of vitamin C may improve absorptionof iron. The low hepcidin levels in patients with iron-deficiency anemia ensure effective iron absorption and the rapid recovery of hemoglobin levels; however, 3 to 6 months of treatment are required for the repletion of iron stores and the normalization of serum ferritin levels. Long-term use of oral iron is limited by side effects, including nausea, vomiting, constipation, and metallic taste; these side effects are frequent and, although not severe, are often worrisome to patients. If treatment with oral iron fails, the reasons may include premature termination of treatment, lack of compliance with the regimen or discontinuation by the patient, or a truly refractory response to treatment. In the latter case, other specific treatments, such as the eradication of infection with Helicobacter pylori or the introduction of a gluten-free diet in patients with celiac disease, may restore the capacity for iron absorption and eliminate the need for supplementation in some patients(41). There are no known markers that can be used to predict which patients will or will not have a response to oral iron therapy.

Parenteral Iron Therapy

The possibility of hypersensitivity reactions (including anaphylaxis) to high-molecular-weight iron dextran has traditionally limited the indications for the intravenous administration of iron. Newly approved, safer iron formulations are modifying this clinical practice. Becausethe use of intravenous iron circumvents the problem of iron absorption, it is more effective and increases hemoglobin levels more quickly than oral iron. Another advantage is that in some patients the total dose required (up to 1000 mg) can be provided in a single infusion. The dose needed is calculated with this formula: body weight in kilograms × 2.3 × hemoglobin deficiency (target hemoglobin level − patient hemoglobin level) + 500 to 1000 mg iron for the repletion of iron stores. The cost of parenteral iron therapy is high, but the number of hospital or clinic visits that are required is significantly decreased. Intravenous administration is also preferred when a rapid increase in hemoglobin level is required or when iron-deficiency anemia caused by chronic blood loss cannot be controlled with the use of oral iron. Active inflammatory bowel disease is an emerging indication for the use of intravenous iron. Intravenous iron is essential in the management of anemia in patients with chronic kidney disease who are receiving dialysis and treatment with erythropoiesis

stimulating agents. The addition of iron supplementation may eliminate or delay the need for these agents in some patients with chronic kidney disease who are not receiving dialysis(42).

Conclusion

The prevalence of iron deficiency anaemia still remains the most common nutritional deficiency throughout the world that negatively impacts on health and development. We must move beyond current ideology, and learn how to better assess those populations at risk for the development of iron deficiency regardless of concurrent medical conditions. Evidence-based practice guidelines need to include diagnostic measures that identify changes in iron status early to avoid progression to iron deficiency anaemia, and specific management goals that include treatment strategies including what constitutes a favourable response to irontherapy along with timelines for treatment.

Corresponding Author

S. PavithraPriyadarshoni

Tutor, Department of Public Health Dentistry,

Reader,Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id: drpavithraphd88@gmail.com

Contact Number: 9176222104

References

1. Carmen Lúcia de Almeida Santos, Marco Akerman, OdivalFaccenda, Lourdes Conceição Martins, Lígia de FátimaNóbregaReato; Iron deficiency during pubertal growth spurt; Journal of Human Growth and Development 2012; 22(3): 341-347
2. Mu-Hong Chen, Tung- Ping Su, Ying-Sheue Chen, Ju-Wei Hsu, Kai-Lin Huang, Wen-Han Chang, Tzeng-Ji Chen and Ya-Mei Bai Chen. Association between psychiatric disorders and iron deficiency anaemia among children and adolescents; BMC Psychiatry 2013, 13:161

3. Devi K, Priya J. Deleterious Functional Impact of anaemia on adolescents: A Survey study. Asian J Pharm Clin Res. 2016; 9(5):150-2.

4. Naeye RL (1981): Teenaged and pre-teenaged pregnancies: consequences of the fetal–maternal competition for nutrients. Pediatrics 67, 146–150.

5. T Leenstra, SK Kariuki, JD Kurtis, AJ Oloo, PA Kager and FO terKuile. Prevalence and severity of anemia and iron deficiency: cross-sectional studies in adolescent schoolgirls in western Kenya; European Journal of Clinical Nutrition 2004: 58,681–691

6. Krebs NF: Dietary zinc and iron sources, physical growth and cognitive development of breastfed infants. J Nutr 2000, 130(2S Suppl):358S–360S.

7. Lind T, Lonnerdal B, Stenlund H, Gamayanti IL, Ismail D, Seswandhana R, Persson LA: A community-based randomized controlled trial of iron and zinc supplementation in Indonesian infants: effects on growth and development. Am J Clin Nutr 2004, 80(3):729–736.

8. Joynson DH, Walker DM, Jacobs A, Dolby AE: Defect of cell-mediated immunity in patients with iron-deficiency anaemia. Lancet 1972,
2(7786):1058–1059.

9. Srikantia SG, Prasad JS, Bhaskaram C, Krishnamachari KA: Anaemia and immune response. Lancet 1976, 1(7973):1307–1309.

10. Beard JL, Borel MJ, Derr J: Impaired thermoregulation and thyroid
function in iron-deficiency anemia. Am J Clin Nutr 1990, 52(5):813–819.

11. Zimmermann MB: The influence of iron status on iodine utilization and thyroid function. Annu Rev Nutr 2006, 26:367–389.

12. Toxqui L, De Piero A, Courtois V, Bastida S, Sanchez-Muniz FJ, Vaquero MP: Iron deficiency and overload. Implications in oxidative stress and cardiovascular health. NutrHosp 2010, 25(3):350–365.

13. Beard J: Iron deficiency alters brain development and functioning. J Nutr 2003, 133(5 Suppl 1):1468S–1472S.

14. Burhans MS, Dailey C, Beard Z, Wiesinger J, Murray-Kolb L, Jones BC, Beard JL: Iron deficiency: differential effects on monoamine transporters. NutrNeurosci 2005, 8(1):31–38.

15. Parks YA, Wharton BA: Iron deficiency and the brain. ActaPaediatrScandSuppl 1989, 361:71–77.

16. Andrews, N.C., 1999. Disorders of iron metabolism. N. Engl. J. Med. 341, 1986 – 1995.

17. Green R., Disorders of iron metabolism. N. Engl. J. Med. 2000 Apr 27; 342(17):1293

18. Ponka, P., Beaumont, C., Richardson, D.R., 1998. Function and regulation of transferrin and ferritin. Semin. Hematol. 35, 35 – 54.

19. G. Papanikolaou, K. Pantopoulos; iron metabolism and toxicity; Toxicology and Applied Pharmacology 202 (2005) 199–211

20. Iron homeostasis: insights from genetics and animal models; Nancy C. Andrews; Nature Reviews Genetics 1, 208–217 (2000)

21. Mastrogiannaki MMP, Matak P, Peyssonnaux C. The gut in iron homeostasis: role of HIF-2 under normal and pathological conditions. Blood 2013; 122: 885-92.

22. Camaschella C. Iron and hepcidin: a story of recycling and balance. Hematology Am SocHematolEduc Program 2013; 2013: 1-8.

23. Zhang AS, Anderson SA, Wang J, Yang F, DeMaster K, Ahmed R, Nizzi CP, Eisenstein RS, Tsukamoto H, Enns CA. Suppression of hepatic hepcidin expression in response to acute iron deprivation is associated with an increase of matriptase-2 protein. Blood 2011; 117: 1687-99.

24. Nemeth E, Tuttle MS, Powelson J, Vaughn MB, Donovan A, Ward DM, Ganz T, Kaplan J. Hepcidin regulates cellular iron efflux by binding to ferroportin and inducing its internalization. Science 2004; 306: 2090-3

25. Managers; World Health Organization - 2001 - apps.who.int

26. Guidelines for adolescent nutrient services (2005): Nutrition needs for adolescents: Mary Story and Jamie Stang; chapter 3, 21-34.

27. Kassebaum NJ, Jasrasaria R, Naghavi M, Wulf SK, Johns N, Lozano R, Regan M, Weatherall D, Chou DP, Eisele TP, Flaxman SR, Pullan RL, Brooker SJ, Murray CJ. A systematic analysis of global anemia burden from 1990 to 2010. Blood 2014; 123: 615-24.

28. Heidelbaugh JJ. Proton pump inhibitors and risk of vitamin and mineral deficiency: evidence and clinical implications. TherAdv Drug Saf 2013; 4: 125-33.

29. Bach V, Schruckmayer G, Sam I, Kemmler G, Stauder R. Prevalence and possible causes of anemia in the elderly: a crosssectional analysis of a large European university hospital cohort. ClinInterv Aging 2014; 9: 1187-96.

30. Aigner E, Feldman A, Datz C. Obesity as an emerging risk factor for iron deficiency. Nutrients 2014; 6: 3587-600.

31.Stefan D. Anker, Josep Comin Colet, Gerasimos Filippatos, Ronnie Willenheimer, Kenneth Dickstein, Helmut Drexler, Thomas F. Lüscher, Boris Bart, Waldemar Banasiak,

Joanna Niegowska, Bridget-Anne Kirwan, Claudio Mori. Ferric carboxymaltose in patients with heart failure and iron deficiency. N Engl J Med 2009; 361: 2436-48.

32. Jagannathan N, Thiruvengadam C, Ramani P, Premkumar P, Natesan A, Sherlin HJ.; Salivary ferritin as a predictive marker of iron deficiency anemia in children. Journal of Clinical Pediatric Dentistry: September 2012, Vol. 37, No. 1, 25-30.

33. Mast AE, Blinder MA, Gronowski AM, Chumley C, Scott MG. Clinical utility of the soluble transferrin receptor and comparison with serum ferritin in several populations. ClinChem1998; 44:45–51.

34. Cooke KS, Hinkle B, Salimi-Moosavi H, et al. A fully human anti-hepcidin antibody modulates iron metabolism in both mice and nonhuman primates. Blood 2013; 122: 3054–61.

35. Punnonen K, Irjala K, Rajamäki A. Serum transferrin receptor and its ratio to serum ferritin in the diagnosis of iron deficiency. Blood 1997; 89: 1052–57.

36. Tessitore N, Solero GP, Lippi G, et al. The role of iron status markers in predicting response to intravenous iron in haemodialysis patients on maintenance erythropoietin. Nephrol Dial Transplant 2001; 16: 1416–23.

37. Brugnara C, Schiller B, Moran J. Reticulocyte hemoglobin equivalent (ret He) and assessment of iron-deficient states. Clin Lab Haematol2006; 28: 303–08.

38. Cook JD. Diagnosis and management of iron-deficiencyanaemia. Best Pract Res ClinHaematol2005; 18: 319–32.

39. Clark SF. Iron deficiency anemia. NutrClinPract 2008; 23:128–141.

40. Auerbach M, Goodnough LT, Picard D, et al. The role of intravenous iron in anaemia management and transfusion avoidance. Transfusion 2008; 48:988–1000.

41. Hershko C, Camaschella C. How I treat unexplained refractory iron deficiency anemia. Blood 2014; 123: 326-33.

42. Bailie GR, Larkina M, Goodkin DA, et al. Variation in intravenous iron use internationally and over time: the Dialysis Outcomes and Practice Patterns Study (DOPPS). Nephrol Dial Transplant 2013; 28: 2570-9.

AnOverviewofOralCanceranditsClinicalSignificance

Hansini B. Ajantha[1],Pavithra Priyadarshoni S[2],Gayatri Devi M[3]

Graduate Student[1],Tutor[2], Department of Public Health Dentistry,Assistant Professor[3], Department of Physiology,

Saveetha Dental College, Saveetha Institute of Technical and Medical Sciences, Saveetha University, Chennai, India

Abstract

Cancer is a disease of the cells in the body, which multiply at an abnormal rate. There are many different types of cancer which arise from different types of cell. Mouth cancer or oral cancer can occur in any part of the mouth, like, on the tongue's surface, in the lips, inside the cheek, in the gums, in the roof and floor of the mouth, in the tonsils, and also in the salivary glands. Oral cancers are part of a group of cancers which is commonly referred to as head and neck cancers. Brain cancer is a cancer category unto itself and is not included in the head and neck cancer group. Most commonly, oral cancers are found in the tongue and in the floor of the mouth. Anyone can get oral cancer but the risk is higher if you are male over age of 40 and use tobacco or alcohol or have a familial history of head or neck cancer. Frequent sun exposure is also a risk for lip cancer. This article gives an insight into oral cancer and its relevance to clinical practice.

Key Words:*Oral cancer, Malignancy, RiskFactors, Tobacco, Biopsy*

Introduction

Oral cancer is one of the ten most common malignancy in the world. According to the Oral Cancer Foundation, someone dies from oral cancer every hour of every day in the United States alone. Over 300,000 new cases of oral cancer are being diagnosed every year, worldwide (1). This serious condition which pertains to the mouth, lips or throat is often highly curable, if diagnosed and treated in the early stages.Mouth or Oral cancer is a sub type of head and neck cancer. It is described as any cancerous tissue growth located in the oral cavity. It may arise as a primary lesion originating in any of the oral tissues or by metastasis from a distant site of origin, or extension from a neighboring anatomic structure, such as the nasal cavity (2). Oral cancer can originate in any of the tissues of the mouth with varied histology types such as teratoma, adenocarcinoma from a major or minor salivary gland, lymphoma from lymphoid tissue, or melanoma from the melanin pigment producing cells of the oral mucosa. 90% of oral cancer are squamous cell carcinomas originating from the tissues that line the mouth and lips. Oral or mouth cancer most commonly involves the tongue and it may also occur on the floor of the mouth, cheek lining, gingiva, lips, or palate/roof of the mouth (3).

SignsandSymptoms

The most common symptoms of oral cancer are a sore or ulcer in the mouth that does not heal in a period of time, and pain and bleeding in the mouth that does not subside easily. In many cases, changes are seen in the oral mucosa before the cancer develops. A precancerous condition or premalignant lesion, is a term used to describe certain conditions or lesions involving abnormal cells which are associated with an increased risk of developing into cancer, which includes oral submucous fibrosis, leukoplakia, erythroplakia, actinic keratosis, oral lichen planus, oral candidiasis, syphilitic glossitis and carcinoma in situ. This means that early treatment of these changes will actually prevent a cancer from developing. (4) Some characteristic signs of oral cancer are, a lump on the lip, tongue or in the mouth or throat, unusual bleeding or numbness in the mouth, pain when chewing or swallowing, poor periodontal condition or dentures not fitting properly, a change in the voice or speech problems and weight loss. If the cancer spreads to other parts of the body, various other symptoms can develop. All of these symptoms can be due to

other conditions, so tests are needed to confirm the diagnosis. (5) Cancer begins with a change in the structure of DNA. DNA provides our cells with a basic set of instructions, such as when to grow and reproduce. A change in DNA structure is known as a mutation, and it can alter the instructions that control cell growth. This means that cells continue to grow instead of stopping when they should. This causes the cells to reproduce in an uncontrollable manner, resulting in a lump of tissue called a tumor.(6,7)There are two ways mouth cancer can spread, directly – the cancer can spread from the mouth and into nearby tissues, such as surrounding skin or into the jaw and through the lymphatic system. The lymphatic system is a series of glands or nodes located throughout the body, which produce many specialized cells, needed by the immune system to fight infection. Oral cancer that spreads to another part of the body is known as metastatic oral cancer. (8, 9)

RiskFactors

The most commonly known risk factors that cause oral cancer include:

Smoking and Alcohol

The two leading causes of oral cancer are smoking cigarettes or other tobacco products, such as pipes or cigars and consuming too much alcohol. Both of these substances are carcinogenic, which means they contain chemicals that can damage the DNA in cells and lead to cancer. (10) The risk of oral cancer increases significantly in those who smokes and drinks heavily. Research has shown that if they smoke 40 cigarettes a day, but do not drink alcohol, they are five times more likely to develop oral cancer than someone who does not drink or smoke. If they do not smoke, but consume an average of 30 pints of alcohol a week, their risk also increases by a factor of five.(11) Smoking more than 40 cigarettes a day and consuming an average of 30 pints of alcohol a week, they are 38 times more likely to develop oral cancer.(12)

Tobacco

Tobacco is used in both smoke and smokeless forms. Smoking forms are more common in western countries compared to the Middle East and eastern countries. Smoking form includes the use of beedis and cigarattes predominantly with various aids like hooka, hookli, chutta, dhumthi, chillum. However the higher pricing of these products compared to other forms makes this more common amongst the middle and upper socioeconomic classes of population. Beedi is a smoking stick made by rolling a dried piece of rectangular temburni leaf (Diospyrosmelanaxylon) with sun-dried, flaked tobacco arranged in a conical shape. The roll is then secured with the thread. Chutta is a kind of reverse smoking wherein the burnt end is kept inside the mouth. It is made up of coarsely prepared roll of tobacco and prevalent in South-eastern parts of India. Dhumthi is another type of smoking forms made by rolling the tobacco leaves inside the leaf of the jackfruit tree. Hooka is made using metallic or wooden pipes and used for smoking tobacco. This device consists of a spherical receptacle, containing water with aromatic substances. Hookli is another form of smoking tobacco device which employs a short clay pipe like device. Chillum is a conical clay pipe used to smoke tobacco and tobacco with ganja in northern parts of India. The narrow end of this device is put inside the mouth and wrapped in a wet cloth to act as a filter. (13) The smokeless forms of tobacco includes betel quid, khaini, mawa, snuff, gutka and pan masala. Betel quid consists of areca nut, slaked lime, tobacco mixed and folded in a betel leaf and placed either in the buccal or labial vestibule. Khaini is prepared by mixing roasted tobacco flakes and slaked lime and the mixture is placed in the lower buccal or labial sulcus. Mawa is a mixture of areca nut, tobacco and slaked lime. Snuff is a blackish brown powder obtained from tobacco through roasting and pulverisation. It is generally used through nasal insufflations or applied on the gums by using a finger. Gutka is a mixture of areca nut, tobacco and few other condiments, which are marketed in different flavors in colorful pouches. Pan Masala may or may not contain tobacco, with areca nut and few other condiments. (14)

Human PapillomaVirus (HPV)

Human Papilloma viruses, belong to a family of viruses that affect the skin and moist membranes lining the body, such as those in the cervix, anus, mouth and throat. An individual can be

infected with Human Papilloma Virus by having sexual conduct with a person already infected. (15, 16) There is evidence that some types of HPV infection could also cause abnormal tissue growth inside the mouth, triggering some cases of mouth cancer. (17, 18) HPV infection is the probable cause of mouth cancer in young people who have few or none of the expected other risk factors.

Diet

There is evidence that a diet high in red meat, processed food and spicy food can increase the risk of developing oral cancer. (19) High consumption of deep fried foods was associated with an elevated risk of oral cancer. Heterocyclic amines and other substances produced by heating meat and fish, particularly at high temperatures, as used in deep frying, have been shown to be mutagenic and carcinogenic in animal models (20).

Diagnosis

The dentist should check the mouth and throat for red or white patches, lumps, swelling or other problems, carefully at the roof and floor of the mouth, back of the throat, and insides of the cheeks, gingiva and lips and also in the lymph node regions. If an individual shows an abnormal area in the oral cavity, a small sample of tissue may be excised. (21) Excising tissue to look for cancer cells is called a biopsy. Usually, a biopsy is done with local anesthesia and sometimes, it is done under general anesthesia. Followed by this, an oral pathologist examines the tissue under a microscope to check for the growth of malignant cells. (22)

ExfoliativeCytology

In this method, the dentist scrapes the affected area and smears the collected tissue onto a glass slide. The sample is then stained with a dye, which aids by making the cells visible under the microscope. If the cells examined are found to be abnormal, then the area can be biopsied. (23)

Incisional Biopsy

Here, the dentist excises a small piece of tissue from the affected area. This is the most common type of biopsy applied to sample areas in the mouth or throat. Biopsy can be done either in the dentist's office or in the operating room, depending on where the tumor is and how easy it is to get a good tissue sample. If it can be done in the dentist's office, the area around the tumor will be anaesthetized before the biopsy is taken and if the tumor is deep inside mouth or throat, the biopsy might be done in the operating room with the patient under general anesthesia. (24)

Fine NeedleAspirationCytology (FNAC)

For this test, the dentist uses a very thin, hollow needle attached to a syringe to aspirate some cells from a lump. These cells are then examined under a microscope to see if malignant cells are present. FNAC is not used to sample abnormal areas in the mouth or throat, but sometimes used when a patient has a neck mass that can be felt or seen on a CT scan. (25)

HPV Testing

In case of nasopharyngeal cancers, the dentists often have the biopsy samples to be tested for HPV infection, as it is likely to be the cause. This information can help the dentist to predict the probable course of the cancer and thetreatment to be done. (26)

ImagingTests

Imaging tests involve the use of x-rays, magnetic fields, or radioactive substances to create/reproduce pictures of the inside structures of the body. Imaging tests are often not used to diagnose oral cavity or oropharyngeal cancers, but they may be done for a number of reasons both before and after a cancer diagnosis, including to help look for a tumor if one is suspected, to learn how far cancer may have spread, to help determine if treatment has been effective and for possible signs of cancer recurrence after treatment. (27)

Computed Tomography (CT)

The computed tomography (CT) scan produces detailed, multiple cross-sectional images of the body. A computer then combines these images into a single image. A CT scan creates detailed images of the soft tissues and organs in the body compared to that of regular X rays. This test can help the dentist to determine the size and location of a tumor, if it is spreading into nearby tissues, and if to lymph nodes in the neck and the test also may be done to look for spread of cancer to the lungs. (27)

Magnetic ResonanceImaging (MRI)

Magnetic resonance imaging (MRI) scans use strong magnets and radio waves instead of x-rays. The energy from the radio waves is absorbed by the body and then released in a specific pattern formed by the type of body tissue and by certain diseases. A computer translates the pattern into a very detailed image of parts of the body. (27) This test can help the dentist to determine the spread of the cancer into adjacent soft tissues and other body parts.

Staging

Staging of oral cancer is important for establishing proper treatment and determining prognosis. Tumors are staged using the TNM system, where T represents the size of the primary tumor, N indicates the status of the regional lymph nodes, and M indicates the presence or absence of distant metastases. (28)

Primary Tumor (T)

TX - Primary tumor cannot be assessed

T0 - No evidence of primary tumor

T_{is} - Carcinoma in situ

T1 - Tumor $\leq$ 2 cm in greatest dimension

T2 - Tumor $\geq$ 2 cm but $\leq$ 4 cm in greatest dimension

T3 - Tumor $\geq$ 4 cm in greatest dimension

T4 - (lip) Tumor invades through cortical bone, inferior alveolar nerve, floor of the mouth or the skin of the face i.e., chin or nose

$T4_a$ - Moderately advanced local disease

(Lip) Tumor invades through cortical bone, inferior alveolar nerve, floor of the mouth or the skin of the face i.e., chin or nose

(Oral cavity) Tumor invades adjacent structures (eg. through cortical bone [mandible or maxilla] into the deep extrinsic muscles of the tongue (genioglossus, hyoglossus, palatoglossus and styloglossus), maxillary sinus or skin of the face

$T4_b$ - Very advanced local disease

Tumor invades masticator space, pterygoid plates, or skull base and/or encases internal carotid artery

Regional LymphNodes (N)

NX - Regional lymph nodes cannot be assessed

N0 - No involvement of regional lymph nodes

N1 - Metastasis in a single ipsilateral lymph node, $\leq$ 3 cm in greatest dimension

N2 - Metastasis in a single ipsilateral lymph node, $\geq$ 3 cm$\leq$ 6 cm in greatest dimension; or in multiple ipsilateral lymph nodes, none $\geq$ 6 cm in greatest dimension or in bilateral or contralateral lymph nodes, none $\geq$ 6 cm in greatest dimension

$N2_a$ - Metastasis in a single ipsilateral lymph node, but $\geq$ 3 cm$\leq$ 6 cm in greatest dimension

$N2_b$ - Metastasis in multiple ipsilateral lymph nodes, none $\geq$ 6 cm in greatest dimension

$N2_c$ - Metastasis in bilateral or contralateral lymph nodes, none $\geq$ 6 cm in greatest dimension

N3 - Metastasis in a lymph node, $\geq$ 6 cm in greatest dimension

Distant Metastasis (M)

M0 - No distant metastasis

M1 - Distant metastasis

Stage Grouping

Stage 0

T_{is} N0 M0

Stage I

T1 N0 M0

Stage II

T2 N0 M0

Stage III

T3 N0 M0

T1 N1 M0

T2 N1 M0

T3 N1 M0

Stage IVA

T4a N0 M0

$T4_a$ N1 M0

T1 N2 M0

T2 N2 M0

T3 N2 M0

$T4_a$ N2 M0

Stage IV B

Any T N3 M0

$T4_b$ Any N M0

Stage IV C

Any T Any N M1

Treatment

Many treatment options for oral cancer are applied, which include radiotherapy, surgery, and chemotherapy. The treatment advised for each case usually depends on various factors such as the exact site and extent of the cancer, and your general health.

Surgery

The most common treatment for oral cancer is surgery. The type of surgery depends on the size of the cancer and its site. The surgery is done by removing the cancerous growth and some of the surrounding normal tissue. The main aim of surgery is by curing the cancer by removing it all and sometimes is used to relieve symptoms if the cancer is an advanced stage (palliative surgery). Surgery with lasers may sometimes be used to remove small oral cancers and this may be combined with a light sensitive drug in treatment known as photodynamic therapy. A surgery called micrographic surgery or Mohs' surgery is done for cancers on the lip. In this surgery, the surgeon removes the cancerous growth in very thin layers. The tissue that has been removed is examined under a microscope during the operation. This technique makes sure that all the cancer cells are removed and only a very small amount of healthy adjacent tissue is removed. (29)

Radiotherapy

Radiotherapy uses high energy beams of radiation, which are targeted on the cancerous tissue for a limited period of time. This kills malignant cells, or stops malignant cells from multiplying further. Two types of radiotherapy are used for mouth cancer, namely, external radiotherapy and internal radiotherapy. (29)

External Radiotherapy

In this method, the radiation is targeted on the cancerous growth from a machine. This is the common type of radiotherapy which is used for many types of oral cancer as a general treatment.

Internal Radiotherapy (Brachytherapy)

This treatment is basically done by placing small radioactive wires adjacent to the malignant growth for a short period of time and then they are removed after a period of time.

Chemotherapy

In chemotherapy, anti-cancer drugs are used to kill the malignant cells, or to stop them from multiplying. Chemotherapy may be used in conjunction with radiotherapy or surgery. Chemotherapy may also be advised if the cancer has spread to other areas of the body. (29)

Conclusion

Of all sites in the head and neck region, lesions found in the oral cavity should be amenable to early detection because the oral mucosa is accessible for viewing by both the patient and general health care professionals. Furthermore, the rich innervation of oral mucosa ensures that even small lesions are likely to elicit tenderness or erythema. Oral cavity lesions are commonly seen in both smokers and alcoholic drinkers, but also an alarming increased frequency is seen along the lateral side of the tongue in non-smokers. Early detection is the first step toward the overall goal of early treatment, which holds the promise of greater efficacy, since single modality treatment is sufficient in many cases. Early diagnosis is facilitated by the accessibility to biopsy without general anesthesia. Educating the general population about oral cancer is a must to bring down mortality and morbidity arising out of it and improve the quality of life of the population.

Corresponding Author

PavithraPriyadarshoni S,

Tutor, Department of Public Health Dentistsry

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email Id: drpavithraphd88@gmail.com

Contact Number: 9176222104

References

1. Scattoloni J. Screening for Oral Cancer: A Brief Evidence Update for the U.S. Preventive Services Task Force. Rockville, MD: Agency for Healthcare Research and Quality; 2004. AHRQ Pub No. 05-0564-B
2. Fritz AG, editor. International classification of diseases for oncology: ICD-O. World Health Organization; 2000.
3. Van der Waal R, Van der Waal I. Oral non-squamous malignant tumors: diagnosis and treatment. Medicina Oral, Patología Oral y CirugíaBucal (Internet). 2007 Nov; 12(7):486-91.
4. Brocklehurst P, Kujan O, Glenny AM, Oliver R, Sloan P, Ogden G, Shepherd S. Screening programmes for the early detection and prevention of oral cancer. Cochrane Database Syst Rev.2010 Nov 10 ;(11).
5. Warnakulasuriya S, Johnson N, Van der Waal I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. Journal of oral pathology & medicine. 2007 Nov 1; 36(10):575-80.
6. Kademani D. Oral cancer. Mayo Clin Proc. 2007;82(7):882–87
7. Petti S. Pooled estimate of world leukoplakia prevalence: a systematic review. Oral oncology. 2003 Dec 1; 39(8):770-80.
8. Napier SS, Speight PM. Natural history of potentially malignant oral lesions and conditions: an overview of the literature. Journal of oral pathology & medicine. 2008 Jan 1; 37(1):1-0.
9. Bagan J. Oral squamous cell carcinoma overview. Oral Oncol. 2009; 45(4-5):301–08.
10. Warnakulasuriya S. Global epidemiology of oral and oropharyngeal cancer. Oral Oncol. 2009; 45(4-5):309–16.
11. Petti S. Lifestyle risk factors for oral cancer. Oral Oncol. 2009; 45(4-5):340–50.

12. Hashibe M, Brennan P, Benhamou S, Castellsague X, Chen C, Curado MP, Maso LD, Daudt AW, Fabianova E, Wünsch-Filho V, Franceschi S. Alcohol drinking in never users of tobacco, cigarette smoking in never drinkers, and the risk of head and neck cancer: pooled analysis in the International Head and Neck Cancer Epidemiology Consortium. Journal of the National Cancer Institute. 2007 May 16; 99(10):777-89.
13. Pillai HS, Jagannathan N. Tobacco- A potential threat to the oral cavity. Int J Pharm PharmSci, 2014, Vol 6, Issue 1, 38-40.
14. Aysha S, Maheswari TU. Occurence of Oral Mucosal Changes among the Smokeless Tobacco Users in Patients Visiting Saveetha Dental College. Journal of Oral Medicine, Oral Surgery, Oral Pathology and Oral Radiology. 2015; 1(3):117-20.
15. Schlecht HP. Oral human papillomavirus infection: hazard of intimacy. JAMA. 2012; 307(7):724–5.
16. Syrjänen S, Lodi G, Von Bültzingslöwen I, Aliko A, Arduino P, Campisi G, Challacombe S, Ficarra G, Flaitz C, Zhou HM, Maeda H. Human papillomaviruses in oral carcinoma and oral potentially malignant disorders: a systematic review. Oral diseases. 2011 Apr 1; 17(s1):58-72.
17. Kreimer AR, Clifford GM, Boyle P, Franceschi S. Human papillomavirus types in head and neck squamous cell carcinomas worldwide: a systematic review. Cancer Epidemiol Biomarkers Prev.2005; 14(2):467–75.
18. Shillitoe EJ. The role of viruses in squamous cell carcinoma of the oropharyngeal mucosa. Oral Oncol.2009;45(4-5):351–5
19. Llewellyn CD, Linklater K, Bell J, Johnson NW, Warnakulasuriya S. An analysis of risk factors for oral cancer in young people: a case-control study. Oral oncology. 2004 Mar 1; 40(3):304-13.
20. Czerniak R, Barnes WS, Weisburger JH. Mutagens and Carcinogens in Food. InDiet, Nutrition and Cancer: A Critical Evaluation 2018 Jan 18 (pp. 115-134). CRC Press.
21. Patton LL, Epstein JB, Kerr AR. Adjunctive techniques for oral cancer examination and lesion diagnosis: a systematic review of the literature. The Journal of the American Dental Association. 2008 Jul 1; 139(7):896-905.

22. Rethman MP, Carpenter W, Cohen EE, Epstein J, Evans CA, Flaitz CM, Graham FJ, Hujoel PP, Kalmar JR, Koch WM, Lambert PM. Evidence-based clinical recommendations regarding screening for oral squamous cell carcinomas. The Journal of the American Dental Association. 2010 May 1; 141(5):509-20.
23. Sankaranarayanan R, Mathew B, Jacob BJ, Thomas G, Somanathan T, Pisani P, Pandey M, Ramadas K, Najeeb K, Abraham E, Trivandrum Oral Cancer Screening Study Group. Early findings from a community-based, cluster-randomized, controlled oral cancer screening trial in Kerala, India. Cancer. 2000 Feb 1; 88(3):664-73.
24. Ramadas K, Sankaranarayanan R, Jacob BJ, Thomas G, Somanathan T, Mahé C, Pandey M, Abraham E, Najeeb S, Mathew B, Parkin DM. Interim results from a cluster randomized controlled oral cancer screening trial in Kerala, India. Oral oncology. 2003 Sep 1; 39(6):580-8.
25. Sankaranarayanan R, Ramadas K, Thomas G, Muwonge R, Thara S, Mathew B, Rajan B, Trivandrum Oral Cancer Screening Study Group. Effect of screening on oral cancer mortality in Kerala, India: a cluster-randomised controlled trial. The Lancet. 2005 Jun 4; 365(9475):1927-33.
26. Ang KK, Harris J, Wheeler R, Weber R, Rosenthal DI, Nguyen-Tân PF, Westra WH, Chung CH, Jordan RC, Lu C, Kim H. Human papillomavirus and survival of patients with oropharyngeal cancer. New England Journal of Medicine. 2010 Jul 1; 363(1):24-35.
27. Harrison LB, Sessions RB, Hong WK, editors. Head and neck cancer: a multidisciplinary approach. Lippincott Williams & Wilkins; 2009.
28. American Joint Committee on Cancer. Lip and Oral Cavity. In: AJCC Cancer Staging Manual, 7th ed. New York, Springer: 2010
29. Sankaranarayanan R. Oral cancer in India: an epidemiologic and clinical review. Oral surgery, oral medicine, oral pathology. 1990 Mar 1; 69(3):325-30.

Anaemia Associated with Chronic Heart Failure

Priyadharshni S[1], Naufal Rizwan[2], E. Roja[3]
Graduate Student[1], Senior Lecturer[2], Department of General Medicine,
Tutor[3], Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Anemia has been identified as an independent prognostic factor of both morbidity and mortality for patients with congestive heart failure (CHF). The association between anemia and adverse outcomes has raised the hypothesis that anemia correction might lead to an improvement in the prognosis of patients with CHF. Nevertheless, data from large randomized trials about the effect of anemia correction on patient outcome are still lacking. Numerous clinical studies, randomized and nonrandomized, have evaluated the efficacy of erythropoietin or iron supplementation for treating anemia in patients with CHF, and their effect on patient symptoms and functional status. The superiority of any of these approaches has not been established yet. This review will discuss different treatment options for anemic patients with CHF, with emphasis on the correction of iron deficiency. Since anemia is an independent risk factor for adverse outcomes in HF, it is reasonable to assume that correction of anemia should improve the prognosis of patients. How- ever, this has not been proved in adequately powered trials yet.

Several studies, both randomized and nonrandomized, have implemented different treatment strategies and investigated the impact of anemia correction on the functional status of patients with CHF. The treatment strategies are: 1) erythropoietin-stimulating agents (ESA), 2) iron supplementation, and 3) both of them. Severe anemia results in the activation of the sympathetic and renin-angiotensin systems, similarly to CHF, potentially leading to exacerbation of these maladaptive responses. Whether this is the case with less severe anemia remains unclear. Anemia is more common in patients with renal failure. The risk of death in anemic patients with CHF and chronic kidney disease (CKD) is increased compared with anemic patients with normal renal function.

Keywords*:Heart failure, patients, anaemia, kidney, haemoglobin.*

Introduction

Anemia is found in about one-third of all cases of congestive heart failure (CHF).Anemia is common and is a powerful independent predictor of death and hospitalization in a broad spectrum of patients suffering from systolic and diastolic dysfunction, new-onset Heart failure and advanced chronic Heart failure. Anemia and chronic renal disease are often present in Chronic Heart failure, one exacerbating the other (cardio-renal anemia syndrome). Chronic renal disease is likely due to the renal vasoconstriction. This reduces the amount of erythropoietin produced in the kidney and leads to anaemia. The anemia itself can worsen cardiac function, as it causes cardiac stress through tachycardia and increased stroke volume, and because it can cause a reduced renal blood flow and fluid retention, adding further stress to the heart. Iron deficiency appears to be the most common cause of Anemia in Heart Failure. Long-standing anemia of any cause can cause left ventricular hypertrophy (LVH), which can lead to cardiac cell death through apoptosis and worsen the CHF. Anaemia thus plays an important role in both the initiation and the progression of heart failure. Recent studies shows that low levels of haemoglobin or haematocrit are associated with a reduced exercise capacity, an increase in hospitalisations and poorer long-term survival.Heart failure (HF) is a common disease associated with poor prognosis. Anaemia is commonly associated with HF due to bone marrow depression, reduced availability of iron and haemodilution, and is sometimes aggravated by too frequent blood testing. Low haemoglobin is very detrimental to the haemodynamic state of the patient with decreased cardiac output as it further diminishes the oxygen supply to the tissues. When anaemia is associated with HF and renal failure, the patient enters a vicious cycle called cardio renal anaemia syndrome. In India, the prevalence of anemia is 2.5% in women and 1% in men associated with chronic heart failure.

Etiology

The etiology of anaemia in HF is multifactorial, including bone marrow depression due to chronic inflammation with production of pro-inflammatory cytokines and induced erythropoietin resistance and reduced availability of iron and heamodilution secondary to sodium and water retention(1).The low iron level, due to reduced content of iron in the diet and also reduced iron absorption, is often present in patients with HF(2).The renin-angiotensin system also plays important role in the regulation of plasma volume and red blood cell volume. Increased angiotensin II signalling in the kidney alters peritubular oxygen

tension, and increases erythropoietin secretion by reducing renal blood flow and increasing proximal tubular reabsorption(3).Angiotensin II may also have direct stimulatory effects on bone marrow erythrocyte precursors. Erythropoietin levels are modestly increased in patients with CHF in proportion to measures of activation of the renin-angiotensin system. Inhibition of the Renin angiotensin system with either ACE inhibitors or angiotensin receptor blockers is associated with decreased erythropoietin production and reduced haemoglobin levels(4). Angiotensin converting enzyme inhibitors, which are commonly used in CHF treatment, can be associated with low hemoglobin, probably by suppression of erythropoietin. Inflammatory cytokines, such as interleukin (IL)-1 and IL-6 and tumor necrosis factor (TNF)-α, which are elevated in severe HF, can also reduce production of erythropoietin or increase resistance to its action.Chronic kidney disease (CKD) is a common comorbidity in patients with CHF, due to low cardiac output and poor organ perfusion. In CKD with HF, where glomerular filtration rate is below 60 mL/ min, erythropoietin production is reduced, resulting into poor hematopoietic and anemia. Finally, concomitant anti-platelet or anticoagulant therapy increases the possibility of gastrointestinal blood loss(5).Inflammatory cytokines, like IL-1 and IL-6 are often raised in patients with severely symptomatic HF patients and may contribute to anemia by various mechanisms, i.e. by (i) promoting iron storage by the reticuloendothelial system, thus not available for hemoglobin synthesis, (ii) increased circulatory levels of inflammatory cytokines, such as IL-6 and TNF-α, found in CHF also inhibits renal production of erythropoietin by activating GATA 2 binding protein and nuclear factor-kB, (iii) increased levels of TNF-α and IL-6 inhibits the proliferation of bone marrow erythroid progenitor cells thus again retarding hematopoiesis(6).

Iron deficiency anemia (IDA) plays an important role in etiology of anaemia in chronic heart failure. Iron deficiency may be absolute or functional. Absolute deficiency of iron is termed when iron stores are depleted as indicated by low serum ferritin, i.e. less than 30 µg/L, which may be due to (i) insufficient dietary iron supply, (ii) intestinal malabsorption, impaired duodenal iron transport, drug interactions (e.g. omeprazole), or food reducing absorption and (iii) gastrointestinal bleeding(7).The possible mechanism of functional IDA appears to be related to hepcidin, a protein which plays central role in the regulation of iron metabolism. Hepcidin levels are elevated in the initial stages of CHF but falls as the disease progresses; low levels are an independent marker of worse prognosis. Hepcidin binds to ferroportin, which is the only protein that is able to export intracellular iron. Ferroportin is degraded by hepcidin preventing iron from re-entering cells. T his reduces iron absorption in

the duodenum and causes iron to be retained in the reticuloendothelial system, reducing its concentration and availability in target tissues. Hepcidin also plays a part in erythropoiesis and in the innate immune response to pathogens(8).

Pathophysiology

Anemia is a problem in various chronic conditions such as renal disease, cancer, infection, and heart disease.These patients are frequently anaemic in the absence of vitamin or mineral deficiencies, hemolysis, or other definable causes. When the etiology of anemia does not appear to be predominantly due to hematinic deficiencies,it is referred to as “anemia of chronic disease.”Patients with anemia usually will have volume overload in vascular system, and oxygen carrying capacity is reduced, in turn leads to compensatory tachycardia and with greater demands on energy expenditure. The increased plasma volume produces eccentric hypertrophy, where ventricular dimension increases disproportionally to the ability to increase wall thickness. Systolic cardiac function may not be altered much in the presence of moderate anemia and in the absence of underlying heart disease. However, in patients with pre-existing damaged heart, systolic function may be low and may add to hemodynamic worsening(9). The ventricular dysfunction is usually reversible with correction of anemia, as seen in a study done in children with severe IDA, where the ratio of LV end-systolic wall stress to the LV volume index (and cardiac index) decreased when hemoglobin concentration was very low, i.e. 6 g/dL, indicating a decrease in LV contractility and LV dysfunction, but this improved after anemia correction with iron replacement, before the rise in hemoglobin level, suggesting that correction of functional abnormalities may also be the result of correction of iron metabolism at the tissue level(10).A circulating hormone called erythropoietin, 80% to 90% of which is produced in the kidneys, is the principal factor that stimulates RBC production. When HF patients have concomitant renal disease, they invariably become anemic owing to erythropoietin deficiency.In chronic HF patients without renal disease, erythropoietin levels may be elevated in response to anemia, but not adequately increased to overcome the anemia.Some degree of erythropoietin resistance may also be present because of elevated plasma levels of inflammatory cytokines.Increased erythropoietin production in HF is thought to be related to decreased renal blood flow and increased proximal tubule sodium reabsorption caused by renin-angiotensin activation. Many HF patients have concomitant renal insufficiency or failure, and consequently may have a combination of factors contributing to the development of anemia. Compared with non

anemic HF patients, those with anemia tend to have a history of hypertension or chronic renal insufficiency. Another identified causes of anaemia in heart failure include an increase in pro inflammatory state including tumor necrosis factor, interleukin IL-1 and IL-6. This pro-inflammatory state can contribute to anemia through a number of mechanisms, including suppression of erythropoietin (EPO) secretion by the kidney, decrease bone marrow responsiveness to EPO and a decrease in iron bioavailability for Hb production. IL-6 is also known to increase the production of hepcidin by the liver, which will lead to a decrease in gastrointestinal iron absorption, further decreasing iron bioavailability(11). Tryptophan is essential for cellular growth. Thus, tryptophan depletion is capable of inhibiting cell proliferation and may contribute to development of T-cell unresponsiveness. Earlier enhanced tryptophan degradation was shown in patients with congestive heart failure and in patients suffering from other chronic diseases. Recently, de- creased hematopoiesis was found in the bone marrow of mice with heart failure.Thus, enhanced degradation of tryptophan could also be the rate-limiting step in hematopoiesis. Consequently , in several diseases with chronic immune activation and concomitant release of IFN- , an increased activity of indoleamine-2,3-dioxygenase could be involved in the drop of hemoglobin and the development of anemia.

Clinical Outcomes

The symptoms of anemia consist of fatigue, mild dyspnea on exertion, and occasionally palpitations. Patients may describe the palpitations as being aware of their heart beating or feeling their heart pounding. Lower Hb concentrations are associated with worse HF symptoms and poorer (higher) NYHA class. Even when it is severe, anemia rarely provokes angina in patients without ischemic heart disease, and, in fact, the prognostic impact of anemia was independent of ischemic heart disease in one large retrospective study. Anemic patients may appear pale, but pallor of the conjunctivae and nail beds is a more reliable sign, particularly in patients of color. An S3 extracardiac sound is often auscultated at the cardiac apex. Jugular venous distention is uncommon, but may occur with peripheral edema and hepatomegaly in the presence of hemodilution as a result of fluid volume overload. Hemodynamic parameters of anemic HF patients in one study revealed lower blood pressure, higher heart rate, and elevated pulmonary capillary wedge pressure. On the basis of HF patient profiles from several studies, patients who are anemic tend to be older, female, and have more severe symptoms and signs of HF. They also tend to have greater cardiac

functional impairment and higher hospitalization rates. In addition, they often have a history of diabetes, renal insufficiency, hypertension, and they are often on diuretic medications(12).In patients presenting with diastolic dysfunction, anemia has been associated with increased mortality, higher likelihood of hospitalization,higher serum concentrations of brain natriuretic peptide, decreased exercise capacity, and longer hospitalizations.It is also note worthy that in a broad population of patients presenting with chronic HF, including patients with diastolic dysfunction, 70% were lymphopenic, suggesting a more widespread disturbance of hematopoiesis in some patients, perhaps due to suppression of the bone marrow by elevated cytokine concentrations. The underlying mechanisms behind this close relationship between anemia and fatal events remain unclear. Potential explanations include neurohormonal hyperactivity, changes in ventricular loading conditions, and a reduced free radical scavenging capacity(13). Chronic anemia may also adversely affect large blood vessels, resulting in arterial hypertrophy and remodeling, occurring as a result of sustained increases in cardiac output. In theory, this may reverse the early vasodi- latation characteristic of anemia and lead to increased systemic vascular resistance, which further contributesto the development of LVH. Early on, these changes may be reversible; however, in conditions such as CKD, they may become permanent. It is important to recognize that, although studies may demonstrate an association between anemia and CVD outcomes, this does not necessarily imply that anemia is the cause and, therefore, a treatable cause of CVD. That is, confounding from unmeasured factors, or residual confounding from measured factors may be playing a role. Two examples include the following: (1) anemia may be associated with an unmeasured risk factor, such as inflammatory status, which in turn is the cause of both the anemia and the causal risk factor associated with

CVD; and (2) anemia may be a marker of the severity of underlying heart disease. For example, in patients with heart failure, anemia may be due to hemodilution associated with the severity of heart failure.

Role of Inflammation

A commonly entertained but still mostly unproven mechanism is that anemia in heart failure resembles anemia of chronic disease as evidenced by iron acquisition in the reticuloendothelial system. Plasma levels of tumor necrosis factor-alpha (TNF-á), interleukin-1, and interleukin-6 are elevated in severely symptomatic patients with heart failure, promoting iron storage by the reticuloendothelial system, likely contributing to the

anemia inflammatory cytokines, such as interleukin-6 and TNF-á, have been shown to inhibit renal production of erythropoietin by activating GATA 2 binding protein and nuclear factor-kB. High levels of TNF-á and interleukin-6 have a tendency to inhibit proliferation of bone marrow erythroid progenitor cells. Hepcidin, an acute phase reactant with intrinsic antimicrobial activity, is synthesized in the liver. Of the two known isoforms, hepcidin-25 plays a key role in iron homeostasis, while the role of hepcidin-20, which lacks the five amino acid sequence, thought to be crucial for iron regulation, remains unclear.Hepatic production of hepcidin is stimulated by interleukin-6, resulting in impaired duodenal iron absorption and down-regulation of ferroportin expression, preventing release of stored iron. Anemia of chronic disease and iron deficiency anemia may coexist in patients with congestive heart failure, complicating the clinical picture, and one may need to measure soluble transferrin receptors and ferritin levels for further delineation.Anemia of chronic disease rather than iron deficiency anemia is far more frequent in patients with heart failure.Although the precise mechanisms are incompletely understood, both inflammatory stress and impaired iron metabolism appear to play a role.

Anemia in an aging population can be further complicated by other factors, including reduced stem cell production, low hematopoietic growth factor production, and altered sensitivity to hematopoietic cytokinesIn addition, higher levels of proinflammatory cytokines, such as interleukin-1, TNF-á, and interleukin-6, are noted in a variety of diseases unique to the elderly population, ie, atherosclerosis, diabetes mellitus, and cancer. Although, there are some data to suggest that carnitine supplementation leads to improvement in anemia and possible reduction in erythropoietin-stimulating agent (ESA) requirements in patients with end-stage renal disease on hemodialysis, the role of carnitine supplementation is yet to be explored in anemic patients with heart failure.

Erythropoietin Production and Resistance

Erythropoietin, a highly glycosylated (40% of total molecular weight) proerythrocytic hormone with a half-life of around 5 hours, is a crucial component of the hematopoietic system in regulation of red blood cell production, and subsequently affects tissue oxygen delivery. Specialised peritubular fibroblasts located in the cortex and outer medulla are responsible for the majority of renal erythropoietin production (90%), while the remainder is synthesized in the liver.Renal hypoxia is the main stimulus for erythropoietin production, resulting in an exponential increase in the number of erythropoietin-producing

cells.Not surprisingly, this response is blunted in patients with chronic kidney disease. Renal blood flow is relatively well maintained in patients with heart failure until the late stages, especially in patients on angiotensin-converting enzyme inhibitors. Renal hypoxia in this setting is more likely due to the presence of local arteriovenous shunts in the intrarenal circulation, resulting in a substantial reduction in oxygen tension and stimulation of erythropoietin production. Defective erythropoietin production was demonstrated in more than 90% of patients with heart failure and laboratory evidence of anemia of chronic disease. In contrast with earlier studies, more recent studies have failed to demonstrate a significant correlation between erythropoietin production and effective renal plasma flow in the setting of heart failure.A weak but significant correlation between erythropoietin production and estimated glomerular filtration rate indicates that renal dysfunction plays a role in the blunted production of erythropoietin in anemic patients with heart failure.

Management of Anaemia Associated with CHF

The primary aim of treatment of HF and anemia is to fulfill the need of oxygen for metabolic performance of the heart and to achieve this, the concentration of hemoglobin should be normal. Though there is no universally agreed target hemoglobin level; however, hemoglobin of 12 g/dL level in patients with CHF is regarded as optimum for adequate myocardial performance. The anemia in HF increases the symptoms of poor functional capacity and is also responsible for frequent rehospitalization and increased mortality. Proper correction of anemia in HF can modify the outcome of patients. It is most important to recognize the cause of anemia like blood loss, poor nutrition and any drug and then manage accordingly.(14)

Iron Therapy

While treating with different intravenous (IV) iron agents, the main concerns are intolerance and occasional anaphylaxis. Most commonly used iron preparation in past was iron dextran complex. Dextran forms outer shell to cover trivalent iron, but it may lead to anaphylactic reaction; hence, nowadays dextran-free preparation are preferred. Other available IV iron preparations are iron hydroxide sucrose, iron gluconate, and iron hydroxide dextran. Recently, new formulations have been introduced such as ferumoxytol in

the US and ferric carboxymaltose in Europe. Several new compounds are also in development but have yet to be tested in patients with CHF. Whether iron replacement therapy helps in clinical improvement, left ventricular function or reduces morbidity and mortality remains another challenge. Many studies were designed with different forms of iron preparation to assess the efficacy and outcome. Oral administration of iron salts is inexpensive and convenient, but unfortunately often poorly tolerated and unreliable. Iron absorption depends on the acidity level in the stomach; thus food, antacids, and proton pump inhibitors impair the response to oral iron. Many patients experience gastric irritation when taking iron on an empty stomach. For this reason, it is our practice to recommend IV iron to most EPO-treated patients who require iron. Prior to the last few years, the only IV iron available was iron dextran, the administration of which was not infrequently associated with symptoms ranging from mild urticaria to full-blown anaphylactoid reactions, which were occasionally lethal. Fortunately, currently available parenteral iron formulations include iron sucrose and ferric gluconate, both of which are extremely safe.The incidence of allergic and other adverse reactions to either of these medications is so low that test doses in advance are not required.(15)

Erythropoietin Stimulating Agents

Erythropoietin, a proerythrocytic hormone has a crucial role in the hematopoietic system in regulation of red blood cell production. It is produced in peritubular fibroblasts located in the cortex and outer medulla of kidney. Renal hypoxia is the main stimulus for erythropoietin production. Erythropoietin levels are modestly elevated in patients with CHF, but the increase is less than that observed in other anemic populations. Erythropoietin stimulates red blood cell production by inhibition of apoptosis of bone marrow erythrocyte progenitors. Bone marrow erythroid progenitor cells escape from apoptosis and proliferate to result in the growth and maturation of proerythroblasts and normoblasts. Subsequently there is increase in reticulocytosis and hemoglobin level. The rationale for using erythropoiesis-stimulating agents (ESAs) is that although anemia in CHF is multifactorial, like low cardiac output related poor renal perfusion and ACE inhibitor therapy, which is given to almost all patients with HF, and has been shown to downregulate erythropoietin system thus contributing to anemia(16).

Conclusion

High morbidity and mortality due to CHF has major impact on health care system. Anemia is one of the common and significant comorbidity in CHF, more so in the elderly population. Anemia itself is an independent prognostic factor in the majority of HF patients. Anemia always has negative impact on course of CHF patients, as oxygen carrying capacity is reduced critically which adds to further jeopardy of failing myocardium. There are several mechanisms contributing to low hemoglobin levels in the HF patients, some of which still remains unexplained; however, available evidences suggest that it is due to complex interaction of underlying iron deficiency due to GI blood losses, and impaired absorption and availability of iron, defective erythropoietin production, resistance, and activation of the renin-angiotensin-aldosterone system, along with the presence of underlying CKD and activation of proinflammatory cytokines. The reduced hemoglobin may merely be a marker for the epiphenomena of advanced HF such as hemodilution due to volume overload, malnutrition from cardiac cachexia, or renal insufficiency. All efforts should be directed for early recognition of anemia in HF patients, discern the pathophysiology of, and test the new therapies for anemia in HF. The correction of anemia is not a substitute for the well-documented effective therapy of CHF but seems to be an important addition to current armamentarium. It is surprising, therefore, that judging from the literature on CHF, such an obvious treatment for improving CHF is so rarely considered. We believe that the correction of the anemia will have an important role to play in the amelioration of cardiac insufficiency, and that this improvement will have significant economic implications as well. Also it is necessary to elucidate whether there are different responses on end points of patients with ischemic HF in comparison to those with nonischemic CHF.

Corresponding Author

E. Roja,

Tutor, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai – 600 077,Tamil Nadu, India.

Email Id: itsrojaa@gmail.com

Contact Number: 91 99941 74480.

References

1. Okonko DO, Anker SD. Anemia in chronic heart failure. J Cardiac Fail 2004; 10: S5–S9.
2. Mustafa I, Leverve X. Metabolic and nutritional disorders in cardiac cachexia. Nutrition 2001; 17: 756–760.
3. Weiss G, Goodnough LT. Anemia of chronic disease. N Engl J Med. 2005;352:1011-23.
4. Macdougall IC, Cooper AC. Erythropoietin resistance: the role of inflammation and pro-inflammatory cytokines. Nephrol Dial Transplant. 2002;17(11):39-43.
5. Nanas JN, Matsouka C, Karageorgopoulos D, et al. Etiology of anemia in patients with advanced heart failure. J Am Cardiol. 2006;48:2485-9.
6. Torre-Amione G, Bozkurt B, Deswal A, . An overview of tumor necrosis factor alpha and the failing human heart.Curr Opin Cardiol. 1999;14:206-10.
7. Jankowska EA, von Haehling S, Anker SD, et al. Iron deficiency and heart failure: diagnostic dilemmas and therapeutic perspectives. Eur Heart J. 2013;34:816-26.
8. Ganz T. Hepcidin, a key regulator of iron metabolism and mediator of anemia of inflammation. Blood. 2003;102:783-8.
9. Gaasch WH. Left ventricular radius to wall thickness ratio. Am J Cardiol. 1979;43:1189-94.
10. Hayashi R, Ogawa S, Watanabe Z, et al. Cardiovascular function before and after iron therapy by echocardiography in patients with iron deficiency anemia. Pediatr Int. 1999;41:13-7.
11. Carson J. Morbidity risk assessment in the surgically anemic patient. Am J Surg. 1995;170(1):32S–36S.
12. De´tivaud l, Nemeth E, Boudjema K, et al. Hepcidin levels in humans are correlated with hepatic iron stores, hemoglobin levels, and hepatic function. Blood 2005; 106:746–748.
13. Ezekowitz J, McAlister F, Armstrong P. Anemia is common in heart failure and is associated with poor outcomes: insights from a cohort of 12,065 patients with new-onset heart failure. Circulation. 2003;107:223–225.
14. Berry C, Norrie J, Hogg K, The prevalence, nature, and importance of hematologic abnormalities in heart failure. Am Heart J. 2006;151:1313–1321.

15. Tang YD, Katz SD. Anemia in chronic heart failure: prevalence, etiology, clinical correlates, and treatment options. Circulation. 2006;113:2454–2461.
16. Ebert BL, Bunn HF. Regulation of the erythropoietin gene. Blood. 1999;94:1864-77.
17. Kannan R., Natarajan S., Surapaneni K.M., Murali A., Senthil Kumar Aiyappan, LalD.V.Homocystinuria in adult siblings. Journal of Clinical and Diagnostic Research. 2012; 6:485-487.
18. TowarShilshiLamkang, Dr. Aruna, S. and Dr. Mangala Gowri, P. "A study to assess sleep disturbance and symptoms among patients with pulmonary arterial hypertension at Saveetha Medical College and Hospital.", International Journal of Development Research,2017;7(09):15286-15289.
19. PrasannaGuru.E, Prevalence of Coronary Atherosclerotic Heart Disease in Metabolic Syndrome Patients, journal of pharmaceutical science and research/J. Pharm. Sci. & Res.2015;7(9):724-726.
20. G. MuthuLaakshmi,Non-Pharmacological Therapies for Treatment of Heart Failure -An Update, journal of pharmaceutical science and research/J. Pharm. Sci. & Res. 2016; 8(7):671-674.

Anatomical Variations in Mandibular First Permanent Molar

Neeraj Surathu[1], Pradeep S[2], Niveda S[3],
Graduate Student[1], Reader[2],Department of Conservative Dentistry and Endodontics
Tutor[3],Department of Public Health Dentistry
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Successful endodontic therapy requires proper diagnosis of root canal morphology along with an understanding of possible variations in canal morphology that affect treatment. Failure to do so can lead to improper endodontic treatment that may eventually result in endodontic failure. Mandibular first molars are treated commonly on the assumption that they have 2 or 3 canals. Studies have however shown a significant incidence of another middle mesial canal in these teeth. Unfilled canals, can compromise the longevity of the tooth, thus being a source of infection.

Key Words: *Mandibular first molar, middle mesial canal, radix entomolaris, c shaped canal, canal anatomy.*

Introduction

The root canal treatment requires proper knowledge of variations in root canal morphology in order to recognise, disinfect and seal all possible root canals. This can be accomplished with proper diagnosis using newer technologies, modification in access preparation, use of operating microscope, enhanced methods of disinfecting and sealing of all canals [1]. The mandibular first molar is the first posterior tooth that erupts and is the tooth that most often requires root canal treatment. This tooth usually has two roots, one mesial and one distal. Usually, the distal root is found to have one root canal and the mesial root is found to have two root canals. But occasionally, it has three roots with two or three canals in the mesial root and one, two, or three canals in the distal root [2].

Teeth do not always have the same internal anatomy. The anatomical variations in number of roots, number of root canals, or even the shape of root canals is often encountered. Many clinicians used to treat every tooth like being a standard one. In these cases, many failures occurred. In time, other practitioners studied the anatomy of root canal systems and their anatomical variations and they found out that there are many different types of teeth. The respective studies provide information that, if taken into account, might improve the outcome of endodontic treatment. The main goal of endodontic therapy is to prevent or heal apical periodontitis. From a biomechanical perspective this means cleaning, shaping, and disinfection that would allow for three-dimensional obturation of the root canal system. However, the complexity of the root canal anatomy presents clinical challenges and difficulties that often jeopardize the primary goal of such therapy. Knowledge of both normal and abnormal anatomy dictates the parameters of root canal therapy and can directly affect the probability of success. Thus, like for any surgical procedure, endodontic therapy should be preceded by a thorough knowledge of pulp chamber and root canal anatomy. Once this complex anatomy has been accessed, the outcome is directly related to the elimination and prevention of microbial contamination

Figure1: Access Cavity of a Mandibular First Molar with 3 Canals.

The tooth generally has two separate roots with a round, or more frequently elliptical, canal in the distal root (Fig. 1) and two canals in the mesial root [5]. Previous in vitro and in vivo reports have indicated that mandibular molars can have more than three root canals. Root canal morphology and configuration have been classified and widely known by Weine and Vertucci[3,9].

The main etiology of failure in endodontic treatment of molars is the partial removal of pulpal tissue of root canal systems, the infection of the remaining tissue and the consequent long-termfailure. To be successful, it is necessary to clean and shape the root canals completely and fill the total area with an inert material. A morphological study of root canals is of extreme importance to effective endodontic treatment, and much attention must be paid to their internal anatomy. In addition, to achieve success in endodontic treatments, the number, localization, shape and direction of root conducts must be considered. With the advent of surgical microscopes, the detection of additional root canals has increased and provided higher pre-visibility to the treatment area, since the complexity of root canal systems is the greatest challenge to the endodontist from both a technical and a microbiological point of view. The first permanent mandibular molars generally present two roots containing two canals on the mesial root and one canal on the distal one. Occasionally, however, they may present a third canal on the mesial root, localized between the mesiovestibular and mesiolingual canal, which deserve special attention during the exploration of the pulp chamber before beginning endodontic treatment. The incidence of this third canal on the mesial root varies from 1% to 15% according to some studies [11]. This third canal may be independent with an individual foramen or may connect to the apical region with the mesiovestibular or mesiolingual canals. Another very common anatomic variation on these molars is the presence of an isthmus on the mesial root, a narrow slit between the buccal and lingual canals of this root, which makes access of the endodontic instruments and auxiliary chemical substances more difficult and maintains microorganisms and residual tissues in this region. The prevalence of an isthmus on the mesial root of first mandibular molars varies from 54% to 89%, and they are localized mainly on medium and apical thirds of the root. There are many recent reports in the literature of notable anatomic variations in third molars, first maxillary molars and second mandibular premolars however, few recent reports have shown cases of treatment of mandibular permanent first molars with

five canals, with three in the mesial root, as in the current thought of Endodontics. Regarding the high incidence of anatomic variations, with the presence of additional root canals and isthmus in these teeth, the importance of an appropriate treatment is critical for successful outcomes in these cases. Thus, this paper aims to report and describe the successful endodontic treatment of a mandibular first molar presenting five root canals.

Possible Variations in Mandibular First Molars

Radix Entomolaris/ Paramolaris

In 2004, De Moor et all presented case reports of a supernumerary root present in the disto lingual aspect of mandibular first molars [4].

The primary aim of endodontic treatment is the elimination of bacteria from the infected root canal and the prevention of subsequent reinfection. This is primarily established by ensuring that proper cleaning and shaping is done followed by proper three dimensional obturation done with a tight coronal and apical seal. Establishing adequate access for cleaning and shaping is an integral part of this procedure. In order to achieve these endodontic objectives, the dental practitioner must be well versed in the knowledge of the anatomy of the tooth, crown, root and root canals and the various possible morphologies and pathologies and how to treat it.

Several authors have reported about the morphology of the mandibular first molars. Most articles have shown that the permanent mandibular first molar usually has three to four root canals present, usually two in the mesial root and one or two in the distal root. Along with the number of root canals, the number of roots may also vary. The majority of first and second mandibular molars are two rooted with two mesial and one distal canals. A major variation occurs when sometime the permanent mandibular first molar has a presence of an extra third root which increases the chances of various morphological appearances in the root canal system. This has a frequency of less than 5% in white Caucasian, African, Eurasian and Indian populations.

For successful endodontic diagnosis and treatment, the dental practitioner must employ proper radiographic techniques to understand the complete root canal anatomy of the tooth to be treated. Radiographs taken at different angulations reveal the basic information regarding the anatomy of a tooth and can thus help to detect any aberrant anatomy such as extra canals / roots. However, a significant constraint in conventional radiography is that it produces a two-

dimensional image of a three-dimensional object, resulting in the superimposition of the overlying structure. To achieve a more detailed understanding of the morphological structure of root canals and their interrelations, more advanced diagnostic tools are required.

The most effective tool in diagnosis of the endodontic system has proven to be the Cone beam computed tomography or CBCT. It is an imaging method employing tomography to generate a three-dimensional reconstruction of the entire tooth at different levels from a single imaging procedure. The advantages of CBCT imaging are that it completely eliminates the superimposition of structural images outside the area of interest and provides a high-contrast resolution and data from a single computed tomography imaging process. Moreover, the images can be viewed in a coronal, sagittal, or even an oblique or curved image planes—a process referred to as multiplanar Reformation (MPR).

RE has an occurrence of less than 5% in the Indian population, and such cases are rarely observed during routine endodontic procedures. We report on six such cases in this paper. RE was observed in the mandibular first molars of three patients being root canal treated. This anatomy was also present on three extracted mandibular teeth which were studied in detail to gain an understanding of their morphological characteristics. Knowledge of such variations can be beneficial in delivering treatment to patients presenting with related diversities in their root canal anatomy[33].

The exact cause of radix entomolaris is still not known. Some authors say that it may be due to disturbance during odontogenesis or may be due to an atavistic origin.

In 35% of cases, four canals are present[6,7]. The distal root contains two canals, one in the buccal and the other in the lingual position. The second distal canal is sometimes found in a separate root in the disto lingual position. It is slightly smaller and mesial to the distobuccal root [9].

Apart from Vertucci’s classification of root canal anatomy variations in mandibular molars, another variation of these teeth is the presence of an extra root. The three roots permanent mandibular first molars are usually present on the distal root and is called as Radix Entomolaris. The prevalence of these three-rooted mandibular first molars appears to be less than 3% in African populations, not to exceed 4.2% in Caucasians, to be less than 5% in Eurasian and Asian populations, and to be higher than 5% (even up to 40%) in populations with Mongolian traits. A total of 18 cases, (12 root filled and six extracted mandibular first molars) with an RE were collected during the years 2000–2003 in patients of Caucasian origin. As far as the access was concerned, entering the root canal in this tooth required a

modification the opening in a disto lingual direction resulting in a trapezoidal opening cavity [4].

C - Shaped Canal

The C-shaped canal was first documented in endodontic literature by Cooke and Cox in 1979,[21] is so named for the cross-sectional morphology of the root and root canal. Instead of having several discrete orifices the pulp chamber of the C-shaped canal is a single ribbon-shaped orifice, which in mandibular molars starts at the mesiolingual line angle and sweeps around the buccal to end at the distal aspect of the pulp chamber[22].

Typically, this canal configuration is found in the teeth with fusion of the roots either on its buccal or lingual aspect. In teeth with this anatomical configuration the pulpal floor is usually situation in a deeper portion and the shape curvature of the canal orifice is unusual. The main anatomic feature of C-shaped canals is the presence of a fin or web connecting the individual canals.

Investigations of root development in mouse molars showed that roots were formed by the meeting of dentine leaflets. The fusing of these leaflets was sometimes irregular, forming accessory canals and occasionally, especially in the third molars the leaflets failed to form. In the case of mandibular molars such failure of dentine leaflet formation resulted in a C-shaped root canal. Once recognized the C-shaped canal provides a challenge with respect to debridement and obturation, especially because it is unclear whether the C-shaped orifice found on the floor of the pulp chamber follows up to the root portion in the apical third. Fused and C-shaped roots may present with narrow grooves that predispose to localized periodontal disease which may in fact be the first diagnostic indication of such anatomic variation. This type of canal configuration is often seen in teeth with fused roots. In such teeth, the floor of the pulp chamber is usually situated deeply and may assume an unusual anatomic appearance[23].

Once it is recognised a tooth with a C shaped canal, it is difficult to establish whether or not the working length of that canal reaches till the apical third of the root or whether it terminates much before[26]. Fused and C-shaped roots may present with narrow grooves that predispose to localized periodontal disease which may in fact be the first diagnostic indication of such anatomic variation[27].

C shaped canal anatomy is a challenge during the preparation and obturation phase and is definitely to be dealt with in a careful manner to prevent any endodontic mishaps. In order to

completely successfully treat the affected tooth one must be follow a strict protocol while attempting this root canal treatment.

For the successful treatment of such C-shape canals following points should be taken into consideration.

Radio graphically it may present as a single fused root or as two distinct roots. Cooke and Cox stated that it is impossible to diagnose C–shaped canals on the radiographs. Radiographs taken while probing the root canal system reveal two characteristics: Instruments tend to converge at the apex; instruments appear clinically and radiographically to perforate the furcation[28].

After routine access cavity preparation and removal of pulp tissue the orifice portions of the slit must be widened considerably early in treatment but not too deeply toward the apex[31].

In C shaped root canal systems generally the mesiolingual canal is fairly distinct from the apical third of the root portion, although it may be significantly shorter than mesiobuccal or distal canals. The mesiobuccal canal may merge with distal canal and both exit through a single foramen or both canal have separate portals of exit [32].

Middle Mesial Canal

Sometimes, the “extra” canal is found in the mesial root, which therefore contains three canals. This is the middle mesial canal [8-16].

Results of the clinical study of 760 first mandibular molars, showed that 20 (2.6%) had three canals in the mesial root. Of these 20 teeth, 13 (65%) had an intermediate canal which joined the mesiobuccal canal in the apical third, 6 (30%) joined the mesiolingual canal in the same location. In only one case did the intermediate or third canal retain its individual nature and end in an independent foramen [13]. The presence of this third canal present in the mesial root is uncommon root morphology and therefore a significant consideration during root canal treatment of such teeth.

The probability of occurrence of middle mesial canal in mandibular first molars ranges from 1% [17] to 15% [18]. Radiographically the number and configuration of root canal system was found to show that on 60 teeth revealed one canal in the mesial root in 5 percent, two in 78.3 percent, three in 13.3 percent and four in 3.3 percent [18].

Aminsobhani M reported 21 cases of mandibular first molar with three mesial canals [19]. Faramarzi F reported a case of mandibular first molar with three mesial canals and broken instrument removal[20].

In many of these cases identification of the middle mesial canal lead to successful endodontic treatment of the decayed tooth. In many cases, failure to locate the canal results in the usual consequences of poor endodontic treatment. In order to ensure proper location of these intermediate canals in the mesial root Fabra H Campos has suggested the following steps: [12,13]

- After preparation of the access cavity the dentinal elevation which is present between the entrance of the mesiobuccal and mesiolingual canals are removed with the help of ultrasonic tips or burs.
- An explorer is used to explore through the groove connecting the mesiobuccal and mesiolingual canal to search for any possible intermediate depression. Also in teeth with vital pulp a bleeding spot can be observed which may indicate MMC (middle mesial canal).
- Catheterise the third canal by using a thin file (#08 or 10) in an alternating 45 degree rotating motion.
- Once the canal is located enlarge canal entrance. Usually this canal may join with the middle third or the apical third with either the mesiolingual or mesiobuccal canal, ending in one foramen.

Taurodontism

Witkop defined taurodontism as "teeth with large pulp chamber in which the bifurcation or trifurcation are displaced apically, so that the chamber has greater apicao-occlusal height than in normal teeth and lacks the constriction at the level of the cementoenamel junction[34]. The term taurodontism was coined by Sir Arthur Keith to describe the "bull-like" condition in teeth in which the tall root trunk encloses a high pulp chamber and short roots [35]. The etiology of taurodontism is unclear. It is thought to be caused by the failure of Hertwig's epithelial sheath diaphragm to invaginate at the proper horizontal level, resulting in a tooth with short roots, elongated body, an enlarged pulp, and normal dentin. Previously, taurodontism was related to syndromes such as Down's and Klinefelter's. Today, it is considered as an anatomic variance that could occur in a normal population regardless of whether any pathology is associated with it [36].A taurodont does not show any significant morphological characteristics that helps in its identification. The radiographic examination is

the only way to visualize a rectangular configuration of the pulp chamber. The apico-occlusal height of the pulp chamber varies depending upon the type of taurodontism [37]. Shaw has classified taurodontism into hypo, meso, and hyper taurodontism on the basis of the apical displacement of the floor of the pulp chamber[38].The teeth most frequently affected are the molars diagnosed most easily by radiograph. Premolars present a lower incidence and the mandibular teeth are found to be more commonly affected [39-42].

Conclusion

The mandibular first molar is the most common tooth to be advised for root canal treatment. In order to successful treat these teeth, proper knowledge of the variations in root canal anatomy is necessary. To successfully identify the root canal anatomy of the tooth to be treated, modern methods of radiography should be considered to ensure all possible variations. The mandibular molar is the first permanent molar to erupt into the oral cavity and it is most often tooth requiring endodontic therapy. The knowledge of root canal system and the most common variations should be kept in mind before starting root canal therapy. With the recent innovations of various operating aids, the coming era can witness more variations in the root canal morphology of permanent mandibular first molar.

Corresponding Author

Niveda S,

Tutor,Department of Public Health Dentistry

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Science

162, Poonamallee High Road,Chennai 600077, Tamil Nadu, India

Email: nive6393@gmail.com

Telephone Number: +91 9952979464

References

1. Ballullaya SV, Vemuri S, Kumar PR. Variable permanent mandibular first molar: Review of literature. Journal of conservative dentistry: JCD. 2013 Mar;16(2):99.
2. Reyhani MF, Rahimi S, Shahi S. Root canal therapy of a mandibular first molar with five root canals: A case report. Iranian endodontic journal. 2007;2(3):110-114.

3. Weine FS, Healey HJ, Gerstein H, Evanson L. Canal configuration in the mesiobuccal root of the maxillary first molar and its endodontic significance. Oral Surgery, Oral Medicine, Oral Pathology. 1969 Sep 1;28(3):419-25.
4. De Moor RJ, Deroose CA, Calberson FL. The radix entomolaris in mandibular first molars: an endodontic challenge. International endodontic journal. 2004 Nov 1;37(11):789-99.
5. Ballullaya SV, Vemuri S, Kumar PR. Variable permanent mandibular first molar: Review of literature. Journal of conservative dentistry: JCD. 2013 Mar;16(2):99.
6. Hartwell G, Bellizzi R. Clinical investigation of in vivo endodontically treated mandibular and maxillary molars. Journal of endodontics. 1982 Dec 1;8(12):555-7.
7. Skidmore AE, Bjorndal AM. Root canal morphology of the human mandibular first molar. Oral Surgery, Oral Medicine, Oral Pathology and Oral Radiology. 1971 Nov 1;32(5):778-84.
8. Weine FS. Case report: three canals in the mesial root of a mandibular first molar (?). Journal of endodontics. 1982 Nov 1;8(11):517-20.
9. Vertucci FJ. Root canal anatomy of the human permanent teeth. Oral surgery, oral medicine, oral pathology. 1984 Nov 1;58(5):589-99.
10. Beatty RG, Krell K. Mandibular molars with five canals: report of two cases. The Journal of the American Dental Association. 1987 Jun 1;114(6):802-4.
11. Bond JL, Hartwell GR, Donnelly JC, Portell FR. Clinical management of middle mesial root canals in mandibular molars. Journal of endodontics. 1988 Jun 1;14(6):312-4.
12. Fabra-Campos H. Unusual root anatomy of mandibular first molars. Journal of Endodontics. 1985 Dec 1;11(12):568-72.
13. Fabra- Campos H. Three canals in the mesial root of mandibular first permanent molars: a clinical study. International endodontic journal. 1989 Jan 1;22(1):39-43.
14. Hülsmann M. Mandibular first premolar with three root canals. Dental Traumatology. 1990 Aug 1;6(4):189-91.
15. Jacobsen EL, Dick K, Bodell R. Mandibular first molars with multiple mesial canals. Journal of endodontics. 1994 Dec 1;20(12):610-3.
16. Pomeranz HH, Eidelman DL, Goldberg MG. Treatment considerations of the middle mesial canal of mandibular first and second molars. Journal of endodontics. 1981 Dec 1;7(12):565-8.

17. Pineda F, Kuttler Y. Mesiodistal and buccolingual roentgenographic investigation of 7,275 root canals. Oral Surgery, Oral Medicine, Oral Pathology. 1972 Jan 1;33(1):101-10.
18. Goel NK, Gill KS, Taneja JR. Study of root canals configuration in mandibular first permanent molar. Journal of the Indian Society of Pedodontics and Preventive Dentistry. 1991 Mar;8(1):12-4.
19. Aminsobhani M, Bolhari B, Shokouhinejad N, Ghorbanzadeh A, Ghabraei S, Rahmani MB. Mandibular first and second molars with three mesial canals: a case series. Iranian endodontic journal. 2010;5(1):36.
20. Faramarzi F, Fakri H, Javaheri HH. Endodontic treatment of a mandibular first molar with three mesial canals and broken instrument removal. Australian Endodontic Journal. 2010 Apr 1;36(1):39-41.
21. Cooke HG, Cox FL. C-shaped canal configurations in mandibular molars. The Journal of the American Dental Association. 1979 Nov 1;99(5):836-9.
22. Jafarzadeh H, Wu YN. The C-shaped root canal configuration: a review. Journal of endodontics. 2007 May 1;33(5):517-23.
23. Gao Y, Fan B, Cheung GS, Gutmann JL, Fan M. C-shaped canal system in mandibular second molars part IV: 3-D morphological analysis and transverse measurement. Journal of endodontics. 2006 Nov 1;32(11):1062-5.
24. Jerome CE. C-shaped root canal systems: diagnosis, treatment, and restoration. General dentistry. 1994;42(5):424.
25. Manning SA. Root canal anatomy of mandibular second molars. International Endodontic Journal. 1990 Jan 1;23(1):40-5.
26. Barril I, Cochet JY, Ricci C. Treatment of a canal with a" C" configuration. Revue francaise d'endodontie: publication officielle de la Societe francaise d'endodontie. 1989 Sep;8(3):47-58.
27. Haddad GY, Nehme WB, Ounsi HF. Diagnosis, classification, and frequency of C-shaped canals in mandibular second molars in the Lebanese population. Journal of Endodontics. 1999 Apr 1;25(4):268-71.
28. Melton DC, Krell KV, Fuller MW. Anatomical and histological features of C-shaped canals in mandibular second molars. Journal of Endodontics. 1991 Aug 1;17(8):384-8.

29. Lambrianidis T, Lyroudia K, Pandelidou O, Nicolaou A. Evaluation of periapical radiographs in the recognition of C- shaped mandibular second molars. International endodontic journal. 2001 Sep 1;34(6):458-62.
30. Cohen S, Burns R. St. Louis: C.V. Mosby; 1994. Pathways of the pulp; pp. 1841–89.
31. Weine FS, Arizona Endodontic Association. The C-shaped mandibular second molar: incidence and other considerations. Journal of Endodontics. 1998 May 1;24(5):372-5.
32. Keith A. Problems relating to the teeth of the earlier forms of prehistoric man.
33. Attam K, Nawal RR, Utneja S, Talwar S. Radix entomolaris in mandibular first molars in Indian population: a review and case reports. Case reports in dentistry. 2012;2012.
34. Sathyanarayana R, Carounanidy U. Taurodontism–Review and an endodontic case report. Endodontology. 2001;13(1):8-10.
35. Tsesis I, Shifman A, Kaufman AY. Taurodontism: an endodontic challenge. Report of a case. Journal of endodontics. 2003 May 1;29(5):353-5.
36. Saini T, Wilson CA. Taurodont molars: review of literature and radiological features. Saudi Dental Journal. 1990;2(2):68-70.
37. Shaw JM. Taurodont teeth in South African races. Journal of anatomy. 1928 Jul;62(Pt 4):476.
38. Schalk- Van Der Weide Y, Steen WH, Bosman F. Taurodontism and length of teeth in patients with oligodontia. Journal of Oral Rehabilitation. 1993 Jul 1;20(4):401-12.
39. Tiku A, Damle SG, Nadkarni U, Kalaskar RR. Hypertaurodontism in molars and premolars: management of two rare cases. Journal-indian society of pedodontics and preventive dentistry. 2003 Dec 1;21(4):131-4.
40. Jafarzadeh H, Azarpazhooh A, Mayhall JT. Taurodontism: a review of the condition and endodontic treatment challenges. International endodontic journal. 2008 May 1;41(5):375-88.
41. Neelakantan P, Subbarao C, Subbarao CV, Ravindranath M. Root and canal morphology of mandibular second molars in an Indian population. Journal of Endodontics. 2010 Aug 1;36(8):1319-22.
42. Mao T, Neelakantan P. Three-dimensional imaging modalities in endodontics. Imaging science in dentistry. 2014 Sep 1;44(3):177-83.

Antidepressant Effects of Ketamine in Depressed Persons-A Short Review

Swetha.S[1],Jothi Priya[2], Jayanth Kumar.V[3],
Graduate Student[1], Senior Lecturer[2],Department of Physiology[2],Reader[3], Department of Oral Medicine and Radiology[3],
Saveetha Dental College, Saveetha Institute Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

The purpose of this review is to evaluate the effects of ketamine in depressed patients which makes it a valuable research tool to identify the response and develop the next generation of fast-acting antidepressants.A growing body of preclinical research suggests that brain glutamate systems may be involved in the pathophysiology of major depression and the mechanism of action of antidepressants. The review aims to assess the treatment effects of a single dose of an N-methyl-d-aspartate (NMDA) receptor antagonist in patients with depression. The N-methyl-D-aspartate (NMDA) antagonist ketamine,an fast acting antidepressants has consistently shown antidepressant effects within a few hours of its administration.The ketamine can exert their antidepressant effects within hours or a few days of their administration.It is a better understanding of the presumed mechanisms associated with these rapid antidepressant effects.

Keywords*: Antidepressant, ketamine, N-methyl-D-aspartate, memory, depression.*

Introduction

Newer antidepressants are essential for the individuals with major depressive disorder (MDD) that does not respond adequately to treatment and because of a delay that appear before the emergence of therapeutic effects. Recent evidence from clinical studies reveals that the NMDA antagonist ketamine is a revolutionary antidepressant because it acts rapidly and is effective for treatment-resistant patients. A single infusion of ketamine stimulates depressive symptoms in treatment-resistant depressed patients within hours and these effects may lastsfor up to 2 weeks(1).Although the discovery of ketamine's effects has reshaped drug discovery for antidepressants, the psychotomimetic properties of this compound limit the use of this therapy to the most severely ill patients.

Major depressive disorder (MDD) is a serious public health problem and one of the most common psychiatric disorders, with a lifetime prevalence of 17% in the United States(1).The currently available antidepressants provide a measurable degree of therapy. 50% of MDD patients do not respond to first–line treatment with conventional antidepressants(2). Moreover, the 3–4 week delay in the onset of therapeutic efficacy leads the patients to suicidal ideation(3).

Recent clinical trials shows that the NMDA antagonist ketamine is a revolutionary novel antidepressant because it acts rapidly and is effective for treatment-resistant patients. Ketamine is a non-competitive antagonist acting at NMDA glutamate receptor. Unlike, classical antidepressants whose therapeutic effects takes weeks to be observed , an acute intravenous injection of ketamine is sufficient to induce quick and long lasting antidepressant effects upto two weeks(4,5). The purpose of this review is to evaluate the effects of ketamine in depressed patients which makes it a valuable research tool to identify the response and develop the next generation of fast-acting antidepressants.A growing body of preclinical research suggests that brain glutamate systems may be involved in the pathophysiology of major depression and the mechanism of action of antidepressants. The review aims to assess the treatment effects of a single dose of an N-methyl-d-aspartate (NMDA) receptor antagonist in patients with depression.

What is Ketamine?

Ketamine is a medication that is used to induce loss of consciousness, or anesthesia. It can produce relaxation and relieve pain in humans and animals. Ketamine belongs to a group of

drugs known as dissociative anesthetics. It is also known as Ketalar, Ketanest, and Ketaset.Other drugs in this category include the hallucinogen, phencyclidine (PCP), dextromethorphan (DXM), and nitrous oxide, or laughing gas.These types of drugs makes a person feel detached from sensations and surroundings, as if they are floating outside their body.It induces a trance like state while providing pain relief, sedation, and memory loss.Heart function, breathing and airway reflexes generally remain functional(6).

Ketamine is an NMDA receptor antagonist with a potent anesthetic effect.It was developed in 1963 as a replacement for phencyclidine (PCP).It started being used for veterinary purposes in Belgium and in 1964 was proven that compared to PCP, it produced minor hallucinogenic effects and shorter psychotomimetic effects. It was FDA approved in 1970, and from there, it has been used as an anesthetic for children or patients undergoing minor surgeries but mainly for veterinary purpose(7).

It is a class III scheduled drug and is approved for use in hospitals and other medical settings as an anesthetic.However, it is also a commonly abused "recreational" drug, due to its hallucinogenic, tranquillising and dissociative effects. Ketamine is safe to use in controlled, medical practice, but it has abuse potential(8). Used outside the approved limits, its adverse mental and physical health effects can be hazardous. Prolonged use can lead to tolerance and psychological addiction.

Structure

In chemical structure, ketamine is an arylcyclohexylamine derivative. Ketamine is a chiral compound. Most pharmaceutical preparations of ketamine are racemic; however, some brands reportedly have (mostly undocumented) differences in their enantiomeric proportions. The more active enantiomer, (S)-ketamine, is also available for medical use under the brand name KetanestS(9).

Indications- (10)

- Debridement, painful dressings, and skin grafting in burn patients, as well as other superficial surgical procedures.
- Neurodiagnostic procedures such as pneumonencephalograms, ventriculograms, myelograms, and lumbar punctures.
- Diagnostic and operative procedures of the eye, ear, nose and mouth, including dental extractions.
- Diagnostic and operative procedures of the pharynx, larynx, or bronchial tree. NOTE: Muscle relaxants, with proper attention to respiration, may be required.
- Sigmoidoscopy and minor surgery of the anus and rectum, and circumcision.
- Extra peritoneal procedures used in gynaecology such as dilatation and curettage.
- Orthopaedic procedures such as closed reductions, manipulations, femoral pinning, amputations, and biopsies.
- As an anesthetic in poor-risk patients with depression of vital functions.
- In procedures where the intramuscular route of administration is preferred.
- In cardiac catheterisation procedures.

Methods of Use

Ketamine is used as an anesthetic due to the hallucinations it may cause.

Since it suppresses breathing much less than most other available anaesthetics.It is the drug of choice for people in traumatic shock who are at risk of hypotension.Low blood pressure is harmful in people with severe head injuryand ketamine is least likely to cause low blood pressure, often even able to prevent it(11,12). The effect of ketamine on the respiratory and circulatory systems is different from that of other anestheticsKetamine is used as a bronchodilator in the treatment of severe asthma(13). Low doses of ketamine reduces morphine use and nausea and vomiting after surgery(14).High quality evidence in acute pain is insufficient to determine if ketamine is useful in this situation(15).

Low-dose ketamine is sometimes used in the treatment of complex regional pain syndrome (CRPS)(16). Ketamine has been tested in treatment-resistant bipolar depression, major depressive disorder, and people in a suicidal crisis in emergency rooms(17).

Administration

The drug is given by a single intravenous infusion at doses less than those used in anesthesia, and preliminary data have indicated it produces a rapid (within 2 hours) and relatively sustained (about 1–2 weeks long) significant reduction in symptoms in some patient (18).Initial studies with ketamine have revealed scientific and clinical interest due to its rapid onset, and because it works by blocking NMDA receptors for glutamate, a different mechanism from most modern antidepressants that operate on other target(19).

Physical Effects

The effects of ketamine are considered as dose dependent. The lower doses of drugs produces varying results when compared to higher doses. A dose of 1-2mg / kg of body weight produces an intense experience lasting about one hour . The effects include a sense of hallucinations, floating and dissociation and stimulation. Larger doses of ketamine may produce "K - hole " , which is likened to an out-of-body or near death experience . Higher doses of ketamine may result in severe respiratory depression , muscle twitches, dizziness,slurred speech , nausea and vomiting (20).One of the most dangerous effects of ketamine is the helpless and confused state of the user after the infusion of the drug . They difficulty with balance , combined with numbness, muscle weakness, and impaired vision.

However, its emergence as a club drug differs from other club drugs (e.g. MDMA) due to its anesthetic properties (e.g., slurred speech, immobilization) at higher doses(21);in addition, there are reports of ketamine being sold as "ecstasy"(22).and also as part of a "postclubbingexperience".Related to its ability to cause confusion and amnesiac effects, ketamine can leave users vulnerable to date rape(23).

Side Effects

Side effects may include one or more of the following (24)

- Cardiovascular: abnormal heart rhythms, slow heart rate or fast heart rate, high blood pressure or low blood pressure.

- Central nervous system: Ketamine is traditionally avoided in people with or at risk of intracranial hypertension (ICP) due to concerns about ketamine causing increased intracranial pressure.
- Dermatologic: Transient erythema, transient morbilliform rash
- Gastrointestinal: Anorexia, nausea, increased salivation, vomiting
- Local: Pain or exanthema of the injection site
- Neuromuscular and skeletal: Increased skeletal muscle tone (tonic-clonic movements)
- Ocular: Double vision, increased intraocular pressure, nystagmus
- Respiratory: Airway obstruction, apnea, increased bronchial secretions, respiratory depression, laryngospasm

These reactions are less common in patients less than 15 years old and greater than 65 years old and when administered intramuscularly.

Conclusions

The development of ketamine for the treatment of depression is an interesting area of investigation which offers a good therapeutic approach. Further, the rapid antidepressant action of ketamine gives us belief that serious depressive episodes may be rapidly eliminated rather than waiting for weeks and months. The development of these therapeutic agents require careful examination of their tolerability and adverse effects. This makes it evident that ketamine may be a viable option for treatment of depressed persons.

Corresponding Author
Jayanth Kumar.V
Reader, Department of Oral Medicine and Radiology,
Saveetha Dental College, Saveetha Institute Medical and Technical Sciences,
Saveetha University, Chennai, India
Email :doctorjayanth@gmail.com
Mobile Number: 8056135807

Reference

1.KesslerRC,ChiuWT, DemlerO,MerikangasKand Walters EE.“Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication”.Arch Gen Psychiatry.2005;62:617–627.

2.Trivedi MH,FavaM,WisniewskiSR,Thase ME,QuitkinF,WardenD. “Medication augmentation after the failure of SSRIs for depression”.N. Engl. J. Med.2006;354:1243–1252.

3.FavaM,RushAJ,AlpertJE,BalasubramaniGK,WisniewskiSR,CarminCN,“Difference in treatment outcome in outpatients with anx- ious versus nonanxious depression: a STAR∗D report”.Am.J.Psychiatry.2008;165:342–351.

4.BermanRM,CappielloA, Anand A,Oren DA,HeningerGR,Charney DS, Krystal JH. “Antidepressant effects of ketamine in depressed patients “. Biol. Psychiatry .2000;47:351-354.

5.Zarate Jr CA, Singh JB, Carlson PJ,BrutscheNE,AmeliR,LuckenbaughDA,Charney DS,ManjiHK.” A randomised trial of an N-methyl-D- aspartate antagonist in treatment- resistant major depression”. Arch. Gen. Psychiatry.2006; 63: 856-864.

6.GreenSM,RobackMG,Kennedy RM, Krauss B."Clinical Practice Guideline for Emergency Department Ketamine Dissociative Sedation: 2011 Update". Annals of Emergency Medicine.2011;57 (5): 449–61.

7.KrügerAD."Current aspects of using ketamine in childhood”.Anaesthesiologie und Reanimation .1998;23 (3): 64–71.

8. HeshmatiF,ZeinaliMB,Noroozinia H,AbbacivashR."Use of ketamine in severe status asthmaticus in intensive care unit".Iranian Journal of Allergy, Asthma, and Immunology. 2003; 2 (4): 175–80.

9.Zarate Jr CA, Singh JB,QuirozJA, De Jesus G,DenicoffKK,Luckenbaugh DA,Manji HK,CharneyDS, “A double blind , placebo controlled study of memantine in the treatment of major depression”. Am.J.Psychiatry.2006;163: 153-155.

10.ManleyG,KnudsonMM,MorabitoD,DamronS."Hypotension, hypoxia, and head injury: Frequency, duration, and consequences". Archives of Surgery.2001; 136 (10): 1118–23.

11.HemmingsenC,NielsenJE."Intravenous ketamine for prevention of severe hypotension during spinal anaesthesia". Acta Anaesthesiologica Scandinavica.1991;35 (8): 755–7.

12.WongDHW,Jenkins LC. "The cardiovascular effects of ketamine in hypotensive states".CanadianAnaesthetists' Society Journal.1975; 22 (3): 339–48.

13.GoyalS, Agrawal A . "Ketamine in status asthmaticus: A review". Indian Journal of Critical Care Medicine.2013; 17 (3): 154–61.

14.BellRF,DahlJB,Moore RA,KalsoEA. "Perioperative ketamine for acute postoperative pain".Pain, Palliative and Supportive Care Group. Cochrane Database of Systematic Reviews.2006;12:167-170.

15.SinB,Ternas T,MotovSM . "The Use of Subdissociative-dose Ketamine for Acute Pain in the Emergency Department.". Academic emergency medicine : official journal of the Society for Academic Emergency Medicine.2015; 22 (3): 251–7.

16.CorrellGE,MalekiJ,GracelyEJ,MuirJJ,HarbutRE."Subanesthetic ketamine infusion therapy: A retrospective analysis of a novel therapeutic approach to complex regional pain syndrome". Pain Medicine.2004; 5 (3): 263–75.

17.TondoL,VázquezGH, Baldessarini, RJ."Options for pharmacological treatment of refractory bipolar depression.". Current psychiatry reports.2014; 16 (2): 431.

18.CaddyC,GiaroliG,WhiteTP,ShergillSS,Tracy DK. "Ketamine as the prototype glutamatergic antidepressant: pharmacodynamic actions, and a systematic review and meta-analysis of efficacy.". Therapeutic advances in psychopharmacology.2014; 4 (2): 75–99.

19.NaughtonM,ClarkeG,O'LearyOF,CryanJF,Dinan TG."A review of ketamine in affective disorders: current evidence of clinical efficacy, limitations of use and pre-clinical evidence on proposed mechanisms of action.". Journal of Affective Disorders.2014;156: 24–35.

20.Joe-LaidlerK,HuntG."Sit down to float: The cultural meaning of ketamine use in Hong Kong". Addiction Research & Theory.2008;16 (3): 259–71.

21.Tanner-SmithEE."Pharmacological content of tablets sold as "ecstasy": Results from an online testing service". Drug and Alcohol Dependence.2006;83 (3): 247–54.

22.CopelandJ,DillonP."The health and psycho-social consequences of ketamine use". International Journal of Drug Policy.2005; 16 (2): 122–31.

23.Berman RM,Cappiello A, Anand A, Oren DA,HeningerGR,Charney DS. “Antidepressant effects of ketamine in depressed patients”. Biol. Psychiatry.2000;47: 351–354.

24.MooreK,Measham F. "Ketamine use: Minimising problems and maximising pleasure". Drugs and Alcohol Today.2006; 6 (3): 29–32.

AntioxidantActivityoftheFlavonoidsintheBerryFamily

Hansini B. Ajantha[1],Anitha Roy[2], Radhika Arjunkumar[3]

Graduate Student[1],Professor and Head[2],Department of Pharmacology,Reader[3],Department of Periodontics.

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Much attention has been focused on the activity of the naturalantioxidants present in fruits and vegetables, because fruits and vegetable consumption is inversely related to the incidence of heart disease and several cancers. Berries contain powerful antioxidants, potential allergens, and other bioactive compounds. Berries contain micronutrients such as vitamin C and folic acid which are essential for health. They may also have additional health benefits as they are also rich in phytochemicals such as anthocyanins and flavonoids, which are responsible for their red, violet, purple and blue colours. Experimental data has lead to numerous health claims implying that they may protect humans against heart disease, cancers and ageing. This review gives an insight about the flavonoids and the antioxidant properties present infew berries, due to flavanoids, which we consume in daily life like strawberries, blueberries, raspberries, gooseberries and blackberries.

KeyWords*:Anthocyanin, AntioxidantActivity,Berries,Flavonoids, PhenolicCompounds.*

Introduction

A major contributing factor to the aetiology of chronic diseases is a poor diet. Diet in the developed world is a complex mixture of foods with highly variable consumption patterns [1]. Berries are important dietary sources of fibre and essential vitamins and minerals. They also contain a vast number of other phytochemicals that have shown marked bioactivities in mammalian cells like oxidative damage, detoxification enzymes, the immune system, blood pressure, platelet aggregation, and anti-inflammatory, antibacterial activities [2]. Berries can also be significant dietary sources of folic acid, a water soluble B vitamin and also may play a role in reducing risk of heart disease and cancer through a range of mechanisms including lowering homocysteine levels, catalysing nitric oxide formation and maintaining DNA stability[3]. In particular, many types of berries contain a high level of vitamin C (ascorbic acid) andphenolic compounds which provide the distinctive and vibrant palate of colours found in dark berries. The most commonly consumed berries are strawberries(Fragariax ananassa), raspberries (Rubusidaeus), blackberries (Rubusspp.), blueberries (Vacciniumcorymbosum), black currants (Ribesnigrum) and red currants (Ribesrubrum). Natural antioxidants present in foods and other biological materials have attracted considerable interest because of their presumed safety and potential nutritional and therapeutic effects. The increasing level of interests in the search for natural replacements of synthetic antioxidants has paved way for evaluation of large number of antioxidant from plant sources [4]. The most current research on antioxidant action focuses on phenolic compounds such as flavonoids and anthocyanins.

PhenolicCompounds

Phenolic compounds are considered as a significant part of the human diet, and are of huge interest in the research field due to their antioxidant properties. Phenolic compounds consists an aromatic ring containing one or more hydroxyl groups. Generally,structures ofphenolic compounds vary from that of a simple phenolic molecule to that of a complex high weight molecular polymer. The antioxidant activity of phenolic compounds depends on the structure, mostly, the number and positions of the hydroxyl groups and the nature of substitutions on the aromatic rings. Phenolic compounds are secondary metabolites which are derivatives of the pentose phosphate, and phenyl propanoid pathways in plants [5]. Phenolic compounds play an important role in growth and reproduction and also provides protection against pathogens and predators, besides contributing towards the colour and sensory characteristics

of fruits and vegetables. Phenolic compounds exhibit a wide range of physiological properties, such as anti-allergenic, antiartherogenic, antiinflammatory, antimicrobial, antioxidant, anti-thromboticand vasodilator effects.

Flavonoids

Flavonoids constitute the largest group of plant phenolics, accounting for more than half of the eight thousand naturally occurring phenolic compounds, many of which are responsible for the bright colors of flowers, fruit, and leaves. Flavonoids are low molecular weight compounds, consisting of fifteen carbon atoms, arranged in a C6–C3–C6 configuration [6], which are labeled A, B, and C (Fig 1). The various classes of flavonoids differ in the level of oxidation and pattern of substitution of the C ring. Individual compounds within a class differ in the pattern of substitution of the A and B rings. Few of the phenolic compounds are flavones, flavanones, isoflavones, flavonols, flavanonols, flavan-3-ols, anthocyanidins, biflavones, chalcones, aurones, coumarins, hydrolyzable tannins, proanthocyanidins (flavan-3-ol oligomers), caffeates, and lignans, which are all plant phenols. Flavonoids generally occur in plants as glycosylated derivatives.Apart from various vegetables and fruits, flavonoids are found in seeds, nuts, grains, spices, and different medicinal plants as well in beverages, such as red wine, tea, and beer. The flavones apigenin and luteolin are common in cereal grains and aromatic herbs such as parsley, rosemary, thyme, while their hydrogenated analogues hesperetin and naringin are almost exclusively present in citrus fruits. The flavonols, quercetin and kaempferol are predominant in vegetables and fruits, where they are found mainly in the skin, with the exception of onions. Isoflavones are found in legumes, including soybeans, black beans, green beans, and chick peas. Alfalfa and clover sprouts and sunflower seeds also contain isoflavones. The flavan-3-ols, catechin, epicatechin, epigallocatechin, and their gallate esters are widely distributed in plants, although they are very rich in tea leaves. Flavan oligomers are present in apples, grapes, berries, persimmon, black currant, sorghum and barley grains. Anthocyanidins and their glycosides, namely, anthocyanins are natural pigments and are abundant in berries and red grapes. Flavonoids play different roles in the ecology of plants. Due to their contribution in producing attractive colors, flavones, flavonols, and anthocyanidins may act as visual signals for pollinating insects. Flavonoids act as catalysts in the light phase of photosynthesis and as regulators of iron channels involved in phosphorylation.A significant role of flavonoids is the scavenging of oxygen-derived free radicals. In vitro experimental systems also showed that flavonoids

possess anti-inflammatory, anti-allergic, anti-viral, and anti-carcinogenicproperties. Apart from their physiological roles in the plants, flavonoids are important components in the human diet, although they are considered as non-nutrients

. The level of intake of flavonoids is high as compared to those of vitamin C, vitamin E and Vitamin A. Flavonoids intake can range between 50 and 800 mg per day, depending on the consumption of vegetables and fruits, and of specific beverages, such asred wine, tea, and unfiltered beer [7]. Thus,variations in consumption of these foods and beverages are mainlyresponsible for the overall flavonoid intake in variousnational diets.

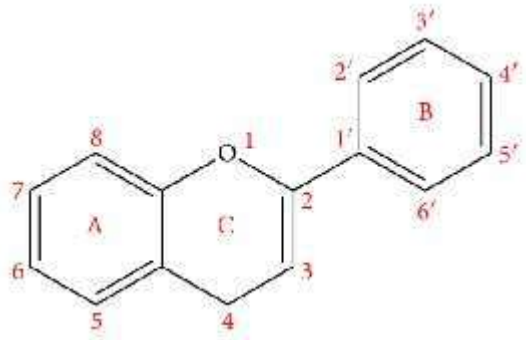


Figure1: Basic structure of flavonids

ClinicalEffects

The different clinical effects of flavonoids are discussed in detail below.

Anti-OxidativeEffects

The best-described property of almost every group of flavonoids is their capacity to act as antioxidants. The flavones and catechins seem to be the most powerful flavonoids for protecting the bodyagainst reactive oxygen species. Body cells and tissues are continuouslyunder threat caused by the damage by free radicals and reactiveoxygen species, which are produced during oxygenmetabolism or are induced by exogenous damage [8, 9]. The mechanismsand the sequence of events by which free radicals interferewith cellular functions are not fully understood, but one of the mostimportant events seems to be lipid peroxidation, which results incellular membrane damage. This cellular damage causes a shift inthe net charge of the cell, changing the osmotic pressure, leading to swelling and eventually cell death. Free radicals can attract variousinflammatory mediators, contributing to a general inflammatory response and tissue damage. To protect themselves from reactive oxygen species, living organisms have developed several effectivemechanisms [10]. The antioxidant-

defense mechanisms of thebody include enzymes such as superoxide dismutase, catalase, andglutatione peroxidase, but also nonenzymatic counterparts such asglutathione, ascorbic acid, and alpha-tocopherol. The increased productionof reactive oxygen species during injury results in consumptionand depletion of the endogenous scavenging compounds.Flavonoids may have an additive effect to the endogenous scavengingcompounds. Flavonoids can interfere with more than three different freeradical producing systems.

Anti-AtherosclerоticEffects

Because of their antioxidative properties, flavonoids are likelyto have a major influence on the vascular system. Oxygen radicalscan oxidize LDL, which tends to injure the endothelial wall and therebypromotes atherosclerotic changes. A study reported that the flavonoids present in regularly consumed foods might reduce the risk of morbidity from coronary heart disease in elderly men [11]. A Japanese studyreported an inverse correlation between flavonoid intake and totalplasma cholesterol concentrations [12]. Oxidative stress and vascular damage are implied to play a significant role in dementia, andregular intake of flavanoids through berries and red wine is reported to prevent the development ofdementia [13].

Anti-InflammatoryEffects

Cyclooxygenase and lipoxygenase play a vital role asinflammatory mediators. They are involved in the release of arachidonic acid, which is a precursor for a general inflammatory response. Neutrophils containing lipoxygenase can create chemotacticcompounds from arachidonic acid, which provoke the release of cytokines. Selected phenolic compounds were shown to inhibit both the cyclooxygenase and 5-lipoxygenase pathways. This inhibition reduces the release of arachidonic acid [14]. The exact mechanism by which flavonoids inhibit these enzymes is not clear. Quercetin, in particular, inhibits bothcyclooxygenase and lipoxygenase activities, thus diminishing the formation of these inflammatory metabolites [15].Another anti-inflammatory feature of the falvonoids, isto inhibit eicosanoid biosynthesis [16]. Flavonoids also has the ability to inhibit both cytosolic andmembranal tyrosine kinase. Integral membrane proteins,such as tyrosine 3-monooxygenase kinase, are involved in a varietyof functions, such as enzyme catalysis, transport acrossmembranes, transduction of signals that function as receptors ofhormones and growth factors, and energy transfer in ATP synthesis.Inhibition of these proteins results in inhibition of uncontrolledcell growth and

proliferation. Tyrosine kinase substratesseem to play key roles in the signal transduction pathway thatregulates cell proliferation. Another significant anti-inflammatory propertyof flavonoids is their ability to inhibit neutrophildegranulation, which is a direct way to diminish the release ofarachidonic acid by neutrophils and other immune cells [17].

Anti-TumorEffects

The antitumor activity of flavonoids is still a point of discussion.Antioxidant systems are frequently inadequate, and damagefrom reactive oxygen species is proposed to be involved in carcinogenesis [18]. Reactive oxygen species can damage DNA,and division of cells with unrepaired or unrepaired damageleads to mutations. If these changes appear in critical genes, suchas oncogenes or tumor suppressor genes, initiation or progressionmay result. Reactive oxygen species can interfere directly with cell signaling and growth. The cellular damage caused by reactive oxygen species can induce mitosis, increasing the risk that damaged DNA will lead to mutations, and can increase theexposure of DNA to mutagens.A studyhas stated that flavonoids, as powerful antioxidants, can inhibitcarcinogenesis. Some flavonoids, such as fisetin, apigenin, and luteolin are stated to be potent inhibitors of rapid cell proliferation [19]. Quercetin and apigenin inhibited melanoma growth and influenced the invasive and metastatic potential in mice [20]. This finding may offer new insights about possible therapies for metastatic disease.

Anti-AngiogeneticEffect

It has been implied that flavonoids has an ability to inhibit angiogenesis. Angiogenesis is a strictly controlled process in the human body. The process of angiogenesis is regulated by a variety of endogenous angiogenic and angiostatic factors. In contrast, pathologic, unregulated angiogenesis occurs in cancer [21]. Angiogenesis inhibitors can interfere with various steps in angiogenesis, such as the proliferation and migration of endothelial cells and lumen formation [22]. The mechanism behind the antiangiogenic effect of flavonoids is still unclear. A possible mechanism could be inhibition of protein kinases [23].

AntiThrombogenicEffects

Platelet aggregation plays a vital role in the development of atherosclerosis and acute platelet thrombus formation, which results in embolization of stenosed arteries. Activated platelets adhering to vascular endothelium, results in generation of lipid peroxides and oxygen free radicals, which in turn inhibit the endothelial formation of prostacyclin and nitrous oxide. Few flavonoids, suchas quercetin, kaempferol, and myricetin were shown to be effective inhibitors of platelet aggregation in dogs and monkeys [24]. Flavonols are particularly antithrombotic because they have the ability to directly scavenge free radicals, thereby maintaining proper concentrations of endothelial prostacyclin and nitric oxide [25]. It is well known that arachidonic acid, which is released in inflammatoryconditions, is metabolized by platelets to form prostaglandin, endoperoxides, and thromboxane A2, leading to platelet activation and aggregation [26]. The vital antiaggregatory effect of flavonoids is inhibition of thromboxane A2formation. Flavonoids affect arachidonic acid metabolism in various ways [27]. Invitro studies showed that flavonoids bind to platelet membranes and may therefore have an accumulative effect over period of time [28].

Anti-ViralEffects

Some of the viruses reported to be affected by flavonoids are herpes simplex virus, respiratory syncytial virus, parainfluenza virus, and adenovirus. Quercetin was stated to exhibit both anti-infective and anti-replicative abilities. The interaction of flavonoids with the different stages in the replication cycle of viruses was previously described [29]. There is some evidence that flavonoids in their glycone form seem to be more inhibitory on rotavirus infectivity than are flavonoids in their aglycone form [30]. Since the worldwide spread of HIV since the 1980s, investigations of the antiviral activity of flavonoids have been mainly focused. The discovery and development of flavonoids as anti-HIV agents has largely expanded in the past two decades. Most of these studies focused on the inhibitory activity of reverse transcriptase, or RNA-directed DNA polymerase [31], but anti integrase and anti-protease activities were also described. Flavonoids have mainly been studied in in vitro experiments, therefore, no clear confirmation of contribution of flavonoids to the treatment of HIV-infected patients has yet been shown [32].

The Berry Family

Blueberry

Blueberries are perennial flowering plants that are usually erect. The flowers are bell-shaped, white, pale pink or red, sometimes tinged greenish. The fruit is considerably small in diameter with a flared crown at the end. They are pale greenish at first, then reddish-purple, and indigo coloured when ripe.They are most commonly found in Europe, Asia, United States, Canada and in Southern Hemisphere.Blueberries are good sources of phenolic acids, principally chlorogenic acid and flavonoids like anthocyanin. They contain the highestantioxidant activityamong the berries [33]. Crude extracts of phenolics, extracted using ethanol as a solvent, from wild blueberry leaves exhibited strong antioxidant properties [34].

Strawberry

The strawberries are first cultivated in France during the late 18^{th} century. They are generally grown in European countries. The fruit is known for its characteristic aroma, bright red colour and juicy texture. It is consumed either fresh or in prepared foods such as preserves, fruit juice, pies, ice creams, milkshakes, and chocolates. Artificial strawberry aroma is also widely used in many industrialised food products.Several studies have shown that the strawberry generally possesses a high level of antioxidant activity, which is linked to the levels of phenolic compounds in the fruit [35, 36, 37]. Wang and Jiao [38]showed that strawberry juice extracts exhibited a high level of antioxidant capacity against free radical species including superoxide radicals, hydrogen peroxide,hydroxyl radicals, and singlet oxygen. Strawberry is one of the most commonly consumed berries. They are an important dietary source of fibre and bioactive compounds, micronutrients and phytochemicals, which are responsible for their bright colour. In particular, strawberries are rich in vitamin C, a handful of strawberries is sufficient to cover the vitamin C recommended daily allowance (RDA) and are among the richest natural food sources of folate [39].Anthocyanins are the most abundant type of polyphenols in strawberry.

Raspberry

The raspberries are perineal plants belonging to a plant species in the genus *Rubus* of the rose family. Raspberries are widely grown in all temperate regions of the World. They are one among the fruits containingthe highest antioxidant levels. The antioxidant activity of raspberries is primarily due to high levels of vitamin C,anthocyanins and ellagitannins. Anthocyaninsare red pigment polyphenols that have been implicated in protection against

coronary heart disease and certain types of cancer [40]. Ellagitanninshave been identified in tea, many medicinal plants, and several fruits, including raspberries.Ellagitannins have been described to have general antioxidant effects and vasorelaxation properties [41, 42]. Raspberry could therefore be considered as a fruit source of variety of potentially healthy compounds.

Gooseberry

The gooseberry is straggling bush with bell shaped flowers. It is commonly found in Europe, north western Africa and Southeast Asia.The fruit has a good source of provitamin A, minerals, vitamin C and vitamin B-complex. The fruit contains high level of fructose, which makes it valuable for diabetics. Its high content of phosphorus levels and dietary fibre is of importance, wherein fruit pectin acts asan intestinal regulator [43].

Blackberry

Blackberry is a perennial shrub with sprawling, woody, and thorny stems. They can reach the height of about 5 meters. It has dark green hairy leaves and flowers are white to pale pink. Fruits are the well-known fleshy black berries. The plant materials contain various type of phytochemical such as alkaloids, flavonoids, tannins, saponins, glycosides, terpenoids, sterols, and carbohydrates. Blackberries are known for their anticancer properties. As they contain antioxidants, they are known to destroy the free radicals that harm cells and can lead to cancer. They also help to protect and strengthen the immunity. Blackberries are also used to make wine, brandy, and flavour liqueurs and cordials and also used as a long-standing home remedy for anaemia, regulates menses, diarrhoea, and dysentery [44].

Conclusion

The study of flavonoids is complex because of the heterogeneity of the different molecular structures and the scarcity of data on bioavailability. Furthermore, insufficient methods are available to measure oxidative damage in vivo and the measurement of objective endpoints remains difficult. While the use of flavanoids as food antioxidants is particularly interesting, practical aspects that need to be considered include extraction efficiency, availability of sufficient raw material, and toxicity or safety considerations.Consuming a diet rich in

antioxidants can improve your health, protect your skin and hair and prevent certain diseases.Antioxidants play a vital role in neutralising harmful by-products of metabolism, free radicals that can lead to cancer and age related diseases. Berries are considered to be the best sources among all the fruits and vegetables containing antioxidants. Berries may reduce the build-up of bad cholesterol that contributes to cardiovascular disease and stroke. It is more effective to get antioxidants through natural diet, as opposed to taking supplements.

Corresponding Author

RadhikaArjunkumar,

Reader,Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id- radhikaarjunkumar@gmail.com

References

1. Julie Beattie, Alan Crozie and Garry G Duthie; Potential Health Benefits of Berries; Current Nutrition &Food Science, 2005, 1, 71-86

2. Duthie GG, Gardner PT, Kyle JA. Plant polyphenols: Are they new magic bullet? Proceedings of the Nutrition Society 2003; 62(3): 599-603

3. Verhaar MC, Stroes E, Rabelink TJ. Folates and cardiovascular disease. Arteriosclerosis Thrombosis and Vascular Biology 2002; 22(1): 6-13.

4. Vaishali M. Popular Medicinal Plants used for Dental Diseases in India. Research Journal of Pharmacy and Technology. 2014 Jul 1; 7(7):805.

5. Randhir, R., Lin, Y.-T., & Shetty, K. Phenolics, their antioxidant and antimicrobial activity in dark germinated fenugreek sprouts in response to peptide and phytochemical elicitors. Asia Pacific Journal of Clinical Nutrition, 2004; 13: 295–307.

6. NagendranBalasundram, KalyanaSundram, Samir Samman; Phenolic compounds in plants and agri-industrialby-products: Antioxidant activity, occurrence, and potential uses, Food Chemistry 2006: 99; 191–203.

7. Pietta PG. Flavonoids as antioxidants. Journal of natural products. 2000 Jul 28;63(7):1035-42.

8. De Groot H. Reactive oxygen species in tissue injury. Hepatogastroenterology 1994; 41:328–32.

9. Grace PA. Ischaemia-reperfusion injury. Br J Surg 1994; 81:637–47.

10. Halliwell B. How to characterize an antioxidant: an update. Biochem Soc Symp 1995; 61:73–101.

11. Hertog MG, Kromhout D, Aravanis C. Flavonoid intake and long-term risk of coronary heart disease and cancer in the seven countries study. Arch Intern Med 1995; 155:381–6.

12. Hertog MG, Feskens EJ, Hollman PC, Katan MB, Kromhout D. Dietary antioxidant flavonoids and risk of coronary heart disease: the Zutphen Elderly Study. Lancet 1993; 342:1007–11.

13. Arai Y, Watanabe S, Kimira M, Shimoi K, Mochizuki R, Kinae N. Dietary intakes of flavonols, flavones and isoflavones by Japanese women and the inverse correlation between quercetin intake and plasma LDL cholesterol concentration. J Nutr 2000; 130: 2243–50.

14. Yoshimoto T, Furukawa M,Yamamoto S, Horie T,Watanabe-Kohno S. Flavonoids: potent inhibitors of arachidonate 5-lipoxygenase. Biochem Biophys Res Commun 1983; 116:612–8.

15. Kim HP, Mani I, Iversen L, Ziboh VA. Effects of naturally-occurring flavonoids and bioflavonoids on epidermal cyclooxygenase and lipoxygenase from guinea-pigs. Prostaglandins Leukot Essent Fatty Acids 1998; 58:17–24.

16. Damas J, Bourdon V, Remacle-Volon G, Lecomte J. Pro-inflammatory flavonoids which are inhibitors of prostaglandin biosynthesis. Prostaglandins Leukot Med 1985; 19:11–24.

17. Tordera M, Ferrandiz ML, Alcaraz MJ. Influence of anti-inflammatory flavonoids on degranulation and arachidonic acid release in rat neutrophils. Z Naturforsch [C] 1994; 49:235–40.

18. Pryor WA. Cigarette smoke radicals and the role of free radicals in chemical carcinogenicity. Environ Health Perspect 1997; 105(suppl): 875–82.

19. Fotsis T, Pepper MS, Aktas E. Flavonoids, dietary-derived inhibitors of cell proliferation and in vitro angiogenesis. Cancer Res 1997; 57:2916–21.

20. Caltagirone S, Rossi C, Poggi A. Flavonoids apigenin and quercetin inhibit melanoma growth and metastatic potential. Int J Cancer 2000; 87:595–600.

21. Fan TP, Jaggar R, Bicknell R. Controlling the vasculature: angiogenesis, anti-angiogenesis and vascular targeting of gene therapy. Trends Pharmacol Sci 1995; 16:57–66.

22. Paper DH. Natural products as angiogenesis inhibitors. Planta Med 1998; 64:686–95.

23. Oikawa T, Shimamura M, Ashino H. Inhibition of angiogenesis by staurosporine, a potent protein kinase inhibitor. J Antibiot (Tokyo) 1992; 45:1155–60.

24. Osman HE, Maalej N, Shanmuganayagam D, Folts JD. Grape juice but not orange or grapefruit juice inhibits platelet activity in dogs and monkeys. J Nutr 1998; 128:2307–12.

25. Gryglewski RJ, Korbut R, Robak J, Swies J. On the mechanism of antithrombotic action of flavonoids. Biochem Pharmacol 1987; 36:317–22.

26. Tzeng SH, Ko WC, Ko FN, Teng CM. Inhibition of platelet aggregation by some flavonoids. Thromb Res 1991; 64:91–100.

27. Landolfi R, Mower RL, Steiner M. Modification of platelet function and arachidonic acid metabolism by bioflavonoids. Structure-activity relations. Biochem Pharmacol 1984; 33:1525–30.

28. Van Wauwe J, Goossens J. Effects of antioxidants on cyclooxygenase and lipoxygenase activities in intact human platelets: comparison with indomethacin and ETYA. Prostaglandins 1983; 26:725–30.

29. Kaul TN, Middleton E Jr, Ogra PL. Antiviral effect of flavonoids on human viruses. J Med Virol 1985; 15:71–9.

30. Bae EA, Han MJ, Lee M, Kim DH. In vitro inhibitory effect of some flavonoids on rotavirus infectivity. Biol Pharm Bull 2000; 23:1122–4.

31. Ng TB, Huang B, Fong WP, Yeung HW. Anti-human immunodeficiency virus (anti-HIV) natural products with special emphasis on HIV reverse transcriptase inhibitors. Life Sci 1997; 61:933–49.

32. Vlietinck AJ, De Bruyne T, Apers S, Pieters LA. Plant-derived leading compounds for chemotherapy of human immunodeficiency virus (HIV) infection. Planta Med 1998; 64:97–109.

33. Ann Marie conner, James J. Luby, Cindy T.S. Tong; Variability and heritability estimates for antioxidant activity, total phenolic content, and anthocyanin content in blueberry progenies;J. Amer. Soc. Hort. Sci. 127(1):82-88, 2002.

34.Marian Naczk, RyszardAmarowicz, RyszardZadernowsk,Ronald B. Pegg, FereidoonShahidi; Antioxidant activity of crude phenolic extracts from wild blueberry leaves; Polish journal ofNutritionSociety2003;62(3):599-6037.

35. Vinson, J. A.; Su, X.; Zubik, L.; Bose, P. Phenol antioxidant quantity and quality in foods: Fruits. J. Agric. Food Chem. 2001, 49, 5315-5321.

36. Sun, J.; Chu, Y. F.; Wu, X.; Liu, R. H. Antioxidant and ant proliferative activities of common fruits. J. Agric. Food Chem.2002, 50, 7449-7454.

37. Wang, S. Y.; Jiao, H. Scavenging capacity of berry crops on superoxide radicals, hydrogen peroxide, hydroxyl radicals, and singlet oxygen. J. Agric. Food Chem. 2000, 48, 5677-5684.

38. Bakker, J.; Bridle, P.; Bellworthy, S. J. Strawberry juice colour: a study of the quantitative and qualitative pigment composition of juices from 39 genotypes. J. Sci. Food Agric.1994, 64, 31–37

39. Carr, A. C.; Frei, B. Toward a new recommended dietary allowance for vitamin C based on antioxidant and health effects in humans. Am. J. Clin. Nutr. 1999, 69, 1086–1107.

40. Kähkönen, M. P.; Heinämäki, J.; Ollilainen, V.; Heinonen, M.Berry anthocyanins: isolation, identification and antioxidant activities. J. Sci. Food Agric. 2003, 83, 1403–1411.

41. Mullen, W.; McGinn, J.; Lean, M. E. J.; MacLean, M. R.Gardner, P.; Duthie, G. G.; Yokota, T.; Crozier, A. Ellagitannin,flavonoids, and other phenolics in red raspberries and their contribution to antioxidant capacity and vasorelaxation properties.J. Agric. Food Chem. 2002, 50, 5191-5196.

42. Okuda, T.; Yoshida, T.; Hatano, T. Ellagitannins as active constituents of medicinal plants. Planta Med. 1989, 55,117-122.

43. Aravind N. Role of Berries as an Antioxidant. Research Journal of Pharmacy and Technology. 2015 Aug 1; 8(8):1001.

44. Rameshwar Verma, Tushar Gangrade, Rakesh Punasiya, and Chetan Ghulaxe; Rubus fruticosus (blackberry) use as an herbal medicine: pharmacogn rev; 2014 Jul-Dec; 8(16): 101–104.

Aphthous Ulcer – A Review

Sarah Banu[1], Mahesh Ramakrishnan[2]

Graduate Student[1], Reader[2], Department Of Pedodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Mouth ulcer is a very common and painful condition, which is categorized as aphthous stomatitis. It is most commonly seen in early childhood and adolescence. These ulcers are small round with circumscribed margins with yellow floor. They are pearl coloured flakes in the mouth or inside the lips. These are oral mucosal lesions and these ulcers are commonly called as Recurrent Aphthous Stomatitis (RAS). There are also different types of ulcers like minor ulcers which are not very large in size and heal within 7-14 days, major ulcers heal slowly which can extend up to weeks or months and the third one is herpetiform ulcer which is multiple pinpoint ulcers that heal within a month. Some reason for the occurrence of ulcers could be stress, trauma, stopping smoking, menstruation and food allergy. Sometimes it could also be due to deficiency of iron, vitamin B12. In children aphthae occurs also due to periodic or high fever, pharyngitis and cervical adenitis.

Key Words: *Aphthous ulcer, minor ulcer, major ulcer, herpetiform*

Introduction

Aphthous ulcer is also called as recurrent aphthous stomatitis or canker sores. It is usually indicated by a redness and swelling or white colour lesion. It is also associated with bleeding in adverse conditions. Severe aphthous ulcer can lead to difficulty in eating [1]. There can be several reasons for the occurrence of aphthous ulcer like stress, nutritional deficiency, genetic and hormonal disturbances [1-3]. These painful lesions usually occur at irregular intervals but have the same sequence of events in every cycle [4]. There are three types of aphthous ulcer such as minor, major and herpetiform. The most common of all is the minor ulcer with an approximate of 80-85% of population [5-8]. The exact etiology and pathophysiology of aphthous ulcer is not yet discovered [9]. Use of tobacco can be said as one of the cause of aphthous ulcer, as it causes injury or chronic irritation to the oral tissues [10]. Many disease are associate with mouth ulcer like gastrointestinal

diseases, Behçet's syndrome, vitamin deficiencies, cyclic neutropenia, Reiter syndrome, hematological disorders, Magic syndrome, PFAPA (periodic fever, aphthous pharyngitis and cervical adenopathy), Sweet syndrome etc[11,12].

Minor ulcers take short time to heal and are small in size. Major ulcers take long time to heal and often lead to scarring. Herpetiform are multiple pinpoint ulcers which heal within a month and are usually found on the tongue.

Definition

Aphthous ulcer is the most common and painful condition which occurs in the oral mucosa. It is basically the breaching of the oral epithelium exposing the nerve endings in the underlying lamina propria. Due to this severe pain is experienced along with soreness, swellings and redness.

Description

Aphthous ulcer is small, round or ovoid ulcers having margins around. They are yellow or grayish in colour. It usually occur only the areas where the skin is not tightly bound to the bone like the inside of lips or cheeks and the dorsum of tongue. The three different types of aphthous ulcers are classified based on the difference of their shape, size, duration and scarring. These are as follows

Minor ulcer is a mild aphthous ulcer whose size varies from 8-10 mm. It is found mostly in non keratinised mucosal surfaces like buccal mucosa, labial mucosa and the floor of the mouth. These will heal in a short period of time like 10-14 days. There may be burning sensation prior to the appearance of the lesion. These do not lead to scar while they cure.

Major ulcer is a rare condition affecting only 10-15% of the population. Its size will reach upto 1cm in diameter. The areas commonly affected by major ulcer are the lips, soft palate and fauces. Sometimes the dorsum of the tongue and gingiva will also be affected [13]. It will last for 6 weeks. This will also lead to scarring.

Herpetiform ulcer is characterized by small multiple ulcers 100 in number. They are 2-3mm diameter. These are small in size and combine to form a large irregular ulcer. These ulcers last for 10-14 days. These are common in women. These usually do not form scars.

Aphthous ulcer can affect the genitals of males and females. It is more common in females than in males. It usually occurs during adolescences or childhood. The aphthous ulcer in the genitals is called as Non Sexually Acquired Genital Ulceration.

Herpes Simplex is an infection. Depending on their position these can be classified as oral herpes or genital herpes. Oral herpes is the sores around the mouth or face. Genital herpes affects the genitals mainly. This is a sexually transmitted disease (STD). Herpes simplex is of two types - HSV-1: This type occurs usually around the mouth and can be transmitted to the genitals during oral sex.
HSV-2: This is mainly on the genitals. It is spread through sexual contact and skin to skin contact. It is a very common and contagious disease.

Associated Factors

The exact etiological reason for the occurrence of aphthous ulcer has not been found yet. But it is s considered to run in the family. In present date it is thought to be caused by the disturbance in the immune system, which will react against the protein of the mucosal tissue. Sodium lauryl sulphate is a detergent found in toothpastes that also cause the growth of ulcer in some individuals [14]. Apart from these some of the associated factors leading to ulcers are nutritional deficiency, trauma, drugs, and hormonal changes.

Nutritional Deficiency

Deficiency in vitamin B12 and iron can lead to decrease in the thickness of the oral mucosa. As the thickness reduces there is maximum chance of it to get worn out and cause lesions.

Genetics

Ulcers due to genetic reasons develop at an early age and are severe in condition. This is seen in 40% of the population [15]. These will be seen at an early stage of life and causes a lot of discomforts.

Trauma

Injury in the oral mucosa due to brushing, disturbances due to teeth etc will also lead to ulcer in the mouth [16]. These reasons will lead to opening of the wound and hence expose the nerve endings leading to lesions. Trauma is basically here referred to as the abberations of the oral mucosa.

Drugs

Many drugs are considered to induce the growth of ulcers in the mouth. Drugs like propionic acid, diclofenac, piroxicam, phenobarbital, sodium hypochloride, phenindione, gold salts and nicorandil [17].

Hormonal Changes

Hormonal changes in women are also associated to ulcers. During menstruation or in the luteal phase of menstrual cycle there are high chances of ulceration [18]. Also by the use of contraceptive pills there is a change in the hormonal levels.

Smoking

In contrary to the causes of ulcers, smoking helps in protection against aphthous ulcers. Smoking is usually considered to be hazardous to health and cause many oral and skin related problems but it has been found out to be protective in function in this case.

It has been found that the smokers have increased keratinization of the oral mucosa. This increase protects the oral mucosa from any kind of infection. It has been assumed that the absorbed constituents from the cigarette lead to increase in keratinization. However hyperkeratosis is considered as premalignant [19]. Even though smoking is one factor which is protective against ulcers, it is cancerous.

Treatment

Several tests can be done to diagnose aphthous ulcer like blood test for blood count, B12, iron. The general treatments taken are use of protective pastes forming a layer on the ulcer reducing irritation and exposure of one of which is Amlexanox paste, avoiding stress, proper intake of vitamins and minerals and antibacterial mouthwash to avoid secondary infection, ignore food stuffs that aggravate ulcers. Benzydamine hydrochloride mouthwash can also be used for treating ulcers.

Topical steroids are also commonly used to treat aphthous ulcer. These include the dexamethasone ointment or to their lesion 3 times daily after meals for 5 days. Topical steroids are usually in the form of paste, cream or lotions, often triamcinolone is in pastes.

Nowadays Low Level Laser Therapy (LLT) is used to treat the aphthous ulcer as it is a fast pain relieving and heals fast. It has fewer side effects than the medicine taken for the treatment of ulcer. It is also cost-effective. It is one of the most appropriate treatments for minor ulcers.

Topical drugs and systemic drugs are used for the treatment of ulcers. Topical drugs are used to treat when the outbreaks are continuous and pain with minor aphthae. The drugs are amlexanox or topical corticosteroids such as triamcinolone acetonide are used to prevent the formation of lesions.

Systemic drugs are used when the outbreak is constant and aggressive with major aphthae and intense pain. Different drugs like colchicine, dapsone, clofazimine and the most commonly used systemic corticosteroids such as prednisone and immune modulators such as thalidomide.

Prognosis

Ulcer can range from minor to a deep one. It causes a lot of discomfort while eating, swallowing which will lead to loss of appetite and weight loss. These do not cause oral cancer or any infection. Aphthous ulcer can usually last for several years before spontaneously disappearing in later life [20].

Conclusion

Aphthous ulcer is a very painful condition which is given least importance but if left unattended can cause severe pain and impact on the quality of life. The duration of a specific ulcer might range from few days to few weeks or sometimes even can last for months. The exact cause of ulcer is unclear. LLLT is a safe, effective and fast way to get rid of ulcers. Topical drugs and systemic drugs are used as treatment.

Corresponding Author

Mahesh Ramakrishnan,

Reader, Department of Pedodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email ID: maheshpedo@gmail.com,

Phone Number: +91 9176873240

Reference

1. Scully C. The diagnosis and management of recurrent aphthous stomatitis: a consensus approach. J Am Dent Ass 2003; 134:200-207.
2. Scully C, Felix DH. Oral medicine: update for the dental practitioner. Aphthous and other common ulcers. Br Dent J 2005; 199(5):259:264.
3. Scully C, Porter SR. Recurrent aphthous stomatitis: current concept of etiology, pathogenesis and management. J Oral Pathol Med 1989; 18(1):21-7.

4. Natah SS, Konttinen YT, Enattah NS, Ashammakhi N, Sharkey KA, Häyrinen-Immonen R. Recurrent aphthous ulcers today: a review of the growing knowledge. Int J Oral Maxillofac Surg 2004; 33: 221–34.
5. Preeti L, Magesh K, Rajkumar K, Karthik R. Recurrent aphthous stomatitis. J Oral Maxillofac Pathol. 2011; 15:252-6.
6. Natah SS, Konttinen YT, Enattah NS, Ashammakhi N, Sharkey KA, Häyrinen-Immonen R. Recurrent aphthous ulcers today: a review of growing knowledge. Int J Oral Maxillofac Surg. 2004; 33:221-34.
7. Femiano F, Lanza A, Buonaiuto C, et al. Guidelines for diagnosis and management of aphthous stomatitis. Pediatr Infect Dis J. 2007; 26:728-32.
8. McBride DR. Management of aphthous ulcers. Am Fam Physician. 2000; 62:149-54, 160.
9. Scully C, Felix DH. Oral medicine--update for the dental practitioner. Aphthous and other common ulcers. Br Dent J. 2005; 199:259–64.
10. Tüzün B, Wolf R, Tüzün Y, Serdaro\uglu S. Recurrent aphthous stomatitis and smoking. International journal of dermatology. 2000; 39:358–60.
11. Baccaglini L, Lalla RV, Bruce AJ, Sartori-Valinotti JC, Latortue MC, Carrozzo M. Urban legends:recurrent aphthous stomatitis. Oral Dis. 2011; 17:755–70.
12. Lalla RV, Choquette LE, Feinn RS, Zawistowski H, Latortue MC, Kelly ET. Multivitamin therapy for recurrent aphthous stomatitis:a randomized, double-masked, placebo-controlled trial. J Am Dent Assoc. 2012; 143:370–6.
13. Cawson RA, Odell EW. Cawson's Essentials of Oral Pathology and Oral Medicine. 8th ed. Philadelphia: Elseivier; 2008.
14. Scully C (2013). Oral and maxillofacial medicine: the basis of diagnosis and treatment (3rd ed.). Edinburgh: Churchill Livingstone. pp. 226–234.
15. Scully C, Porter S. Oral mucosal disease: Recurrent aphthous stomatitis. Br J Oral Maxillofac Surg. 2008; 46:198–206.
16. Jurge S, Kuffer R, Scully C, Porter SR. Recurrent aphthous stomatitis. Oral Dis. 2006; 12:1–21.
17. Natah SS, Konttinen YT, Enattah NS, Ashammakhi N, Sharkey KA, Häyrinen-Immonen R. Recurrent aphthous ulcers today: A review of growing knowledge. Int J Oral Maxillofac Surg. 2004; 33:221–34.
18. McCartan BE, Sullivan A. The association of menstrual cycle, pregnancy and menopause with recurrent oral aphthous stomatitis: A review and critique. Obstet Gynecol. 1992; 80:455–8.

19. McRobbie H, Hajek P, Gillison F. The relationship between smoking cessation and mouth ulcers. Nicotine Tob Res. 2004; 6:655–59.
20. Sherlin HJ, Ramalingam K, Natesan A, Ramani P, Premkumar P, Thiruvenkadam C. Lichen sclerosus of the oral cavity. Case report and review of literature. Journal of Dermatological Case Reports. 2010;4(3):38-43.
21. Nagappan N, John J. Antimicrobial efficacy of herbal and chlorhexidine mouth rinse: a systematic review. J Dent Med Sci 2012; 2:5–10.

Assessment of Pain during Orthodontic Therapy – Review

Abiraamasri BL[1],Saravana Pandian[2], DhanalakshmiRavikumar[3],
Graduate Student[1], Senior Lecturer[2], Department of Orthodontics and DentofacialOrthopaedics, Senior Lecturer[3],Department of Pedodontics and Preventive Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

The aim of the review is assessment ofpain and pain control measures during orthodontic therapy. Pain and discomfort are the two main clinical symptoms appreciated by the patient who is undergoing orthodontic therapy mainly fixed appliance. Pain during orthodontic treatment can discourage the patient from seeking treatment and discontinue the treatment.To compare the different methods ofpain control assessment during fixed orthodontic therapy.

Key word: *orthodontic therapy, pain control,painscale,noxiousstimuli,pain control assessement.*

Introduction

Pain is subjective response with a large individual variation and it's a sensation evoked by noxious stimuli. Pain and discomfort are the two main clinical symptoms experienced by orthodontic patients usually after placement of fixed appliance for 2-4 days [1]. Pain does not localize only to the supporting tissues (PDL) due to force applied to the teeth, but also to surrounding soft tissues, which is due to attrition caused by arch wires and brackets. Pain being a major concern for the patients now a days and it's the main reason for discontinuing orthodontic treatment and discourage patients from seeking treatment [2]. According to O'Connor 2000, pain as a greatest dislike during treatment and fourth among major fear and apprehension prior to orthodontic treatment.Pain depends on

certain factors, that include age, sex, stress, emotional state, magnitude Of force applied etc. According to Amy M. Krukemeyer et al 2009, in his survey he has stated that more than 58% of patients strongly agreed and agreed with the statement “I have pain for few days after an appointment” and 20% answered with a neutral fashion but no one disagreed with the statement.

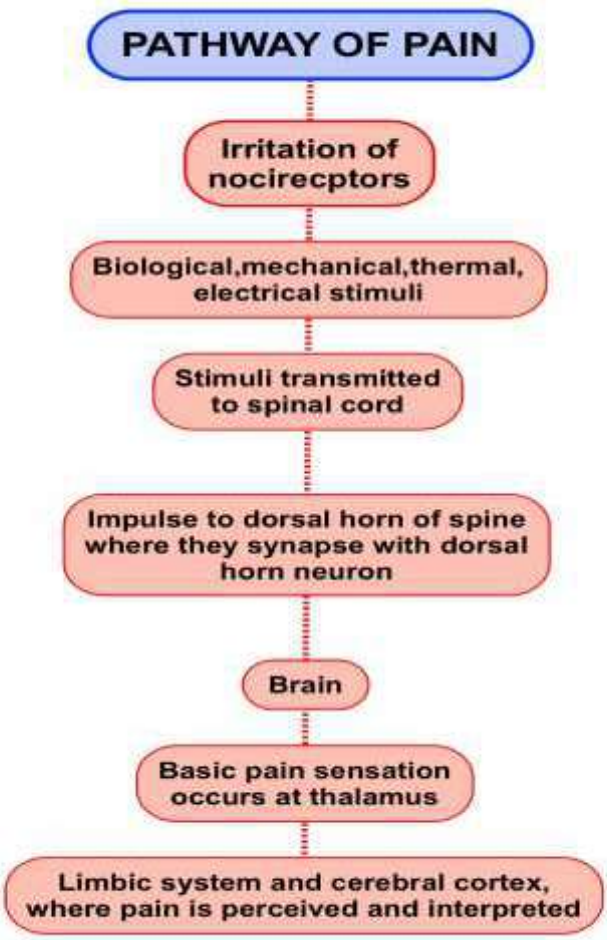


Figure 1: Pathway of Pain

Causes

Pain depends on certain factors and it is caused during placement of separator, placement of arch wires and activation, application of force for tooth movement and during debonding. Pain experienced during fixed appliance is more when compared to removal appliance therapy [3]. Beyond these factors, other factors include age, gender, physiological and psychological well beingof the individual. It has been reported that

adults experience more pain than pre-adolescents and females experience more pain when compared to male [4].

Pain during orthodontic separation

During orthodontic separators like brass wires, elastomeric and spring type steel separator will cause pain. Pain starts within 4 hours of placement and subsides within 5 days of placement but pain being at its peak at 2ndday [5].

Pain during arch wire placement

Pain experienced during arch wire placement varies with different types of arch wires used. Pain usually starts after 4 hours of placement and lasts for 2-3 days and it shows diurnal variation – evening and nighttime elevated pain [6]. It has been reported that anterior teeth experiences more pain than posterior teeth as anterior teeth is involved in functional movements like biting, and difference in root surface anatomy. Likewise pain in lower jaw is more than in upper arch [7].

Pain during arch wire activation

Pain exerted during activation often leads to avoidance of chewing hard food, which in turn leads to deficient dietary intake [8]. Through different experimental studies, masseter muscle activity after activation of arch wire has been evaluated and stated that the activity of masseter muscle has been reduced due to external stimuli. Pain elicited during activation of arch wires affects dietary intake and basic day to day like activities [9].

Pain during application of force

Degree of pain and magnitude of applied force are interrelated. Higher the magnitude, higher the pain experienced by the patients. There is no significant relation between pain and severity of malocclusion. However be the malocclusion, mild to severe patients undergoing fixed appliance therapy will experience pain [10].

Pain during Debonding

Tooth mobility and force applied during debonding are the two main factors responsible for pain during debonding [11]. Pain during debonding is comparatively minimal and can be minimized by placement of occlusal wax rims and cotton rolls.

Other Factors

There is a relationship between physiological and psychological aspect of individual with pain perception during orthodontic therapy. Pain experienced by the individual doesn't completely depend on magnitude of force but it also relies on psychological well beingof the individual [12]. Pain perception with age in orthodontic treatment is a bias because Of different treatment approaches for different age groups. But some studies have revealed that adult patients experience more pain than young individuals [13].

Pain Mechanism

Tooth movement during orthodontic treatment causes inflammatory changes in the periodontium, which stimulates the release Of various biochemical mediators responsible for causing pain. Orthodontic pain is due to change in blood flow caused by orthodontic appliance and are related with the release Of various biochemical mediators like histamine, dopamine, serotonin etc.[14]. Initial step in pain is the irritation ofnocireceptors, which are free nerve endings that responds to pain stimuli. Nocireceptorsare seen in locomotory organs, skin, cornea and dental pulp. The stimulus is Of various sources – biological, mechanical, electrical and chemical stimuli. The stimuli are transmitted to spinal cord then to the central areas of the brain, where pain perception takes place. Impulses run to dorsal horn of spine where they synapse with dorsal horn neuron and enter the brain. Pain is carried by two fibers- A delta fibers and C fibers. Delta fibers are the large fibers usually involved in production of sharp and well defined pain, they are myelinated and have capacity to travel at a rate Of 20 meters/second to the central nervous system. Stimulated by electric shock, physical blow and cut. C fibers are demyelinated fibers and are thin and are easily susceptible to damage. Absence of myelin sheath conducts pain stimuli at a slower rate at about 2 meters/second. Pain will be rapid and pulsating [15,16].

Pain classification

According to orthodontic mechanics by Burstone in 1962 into two ways

(i) Relationship of application of force

(ii) Time of onset.

According to application of force

1) First degree – teeth moved by appliance
   Eg.Band pusher, force gauge.
2) Second degree – pain during clenching and biting.
3) Third degree – unable to bite or chew food of normal consistency.

According to time of onset

1) Acute/immediate- sudden application of heavy force.
2) Chronic/delayed- due to light to heavy force and the intensity decreases over time [17].

Pain Assessment

Pain assessment is done by measuring the intensity of pain through visual analog scale (VAS), which has a rating scale with minimum constraints to evaluate pain perception. VAS is a line whose ends are anchored and measures the intensity of pain by grading scale from 0-10. The individual with pain is expected to a mark on the scale corresponding to the pain experienced by them from 0-10 (0 as no pain and 10 as severe unbearable pain). Pain score of the individual is assessed from the pain scale [18].

Pain management

Relieving pain is one of the most important because pain causes depression, irritation and outburst of anger. It doesn't allow the individual to lead a normal life with family leading to isolation, elimination from social life and dramatically reduce sense of mind [19]. Various modalities include pharmacological approach, physical therapy and invasive method.

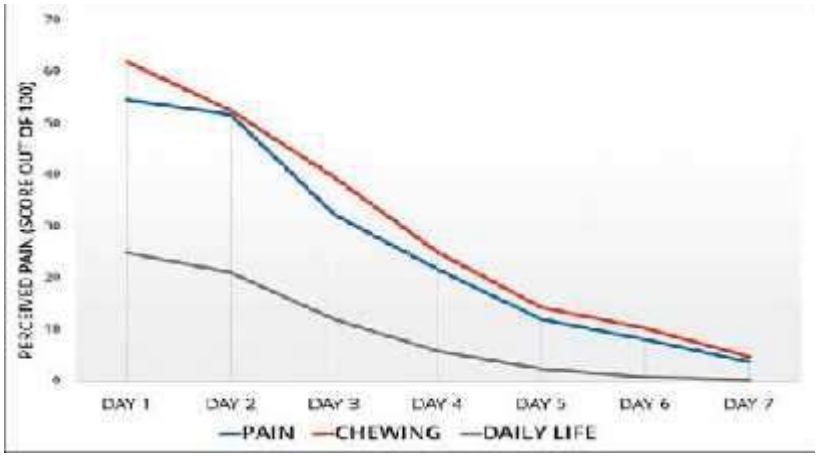


Figure 2: Pain Scale

(i) **Pharmacological approach**

Pharmacological approach includes opioid analgesics – paracetamol and NSAIDS (ibuprofen, aspirin). Analgesic will inhibit the synthesis of prostaglandins at the site of injury. It also includes anesthetic gel, bite wafers and vibratory stimulation. There is no universal recommendation for the use of analgesic for orthodontic pain. Low dose for the first 2-3 days will not affect or interfere with tooth movement. Contraindicated in patients who are allergic to selective drugs [20].

(ii) **Physical therapy**

It includes method of physical treatment- cryotherapy (cold), thermotherapy (heat), laser therapy and manual techniques. Physical treatment doesn't completely eradicate the pain but it helps to improve the life of the individual [21].

(iii) **Invasive methods**

Invasive methods are carried out only when there is no response or relief for the individual through the other pain management methods. In invasive method the management of pain is through local anesthesia – nerve blocks by intrathecal administration of drugs, neurosurgery and neurolysis [22].

Literature Of review

Angelopoulou, conducted a meta-analysis to evaluate the effect of non-steroidal anti-inflammatory drugs (NSAIDs) in managing pain. Among 1,127 studies, seven were included. No statistically significant difference was found between ibuprofen and paracetamol (acetaminophen) administration in relieving pain. It was evident from the literature that ibuprofen lowered pain at two hours and six hours after intervention but not at 24 hours when pain peaks. To some extent, ibuprofen relieves pain in the initial stage of treatment. Hence, it was considered a safe and effective drug compared to other NSAIDs and also had comparatively fewer adverse effects. A report based on a literature review states that NSAIDs impede tooth movement and also increase the risk of root resorption. Therefore, paracetamol (acetaminophen) was considered the safest NSAID that had no influence on the range of tooth movements as well as root resorption and other adverse effects that might occur within the oral cavity [26].

M. Abu Al-Melh and Andersson, the effect of lidocaine/prilocaine topical anesthetic was investigated on pain and discomfort attributed to the insertion of elastomeric separators. Hence, the overall mean discomfort/pain score was found to be significantly lower ($p<0.001$) with the topical anesthetic as compared to the placebo group. Therefore, this methodology might be beneficial for patients with a low pain threshold as it could potentially relieve pain and discomfort after the placement of orthodontic elastomeric separators [27].

In 2003, a study made with 200 adult patients, evaluated the reasons which led them to initially reject the suggestion to undergo orthodontic treatment. The researchers found out the reasons to be, from the highest to the lowest prevalence: Long treatment time, the

discomfort of wearing orthodontic appliances, rejection to the anti-esthetic appearance of brackets, concerns about pain, and fear of disappointment with the final treatment result [29].

In 2005, a satisfaction questionnaire to be answered by 100 patients treated in the Academic Center of Dentistry in Amsterdam three years after they completed their orthodontic treatment. In that study it was observed that the most important issue in determining patient satisfaction was the good orthodontist/ patient relationship during treatment and that female patients revealed lower satisfaction levels with the dentofacial improvements achieved by treatment when compared to male patients [29].

Conclusion

The experience of pain is a complex phenomenon that comprises of behavioral, emotional, physical and interpersonal aspect. Psychological factors also play an important role in pain, where in chronic pain it leads to disability and distress associated with pain. Pain differs from patient to patient, as they vary in magnitude, threshold level and amount of force applied. There is a evidence that education and cognitive behavioral intervention for pain can improve the attitude of individual towards orthodontic treatment, eliminate disability and distress associated with pain.

Corresponding author

Dhanalakshmi Ravikumar,

Senior Lecturer,Department Of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University,Chennai, India

162, Poonamallee High Road,Chennai, 600077, Tamil Nadu, India

Email id: dhana9677@gmail.com

Phone Number: 9597011442.

Reference

1. AshamA A , Southard K A Orthodontic pain . American Journal Of Orthodontics and Dentofacial Orthopedics,2004 .125 :18.
2. VinodKrishnan, Orthodontic pain: from causes to management — a review. European Journal of Orthodontics ,2007;29: 170–179.
3. Paulina Świeboda1, Rafał Filip2, Andrzej Prystupa3, Mariola Drozd4, Assessment Of pain: types, mechanism and treatment. Ann Agric Environ Med. 2013; 1: 2–7.
4. Oliver R G , Knapman Y MAttitudesto orthodontic treatment . British Journal of Orthodontics 1985 ;12 : 179 – 188.
5. NganP, Kess B , Wilson S Perception Of discomfort by patients undergoing orthodontic treatment . American Journal Of Orthodontics and Dentofacial Orthopedics, 1989 ; 96 : 47 – 53.
6. Bergius M ,Kiliardis S , Berggren U Pain in orthodontics: a review and discussion Of the literature . Journal of Orofacial Orthopedics,2000 ;61: 125 – 137.
7. BondemarkL , Fredriksson K , Ilros S Separation effect and perception of pain and discomfort from two types of orthodontic separators. World Journal Of Orthodontics, 2004;(5) : 172 – 176.
8. Firestone A R , Scheurer P A , Bürgin W B Patient's anticipation Of pain and pain-related side effects, and their perception Of pain as a result Of orthodontic treatment with fixed appliances . European Journal of Orthodontics 1999 ; 21 : 387 – 396.
9. ScheurerPA,Firestone A R , Bürgin W B Perception Of pain as a result Of orthodontic treatment with fixed appliances . European Journal of Orthodontics 1996 ; 18 : 349 –357.
10. HixonE H , Aitikian H , Callow G , McDonald H , Tacy R J Optimal force, differential force, and anchorage . American Journal Of Orthodontics 1969;55 :437 – 451.
11. Williams O L , Bishara S E Patient discomfort levels at the time of debonding. A pilot study . American Journal of Orthodontics and Dentofacial Orthopedics1992; 101 : 313 – 317.

12. Jones M, Chan C. The pain and discomfort experienced during orthodontic treatment: a randomized controlled clinical trial of two initial aligning arch wires. Am J OrthodDentofacialorthop. 1992;102(4):373-81.
13. Jones M L , Richmond S Initial tooth movement: force application and pain — a relationship? American Journal of Orthodontics1985 ; 88 :111 – 116.
14. ErdinçAM, Dinçer B. Perception of pain during orthodontic treatment with fixed appliances. Eur J Orthod. 2004;26(1):79-85.
15. Schmelz M, Schmidt R, Bickel A, Handwerker HO, Torebjörk HE. Specific C-receptors for itch in human skin. J. Neurosci. 1997; 17: 8003–8008.
16. Besson J. The neurobiology of pain. Lancet. 1999; 353: 1610–1615.
17. Helms J. Barone C. Physiology and Treatment of Pain. Critical Care Nurse. 2008; 28 (6): 38–49.
18. Apkarian A, Bushnell M, Treede R, Zubieta J. Human brain mechanisms of pain perception and regulation in health and disease. Eur. J. Pain. 2005; 9: 463–484.
19. Burstone C J . The biomechanics of tooth movement. In: Kraus B S , Riedel R A (eds). Vistas in orthodontics. Lea andFebiger , Philadelphia .1962 ;1: 197 – 213.
20. Brown D F , Moerenhout R G The pain experience and psychological adjustments to orthodontic treatment of preadolescents, adolescents and adults . American Journal of Orthodontics and Dentofacial Orthopedics 1991; 100 : 349 – 356.
21. Seymour RA, Simpson JM, Charlton JE, Phillips ME. An evaluation of length and end-phrase of visual analogue scales in dental pain. Pain. 1985;21(2):177-85.
22. Twycross R., Harcourt J., Bergl S. A survey of pain in patients with advanced cancer. J Pain Symptom Manage. 1996;12(5): 273–282.
23. Williams S, Dale J. The effectivenessof treatment for depression/ depressive symptoms in adults with cancer: a systematic review. Br J Canc. 2006; 94: 372–390.

24. Kiliaridis S, Bergius M. Pain and discomfort in orthodontics. In: Graber TM, Eliades T, Athanasiou AE. Risk management in Orthodontics: experts' guide to malpractice. Chicago: Quintessence; 2004;3:131-143.
25. Langley GB, Sheppeard H. Problems associated with pain measurement in arthritis: comparison of the visual analogue and verbal rating scales. ClinExpRheumatol. 1984;2(3):231-234.
26. Angelopoulou M.V., Vlachou V., Halazonetis D.J. Pharmacological management of pain during orthodontic treatment: A meta-analysis. Orthod. CraniOfac. Res. 2012;15(2):71–83.
27. Al-Melh M.A., Andersson L. The effect of a lidocaine/prilocaine topical anesthetic on pain and discomfort associated with orthodontic elastomeric separator placement. Prog. Orthod. 2017;18(1):1.

Association between CRP and Cardiovascular Diseases - A Review

Maria Kurian[1] , Naufal Rizwan[2], Sreedevi Dharman[3]

Graduate Student [1] ,Senior Lecturer[2], Department ofGeneral Medicine

Senior Lecturer[3], Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

The aim of this review is to assess the relationship between C reactive protein and cardiovascular diseases. Physical activity is associated with a reduced incidence of coronary disease, but the mechanisms mediating this effect are not defined. Inflammation has been seen to show an interest in the pathogenesis of cardiovascular disease. Some of the beneficial role of physical activity may result from its effects on the inflammatory process.Cardiovascular disease (CVD) is currently the leading cause of death and disability in the developed world, and will soon overtake infectious disease as the pre-eminent cause of death worldwide. A substantial body of evidence has suggested that four modifiable risk factors — smoking, diabetes, hypertension and hyperlipidaemia account for a significant proportion of CVD, and that reduction of these risk factors leads to reduction in morbidity and mortality. Interleukin-6 (IL-6) and C-reactive protein (CRP) are markers of systemic vascular inflammation that herald atherothrombosis and may have important interrelationships with traditional cardiovascular risk factors. Although markers of systemic inflammation may have a role in the development of hypertension, supportive clinical data remain limited. IL-6 and CRP were measured from baseline bloods. After multivariate adjustment and strong confounding by body mass index, IL-6 was found weakly associated and CRP strongly associated with hypertension risk.

Keywords - *Cardiovascular disease, CRP, Inflammation, Interleukin, Risk factors.*

Introduction

Inflammation is seen to play an important role in the etiology of coronary heart disease (CHD) [1, 2], but the mechanism by which it acts is poorly understood. A short-term inflammatory response is produced by exercise, but from both cross-sectional and longitudinal studies, it is discovered that exercise demonstrates a long-term “anti-inflammatory” effect. This anti-inflammatory response adds to the good effects of regular physical activities. Inflammatory reactions that occur in coronary plaques are seen to play an important role in the development of acute atherothrombotic events. It is seen that inflammation anywhere else is also correlated to atherogenesis and also its thrombotic complications. In recent studies it is noted that systemic markers of inflammation can identify people who are at a high risk of cardiovascular diseases.

Considerable interest has been focused on C-reactive protein (CRP) which is a systemic marker of inflammation in order to improve the cardiovascular risk prediction.CRP has been used to detect the incidence of myocardial infarction, stroke, peripheral arterial disease, and also sudden cardiac death in a patient. CRP levels also have the ability to predict risk of both recurrent ischemia and death in the patients having stable and unstable angina or in those arriving at the emergency rooms showing acute coronary syndromes. It has come to the conclusion that atherothrombosis, is a chronic inflammatory disease in addition to being caused due to lipid accumulation. This is supported by various studies showing clinical evidence of the same [3].

In clinical application, CRP is seen to be a better predictor of cardiovascular diseases when compared to the LDL cholesterol. CRP levels of <1, 1 to 3, and >3 mg/L corresponds to low-, moderate-, and high-risk groups respectively; this is calculated by using widely available high-sensitivity assays. There are a few instances where individuals may present with LDL cholesterol below 130 mg/dL but their CRP levels >3 mg/L. This denotes that they belong to the high-risk group but are often missed out in clinical practice. By making CRP evaluation mandatory in addition to cholesterol evaluation, it forms a simple and inexpensive method by which risk prediction can be improved.

Recently there has been evidence sating that inflammation plays a role in the atherosclerotic process [4]. The predictors of the disease was thought to be cytokines and acute-phase reactantshence they were examined. C-reactive protein (CRP) was found to be highly sensitive

and has assay characteristics that make it easy for clinical use. CRP has been shown to have a dose-response relationship to coronary heart disease. It was also noted that CRP is independent of all other major risk factors [5]. There is well documented evidence which shows that physical activity plays an important role in preventing coronary heart disease [6].

Several studies have shown that elevated plasma levels of fibrinogen, C-reactive protein (CRP), and interleukin-6 (IL-6) are associated with the risk of CHD and the severity of atherosclerosis [7, 8]. Whether these molecules play a causative role, or simply act as markers of the acute phase reaction, is debatable [9]. High blood pressure (HBP) is one of the most important risk factors for cardiovascular–renal disease.In spite of its high health impact, primary prevention of HBP is partly hampered because of a limited knowledge of HBP risk factors [10].C-reactive protein has been shown to be associated to HBP in a few well-controlled studies [11, 12, 13]. On the other hand, results from studies on the association between interleukin-6 (IL-6) and HBP have been contradictory [14, 15].The interplay between the inflammatory process, cardiovascular risk factors, and atherothrombosis is complex [16].Despite evolving theories regarding IL-6 and CRP in the central mechanisms of inflammation and atherogenesis [17],there exists a paucity of data coherently relating clinical cardiovascular risk factors with circulating levels of systemic vascular inflammatory markers in healthy individuals. It is seen that there is astrong and association between the CRP levels and the clinical features of atherothrombotic disease, this was described in studies of patients suffering from acute myocardial ischemia [18]or myocardial infarction [19, 20],stable and unstable angina pectoris [21],and myocardial infarction or recurrent ischemia among those hospitalized with angina pectoris [22].The aim of this review is to understand the correlation between the systemic inflammatory marker CRP and cardiovascular diseases.

C-Reactive Protein

CRP was first discovered in 1930 by William Tillet and Thomas Francis at the Rockefeller Institute for Medical Research, in New York. In studying the blood of patients suffering from acute Streptococcus pneumonia infection, it was found that the sera of these patients formed a precipitin with an extract from the streptococcal bacterium. The extract was originally labeled

Fraction C, and was later confirmed as a polysaccharide. Hence, as a result of its reactivity with the C polysaccharide of the Streptococcus cell wall, the 'substance' in the sera was named CRP.A decade later, Oswald Avery and Maclyn McCarty-the research team who originally described the "transforming principle" and the concept that genes are made of DNA also described CRP as an "acute-phase reactant" that was increased in serum of patients suffering from a spectrum of inflammatory stimuli, including myocarditis and the inflammation associated with rheumatic fever [23].

CRP belongs to the pentraxin family of calcium dependent ligand-binding plasma proteins. The human CRP molecule is composed of five identical non-glycosylated polypeptide subunits each containing 206 amino acid residues.

C-reactive protein (CRP) is a derived from the liver and plays an important role in the innate immune response. CRP is known to have a long plasma half-life period. This is helpful as it acts as a marker for atherothrombotic disease. A large number of studies had been carried out in individuals who had no history of cardiovascular diseases, it was seen that a single CRP level test done is able to predict the vascular effects of the individual. The relationship noted between a patient's CRP levels and risk of cardiovascular disease has seen to be consistent with various studies conducted in USA and Europe. It also supports the fact that CRP is independent of the various risk factors such as age, smoking, cholesterol levels, blood pressure, and diabetes. These effects are present among women as well as men, among the elderly as well as those in middle age, among smokers and non-smokers, and among those with and without diabetes. CRP levels have long-term predictive value. CRP still acts as a strong predictor of risk for cardiovascular diseases even after the initial blood samples were collected 20 years ago [24].

The recent use of highly sensitive CRP assays, with international reference standards set by the World Health Organization (WHO) [25]has established international reference standards using which the highly sensitive CRP assays are done and calculated. By this method, it simplifies the use of CRP as an internationally accepted predictor of cardiovascular events. Large number of prospective studies done in apparently healthy individuals confirmed the prognostic relevance of CRP to (i) the risk of cardiovascular disease in men [26],women [27],and the elderly [28] ; (ii) the risk of fatal coronary disease among smokers with multiple risk factors for atherosclerosis[29]; (iii) the development of peripheral vascular disease [30]; and (iv) the risk of

coronary heart disease (CHD) in a large cohort of initially healthy middle-aged men [31]. The synthesis of CRP by the liver is largely regulated by IL-6. The activated leukocyte is widely assumed to be the major source of circulating IL-6, with additional contributions from fibroblasts and endothelial cells [32].

C-reactive protein (CRP) is not seen to be directly involved in the coagulation process; it acts as a sensitive marker for inflammation [33], tissue damage and also infections [34]. CRP is seen to have a plasma half-life of about 19 hours. CRP half life remains identical under all conditions. It is in rapid in contrast to the other elements seen such coagulation proteins and all major acute-phase reactants, hence synthesis rate of CRP acts as the direct determinant of its plasma concentration [35]. Excellent anti-CRP antibodies and a well-established World Health Organization (WHO) international reference standard for CRP [36] are available, so precise, sensitive, and robust clinical serum/plasma assays can be readily undertaken [37, 38].CRP measurement thus has many advantages in the detection and monitoring of the acute-phase response in general and the relation to atheroma and its complications in particular. The recent use of the highly sensitive CRP assays was done in a large prospective study among patients with angina pectoris [39]and in initially healthy subjects [40]. There was a consistent positive association between baseline CRP levels and cardiovascular diseases. In order to prove the correlation between the CRP levels of an individual to coronary heart disease (CHD) risk, the serum CRP in 936 initially healthy men were drawn from a random sample of the general population and were measured at Augsburg in 1984 to 1985.8 years later a follow up was done where they were able to prove the prognostic significance of CRP values which were able to show the occurrence of a first major coronary event in these men [41].

Inflammatory Markers

In an effort to better identify patients at high risk for cardiovascular events; several markers of risk have been proposed to be used for screening patients and identifying those who have a high risk for cardiovascular diseases. The various markers includehomocysteine and fibrinogen levels, fibrinolytic capacity, and levels of apolipoprotein A-I, apolipoprotein B-100, and lipoprotein. However, the clinical value of many of these markers has been limited because of inadequate standardization of assay conditions, inconsistency of prospective data, or lack of evidence of

significant improvement in the prediction of risk over that afforded by standard lipid screening alone. As atherosclerosis is recognized as an inflammatory process, various plasma markers of inflammation are being investigated to see if they could behave as a marker for risk of cardiovascular diseases. There are markers of systemic inflammation produced in the liver which are, high-sensitivity C-reactive protein and serum amyloid A. Cytokines such as interleukin-6; and adhesion molecules such as soluble intercellular adhesion molecule type 1 are also seen.It is seen that these factors are proposed to predict cardiovascular diseases; however, the prognostic value remains uncertain [42].

Relationship between CRP and CVD

There has been much interest in the prognostic significance of raised levels of C-reactive protein in patients with angina [43], with the proposal that it points to release of IL-6 by activatedmacrophages in an unstable plaque [44]. More recently, however, the observations that raised concentrations of CRP in healthy subjects predicted the incidence of CHD over a period of years [45] have suggested a role for inflammation in the initiation of atherosclerosis as well as in the precipitationof an acute event. Patients with COPD had higher levels of CRP than control subjects in all studies. Even in population based studies, which are less susceptible to selection bias, a strong relationship was observed between CRP and COPD which suggests that COPD is, indeed, a risk factor for increased CRP in the community.In addition to lipid screening and the Framingham Risk Score, CRP is capable of predicting the risk for cardiovascular diseases, this gives additional prognostic information.

In outpatient settings, the primary use of CRP should be at the time of cholesterol screening, when knowledge of CRP can be used as an adjunct for global risk assessment. For individuals with LDL levels above 160 mg/dL and for whom the guidelines already call for therapeutic intervention, an elevated CRP level should aggressively encourage physicians and patients to institute pharmacological therapy in those instances where none is currently being used or where compliance is poor [46].

For individuals with LDL levels between 130 and 160 mg/dL, and if the CRP level is seen to be high, then it acts as an extra finding which leads to a higher risk for cardiovascular disease..

For individuals with LDL levels below 130 mg/dL, the finding of an elevated CRP implies substantially higher risk than predicted on the basis of LDL alone. Individuals who present with low LDL levels and high CRP levels have a higher risk of having the metabolic syndrome and fasting glucose levels must be recorded for these patients. Randomized trial is done on a large scale so that evidence is obtained before advising the patient for the statin therapy.

Framingham Risk Score assesses the patients, and only those who have intermediate risk are subjected to CRP level test. By this method unnecessary testing procedure can be avoided for patients.Clinicians can selectively pick a few individuals for whom CRP is calculated. The clinician makes the choice based on the calculated 10-year Framingham risk when it is seen to be between 5% and 20%. This approach is seen to require a second office visit and also a second phlebotomy; hence it is less efficient and perhaps less cost-effective [47].

Aggressive therapies must be started for secondary prevention; here the role of CRP is less certain. Hence LDL evaluation method is carried out so as to assess the statin efficacy.

In the setting of acute coronary ischemia and unstable angina, the role of CRP is rapidly evolving. There have been many studies conducted which shows that CRP has the ability to predict early and late death in cases of acute coronary ischemia, thus it enhances the predictive ability in addition to cardiac troponin. Knowledge of inflammatory status has been seen to be useful in distinguishing patient subgroups which are more or less likely to gain from an aggressive approach when compared to a conservative management approach [48]. However, appropriate clinical cut-points for CRP in the setting of acute ischemia remain uncertain, as does the timing of CRP evaluation in relation to the onset of ischemia. In the emergency room setting, the most common use of CRP is to detect those with chest pain syndromes and those who have negative troponin levels. If CRP level is seen to be increased in this setting, it is generally associated with long term and short term risks. More evaluation tests need to be conducted for the same. Current data states that the patients in the emergency room setting who have negative troponin levels and negative CRP levels are highly unlikely to have any flow limiting coronary disease [49].

The synthesis of CRP is predominantly under the control of IL-6 [50] which in turn has been assumed to originate largely from activated leukocytes, either in the vessel wall itself or at a

remote site of infection [51]. The correlations between these antibody titers and concentrations of fibrinogen, TNF-a, and IL-6 were weaker and generally insignificant. Endothelial dysfunction indicators were seen with the help of raised levels of CRP and also raised levels of cytokines. Tracy et al have stated that there are associations of CRP levels and a variety of measures of procoagulant activity and fibrinolysis [52]. They have suggested that this is either because of inflammation in underlying atherothrombotic disease or due to the inflammatory cells which are activated by the ongoing coagulation processes.

Goals of Screening

Cardiovascular screening programs should have their primary aim to identify the high-risk individuals. These high risk individuals can be then advised for smoking cessation, diet, exercise, and blood pressure control. It is a known fact that lifestyle recommendations is directly associated with the risk for cardiovascular disease. Using both the CRP level and the lipid evaluation, there is improved prediction of the risks for cardiac diseases seen.

It has not been found out that reduction of the CRP levels would in turn reduce the rates of the cardiac events. Studies are currently being done for the same. It has been found out that when the cardiovascular risk is reduced, there is also a reduction seen in the CRP levels. For example, reduction of weight, healthy diet, regular exercise and cessation of smoking has seen to lower both the cardiac risk as well as the CRP level.

There are several drugs which have the ability to reduce the cardiac risks which have an influence on the CRP level. Statin drugs are capable of that action; various studies have been conducted using these drugs which show an average reduction of CRP levels seen around 6 weeks after the usage of the drug. Aggressive LDL reduction is done as a critical treatment goal. LDL level evaluation must be done on a regular basis so as to monitor the statin compliance of the patients. Hence it is still regarded as one of the primary evaluation tests. LDL levels have seen to be reduced with the help of statins, but it is noted in some cases where the CRP levels reduce and other cases where the CRP levels remain unchanged [53].

Measuring CRP

In clinical practice, if the CRP level is found to be less than 10 mg/L, a single assessment is sufficient. In cases of major infections, trauma or even hospitalization, there is a raise in the CRP levels greater than 10 mg/L. This value must be ignored and another test must be scheduled on another date when the patient is seen to be clinically stable. It has been advised by many to use two measures of CRP level and then using the lower value of the two, or the average value of the two as the final level of CRP. Cholesterol evaluation is also done in order to provide a better picture on the risk of cardiac disease. There have been a few cases where the CRP is highly elevated, systemic inflammatory conditions such as lupus, inflammatory bowel disease or endocarditis must be considered. In these cases there is an increase of the erythrocyte sedimentation rate seen [54].

CRP levels are seen to be stable for long periods of time. It is not affected by food intake etc. hence it is not required to take fasting blood samples to estimate it. CRP is an acute phase reactant and its variations are seen to be similar to those associated with cholesterol screening in individuals. The individual is said to be out of risk if the CRP is within normal clinical limits [55].

The normally used assays to assess CRP are not sensitive enough to provide accurate cardiac disease risk prediction. In order to prevent this problem, highly sensitivity CRP assays were introduced and is now widely used [56]. The cost of CRP screening is comparable to that of standard cholesterol evaluation and far less than almost all other alternative approaches to cardiovascular screening under consideration. Owing to its low cost and also its long term of use, CRP screening is seen to be very efficient [57]. In many settings, the inexpensive approach of adding CRP to LDL screening may yield immediate cost-savings in terms of negative predictive value and the subsequent avoidance of unnecessary clinical testing, particularly when compared with far more expensive screening approaches such as electron beam calcium tomography or MRI.

It was noted that the CRP levels which were well within the range detected using the highly sensitivity assays have shown specificity for vascular events [58]. Some doctors have started using CRP as part of a normal routine annual check up.

Comparing CRP to Other Risk Predictors

Apart from CRP evaluation other methods such as, sophisticated measures of cytokine activity, cellular adhesion and immunologic function are able to predict myocardial infarction and stroke as they show elevated levels in those with increased vascular risk. These other methods to assess risk is not used a lot as the assays are either not appropriate for clinical use or that the protein of interest in use has a very short life period. Fibrinogen is a biomarker which is involved in both inflammation as well as thrombosis. Measuring fibrinogen is not commonly done due to limit in methodology and as it is not standardised. Systemic inflammation can be measured by using white blood cell count or by using the erythrocyte sedimentation rate. This has been proved unreliable in the clinical setting and hence is not in use. CRP evaluation is preferred [59]. Highly sensitive assays have been developed for CRP and are being standardised across the commercial platforms.Due to the high stability of CRP, it is possible to measure accurately its levels seen in both fresh and frozen plasma without the need of being collected in some special method. The stability of CRP is seen to be due to its highly stable pentraxin structure. CRP having a plasma half life period of about 19 hours owes to it being stable [60].

Evaluation of other markers such as lipoprotein(a) and homocystein are done in cases where there is premature and unexplained atherosclerosis. It is seen that the reliability of assessing CRP levels is greater when compared to these other markers. Studies indicate that CRP is a better predictor of risk when compared to the resonance based evaluation of LDL concentration and particle size [61].

Conclusion

CHD is the leading cause of death and disability in developed nations and is increasing rapidly in the developing world. Up to half of all events associated with CHD are reported to occur in apparently healthy individuals who have few or none of the traditional risk factors, including dyslipidemia. As a result, attention has increasingly turned to the role of other factors, such as inflammation, in the development of atherosclerosis and CHD. In conclusion, CRP levels seem to be correlated with levels of heart disease risk. In fact, CRP seems to predict cardiovascular risk at least as well as cholesterol levels do, thus enabling early diagnosis of CVDs and may provide new directions for prevention of cardiovascular events. .

Corresponding Author

Sreedevi Dharman

Senior Lecturer, Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

E-mail: sanjamrut@gmail.com

Telephone Number: +919841009003

References

1. Munro JM, Cotran RS. The pathogenesis of atherosclerosis: atherogenesis and inflammation. Laboratory investigation; a journal of technical methods and pathology. 1988 Mar; 58(3):249-61.
2. Alexander RW. Inflammation and coronary artery disease. N Engl J Med. 1994; 331:468–469.
3. Thompson SG, Kienast J, Pyke SD, Haverkate F, van de Loo JC. Hemostatic factors and the risk of myocardial infarction or sudden death in patients with angina pectoris. New England Journal of Medicine. 1995 Mar 9; 332(10):635-41.
4. Ross R. Atherosclerosis—an inflammatory disease. New England journal of medicine. 1999 Jan 14;340(2):115-26.
5. Pearson TA, Mensah GA, Alexander RW, Anderson JL, Cannon RO, Criqui M, Fadl YY, Fortmann SP, Hong Y, Myers GL, Rifai N. Markers of inflammation and cardiovascular disease: application to clinical and public health practice: a statement for healthcare professionals from the Centers for Disease Control and Prevention and the American Heart Association. Circulation. 2003 Jan 28; 107(3):499-511.

6. Verma S, Wang CH, Li SH, Dumont AS, Fedak PW, Badiwala MV, Dhillon B, Weisel RD, Li RK, Mickle DA, Stewart DJ. A self-fulfilling prophecy: C-reactive protein attenuates nitric oxide production and inhibits angiogenesis. Circulation. 2002 Aug 20; 106(8):913-9.
7. de Maat MP, Pietersma A, Kofflard M, Sluiter W, Kluft C. Association of plasma fibrinogen levels with coronary artery disease, smoking and inflammatory markers. Atherosclerosis. 1996 Apr 5; 121(2):185-91.
8. Berk BC, Weintraub WS, Alexander RW. Elevation of C-reactive protein in "active" coronary artery disease. The American journal of cardiology. 1990 Jan 15; 65(3):168-72.
9. Yudkin JS, Stehouwer C, Emeis J, Coppack S. C-reactive protein in healthy subjects: associations with obesity, insulin resistance, and endothelial dysfunction: a potential role for cytokines originating from adipose tissue? Arteriosclerosis, thrombosis, and vascular biology. 1999 Apr 1;19(4):972-8.
10. Bautista LE, Vera LM, Arenas IA, Gamarra G. Independent association between inflammatory markers (C-reactive protein, interleukin-6, and TNF-α) and essential hypertension. Journal of human hypertension. 2005 Feb; 19(2):149.
11. Bautista LE, Lopez-Jaramillo P, Vera LM, Casas JP, Otero AP, Guaracao AI. Is C-reactive protein an independent risk factor for essential hypertension? Journal of hypertension. 2001 May 1; 19(5):857-61.
12. Chul Sung K, Suh JY, Kim BS, Kang JH, Kim H, Lee MH, Park JR, Kim SW. High sensitivity C-reactive protein as an independent risk factor for essential hypertension. American Journal of Hypertension. 2003 Jun 1; 16(6):429-33.
13. Bautista LE, Atwood JE, O'Malley PG, Taylor AJ. Association between C-reactive protein and hypertension in healthy middle-aged men and women. Coronary artery disease. 2004 Sep 1; 15(6):331-6.
14. Rifai N, Joubran R, Yu H, Asmi M, Jouma M. Inflammatory markers in men with angiographically documented coronary heart disease. Clinical Chemistry. 1999 Nov 1; 45(11):1967-73.
15. Chae CU, Lee RT, Rifai N, Ridker PM. Blood pressure and inflammation in apparently healthy men. Hypertension. 2001 Sep 1; 38(3):399-403.

16. Yudkin JS, Stehouwer C, Emeis J, Coppack S. C-reactive protein in healthy subjects: associations with obesity, insulin resistance, and endothelial dysfunction: a potential role for cytokines originating from adipose tissue? Arteriosclerosis, thrombosis, and vascular biology. 1999 Apr 1; 19(4):972-8.
17. McCarty MF. Interleukin-6 as a central mediator of cardiovascular risk associated with chronic inflammation, smoking, diabetes, and visceral obesity: down-regulation with essential fatty acids, ethanol and pentoxifylline. Medical hypotheses. 1999 May 1; 52(5):465-77.
18. Berk BC, Weintraub WS, Alexander RW. Elevation of C-reactive protein in "active" coronary artery disease. The American journal of cardiology. 1990 Jan 15; 65(3):168-72.
19. De Beer FC, Hind CR, Fox KM, Allan RM, Maseri A, Pepys MB. Measurement of serum C-reactive protein concentration in myocardial ischaemia and infarction. Heart. 1982 Mar 1; 47(3):239-43.
20. Pietilä K, Harmoinen A, Hermens W, Simoons ML, Van de Werf F, Verstraete M. Serum C-reactive protein and infarct size in myocardial infarct patients with a closed versus an open infarct-related coronary artery after thrombolytic therapy. European heart journal. 1993 Jul 1; 14(7):915-9.
21. Haverkate E, Thompson SG, Pyke SD, Gallimore JR. Production of C-reactive protein and risk of coronary events in stable and unstable angina. The Lancet. 1997 Feb 15; 349(9050):462-6.
22. Thompson SG, Kienast J, Pyke SD, Haverkate F, van de Loo JC. Hemostatic factors and the risk of myocardial infarction or sudden death in patients with angina pectoris. New England Journal of Medicine. 1995 Mar 9; 332(10):635-41.
23. Liuzzo G, Biasucci LM, Gallimore JR, Grillo RL, Rebuzzi AG, Pepys MB, Maseri A. The prognostic value of C-reactive protein and serum amyloid a protein in severe unstable angina. New England journal of medicine. 1994 Aug 18; 331(7):417-24.
24. Biasucci LM, Vitelli A, Liuzzo G, Altamura S, Caligiuri G, Monaco C, Rebuzzi AG, Ciliberto G, Maseri A. Elevated levels of interleukin-6 in unstable angina. Circulation. 1996 Sep 1; 94(5):874-7.
25. WHO Expert Committee on Biological Standardization. WHO Technical Report Series 760. Geneva, Switzerland: World Health Organization;1987.

26. Ridker PM, Cushman M, Stampfer MJ, Tracy RP, Hennekens CH. Inflammation, aspirin, and the risk of cardiovascular disease in apparently healthy men. New England journal of medicine. 1997 Apr 3; 336(14):973-9.
27. Ridker PM, Buring JE, Shih J, Matias M, Hennekens CH. Prospective study of C-reactive protein and the risk of future cardiovascular events among apparently healthy women. Circulation. 1998 Aug 25; 98(8):731-3.
28. Tracy RP, Lemaitre RN, Psaty BM, Ives DG, Evans RW, Cushman M, Meilahn EN, Kuller LH. Relationship of C-reactive protein to risk of cardiovascular disease in the elderly: results from the Cardiovascular Health Study and the Rural Health Promotion Project. Arteriosclerosis, thrombosis, and vascular biology. 1997 Jun 1; 17(6):1121-7.
29. MRFIT Research Group, Kuller LH, Tracy RP, Shaten J, Meilahn EN. Relation of C-reactive protein and coronary heart disease in the MRFIT nested case-control study. American journal of epidemiology. 1996 Sep 15; 144(6):537-47.
30. Ridker PM, Cushman M, Stampfer MJ, Tracy RP, Hennekens CH. Plasma concentration of C-reactive protein and risk of developing peripheral vascular disease. Circulation. 1998 Feb 10; 97(5):425-8.
31. Koenig W, Sund M, Fröhlich M, Fischer HG, Löwel H, Döring A, Hutchinson WL, Pepys MB. C - reactive protein, a sensitive marker of inflammation, predicts future risk of coronary heart disease in initially healthy middle-aged men: results from the MONICA (Monitoring Trends and Determinants in Cardiovascular Disease) Augsburg Cohort Study, 1984 to 1992. Circulation. 1999 Jan 19; 99(2):237-42.
32. Heinrich PC, Castell JV, Andus T. Interleukin-6 and the acute phase response. Biochemical journal. 1990 Feb 1; 265(3):621.
33. Savitha P, Shasmitha R. Comparison of Salivary CRP Level in Chronic Periodontitis and Healthy Individual. Journal of Pharmaceutical Sciences and Research. 2015 Sep 1; 7(9):729.
34. Pepys MB. The acute phase response and C-reactive protein. Oxford textbook of medicine. 1995; 2:1527-33.
35. Vigushin DM, Pepys MB, Hawkins PN. Metabolic and scintigraphic studies of radioiodinated human C-reactive protein in health and disease. The Journal of clinical investigation. 1993 Apr 1; 91(4):1351-7.

36. WHO Expert Committee on Biological Standardization. WHO Technical Report Series 760. Geneva, Switzerland; 1987.
37. Macy EM, Hayes TE, Tracy RP. Variability in the measurement of C-reactive protein in healthy subjects: implications for reference intervals and epidemiological applications. Clinical chemistry. 1997 Jan 1; 43(1):52-8.
38. Wilkins J, Gallimore JR, Moore EG, Pepys MB. Rapid automated high sensitivity enzyme immunoassay of C-reactive protein. Clinical chemistry. 1998 Jun 1; 44(6):1358-61.
39. Haverkate E, Thompson SG, Pyke SD, Gallimore JR. Production of C-reactive protein and risk of coronary events in stable and unstable angina. The Lancet. 1997 Feb 15; 349(9050):462-6.
40. MRFIT Research Group, Kuller LH, Tracy RP, Shaten J, Meilahn EN. Relation of C-reactive protein and coronary heart disease in the MRFIT nested case-control study. American journal of epidemiology. 1996 Sep 15; 144(6):537-47.
41. Ridker PM, Cushman M, Stampfer MJ, Tracy RP, Hennekens CH. Inflammation, aspirin, and the risk of cardiovascular disease in apparently healthy men. New England journal of medicine. 1997 Apr 3; 336(14):973-9.

42. Tracy RP, Lemaitre RN, Psaty BM, Ives DG, Evans RW, Cushman M, Meilahn EN, Kuller LH. Relationship of C-reactive protein to risk of cardiovascular disease in the elderly: results from the Cardiovascular Health Study and the Rural Health Promotion Project. Arteriosclerosis, thrombosis, and vascular biology. 1997 Jun 1; 17(6):1121-7.
43. [Maseri A, Biasucci LM, Liuzzo G. Inflammation in ischemic heart disease. BMJ: British Medical Journal. 1996 Apr 27; 312(7038):1049.
44. Haverkate E, Thompson SG, Pyke SD, Gallimore JR. Production of C-reactive protein and risk of coronary events in stable and unstable angina. The Lancet. 1997 Feb 15; 349(9050):462-6.
45. Ridker PM, Cushman M, Stampfer MJ, Tracy RP, Hennekens CH. Inflammation, aspirin, and the risk of cardiovascular disease in apparently healthy men. New England journal of medicine. 1997 Apr 3; 336(14):973-9.

46. Meisinger C, Baumert J, Khuseyinova N, Loewel H, Koenig W. Plasma oxidized low-density lipoprotein, a strong predictor for acute coronary heart disease events in apparently healthy, middle-aged men from the general population. Circulation. 2005 Aug 2; 112(5):651-7.
47. Langstaff S. C-reactive protein is a potent predictor of mortality independently and in combination with troponin T in acute coronary syndromes: A Timi 11a substudy. Annals of Emergency Medicine. 1998 Nov 1; 32(5):638-9.
48. Lindmark E, Diderholm E, Wallentin L, Siegbahn A. Relationship between interleukin 6 and mortality in patients with unstable coronary artery disease: effects of an early invasive or noninvasive strategy. Jama. 2001 Nov 7; 286(17):2107-13.
49. Morrow DA, Rifai N, Antman EM, Weiner DL, McCabe CH, Cannon CP, Braunwald E. C-reactive protein is a potent predictor of mortality independently of and in combination with troponin T in acute coronary syndromes: a TIMI 11A substudy. Journal of the American College of Cardiology. 1998 Jun 1; 31(7):1460-5.
50. [50]Heinrich PC, Castell JV, Andus T. Interleukin-6 and the acute phase response. Biochemical journal. 1990 Feb 1; 265(3):621.
51. Alexander RW. Inflammation and coronary artery disease. N Engl J Med. 1994; 331:468–469.
52. Tracy RP, Psaty BM, Macy E, Bovill EG, Cushman M, Cornell ES, Kuller LH. Lifetime smoking exposure affects the association of C-reactive protein with cardiovascular disease risk factors and subclinical disease in healthy elderly subjects. Arteriosclerosis, thrombosis, and vascular biology. 1997 Oct 1; 17(10):2167-76.
53. Danesh J, Collins R, Peto R. Chronic infections and coronary heart disease: is there a link? The lancet. 1997 Aug 9; 350(9075):430-6.
54. Ridker PM, Hennekens CH, Buring JE, Rifai N. C-reactive protein and other markers of inflammation in the prediction of cardiovascular disease in women. New England journal of medicine. 2000 Mar 23; 342(12):836-43.
55. Ockene IS, Matthews CE, Rifai N, Ridker PM, Reed G, Stanek E. Variability and classification accuracy of serial high-sensitivity C-reactive protein measurements in healthy adults. Clinical chemistry. 2001 Mar 1; 47(3):444-50.

56. Roberts WL, Moulton L, Law TC, Farrow G, Cooper-Anderson M, Savory J, Rifai N. Evaluation of nine automated high-sensitivity C-reactive protein methods: implications for clinical and epidemiological applications. Part 2. Clinical chemistry. 2001 Mar 1; 47(3):418-25.
57. Blake GJ, Ridker PM, Kuntz KM. Projected life-expectancy gains with statin therapy for individuals with elevated C-reactive protein levels. Journal of the American College of Cardiology. 2002 Jul 3; 40(1):49-55.
58. Rifai N, Buring JE, Lee IM, Manson JE, Ridker PM. Is C-reactive protein specific for vascular disease in women? Annals of internal medicine. 2002 Apr 2; 136(7):529-33.
59. Aroonika SB, Vishnu P, Harsha L, Deepika V. Estimation of Serum C – Reactive Protein Levels in Patients with and without Chronic Periodontitis – A Preliminary Study, Int. J. Pharm. Sci. Rev. Res., September – October 2015; 34(1): 24-26.
60. Gussekloo J, Schaap MC, Frölich M, Blauw GJ, Westendorp RG. C-reactive protein is a strong but nonspecific risk factor of fatal stroke in elderly persons. Arteriosclerosis, thrombosis, and vascular biology. 2000 Apr 1; 20(4):1047-51.
61. Ridker PM, Rifai N, Pfeffer MA, Sacks F, Braunwald E. Long-term effects of pravastatin on plasma concentration of C-reactive protein. Circulation. 1999 Jul 20; 100(3):230-5.

Astragalus- A Review

Karthic .A [1], Lakshmi Thangavelu [2], Remmiya Mary Varghese [3]
Graduate Student [1], Assistant Professor [2], Department of Pharmacology
Senior Lecturer [3], Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Astragalus is a plant native to Asia. Astragalus has been utilized as a part of Traditional Chinese Medicine for a large number of years, regularly in mix with different herbs, to tone and reinforce the body. The Chinese name of the herb,huang qi, means "yellow leader", because the root is yellow and it is considered to be one of the most important herbs in traditional Chinese medicine. The root of the plant is used medicinally.In Chinese medicine, they traditionally convert astragalus into a decoction, it means the roots of astragalus are boiled in water and then removed. It is usually combined with other herbs, such as ginseng. In some health food stores it is found in supplement form. The potential utilisation of this herb and its compound constituents in medicines of fiery sicknesses and growths has been effectively explored lately. Astragalus-based medicines have exhibited noteworthy improvement of the danger actuated by other simultaneously controlled standard medications (e.g., immunosuppressants and tumour chemotherapeutics). The significant parts of Astragalus membranaceus are polysaccharides, flavonoids, and saponins. Contemporary utilisation of Astragalus membranaceus for the most part centers around its immunomodulating, hostile to oxidant, and calming, and also anticancer impacts

Key Words: *Astragalus membranaceous, Huang Qi, Bei Qi, Hwanggi, Milk Vetch.*

Introduction

Astragalus which is also called as milk vetch, locoweed etc belongs to the legume family Fabaceae and subfamily faboideae. It is said to be the largest genus of plants. Some species like lepidoptera uses astragalus as a food plant. It also has some uses as herbal medicine. Ithas been used in China as a popular herbal tonic for centuries. It strengthens the body's resistance to disease and increases energy by enhancing cell growth and longevity and simulating the synthesis of antibodies. It has a tonic effect on the liver, heart, lungs and kidneys. It lowers blood pressure and increases circulation. It enhances the production of interferon, which prevents viruses from replicating inside the body [1, 2].

It comes from the root of a perennial plant plant in the pea family that grows in the northern and eastern parts of China as well as in Mongolia and Korea. The root is usually harvested from four-year-old plants[3]. Astragalus is the source of a popular medicine called huang qi available in any drugstore in China for use against colds, flus and other respiratory infections. Astragalusis still widely used in China for treatment of chronic hepatitis and as an adjunctive therapy for cancer and as a folk or traditional remedy for heart disease [4, 5]. Astragalus has also been promoted for stimulation of the spleen, liver, lungs, circulatory, and urinary system, to help treat arthritis, asthma, and nervous conditions and to lower blood sugar levels and blood pressure.

Astragalus root has been used in traditional Chinese medicine for centuries as a restorative tonic; it is considered a sweet, warming herb with effects on many organs. It is used either alone or with other herbs to help with aging, improve energy, and stimulate the immune system during conditions such as the common cold, blood disorders, cancer and HIV/AIDS [6, 7]. It is also used as an adaptogen, which is meant to increase general resistance to stress and disease, and normalise disturbances in your body's ability to balance itself. The root is sold as a supplement in tablets of different dosages [8, 9].Among its diversified clinical applications; the potential use of this herb and its chemical constituents in treatments of inflammatory diseases and cancers has been actively investigated in recent years. Astragalus-based treatments have demonstrated significant amelioration of the toxicity induced by other concurrently administered orthodox drugs (e.g., immunosuppressants and cancer chemotherapeutics). The major components of Astragalus membranaceus are polysaccharides, flavonoids, and saponins[10, 11]. Contemporary use of Astragalus membranaceus mainly focuses on its immunomodulating, anti-oxidant, and anti-

inflammatory, as well as anticancer effects[12, 13, 14]. In pharmacological studies, crude extracts of Astragalus, as well as isolated constituents showed anti-inflammatory, immuno stimulant, anti oxidative, anti-cancer, anti diabetic, cardio protective, hepato protective and antiviral activities[15,16].

Astragalaus-A Supreme Protector

Astragalus -Radix Astragali

Other common names: milk vetch, huang qi

Botanical name: Astragalus membranaceus

Family: Fabaceae (pea)

Parts used: root

Properties: sweet taste, warming and moist, immunomodulator, antioxidant, hepatoprotective, cardio protective, adaptogen, diuretic

Used for: immune system dysfunction (from frequent colds and flu, to HIV, to cancer), angina, hypertension, hepatitis, fatigue, asthma, prolapsed organs, weak limbs, hepatitis, anaemia

Plant preparations: decoctions, cooked with food, powdered, capsules, tincture

Astragalus originally comes to us from China but it has quickly integrated itself into western herbalism. In a recent poll of practicing herbalists it placed as 16th in the top 50 herbs commonly used by western herbalists.

It’s important to note that the plant we use for medicine is a specific genus and species. There are over 2,000 different species in the Astragalus genus. Some of these plants are toxic and none are known to have the same qualities as Astragalus membranaceus, although a few are used medicinally.

How Does It Work?

There's a special chemical in astragalus that actually slows down the aging process right where it happens, inside of our cells, where the blueprint of our cells resides.

Recent research has shown that this special chemical derived from astragalus can "turn on" an enzyme called telomerase (hTERT). Telomerase acts to maintain or lengthen telomeres, which extend the lifespan of your DNA. If you imagine DNA as a shoelace, telomeres are the plastic aglets at each end. It serves as a protector for your DNA because it keeps it from fraying or damaging. As you age, your telomeres shorten due to wear and tear, which gives your cells an expiration date. However, telomerase helps to preserve telomeres by making them longer. Telemerase is usually "off" in adult cells, except in immune cells, in egg and sperm cells, and in malignant cells, like those found in cancer.

The length of your telomeres is important: Researchers have discovered correlations between telomere length and susceptibility to certain aging-related diseases, like cardiovascular diseases (heart attacks, atherosclerosis, and strokes), diabetes, and cancer.

Medical Uses and Indications

1. Astragalus has been used for the following:
2. Adaptogen -- protects the body from stress and disease
3. Anemia -- One early study suggested astragalus may improve blood counts in people with aplastic anemia. The study was poorly designed, so more research is needed.
4. Colds and influenza -- In TCM, astragalus is used as part of an herbal combination to prevent or treat colds, although TCM theory holds that, in some cases, it may make colds worse. Evidence in animal and laboratory tests suggests it may act against viruses like the ones that cause colds.
5. Diabetes -- Astragalus appears to lower blood sugar. More studies are needed to determine whether it can help treat diabetes.
6. Fatigue or lack of appetite from chemotherapy -- Some studies suggest astragalus may help reduce side effects from chemotherapy. The studies have not been well designed, however. More research is needed.
7. Heart disease -- Several studies suggest that astragalus may act as an antioxidant and help treat heart disease. Other studies suggest astragalus may help lower cholesterol levels.
8. Hepatitis -- A few studies have used a combination of herbs containing astragalus to treat hepatitis. Results have been mixed.
9. Kidney disease -- Preliminary research suggests astragalus may help protect the kidneys and may help treat kidney disease. More studies are needed.

10. Seasonal allergies -- One study found that astragalus may help reduce symptoms in people who have allergic rhinitis or hay fever.

Available forms

Astragalus root may be available in a variety of forms:

1. Tincture (liquid alcohol extract)
2. Capsules and tablets, standardized and non-standardized
3. Injectable forms for use in hospital or clinical settings in Asian countries
4. Topically for the skin

Research on Astragalus

Biotechnology firms are working on deriving a telomerase activator from Astragalus. The chemical constituent cycloastragenol (also called TAT2) is being studied to help combat HIV, as well as infections associated with chronic diseases or aging. However, the National Institutes of Health states: "The evidence for using astragalus for any health condition is limited. High-quality clinical trials (studies in people) are generally lacking. There is some preliminary evidence to suggest that astragalus, either alone or in combination with other herbs, may have potential benefits for the immune system, heart, and liver, and as an adjunctive therapy for cancer".

Research at the UCLA AIDS Institute focused on the function of cycloastragenol in the aging process of immune cells, and its effects on the cells' response to viral infections. It appears to increase the production of telomerase, an enzyme that mediates the replacement of short bits of DNA known as telomeres, which play a key role in cell replication, including in cancer processes.

According to the recent research, one such species of astragalus include Astragalus radix, and their chemical composition and pharmacological activities have been widely reported. Polysaccharide, triterpene and flavonoids are regarded to its effective components.Pharmacological experiments and clinical reports show that Astragali Radix has good effects on immune-regulation, anti tumour, anti-virus, diabetes, anti-aging and so on[17].

How to Take Astragalus

Paediatric

There is not a lot of scientific evidence about giving astragalus to children, so ask your doctor first. According to TCM, you should not give astragalus to a child with fever because the herb may make the fever last longer or grow stronger. Dosage should be determined by your doctor.

Adult

Dosage depends on condition being treated, age, and weight. Work with your physician to determine the safest and most effective dosage for you. Higher doses may suppress the immune system. For best results, use a standardised astragalus supplement. Recommended doses are as follows:

1. Standardized extract: 250 - 500 mg, 3 - 4 times a day standardized to 0.4% 4-hydroxy-3-methoxy isoflavone 7-sug.
2. Decoction (strong boiled tea): 3 - 6 g of dried root per 12 oz water, 3 times per day
3. Fluid extract (1:1) in 25% ethanol: 2 - 4 mL, 3 times a day
4. Powdered root: 250 - 500 mg, 3 - 4 times per day
5. Ointment: 10% astragalus applied to surface of wound. Do not apply to open wound without your doctor's supervision.
6. Tincture (1:5) in 30% ethanol: 20 - 60 drops, 3 times a day.

Effects and Safety Concerns

People with autoimmune diseases, such as Crohn's disease, multiple sclerosis, psoriasis, rheumatoid arthritis, type 1 diabetes or systemic lupus erythematous shouldn't use astragalus unless recommended by a qualified healthcare practitioner. People who have had transplant surgery should not use astragalus. The safety of astragalus in pregnant or nursing women or children isn't known.

Possible Interactions

1. Astragalus may interfere with the effectiveness of corticosteroid medications, such

Nasacort (triamcinolone)

2. Beconase, Vancenase (beclomethasone)

3. Decadron (dexamethasone)
4. Deltasone (prednisone)
5. hydrocortisone
6. Medrol (methylprednisolone)
7. prednisolone

Astragalus - Cure Disease

Immune System and Cancer

Astragalus root has been studied extensively for its effect on the immune system. It has been shown to reduce the occurrence of common respiratory illnesses, inhibit tumour growth and bolster immune system activity in general.

The studies of its use in cancer patients is astounding. It is frequently being used alongside chemotherapy to alleviate the side effects of the chemotherapy treatments. It has also been shown to inhibit the growth of tumour and bolster the immune system[1].

Research shows Astragalus root stimulates the immune system in many ways. It increases the number of stem cells in bone marrow and lymph tissue and encourages their development into active immune cells. It appears to help trigger immune cells from a “resting” state into heightened activity. One study showed Astragalus root helps promote and maintain respiratory health. It also enhances the body’s production of immunoglobulin and stimulates macrophages. Astragalus can help activate T-cells and natural killer (NK) cells[19,20].

Longevity

Astragalus seems to prolong the life of cancer patients by supporting the immune system and inhibiting cancer growth. Scientific studies have also shown that specific constituents within astragalus are highly anti-oxidant, which slows the rate of aging.

A telomere is a structure found at the end of a chromosome and is a region of repetitive DNA. Its job is to prevent deterioration of the chromosome. Shortened telomeres are associated with poor health and aging. Astragalus has also shown that it can slow telomere shortening, giving us further clues as to how to promotes longevity.

For the Heart

Astragalus has been studied extensively for its effects on improving heart function, even in patients with extreme cases such as congestive heart failure. It can also inhibit the formation of lipid peroxides and decrease blood coagulation. Another study shows that it can strengthen left ventricular function [3,4].

As a Hepatoprotective

Astragalus root has been scientifically shown to decrease the replication of the hepatitis B virus[5]. It has also been shown to prevent damage to the kidneys and liver that has been caused by either medications or virus infections.

For the Blood and Kidneys

Astragalus is also a blood tonic. It helps to regulate fluid metabolism, and those who consume it regularly are said to rarely suffer from fluid retention and bloating.

Conclusion

Astragalus root has specific and powerful applications for cancer and immune system support, however because it also contains so many protective properties (heart, liver, kidneys) it is a wonderful herb to consider for preventive care.

Corresponding Author

Remmiya Mary Varghese

Senior Lecturer, Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India
Email Id-remmiya@gmail.com
Phone Number :9840488439

Reference

1. Smith FP, Stuart GA. Chinese Medicinal Herbs, Georgetown Press. San Francisco, CA, 1973.
2. Yang S. The divine farmer's materia medica: a translation of the Shen Nong Ben Cao Jing. Blue Poppy Enterprises, Inc.; 1998.
3. Dharmananda S, The Jin-Yuan medical reforms, START Manuscripts, ITM, Portland, OR. 2001.
4. Huang Bingham and Wang Yuxia. Thousand Formulas and Thousand Herbs of Traditional Chinese Medicine, Heilongjiang Education Press, Harbin, 1993, vol(2).
5. Yang Yifan, Chinese Herbal Medicines Comparison ons and Characteristics, Churchill Livingstone, London, 2002.
6. State Administration of Traditional Chinese Medicine, Advanced Textbook on Traditional Chinese Medicine and Pharmacology, New World Press, Beijing,1995 vol(2).
7. Zhu YP, Chinese Materia Medica: Chemistry, Pharmacology, and Applications, Harwood Academic Publishers, Amsterdam,1998.
8. Dharmananda S, Flavonoids and Health, START Manuscripts, ITM, Portland, OR, 1997.
9. Matkowski A,., Flavonoids and phenol carboxylic acids in the oriental medicinal plant Astragalus membranaceus acclimated in Poland, Zeitschrift für Naturforschung 2003; 58(7-8): 602--604.
10. Tang W and Eisenbrand G, Chinese Drugs of Plant Origin, Springer-Verlag, Berlin, 1992.
11. Dharmananda S, Platycodon and other Chinese herbs with triterpene glycosides, START Manuscripts, ITM, Portland, OR, 2000.
12. Dharmananda, S, The physiological responses to immunologically-active polysaccharides, START Manuscripts, ITM, Portland, OR, 1999.
13. Ou Ming, Chinese-English Manual of Commonly Used Herbs in Traditional Chinese Medicine, Hong Kong, Joint Publishing Company,1989.

14. Auyeung KK, Han QB, KO JK. Astragalus membranaceus: a review of its protection against inflammation and gastrointestinal cancers. The American journal of Chinese medicine. 2016;44(01):1-22.
15. Li X, Qu L, Dong Y, Han L, Liu E, Fang S, Zhang Y, Wang T. A review of recent research progress on the astragalus genus. Molecules. 2014 Nov 17;19(11):18850-80.
16. Fu J, Wang Z, Huang L, Zheng S, Wang D, Chen S, Zhang H, Yang S. Review of the botanical characteristics, phytochemistry, and pharmacology of Astragalus membranaceus (Huangqi). Phytotherapy Research. 2014 Sep 1; 28(9):1275-83.
17. Zhang Q, Gao WY, Man SL. Chemical composition and pharmacological activities of astragali radix. Zhongguo Zhong yao za zhi= Zhongguo zhongyao zazhi= China journal of Chinese materia medica. 2012 Nov; 37(21):3203-7.
18. Wu JJ, Sun WY, Hu SS, Zhang S, Wei W. A standardized extract from Paeonia lactiflora and Astragalus membranaceus induces apoptosis and inhibits the proliferation, migration and invasion of human hepatoma cell lines. International journal of oncology. 2013 Nov 1; 43(5):1643-51.
19. Chen J, Wu XT, Xu YQ, Zhong Y, Li YX, Chen JK, Li X, Nan P. Global transcriptome analysis profiles metabolic pathways in traditional herb Astragalus membranaceus Bge. var. mongolicus (Bge.) Hsiao. BMC genomics. 2015 Dec; 16(7):S15.
20. Cheng J, Xiao S, Liu T. Analysis of active patents to investigate the frequency and patterns of Chinese herbal extract combinations claiming to treat heart disease. Journal of Traditional Medical Sciences. 2016 Apr 1; 3(2):81-90.
21. Ravikumar C. Review on herbal teas. J Pharm Sci & Res. 2014;6(5):236-8.

Asymptomatic Impacted Third Molar: Removal Or Retention? - A Questionnaire Based Study

Subasree.S [1], Kathiravan.S [2], Sangeetha.S [3]

Graduate Student [1], Senior Lecturer [2],Department of Oral and Maxillofacial surgery, Reader [3],Department of Prosthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Aim

To study the decisions made by dentists on removal or retention of asymptomatic impacted third molar.

Materials and method

This study recruited 100 participants (50 OMS & 50 other speciality dentists) among the Dentistsof Saveetha Dental College.The judgement on removal or retention of asymptomatic impacted third molar under various situations was assessed using a questionnaire which had 15 questions.The response was calculated in percentage and results were tabulated.Bar graphs were also plotted comparing the response given by the OMS and others for each situation.

Results

Most of the decisions made by the dentists were in accordance with clinical guidelines for management of unerupted third molars.Whereas some of the decisions were in contrary to the guidelines probably due to lack of knowledge about the guidelines among the dentists or Becauseof experience which has influenced their decisions.These conflicting results have hindered the decision making process.

Conclusion

All asymptomatic & pathology free impacted third molars need not be considered forprophylactic removal but should be reviewed periodically.

Keywords

Asymptomatic, Impaction, Wisdom tooth, Asymptomatic, Prophylaxis

Introduction

Generally third molars erupt between 18 and 24 years of age, although there can be vast Differencein eruption time.Approximately in 25% of adults one or more third molars are Absent [1-4] but in most of the elderly patients they remain impacted. The prevalence of Impacted third molars are influenced by age, gender and race. The failure of third molar Eruptionis a very common problem [5-7] and the removal of impacted third molar teeth is Oneof the surgical procedures carried out frequently in the dental clinics.Surgical removal Of impacted third molarmay be associated with certain complications postoperatively; These complications are more common in the mandible than in themaxilla; They may include haemorrhage, alveolar osteitis, nerve damage, delayedHealing, periodontal defects in adjacent tooth and infection. It is not mandatory that allthird molars should be removedeven in the absence of pathological findings, hencepatients need not unnecessarily have to accept harmful consequences associated with the surgery.

Approximately 75% of individuals who undergo regular dental care have their third molar extracted [8].Apart from pathological conditions, other criteria that justify the decision to extract third molars isfor orthodontic, prosthetic or restorative purposes [9, 10].In additions, the risks associated with the surgery are accepted by most of the surgeons, when there is clinical or radiological confirmation of periodontitis, caries, pericoronitis, damagingeffects on second molars [11,12].

Although a number of studies have been reported on third molar extraction, the inconsistent results hinder the decision-making process. This study aims in accessing the decision making of the dentists and understanding the justification for removal or retention ofasymptomatic impacted third molars under various conditions.

Materials & method

This study recruited 100 participants among the dentists of Saveetha Dental College. The participants included 50 Oral & Maxillofacial Surgeons and 50 other speciality dentists.The judgement on removal or retention of asymptomatic impacted third molar under various situations was assessed using a questionnaire which had 15 questions.The various situations

were - asymptomatic impacted third molar in a 18 year old adult, prior to orthodontic treatment to prevent late anterior crowding, pericoronitis, associated with risk of periodontal defects post operatively, fear of second molar caries, risk of root resorption in second molar, riskof development of cyst or tumors, risk of permanent parasthesia, impediment in orthognathicsurgery or prosthetic replacement.The response was calculated in percentage and results were tabulated.Bar graphs were also plotted comparing the response given by the OMS and others for each.

Results

In this study, 98% of the Oral & Maxillofacial surgeons and 55% of other speciality dentisthave the opinion that removal of asymptomatic impacted third molar is not ideal in a 18 year old adult.Whereas 2% of Surgeons and 45% of other speciality dentists believe that prophylactic removal of the impacted third molar is a better decision in this situation.

About 80% of other speciality dentists chose that extraction of the impacted third molar Prior to orthodontic treatment has to be done to prevent late anterior crowding.Whereas 87.5% of the surgeons feel that third molar can be retained as it will not cause anterior crowdingon eruption. 98% of surgeons and 85% of other dentists who participated in this study thinks that asymptomatic impacted third molar should be removed if it is associated withhigh incidence periodontal defect distal to second molars.Whereas 2% of surgeons and 15% of other dentists believe that it can be retained in such condition to prevent further complications.88% of surgeons & 55% of other dentists feel that it is better to retain the asymptomaticimpacted third molar which is associated with healthy periodontium.

About 20% of surgeons & 75% other speciality dentists consider extraction of the impactedthird molar to be the ideal treatment incase of pericoronitis.Whereas 80% of surgeons& 25% of other dentists feel that it is not necessary to remove the impacted third molarto manage pericoronitis. 96% of surgeons & 92% other dentists in this study opted removalof impacted third as the treatment to prevent second molar caries. 100% of surgeons & 98% of other dentists believe that root resorption in relation to second molar can be preventedon removal of the impacted third molars.

In the present study only 37.5% & 15% of the surgeons chose extraction of impacted third molar is better when it is associated with the risk of cyst formation & neoplasm development respectively.About 52.5% of the surgeons & 60% of the other dentists feel that it is better to retainthe asymptomatic impacted third molar as it can cause permanent parasthesia post operativelyin some cases.98% & 52% of the surgeons chose removal of the impacted third molarcan be done if it impedes orthognathic surgery & prosthetic replacement respectively.

The dentists belonging to other specialities showed greater tendency towards extraction of asymptomatic impacted third molars when compared to the surgeons. (Table 1)
Bar graphs were plotted to compare the decisions made in each situation by the surgeons & other dentists. (Fig 1 &2)

Table 1: Decisions made by Surgeons & other dentists

Questions Asymptomatic impacted Third molar	Response	Oral & Maxillofacial surgeons	Other speciality Dentists
In a 18 year old patient	Removal	2%	45%
	Retention	98%	55%
Associated with risk of late anterior crowding	Removal	12.5%	80%
	Retention	87.5%	20%
Associated with higher Incidence of periodontal Defect distal to 2nd molar	Removal	98%	85%
	Retention	2%	15%
Associated with healthy Periodontium	Removal	12%	45%
	Retention	88%	55%
Associated with mild Pericoronitis	Removal	20%	75%
	Retention	80%	25%
Associated with risk of Second molar caries	Removal	96%	92%
	Retention	4%	8%
Associated with risk of Root resorption of second molar	Removal	100%	98%
	Retention	-	2%

Associated with risk of cyst development	Removal	37.5%	75%
	Retention	62.5%	25%
Associated with risk of Neoplasm	Removal	15%	55%
	Retention	85%	45%
Associated with risk of Permanent parasthesia	Removal	47.5%	40%
	Retention	52.5%	60%
Impeding orthognathic Surgery	Removal	98%	93%
	Retention	2%	7%
Impeding denture Construction/implant Placement	Removal	52%	65%
	Retention	48%	35%

Figure 1: Bar graphs showing decisions made by surgeons and other dentists.

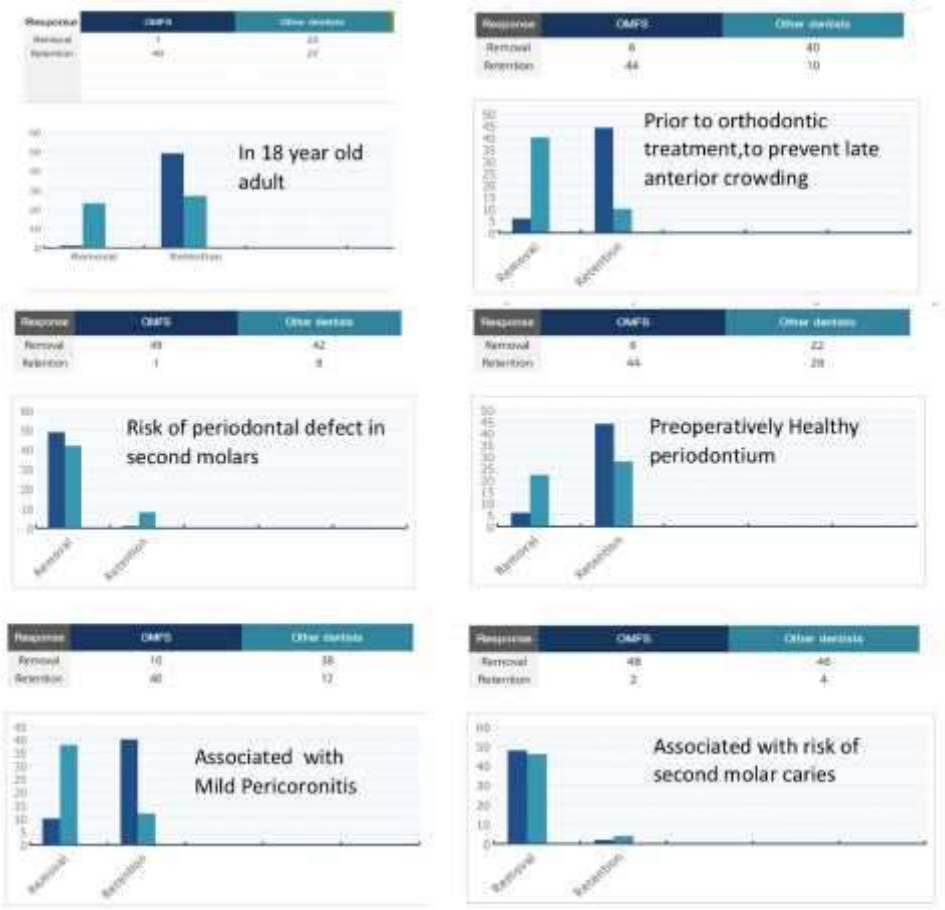


Fig 2: Bar graphs showing decisions made by surgeons and other dentists

Discussion

Tooth impaction has been a common phenomenon nowadays. Tooth impaction is a pathological situation in which a tooth is unable to erupt into its normal functioning position due to lack of space. The impaction can be mesioangular, distoangular; horizontal, transverse or vertically angulated .It is often associated with pain, pericoronitis, root resorption, cystic lesions, etc. However, there is considerable variation in the prevalence and distribution of impacted teeth in different regions of the jaw. Factors affecting the prevalence can be the age-group, timing of dental eruption, and the radiographic criteria for dental development and eruption[13]. According toSwarna et al study, third molar impactions have a mandibular predisposition, and showed a predilection towards females than males. Agenesis of third molars was more common in females than in males and was more common on the right side.The most common pattern of impaction was mesio-angular followed by vertical which is more common on the left side, horizontal which is common on the right side [14].

Mead defined an impacted tooth as a tooth which is prevented from erupting into Positionbecause of malposition, lack of space, or other impediments [15]. Peterson describedimpacted tooth as tooth that fails to erupt into the dental arch within the expected time.Later Farman proposed that impacted teeth are those that are prevented from eruptiondue to a physical barrier within the path of eruption [16]. It should be noted that any normally erupted teeth would have been unerupted or partially erupted at certain stage in the process of eruption.Hence,all unerupted or partially erupted teeth should not be considered as impacted teeth [17]. Impacted third molars may be associated with certain pathological changessuch as infections, dental caries, destruction of adjacent teeth, cysts and tumours. Although impacted third molars do not necessarily cause some of these pathological changes (such as dental caries), the impaction may increase the risk of disease, particularly when oral hygiene is poor.

As the third molars erupt completely into position anytime between 18 to 24 years,it is notideal to extract the asymptomatic impacted third molar at the age of 18.In the present study, 98% of OMS & 55% of other speciality dentists have said the asymptomatic impacted third molar need not be removed.Whereas 45% of other dentists have said that removal is better, as they probably are unaware that a tooth which impacted at 18 years of age can fully eruptby 25 years and also post operatively complications may occur after surgical extractions.

In this study, 87.5% surgeons have chosen retention of asymptomatic impacted third molar preceding orthodontic treatment.In a randomized study carried out by Harradine et al showed that the removal of third molars to prevent late incisor crowding cannot be justified [18]. Southard in his study concluded that the force generated by eruption of third molar is insufficient to significantly affect anterior crowding [19].The National Institute of Clinical Excellence (NICE) in 2000 (20) & the Scottish Intercollegiate Guidelines Network (SIGN) in 1999 reviewed in 2005 (21) did not consider late anterior crowding as a reason to justify the prophylacticremoval of third molars.About 80% of other dentists have opted for extraction of the impacted third molar prior to orthodontic treatment to prevent late anterior crowding.This is probably because they are unaware of the clinical guidelines for management of asymptomatic impacted third molar.

According to SIGN, there is a strong indication for removal of impacted third molar whenit is associated with periodontal disease in relation to second molar. In this study about98% of OMS & 85% of other dentists have opted for removal in such condition.There isa debate about the development of periodontal defect at the distal surface of the second molarsafter extraction of the impacted third molars. Some authors showed improvement of periodontalhealth distal to the adjacent second molar, while others have revealed attachment loss and reduction in level of alveolar bone [22].Only 12% of OMS opted removal, whereas 45% of other dentists chose for extraction of impacted third molar with healthy periodontium preoperatively. This is because they probably lack the knowledge about development of periodontaldefect after removal of impacted third molar.

Not all cases of pericoronitis should be treated with extraction.First episode of pericoronitis, unlesssevere should not be indicated for surgical removal of the third molar[23].About 80% of the OMS in this study are aware about this, but only 20% have opted retention of the asymptomaticimpacted third molar in case of mild pericoronitis. This is again because other specialitydentists are not familiar with the NICE guidelines.

About 96% of OMS & 92% of other dentists in this study have chosen removal of asymptomatic impacted third molar if it is associated with second molar caries.According to SIGN guidelines, there is a strong indication for removal of impacted third molar when there iscaries in the adjacent second molar, which cannot be satisfactorily treated without it's extraction [22].Walmsley et al reported that when second molar is restored but third molar is retained, recurrent caries can develop in the second molar extending to impacted adjacent thirdmolar causing loss of both the teeth [24].Thus prophylactic removal of impacted third molar which is mesioangular in position may be considered to prevent distal caries formation. Dentists who opted retention are not aware that mesioangular type of impactions can causesecond molar caries.

In this study, about 100% OMS & 98% other dentists chose removal of the impacted third molar if it is associated with the risk of root resorption of second molars.According to SIGN guidelines, third molar removal should be considered incase of root resorption of the second molar where it would be due to the third molar [22].Majority of the participants have opted thisdecision.

Around 62.5% of OMS have chosen retention of asymptomatic impacted third molar

whichis associated with risk of developing cysts which is in contrary to the clinical guidelines.This decision is probably opted as the incidence of cyst development on retention of the impacted third molar is rare (1.65%), according to Friedman et al [25].Whereas 75% of other dentists have chosen removal in such condition, possibly because they are not aware about the rareness of cyst development on retention of impacted third molars.

Similarly, tumors developing around the impacted third molar are relatively low (1.16%) [26] and so the fear of neoplasm cannot be considered as the justification for removal of asymptomaticimpacted third molar.About 85% of OMS & 55% of other dentists have given thesame opinion, which is not in accordance to the NICE guidelines. Whereas, 45% of other Dentistshave opted removal as they are again not aware about the rareness of tumors that developwhen impacted third molars are retained.

Incidence of permanent parasthesia after surgical extraction of impacted third molar rangesfrom 0.33 - 1% which is quiet rare. About 52.5% of OMS & 60% of other dentists have chosen retention of the impacted third molar maybe because the conditions where there ishigh risk of nerve injury (deflection of mandibular canal) should be taken into consideration.Whereas 47.5% of OMS opted removal maybe because they are aware that propersurgical method without excessive removal of bone & vertical sectioning of impacted teeth will prevent permanent parasthesia. But 40% of other dentists have opted removal probably because they are not aware that permanent parasthesia can occur postoperatively.

According to AAOMS, there is a view that when an impacted third molar may Complicateorthognathic surgery,then it is reasonable to remove that tooth, provided the risks of complications do not outweigh the benefits [27].About 98% of OMS & 93% of other dentistsin this study have also opted removal, which is in according to the report submitted WithAAOMS.

According to SIGN guidelines, removal of an impacted third molar close to the alveolar surface should be considered preceding denture construction or implant placement.About

52% of OMS & 65% of other dentists have chosen removal as their decision which is in agreementwith the clinical guidelines.Whereas 48% of OMS & 35% of other dentists feel thatprosthetic replacement is not a justification for removal of asymptomatic impacted third molarconsidering the complications which may arise after surgery.

There are no previous questionnaire based study carried out among the dentists about theirdecision on management of asymptomatic impacted third molars.This is a first study of thatkind.It is understood from the results that majority of the OMS have good knowledge aboutthe clinical guidelines for management of unerupted third molar than other speciality dentists.There are some conditions where decisions made by the dentists are not in agreement with the guidelines, probably because their experience influences their decision.The dentists belonging to other speciality lack proper decision making ability & showed greater tendency towards extraction of asymptomatic impacted third molars.Even though they do not have amajorrole in decision making about the management of asymptomatic impacted third molar, they are the ones who are going to refer the patients to a surgeon when they identify anasymptomaticimpacted third molar. Hence, some methodology should be employed forimplementationof clinical guideline on management of asymptomatic mandibular thirdmolar which can improve dentists' knowledge on this topic and their decision making ability.

Conclusion

From this study it is conclusive that general dental practitioners show greater tendency Towardsextraction when compared to Oral & Maxillofacial surgeons.All asymptomatic & Pathologyfree impacted third molars need not be considered for prophylactic removal but Shouldbe reviewed periodically.

Corresponding Author

Sangeetha.S

Reader,Department of Prosthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

E-Mail: dr.sangeethakiruba@gmail.com

Telephone Number: +919894067220

References

1. Rantanen AV. The age of eruption of the third molar teeth. Acta Odontology Scand 1967; 25: Suppl 48.
2. Song F, Landes DP, Glenny AM, Sheldon TA. Prophylactic removal of impacted third molars: an assessment of published reviews. British dental journal. 1997 May 10; 182(9):339.
3. von Wowern N, Nielsen HO. The fate of impacted lower third molars after the age of 20. A four-year clinical follow-up. International journal of oral and maxillofacial surgery. 1989 Oct 1; 18(5):277-80.
4. Levesque GY, Demirjian A, Tanguay R. Sexual dimorphism in the development, emergence, and agenesis of the mandibular third molar. Journal of Dental Research. 1981 Oct; 60(10):1735-41.
5. Garcia RI, Chauncey HH. The eruption of third molars in adults: a 10-year longitudinal study. Oral Surgery, Oral Medicine, Oral Pathology. 1989 Jul 1; 68(1):9-13.
6. Bell GW. Use of dental panoramic tomographs to predict the relation between mandibular third molar teeth and the inferior alveolar nerve: Radiological and surgical findings, and clinical outcome. British Journal of Oral and Maxillofacial Surgery. 2004 Feb 1; 42(1):21-7.
7. Hugoson A. The prevalence of third molars in a Swedish population: an epidemiological study. Community Dent Health. 1988; 5:121-38.

8. Schulhof RJ. Third molars and orthodontic diagnosis. Journal of clinical orthodontics: JCO. 1976 Apr; 10(4):272-81.
9. Chaparro-Avendaño AV, Pérez-García S, Valmaseda-Castellón E, Berini-Aytés L, Gay-Escoda C. Morbidity of third molar extraction in patients between 12 and 18 years of age. Medicina oral, patologia oral y cirugia bucal. 2005; 10(5):422-31.
10. Kruger E, Thomson WM, Konthasinghe P. Third molar outcomes from age 18 to 26: findings from a population-based New Zealand longitudinal study. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics. 2001 Aug 1; 92(2):150-5.
11. Marciani RD. Third molar removal: an overview of indications, imaging, evaluation, and assessment of risk. Oral and Maxillofacial Surgery Clinics. 2007 Feb 1; 19(1):1-3.
12. Bagheri SC, Khan HA. Extraction versus nonextraction management of third molars. Oral and maxillofacial surgery clinics of North America. 2007 Feb 1; 19(1):15-21.
13. Kumar MS, Aysha S. Angulations of impacted mandibular third molar: a radiographic study in Saveetha Dental College. J Pharm Sci Res. 2015; 7:981-3.
14. Sivaramakrishnan SM, Ramani P. Study on the Prevalence of Eruption Status of Third Molars in South Indian Population. Biology and Medicine. 2015 Oct 1; 7(4):1.
15. Archer WH. Oral Surgery: A Step-By-Step Atlas of Operative Techniques, WB Saunders Company. Philadelphia, 1966; 4th ed. Chapter 4: 122. 1966; 237.
16. Peterson LJ. Principles of Management of Impacted Teeth. In: Peterson LJ, Ellis E III, Hupp JR, Tuker MR, editors. Contemporary Oral and Maxillofacial Surgery, 3rd ed. St. Louis: Mosby; 1998. P. 215-48.
17. Agarwal KN, Gupta R, Faridi MM, Kalra N. Permanent dentition in Delhi boys of age 5-14 years. Indian Pediatr. 2004 Oct; 41(10):1031-5.
18. Mettes TD, Ghaeminia H, Nienhuijs ME, Perry J, Van der Sanden WJ, Plasschaert A. Surgical removal versus retention for the management of asymptomatic impacted wisdom teeth. Cochrane Database Syst Rev. 2012 Jan 1; 6.
19. Harradine NW, Pearson MH, Toth B. The effect of extraction of third molars on late lower incisor crowding: a randomized controlled trial. Br J Orthod. 1998 May; 25(2):117-22.

20. Southard TE, Southard KA, Weeda LW. Mesial force from unerupted third molars. American Journal of Orthodontics and Dentofacial Orthopedics. 1991 Mar 1; 99(3):220-5.
21. NICE. Guidance on the Extraction of Wisdom Teeth. London: National Institute for Clinical Excellence; 2000.
22. SIGN. Management of Unerupted and Impacted Third Molar Teeth. Edinburgh: Scottish Intercollegiate Guidelines Network; 1999.
23. Marmary Y, Brayer L, Tzukert A, Feller L. Alveolar bone repair following extraction of impacted mandibular third molars. Oral Surgery, Oral Medicine, Oral Pathology. 1986 Apr 1; 61(4):324-6.
24. Sheikh MA, Riaz M, Shafiq S. Incidence of distal caries in mandibular second molars due to impacted third molars-A clinical and radiographic study. Pakistan Oral & Dental Journal. 2012 Dec 1; 32(3).
25. Al-Khateeb TH, Bataineh AB. Pathology associated with impacted mandibular third molars in a group of Jordanians. Journal of oral and maxillofacial surgery. 2006 Nov 1; 64(11):1598-602.
26. Friedman JW. 20th century dental (mal) practice in the 21st century. J Calif Litigation. 2004; 17:37–40.サ
27. Jay W. Friedman, DDS, and MPH.The Prophylactic Extraction of Third Molars: A PublicHealth Hazard, American Journal of Public Health. 2007; 97(9):1554-1559.
28. AAOMS. Report of a workshop on the management of patients with third molar teeth. J Oral Maxillofacial Surg 1994; 52:1102-12.

Awareness of Complications in Chronic Kidney Disease Patients

Nivedha Srinivasan [1],Halleys Kumar [2],S.Niveda [3]

Graduate Student [1], Senior Lecturer [2], Department of General Medicine,

Tutor [3],Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Chronic kidney disease includes conditions that damage your kidneys and decrease their ability to keep you healthy by doing the jobs listed. If kidney disease gets worse, wastes can build to high levels within the blood.Earlier recognition of chronic kidney disease (CKD) could slow progression, prevent complications, and reduce cardiovascular-related outcomes. However, current estimates of CKD awareness indicate that both patient- and provider-level awareness remain unacceptably low. Many of the factors that are possibly associated with CKD awareness, which could help guide implementation of awareness efforts, have yet to be fully examined.

***Key words**: Chronic kidney disease, cardiovascular, blood, Complications*

Introduction

Chronic kidney disease (CKD) is a public health concern, affecting an estimated 35% of Indian population [1]. CKD is associated with an increased risk of cardiovascular disease and chronic renal failure. Better management of CKD can slow progression of renal dysfunction, prevent metabolic complications, and reduce cardiovascular-related outcomes [2]. Physician awareness of disease is critical for implementation of evidence-based therapies, and patient awareness is a major determinant of adherence to those therapies. Long term hypertension is a risk factor for many diseases, including heart disease, stroke and kidney failure

Awareness of CKD remains unacceptably low, despite recent attempts to increase awareness through dissemination of clinical practice guidelines and recommendations for patients with CKD or its risk factors to providers [3-5], community awareness events such as world kidney day [6,7], and free screening efforts for high-risk individuals like the kidney early evaluation program (keep) [8,9]. Earlier recognition of CKD could lead to slow progression, prevent complications, and reduce cardiovascular-related outcomes; additionally, early referral to nephrologists has been shown to improve outcomes for those who progress to end-stage renal disease.

We examined whether well-known manifestations of kidney disease were associated with greater individual CKD awareness. We hypothesized that participants who exhibited markers of kidney dysfunction for any given level were more likely to have a provider who detected CKD and communicated the individual's CKD status to the participant, resulting in greater individual awareness of CKD.

Materials and Method

A questionnaire based study on 50 individual CKD patients was conducted and compared. To find the epidemiological variations with relation to CKD among the population using a questionnaire based study within Saveetha medical college.

Self-reported sociodemographics (age, gender, race/ethnicity, marital status, social support, primary language, education, and income), access to care (insurance and routine site for medical care), and diagnoses (CKD and Diabetes) were obtained during interviews.

Questions like at which stage where they were diagnosed with ckd and the awareness of the symptoms of CKD such as bone diseases, anemic, heart diseases, edematous lesions were asked in the questionnaire.

Results

The questionnaire based study was conducted among 50 individuals who were either diagnosed with CKD or was a relative to the patient diagnosed with CKD. It was found out that 68% of the patients were diagnosed with chronic kidney disease at earlier stage itself.

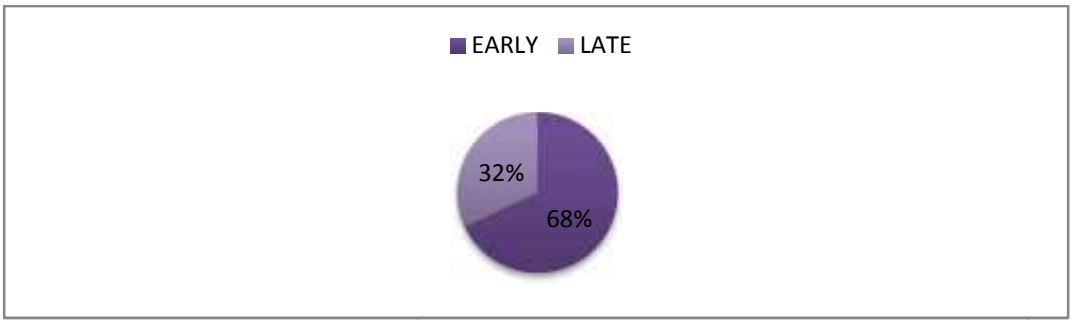


Figure 1: Pie chart depicts the stage at which CKD was diagnosed

When the prevalence risk ratio was conducted, awareness was less in males when compared to females.

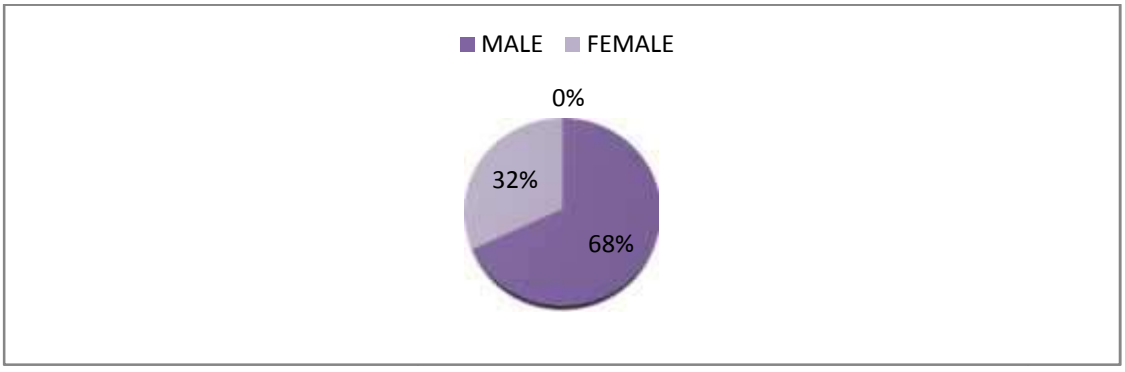


Figure 2: Pie Chart depicts the awareness of CKD in males and females.

Half of the cases of CKD (49.1%) were not associated with any of the measured risk factors of hyper tension, diabetes or HIV.

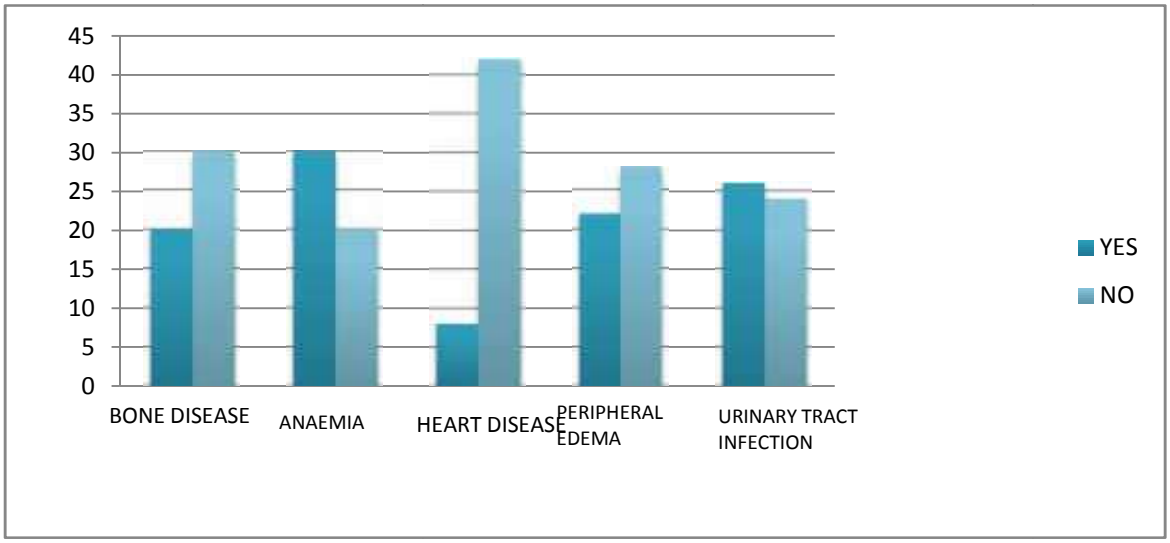


Figure 3: Bar graph depicts the awareness of risk factors associated to CKD

Many patients were aware of the urinary tract infection and anemia due to CKD. But a major amount of patients were not aware that heart, bone diseases and peripheral edema were also symptoms of CKD.

Discussion

Although research among CKD patients has shown that greater knowledge of their condition leads to better clinical outcomes [10,11] little data are available on whether awareness of earlier-stage CKD is associated with better clinical outcomes. There is some evidence that those with higher perceived risk for ckd are more likely to seek information, speak to their doctor about ckd, and get tested [12]. Interestingly, those with the highest and lowest perceived risk were less likely than those with intermediate perceived risk to have controlled blood pressure [13]; for those with the highest perceived risk, psychological factors such as those mentioned above may explain this result. Awareness of CKD status was not generally associated with achieving target blood glucose regardless of diabetes status in the keep study [14], but CKD awareness was associated with lower hemoglobin a1c in a group of primary care patients with diabetes and ckd [15]. Further research is necessary to determine whether—and for whom—CKD awareness leads to better self-management and better clinical outcomes (particularly, decreased hospitalization, cardiovascular events, and/or mortality). Additionally, the possibly negative consequences of CKD knowledge and awareness, including excess worry about risk and decreased quality of life

and depression associated with the diagnosis of chronic disease, should also be explored and weighed against any improvements in clinical outcomes.

Surveys demonstrate high levels of knowledge of diabetes and hypertension as risk factors, with 99% of family practice and internal medicine trainees identifying them as strong risk factors in web-based questionnaires [16, 17]. Minority status, age, and family history of CKD were less well-recognized as risk factors. A survey of Pcps practicing in predominantly African-American communities also revealed that a third of respondents (34.4%) did not consider family history of kidney disease to be associated with increased risk of CKD and 22% did not consider African American race to be a risk factor for development of CKD [18].

Early referral of patients to nephrologists is optimal, but given the relatively small number of practicing nephrologists nationwide, nephrologists cannot exclusively manage all patients with CKD [19]. The awareness of CKD is unacceptably low and knowledge of CKD management is particularly poor among the patients even though the data is very limited. Further exploration of the factors associated with decreased CKD knowledge and the impact of CKD awareness on individual patient outcomes and public health outcomes is necessary to guide awareness efforts moving forward [20].

Conclusion

CKD in its early stages is usually silent and without remarkable symptoms, patients may not be aware of their disease. Even persons who have been identified as having early-stage CKD by evidence of kidney damage or reduced kidney function through regular screening may not understand their diagnosis or recognize the importance of treatment. Better management of CKD can slow the progression of CKD, prevent complications, and reduce cardiovascular-related outcomes.

Corresponding Author

S.Niveda,

Tutor, Department of Public Health Dentistry

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email: drnivedasubash@gmail.com,

Telephone Number: +919952979464

References

1. Plantinga Lc, Boulware Le, Patient Awareness of Chronic Kidney Disease: Trends and Predictors. Arch Intern Med 168:2268–2275
2. Sarnak Mj, Levey As: Kidney Disease As A Risk Factor For Development Of Cardiovascular Disease: A Statement From The American Heart Association Councils On Kidney In Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, And Epidemiology And Prevention. Hypertension 2003.42:1050–1065.
3. American Da. Standards of Medical Care in Diabetes-2009. Diabetes Care. 2009;32:S13–S61
4. Chobanian Av, BakrisGl, Black Hr, Cushman Wc, Green La, IzzoJl, Jr, Jones Dw, MatersonBj, Oparil S, Wright Jt, Jr, RoccellaEj. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment Of High Blood Pressure: The Jnc 7 Report. Jama. 2003; 289(19):2560–2572.
5. LeveyAs, Atkins R, Coresh J, Cohen Ep, Collins Aj, Eckardt Ku, Nahas Me, JaberBl, Jadoul M, Levin A, Powe Nr, Rossert J, Wheeler Dc, Lameire N, Eknoyan G. Chronic Kidney Disease As A Global Public Health Problem: Approaches And Initiatives - A Position Statement From Kidney Disease Improving Global Outcomes. Kidney Int. 2007;72(3):247–259
6. LeveyAs, AndreoliSp, Dubose T, Provenzano R, Collins Aj. Chronic Kidney Disease: Common, Harmful and Treatable--World Kidney Day 2007. Am J Nephrol. 2007; 27(1):108–112.
7. Chin Hj, AhnJm, Na Ky, ChaeDw, Lee Tw, HeoNj, Kim S. The Effect Of The World Kidney Day Campaign On The Awareness Of Chronic Kidney Disease And The Status Of Risk Factors For Cardiovascular Disease And Renal Progression. Nephrol Dial Transplant. 2009 In Press.

8. Bakris G, Collins Aj. Executive Summary: Kidney Early Evaluation Program (Keep) 2007 Annual Data Report. Am J Kidney Dis. 2008; 51(4):S1–S2.
9. VassalottiJa, Li S, Chen Sc, Collins Aj. Screening Populations at Increased Risk ofCkd: The Kidney Early Evaluation Program (Keep) And The Public Health Problem. Am J Kidney Dis. 2009; 53(3 Suppl 3):S107–S114.
10. Cavanaugh Kl, MerkinSs, PlantingaLc, Fink Ne, Sadler Jh, Powe Nr. Accuracy Of Patients' Reports Of Comorbid Disease And Their Association With Mortality In Esrd. Am J Kidney Dis. 2008; 52(1):118–127.
11. Grubbs V, Gregorich Se, Perez-Stable Ej, Hsu Cy. Health Literacy and Access To Kidney Transplantation. Clin J Am SocNephrol. 2009;4(1):195–200.
12. Waterman Ad, Browne T, Waterman Bm, Gladstone Eh, Hostetter T. Attitudes And Behaviors Of African Americans Regarding Early Detection Of Kidney Disease. Am J Kidney Dis. 2008; 51(4):554–562.
13. Boulware Le, Carson Ka, Troll Mu, Powe Nr, Cooper La. Perceived Susceptibility To Chronic Kidney Disease Among High-Risk Patients Seen In Primary Care Practices. J Gen Intern Med. 2009;24(10):1123–1129
14. Cavanaugh Kl, Wright J, WallstonKe, Elasy Ta, Ikizler Ta, Rothman Rl. Effect OfCkd Awareness On Diabetes Control And Self-Care Is Modified By Health Literacy [Abstract] J Am SocNephrol. 2006; 20 421a.
15. Whaley-Connell A, Sowers Jr, Mccullough Pa, Roberts T, Mcfarlane Si, Chen Sc, Li S, Wang C, Collins Aj, Bakris Gl. Keep Investigators: Diabetes Mellitus And Ckd Awareness: The Kidney Early Evaluation Program (Keep) And National Health And Nutrition Examination Survey (Nhanes) Am J Kidney Dis. 2009;53(44):S11-S21
16. Agrawal V, GhoshAk, Barnes Ma, Mccullough Pa. Perception Of Indications For Nephrology Referral Among Internal Medicine Residents: A National Online Survey. Clin J Am SocNephrol. 2009;4(2):323–328.
17. Lenz O, Fornoni A. Chronic Kidney Disease Care Delivered By Us Family Medicine and Internal Medicine Trainees: Results From An Online Survey. 2006; 4:30.
18. Lea Jp, McclellanWm, Melcher C, Gladstone E, Hostetter T. Ckd Risk Factors Reported By Primary Care Physicians: Do Guidelines Make a Difference? Am J Kidney Dis. 2006; 47(1):72–77.

19. M. Ashfaq Ahmed. Awareness Of Blood Pressure And Its Treatment In Op Patients Of Saveetha Dental College, Int. J. Pharm. Sci. Rev. Res.2017; 43(1)
20. T. Lakshmi, ThirumalaikumaranRathinam.D. Ezhilarasan, Novel Strategies for The Management Of Renal Replacement – A Review 2017; 7(1)

Beneficial Effects of Murraya Koeinjii inModern Medicine

Vidhya.T.K[1],Anitha Roy[2], Saravana Pandian[3],

Graduate Student[1],Head[2], Department of Pharmacology, Senior Lecturer[3], Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Murraya koienjii is also known as curry leaves. Several studies have been done regarding the therapeutic uses of Murraya koienjii. This study is to compile the overall usefulness of this plant. Leaves are used as herb in ayurvedic medicine and it has an anti-diabetic properties. Murraya koeinjii is widely used as a nephroprotective against kidney infirmities in Malaysia. Dementia is a common disorder in older patients, leaves have many beneficial effects, it is used in the management of alzheimers disease and dementia. Girinimbine, a carbazole alkaloid isolated from stem bark is also used in the treatment of breast cancer. Leaves are also used in the growth of hair and helpful for good vision. Pharmacological activities of the plant are activity on heart, anti-diabetic and cholesterol reducing property, anti-microbial activity, anti-ulcer activity, anti -oxidative property, cytotoxic activity, anti-diarrhoea activity, phagocytic activity and many more medicinal values.Being a plant with much therapeutic potential to bring an awareness of this plant.

Keywords: Murraya Koienjii, anti bacterial, anti ulcer, anti oxidative, anti diarrhoea.

Introduction

India is one of the pioneers in the discovery of herbal medicines for the treatment of various ailments. *Murraya koemji*i is an important spice in Asia. It belongs to family *Rutaceae*. A few metres high shrub; the main stem, dark green to brownish, with numerous dots on it; its bark can be peeled off longitudinally, exposing the white wood underneath; the girth of the main stem is 16 cm(1). Leaves, exstipulate, bipinnately compound, 30 cm long, each bearing 24 leaflets, having reticulate venation; leaflets, lanceolate, 4.9 cm long, 1.8 cm broad, having

0.5-cm-long petiole(2). Murraya koeinjii leaves are used as flavouring, condiment, and folk medicine for the treatment of various metabolic and infectious disease(3,4).It is an antiemetic, anti-diarrhoeal, febrifuge and blood purifier, phytochemical screening of Murraya koeinjii leaves revealed the presence of some vitamins(A,B), carbazole alkaloid(kurryam, koenimbine and koenine) , trepenoids, phenolic compounds, and mineral contents such as calcium, iron, zinc and vanadium. Besides it has no side effects and can cure the patient for any kind of infectious disease. (5) The green leaves are stated to be eaten raw for curing dysentery, and the infusion of the washed leaves stops vomiting. Curry leaves are also used in calcium deficiency. Its nutritional value benefits both the young and the old alike(6). Women who suffer from calcium deficiency, osteoporosis etc. can find an ideal natural calcium supplement in curry leaves. Fresh juice of curry leaves, with lime juice and sugar, is an effective medicine in the treatment of morning sickness, nausea and vomiting (7). In the present review, Murraya koeinjii was chosen since it is one of the most widely acclaimed remedies for the treatment of many disease.

Figure 1: Inflorescenceand Leavesof MurrayaKoenjii

Traditional Uses

Fresh leaves, dried leaf powder, and essential oil are widely used for flavouring soups, curries, fish and meat dishes, eggs dishes, traditional curry powder blends, seasoning and ready to use other food preparations. The essential oil is also utilized by soap and cosmetic aromatherapy industry.16 Curry leaves are boiled with coconut oil till they are reduced to blanked residue which is then used as an excellent hair tonic for retaining natural hair tone and stimulating hair growth. It is traditionally used as a whole or in parts as antiemetics, antidiarrheal, febrifuge, blood purifier, antifungal, depressant, anti-inflammatory, body aches, for kidney pain and vomiting.

Phytochemistry

Mature leaves contains 63.2 % moisture, 1.15 % total nitrogen, 6.15 % fat, 18.92 % total sugars, 14.6 % starch, 6.8 % crude fiber, ash 13.06 %, acid insoluble ash 1.35 %, alcohol soluble extractive 1.82%, cold water (20˚C) extractive 27.33% and a maximum of hot water soluble extractive 33.45%. 30 Constituents that have been stimulated the most interest includes a wide range of carbazole alkaloids, essential oil and carotenoids. The following major group of bioactive constituents summarizes the constituents of murraya.

Carotenoids

Leaves contain 9744 ng of lutein, 212 ng of α-tocopherol and 183 ng of carotene/g of fresh weight. 31 21.4 mg/100 g of total carotene, 7.1 mg/100 g of β-carotene is reported by Bhaskaracharyet. al. 32 E. Siong Tee has reported 14570-µg/100 g of total carotenoids in leaves as measured by HPLC. Out of total carotenoids, lutein content was 5252 and β-carotene content was 9328 µg /g. 33

Carbazole Alkaloids

Leaves: Tachibana et.al has isolated 8, 10’-{3,3’,11, 11’-tetrahydro-9,9’ dihydroxy- 3,3’,5, 8’-tetra methyl –3,3’-bis (4-methyl-3-pentenyl)}bispyrano (3,2 a) carbazole (a dimericcarbazole alkaloid) from methylene chloride Vandana Jain International journal of ayurvedic & herbal medicine 2(4) aug . 2012(607-627) 609 extract of M. koenigii leaves together with six known alkaloids; koenimbine, O- methyl murrayamine, Omethylmahanine, isomahanine and bismahanine and bispyrayafoline. 34,35 From dried leaves glycozoline,36 1-formyl –3 methoxy- 6-methyl carbazole and 6, 7- dimethoxy- 1- hydroxy- 3-methyl

carbazole 37 was isolated. Koenigine, koenine, koenidine and (-) mahanine were isolated from acetone extract of leaves.Form the hexane extract of leaves Joshi et.al has isolated mahanimbine, isomahanimbine, koenimbidine and murrayacine.Isomahanimbicine was isolated form petroleum ether extract of leaves of M. koenigii specifically collected in the month of February 38. Euchrestine B, mahanine, mahanimbicine, mahanimbine 35, bismurrayafoline E 35,40, mahanimbicine, bicyclomahanimbicine 41, cyclomahanimbinebicyclomahanimbine, mahanimbidine 42, mukonicine 43, 8, 8"-biskoenigine, new binary carbazole alkaloid along with its monomer koenigine 44 and a minor alkaloid mahanine 45 were identified and isolated from leaves of M. koenigii. Gupta et.al has reported the presence of murrayanine (0.32%), glycoside scopolin (0.25%), free glucose (3.5%) and ash (10.4%) 46. Arial part is reported to contain murrayanine and 8,8" biskoenigine 47. Patroleum ether extract of leaves was used to isolate carbazole alkaloids, mahanimbine(3,5- dimethyl-3-(4-methylpent-3-enyl)-11H-pyrano[5,6-a] carbazole)48 . Methanolic extract of M. Koenigii was subjected to qualitative thin-layer chromatography and HPLC using different solvent system by Gupta et.al. Spectral analysis (IR, 1H NMR, 13C NMR and MS) was carried out to establish the structure. The structures of these 6-bioactive compounds confirmed as carbazole alkaloids- Mahanimbine, Girinimbine, Isomahanimbine, Murrayazoline, Murrayazolidine, and Mahanine, by the spectrometric data. 49 Stem: From alcohol extract of stem bark Saha et.al has isolated koenigine- quinone A and koeniginequinone B, structures were established as 7- methoxy- 3 methyl carbazole- 1,4-quinone and 6, 7-dimethoxy-3-methyl carbazole-1, 4- quinone respectively 50. 9-carbethoxy-3-methyl carbazole and 9- formyl –3- methyl carbazole were identified form M. koenigii by Chakrabortyet. al 51. Me- 2- methoxy carbazole –3- carboxylate and 1- hydroxy –3- methyl carbazole were isolated form stem bark52. Mukonal, a probable biogenetic intermediate of pyranocarbazole alkaloid was detected in stem bark.53 From stem bark Murrayazolinol (a minor carbazole alkaloid)54, mahanimbinol55 , murrayazolidine56, 57, murrayacinine58, mukonidine59, murrayazolinine60, murrayanine, girinimibine and mahanimbine61, girinimbinol and mahanimbilol62 (possible biogenetic precursors of girinimbine and mahanimbine) has also been identified and isolated. Roots: Murrayanol, murrayagetin, marmesin- 1"- O- rutinoside were isolated form root extract63. Three monomeric and five binary carbazole alkaloids named mukoenine- A, -B and C and murrastifoline –F. bis – 2- hydroxy3- methyl carbazole, bismahanine, bi koeniquinone- A and bismurrayaquinone A were isolated form root and stem bark64. Koenoline (1- methoxy-3-hydroxy methyl carbazole) was isolated form the root bark65, Mukoline, mukolidine were

isolated form the benzene extract of roots63. Roots were also found to contain girinimbine39, 66. Seeds: Mahanimbine, girinimbine, koenimbine, isomahanine and mahanine were isolated form seeds of M. koenigii from Marassana, Sri Lanka 67. 2- methoxy-3- methyl carbazole was isolated form petroleum ether extract of seeds68 . Mandal et.al isolated three bioactive carbazole alkaloids, kurryam (I), Koenimbine (II) and koenine (III) with structural confirmation with 2D-NMR spectra69 Fruits: Mahanimbine and koenimbine were isolated from petroleum ether extract of fruits 38,70. Isomahanine and murrayanol were isolated form fruits by Reischet. al along with five previously reported carbazole alkaloids mahanimbine, murrayazolidine, girinimbine, koenimbine and mahanine71. Coumarin: Indicolactone, anisoalctone and 2', 3' epoxy indicolactone (a furocoumarin lactone) were isolated from the seeds. This represents the first furocoumarin with a mono terpenoid lactone chain in the genus Murraya72. Vandana Jain International journal of ayurvedic & herbal medicine 2(4) aug . 2012(607-627) 610 Adebajoet. al has reported xanthotoxin, isobyaknagelicol, byakangelicol and isogosferol as minor furocoumarins in seeds of M. koenigii73. Isoheraclenin, isoimperatonin, oxypeucedanin, isopimpinellin and bergaptan were isolated form seeds of Marassana village, Sri Lanka, suggesting it as a new chemical race74. A new coumaringalactosidemarmesin- 1'-O-β-D-galactopyranoside, osthol and umbelliferone were isolated form ethanol stem bark extract75 . 3- (1,1- dimethyl allylxanthyletin) was isolated form petroleum ether extract of stem bark of M. koenigii76.

Pharmacological Activities

Antifungal Activity

Acetone extract of M. koenigii is active against Aspergillus niger, benzene extract is most active against Alternaria solani and Helminthosporium solani and ethanol extract is active against Penicillum notatum(8)

Antioxidant Activity

Antioxidants stop unwanted oxidation in the body, which involve the formation of free radicals and further deteriorate the condition of the body .(9) Reactive oxygen species (ROS), causing damage to DNA, proteins and lipids, have been associated with carcinogenesis, coronary heart disease and many other health problems. . The most commonly added antioxidants are synthetic phenols, such as butylated hydroxy toluene (BHT) and butylated hydroxy anisole (BHA). Their safety however is doubtful. (10).

Cytotoxic Activity

Alkaloid Koenoline isolated from the root bark of M.koenigii is found to exhibitcytotoxic activity against KB cell culture system (11). Carbazole alkaloids isolated from the stems are found to effects the growth of the human leukemia cell line HL-60. Mahanine, Pyrafoline-D and murrafoline-I (Carbazole alkaloids) showed significant cytotoxicity against HL-60 cells and also induced loss of mitochondrial membrane potential.

Radiation Protection Activity

The effect of 4 Gy gamma radiation 30 min after the last injection of 100 mg/kg of methanolic extract of M. koenigii for 5 consecutive days was observed on adult Swiss albino mice. The extract itself increased the glutathione and enzymes levels, whereas radiation significantly reduced all values. Pretreatment with the extract reduced lipid peroxidation rate induced by radiation. The result demonstrated that M. koenigii leaves possess good antioxidant activity in vitro and are able to protect against radiation-induced depletion in cellular antioxidants152 Themethanolic extract showed protection against gamma radiation and cyclophosphamide-induced chromosomal damage in Swiss albino mice at a single dose of 100 mg/kg body weight153

Antiulcer Activity

Anti ulcer activity was observed using aqueous extract at doses of 200 and 400 mg/kg. It produced significant inhibition of gastric lesion induced by non-steroidal anti-inflammatory drugs and pylorus ligation-induced ulcer. The extract reduced ulcerative lesion, gastric volume and free and total acidity but raised the pH value of gastric juice in pylorus ligation model. The results obtained suggested that the extract possesses significant antiulcer activity154.

Antidiarrhoeal Activity

Bioactive alkaloids, kurryam and koenimbine obtained from fractionated n-hexane extract of the seeds of M. koenigii were found to exhibit inhibitory activity against castor oil-induceddiarrhoea and prostaglandin E2-induced enteropooling in rats in charcoal meal test in

Wister rats, these compounds were found to exhibit significant reduction in gastrointestinal motility(13). The bioassay guided fractionation of the n-hexane extract of the seeds of M. koenigii resulted in the isolation of three pure compounds of bioactive carbazole alkaloids, kurryam, koenimbine and koenine(14,15). Of the three compounds kurryam and koenimbine exhibited significant inhibitory activity against castor oil-induced diarrhoea and PGE2-induced enter pooling in rats. The compounds also produced a significant reduction in gastro-intestinal motility in the charcoal meal test in Wister rats (16).

Antiulcer Activity

Antiulcer activity of aqueous and ether extracts of M. koenigii was studied in reserpine induced gastric ulcer model in albino rats. Extracts were effective in gastric ulceration and suggested as protective as ranitidine (17). Crude aqueous extract of leaves showed anti-ulcer activity which was evaluated by using models of acute gastric lesions induced by ethanol induced, aspirin induced, cold restrain stress and pylorus ligation in rats(18) Animals were pretreated with doses of 200 mg/kg and 400 mg/kg of aqueous extract which showed efficient reduction in lesion index, total affected area and percentage of lesion in comparison with control group in the ethanol induced, aspirin induced, cold restrain stress induced ulcer and pylorus ligation models. These observations provide a confirmation about aqueous extract of leaves of M. koenigii can act as good anti-ulcer drug (19) .

Vasodilating Activity

Crude aqueous leaf extract of M. koenigii was prepared which showed a dose dependent negative chronotropic effect on cardiovascular system of frog heart preparations which might be due to its direct actions on the heart and blood vessels. Potassium ion concentration was also found to be very negligible by flame photometry, indicating no involvement of potassium ions.(20) The aqueous leaf extract possesses vasodilatory effect which is independent of muscarinic, histaminergic and ß-adrenergic receptor as it increased the number of drops/minute in frog hind limb perfusion experiment and also does not possess α-adrenergic receptor antagonistic activity. The aqueous leaf extract showed significant effect at concentration of 1 mg/ml (21). Crude ethanolic extract of fresh leaves of M. koenigii showed dose dependent positive ionotropic effect on an isolated frog heart. The response to M. koenigii 62.5 - 1000 µg was not affected in either way by theophylline, imidazole, propanolol and sildenafil. The changes in potassium and sodium concentration did not alter.

The result suggested that M. koenigii induced positive ionotropic effect possibly by increasing availability of calcium from extra cellular sites (22)

Hypocholesterolemic Activity

Hypocholesterolemic activity was checked in aged mice, which was done by using crude ethanol extract of plant leaves of M. Koenigii. The experiment was confirmed by observing a decrease in cholesterol level in dose dependent manner in aged mice. The dose of 500 mg/kg was found more efficient than the 300 mg/kg and was comparable with the standard cholesterol reducing agent, Simvastatin. Carbazole alkaloids a major phytochemical constituent of plant found to have lipid lowering etc (23) .

Analgesic and Antinociceptive Activity

The methanolic extract of leaves showed analgesic effect in hot plate model and formalin induced paw licking response in mice. The activity might be linked to the processes involved in the prevention of sensitization of nociceptors, down regulation of the sensitized nociceptors or blockade of the nociceptors at peripheral and central levels. Methanol extracts were taken at different concentrations, viz. 100mg/ml, 200mg/ml and 400 mg/ml. Among these 400 mg/ml showed prolific results (24) .

Antidiabetic Activity

Curry leaves are rich in many minerals and trace minerals such as Iron, zinc and copper. Minerals found in curry leaf extract are important for maintaining normoglycemia, or the normal glucose content of the blood (25). This is done by the activation of pancreatic beta cells, which are responsible for the creation of insulin. While the nutrients in curry account for only about 1 to 2 percent of the required daily intake for these elements, they are bioavailable, or readily usable by the body. Therefore, the researchers suggested that curry leaves may be useful for the management ofdiabetes.

Effect on Dental Caries

Feeding of murraya leaf extract in golden hamsters showed lower caries scores compared to control group128. Murraya extract or isomahanine, murrayanol and mahanine incorporated in foods (such as candies, biscuits, cakes, chewing gums, juices) showed 86.2% inhibition of methyl suphydral formation by cultured Fusobacterium nucleatum 126. M. koenigii leaf extract containing mahanin, isomahanin or murrayanol as active ingredient formulated in

toothpaste, was found to be useful as oral disinfectant to protect against dental caries and periodontal disorders. They are also effective against Streptococcus mutans and Porphyromonas gingivalis.

Anticancer Activity

Koenoline isolated form root bark exhibited cytotoxic activity against the KB cell culture test system98. 9- formyl-3 methyl carbazole displayed weak cytotoxic activity against both mouse melanoma B 16 and adriamycin resistant P 388 mouse leukemia cell lines51. The effects of extracts of M. koenigii in in-vitro (short term incubation method and in-vivo (Dalton's ascitic lymphoma (DAL) anticancer models have been evaluated in male Swiss albino mice. DAL cells were injected intraperitoneally (106 cells) to the mice125. The anticarcinogenic potential of curry leaf using benzo (a) pyrene induced fore stomach and 7, 12 dimethyl benz (a) anthracene (DMBA) induced skin papillomas was studied. Chemo protective responses were measured as decrease in tumor burden (papillomas/mouse) and % of tumor bearing animals in both the models. Increase in level of acid soluble sulphydral compounds, glutathione S- transferase and DTdiaphorases were also measured. Antioxidant parameters (reduced glutathione, Super Oxide dismutase, catalases, glutathione peroxidase and glutathione reductase) were also elevated131. The in-vitro anti-tumour promoting activity and antioxidant properties of Girinimbine isolated from the stem bark of Murraya koenigii was studied by Yih et.al. The in vitro anti-tumour promoting activity of girinimbine was determined by measuring the percentage inhibition of induced early antigen (EA) of EBV on the surface of Raji cells132 M. koenigii has been found to induce apoptosis in human myeloid cancer cell (HL-60). Results shows that mahanine down-regulates cell survival factors by activation of caspase-3 through mitochondrion-dependent pathway, and disrupts cell cycle progression133 . Another study reported that mahanine, purified from the leaves of M. koenigii , has a dose- and time-dependent antiproliferative activity in acute lymphoid (MOLT3) and chronic myeloid (K562) leukemic cell lines and in the primary cells of leukemic and myeloid patients, with minimal effect on normal immune cells including CD34 (+) cells134.

Conclusion

Murraya koenjii have an significant anti-stress level.Extract from various parts of curry leaf have numerous medical applications, modern drugs are developed after extensive investigation of its bioactivity, and its action, pharmacotherapuetics and toxicity.crudeorganic

extracts of leaves of Murraya Koenigii have some pharmacological activities and found to possess anti-diabetic, cholesterol reducing property, anti-diarrhea activity, cytotoxic activity antioxidant property, antiulcer activity antimicrobial, antibacterial potential and many more usefulmedicinal properties. Murraya koemjii is a rich source of biologically active carbazole alkaloids which would attract the attention of chemist and pharmacologist and plays a significant role in future research in medical science. Keeping in view the tremendous pharmacological activities and wealth of literature available, M. koenigii may be utilized to alleviate the symptoms of variety of diseases as evident form the pre-clinical data. The available literature and wide spread availability of M. koenigii in India thus makes it an attractive candidate for further pre-clinical and clinical research.

Corresponding Author

Saravana Pandian,

Senior Lecturer, Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id- sporthoresearch@gmail.com

Telephone Number: +91 9003101627

Referance

1. Tembhurne SV, Sakarkar DM. Hypoglycemic effects of fruit juice of Murraya koenigii (L) in alloxan induced diabetic mice. Int J Pharm Tech Res. 2009 Oct;1(4):1589-93.

2. Prajapati ND. Handbook of medicinal plants.Agrobios; 2003.

3.Abujam SS, Shah RK. Study on the ethnomedicinal system of local people of Dibrugarh, Assam. International Journal of Pharmaceutical Innovation. 2012;2:17-28.

4. Adams RP. Identification of essential oil components by gas chromatography/mass spectrometry.5 online ed. Texensis Publishing; 2017.

5.Arulselvan P, Subramanian SP. Beneficial effects of Murraya koenigii leaves on antioxidant defense system and ultra structural changes of pancreatic β-cells in experimental diabetes in rats. Chemico-Biological Interactions. 2007 Jan 30;165(2):155-64.

6. Kumar MM, Vilas SR, Goojar M, Prajapati N, Pathak K. Anti-fungal potential of leave extracts of Murraya koenigii. International Journal of Research in Ayurveda and Pharmacy (IJRAP). 2010;1(2):549-52.

7. Tachibana Y, Kikuzaki H, Lajis NH, Nakatani N. Antioxidative activity of carbazoles from Murraya koenigii leaves. Journal of agricultural and food chemistry. 2001 Nov 19;49(11):5589-94.

8. Fiebig M, Pezzuto JM, Soejarto DD, Kinghorn AD. Koenoline, a further cytotoxic carbazole alkaloid from Murraya koenigii.Phytochemistry. 1985 Nov 26;24(12):3041-3.

9. Ito C, Itoigawa M, Nakao K, Murata T, Tsuboi M, Kaneda N, Furukawa H. Induction of apoptosis by carbazole alkaloids isolated from Murraya koenigii. Phytomedicine. 2006 May 9;13(5):359-65.

10. Parmar S, Gangwal A, Sheth N. Mast cell membrane stabilization and anti-histaminic actions possible mechanism of action of anti-inflammatory action of Murraya koenigii. J Curr Pharm Res. 2010;2(1):21-5.

11. Mandal S, Nayak A, Kar M, Banerjee SK, Das A, Upadhyay SN, Singh RK, Banerji A, Banerji J. Antidiarrhoeal activity of carbazole alkaloids from Murraya koenigiiSpreng (Rutaceae) seeds. Fitoterapia. 2010 Jan 1;81(1):72-4.

12. Furukawa H, Ito C, Yogo M, Wu TS. Structures of murrayastine, murrayaline, and pyrayafoline; three new carbazole alkaloids from Murraya euchrestifolia.Chemical and pharmaceutical bulletin. 1986 Jun 25;34(6):2672-5

13. Parmar C, Kaushal MK. Wild fruits of the Sub-Himalayan region. Wild fruits of the Sub-Himalayan region.. 1982.

14.Kadam SH, Dombe S, Naikwadi P, Patil M. Cardiovascular Effects of Aqueous Extract of Murraya koenigii on Isolated Perfused Frog Heart Preparation. Journal of Pharmacy Research. 2011;4(2):462-3.

15. Shah KJ, Juvekar AR. Positive inotropic effect of Murraya koenigii (Linn.) Spreng extract on an isolated perfused frog heart.

16. Dineshkumar B, Mitra A, Mahadevappa M. Antidiabetic and hypolipidemic effects of mahanimbine (carbazole alkaloid) from Murraya koenigii (rutaceae) leaves. International Journal of Phytomedicine. 2010 Jan 1;2(1).

17. Tembhurne SV, Sakarkar DM. Beneficial Effects of Ethanolic Extract of Murraya Koenigii Linn. Leaves in Cognitive Deficit Aged Mice Involving Possible Anticholinesterase and Cholesterol Lowering Mechanism.International Journal of PharmTech Research. 2010 Jan;2(1):181-8.

18.Chowdhury JU, Bhuiyan MN, Yusuf M. Chemical composition of the leaf essential oils of Murraya koenigii (L.) Spreng and Murraya paniculata (L.) Jack. Bangladesh Journal of Pharmacology. 2008 May 18;3(2):59-63.

19. Ajay S, Rahul S, Sumit G, Paras M, Mishra A, Gaurav A. Comprehensive review: Murraya koenigii Linn. Asian Journal of Pharmacy and Life Science ISSN. 2011;2231:4423

20. Shivkanya J, Shilpa P, Sangita K, Neeraj F. Pharmacognostical studies and antibacterial activity of the leaves of Murraya koenigii. Phcog J. 2009;1(3):211.

21. Syam S, Abdul AB, Sukari MA, Mohan S, Abdelwahab SI, Wah TS. The growth suppressing effects of girinimbine on HepG2 involve induction of apoptosis and cell cycle arrest. Molecules. 2011 Aug 23;16(8):7155-70.

22. Shirwaikar A, Ram HN, Mohapatra P. Antioxidant and antiulcer activity of aqueous extract of a polyherbal formulation.

23. Mandal S, Nayak A, Kar M, Banerjee SK, Das A, Upadhyay SN, Singh RK, Banerji A, Banerji J. Antidiarrhoeal activity of carbazole alkaloids from Murraya koenigiiSpreng (Rutaceae) seeds. Fitoterapia. 2010 Jan 1;81(1):72-4.

24. Molly J, Edison S, Vijajaraghavan R. Effect of Murraya Koenigii (Curry Leaves) Powder on the Liver and Renal Functions in Women with Hyperlipidemia.

25. Molly J, Edison S, Vijajaraghavan R, Ajith TA. Curry Leaf Powder and Cucumber Slices AttenuateHyperlipidemiainPostMenopausal Women: A Randomized Control Single Blinded Study. International Journal of Pharmaceutical Sciences and Research. 2017 Apr 1;8(4):1858.

Biodentine as a Pulpotomy Agent – A Review

Akshay Satwik T[1], Madhusudan Vasantharajan[2], Sindhu Ramesh[3]
Graduate Student[1], Senior Lecturer[2], Department of Pedodontics, Professor and Head[3],Department of Conservative Dentistry and Endodontics,
Saveetha Dental College , Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

The purpose of this text was to review the clinical applications of biodentine as a pulpotomy agent within the medical specialty practice.Biodentine may be a calcium-silicate primarily based material that has drawn attention in recent years and has been advocated to be employed in varied clinical applications, like root perforations, apexification, resorptions, retrograde fillings, pulp capping procedures, and dentin replacement. There has been sizeable analysis performed on this material since it's launching; but, there's scarce range of review articles that collates info and knowledge obtained from these studies. the aim of this text was to review the clinical applications and blessings of biodentine within the medical specialty practice. Electronic search of English scientific papers from 1992 to 2015 was accomplished exploitation saloon master's degree program. the subsequent search terms used were clinical applications, biodentine, medical specialty dental medicine, children, advantages, dentin substitute, pulp medical care, root filling, and tooth repair. because of its major blessings and distinctive options additionally as its ability to beat the disadvantages of alternative materials, biodentine has nice potential to revolutionize the various aspects of managing each primary and permanent in odontology additionally as operative dental medicine.

Key Words:*Clinical applications,Biodentine,medical specialty dental medicine,pulpotomy , infections*

Introduction

Pulpotomy

Pulpotomy may be a technique that has been accustomed treat primary teeth of that the requirement for additional and additional new materials isn't ending particularly within the field of dental medicine. varied materials are developed, tested and standardized to get most profit permanently clinical performance. One such new material is that the latest bioactive calcium-silicate primarily based material (biodentine), that was recently introduced by Septodont Company and will conciliate high mechanical properties with glorious biocompatibility, additionally as a bioactive behavior (1).

Biodentine may be a calcium-silicate primarily based material that has drawn attention in recent years and has been advocated to be employed in varied clinical applications, like resorptions , retrograde fillings ,root perforations, apexification, pulp capping procedures, and dentine replacement. Biodentine may be a new bioactive cement, kind of like the wide used MTA (1). Biodentine has many blessings that embody smart protection ability, adequate compressive strength, and short setting time, which offer a major clinical advantage over alternative comparable materials (2,3). it's biocompatible and conjointly shows bioactivity (4,5).

The commercialised tricalcium salt of biodentine is totally different from the same old dental atomic number 20 salt “Portland Cement” materials. The producing method of the active biosilicate technology eliminates the metal impurities (such as aluminates and alternative impurities) seen within the “Portland Cement” atomic number 20 silicates. Therefore, the mechanical properties area unit improved in biodentine by dominant the purity of the atomic number 20 salt through this Active Biosilicate Technology. Therefore, it's been developed and made with the aim of transferral along the high biocompatibility and bioactivity of atomic number 20 silicates, with increased properties, that build it additional distinctive than the other atomic number 20 silicate-based materials (2-5).

Biodentine is out there as powder in an exceedingly capsule and liquid in an exceedingly measuring instrument. There area unit 2 sorts of boxes out there within the market. Box is containing fifteen capsules & fifteen single-dose containers and another smaller box that contains solely five capsules & five single-dose containers (4)The powder is principally

composed of tricalcium salt (main core), dicalcium salt, carbonate, and iron chemical compound additionally as zirconia because the radiopacifier. The liquid contains water, salt (as setting accelerator) and a changed polycarboxylate (as superplasticising or water reducing agent) (5).

Biodentine was developed supported the foremost biocompatible chemistry out there for dental materials: atomic number 20 salt, which might set within the presence of water [5]. The atomic number 20 salt can act with water resulting in the setting and hardening of the cement. This association method can turn out hydrous atomic number 20 salt (CSH) gel. As a part of its chemical setting reaction, hydroxide is additionally shaped (5) in contact with phosphate ions, it creates precipitates that match hydroxyapatite (6).

This dissolution method happens at the surface of every grain of atomic number 20 salt. The non reactedtricalcium salt grains area unit enclosed by layers of CSH gel, that area unit comparatively impervious to water, thereby retardation down the results of any reactions. Gradually, the CSH gel fills within the areas between the tricalcium salt grains. Later on, the hardening method results from the formation of crystals that area unit deposited in an exceedingly concentrated resolution (6).

Biodentine attracted attention within the field of dental medicine because of its quick setting time, high biocompatibility, high compressive strength, glorious protection ability, and simple handling additionally as its versatile usage in each dental medicine repairand restorative procedures while not inflicting any staining of the treated teeth [4-6,10]. However, it's conjointly been well-tried that biodentine has a superb antimicrobial properties because of its terribly high hydrogen ion concentration (pH=12). additionally to it, it's rather more value effective as compared to similar materials (7).

Many in vivo and in vitro studies support its bioactivity additionally as its triple-crown performance in several clinical applications(8).On the opposite hand, all the out there clinical studies and case reports unconcealed glorious results for its use in human primary teeth(9).

Due to its improved material properties (short setting time, higher mechanical properties, and straightforward and technology use) additionally as its ability to beat the drawbacks of the

many alternative materials, biodentine could be a stimulating and promising various to the present materials for dentin-pulp advanced regeneration. Biodentine has the potential of creating major contributions within the field of dental medicine by maintaining the teeth in an exceedingly healthy state through various exciting clinical applications (9).Therefore, biodentine guarantees to be one in every of the foremost versatile materials of this century within the field of dental medicine.(10).

The purpose of this text was to review the clinical applications and blessings of biodentine within the medical specialty practice. the garland pulp has been affected or infected by dental caries or traumatized, whereas the radicular pulp remains healthy (10).

Clinical Applications OfBiodentine

Biodentine individuation not solely lies in its innovative bioactive and "pulp-protective" chemistry, however conjointly in its universal application on each crown and root. In the space of the dental crown, it is indicated for pulp capping, treatment of deep unhealthy lesions exploitation the sandwich technique,pulpotomy and conjointly as temporary enamel restoration or permanent dentine replacement.[2] Its use in root includes managing perforations of forking, external and internal reabsorption, apexification and retrograde root canal obturation. In addition to that, it may be used conjointly as bone substitute material for implant stabilization. On the alternative hand, biodentine isn't suggested in massive or esthetic build-ups.(11-15).

BiodentineAs A Pulpotomy Agent

Pulpotomy is another wide used important pulp medical care methodology within which biodentine is advocated to be used .This treatment methodology is that the most often accepted clinical procedure in medical specialty dental medicine once the garland pulp tissue is inflamed and a right away pulp capping isn't an appropriate possibility (15).

In comparison to formocresol in primary teeth pulpotomy, biodentine may be a regenerative material that maintains pulp vitality whereas formocresol may be a devitalizing agent. However, biodentine needed less time for the pulpotomy procedure .whereasformocresol acts solely as dressing material, that desires a restorative material to seal the pulp chamber, biodentine acts at the same time as each dressing and filling material .Thus,

biodentineeliminates the requirement for a filling material within the pulp chamber of pulpotomized teeth. whereasformocresol needs 3–5 minutes application before the cotton pellet is removed, with biodentine the pulp chamber is crammed right away . Moreover, throughout the removal of formocresol-soaked cotton pellet, there's a clear stage of the cotton fibers adhering to clot, leading to reoccurrence of hemorrhage. This doesn't occur with biodentine because it is applied directly while not cotton pellet(16).

In 2012, Shayegan investigated the inflammatory cell response and arduous tissue formation when biodentine pulpotomy in primary pig teeth. when ninety days, they found that the pulp tissue was traditional with none signs of inflammation and nine out of ten teeth showed thick calcification beneath the pulpotomy website. They all over that biodentine has bioactive properties, encourages arduous tissue regeneration, and provoke no signs of moderate or severe pulp inflammation response .

In support to the aforesaid favorable biological results, Marijana , all over that the therapeutic effects of biodentine when important pulp medical care in Vietnamese pigs area unit favorable. Biodentine has the potential of creating major contributions to maintaining pulp vitality in patients judiciously chosen for pulpotomy treatment.Therefore, this distinctive material could be a stimulating various to the present materials for dentin-pulp advanced regeneration.

A survey of the out there literature shows that there area unit nonetheless few revealed case reports and clinical trials with several non-published in progress clinical trials that embody the usage of biodentine in pulpotomy.All these studies showed biodentine as a good and promising various for the present pulpotomy medicaments.

In multiple case reports, Lavaud , showed a triple-crown results of biodentine with none clinical or imaging symptoms once it's used for primary teeth pulpotomy (9 months of follow up), indirect capping on a hypomineralized molar (12 months of follow up), and apexogenesis(14 months of follow up). In another revealed case report, Villat et al. performed a partial pulpotomy in AN immature second right tooth of a 12-year-old patient .After vi months, the patient didn't report any pain or complains on the observation amount. moreover, the authors detected homogenous dentin bridge formation additionally as continuation of root

development .Accordingly, they commented that quick favorable pulpal response render this material an appropriate alternative compared to alternative materials.

Recently at the twelfth Congress of European Academy of medical specialty dental medicine (EAPD) in Polska, Rubanenko, given their preliminary results of comparison biodentine versus formocresol as dressing agents in pulpotomized primary molars.They incontestable a hit rate of 100 percent for biodentine whereas that of formocresol was ninety four .Additionally, Cuadros confirmed these attention-grabbing preliminary results of biodentine in humans and stressed that biodentine appears to be a promising various to be used in pulpotomies of primary molars with 100 percent clinical and photography success when vi months of follow up .On the opposite hand, Rajasekharan, given the results of their randomised management run and showed clinical additionally as photography success in ninety four, 73% of biodentine treated teeth . They all over, “there was no vital distinction between the new product biodentine as compared to the well-known merchandise (mineral oxide combination (MTA) or Tempophore)” .In evaluating this preference dental medicine material in kids amongst Flemish medical specialty dentists, Vandenbulcke, found that biodentine was the foremost most well-liked pulpotomy material in each primary and immature permanent teeth .

Nowicka , studied the response of Biodentine direct pulp-capping in twenty eight caries-free inframaxillary and jaw permanent intact human molars scheduled for extraction for dental medicine reasons when mechanical exposure. when vi weeks, the teeth was extracted, stained with hematoxylin-eosin. They found majority of specimens showing an entire dentinal bridge formation ANd an absence of inflammatory pulp response. Layers of well-arranged odontoblast-like cells and odontoblast were found to create hollow dentin beneath the osteodentin. They conjointly found no statistically vital variations between the Biodentine and MTA experimental groups(15-16).

Han and Okiji compared Biodentine and white ProRoot MTA in terms of Ca and Si uptake by adjacent passage dentine and ascertained whereas each materials shaped tag-like structures, dentine component uptake was additional outstanding for Biodentine than MTA.The same authors in another study showed higher atomic number 20 unharness for Biodentine as compared with MTA. The tag-like structures shaped were composed of Ca and P-rich and Si-

poor materials.(17). Laurent et al. evaluated its genotoxicity, toxicity, and effects on the target cells' specific functions and located that it didn't have an effect on the pulp embryonic cell specific functions like mineralization, additionally as expression of albuminoid I, dentin sialoprotein, and Nestin.

PérardM , assessed the biological effects of Biodentine is employed in pulp-capping treatment, on pseudo-odontoblastic and pulp cells. They conjointly evaluated the results of Biodentine and MTA on organic phenomenon in civilized spheroids, and located Col1a1 expression levels (responsible for matrix secreton) were slightly lower in cells civilized within the presence of MTA than in those civilized within the presence of Biodentine and also the management cells. They all over that each Biodentine additionally as MTA area unit each appropriate for pulp-capping (18).

VillatC , performed partial pulpotomy exploitation Biodentine in AN immature second right inframaxillary tooth and incontestable a quick tissue response each at the pulpal and root dentin level with formation of a radiopaque bridge at intervals 3-6 months. They advised the utilization of tricalcium salt cement ought to be used as a conservative intervention within the treatment of symptomatic immature teeth (19).

Biodentine Used As Pulp Capping Material

Biodentine was conjointly used as direct pulp capping material. BiodentineTM showed:

• a superb tolerance.

• the flexibility to save lots of pulp vitality even in tough cases: the vitality take a look at was positive at every recall.

Moreover, Biodentine is employed in direct pulp capping indications with an honest success rate .it's necessary to underline that BiodentineTM was employed in contact with pulp tissue in an exceedingly patient older than twenty one and maintained the pulp alive.

BiodentineAs A Dental Repair Material

The dental medicine indications of Biodentine area unit kind of like the same old atomic number 20 salt primarily based materials, just like the Portland cements (i.e. ProRoot MTA). this sort of product is already well documented.

Several physical, chemical and biological properties area unit comparable as summarised within the presymptomatic section. However, Biodentine has some options that area unit superior to MTA.

- Biodentine consistency is best suited to the clinical use than MTA's.
- Biodentine presentation ensures a higher safety and handling than MTA.
- Biodentine doesn't need a 2 step obturation as within the case of MTA. because the setting is quicker, there's a lower risk of microorganism contamination than with MTA.

Adding to its ability to be used as dentine substitute, Biodentine may safely be used for every indication wherever dentine is broken . Therefore, its a bonus for the practician and also the patient (20-25).

The tolerance and effectuality of BiodentineTM in vi dental medicine procedures, when three months and when a pair of years follow-up is in progress:

- Direct pulp capping following unhealthy pulp exposure
- Direct pulp capping following dental trauma/injury to healthy pulp (partial pulpotomy)
- Repair of perforated root canals and/or pulp chamber floor
- Retrograde dental medicine surgery.
- Pulpotomy in primary molars
- Apexification .

Conclusion

The clinical applications of biodentine material are mentioned. Biodentine is a superb material with countless qualities needed of a perfect material. The necessary applications of biodentine in medical specialty dental medicine embody dentin substitute, pulp capping, pulpotomy, apexification, and repair material of perforation and reabsorption additionally as root finish filling material. It is another to formocresol in pulpotomy thanks to the tissue irritating, cytotoxic and agent effects of formocresol that area unit resolved with biodentine. However, it is another to hydroxide or MTA in pulp capping, pulpotomy, and apexification as a result of biodentine is extremely triple-crown within the formation of a dentin bridge that's

quicker and thicker with lesser defects. whereas it's stronger automatically, less soluble and produces tighter seals than hydroxide biodentine conjointly avoids the drawbacks of MTA, i.e. extended setting time, tough handling characteristics, high cost, and potential of discoloration.Accordingly, biodentine could be a stimulating various to the present materials for dentin-pulp advanced regeneration (26).

Due to its major blessings and distinctive options additionally as its ability to beat the disadvantages of alternative materials, biodentine has nice potential to revolutionize the various aspects of managing each primary and permanent in odontology additionally as operative dental medicine. On the opposite hand, any studies area unit required to increase the long run scope of this material relating to the clinical applications."(27).

Corresponding Author

Sindhu Ramesh,

Professor and Head,Department of Conservative Dentistry and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162,Poonamallee High Road, Chennai – 600077, Tamil Nadu, India.

Email – drsinramesh@gmail.com

Telephone Number : 9840136543

References

1. Nowicka A, Lipski M, Parafiniuk M, Sporniak-Tutak K, Lichota D, Kosierkiewicz A. Response of human dental pulp capped with biodentine and mineral trioxide aggregate. J Endod 2013;39:743-7.
2. Han L, Okiji T. Uptake of calcium and silicon released from calcium silicate-based endodontic materials into root canal dentine. Int Endod J 2011;44:1081-7.
3. Koubi G, Colon P, Franquin JC, Hartmann A, Richard G, Faure MO. Clinical evaluation of the performance and safety of a new dentine substitute, Biodentine, in the restoration of posterior teeth - a prospective study. Clin Oral Investig 2013;17:243-9.
4. Laurent P, Camps J, De Méo M, Déjou J, About I. Induction of specific cell responses to a Ca(3) SiO(5)-based posterior restorative material. Dent Mater 2008;24:1486-94.

5. Laurent P, Camps J, About I. Biodentine induces TGF-b1 release from human pulp cells and early dental pulp mineralization. Int Endod J 2012;45:439-48.
6. Nowicka A, Lipski M, Parafiniuk M, Sporniak-Tutak K, Lichota D, Kosierkiewicz A. Response of human dental pulp capped with biodentine and mineral trioxide aggregate. J Endod 2013;39:743-7.
7. Han L, Okiji T. Bioactivity evaluation of three calcium silicate-based endodontic materials. Int Endod J 2013;46:808-14.
8. Pérard M, Le Clerc J, Watrin T, Meary F, Pérez F, Tricot-Doleux S. Spheroid model study comparing the biocompatibility of Biodentine and MTA. J Mater Sci Mater Med 2013;24:1527-34.
9. Villat C, Grosgogeat B, Seux D, Farge P. Conservative approach of a symptomatic carious immature permanent tooth using a tricalcium silicate cement (Biodentine): A case report. Restor Dent Endod 2013;38:258-62.
10. Zanini M, Sautier JM, Berdal A, Simon S,Biodentinee induces immortalized murine pulp cell differentiation into odontoblast-like cells and stimulates biomineralization. J Endod 2012;38: 1220-1226.
11. Malkondu O, kazandag M, Kazazoglu E,A Review on Biodentine, a Contemporary Dentine Replacement and Repair Material. Biomed Res Int 2014;12: 1-10.
12. GrechL, Mallia B, CamilleriJ,Characterization of set Intermediate Restorative Material, Biodentine, Bioaggregate and a prototype calcium silicate cement for use as root-end filling materials. IntEndod J,2013; 46: 632-641.
13. Bachoo IK, Seymour D, Brunton P, A biocompatible and bioactive replacement for dentine: is this a reality? The properties and uses of a novel calcium-based cement. Br Dent J 2013;214: 1-7.
14. Priyalakshmi S, Ranjan M,Review on Biodentine-A Bioactive Dentin Substitute. J Dent Med Sci,2014; 13: 13-17.
15. Camilleri J, Sorrentino F, DamidotD,Investigation of the hydration and bioactivity of radiopacifiedtricalcium silicate cement, Biodentine and MTA Angelus. Dent Mater,2013; 29:580-593.
16. GrechL, Mallia B, CamilleriJ,Investigation of the physical properties of tricalcium silicate cement-based root-end filling materials. Dent Mater 2013;29: 20-28.

17. Camilleri J, Grech L, Galea K. Keir D, Fenech M, Formosa L, Damidot D, Mallia B Porosity and root dentine to material interface assessment of calcium silicate-based root-end filling materials. Clin Oral Investig 2014;18: 1437-46.
18. Shamkhalov GS, Ivanova EV, Dmitrieva NA, AkhmedovaZR,Comparative study of antimicrobial activity of "Biodentine" and “Rootdent” cements and “Futurabond HP” adhesive. Stomatologiia (Mosk) 2013;92: 37-39.
19. Koubi S, Elmerini H, Koubi G, Tassery H, Camps J,Quantitative evaluation by glucose diffusion of microleakage in aged calcium silicate-based open sandwich restorations. Inter J Dent 2012;1:1-6.
20. Kokate SR, PawarAM,An in vitro comparative stereomicroscopic evaluation of marginal seal between MTA, glass inomer cement &biodentine as root end filling materials using 1% methylene blue as tracer. Endodontology,2012; 24: 36-42.
21. Gjorgievska E, Nicholson J, Apostolska S ,Interfacial properties of three different bioactive dentine substitutes. MicroscMicroanal,2013;19: 1450-1457.
22. Tran X, Gorin C, Willig C, Baroukh B, PellatB.,Effect of a calcium-silicate-based restorative cement on pulp repair. J Dent Res,2012;91: 1166-1171.
23. Marijana P, Prokic B, Prokic BB, Jokanovic V, Danilovic V. Histological evaluation of direct pulp capping with novel nanostructural materials based on active silicate cements and Biodentine on pulp tissue. Acta Veterineria,2013; 63: 347-360.
24. Shayegan A, Jurysta C,Atash R, Petein M, Abbeele Av, Biodentine used as a pulp-capping agent in primary pig teeth. Pediatr Dent 2012;34: 202-208.
25. Aggarwal V, Singla M, Miglani S, Kohli S,Comparative evaluation of push-out bond strength of ProRoot MTA, Biodentine, and MTA Plus in furcation perforation repair. J Conserv Dent2013;16: 462-465.
26. Govindaraju L, Neelakantan P, Gutmann JL. Effect of root canal irrigating solutions on the compressive strength of tricalcium silicate cements. Clinical oral investigations. 2017;21(2):567-71.
27. Karthikeson PS, Jayalakshmi S. Pulp Capping Agents-A Review. Journal of Pharmaceutical Sciences and Research. 2016;8(6):525.

Biohazards Associated with the Materials used inDentistry

B.Valli[1],Anand Selvaraj[2],Jayanth Kumar V[3]

Graduate Student[1],Professor[2], Department of Prosthodontics,Reader[3], Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences
Saveetha University, Chennai, India

Abstract

The materials generally used for restoration like amalgam, composite etc. are considered as bio hazardous materials. These materials are manufactured with the aim to be insoluble and stable. The acrylic components of composite material is also toxic enough to cause systemic problems. Some of the dental restorative materials can cause harm to the patients and dental personnel. It can cause allergic contact dermatitis ,conjunctival symptoms and asthma. Once the environmental pH changes, the changes in the other parameters causes corrosion and abrasion can allow the bacteria to use the mercury in amalgam and toxicate it. The minute components mixed with these materials leach out of permanent restorative material and tend to cause toxic reactions.

Key Word:*materials, dentistry, biohazards, infection, prevention*

Introduction

A biological agent or infection that constitutes a hazard to human or the surrounding environment is called a biological hazard.There are atleast 193 important biological agents that show infectious allergenic , toxic or carcinogenic activities in the working population .Toxic substances enter our body through absorption , ingestion , inhalation and injection . Absorption can take place through the skin or eyes causing burns , sores , ulcer and other infections . Dentists and dental patients maybe exposed to variety of microorganisms via blood or oral or respiratory secretions[1] the microbes penetrate the body through a cut on the skin while performing either a medical procedure , or a dental procedure . The biological hazards are established by infectious agents of human origin and embrace viruses, bacterium

and fungi. Transmissible diseases presently of greatest concern to the dental skilled are HIV, HBV, HCV and tubercle bacillus. A dental practitioner will become infected either directly or indirectly, i.e by a cut or wound, needle stick injury, aerosols of secretion, gingival fluid and natural organic dirt particles.The following are the important entry points of infection: cuticle of hands, oral epithelial tissue, nasal epithelial tissue, epithelial tissue of upper airways, bronchial tubes, alveoli and mucous membrane epithelium. so as to overcome from the infection that has been spread, an intensive information concerning the infection, mode of transmission and safety measures is important. throughout several dental procedures, the utilization of a rubber dam can eliminate nearly all contamination arising from secretions such as saliva or blood[2].

Prevention from contamination and cross-infection will be done by effective sterilization of instruments with the use of autoclave before and after use. Legnani et al. created an assessment of the aerosol contamination that arouse from dental procedures. Air contamination was measured by means of the Surface Air System technique and also the "plate" technique (Air microbial Index). it had been proven that in operating hours the common air microorganism load raised over 3 times, and also the air load levels were 1.5 times (aerobic bacteria) and 2 times (anaerobes) bigger as compared to the initial load[3].Hazardous waste includes X-ray fixers and film, chemical disinfectants, sharps like needles, blood-soaked dressings, mercury, silver, lead, numerous solvents and different chemicals. paper, plastic, latex and glass, a lot of of that is contaminated with body fluids, comprise a huge amount of dental waste[4,5]. a big increase in the amount of dental solid waste generated has been noted because of the increased use of plastic barriers, gloves and masks, believed to compromise regarding 90th of the solid waste[6]. This is often because of enhanced safety measures enforced further because the replacement of reusable things with disposable ones[5]. Other hazardous waste made in minor quantities includes fluorescent lamps, unused chemicals, medication and extremely inflammable cleaners for developer systems which will contain chromium, vapour steriliser chemicals and formaldehydes[7] . Mercury from amalgam is taken into account the foremost problematic waste because of its noxious potential[8] . Mercury will enter the surroundings as solid waste by the disposal of extracted teeth also as through the effluent collection system by the disposal of amalgam particles throughout dental operations[7,9].The Occupational Safety and Health Administration (OSHA) has revealed controlling occupational Exposure to

BloodbornePathogens in dental medicine. These osha guidelines are made to safeguard the worker, not the patient. The osha blood- borne infectious agent standard could be a comprehensive rule that sets forth specific necessities. Osha guidelines are made to stop the transmission of blood-borne diseases to workers. It includes needs for an exposure management set up, exposure management precautions, laundry procedures, necessary serum hepatitis vaccinations, housekeeping standards, and waste disposal rules. The Aim of this article is to review the biohazards of the materials used in dentistry and the proper methodology to dispose the waste.

Classification of Biohazardous Materials used in Dentistry[10]

Hazardous materials from dental offices may include:

1. Photo processing wastes - X-ray film , lead foils and shields , X-ray fixer , developer and cleaner .
2. Chemical sterilants wastes - also known as chemiclave solutions (or) disinfectants.
3. Line cleaner wastes
4. Universal wastes : batteries , fluorescent lamps , mercury thermometers , etc.
5. Amalgam capsules : (i) scrap (non-contact) amalgam . (ii) amalgam capsules (empty , leaking or unusable) (iii) contact amalgam (eg . Extracted teeth)
(iv) amalgam pieces captured by vacuum pump filters and screens.
6. Biomedical waste : blood-soaked dressings, used gloves , extracted teeth

Photo Processing Waste

Silver: A heavy metal which enters the body through disposal of dental waste. The radiographic waste contains silver. Less amount of waste is generated from dental offices and the concentration if silver in X-ray film ranges from 8g-12g/L [11].There is high level of silver in the X-ray films that are not developed and to avoid these it should be sent to proper X-ray disposal company , this can be avoided by using digital X-ray[12]

Lead : the X-ray packets contain lead, leachable ions and lead aprons. High doses of lead intake lead to reproductive toxicity, neurotoxicity, carcinogenicity, hypertension, renal function, immunology, toxicokinetics, etc[13]. Use of X-ray machines in dental offices

predispose dentists to suffer from ionizing radiation[14,15]. Lead aprons, periodic maintenance of the X-ray machine and radiation level sensors deal with radiation dangers[15,16]. The way to reduce the toxicity released by these methods and save people from the after effects caused by this is ,primarily to avoid the usage of such things. e.g. use of digital X-rays compared to the conventional film, developer/fixer combination[17].

Approximately over half the dental clinics have X-ray units. All of these clinics used one processing solution for the X-ray unit that was disposed of down the drain. However, no information on the composition or concentration of the used solution was provided on the packaging bottle[18]. As most dental clinics in several developing countries manufacture little amounts of waste separately, the silver recovery unit designed to fulfil the standards is required instead of the additional usually designed units that handle larger and continuous quantities of waste. Dentists ought to then follow the correct equipment during surgical procedure and maintenance procedures to make sure the quantity of silver remaining within the effluent is at proper levels. Moreover, lead foil, that are used to protect the X- ray film, ought to be treated as hazardous waste and not disposed of with the regular waste as is the current follow[19]. Lead aprons, used on patients throughout X-ray filming, contain lead that is hazardous. Therefore, lead aprons ought to be reused and came to the manufacturer once the helpful life is over.

Chemical Sterilants Waste

Workplace Hazardous Materials Information System (WHMIS) trains the staff those who need to work in such places . Dental waste has been demonstrated to contribute significantly to pollution of the environment if poorly treated[20] and that it can lead to cross-infection risks[21] such as deadly HIV and Hepatitis among others[22]. Chromium , cadmium etc are called heavy metals which are considered to be hazardous. Chromium causes respiratory damage , kidney failure while cadmium causes lung cancer , kidney damage[23]. Rubber dams , gloves , mask , headcaps are mandatory equipments for any dental procedure but the use of these materials releases few toxic gases like Chlorofluorocarbons , dioxin etc which causes neurological cancers, respiratory problems[24].Dental waste do not generally contain food remains to any large extent , but more of plastics (Polyvinyl Chloride PVC, Polyethylene, (PE), polypropylene (PPE). Any untreated waste should be properly labelled and handled by a waste hauler[25]. Contaminated sharps must be placed in a puncture-

resistant rigid container and treated prior to disposal.One should not pour sterilants into a septic system as this may significantly disrupt the bacteria which normally breakdownwastes[26,27].

Line Cleaners

Line cleaners generally contain alcohols and/or other hazardous materials. Products with such ingredients that go unused are considered hazardous waste and should be disposed properly.

• Disposal - It is done by City-sponsored Very Small Quantity Generated Program.

Universal Wastes

Needles, scalpels, glass carpules, burs, acid etch tips, files, blades and other similar objects , Their waste management includes collection in a red or yellow puncture resistant container with a lid that cannot be removed[27]. Aerosols cause airborne infections because they can stay airborne and has the potential to cause respiratory passage diseases. The various instruments that can cause airborne diseases ultrasonic scaler , air polisher , air driven hand pieces etc[28-30]. Autoclaving must be done to prevent contamination .Legnani.et.al made an assessment of the aerosol contamination resulting from dental Treatments. It was proved that during working hours the average air bacterial load increased over three times, and the air load levels were 1.5 times (aerobic bacteria) and 2 times (anaerobes) greater as compared to the initial load[31]. The practician who is using a disposable sharp item should be liable for its immediate safe management or disposal it is use. This should be at the point of use (i.e. the operatory or treatment room) unless transferred in an acceptable container. Used disposable needle syringe combinations, empty or partly used cartridges of anaesthetic agent solution, burs, needles, surgical knife blades, orthodontic bands, endodontic files and different single-use sharp things should be discarded in an approved clearly labeled , puncture and leak-proof containers.

Dental assistants ought to be trained to examine that sharps resembling burs and orthodontic wires are removed by the operator before commencing the transformation procedure.Burs ought to be faraway from hand-pieces before removing the hand-piece from the dental unit, to cut back the danger of sharps injury.Disposable sharps, if not placed by the operator into a sharps bin placed at the chairside, may instead be placed when use during a specific puncture-

proof dish to minimizerisk.Sharps containers should be placed in a very safe position inside the clinic to avoid accidental tipping over and should be out of the reach of young children.

Amalgam Wastes

Two main kinds of dental restoration practices are used: dental composite (otherwise referred to as white fillings), a mixture of glass or ceramic particles spread during a photo-polymerizable synthetic organic resin matrix, and amalgam, consisting of roughly 1:1 mixture of metallic mercury and a pulverized alloy consisting of silver, tin, copper, zinc additionally to different metals . it's thus because of the high concentration of mercury that this latter waste is assessed as hazardous. However, the utilization of elemental mercury in amalgam rather than the safer encapsulated amalgam is usually most preferred because it is less costly, even though accidental spills will put the dental personnel in danger of mercury toxicity.Mercury is neurotoxic , nephrotoxic and bio accumulative element and one of the main source of it in dentistry is amalgam. It can get into the environment through waste water, scrap amalgam or vapors. Vaporous mercury waste management includes: (i) storing unused elemental mercury in a tightly sealed container, (ii) contacting a certified biomedical waste carrier (CWC) for recycling or disposal, (iii) using a “mercury spill kit” in case of a spill of mercury, (iv) reacting unused elemental mercury with silver alloy to form scrap amalgam, (v) not placing elemental mercury in the garbage, and (vi) not washing elemental mercury down the drain. Humans are continuously exposed to mercury via food, air, water[32].During the placement and removal of dental amalgam restorations, a variety of waste products is generated:(i)elemental mercury vapour–released from dental amalgam alloy,(ii)dental amalgam scrap–the amalgam particles that have not come into contact with the patient,(iii)amalgam waste–the particles that have come into contact with patient secretions,(iv)amalgam sludge–the fine particles present in dental office wastewater, commonly trapped in chair-side traps and vacuum filters[3,33].Although amalgam separators in the certification process are required to remove at least 95% of incoming mercury in a standardized laboratory test, their efficiency in practical use has not been properly investigated mercurypoisoning[34] can be characterized by tumours of the face, arms or legs and may be associated with progressive, tremulous illegible handwriting with slurred speech[35]. The amalgam waste ought to stay in a very properly labeled sealed container that's appropriate for storing contaminated amalgam waste and will be sent for the contents to

be recycled, chiefly mercury and silver. Used and empty amalgam capsules is also disposed of as solid waste since they're non-hazardous.

Blood-Soaked Dressings

All biomedical wastes should be color coded and marked with the workplace hazardous Material information system (WHMIS) biohazard image. medicine wastes will solely be transported by a corporation with correct certification. biomedical wastes are occupational health and safety concerns for those who come in close contact with them[36]. Non-dripping gauze and extracted teeth don't seem to be thought of biomedical; but teeth with amalgam restorations can not be placed within the incinerator for disposal.cotton and extracted teeth, within the trash with the regular waste. Swabs or dressings that are contaminated with blood or different body fluids are regulated by infectious waste rules . These ought to be placed in sealed, durable impermeable bags to stop escape of the contained things.Human anatomic waste includes all the body parts, biopsy undergone tissues and also extracted teeth. All thesemust be disposed in yellow bags or containers[37].

Standard Precautions to be Followed by a Dental Practitioner

There are several ways of precautions to be administered by the dentist . Amongst them the two basic precautions to be followed are the following -

1. Hand hygiene
2. Personal protective equipment

Hand Hygiene

Hand hygiene may be a general term applying to processes reaching to cut back the quantity of microorganismsonhands. This includes either the applying of a dry antimicrobial agent, e.g. alcohol-based hand rub (ABHR), to the surface of the hands, or the employment of soap/solution (plain or antimicrobial) and water, followed by patting dry with single-use towels.The HHA protocol is to use an ABHR for all clinical situations wherever hands are visibly clean. The traditional routine in practice ought to be for dental employees to use ABHR between patient appointments and through interruptions with intervals the one appointment. ABHR is applied onto dry hands and rubbed on for 15-20 seconds, after which period the hands are dry.

Hand hygiene is needed once removal of gloves. This should be done before the dentist writes or types up patient notes. Practitioners shouldn't shake a patient's hand once greeting them when they are in the reception while not having first completed hand hygiene.ABHR usually will be used as often as is needed. However,a compatible moisturiser ought to be applied up to four times per day. ABHR should solely be used on dry skin;wet hands dilutes the product therefore decreasing its effectiveness.

Personal Protective Equipment

There are a list of personal protective equipments to be used by the dentist-

1. Gloves
2. Mask
3. Eye protection
4. Protective clothing
5. Footwear

Gloves

Dental practitioners and clinical support employees should wear gloves whenever there's risk of exposure to blood, saliva or different body secretions or when hands can come in contact with secretion membranes. This implies gloves should be worn for all clinical procedures. Wearing gloves doesn't replace the necessity for hand hygiene because hands might still become contaminated because of producing defects in new gloves that weren't obvious to the user, or due to injury (such as tears and pinpricks) that occur to the gloves throughout use.Dental materials that are madeof components of methacrylate and its polymer are found to be important reason for contact dermatitis, aggravation of skin conditions, eyes or mucous membranes, allergic dermatitis, asthma and paresthesia in the fingers[38].Disposable gloves employed in patient care should not be washed before or once use nor ought to they be reused. A brand new pair of gloves should be used for every patient and altered as soon as they're cut, torn or perforated.

Masks

Dental procedures will generate huge quantities of aerosols of 3 microns or less in size and variety of diseases could also be transmitted via the airborne (inhalational) route. In the

dental surgery surroundings, the foremost common causes of airborne aerosols are the high-speed air rotor handpiece, the supersonic scaler and also the three-way syringe. The aerosols created could also be contaminated with microorganism and fungi from the oral cavity (from saliva and dental bio films),as well as viruses from the patient's blood[30].Therefore, dental practitioners and clinical support employees should wear appropriate fluid-resistant surgical masks that block particles of 3 microns or less in size. However, it's recommended that masks be worn during all the times while treating patients to stop contamination of the operating area with the operator's respiratory or nasal secretions/organisms.

Eye Protection- Dental practitioners and clinical support employees should wear protective eyewear to safeguard the mucous membranes of the eyes throughout procedures wherever there's the potential for penetrating trauma or exposure to aerosols, splattering or spraying with blood, saliva or body substances.Eyewear protects the eye from a broad variety of hazards as well as projectiles and may be worn for many clinical procedures[27].Protection from projectiles is especially necessary during scaling, while using rotary instruments (particularly when removing existing restorations), cutting wires and cleansing instruments and equipment.

Protective Clothing

Protective clothing (e.g. disposable gown), ought to be worn when treating patients while it's possible aerosols or splatter are likely to be generated or once contaminated with blood or saliva. the foremost appropriate style of protective clothing and equipment used varies according to the nature of the procedure and may be a matter of skilled judgement. Wherever there's a risk of enormous splashes with blood or body substances, imperviable protective clothing should be worn[33]. The protective gown worn within the clinical space should be removed before consumption of food, drinking, taking a break or leaving the clinical premises.

Footwear- Dentists and clinical support employees like the assistant ,receptionistand the other staffs should wear closed footwear which will help them protect from injury or contact with sharp items such as the accidentally dropped sharps or spilt chemicals[32].

Conclusion

Sufficient knowledge and adequate information regarding occupational hazards and its prevention will contribute in providing quality care to patients without any doubt. The global literature focuses strictly on control of infections and appropriate management of potentially infected materials, owing to the high profile of dentistry regarding infection transmission. The dentist should follow the proper precautions that will protect them and their patients from getting affected through the hazardous waste obtained from the clinic.

Corresponding Author

Jayanth Kumar V,

Reader, Department of Oral Medicine and Radiology,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai -600077, Tamil Nadu, India.

Telephone Number: +91 8056135807

Email Id: doctorjayanth@gmail.com

References

1. M, Wegman DH. Prevelance rates and odds ratios of shoulder – neck diseases in different occupational groups .Br J Ind Med 1987 ; 44:602-10
2. Jolanta S. Occupational Hazards of Dentistry. Ann Agric Environ Med. 1999; 6: 13-19.
3. Legnani P, Checchi L, Pelliccioni GA, D'Achille C: Atmospheric contamination during dental procedures. Quintessence Int 1994; 25: 435-39.
4. Svenson, B., Soderfeldt, B. &Grondahl, H.G. (1996) Attitudes of Swed- ish dentists to the choice of dental X-ray film and collimator for oral radiology. Dentomaxillofac. Rad., 25(3), 157–161.

5. Treasure, P. & Treasure, E.T. (1997) An investigation of the disposal of hazardous wastes from New Zealand dental practices. Community, Dent. Oral Epidemiol. 24, 328–331.
6. Farmer, G.M., Stankiewicz, N., Michael, B., Wojcik, A., Lim, Y., Ivkovic, D. &Rajakulendran, J. (1997) Audit of waste collected over one week from ten dental practices. A pilot study. Aust. Dent. J., 42(2), 114–117.
7. Arenholt-Bindslev, D. (1998) Environmental aspects of dental filling materials. Eur. J. Oral Sci., 106, 713–720.
8. Al-Khatib, I. A. &Darwish, R. (2004) Assessment of amalgam waste management in dental clinics in Ramallah and Al Bireh cities in Palestine. Int. J. Environ. Heal. R., 14(3), 179–83.
9. Chin, G., Chong, J., Kluczewska, A., Lau, A., Gorjy, S. and Tennant, M. 2000. The environmental effects of dental amalgam. Aust. Dent. J., 45(4), 246–249.
10. Ayatollahi J, Bahrololoomi R, Ayatollahi F. Vaccination of dentist and other oral health care providers. J Den Med. 2005;18:5–14.
11. J Am Dent Assoc, 2003; 134(8): 1095-6.
12. Hörsted-Bindslev P. Amalgam toxicity-environmental and occupational hazards. J Dent. 2004;32:359–65.
13. Clifton JC., 2nd Mercury exposure and public health. Pediatr Clin North Am. 2007;54:237–69.
14. Gidlow DA. Lead Toxicity. Occup Med (Lond) 2004;54:76–81.
15. Rubel DM, Watchorn RB. Allergic contact dermatitis in dentistry. Aust J Dermatol. 2000;41:63–9.
16. Shuhaiber S, Einarson A, Radde IC, Sarkar M, Koren G. A prospective controlled study of pregnant veterinary staff exposed to inhaled anesthetics and X-rays. Int J Occup Med Environ Health. 2002;15:363–73.
17. Szymanska J. Occupational hazards of dentistry. Ann Agric Environ Med. 1999;6:13–9.
18. Prüss, A. E., Giroult, E. &Rushbrook, P. eds. (1999) Safe Management of Wastes from Healthcare Activities. WHO handbook. Geneva,World Health Organization.
19. Minnesota Technical Assistance Program (MnTAP) 1995. Managing Waste Generated by Dental Clinics.

20. Adedigba, M.A., Afon, A.O., Abegunde, A.A., Nwhator, S.O., Bamise, C.T.. Assess- ment of dental waste management in a Nigerian tertiary hospital. Waste Manage- ment& Research 2010; 28: 769–777
21. Adedigba, M.A., Afon, A.O., Abegunde, A.A., Nwhator, S.O., Bamise, C.T.. Assess- ment of dental waste management in a Nigerian tertiary hospital. Waste Manage- ment& Research 2010; 28: 769–777
22. Coker, A, Sangodoyin, A. Sridhar, M., Booth, C., Olomolaiye, P. & Hammond, F. Medical waste management in Ibadan, Nigeria: obstacles and prospects. Waste Man- agement, 2009; 29, 804–811.
23. Rao, P.H., Report: Hospital Waste management – awareness and practices: a study of three states in India. Waste Management and Research, 2008; 26, 297–303.
24. Punchanuwat K., Drummond, B.K. & Treasure, E.T.. An investigation of the disposal of dental clinical waste in Bangkok. International Dentistry Journal, 1998; 48, 369–373.
25. Floret, N., Mauny, F., Challier, B., Arveux, P., Cahn, J.-Y. &Viel, J.F. Dioxin emis- sions from a solid waste incinerator and risk of non-Hodgkin lymphoma. Epidemiol- ogy, 2003; 14, 392–398.
26. Colorado Department of Public Health and Environment. Compliance Bulletin for Hazardous Waste, Photographic, X-ray and Dental Wastes. 7/97 CHW-012.
27. Pasupathi P, Sindhu S, Ponnusha BS, Ambika A. Biomedical waste management for health care industry. Int J Biol Med Res. 2011;2:472–86.
28. Blenkharn JI. Standards of clinical waste management in UK hospitals. J Hosp Infect. 2006;62:300–3.
29. Micik RE, Miller RL, Mazzarella MA, Ryge G. Studies on dental aerobiology: bacterial aerosols generated during dental procedures. J Dent Res 1969; 48: 49-56.
30. Miller RL, Micik RE. Air pollution and its control in the dental office. Dent Clin North Am 1978; 22: 453-76.
31. HarrelSK , Molinari J. Aerosols and splatter in dentistry: A brief review of the literature and infection control implications. J Am Dent Assoc 2004; 135: 429-37.
32. Hiltz M. The environmental impact of dentistry. J Can Dent Assoc, 2007; 73(1): 59-62.

33. Condrin AK. The use of CDA best management practices and amalgam separators to improve the management of dental wastewater. J Calif Dent Assoc, 2004; 32(7): 583-92.
34. Hylander LD, Lindvall A, Gahnberg L. High mercury emissions from dental clinics despite amalgam separators. Sci Total Environ, 2006; 362(1-3): 74-84.
35. Leggat PA, Kedjarune U, Smith DR. Occupational health problems in modern dentistry: a review. Industrial Health. 2007; 45: 611-21.
36. Mutter J. Is dental amalgam safe for humans? The opinion of the scientific committee of the European Commission. Journal of Occupational Medicine and Toxicology. 2011 ;6: 1-17.
37. R Monalisa, S Anand. Incidence of Adverse effects of dental materials on dentists survey based on questionnaire. Research J. Pharm. and Tech 2016; 9(10):1599-1601.
38. N. Ashikariswana et al /J. Pharm. Sci. & Res. Vol. 8(9), 2016, 1070-1072.

Biological Properties of Turmeric

Priyadharshni S[1], Anitha[2], E. Roja[3]
Graduate Student[1], Reader[2], Department of Biochemistry,Tutor[3], Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Turmeric is traditionally used as a spice that comes from the root curcuma long, a member of the ginger family. The main ingredient in turmeric is curcumin. The characteristic yellow colour of turmeric is due to curcumin. Turmeric forms a part of most Indian curry powder. A wide range of biological activities like anticancer, antimicrobial, antiinflammatory, anticarcinogenic, antimutagenic, antifertility, antiprotozoal, antidiabetic and free radical scavenging activity of the plant suggests a logical basis for its traditional use in foodstuff. Turmeric powder is used to treat a wide variety of diseases. In Unani medicine, turmeric has been used for conditions such as liver obstruction and jaundice and has been applied externally for ulcers and inflammation. Safety evaluation indicates that both turmeric and curcumin are well tolerated at a very high dose without any toxic effects. Thus, both turmeric and curcumin have the potential for the development of modern medicine for the treatment of various diseases. Turmeric is highly estimated by the Indians and has religious importance because of its yellow colour resembling sunlight.

Keywords:*Biological activities, disease, curcumin, healing, powder*

Introduction

Turmeric is a medicinal plant extensively used in Ayurveda, unani and Sidda medicine as home remedy for various diseases.Turmeric is one of the nature's powerful healers It is also considered as auspicious and is a part of religious rituals[1]. Turmeric has been used for 4000 years to treat a variety of ailments. Turmeric is used as a food preservative, food additive and colouring agent[2]. Since its initial medical use in Ayurveda to bring "heat"to the body'shot/cold balance, many health conditions have been treated by turmeric.Its main bioactive compound, curcumin, is thought to be responsible for the many anti-inflammatory and antioxidant properties which benefit conditions as broad as healing wounds, cholesterol, and triglyceride reduction, reduction of sclerosing of bile ducts in the liver in cirrhosis and improving man digestive conditions such as colic[3]. Curcumin in turmeric has been shown to stimulate the production of bile by the gall bladder. Curcumin is also a powerful antioxidant;antioxidant scavenge damaging particles in the body known as free radicals, which damage cell membrane tamper with DNA and even cause cell death. The colour of turmeric is due to curcumin. As Indian culture worshipped solar system turmeric is specially protected as it contains sun coloured yellow dye. Turmeric has 46 synonyms but it is best known as Haridra in Sanskrit and Pita: yellow, Gauri:brillant, Rajini:night in Hindi. It belongs to group of aromatic spices and when added to various food preparations preserved their freshness and nutritive value. Curcumin is the principalcurcumoid found in turmeric[4]. Turmeric held a place of honour in Indian traditional Ayurveda medicine. In Ayurveda, it was prescribed for the treatment of many medical problems ranging from constipation to skin diseases. It was used as digestive aid and treatment for fever, inflammation, wounds, infections, dysentry, arthritis, injuries, trauma, jaundice, and other liver problems. In unani turmeric is considered to be safest herb of choice for blood disorders since it purifies, stimulates and builds blood. Turmeric is relatively broad spectrum anti-fungal. Turmeric exhibits antioxidant activity and protect from free radical damage. Curcuma also exhibits anti tumor activities and prevent cancer. It inhibits topoisomerase enzyme, which is required for cancer[5]. Turmeric and its constituents have the potential for the development of modern medicine of the treatment of various diseases[6].

Plant Morphology

The turmeric (curcuma longa) plant, a perennial herb belonging to the ginger family, is cultivated extensively in south and southeast tropical as Saveetha University[7].Turmeric was described as c.longa by linnaeus and its taxonomic position is as follows:

Class -Liliopsida
Subclass -Commelinids
Order - Zingiberales
Family - Zingiberaceae
Genus - Curcuma
Species- Curcuma longa

The wild turmeric is called C.aromatica and the domestic species is called C. Longa[8].

India is the largest producer of turmeric in the world (93.7% of the total world production) and is cultivated in 150,000hectares in India. Maximum area under turmeric is in AndhraPradesh, followed by Maharashtra, Tamil Nadu, Orissa, Karnatakaand Kerala. It is found throughout the south and south east Asia with a few species extending to China, Australia and the South Pacific[8]. The rhizome is a yellowish-brown with a dull orange interior that looks bright yellow when powdered. Rhizome measures 2.5-7.0 cm (in length), and 2.5 cm (in diameter) with small tuber branching off [9].

Antioxidant Property

The antioxidant activity of curcumin was reported as early as 1975. It acts as a scavenger of oxygen free radicals. It can protect haemoglobin from oxidation.In vitro, curcumin can significantly inhibit the generation of reactive oxygen species (ROS) like superoxide anions, nitrite radical generation by activated macrophages[10]. Curcumin prevents oxidative damage during indomethacin induced gastric lesion not only by blocking inactivation of gastric peroxidase, but also by direct scavenging of hydrogen peroxide and OH (unpublished observation). Since ROS have been implicated in the development of various pathological conditions[11],curcumin has the potential to control these diseases through its potent antioxidant activity. Curcumin exerts powerful inhibitory effect against hydrogen peroxide-induced damage in human keratinocytesand fibroblasts[12]. Contradictory to the above-mentioned antioxidant effect, curcumin has pro oxidantactivity. Kelly reported that curcumin not only failed to prevent single strand DNA breaks by hydrogen peroxide, but also caused

DNA damage. As this damage was prevented by antioxidant alpha-tocopherol, the pro oxidant role of curcumin has been proved(13). Reactive oxygen species (ROS) are associated with many biological phenomena such as inflammation, carcinogenesis and aging (Amin, 2012). Todd (1985), has reported the antioxidant effect of hexane and methanol extracts of rhizome at a concentration of 0.1%. They found that hexane extract of dried rhizome at a concentration of 0.06% was inactive when tested on lard however, the methanol extract was active (Lee et al., 1982). According to a study by Shalinin et al. (1987), hot water extract of a commercial sample of tuber was active vs. protection of DNA against per oxidative injury. Water extract of rhizome was active on rat brain vs. Fe2+/ ascorbate Fe2+/ TBH induced lipid per oxidation. The biological activity was highly dose dependent, IC50 (median inhibitory concentration) 100 mcg/ml. The extract was also active vs. lipid per oxidation induced by TBARS, IC50 50 mcg/ml which means that the activity depends on the doses and varies with dose variation (Selvam, 1995).

Anti-Inflammatory

Curcumin is effective against carrageenin-induced oedema in rats and mice(14). The natural analogues of curcumin, viz. FHM and BHM, are also potential inflammatory agents[15]. The volatile oil and also the petroleum ether, alcohol and water extracts of C.longa show inflammatory effects[16]. The anti rheumatic activity of curcumin has also been established in patients who showed significant improvement of symptoms afteradministration of curcumin[17]. That curcumin stimulates stress-induced expression of stress proteins and may act in a way similar oxylated phenols and an enol form of beta-diketone;the structure shows typical radical-trapping ability as a chain-breaking antioxidant[18]. A cohort study of 32 patients found that curcumin was as effective as corticosteroids for chronic anterior uveitis (as demonstrated by improved vision, decreased keratic precipitates, and a break of synchiaeassessedby slit lamp examination)[19].Another small cohort study cumin reduced or resolved inflammatory orbital pseudotumor (as evidenced by reduced ocular swelling, normal ocular movements, and absence of diplopia) [20].

There is a great number of papers in the literature relating the activity of compounds extracted from C. longa L. being potent inhibitors of inflammation. These substances can be classified as curcuminoids, analogues of diarylheptanoids. There are two models of inflammation to be studied: chronic models (cot- ton pellet and granuloma pouch), where the inflammation and granulomas develop during a period of time (several days), indicating the proliferative phase of inflammation; and acute models, where acute effects of anti-

inflammatory agents can be studied, testing their inhibitory action on the development of rat paw edema.

Mukophadhyay (1982) demonstrated the activity of curcumin and other semi-synthetic analogues (sodium curcuminate, diacetylcurcumin, triethylcurcumin and tetrahydrocurcumin) in carrageenin-induced rat paw edema and cotton pellet granuloma models of inflammation in rats. In these experiments the authors used ferulic acid and phenylbutazone (reference drug). Curcumin and its analogues showed similar action in carrageenin- induced paw edema in rats; however the sodium curcuminate was the most potent analogue and was more water-soluble than curcumin. Among the curcumin analogues, triethylcurcumin was the most potent anti-inflammatory in the chronic model of inflammation, when compared with the others and with the drug reference; and tetrahydrocurcumin showed no activity. In the acute inflammation condition, all the substances were more effective. The authors concluded that the activity of the compounds used in these experiments, would depend on the model of inflammation. Arora (1971) investigated the anti-inflammatory activity in different fractions of the petroleum ether extract of the rhizomes of turmeric (two constituents) in animals. They found that the extracts reduced the granuloma growth and no toxic effects were observed. Chandra and Gupta (1972) demonstrated the anti-inflammatory and anti-arthritic actions of volatile oil of C. longa L. Ghatak and Basu (1972) showed the action of sodium curcuminate as an anti-inflammatory agent, being better than curcumin and hydrocortisone acetate, in experimental inflammation induced by carrageenin and formalin in albino rats (ED50 = 144μg/kg), its more soluble in water than curcumin and no side effects were observed.

Pharmacological actions of curcumin as an anti- inflammatory agent have been examined by Srimal and Dhawan (1973). In this work, the authors re- ported that the compound was effective in acute as well as chronic models of inflammation. The potency of this drug is approximately equal to phenylbutazone in the carrageenin-induced edema test, but it is only half as active in the chronic experiments. It was observed that curcumin was less toxic than the reference drug (no mortality up to a dose of 2 g kg-1). Huang (1992) examined the inhibitory effects of curcumin on the proliferation of blood mononuclear cells and vascular smooth muscle cells. In blood mononuclear cells, curcumin was capable to impair the response of cells to mitogen, PHA and the response to alloantigen, MLR. The investigators suggested that curcumin could be use clinically in transplant atherosclerosis. The cinnamicacid derivatives were less active than curcumin. Ammon (1992) demonstrated curcumin as an inhibitor of leucotriene formation in rat peritoneal polymorph nuclear

neutrophils (PMNL), with an EC50 of 27 x 10-7 M, in contrast, the hydrocortisone did not show any effect.

Curcumin enhances wound healing Tissue repair and wound healing are complex processes. Curcumin has very effective wound healing activity, examined in rats and guinea pigs. In situ hybridisation and PCR analysis shows an increase in the mRNA transcripts of transforming growth factor beta 1 (TGFβ1) and fibronectin in Curcumin treated wounds. Transforming growth factor beta 1 enhance wound healing, therefore it is possible that Curcumin modulates TGF β1 activity.

Anti-Fertility

Petroleum ether and aqueous extracts of turmeric rhizomes show 100% anti-fertilityeffect in rats when fed orally[21]. Implantation is completely inhibited by these extracts. Curcumin inhibits 5a-reductase, which converts testosterone to 5adihydrotestosterone, thereby inhibiting the growth of flank organs in hamster[22]. Curcumin also inhibits humansperm motility and has the potential for the development of a novel intravaginalcontraceptive[23]. Turmeric regulates menses, decreases intensity and pain of periods, decreases amenorrhea and decreases uterine tumours. Turmeric is a mild and supportive uterine stimulant.

Anti-Cancer

Curcumin acts as a potent anti-carcinogeniccompound.Among various mechanisms, induction of apoptosis plays an important role in its anti-carcinogenic effect.It induces apoptosis and inhibits cell-cycle progression, both of which are instrumental in preventing cancerous cell growth in rat aortic smooth muscle cells[24]. The mechanism responsible for apoptosis involves inhibition of cell signalling pathway genes like DNA damage(25). Turmeric inhibits the topoimerase enzyme, which is required for the replication of cancer and parasite cells. It strongly inhibits DNA and RNA synthesis and increases mitochondrial membrane permeability ; a very significant property in the apoptosis of proliferating cells. It can also prevent proliferation by cell cycle arrest in the G2/M phase in a variety of malignant tumours [26]. G2/M arrest renders cells more susceptible to the cytotoxic effects of radiation, suggesting that curcumin may find significance as a radio sensitizer[27].The ability to inhibit COX-2 gene over-expression which is implicated in the carcinogenesis of many different tumours, has suggested a plausible role of curcumin to protect children against leukaemia. Curcumin was shown to induce apoptosis among

leukaemia B lymphoma cells and inhibits the multiplication of leukaemia cells in laboratory studies[28]. Curcumin also suppresses human breast carcinoma through various pathways.

Anti-Microbial

Turmeric extract and the essential oil of Curcuma longa inhibit the growth of a variety oil of bacteria , parasites, and the pathogenic fungi. The aqueous extract of turmeric rhizomes hasanti-microbial effects [29]. Both curcumin and the oil fraction suppress growth of several bacteria like streptococcus, staphylococcus, lactobacillus, etc[30].Ether and chloroform extracts and oil of C.longa have anti-fungaleffects. Crude ethanol extract also possess anti-fungalactivity. Turmeric oil is also active against Aspergillusflavus, A.parasiticus, Fusariummomiliforme. The ethanol extract of the rhizomes has anti-Entamoebahistolytica activity. Curcumin has anti-Leishmania activity in vitro[31].Several synthetic derivatives of curcumin have anti-L.amazonensis effect. Anti-plasmodium falciparum and anti-L.major effects of curcumin have also been reported. Turmeric has shown to have anti -viral activity[32].It acts as an efficient inhibitor. Curcumin prevents galactose-induced cataract formation at very low doses. Both turmeric and curcumin decreases blood sugar level in alloxan-induced diabetes in rat.Curcumin also decreases advanced glycaton end products inducedcomplications in diabetes mellitus [33].

Anti-Viral Activity

Cai (1988), showed in a study that hot water extract of dried rhizome in cell culture was active on vesicular stomatitis virus. The prescription included 10g each of Curcuma longa rhizome, Rheum officinale root, Cimicifugafoetida rhizome, Anemarrhenaasaphodeloides rhizome, Areca catechu seed, Magnolia officinalis bark and Scutellariabaicalensis root along with 5g Amomumtsaoko fruit, together with insects Bombyxmori and Cryptotympanapustulata. May et al. (1978), reported that water extract of dried rhizome in cell culture at a concentration of 10% was inactive on Herpes Virus Type- 2, Influenza virus A2 (Manheim 57), Polio-II and Vaccinia virus.

Anti-Microbiological Activity

Chang (1989), carried out a study to evaluate the antimutagenic effect of hot water extract of dried rhizome in comparison with Aflatoxin B1 induced mutagenesis and Aflatoxin and Mitomycin induced mutagenesis. Chang and co-workers (1989), found that hot water extract of dried rhizome on agar plate at concentration of 40 mg/plate and at a minimum toxic dose were inactive on Salmonella typhimurium TA100 vs Aflatoxin B1 induced mutagenesis. Metabolic activation had no effect on the results. Dried rhizome extract on agar plate at a concentration of 50 mg/ml was inactive on Salmonella typhimurium TA1535 vs Aflatoxin and Mitomycin induced mutagenesis. Another study by Deshpande.(1995) shows that water extract of rhizome at a concentration of 0.33 mg/ml was active on rat liver microsomes and the formation of labeledbenzo[a]pyrene-DNA adducts was inhibited. Infusion at a concentration of 2 mcg/ plate on agar plate was active on Salmonella typhimurium TA100. 1-methyl-3 nitro-1- nitroguanidine-induced mutagenesis was inhibited by 25%. There was a 38% inhibition of 4-nitro-Dphenylenediamineinduced mutagenesis of S. typhimurium TA98. Infusion of rhizome administered intra-gastric to mice at a dose of 3mg/animal was active. The incidence of benzo[a]pyrene induced bone marrow micro nucleated cells was decreased 40% by pre-treatment with the extract (Azuine, 1992). Powdered rhizome at a concentration of 0.033 mg/ml was active on rat liver microsomes. Formation of labeledbenzo[a]pyrene-DNA adducts was inhibited (Deshpande et al., 1995). Powdered rhizome administered intra-gastric to rats at a dose of 0.5% of the diet was active. Animals fed the diet for one month before being given 3-methylcholanthrene intraperitoneally, produced urine with reduced mutagenicity on S. typhimurium strains TA100 and TA98, with or without activation with S9, as assessed by Ames test (Polasa., 1991).

Toxicological Properties

Curcumin is considered to have a low toxicity in man and animals. In a clinical trial with 25 volunteers, administration of up to 8 gm of Curcumin per day has no apparent toxic sign. In another clinical trial in which humans were given 1.25-2.5 gm Curcumin per day confirmed the apparent safety of the substance (Chainani-Wu, 2003). There are no reports of adverse effects of either Curcumin or its analogues except for rare cases of contact dermatitis. Many women in Asia apply turmeric to their skin in an effort to minimize unwanted hair growth, but few experience dermatitis. Oral administration of Curcumin to rats at doses up to 5 g/kg caused no overt signs of toxicity. The American Herbal Association classifies turmeric as a

menstrual stimulant and some sources recommended avoiding Curcumin in pregnancy. Its use is not recommended during breast-feeding, as effects on breast-feeding infants are unknown (Oetari, 1996). Turmeric may have an antiplatelet activity (Shah, 1999), and its concurrent use with anticoagulants may lead to an additive effect. Although there are no reports of this in humans, its use should be avoided in patients with bleeding disorders and bile duct obstruction and should only be used under the supervision of a physician in patients with gallstone[34].

Adjuvants

Piperine is known to inhibit hepatic and intestinal glucuronidation. When combined with piperine, the elimination half-life and clearance of curcumin were significantly decreased, resulting in an increase of bioavailability to 154% that of curcumin alone in rats. In contrast, the increase in bioavailability was 2000% in humans, clearly showing that the effect of piperine on bioavailability of curcumin is much greater in humans than in rats. A human volunteer trial conducted by our group revealed the enhancing effect of piperine on serum curcumin level. Six healthy adult male human volunteers took 2 g of curcumin with or without 5 mg piperine (as Bioperine1) in this cross-over design study. Three subjects were randomized to receive curcumin only, while the remaining three received the curcumin + piperine combination. One week following initial drug administration, volunteers were crossed over to the other therapy and blood samples were obtained for evaluation. The presence of piperine was found to double the absorption of curcumin.

The effect of piperine on tissue uptake of a radiolabeled fluoropropyl-substituted curcumin was evaluated in mice. Mice that received piperine had 48% greater brain uptake of curcumin after 2 min than mice that did not receive piperine. However, the uptake in other organs was not found to be significantly improved by piperine in this study; the authors think this observation can be explained by the poor solubility of piperine in 10% ethanolic saline (injection medium).

Some other agents that showed a synergistic effect when used in combination with curcumin in various in vitro studies look promising for further evaluation. Five patients with familial adenomatous polyposis who had undergone colectomy received curcumin 480 mg and quercetin 20 mg orally 3 times a day. The number and size of polyps were assessed at baseline and after therapy. All five patients had decreases in polyp number and size, 60.4% and 50.9%, respectively, from baseline after a mean of 6 months of this treatment. Though

the authors did not compare the effects of this combination treatment with those of the single agents, this study at least throws light on the therapeutic value of this combination[35].

The synergistic inhibitory effect of curcumin and genistein against pesticide-induced growth of estrogen-dependent MCF-7 breast carcinoma cells has been reported. It was showed that a combination of curcumin and genistein completely inhibited the cellular proliferation induced by an individual pesticide or a mixture of pesticides, and that the inhibitory effect was superior to the individual effects of either curcumin or genistein. Curcumin uptake within rat skin after topical application of a curcumin hydrogel, with or without eugenol or terpineol pretreatment, was evaluated in an in vivo study. The effects of eugenol and terpeneol as enhancers of skin curcumin absorption were demonstrated; 8h after application, curcumin levels in skin were 2.2- and 2.5-fold greater, respectively, in mice that received eugenol or terpeniolpretreatment than in mice that received curcumin alone. These observations indicate that these absorption- enhancing agents may also be effective as adjuvants. Epigallo-catechin-3-gallate, a component of green tea, could counteract certain activities attributed to curcumin. BCM-95 (also called Biocurcumax) curcuminoids combined with turmeric oil (turmerons) in a specific proportion enhanced the bioavailability and showed better absorption into blood and had longer retention time than curcumin alone. Currently a multicenter, phase II, randomized, double-blinded, placebo-controlled clinical study is ongoing to assess the efficacy and safety of BCM-95 in oral premalignant lesions or cervical cancer.

Conclusion

Now-a-days, economic and medicinal importance of turmeric (Curcuma longa Linn.) is an established fact, besides further research methodology is practiced to reach the extreme and extensive value of its diverse pharmacological and other uses. It has been appreciably used in traditional medicine as a household remedy for various diseases. Its anti-inflammatory, anticancer and anti-oxidant properties if exploited efficiently may benefit the mankind in colorful ways. It has a low toxic effect on body hence large doses can be given without any fear of toxicity that reflects its broad therapeutic index. This plant has benefited us of its various medicinal values besides other utilities.Turmeric has been used in Ayurveda medicine since ancient times, with various biological applications. Although some work has been done on the positive medical applications, no studies for drug-development have been carried out as yet. Although the crude extract has numerous medical applications,

clinical applications can be made only after extensive research on its bioactivity, mechanism of action, pharmacotherapeutics and toxicity studies. However, as curcumin is now available in pure form, which a shows a wide spectrum of biological activities, it would be easier to develop new drugs from this compound after extensive studies on its mechanism of action and pharmacological effects. Recent years have seen an increased enthusiasm in treating various diseases withnatural products. Curcumin is a non-toxic, highly promising anti-oxidant compound having a wide spectrum of biological functions. It is expected that curcumin may find application as a novel drug in the near future to control various diseases, including inflammatory disorders, carcinogenesis and oxidative stress-induced pathogenesis. Thus, Turmeric holds promises of holding significant clinical benefits.

Corresponding Author

E. Roja,

Tutor, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai – 600 077, Tamil Nadu, India

Email Id: itsrojaa@gmail.com

Contact Number: +91 99941 74480.

References

1. D.NidaFathimaJ.Curcumin – role in cancer. Pharm. Sci. & Res.2014; 6(11):351-355.
2. PrasannaNeelakanta,NithyaJagannathan, NabeelNazar“Ethnopharmacological approach in Endodontic Treatment: A Focused Review”, Int. J. Drug Dev. & Res., 2011;3(4): 68-77.
3. Roughley, P. J. and Whiting, D. A., Experiments inthe biosynthesis of curcumin. J. Chem. Soc., 1973; 20: 2379–2388.
4. Sasikumar B, Genetic Resources of Curcuma:Diversity characterization and utilization, Plant Genet Res, 2005;3(2);230-251. 10.Sharma, O.P.Antioxidant activity of curcumin and related compound. Biochem. Pharmacol.,1976;25:1811-1812.
5. Bandyopadhyay, U., Das, D. and Banerjee, R. K., Reactive oxygen species: oxidative damage and pathogenesis. Curr Sci., 1999; 77; 658–666.

6. See, P., Lee, and S. T. and Chan, S. Y., Protective effects of curcumin against oxidative damage on skin cell in vitro: its implication for wound healing. J. Trauma, 2001; 51; 927–931.
7. Kelly, M. R., Xu, J. Alexander, K. E. and Loo, G., Disparate effects of similar phenolic phytochemicals as inhibitors of oxidative damage to cellular DNA. Mutat. Res., 2001; 485; 309–318.
8. Srimal, R. C. and Dhawan, B. N., In Development of Unani drugs from Herbal Sources and the Role of Elements in their Mechanism of Action (ed. Arora, B. B.), Hamdard National Foundation Monograph, New Delhi, 1985;201-212.
9. SrihariRao, T., Basu, N. and Siddqui, H. H., Anti-inflammatory activity of curcumin analogues. Indian J. Med. Res., 1982; 75, 574– 578.
10. Yegnanarayan, R., Saraf, A. P. and Balwani, J. H., Comparison of anti-inflammatory activity of various extracts of Curcuma longa (Linn). Indian J. Med. Res., 1976;64, 601–608.
11. Deodhar, S. D., Sethi, R. and Srimal, R. C., Preliminary study on antirheumatic activity of curcumin (diferuloylmethane). Indian J. Med. Res., 1980;71; 632–634.
12. Sreejayan Rao, M. N., Curcuminoids as potent inhibitors of lipid peroxidation. J. Pharm. Pharmacol., 1994; 46;1013–1016.
13. Lal B, Kapoor AK, Asthana OP.Efficacy of curcumin in the management of chronic anterior uveitis. Phytother Res. 1999;13; 318-322.
14. Lal B, Kapoor AK, Agrawal PK. Role of curcumin in idiopathic inflammatory orbital pseudotumours. Phytother Res. 2000;14:443-447.
15. Garg SK: Effect of Curcuma longa (rhizomes) on fertility in experimental animals. Planta Med.; 1974; 26: 225–227.
16. Rithaporn T, Monga M and Rajasekharan M: Curcumin: a potential vaginal contraceptive. Contraception; 2003; 68: 219–223.
17. Liao S, Lin J, Dang MT, Zhang H, Kao YH, Fukuchi J and Hiipakka RA: Growth suppression of hamster flank organs by topical application of catechins, alizarin, curcumin and myristoleic acid. Arch. Dermatol. Res.2001; 293: 200–205.
18. Chen, H. W. and Huang, H. C., Effect of curcumin on cell cycle progression and apoptosis in vascular smooth cells. Br. J. Pharmacol., 1998;124:1029–1040.
19. Hour TC, Chen J and Huang CY: Curcumin enhances cytotoxicity of chemotherapeutic agents in prostate cancer cells by inducing p21 (WAF1/ CIP1)

and C/EBPbeta expressions and suppressing NF-kappa activation. Prostate; 2002; 51: 211-218.

20. Kuttan R, Bhanumathy P, Nirmala K and George MC: Potential anticancer activity of turmeric (Curcuma longa). Cancer Lett; 1985; 29: 197–202.
21. Bush JA, Cheung KJ and Lee G: Curcumin induces apoptosis in human melanoma cells through a Fasrecep-tor/caspase-8 pathway independent of p53. Exp Cell Res; 2001; 271: 305-314.
22. Cheng AL, Hsu CH and Lin JK: Phase I clinical trial of curcumin, a chemopreventive agent, in patients with high-risk or pre-malignant lesions. Anticancer Res; 2001; 21: 2895-2900.
23. BanerjeeA and Nigam SS: Antimicrobial efficacy of the essential oil of Curcuma longa. Indian J. Med. Res.1978; 68: 864–866.
24. Kumar S, Narain U, Tripathi S and Misra K: Synthesis of curcuminbioconjugates and study of their antibacterial activities against beta-lactamaseproducing microorganisms. Bioconjug. Chem.; 2001; 12: 464–469.
25. Koide T, Nose M, Ogihara Y, Yabu Y and Ohta N: Leishmanicidal effect of curcumin in vitro. Biol. Pharm. Bull.; 2002; 25: 131–133.
26. De Clercq E: Current lead natural products for the chemotherapy of human immunodeficiency virus (HIV) infection. Med. Res. Rev; 2000; 20: 323–349.
27. Sajithlal GB, Chittra P and Chandrakasan G: Effect of curcumin on the advanced glycation and cross-linking of collagen in diabetic rats. Biochem. Pharmacol; 1998;56: 1607–1614.
28. McMichael AJ, McCall MG, Hartchorne JM, Woodings TL: Patterns of gastrointestinal cancer in European migrants to Australia: the role of dietary change. Int J Cancer 1980;5:431-437.
29. Chattopadhyay I, Biswas K, Bandyopadhyay U, Banerjee RK: Turmeric and curcuminBiological actions and medicinal applications. Curr Sci 2004; 87:44-50
30. Aggarwal, B.B, Kumar, A., Bharti, A.C., Anticancer potential of curcumin in: Preclinical and studies, Anticancer Res.2003; 23: 363- 393.
31. Aggarwal BB, Sundaram C, Malani N, and Ichikawa H: Curcumin: The Indian solid gold. AdvExp Med Biol 2007; 595:1-75.
32. Ammon HP, Wahl MA: Pharmacology of Curcuma longa. Planta Med 1991; 57:17.

33. Jurenka JS. Anti-inflammatory properties of curcumin, a major constituent of Curcuma longa: a review of preclinical and clinical research. Altern Med Rev. 2009; 14(2):141-153.
34. Goel A, Aggarwal BB. Curcumin, the golden spice from Indian saffron, is a chemosensitizer and radiosensitizer for tumours and chemoprotector and radioprotector for normal organs. Nutr Cancer. 2010; 62(7):919-930.
35. Murphy EA, Davis JM, McClellan JL, Gordon BT, Carmichael MD. Curcumin's effect on intestinal inflammation and tumorigenesis in the Apc (Min/+) Mouse. J Interferon Cytokine Res. 2011;31(2):219-226.

Biomedical Waste Management

RathnaSubhashini M.H[1], Vishnu Priya[2], Raghu Sandhya[3]
Graduate Student[1],Professor and Head[2],Department of Biochemistry,Reader[3],Department of Conservative Dentistry and Endodontics
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Proper handling and disposal of medicine wastes plays a vital parts of health care work place infection management program. Effective waste disposal isachieved solely by considering the assorted parts of the waste management system as an integral a part of hospital designing and planning. Medical care is important for our life and health, however the waste generated from medical activities represents a true drawback of living nature and human world. Improper management of waste generated in health care facilities causes an instantaneous health impact on the community, the health care employees and on the atmosphere. Indiscriminate disposal of biomedical waste management and exposure to such waste possess serious threat tothe atmosphere and to human health that needs specific treatment and management before its final disposal. Most of the foundations world-wide don't seem to be specific for dental biomedical waste management and hinder simple understanding by dental practitioners.Considering its disposal and ill-effects on health and the atmosphere, a method of dental waste segregation protocol was needed. The current review deals with the definition, source, classification, methods to dispose and problems related to handling and disposal of bio medical waste.

Key Words: *Biomedical, waste, management, disposal, practice*

Introduction

Biomedical waste management has emerged as a problem of major concern not only to hospitals, institution authorities but also to the surroundings. The biomedical wastes generated from health care units depends on a variety of things like waste management strategies, forms of health care units, occupancy of tending units, specialization of tending units, quantitative relation of reusable things in use, handiness of infrastructure and resources etc [1]. Health care facilities like laboratories, clinics, nursing homes, medical, dental,and veterinary hospitals, generate a waste stream varied in its composition.Among these facilities the hospitals contribute most wastes [2].The proper management of biomedical waste has become a worldwide humanitarian topic today. Hazards of poor biomedical waste management have aroused as a major concern in affecting human and the environment [3]. Hospital waste could be a potential jeopardy to the health care staff, public and flora and fauna of the realm [4].According to Biomedical Waste (Management and Handling) Rules, 1998 of India “Any waste which is generated during the diagnosis, treatment or immunization of human beings or animals or in research activities pertaining thereto or in the production or testing of biological".Inadequate and inappropriate handling of health-care waste could have serious public health consequences and a major impact on the environment. Considering the increasing range of dentists in urban areas and magnified awareness regarding the dental treatment amongst the general public, it is more important to create awareness on biomedical waste management and make it anintegral feature of dental services.The current review deals with the definition, source, classification, methods to dispose and problems related to handling and disposal of bio medical waste management

Need for Biomedical Waste Management

The content of infectious waste within the total waste generated in an hospital may be 20 percentage. However it haspotential to infect whole of hospital waste if it is not transported in and prescribed manner [4]. Waste generated at hospitals and health care facilities is totally different from, general municipal waste. The municipal waste may require only one system for assortment, transportation and final disposal. Against this the biomedical waste may have additional systems, since it includes body components, human

and animal tissues, radioactive material, gauze, cotton, plastics, plaster-of-paris casts, infected liquid waste, blood and laboratory wastes. Medical waste generated at totally different health care facilities presents environmental and public health risks. Inappropriate treatment and disposal will unfold infectious diseases like TB, hepatitis, typhoid, HIV infection, or perhaps AIDS [2,5]. Injuries from sharps leading to infection among all categories of hospital personnel and waste handler. Risk of infection outside hospital for waste handlers, scavengers and at time general public living in the hospitals vicinity is always high.

Classification of Biomedical Waste

The World Health Organization (WHO) has classified biomedical waste into eight categories:

1. General Waste
2. Pathological
3. Radioactive - It is genotoxicand thus may affect genetic material. The effects of exposure can range from headache, dizziness, and vomiting to malignancies.
4. Chemical wastes
5. Pharmaceuticals - Hazards from chemical and pharmaceutical waste [5]. Many of the chemicals are toxic, genotoxic, corrosive, flammable, reactive, explosive or shock sensitive. Although present in small quantity they may cause intoxication, either by acute or chronic exposure and injuries, including burns. Reactive chemicals may form highly toxic secondary compounds
6. Sharps -Pathogens in infectious waste may enter the human body through a prick, abrasion or cut in the skin, through mucous membranes by inhalation or by ingestion. There is a particular concern about infection with Human immunodeficiency virus and hepatitis virus B and C, for which there is a strong evidence of transmission via health-care waste. [5].
7. Infectious to potentially infectious waste
8. Pressurized containers

Sources of Biomedical Waste

The sources of health-care waste are classified as major or minor according to the quantities created. [1-5]

Major Sources

- Govt. hospitals/private hospitals/nursing homes/ dispensaries.
- Primary health centers.
- Medical colleges and research centers/ paramedic services.
- Veterinary colleges and animal research centers.
- Blood banks/mortuaries/autopsy centers.
- Biotechnology institutions.
- Production units.

Minor Sources

- Physicians/ dentists' clinics
- Animal houses/slaughter houses.
- Blood donation camps.
- Vaccination centers.
- Acupuncturists/psychiatric clinics/cosmetic piercing.
- Funeral services.
- Institutions for disabled persons

Table 1: Steps in management of health care wastes

Color cryptography	**Type of Material**	**Waste Category**	**Treatment option**
Yellow cover	Plastic bag	Human Anatomical Waste, Animal Waste. Human tissues, organs, body parts including the waste generated from animals used in experiments or testing in veterinary hospitals or colleges or	Incineration or burning

		animal houses and fetus below the viability period. **Expired or Discarded Medicines** **Chemical Waste:** Chemicals used in production of biological and used or discarded disinfectants. Discarded linen, mattresses, beddings contaminated with blood or body fluid. **Chemical Liquid Waste :** Liquid waste generated due to use of chemicals in hospitals and health care centers.	
Red cover	Plastic bag	Infected /Non-Infected Plastics Contaminated Wastes generated from disposable items such as tubing, bottles, intravenous tubes and sets, catheters, urine bags, syringes (without needles and fixed needle syringes) and vaccutainers with their needles cut) and gloves	Shredding after Autoclaving
Blue cover	Plastic bag	Broken or discarded and contaminated glass including medicine vials and ampoules except contaminated cytotoxic wastes.	Autoclaving /Destruction
Black cover	Carboy bag	Needles, syringes with fixed needles, needles from needle tip cutter or burner, scalpels, blades, or any other contaminated sharp object that may cause puncture and cuts. This includes both used, discarded and contaminated metal sharps	Autoclaving /Destruction

Problems Associated with Biomedical Waste

A major issue related to current Bio-Medical waste management in many hospitals is that the implementation of Bio-Waste regulation is unsatisfactory as some hospitals are disposing of waste in a haphazard, improper and indiscriminate manner. Various communicable diseases spread through water, sweat, blood, body fluids and contaminated organs and it is very important to be disposed properly [4-6]. The bio medical waste scattered in and around the hospitals invites flies, insects, rodents and dogs that are responsible for the spread of communicable disease like plague and rabies. Improper disposal of disposable syringes, needles, IV infusion sets and other articles like glass bottles without proper sterilization are responsible for the transmission of diseases. It became the primary responsibility of Health administrators to manage hospital waste in the safest and eco-friendly manner[6].The waste, if allowed to accumulate, is a health hazard, because it decomposes and favors fly breeding, it attracts rodents and vermin, the pathogens, which may be present in the waste may be conveyed back to man's food through flies and dust. There is a possibility of water and soil pollution. Heaps of refuse present an unsightly appearance and nuisance from bad odors [7].

Biomedical Waste Management Process

There is a big network of Health Care Institutions in India. The hospital waste like body parts, organs, tissues, blood and body fluids along with soiled linen, cotton, bandage and plaster casts from infected and contaminated areas should be properly disposed [8]. This involves the following steps,

1. Waste collection
2. Segregation
3. Transportation and storage
4. Treatment & Disposal
5. Transport to final disposal site
6. Final disposal

Treatment and Disposal Methods

Incineration Technology

It is a high temperature thermal process involving combustion of the waste under controlled condition for converting them into inert material and gases. Incinerators can be oil fired or electrically powered or a combination of both. Three types of incinerators are used for hospital waste - multiple hearth type, rotary kiln and controlled air types. All the types can have primary and secondary combustion chambers to ensure optimal combustion.It is a controlled combustion processin which the waste is completely oxidized and harmful microorganisms present in it are destroyed/ denatured under high temperature[7-9]

Non-IncinerationTechnology

Non-incineration treatment includes four basic processes: thermal, chemical, irradiative, and biological. The main purpose of the treatment technology is to decontaminate waste by destroying pathogens [10-12].

Autoclaving

- ✓ The autoclave operates on the principle of the standard pressure cooker.
- ✓ The process involves steam at high temperatures.
- ✓ The steam generated at a high temperature penetrates waste material and kills all the micro organism .

In the Gravity type, air is evacuated with the help of gravity alone. The systemoperates with temperature of 121^{o} C and steam pressure of15 psi for 60-90 minutes. Vacuum pumpsare used to evacuate air from the Pre vacuum autoclave so that the time cycle can be reduced to 30-60 minutes. It operates at about 132^{o} C.Retort type autoclaves are designed to work at much higher steam temperature and pressure. Autoclave treatment has been recommended for microbiology and biotechnology waste, sharps and solid wastes. Autoclave treatment has been recommended for microbiology and biotechnology waste, waste sharps, soiled and solid wastes [11-16]. Common items from a hospital that should be autoclaved for sterilization include:

- Gloves, gauze, gowns, or other items worn by patients or doctors.
- Used bandages, Band-Aids, and gauze.
- Used or contaminated sharps (needles, syringes, scalpels, medical knives, etc.)

Microwave Irradiation

The microwave is based on the principle of generation of high frequency waves.These waves cause the particles within the waste material to vibrate, generating heat.This heat generated from within kills all pathogens.Irradiation attempts to sterilize waste by exposing it to gamma rays that are fatal to bacteria. A radioactive isotope of cobalt is employed. This is basically the same radiation source used for radiation treatment for cancer. In irradiation for sterilization of equipment or treatment of waste, the radiation is intended to kill pathogens, or probably any living cells. Ultra violet rays is lower frequency and generally less lethal than gamma radiation.

Chemical Methods

Chemical disinfection, primarily through the use of chlorine compounds, kills microorganisms in medical waste and can sometimes oxidize hazardous chemical constituents. Chlorine has been used for many disinfecting processes for years. Ethylene oxide treatment is used for disinfection and in treatment of medical waste. Ethylene oxide treatment is used more often to sterilize equipment that will be reused. This gas, mixed with air at a ratio of at least 3% EtOgas, forms an explosive mixture. Most of the time, it is mixed with Nitrogen or CO2.1 % of hypochlorite solution can be used for chemical disinfection.

Plasma Pyrolysis

Plasma pyrolysis is a technology for safe disposal of medical waste. It is an eco-friendly technology, which converts organic waste into commercially useful byproducts. The heat generated by the plasma enables disposal of all types of waste including municipal solid waste, biomedical waste and hazardous waste in a safest and most reliable manner. Medical wastes arepyrolysed into CO, H2, and hydrocarbons when it comes in contact with the plasma-arc. These gases are burned and produce a high temperature (around 1200^{o}C) [13-17]

Benefits of Biomedical Waste Management

- ✓ Cleaner and healthier surroundings.
- ✓ Reduction in the incidence of hospital acquired and general infections.
- ✓ Reduction in the cost of infection control in the hospital.

- ✓ Reduction in the possibility of disease and death due to reuse and repackaging of infectious disposables.
- ✓ Low incidence of community and occupational health hazards[18-20]
- ✓ Reduction in the cost of waste management and generation of revenue through appropriate treatment and disposal of waste.
- ✓ Improved image of the healthcare establishment and increase the quality of life[23].

Legislative Aspect in Relation to Biomedical Waste

Various central legislation related to biomedical waste management in India are as follows

- ✓ The water (prevention and control of pollution) Act, 1974
- ✓ The Air (prevention and control of pollution) Act, 1981
- ✓ The Environment(Protection) Act,1986
- ✓ The hazardous waste(management and handling) rules,1998
- ✓ The Biomedical waste(management and handling) rules,1998
- ✓ Municipal Solid waste (management and handling) rules, 2000
- ✓ The Biomedical waste(management and handling) rules Amendment ,2000 and 2003
- ✓ The Bio-medical Waste (Management and Handling) Rules, 2011 [Draft].

Recommendations

- ✓ Bio-medical waste should not be mixed with other waste of Municipal Corporation.
- ✓ Private hospitals should be allowed to use incinerator. For this purpose a specific fee can be charged from private hospitals.
- ✓ As provided by bio-medical waste rules, the whole of the waste should be segregated into colors due to their hazardous nature.
- ✓ Bio-medical waste management board can be established in every districts.
- ✓ Housekeeping staff should wear protective devices such as gloves, face masks, gowned, while handling the waste.
- ✓ There is biomedical waste label on waste carry bags.
- ✓

Conclusion

The segregation of waste at its supply is the key step to protect the environment. The challenge is to manage growing quantities of medical specialty waste that transcend past practices. If we would like to safeguard the environment and health of community we should sensitize ourselves to the present necessary issue. As a dentist we tend to do have some responsibility toward our surroundings and descendents. The method we dispose waste affects the standard of the atmosphere that we should expire to living standard of the future generations..The authoritative bodies in Asian nations and alternative developing nations ought to strictly implement and monitor biomedical waste management consistent with their needs during a systematic and oversimplified manner. The governmental bodies ought to take responsibility of creating these services obtainable to the active dentists furthermore at dental hospitals.

Corresponding Author

Raghu Sandhya

Reader,Department of Conservative Dentistry and Endodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Mail Id: drsandhyaendo@gmail.com

Contact Number: +91 9884610410

Reference

1. Mathur P, Patan S, Shobhawat S. Need of biomedical waste management system in hospitals-an emerging issue-a review. Curr World Environ. 2012;7(1):117-24.
2. Mandal SK, Dutta J. Integrated bio-medical waste management plan for Patna city. Institute of Town Planners, India Journal. 2009;6(2):1-25.
3. Singh VP, Biswas G, Sharma JJ. Biomedical waste management-an emerging concern in Indian hospitals. Indian Journal of Forensic Medicine &Toxicology. 2007;1(2):39-44.
4. Chandra H. Hospital waste: An environmental hazard and its management. C. Newsletter of ISEB, India. 1999 Jul;5(3):80-5.

5. Kishore J, Ingle GK. Biomedical Waste Management in India. primates. 1999;17(15-16):1869-82.
6. Ngai EW, Moon KK, Riggins FJ, Candace YY. RFID research: An academic literature review (1995–2005) and future research directions. International Journal of Production Economics. 2008 Apr 1;112(2):510-20.
7. Akter N. Medical waste management: a review. Environmental Engineering Program, School of Environment, Resources and Development Asian Institute of Technology, Thailand. 2000 Jan:1-25.
8. Gravers PD. Management of Hospital Wastes-An overview. InProceedings of National workshop on Management of Hospital Waste 1998 (pp. 16-18).
9. Sunil Kumar VC, Manjunatha M, Badami V, Pradeep PR. Biomedical waste management: A review. J Oral Health Comm Dent. 2012;6:141-4.
10. Thornton J, McCally M, Orris P, Weinberg J. Hospitals and plastics. Dioxin prevention and medical waste incinerators. Public Health Reports. 1996 Jul;111(4):298.
11. Agrawal AG, Singh R. Understanding and simplifying bio-medical waste management: A training manual for trainers. InUnderstanding and simplifying bio-medical waste management: a training manual for trainers 2005 Jan. Toxics Link.
12. Riswana NA. Knowledge and Attitude in Regards to Dental Care Waste Management amongst Dental Students-Questionnaire Study. Journal of Pharmaceutical Sciences and Research. 2016 Sep 1;8(9):1070.
13. Col SR, Col SB, Col VS. Biomedical waste management: an infrastructural survey of hospitals. Medical Journal Armed Forces India. 2004 Oct 1;60(4):379-82.
14. Chitralekha M, Agrawal S. waste management: A Social Perspective A Study reference to Hazardous bio medical waste. In2010 International Conference on Economics, Business and Management, IPEDR 2011 (Vol. 2).
15. Sikka S. Biomedical Waste in Indian Context. B-6/15 Rajouri Garden, New Delhi. 110 027, India; 2006.
16. Sharma S, Chauhan SV. Assessment of bio-medical waste management in three apex Government hospitals of Agra. Journal of Environmental Biology. 2008 Mar 1;29(2):159.

17. Samaddar S, Kundu N. Biomedical Waste Management: An Infrastructural case study of Kolkata based Hospitals.
18. Amin R, Gul R, Mehrab A. HOSPITAL WASTE MANAGEMENT. Professional Medical Journal. 2013 Nov 1;20(6)
19. Ross DS. Emerging need for bio-medical wastes management in hospitals. International Journal of Management, IT and Engineering. 2014 Jul 1;4(7):421.
20. Dutta R, Prashanth R, Parasuraman G, Jain T, Raja D, Dcruze L. Knowledge, attitude and practice of bio-medical waste management among private practitioners in Poonamallee taluk, Chennai. International Journal Of Community Medicine And Public Health. 2017 Jul 22;4(8):2930-3.
21. Hegde V, Kulkarni RD, Ajantha GS. Biomedical waste management. Journal of Oral and Maxillofacial Pathology. 2007 Jan 1;11(1):5.
22. Thota MM, Bathala LR, Theruru K, Shaik S, Jupidi B, Rayapati S. " There's plenty of room at the bottom": The biomedical waste management in dentistry. Journal of Dr. NTR University of Health Sciences. 2014 Jul 1;3(3):149.
23. Shah C. Biomedical Waste Management: A Move towards Green Environment. Asia Pacific Journal of Management & Entrepreneurship Research. 2012 Nov 1;1(3):5.
24. Byahatti SM, No P. Hospital Waste Management. Guident. 2012 Feb 1;5(3).

Bleeding Disorders in Dental Care

Nazia Zareen.I[1], Jothi Priya[2], L. Leelavathi[3]

Graduate Student[1], Senior Lecturer[2], Department of Physiology, Senior Lecturer[3],Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Bleeding disorders are a group of disorders that share the inability to form a proper blood clot. They are characterized by extended bleeding after injury, surgery, trauma .Initial recognition of a bleeding disorders which may indicate presence of systemic pathological process may occur in dental practice. Oral care providers must be aware of the impact of bleeding disorders on the management of dental patients.With appropriate management strategies,nearly all bleeding disorder patients can benefit from the full range of dental procedures available to establish and maintain good oral health. Patient with bleeding disorders places a major challenge for dentists. Hence, adequate understanding of the medical conditions is essential to reduce the risk of dangerous complications and by providing required replacement therapy for the common bleeding disorders before the dental procedure and by theselecting the appropriate conservative treatment approaches, use of local hemostatic measure to facilitate haemostatis. From this review we can understand the types and management of bleeding disorders and its effects on the delivery of oral health care.

Key Word:*Bleedingdisorders, von willebranddisease,mennorahgia,thrombocytopenias,streptokinase.*

Introduction

Bleeding disorders are due to altered ability of blood vessels, platelets, and coagulation factors to maintain haemostasis. Most of the bleeding disorders are iatrogenic.These may be inherited due to genetic transmission or acquired secondary to diseases affecting vascular wall integrity, platelets, and coagulation factors or due to drugs, radiation or chemotherapy[1]. Three factors of haemostatis are vascular, platelet and coagulation phase is followed by fibrinolytic phase which forms fibrin as clot.

Vascular phase results in vasoconstriction of arteries and veins at the site of injury , it also builds up extra vascular pressure by blood lost from cut vessels and it lasts for seconds[2,3].

Platelet phase – it also known as primary haemostatis in which platelets interact with elements of the damaged vessel wall, leading to the initial formation of a “platelet plug”[2,3]

Coagulation phase - it takes place more slowly than other phases where blood lost into surrounding area and coagulates through both extrinsic and common pathways. Fibrin secure the primary platelet plug, exceptionally in larger blood vessels where the platelet plug is inadequate only to block hemorrhage[2,3].

Fibrinolytic phase – activates simultaneously with the coagulation and functions to maintain fluidity of blood during coagulation, serves as clot lysis and anti thrombic agents are released[2,3].

Coagulation casade –development of blood clot is changeover from a soluble protein fibrinogen into insoluble fibrin under the response of a thrombin. The conversion of prothrombin to thrombin involves a series of plasma serine proteases that normally exist ininactive and pro-enzyme forms[2,3].

Dentists may encounter patients with various types of bleeding disorders in their daily practice[4].Clinical bleeding can be presented in two forms:the first can occur during surgery and the second can manifest several days after the procedure.In both situations,the clinician need to take immediate action to control the haemorrhage and stabilize the patient[5].Dental procedures are associated with post operative bleeding .Procedures like extractions,oral surgeries

and periodontal surgery are the most common invasive procedures[6].In order to control haemostasis following an injury to blood vessel,a series of events is iniated are as follows:

- Local vasoconstriction
- Adhesion and aggregation of platelets
- Activation of the clotting casade to create a fibrin clot
- Activation of coagulation inhibitors to restrict coagulation to the site of the injury;
- Fibrinolysis occurs later to restore vessel potency[4].

Proper medical and dental reports of the patient is necessary before treatment ,especially if an invasive dental procedure is planned.Many bleeding disorders, such as hemophilia and Von Willebrand's disease run in a families; therefore a family history of bleeding disorders should be carefully elicited[7].This article is review on to understand causes of bleeding disorders prevailed during dental procedures.

Type of Bleeding Disorders

Congential Coagulation Defects

Haemophilia A is due to deficiency of clotting factor VIII. It is an inherited X-linked recessive trait found in 1:5,000 in the male population. Symptoms may include delayed bleeding,epitaxis,spontaneous gingival bleeding and hemarthrosis[9].Severe bleeding is seen when F VIII level is less than1% of normal. Severe hemorrhage leads to joint synovitis and hemophilic arthropathies, intramuscular bleeds, pseudotumors (encapsulated hemorrhagic cyst).Moderate bleeding is found when F VIII levels are 1% to5% of normal[10].Treatment for hemophilia is by administration of a intravenous replacement of Factor VIII or IX using purified plasma derived concentrates or rather by recombinant factor concentrates. Desmopressin (DDAVP) is used to achieve a transient increase in factor VIII level through the release of endogenous factor VIII in patients with hemophilia A and Von Willebrand's disease[11]. Haemophilia B is due to deficiency of F IX(Christmas factor).It is managed by replacement therapy with highly purified,virally inactivated factor IX concentrates. Factor IX replacementtreatment prothrombin complex concentrates is used [9].Von Willebrand's disease

is a unique disorder that was described originally by Erik Von Willebrand in 1926.It is an autosomal dominant trait with varying penetrance,defect is found in the F VIII protein complex. It is classified as type I to type IV and may vary in severity. For mild condition,use of DDAVP may be sufficient,bt severe disease warrants factor VIII replacement [11].

Rare Coagulation Factor Deficiencies

Rare coagulation factors deficienciesare Afibrinogenaemia, Factor II, Factor V, Factor XI and Factor XIII. These disorders are inherited as autosomal recessive traits and unambiguous clinically in homozygotes or compound heterozygotes. Treatment for rare coagulation factor deficiencies are fresh frozen plasma is used as the source for others(eg Factor V)[6].Fresh whole blood platelet concentrates, factor VIII concentrates[11].

Acquired Coagulation Abnormalities

Patients on eternal anti coagulant therapy with either warfarin or heparin are at increased liablity of bleeding with trauma or surgical procedures. In liver diseases,the synthesis of clotting factors may be reduced due to parenchymal damage or obstruction[6]. Management benefits for hemostatic shortage in liver diseasesincludes vitamin K and fresh frozen plasma in fusion for prolonged prothrombin time and partial thrombo plastin time;cryoprecipitate for replacement of factor VIII deficiency; and replacement therapy for disemminated intravascular coagulation[11]. Drug doses frequently needed to be modified in these patients due to impaired liver function. Warfarin, low molecular weight heparin and dicumarol are the commonly used anticoagulant drug[6].

Platelet Disorders

The large number of platelet disorder can be inherited and acquired and it is broadly categorized as defects of number(thrombocytopenia)or of function. Its classification is somewhatbased on the platelet disorders which are characterized by both decreased number and function[6]. Thrombocytopenias are primarily managed acutely with transfusions of platelets to maintain the minimum level of 10,000 to 20,000/mm^3 necessary to prevent spontaneous hemorrhage.

Congential Platelet Disorder

Congential abnormalities of platelet function or production are rare.Glannzmann's thrombocytopenia is a qualitative disorder characterized by a deficiency in the platelet membrane glycoproteins IIa and IIIb. Clinical signs include are brusing,epistaxis,gingival hemorrhage,and menorrhagia.Treatment of oral surgical bleeding involves platelet transfusion and use of antifibrinolytics and local heamoistatic agents[13,14].

Acquired Platelet Disorder

Idopatic or immune thrombocytopenia purpura(itp) and thrombotic thrombocytopenia purpura (ttp) clinical science are petechiae and purpura over the chest, neck and limbs . mucosal bleeding occur in oral cavity and git tracts. Thrombocytopenia is a component of other hematologic disease such as myelodysplastic disorder,aplastic anemia,and leukemia[13]. Alcohol can induce thrombocytopenia. Drug induced platelet is also part of thrombocytopenia,a large number of drugs attenuate platelet activity. The most common is aspirin,acetylates COX which blocks TxA_2 release from activated platelets.Other nonsteroidal anti-inflammatory drugs (NSAID'S)such as ibuprofen,naproxen,ketorolac and indomethacin inhibit COX.[6].

Vascular Disorders

Vascular defects are rare and usually associated with mild bleeding confined to skin or mucosa. It relates diseases such as epithelium and connective tissue of blood vessels. Scurvy (vitaminC deficiency) affects the formation of connective tissue and also perivascular connective tissue network and weakening of capillaries will lead to hemorrhage[15]. It can be treated with laser ablation and embolization.

Fibrinolytic Disorders

Disorders of fibrinolytic system can lead to hemorrhage when clot breakdown is enhanced ,or excessive clotting and thrombosis when clot breakdown mechanisms are retarded. They are two types of fibrinolysis are primary fibrinolysis and reduced fibrinolysis which is caused due to plasminogen activators,prostate carcinoma is caused when there is rise in plasmin levels and fibrinolysis. Medications are streptokinase,urokinase,TPA is used to accelerate clot lysis in patient with acute thromboembolism[17].

Laboratory Tests

Quantitative

Normal platelet count – 1,50,000 – 4,40,000/mm^3

Thrombocytopenia - < 150,000/mm^3

Bleeding time = 3-5 min

Clotting time = 4- 10 min

Partial thromboplastin time = 25-45 seconds

Thrombin time = 9-13 seconds

Qualitative

Ivy bleeding time = 1-6 min

Closure time from PFA 100 = 60-120 seconds [12].

Dental Management

Managing a patient with bleeding disorders is most important during dental treatment as it requires the infusions of clotting factor and medications to control bleeding. Based on the severity of conditon, invasive procedures to be planned in order minimise the hemostatis. Patient undergoing coagulation factors replacement or other anti platelet drugs must be noticed and stopped for certain periods and then proceed with treatment.

Pain Management

Dental pain can be contolled with analgesics and especially acetaminophen, codeine and cox-2 inhibitors must be advised carefully after a consulatnt of physician. During invasive procedures local anaesthic agent does not cause severe blood loss as it is combined with vasoconstrictors[11].Nerve block techniques is contraindicated in coagulopathies patient so prophylaxis is provided. Patient can be treated with general anaesthesia in case of factor

replacement defects. Infiltrations, intra ligamentary, intra osseous or intra pulpal injections are still safer[18].

Surgical Procedures

Extraction of tooth or other minor surgical procedures causes severe bleeding. Local hemostatic agents such as pressure packs, sutures, oxygel, surgicel, bone wax and other topical agents which are required during surgical treatments[23]. Patients undergoing warfarin therapy must be measured by INR [international normalised ratio] , if its greater than 3 treatment can be proceeded. Heparin taking patients can be treated in between dialysis as its short span is for 5 hours. Upatake of anti platelet drug should be stopped in order to lessen the increased risk of bleeding. Careful pre-operative planning and use of antifibrinolytic agents will reduce post operative complications. Desmopressin and tranexamic acid are primary alternatives[11].

Periodontal Procedures

Healthy periodontal tissue is essential to prevent bleeding and tooth loss. Periodontal problems is more troublesome in patients who has bleeding disorders experiences inflamed and hyperemic gingival tissues will be a source for the increased prospect of bleeding[10]. People will denied normal oral hygiene prophylaxis such as brushing of teeth, flossing etc. Patient undergoing factor replacement will require nerve blocks and periodontal packing , mouthgards will protect the surgical site from excessive bleeding. To achieve hemostatis post operative antifibrinolytic mouth washes can be used. On combination of 30% tricholor acetic acid with tranexamic acid was used to delay the bleeding in moderate hemophilia.Extremely inflamed and bloated gingival tissues are treated priorly with chlorhexidine oral mouthwashes or by gross debridement with a cavitron or hand instruments to allow gingival shrinkage prior to deep scaling[24].

Restorative Procedures

Usage of rubber dam, saliva ejectors and high speed suction can cause bleeding in ginvival tissue or oral mucosae so it must be handled carefully. Endodontic therapy might require factor replacement therapy. Restorative procedure can be undergone routinely by providing care,taken to protect the mucosa. Chances of severe bleeding is caused due to use of matrix bands or wooden wedges its controlled by application of topical agents[11].

Prosthodontic Therapy

Patient with bleeding disorders can usedentures as long as they are comfortable. Partial dentures isadvised so that the periodontal health of remaining natural teeth is maintained. Replacement of teeth does not involve much bleeding, oral tissues should be handled carefully on prosthesis fabrication and trial stages. Trauma should be minimal to soft tissues on post insertion process[17].

Orthodontic Treatment

Orthodontic therapy can take place without much bleeding complications. Care should be taken on using appliances which do not impinge soft tissue and provide atraumatic oral hygiene[11].

Oral Infections

Oral infections usually caused by streptococcus viridans, anaerobic gram positive cocci and anaerobic gram negative rods. Antibiotic regimes will control all these groups. Pencillin is first line drug regime to control oral infections. Metranidazole is combined with pencillin to treat aerobic and anaerobic bacteria present in oral cavity[11].

Conclusion

During anticoagulant medications, patient should be reviewed during their dental procedure periodically inorder to minimize the risk of thromboembolic complications. Dentist faces increasing numberof problem related to inherited,acquired and drug related associated with abnormal hemaostatic function. So we must maintain clear and open communication with patient and hematologist to ensure the contemporary information on the severity and control of the patients condition on and after dental procedures.

Corresponding Author

L. Leelavathi,

Senior Lecturer, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Mobile Number: +91 9443702882

Email id: karleela81@gmail.com

Reference

1. AbhirupGoswami, Amitava Bora, Gautam Kumar Kundu,Samiran Ghosh, AnupamGoswam, “Bleeding disorders in dental practice: A diagnostic overview” Journal of the International Clinical Dental Research Organization ,2014 6(2): 143-150.
2. Li le JW, Falace DA, Miller CS, Rhodus NL, editors. Dental Management of the Medically Compromised Patient. 8th ed.; 2012.

3. Morgan GE Jr, Mikhail MS, Murray MJ, editors: Clinical Anaesthesiology. 5th ed. Lange; 2013.
4. Mohamed Hani Nouri Dalati, Zaki Kudsi, LaraTaher Koussayer, Mohamad Fadi Dalati and Mohamad Fouad Mawla, “Bleeding disorders seen in the dental practice”dent update 2012;39:266-270.
5. Philip Vassilopoulos &Kent Palcanis, “Bleeding disorders and periodontology’’periodontoly 2000,2007:44;211-223.
6. Sara Israels, Nora Schwetz,RN, Ron Boyar, Archie Mcnicol, “Bleeding disorders:characterization,dental considerations and management” .JCDA2006;72(9):827
7. Zaki Kudasi, Mohamed Hani Nouri Dalati, Lara Taher Koussayer, Mohamad Fadi Dalati and Mohamad Fouad Mawla, “Management of bleeding disorders in the dental practice:managing patients on anticoagulants”dent update2012;39(5):358-363.
8. Bolton-Maggs PH, Pasi KJ. Haemophilias A and B. Lancet 2003; 361(9371):1801–9.
9. Hassan Mansouritorghabeh“ Clinical and Laboratory Approaches to Hemophilia A” IJMS 2015;40(3).
10. Patton LL. Bleeding and clotting disorders. In: Burket’s oral medicine: diagnosis and treatment. 10th ed. Hamilton (ON): BC Decker; 2003; 454–77.
11. Flora Peyvandi, “Rare coagulation disorders: an emerging issue”BloodTransfusnov 2007; 5(4): 185-186.
12. Anurag Gupta, Joel B.Epstein,RobertJ.Cabay,”Bleeding disorders of importance in dental care and related patient management” ,JCDA 2007;73(1);77-83.
13. F. Peyvandi, R. J. Kaufman, U. Seligsohn “Rare bleeding disorders” Haemophilia (2006);12 (3): 137–142.
14. Drachman JG. Inherited thrombocytopenia: when a low platelet count does not mean ITP. Blood 2004; 103(2):390–8.
15. Geddis AE, Kaushansky K. Inherited thrombocytopenias: toward a mo- lecular understanding of disorders of platelet production. Curr Opin Pediatr 2004; 16(1):15–22.
16. Flint SR, Keith O, Scully C. Hereditary hemorrhagic telangiectasia: family study and review. Oral Surgery Oral Med Oral Pathol 1988; 66:440–4.

17. M.Stubbs, J Lloyd, “A protocol for the dental management of von willebrand’s disease,haemophilia A and haemophiliaB”, Australian dental journal2001;46(1):37-40
18. Lauren L. Patton, “Bleeding and clotting disorders”principles of medicine ;454-477.
19. Rackoz M, Mazar A, Varon D, Spierer S, Blinder D, Martinowitz U. Dental extractions in patients with bleeding disorders. The use of fibrin glue. Oral Surg Oral Med Oral Pathol 1993; 75(3):280–2.
20. Webster WP, Courtney RM. Diagnosis and treatment of peri- odontal disease in the hemophiliac. In: Proceedings, Dental Hemophilia Institute, January, 1968. The National Hemophilia Foundation, U.S.A.

Bombay Blood Group

Bharadwaj.B [1] , Gowri Sethu [2] , Balakrishna R.N [3]
Graduate Student [1],Professor [2], Department of Physiology
Senior Lecturer [3], Department of Oral and Maxillofacial Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Among the available blood group system, ABO system was first to be identified and Rh blood group system was the fourth one. Both are important for safe blood transfusion. A separate group called the Bombay blood group was categorised as the O group because it would not show any reaction to anti-A and anti-B antibodies similar to the O group. When a cross matching with O group is done, then it would show cross reactivity or incompatibility. Therefore, reverse grouping or serum grouping has to be performed to detect this group. Incompatibility in the context of blood groups is due to the binding of plasma antibody with red cell antigen, thereby causing a reaction. In laboratory tests this reaction is most commonly visualised by agglutination of the red cells. Therefore, proper blood matching should be done to avoid various complications.This article explains the importance of blood matching, the character of Bombay blood group and its complications as well.

Key Words: Bombay blood group, Cross matching, Serum grouping, Reverse grouping, Coagulation.

Introduction

Blood can save billions of lives. The need for blood and blood products in a country depends on the population, health care structure, prevalence of diseased conditions requiring regular transfusions, such as haemophilia and thalassaemia.The availability of surgical centresthat uses modern sophisticated techniques creates awareness amongst clinicians regarding judicious use of blood[1].The main objective of the blood bank is safe transfusion of blood from the donor to recipient without harming both. Landsteiner discovered ABO boood group system in 1901 and Rh system in 1940after which blood transfusion became much safer. Determining blood grouping and cross matching is an important prerequisite for blood transfusion. Nearly, 400 red cell antigens have been discovered till now. Among the available blood group system, the ABO system was identified first and Rh blood group system was the fourth one. Both are considered to be important for safe blood transfusion [2]. If proper blood grouping or testing practices are not followed, it can lead to people with Bombay blood group not being detected. This group would be categorised as the O group because it would not show any reaction to anti-A and anti-B antibodies similar to the O group. When a cross matching with O group is done, then it would show cross reactivity or incompatibility. Thus, reverse grouping or serum grouping has to be performed to identify this group.

There are four main blood groups identified. They are A, B, AB, and O [3]. If an individual has type “A” blood, this refers to them having an “A” antigen on the red blood cell. Subsequently, the patient with this type will have antibodies to type “B”. Those with type “O” blood type have no antigens present, but have antibodies to both “A” and “B” antigens. This is why type “O” blood is referred to as the universal donor. Conversely, those with AB blood types have no antibodies and therefore are considered as universal recipients of blood.

Bombay phenotype was first discovered in Bombay, India in 1952. In this blood group, no “A” or “B” antigens are detected on red blood cells or in other secretions and hence it is similar to “O” blood group. In Bombay phenotype, there is a void of A antigen, B Antigen as well as H antigen [4]. It is found that A and B blood groups are formed from the precursor carbohydrate H antigen. All blood cells, except for Bombay typeexpresses the H antigen. People having this blood type are mostly confined to Southeast Asia. About $1/10^{th}$of the population in that region have Bombay blood group. Mostly this Bombay phenotype is inherited by the offsprings from their parents. Caucasian incidence is 1/250, 000 [5].

When Bombay blood group is misdiagnosed, fatal hemolytic transfusion reactions occur. For this reason, and because of its rarity, inaccurate interpretation has led to these adverse effects[6].

In 1952 that Doctors Y.M. Bhende, H.M. Bhatia, C.K. Deshpande in Seth Gordhandas Sunderdas Medical College, Mumbai published an article in The Lancet (May 3, 1952) about two patients (X, a railway worker and Y, a stab wound victim) who required blood transfusion. No blood groups worked for them. The moment their blood samples were mixed with any other blood group types, the blood showed coagulation or clumping. Around 160 donors were tried for blood group matching and finally found one from Mr. Z, a resident of Bombay, suited the type of both patient X and Y. Thus, this donor blood group was then named as the 'Bombay Blood Type also called as hh group.

In 1955 Levine and associates reportedthe genetic suppression of blood group B in an American family in which 3 members were of the Bombay phenotype Oh- This convenient symbol was designated by Levine for persons with "Bombay" blood. In addition to the genetic suppression of blood group B, there was also evidence of suppression of the secretor gene »Se and the H antigen. In order to explain the phenomenon of the "Bombay" blood, 2 hypothetical genes were invoked, X and x. The former is very common and the latter very rare, but gene x in the homozygous state (xx) is responsible for the suppression of blood group substance B, the secretor gene Se and H antigen[6]. The characteristic features of Bombay blood group are absence of H, A and B antigens, no agglutination with anti-A, anti-B, or anti-H lectin, presence of anti-A, anti-B, anti-AB and potent wide thermal range anti-H in the serum, A, B, H non-secretor (no A, B, or H substances present in saliva) absence of H enzyme in serum and H antigen on red cell, presence of A or B enzymes in serum and red cell. Thus, the phenotype of Bombay blood group is considered to be recessive and the red cells of the Bombay group are compatible only withthe serum from another Bombay individual. This article aims at emphasizing the importance to detect the Bombay blood group and its need during emergencies.

Incompatibility

Incompatibility of blood groups is due to the binding of plasma antibody with red blood cell antigen and thereby resulting in antigen-antibody reaction. In laboratory tests, this clumping reaction is most commonly seen by agglutination of the red blood cells. In the body, an antigen-antibody reaction occurs as a result of errors in blood transfusion or pregnancy. These errors ultimately result inaccelerated destruction of red blood cells. It is therefore important to detect incompatibility between the plasma of a patient and the red blood cells of a potential blood donor before transfusion, to avoid a transfusion reaction. Incompatibility occurs in pregnancy when the mother is Rh negative and the foetusbeing Rh positive.

Antigen- antibody reaction occurs as the red blood cells crosses the placenta. This reaction occurs when the foetus has inherited a bloodgroupantigenfromthefatherwhichis ‘foreign’ to the mother. ABO system has the most important blood groups for blood transfusion. In pregnancy the D antigen of the Rh system from the foetus causes the reaction, ultimately resulting in erythroblastosis fetalis.

Antibodies are naturally produced in individuals who have never been transfused with red blood cells carrying the particular antigen or been pregnant with a foetus carrying the particular antigen. On studying about the presence of such antibodies, there can be some cells which may be capable of making specific antibody in the absence of antigenic stimulus or those antibodies, such as production of anti-A and anti-B as an immune response to substances in the environment which are antigenically similar to human blood group substances. For instance, the glycoproteins present on the surface of bacteria present in the gut are antigenically similar to the A and B antigens. Immune antibodies are formed and produced in response to an antigenic stimulus by a foreign antigen which may be due to blood transfusion or pregnancy. Any blood group can cause immune antibody production but out of all the blood groups the Rh system are the most common due to the presence of anti-D. Antibodies that are capable of causing an antigen-antibody reaction to blood transfusion or those which are harmful to a foetus are said to be clinically significant.

Blood group antigens are inherited characters by offsprings from their parents. These are detected by specific antibodies and may be protein or carbohydrate in nature. Though some are present on other cells and tissues of the body, the blood groups that will be referred to be those intrinsic to the surface of red cells. The genetic mechanism involved in the production of protein and carbohydrate antigens is completely different. Protein antigens (i.e. Rh) are direct products of their appropriate gene. However, the genes controlling the attachment of an immunodominant sugar to a cell membrane component encode transferase enzymes. Thus, carbohydrate antigens (i.e. ABO) are indirect products of the defining gene.

Laboratory Detection of Antigen-Antibody Reaction

Blood group antibodies are usually identified by a number of methods, known as serological techniques. Most of the methods utilise plasma or serum from the patient. Out of all the most commonly used is indicator of antigen–antibody interaction in blood grouping which results in agglutination, although haemolysis also indicates that antigen–antibody interaction has taken place.The agglutination reaction occurs when the repulsive forces that normally keep red blood cells apart gets overcome and also the multivalent IgM and bivalent IgG molecules

crosslink the red blood cells. The structure of the IgM pentamer which has 10 binding sites allows crosslinking more readily than the IgG monomer. IgM blood group antibodies are hence capable of acting as ‘direct’ agglutinins. Thus, if serum containing antibody is mixed with red blood cells possessing the specific antigen the cells will clump together ‘directly’, without an addition of anything else. This occurs because the pentameric structure of IgM ensures that the antibody molecules are close enough to link with antigens on two red blood cells and bind them together.Except IgG anti-A and anti-B, most IgG antibodies do not act as direct agglutinins. This is because there are a number of A/B antigen sites on red blood cells, which is about 100 times greater than D antigen sites. Indirect antiglobulin technique (IAGT) and potentiators like proteolytic enzymes can be used for agglutination ofIgG-sensitized cells.The action of proteolytic enzymes like papin on red blood cells may trigger agglutination in two different ways. One is it reduces the surface charge and allows red blood cells to come closer together and another by removing the structures which sterically interfere with the access of antibody molecules. However, we should realise that some blood group antigens are destroyed by papain and thus it cannot be used for the detection of all blood group antibodies.The antiglobulin test (AGT) was actually developed in 1945 and it still remains the most important and gold standard test for detecting clinically significant blood group antibodies. This test was named after its inventor and the test is called as Coombs test. The AGT test can be used as an indirect test (IAGT) to detect the presence of antibodies in the patient plasma or as a direct test (DAGT) to detect antibodies bound to red blood cells in the body. This test was very much useful in detection of red blood cells of babies with haemolytic disease of the newborn or patients with certain types of autoimmune haemolytic anaemia. In the AGT, agglutination is visualized by the addition of antihuman globulin (AHG) to the red blood cells which have sensitised antibody on their surface. It is usually washed in saline to remove residual unbound plasma proteins. In AGT unbound plasma proteins will bind with AHG and inactivate the reagent and hence washing with saline is an important procedure. AHGreagentsusually contain anticomplement (C3) as well as anti-IgG and also contain antibodies to human immunoglobulins. Methodology for performing the AGT has evolved since the usage of opaque glass tile. The tile is now replaced by the test tube, which today has largely been used bythe test name as ‘geltest’or‘column agglutination’ technology. In this the plasma proteins do not come into direct contact with the AHG, thereby neglecting the need for the washing procedure.

Screening of H-Deficient Phenotype

The presence of anti-H antibodies in sera was the first criterium used to define the Bombayphenotype.TheabsenceofH antigenonredcelsfromindividualswithcirculating anti-H was confirmed by hem-agglutination using the lectins Ulex europaeus and Lotus tetragonolobus and human anti-H sera from known Bombay subjects. With this protocol, the classical Bombay non-secretors were well detected. However, those with H-deficient erythrocytesandnormalABH secretionintheirsalivawerenotdetectedbecausethey generally have very low or undetectable titers of anti-H. This protocol is justified, in routine large-scale typing, for clinical reasons because the classical Bombay non-secretors with high titers of anti-H antibodies are susceptible to transfusion accidents if transfused with non-Bombay blood, while H-deficient secretors have a much lower risk of transfusion accidents. Although H-deficient secretors are interesting from a scientific point of view, they were unfortunately missed in our study[8, 9].

Genetics of Bombay Phenotype

Ceppellini suggested that this blood group results from the presence of homozygous recessive gene which inhibits the formation of A,B and H antigen. Subsequently, Watkin and Morgan postulated that the H antigen might be the product of the gene independent of A-B-O system and that the Bombay phenotype is the rare homozygous hh. Levine demonstrated the effective suppression of expression B and secretor gene by the Bombay phenotype extracted from a family of Italian-American. This same family also showed that the H-h locus was not linked to the A-B-O locus. A total of thirteen families with individuals of the Bombay phenotype have been reported from India. These have occurred in multiple castes and in both Hindus and muslims. Inaddition, Italian-American, Irish, French-American, English-American and German family studies have been reported. Although the frequency of the h gene is quite low, it appears to be present in widely separated population groups.

The results of family studies, serologic and biochemical investigations have shown that the A,B,H and Lewis antigens are the products of sequential action of transferases that modify a common precursor substance. The products of A and B genes are glycosyl transferases that add either N-acetylene-galactosamine or galactose to the terminal galactose residue of the H-substance. A fucosyl transferase is the product of H gene and adds a fucose residue[10].

In Bombay phenotype A, B, and H antigens are absent on its red blood cells and the serum of these individuals have anti-A and anti-B.But the serum that agglutinates red blood cells of

‘O’ group individuals, occurs due to presence of strongly reactive anti-H antibodies. It was identified that the h allele is a result of mutation of the H gene (FUTI) that would express the H antigen on the red blood cell of ABO blood groups. Bombay phenotype is homozygous (hh) for the 725G mutation in the FUT1 coding region; the consequence of this mutation is the production of an inactivated enzyme, the enzyme needed in the production of the H antigen. The H substance is bio-chemically produced by the binding of Fucose to the surface glycoproteins. This process iscatalysed by fucosyltransferase. When N-acetyl-galactosamine binds to the H substance, Blood group A gets formed. Blood group B is formed when galactose binds to the H substance. If neither occurs, that is, if the substance does not bind to H, then resultant will be ‘O’ blood group. Individuals that fail to express H transferase (FUT1) lack the H antigen, which is the foundation of Bombay phenotype. Few individuals cannot synthesize A or B antigenic structures as they lack the expression. It is also found that ABH antigens are absent from both their erythrocytes and secretions.

The practice of tribal and territorial endogamy is small population leads to smaller gene pools. Consequently, increased expression of homozygous genetic characters like Bombay phenotype occurs. Many studies show that in India consanguineous marriage is commonly practiced.During cell grouping or forward grouping, Bombay blood group would be categorized as O group because it would not show any reaction to anti-A and anti-B antibodies just like a normal O group would do. Cross reactivity or incompatibility was seen when cross matching with different blood bags of O group was done. Hence a thorough evaluation should be done for identification. Therefore, for detection of Bombay blood group, reverse grouping or serum grouping has to be performed. Thus, this method which is serum grouping or reverse grouping has to be performed in every transfusion medicine department, blood bank or blood donor centres so as to reduce the risk of fatal hemolytic transfusion reactions.The concern with Bombay blood group is that the individuals having this group can only receive either autologous blood donation or blood from an individual of Bombay phenotype [11]. In case of emergency situation, it is important to detect Bombay blood group as it may cause lethal effects on the recipient. Hence individuals identified with Bombay blood group should make all their family members and relatives tested for the blood group. They should also register themselves with leading blood banks so that they can be helpful in case of emergencies [12]. In other suggestions, cryopreservation facilities for rare donor units have been recommended.In the event that the blood banks can borrow or trade uncommon blood units in times of require, a part of issues related to uncommon blood bunches just like the Bombay blood group can be unraveled. Typically, as it were conceivable in case each

blood bank features a huge number of committed customary deliberate givers [13]. In the absence of blood donor registry, transfusion management of patients needing immediate surgery can be challenging. Acute normovolemic hemodilution (ANH) is a blood conservation technique that entails the removal of blood from a patient shortly after induction of anesthesia, with maintenance of normovolemia using crystalloid and/or colloid replacement. The blood is infused into the patient during or shortly after the surgical procedure [14]. ANH is available, cost effective for suitable patients and reduces blood viscosity [15]. Newer research with stem cells has provided an opportunity to produce a universal bloodgroup donor, in vitro, thus enabling cellular replacement therapies, once the safety issue is resolved [16].

Prevalence of Bombay Blood Phenotype

Bombay blood group was first reported in 1952. Bombay phenotype is seldom, since it is found to occur in about 1 in 10,000 individuals in India and 1 in 100,000 individuals in Europe.Regarding the distribution and spread of the Bombay phenotype in different states of India, it is found that this type of blood group is more common in the states of Western and Southern parts of India as compared to other states. Most cases belonged to Maharashtra from the 179 cases recorded at that time. A slightly higher frequency of the Bombay phenotype was also found in the neighboring state of Karnataka (15 cases), Andhra Pradesh (8 cases), Goa (6 cases), Gujarat (5 cases), Uttar Pradesh (5 cases), and so on in the decreasing order[17]. A person with this rare group should always be cautious and alert. People with this blood group can donate blood to any ABO group, but as this blood group is rare one shouldn’t be advised to donate to any other groups. And they should maintain healthy body to avoid Health problems.

Fate of Bombay Blood Group

In Bombay Blood group people, without fucose, neutrophils lack sialyl Lex and thus cannot roll and ingest bacteria. Thus, these patients can have high white blood cell count and severe recurrent infections. This condition is called leukocyte adhesion deficiency II or congenital disorder of glycosylation II[18]. Since this blood group is found to be rare, it would be difficult even for the blood banks to acquire these groups in their bank and use them in case of emergencies. Those anticipating the need for blood transfusion (e.g. in scheduled surgery) may bank blood for their own use (i.e. an autologous blood donation), but this option is not available in case of accidental injury.

If patients with anti-H in their circulation receive transfusions of blood that contains the H antigen (e.g., blood group O), they are at risk of suffering an acute hemolytic transfusion reaction.The maternal production of anti-H during pregnancy in an O_h phenotype mother could cause hemolytic disease in a foetus who did not inherit the mother's Bombay phenotype[19,20].

Conclusion

Various ponders have appeared that the antigenic determinant of ABO blood gather framework is closely related in biochemical terms to the antigenic determinants of the Hh, P, Lewis and Ii blood bunch framework. The blood group antigens of each of this system are formed by the addition of specific sugar to the oligosaccharide precursor chain which may be bound through sphingosine to fatty acids (glycolipid) or through serine or threonine to a peptide chain (glycoprotein). The direct gene product of each of these blood group systems are the glycosyltransferase enzyme which catalyse the addition of specific sugar thus conferring the specified blood group activity to the glycolipid or glycoprotein molecule.When considering such patients for transfusion, as it were blood of indistinguishable Bombay sort can be securely transfused.During cell grouping or forward grouping, Bombay blood group would be categorized as O group because it would not show any reaction to anti-A and anti-B antibodies just like a normal O group would do.When a cross matching with distinctive blood sacks of O gather is done, at that point it would appear cross-reactivity or contradiction[8]. Hence a thorough evaluation should be done for identification. Therefore, for detection of Bombay blood group, reverse grouping or serum grouping has to be performed. Thus, this method which is serum grouping or reverse grouping has to be performed in every transfusion medicine department, blood bank or blood donor centres so as to reduce the risk of fatal hemolytic transfusion reactions.Bombay type is very peculiar because in routine tests for blood grouping it is an O type, but during cross matching procedure donor's O-type cells get clumped with the serum of Bombay type recipient, as Bombay type possesses suppressed H-gene. When considering such patients for transfusion, only blood of identical Bombay type can be safely transfused. Therefore, it becomes a real challenge for the anesthesiologist to manage such type patients without having units of red packed cell. And this article reveals the true importance of blood group matching and serum matching prior to blood transfusion. Even though the probability of occurrence of Bombay blood group is very less, we should never assume things. This will always provide a safe and quality practice.

Corresponding Author

Balakrishna R.N

Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamalle High Road,Chennai 600077,Tamil Nadu, India

E-mail Id – balakrishdr8394@gmail.com

Mobile Number: 8939896729

References

1. Amurdhavani.B.S. Awareness of Blood Donation among Female College Students- A Survey.J. Pharm. Sci. & Res. 2016;8(8): 764-766.
2. Ramamoorthy B, Varghese Ss, Ramesh. A relationship between periodontal disease and ABO blood group phenotypes-a cross sectional retrospective study. International Journal of pharmacy and Pharmaceutical Sciences 2015;7(11):386-388.
3. Thrumiaya T, Gayathri R, Priya VV. Efficacy and accuracy of ABO blood group determination from saliva. Journal of Advanced Pharmacy Education & Research|. 2017;7(2): 41-43.
4. Das S, Kumar HRA. Bombay Blood A Rare Entity. Journal of Clinical Biomedical Science 2011;1(3):122– 5.
5. Dipta TF, Hossain AZ. The Bombay blood group: are we out of risk? Mymensingh Med J 2011;20(3):536–40.
6. Dipta TFA, Hossain KN, Khatun A. Bombay Phenotype: Report of 2 Cases. Journal of Bangladesh College of Physicians and Surgeons 2011;29(4):241– 3.
7. Levine, P.Robinson, E.A.Celano, M.Briggs, Falkinburg.Gene interaction resulting in suppression of blood group substance B .Blood, 1955;10:1100-1108.
8. Exner DV, Dries DL, Domanski MJ, Cohn JN. Lesser response to angiotensin converting–enzyme inhibitor therapy in black as compared with white patients

with left ventricular dysfunction. New England Journal of Medicine.

2001;344(18):1351-7.

9. Solomon J M, Waggoner R, Ley Shon WC. A quantitative immunogenetic study of gene suppression involving A,and H antigens of the erythrocyte without affecting

secreted blood group substances.The ABH phenotypes Amh and Omh. Blood1965;25:470-485.

10. H Rubiskom, Laluhaj, Mergancov, Zakovicovas. New variants in the ABOH blood group system due to interaction of recessive genes controlling the formation of H antigen in erythrocytes: the “Bombay"-like phenotypes OHm, OHmB and OHmAB. Vox Sang1970;19:113-122.
11. Edmond J Yunis, Janet and Robert. Genetic of the Bombay phenotype. Blood journal. 1969;3:1.
12. Dipta TFA, Hossain KN, Khatun A. Bombay Phenotype: Report of 2 Cases. Journal of Bangladesh College of Physicians and Surgeons 2011;29(4):241– 3.
13. Balgir RS. Identification of a rare blood group, “Bombay (Oh) phenotype,” in Bhuyan tribe of Northwestern Orissa, India. Indian J Hum Genet 2007;13(3):109–13.
14. Malhotra S, Dhawan HK, Jain A, Sachdev S, Marwaha N. Acute Hemolytic Transfusion Reaction in a Patient with Bombay Phenotype: Implications for ABO Grouping. Indian J Hematol Blood Transfus 2014;30(1):108–10.
15. Khan M. Bombay Blood Group: A Case Report. The Pacific Journal of Science and Technology 2009;10(1):333–7.
16. O’Donnell JS, McKinnon TA, Crawley JT, Lane DA, Laffan MA. Bombay phenotype is associated with reduced plasma-VWF levels and an increased susceptibility to ADAMTS13 proteolysis. Blood 2005;106(6):1988–91.
17. Shrivastava M, Navaid S, Peethambarakshan A, Agrawal K, Khan A. Detection of rare blood group, Bombay (Oh) phenotype patients and management by acute normovolemic hemodilution. Asian J Transfus Sci 2015;9(1):74–7.
18. Etzioni A, Tonetti M. Leukocyte adhesion deficiency II-from A to almost Z. Immunol Rev. 2000;178:138–47.
19. Bhende YM, Deshpande CK, Bhatia HM, Sanger R, Race RR, Morgan WT. A “new” blood group character related to the ABO system. Lancet. 1952;1:903–4.
20. Bhattacharya S, Makar Y, Laycock RA, Gooch A, Poole J, Hadley A. Outcome of consecutive pregnancies in a patient with Bombay (Oh) blood group. Transfus Med. 2002;12:379–82.

Brachial Plexus Injury

Anitha.D.R [1], Saravana Kumar [2], Sri Sakthi.D [3]
Graduate Student [1],Lecturer [2],Department Of Anatomy,
Reader [3],Department Of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

To Review The Function, Anatomy, Causes, Classification, Diagnosis And Treatment Of Brachial Plexus Injury. The Brachial Plexus Is A Network Of Nerve Running From The Spine .It Is Formed By The Ventral Rami Of The Lower Four Cervical And First Thoracic Nerve Roots (C5-C8, T1). It Proceeds Through The Neck, The Axilla (Armpit Region), And Into The Arm. It Is A Network Of Nerves Passing Through The Cervico-Axillary Canal To Reach Axilla And Innervates Brachium (Upper Arm), Ante Brachium (Fore Arm) And Hand.The Brachial Plexus Is Responsible For Cutaneous And Muscular Innervation Of The Entire Upper Limb, With Two Exceptions: The Trapezius Muscle Innervated By The Spinal Accessory Nerve And An Area Of Skin Near The Axilla Innervated By The Intercostobrachial Nerve .Lesions Can Lead To Severe Functional Impairment. Brachial Plexus Injuries Affects The Nerves Which Send Signals From The Spine To Your Shoulder.Brachial Plexus Injury Is An Umbrella Term For A Variety Of Conditions That May Impair Function Of The Brachial Plexus Nerve Network. The Majority Of Paediatric And Adult Brachial Plexus Injuries Are Caused By Trauma. A Brachial Plexus Injury Occurring During Birth Is Called Birth Related Brachial Plexus Palsy Or Obstetric Brachial Plexus Palsy.

Key Words: Brachial Plexus , Ventral Rami , Obstetric Brachial Plexus Palsy.

Introduction

The Brachial Plexus Is The System Of Nerves That Sends Signals From Spine To Shoulder, Arm And Hand. Brachial Plexus Damage Happens When These Nerves Are Extended, Compacted, Or In The Most Genuine Cases, Tore Separated Or Torn Far From The Spinal String. It Is The System Of Nerves Which Goes Through The Cervical Spine, Neck, Axilla And Afterward Into Arm Or It Is A System Of Nerves Going Through The Cervico-Axillary Waterway To Achieve Axilla And Innervates Brachium (Upper Arm), Bet Brachium (Lower Arm) And Hand.It Is A Physical Nerve Plexus Shaped By Intercommunications Among The Ventral Rami (Roots) Of The Lower 4 Cervical Nerves (C5-C8) And The Main Thoracic Nerve (T1).Minor Brachial Plexus Wounds, Known As Stingers Or Burners, Are Regular In Contact Sports, For Example, Football. Children Some Of The Time Maintain Brachial Plexus Wounds Amid Birth[1]. Different Conditions, For Example, Aggravation Or Tumors, May Influence The Brachial Plexus.The Most Serious Brachial Plexus Wounds As A Rule Result From Auto Or Bike Mishaps. Serious Brachial Plexus Wounds Can Leave Arm Deadened, With Lost Capacity And Sensation. Surgical Methods, For Example, Nerve Unites, Nerve Exchanges Or Muscle Exchanges Can Help Reestablish Work.

Injuries To The Brachial Plexus Are Normal And Are The Most Serious Of All SoresOf Fringe Nerves. Brachial Plexus Wounds Or Injuries Can Happen Because Of Shoulder Injury, Tumors, Or Irritation. The Uncommon Parsonage-Turner Disorder Causes Brachial Plexus Aggravation Without Clear Damage However With By The By Impairing Side Effects. Brachial Plexus Sores Can Be Delegated Either Awful Or Obstetric. Obstetric Wounds May Happen From Mechanical Damage Including Shoulder Dystocia Amid Troublesome Labor. Horrendous Damage May Emerge From A Few Causes. "The Brachial Plexus Might Be Harmed By Tumbles From A Stature On To The Side Of The Head And Shoulder, Whereby The Nerves Of The Plexus Are Brutally Extended .The Brachial Plexus May Likewise Be Harmed By Coordinate Viciousness Or Discharge Wounds, By Savage Footing On The Arm, Or By Endeavors At Diminishing A Disengagement Of The Shoulder Joint"[2,3].

Function

The Brachial Plexus Is In Charge Of Cutaneous And Solid Innervation Of The Whole Upper Appendage, With Two Special Cases: The Trapezius Muscle Innervated By The Spinal Embellishment Nerve (Cn Xi) And A Territory Of Skin Close To The Axilla Innervated By The Intercostobrachial Nerve.

Clinical Anatomy

The Plexus Comprises Of Roots, Trunks, Divisions, Cords And Branches[4].

1. **Roots:** These Are Consititued By The Front Essential Rami Of Spinal Nerves C5,6,7,8 And T1 With Commitments From The Foremost Essential Rami Of C4 And T2. The Root Of The Plexus May Move One Fragment Either Upward Or Descending Bringing About A Pre Fixed Pleus Or Post Fixed Plexus Separately. Ina Prefixed Plexus, The Commitment By C4 Is Vast And In That From T2 Is Regularly Truant. In A Post Settled Plexus, The Commitment By T1 Is Substantial, T2 Is Constantly Present, C4 Is Truant, And C5 Is Decreased In Estimate. The Roots Join To Shape Trunks As Takes After:

2. Upper Trunk Is Framed By C5 And C6

Middle Trunk Is Framed By C7

Lower Trunk Is Framed By C8 And T1

3. **Divisions Of The Trunks** Each Trunk Separates Into Ventral And Dorsal Divisions (Which At Last Supply The Front And Back Parts Of The Appendage). These Divisions Join To Shape Strings.

4. **Cords**: It Frames 3 Cords

1. The Posterior Cord Is Framed From The Three Back Divisions Of The Trunks (C5-C8,T1)

2. The Lateral Cord Is The Foremost Divisions From The Upper And Center Trunks (C5-C7)

3. The Medial Cord Is Just A Continuation Of The Foremost Division Of The Lower Trunk (C8,T1).

The Different Characterisations Of Brachial Plexus Damage Are As Follows[5]:

1. Leffert Arrangement Of Brachial Plexus Damage

2. Millesi Order Of Brachial Plexus Damage

3. Classification On Anatomical Area Of Damage.

Leffert Grouping Of Brachial Plexus Damage: It Depends On Etiology And Level Of Damage And Is As Per The Following

•I Open (Ordinarily From Cutting)

•Ii Closed (Ordinarily From Bike Mishap)

•Iia Supraclavicular

•Preganglionic

•Avulsion Of Nerve Roots, Ordinarily From Rapid Wounds With Different Wounds And Loc;

•No Proximal Stump, No Neuroma Arrangement (NegTinel's)

•Pseudomeningocele, Denervation Of Neck Muscles Are Normal

•Horner’s Sign (Ptosis, Miosis, Anhydrosis)

•Postgangionic;

•Roots Stay In Place;

•Usually From Footing Wounds;

•There Are Proximal Stump And Neuroma Arrangement (PosTinel’s)

•Deep Dorsal Neck Muscles Are In Place, AndPseudomeningoceles Won’t Create;

•Infraclavicular Lesion:

•Usually Includes Branches From The Trunks (Supraclavicular);

•Function Is Influenced In View Of Trunk Included;

• Iii Radiation Actuated

•Iv Obstetric

•Iva Erb’s (Upper Root) - Server's Tip Hand;

•IvbKlumpke (Bring Down Root)

Millesi Characterization Of Brachial Plexus Damage: It Is Primarily Isolated Into 4

I: Supraganglionic/Preganglionic.

Ii: Infraganglionic/Postganglionic

Iii: Trunk.

Iv: Cord

Characterization On Anatomical Area Of Damage:

•Upper Plexus Paralysis (Erb’s Paralysis In The Obpi Cases) Includes C5-C6+/ - C7roots.

•Lower Plexus Paralysis (Klumpke’s Paralysis) Includes C8-T1 Roots (And Here And There Likewise C7)

•Total Plexus Injuries Include All Nerve Roots C5-T1

•Some Creators Have Incorporated A Fourth Kind, A Moderate Sort That Principally Includes The C7 Root.

Upper Trunk Plexus Damage

Upper-Trunk Paralysis Happens When The Edge Between The Shoulder And The Neck Persuasively Extends, For Example, When A Fall Powers The Shoulder Down And The Go To The Contrary Side.

Patients With Upper-Trunk Paralyses Can't Utilize The Shoulder To Raise The Arm Far From The Body, Have Shortcoming In The Arm, And Might Be Not Able Twist The Arm At The Elbow[6,7]. There Might Be Loss Of Sensation In The Shoulder, Outside Of The Arm, And The Thumb.

Extreme Upper-Trunk Damage May Deaden The Shoulder Muscles (Deltoid Muscle And Rotator Sleeve), And In Addition The Muscle In The Upper Arm (Biceps.)

Lower Trunk Plexus Damage

Lower-Trunk Paralysis Happens When The Edge Between The Arm And The Chest Divider Persuasively Enlarges. This May Harm The Lower Nerves And The Lower Trunks.

Patients With A Lower-Trunk Paralysis Will Commonly Keep Up Shoulder And Elbow Quality, Yet Will Lose Hand Work. After Some Time, This Will Make The Fingers Contract Into A Paw Position, And The Patient Won't Have The Capacity To Perform Fine Engine Assignments. Patients Likewise Regularly Have Hand Deadness In At Any Rate The Ring Finger And Little Finger[7].

Pan Plexus Damage

Pan Plexus Paralysis May Happen If The Power Of The Damage Is Outrageous. In Dish Plexus Paralysis, All Levels Of The Nerves And Trunk Are Harmed. This Outcomes In Entire Loss Of Motion Of The Arm And Hand, Which Is Regularly Alluded To As "Thrash Limb."[7].

Causes

Damage To The Upper Nerves That Make Up The Brachial Plexus Has A Tendency To Happen When Bear Is Constrained Down While Your Neck Extends Way Up Yonder, Into The Clouds From The Harmed Bear. The Lower Nerves Will Probably Be Harmed

When Your Arm Is Constrained Over Your Head[8]. These Wounds Can Happen In A Few Ways, Including:

•Contact Sports. Numerous Football Players Encounter Burners Or Stingers, Which Can Happen When The Nerves In The Brachial Plexus Get Extended Past Their Point Of Confinement Amid Crashes With Different Players.

•Difficult Births. Babies Can Maintain Brachial Plexus Wounds When There Are Issues Amid Birth, For Example, A Breech Introduction Or Delayed Work. On The Off Chance That A Newborn Child's Shoulders Get Wedged Inside The Birth Trench, There Is An Expanded Danger Of A Brachial Plexus Paralysis. Regularly, The Upper Nerves Are Harmed, A Condition Called Erb's Paralysis. Add Up To Brachial Plexus Birth Paralysis Happens When Both The Upper And Lower Nerves Are Harmed.

•Trauma. A Few Kinds Of Injury — Including Engine Vehicle Mischances, Bike Mishaps, Falls Or Slug Wounds — Can Bring About Brachial Plexus Wounds.

•Inflammation. Irritation May Make Harm The Brachial Plexus. An Uncommon Condition Known As Parsonage-Turner Disorder (Brachial Plexitis) Causes Brachial Plexus Aggravation With No Injury And Results In Loss Of Motion Of A Few Muscles Of The Arm.

•Tumours. Noncancerous (Kindhearted) Or Destructive Tumors Can Develop In The Brachial Plexus Or Put Weight On The Brachial Plexus Or Spread To The Nerves, Making Harm The Brachial Plexus.

•Radiation Treatment. Radiation Treatment May Make Harm The Brachial Plexus.

Signs And Symptoms

Signs And Manifestations May Incorporate A Limp Or Deadened Arm, Absence Of Muscle Control In The Arm, Hand, Or Wrist, And Absence Of Feeling Or Sensation In The Arm Or Hand. Signs And Side Effects Of Brachial Plexus Damage Can Change Enormously, Contingent Upon The Seriousness And Area Of The Damage. Normally Just A Single Arm Is Affected[9,10].

Less Extreme Wounds

Minor Harm Regularly Happens Amid Contact Sports, For Example, Football Or Wrestling, When The Brachial Plexus Nerves Get Extended Or Compacted. These Are Called Stingers Or Burners, And Can Create The Accompanying Side Effects:

•A Feeling Like An Electric Stun Or A Torching Sensation Shooting In Arm

•Numbness And Shortcoming In Arm

These Side Effects Normally Last Just A Couple Of Moments Or Minutes, However In A Few People May Wait For Quite A Long Time Or More.

Progressively Serious Wounds

Progressively Extreme Side Effects Result From Wounds That Truly Harm Or Even Tear Or Burst The Nerves. The Most Genuine Brachial Plexus Damage (Separation) Happens When The Nerve Root Is Torn From The Spinal Rope.

Signs And Side Effects Of Increasingly Serious Wounds Can Include:

•Weakness Or Failure To Utilize Certain Muscles Close By, Arm Or Shoulder

•Complete Absence Of Development And Feeling In Arm, Including Shoulder And Hand

•Severe Pain

Albeit A Few Systems Represent Brachial Plexus Wounds, The Most Widely Recognised Is Nerve Pressure Or Extend. Newborn Children, Specifically, May Endure Brachial Plexus Wounds Amid Conveyance And These Present With Run Of The Mill Examples Of Shortcoming, Contingent Upon Which Part Of The Brachial Plexus Is Included. The Most Extreme Type Of Damage Is Nerve Root Separation, Which Brings About Entire Shortcoming In Comparing Muscles. This For The Most Part Goes With High-Speed Impacts That Happens Amid Engine Vehicle Or Bike Mishaps.

Mechanism Of Injury

Genuine Plexus Wounds Are Because Of Footing On The Plexus. These Are The Aftereffect Of Joined Neck Augmentation And In Addition Ipsilateral Side Flexion And Ipsilateral Pivot. The Standard Component Of Horrible Damage Includes A Generally

Rapid Effect. This Component Causes Serious Footing Powers On The Brachial Plexus Particularly In The Upper Trunk And Can Bring About One Of Three Scenarios[11] ;

Neuropraxia

Mellow Footing Which Can Bring About A Neuropraxia Causing The " Burners And Stingers" Surely Understood And Adored In Rugby Circles. As The Name Proposes The Manifestations Are Portrayed By A Copying Or Stinging Agony Joined By Deadness, Paraesthesia And Muscle Shortcoming Normally Influencing The Entire Arm From The Upper Filaments Of The Trapezius To The Fingertips. Rehashed Neuropraxias At A Similar Level Can Bring About Huge Shortcoming Influencing The Deltoid And Biceps.

Axontmesis

Direct Power And Footing Can Bring About An Axontmesis Where There Is Disturbance Of The Axon And The Myelin Sheath With Protection Of The Sinewy Epineurium.

Neurotmesis

Neurotmesis Isn't Extremely Regular In Rugby Yet Is Seen Frequently In Engine Cycle Hustling And Is Once In A While Connected With The Noteworthy Injury To The Navigating Significant Veins, Bone Structure And Muscles.

Investigation

Clinical Examination Is Important To Recognise Dynamic Wounds To The Head, The Chest And Stomach Area. At The Point When There Is Clinical Doubt Of Harm To The Curve Of The Aorta Arteriography Is Fundamental; It Must Be Recalled That A Blade Or Rocket Does Not Regard Tissue Planes. We Have Seen An Aneurysm Of The Axillary Supply Route After Rocket Damage To The Contrary Arm And An Arteriovenous Fistula In The Back Triangle Of The Neck After A Rocket Entered The Arm And Left Underneath The Spine Of The Scapula.

Angiography, In Any Case, Isn't Generally Vital Before Critical Surgery. Honest Seeping From An Open Injury Or A Growing Swelling With Or Without A Bruit Demonstrates A Blood Vessel Damage. A Boisterous Bruit Proposes An Arteriovenous Fistula. The Clinical Determination Of Break Of The Axillary Vein In A High-Vitality

Shut Infraclavicular Sore Is Normally Direct: The Back Triangle Of The Neck Isn't Swollen, The Subclavian Heartbeat Can Be Felt, There Is An Extending Swelling Beneath The Clavicle And There Is No Brachial Heartbeat. In Such A Case Angiography Would Cause Superfluous Postponement.

Diagnosis Of The Neural Damage.

The Fundamental Component In Analysis Is The Qualification Amongst Pre-And Postganglionic Sores. In Preganglionic Burst, The Afferent Nerve Filaments With Cell Bodies In The Dorsal Root Ganglion Don't Degener-Ate. Just Efferent Strands To Skeletal Or Smooth Muscle Experience Wallerian Degeneration. It Is Presently Of More Than Scholastic Intrigue That A Preganglionic Or Intradural Sore Is Analyzed All The More Precisely As Either A Crack Of The Rootlet Fringe To The Focal Fringe Transmission Zone (Bert-Hold, Carlstedt And Corneliuson 1993) Or A Genuine Separation From The Spinal String, Which Is Damage To The Focal Apprehensive System[12]. Up To This Point, This Refinement Could Be Made Just By Investigation Of The String Or Histological Examination Of The Tips Of Separated Rootlets, Yet It Is Likely That Upgrades In Mri Systems Will Make The Finding Conceivable.

An Exact History And Physical Examination Permit A Neurological Finding As A Rule. The Relative Brutality Of The Damage And Gauge Of The Power Connected To The Appendage Are Especially Significant[13,14,15]. The Trademark Torment From A Preganglionic Damage Begins On The Main Day In Around Half Of Cases; The Patients Depict A Steady Pounding Or Consuming Agony In The Hand With Superimposed 'Lightning Stuns' Shooting Down The Appendage. Direct Scraped Spots From The Neck To The Shoulder, Swelling In The Back Triangle And Vascular Damage All Propose That A Savage Footing Power Has Been Connected.

The Degree Of Nerve Harm Is Effortlessly Learned By The Individuals Who Have Contemplated The Mrc Notice Aids To Examination Of The Fringe Sensory System (1976); All Specialists Treating Wounds Ought To Be Comfortable With This Work[16]. Assurance Of The Level Of The Injury, Regardless Of Whether It Is Intradural Or A Postganglionic Crack, Is More Troublesome, Yet Entire Thoughtful Loss Of Motion Is An Indication Of Break. Pre-Ganglionic Damage Of The Upper Three Foundations Of The Plexus Is Proposed By Loss Of Sensation Over The Clavicle, With Loss Of Motion

Of The IpsilateralHemidiaphragm, Serratus Foremost And Trapezius; A Bernard-Horner Sign Shows Comparative Damage To The Lower Two Roots[17]. An Unequivocally Positive Tinel Sign Proposes A Postganglionic Crack. Percussion Of The Back Triangle Of The Neck Makes The Patient Feel Sticks And Needles Transmitting To The Elbow When The Fifth Cervical Nerve Has Been So Harmed; If These Sensations Reach Out To The Thumb And Forefinger, At That Point The 6th Cervical Nerve Has Been Harmed. Plain Radiographs Of The Neck And The Chest Are Constantly Valuable. Tilting Of The Cervical Spine Far From The Side Of Damage Is Suggestive Of Finish Intradural Damage; Breaks Of The Transverse Procedure Of C7 Or Of The Principal Rib May Show Intradural Damage Of The Lower Two Roots. Variety In The Life Structures Of The Brachial Plexus Is A Noteworthy Trap In Clinical Conclusion. It Isn't Exceptional To Locate The Advanced Extensors Innervated By The Primary Thoracic Nerve; On The Other Hand, Numerous Patients With In Place C8 And T1 Have Loss Of Motion Of All Extensor Muscles. 'Pre' And 'Post' Obsession Of The Plexus Is Once In A While Critical.

Advanced Examinations

Physiological Analysis Depends On Bonney's Finding That Afferent Axons Stay Myelinated On The Off Chance That They Are Still In Progression With The Dorsal Root Ganglion. In The Event That Intradermal Histamine Prompts A Flare In Soporific Skin Then The Spinal Nerve Providing That Skin Has Been Separated From The Spinal String. On The Off Chance That A Tactile Activity Potential Can Be Recorded From The Incitement Of A Sedative Forefinger Then The 6th Cervical Nerve Has Been Harmed Along These Lines. These Examinations Are Valuable Simply After Wallerian Degeneration Has Occurred, No Less Than Three Weeks After Injury[18,19]. Early Myelography, Inside A Couple Of Days Of Damage, Might Be Hard To Decipher When There Has Been A Tear Of The Dura, Yet A Decent Quality Myelogram Is To A Great Degree Valuable After Around Seven Days And Surprisingly Better When Joined With Ct. The Show Of Evoked Possibilities From Scalp Cathodes By The Incitement Of Uncovered Spinal Nerves Prohibits Separation. Kline (1989) Has Demonstrated The Estimation Of Intraoperative Nerve Activity Possibilities In The Analysis Of Sores In Progression, Outstandingly Those Caused By Discharge Injuries[20].

Mri Is Important In Showing Sores Of The Spinal String And Of Damage Inside The Back Triangle. It Can Recognise Denervated, Incompletely Denervated And Innervated

Muscle (Fleckenstein Et Al 1993)[21]. Francel Et Al (1995) Have Stretched Out Its Utilization To The Determination Of Birth Injuries Of The Brachial Plexus[22].

Treatment

Treatment For Brachial Plexus Wounds Incorporates Orthosis/Supporting, Word Related Or Exercise Based Recuperation And, At Times, Surgery. Some Brachial Plexus Wounds May Recuperate Without Treatment. Numerous Newborn Children Enhance Or Recuperate Inside A Half Year, However Those That Don't Have An Extremely Poor Viewpoint And Will Require Assist Surgery To Endeavor To Adjust For The Nerve Shortfalls. The Capacity To Twist The Elbow (Biceps Work) By The Third Month Of Life Is Viewed As A Pointer Of Plausible Recuperation, With Extra Upward Development Of The Wrist, And Also Rectifying Of Thumb And Fingers A Considerably More Grounded Marker Of Superb Unconstrained Change. Delicate Scope Of Movement Practices Performed By Guardians, Joined By Rehashed Examinations By A Doctor, Might Be All That Is Important For Patients With Solid Markers Of Recuperation.

Comprehensively Surgery For These Is Separated In Two General Classes:

•Surgery For Nerve Repair

•Secondary Methodology

At Whatever Point Practical Relying Upon Timing, Surgery For Nerve Repair Outweighs Every Other Methodology Since Time Is Of The Embodiment. When Different Wounds Are Managed Essentially The Patient Ought To Have The Soonest Conceivable Nerve Repair.

Optional Techniques Are Done After Nerve Repairs Or In Late Cases As A Substitute To Reestablish Work. Whichever Way All Patients Can Be Offered Some Treatment At All Stages.

Surgery For Nerve:

Extensively Arranged In To:

•Intra Plexus Repair

•Extra Plexus Repair

•Distal Nerve Exchanges

•Contralateral C7 Exchange.

Frequently A Mix Of These Might Be Advertised.

Intra Plexal Repair

In Instances Of Post Ganglionic Damage Where Contributor Roots Are Accessible, The Root Stumps Are Joined To Distal Targets Which Might Be Trunks, Ropes Or Individual Nerves With The Assistance Of Autologous Nerve Unites. These Might Be Free Joins Or Vascularised Unites. As A Rule Short Joins Improve The Situation Than Long Unites; Notwithstanding, Such A Decision Isn't Generally Accessible.

Extra Plexal Repair

Nerves Not Emerging From The Plexus Are Utilized As Giver Nerves. Great Case As Being, Intercostal Nerves (Icn) To Musculo Cutaneous Nerve (Mcn) For Biceps And Spinal Accessory Nerve (San) To Supra Scapular Nerve (Ssn) For Rotator Sleeve Reinnervation. A Few Creators Utilize Phrenic Nerve. Despite The Fact That They Guarantee That All Parameters Continuously Recoup To Preoperative Status Levels Inside 1 Year; The Loss Of Stomach Work Prevents Numerous Others From Utilizing It. Bhandari Et Al Have Demonstrated That However The Phrenic Exchange Produces The Coveted Engine Result, The Long Haul Follow Up Of The Patients Demonstrated A Relentless Pneumonic Capacity Shortage Even Toward The Finish Of Quite A Long While In Extremely Fit Youthful Adults[23]. Their Information And Conclusions Are Repeated Beneath.

Methodologies For Recreation

Methodologies For Grown-Ups And Kids (Birth Brachial Plexus Injury Bbpi) Vary Extensively. The Creator Has Examined The Bbpi Systems Somewhere Else In Detail. Youngsters Have A Far More Prominent Recovery Limit And Separation To Movement

For The Recovering Axons Is Far Littler. Hence Conceivably Add Up To Recreation Up To And Including The Hand Inborn Muscles Is Possible Even In An Every One Of The 5 Root Damage If Worked In Time[24]. This Is Once In A While Doable In A Grown-Up Add Up To Paralysis.

Conclusion

The Vital Advances Of The Most Recent 30 Years Include: Refinement Of Conclusion; Re-Learning And Re-Use Of The Standards Of The Treatment Of Wounds And Vascular Injuries; Enhanced Strategies For Nerve Repair And Exchange; And, In Particular, An Approach Of Earnest Investigation And Repair. Great Useful Recuperation Can Sensibly Be Normal In Patients With Either Pre-Or Postganglionic Harm To The Fifth, 6th And Seventh Cervical Nerves, And In Place Eighth Cervical And First Thoracic Nerve. On The Off Chance That Specialists Repudiate The Destructive Strategy Of Delaying Most Patients Can Accomplish Rebuilding OfThoracoscapular Control, Glenohumeral Control, Elbow Flexion And Wrist Expansion, With Ordinary Sensation Inside The Middle Domain Of The Hand And The Help Of Torment. Because Of The Expansive Range Of Brachial Plexus Wounds, It Is Hard To Gauge The Rate Of Unconstrained Recuperation. The Potential For Unconstrained Recuperation Relies Upon The Sort And Seriousness Of Damage. In This Way, Visualization Must Be Surveyed For Every Patient Separately In View Of The Sort And Seriousness Of Their Damage, And The Movement Of Any Unconstrained Recuperation That Might Be Occurring.Depending On The Level Of Seriousness, Some Nerve Wounds Can Mend Without Anyone Else. On The Off Chance That This Isn't A Feasible Alternative For A Specific Patient, A Surgical Choice Might Be Prescribed By The Doctor. Physical Restoration Treatment Is Dependably Part Of The Recuperation Procedure For A Brachial Plexus Injury.The Level Of Practical Weakness And Potential For Recuperation Rely Upon The System, Type, Intricacy Of The Brachial Plexus Damage, And Time From Damage. The Most Imperative Choice Your Specialists Will Make Is Deciding Whether And When Surgical Mediation Ought To Happen.

Corresponding Author

D.Sri Sakthi

Reader,Department Of Public Health Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai – 600077,Tamil Nadu, India

Mobile - +91 9962787531

Email: Drsrisakthiphd@Gmail.Com

Reference

1. Thatte Mr, Agashe M, Rathod C, Lad P, Mehta R. An Approach To The Supraclavicular And Infraclavicular Aspects Of The Brachial Plexus. Tech Hand UpExtrem Surg. 2011; 15:188–97.
2. Gu Y, Meng K. Use Of The Phrenic Nerve For Brachial Plexus Reconstruction. Clin Orthop Relat Res. 1996; 323:119–21.
3. Xu Wd, Gu Yd, Lu Jb, Yu C, Zhang Cg, Xu Jg. Pulmonary Function After Complete Unilateral Phrenic Nerve Transection. J Neurosurg. 2005;103:464–7.
4. Erb Wh. UeberEineEigenthumliche Localisation Von LahmungenIn Plexus Brachialis. VerhDtschNatur Med. 1874;2:130.
5. Thorburn W. A Clinical Lecture On Secondary Suture Of The Brachial Plexus. Br Med J. 1900;1:1073–5.
6. Harris W, Low Vw. On The Importance Of Accurate Muscular Analysis In Lesions Of The Brachial Plexus And The Treatment OfErb's Palsy And Infantile Paralysis Of The Upper Extremity By Cross-Union Of Nerve Roots. Br Med J. 1903;2:1035.
7. SeddonHj. The Use OfAutogenous Grafts For The Repair Of Large Gaps In Peripheral Nerves. Br J Surg. 1947;35:151–67.
8. Stevens Jh. Brachial Plexus Paralysis. In: Codman Ea, Editor. The Shoulder. Boston: Privately Published; 1934. Pp. 344–50.
9. Narakas A. Surgical Treatment Of Traction Injuries Of The Brachial Plexus. Clin Orthop. 1978;133:71–90.
10. Narakas A. Brachial Plexus Surgery. Orthop Clin North Am. 1981;12:303–23.

11. Millesi H. Surgical Management Of Brachial Plexus Injuries. J Hand Surg. 1977;2:367–78.
12. Berthold C-H, Carlstedt T, Corneliuson O. The Central-Peripheral Transition One. In: Dyck Pj, Thomas Pk, Eds. Peripheral Neuropathy. 3rd Ed. Philadelphia, Wb Saunders, 1993;1:73-91.
13. Dong Z, GuYd, Zhang Cg, Zhang L. Clinical Use Of Supinator Motor Branch Transfer To The Posterior Interosseous Nerve In C7-T1 Brachial Plexus Palsies. J Neurosurg. 2010;113:113–7.
14. BertelliJa, Ghizoni Mf, Tacca Cp. Transfer Of The Supinator Muscle To The Extensor PollicisBrevis For Thumb Extension Reconstruction In C7-T1 Brachial Plexus Palsy. J Hand Surg Eur. 2010;35:29–31.
15. Doi K, Sakai K, Kuwata N, Ihara K, Kawai S. Double-Muscle Technique For Reconstruction OfPrehension After Complete Avulsion Of Brachial Plexus. J Hand Surg. 1995;20:408–14.
16. Medical Research Council. Aids To Examination Of The Peripheral Nervous System. Memorandum No. 45, London.Hmso, 1976.
17. Carlstedt T, Grane P, HallinRg, Noren G. Return Of Function After Spinal Cord Implantation Of Avulsed Spinal Nerve Roots. Lancet. 1995;346:1323–5.
18. CarlstedtTp, Anand P, Hallin R, MisraPv, Noren G, Seferlis T. Spinal Nerve Root Repair And ReimplantationOfAvuled Ventral Roots Into The Spinal Cord After Brachial Plexus Injury. J Neurosurg. 2000;93:237–42.
19. 19)BertelliJa, Mira Jc. Brachial Plexus Repair By Peripheral Nerve Grafts Directly Into The Spinal Cords In Rats. Behavioral And Anatomical Evidence Of Functional Recovery. J Neurosurg. 1994;81:107–14
20. Kline Dg. Civilian Gunshot Wounds To The Brachial Plexus. J Neurosurg 1989;70:166-74.
21. Fleckenstein J, Watumull D, Conner Ke. Denervated Human Skeletal Muscle Mr Imaging Evaluation. Radiology 1993;187:213-8.
22. Francel Pc, Koby M, Park Ts. Fast-Spin Echo Magnetic Resonance Imaging For Radiological Assessment For Neonatal Brachial Plexus Injury. J Neurosurg 1995;83:461-6.

23. Vignesh, Santhosh Kumar. Management Of Haemorrhage In Oral Surgery.J. Pharm. Sci. & Res.2016:8(7);642-644.
24. Santhosh Kumar. Efficacy OfArticaine Over Lidocaine. J. Pharm. Sci. & Res.2015:7(11);956-959.

Bruxism

Subhasri Raman [1], Madhulaxmi M [2]

Graduate Student [1], Reader [2], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Bruxism is an oral parafunctional habit prevalent in all age groups which is characterized by the involuntary teeth grinding or clenching that may occur at any time of the day. It has a multifactorial aetiology associated with occlusal and psychological factors and habits that can compromise the orthognathic system that may bring negative consequences. Bruxism can be classified as awake or sleep bruxism. Patients with sleep bruxism are more likely to experience jaw pain and limitation of movement, than people who do not experience sleep bruxism. Faulty occlusion is one of the most common causes of bruxism that further leads to temporomandibular joint pain. This review focuses upon various aspects of bruxism and its treatment modalities.

Key Words: *Tooth grinding, Wear facets, Malocclusion, Temporomandibular disorders, Muscle fatigue*

Introduction

Bruxism has been considered a detrimental motor activity known to potentially cause over activity of the stomatognathic structures.Tooth grinding (bruxism) has been of prime importance to dentist as it leads to significant tooth damage, breakage of dental restorations, induction of temporal headache, temporomandibular disorders and orofacial muscle fatigue [1].Activities of the masticatory system can be divided as Functional and Parafunctional. Function includes chewing, speaking and parafunctionalincludes clenching or grinding of the teeth (referred to as bruxism). Parafunctional activity oftenleads muscle hyperactivity. The functional activities are very controlled muscle activities, which allow the masticatory system to perform necessary functions with minimum damage to the structures of this system. These functions are often disturbed by interfering tooth contact leading to inhibition of functional muscle activity. Therefore, functional activities are considered to be directly influenced by the occlusion.

The term parafunction was introduced by Drum [2] to suggest distinction between occlusal stress exerted during mastication and swallowing and occlusal stress which are brought into action outside of the normal function. Parafunctional activities are non functionaloromandibular or lingual activities that includes jaw clenching, bruxism, tooth grinding, tooth tapping, cheek biting lip biting, object biting etc. that can occur alone or in combination and are different from functional activities like chewing, speaking and swallowing.

The term ‘la bruxomanie’ was first introduced by Marie Pietkiewicz in 1907 [3]. It was latter adopted as ‘bruxism’ to describe gnashing and grinding of the teeth occurring without a functional purpose. Glossary of Prosthodontic Terms (GPT-8) [4] defines bruxism as parafunctional grinding of teeth or an oral habit consisting of involuntary rhythmic or spasmodic non functional gnashing, grinding or clenching of teeth in other than chewing movements of the mandible which may lead to occlusal trauma. Bruxism can occur during wakefulness or during sleep. Bruxism during daytime is commonly a semivoluntary ‘clenching’ activity and is also known as ‘Awake Bruxism’ (AB) or Diurnal Bruxism (DB). AB can be associated with life stress caused by familial responsibility or work pressure. Bruxism during sleep either during daytime or during night is termed as ‘Sleep Bruxism’ (SB). SB is an oromandibular behaviour that is defined as a stereotyped movement disorder occurring during sleep and characterized by tooth grinding and/or clenching [5]. Sleep

bruxism was recently classified as sleep related movement disorder according to recent classification of Sleep Disorders. Historically, occlusal/articulation and skeletal factors were believed to constitute the greatest risk for bruxism, but modern studies have failed to demonstrate a consistently significant relationship between such factors and bruxism. Factors which have been implicated as having an increased risk for bruxism include lower age, female gender, tobacco, alcohol and caffeine usage, psychosocial factors (e.g. stress and anxiety), sleeping disorders (e.g. obstructive sleep apnea), genetics and certain medications or drugs. Some authors have emphasized that bruxism during sleep and during wakefulness should be regarded as two separate entities, probably with different etiologies, and with different presumed risk factors. The American Academy of Sleeping Disorders proposed the terms sleep and awake bruxism [3]. Even though most of the literature does not differentiate between sleep and awake bruxism, studies in sleep-laboratories are thought to produce research of higher quality (sometimes called the ‘‘gold standard’’) than other types of studies, many of which are based on self-reports. It follows that self-report is not an adequate measure of sleep bruxism because of diagnostic bias and confounders. At the practical level, however, the process of diagnosing sleep bruxism by means of polysomnography (PSG) is complicated, while detecting awake bruxism is easier as the patient can report it after becoming, or being made aware of the habit. However, there are some promising recent developments in portable EMG measuring devices for diagnosing bruxism which correlate well with the gold standard, viz. PSG [5].

Prevalence rate of AB and SB is about 20 and 8–16% respectively in adult population [6]. AB is found to occur predominantly among females while no such gender difference is seen for sleep bruxism [7]. Onset of SB is about 1 year of age soon after the eruption of deciduous incisors [8]. The disorder is appearing more frequently in the younger population. The prevalence in children is between 14 to 20%. In adults aged above 60 years and over only 3% are being aware of frequent grinding [9].

Most of the functional activity of mandible occurs at or near the centric occlusion position. The forces related to the functional activity are distributed to many teeth that minimize potential damage to a single tooth. Bruxism occurs in eccentric positions. Few tooth contacts occur during the activity and in this activity, the mandibular position is far from its stable position. This position of mandible causes more strain on the masticatory system, making it more susceptible to breakdown. This causes the application of heavy forces to a few teeth

[10]. This review aims on enumerating the various methods of detecting bruxism and its treatment options.

Aetiology

Bruxism is considered to have multifactorial aetiology. Sleep Bruxism and grinding have been associated with peripheral factors such as tooth interference in dental occlusion, psychosocial influences such as stress or anxiety and central or pathophysiological causes involving brain neurotransmitters or basal ganglia [11].

Central or Pathophysiological Factors

More and more pathophysiological factors are suggested to be involved in the precipitation of bruxism. As the bruxism often occurs during sleep, the physiology of sleep has been studied extensively especially the ‘arousal response’ in search of possible causes of disorder. Arousal response is a sudden change in the depth of the sleep during which the individual either arrives in the lighter sleep stage or actually wakes up. Such a response is accompanied by gross body movements, increased heart rate, respiratory changes and increased muscle activity. Macaluso et al in their study showed 86% of bruxism episodes were associated with arousal response along with involuntary leg movements. This shows that bruxism is a part of arousal response indeed [12].

Recently it is derived that disturbances in central neurotransmitter system may be involved in the aetiology of the bruxism [13]. It is hypothesized that the direct and indirect pathways of the basal ganglion, a group of five sub cortical nuclei that are involved in the coordination of movements is disturbed in bruxer. The direct output pathway goes directly from the stratum to the thalamus from where afferent signals project to the cerebral cortex. The indirect pathway on the other hand passes by several other nuclei before reaching it to the thalamus [14]. If there is imbalance between both the pathways, movement disorder results like Parkinson’s disease. The imbalance occurs with the disturbances in the dopamine mediated transmission of action potential. In case of bruxism there may be an imbalance in both the pathways. Acute use of dopamine precursors like L-dopa inhibits bruxism activityand chronic long term use of L-dopa results in increased bruxism activity. SSRTs (serotonin reuptake inhibitors) which exert an indirect influence on the dopaminergic system may cause bruxism after long term use [13]. Amphetamine which increases the dopamine concentration by facilitating its release has been observed to increase bruxism. Nicotine stimulates central

dopaminergic activities which might explain the finding that cigarette smokers report bruxism two times more than the non smokers [15].

Psychosocial Factors

Number of studies is published in the literature regarding the role of psychosocial factors in the aetiology of bruxism but none of these describe the conclusive nature because of the absence of large scale longitudinal trials. Bruxers differs from healthy individuals in the presence of depression, increased levels of hostility and stress sensitivity. Bruxing children are more anxious than non bruxers. A multifactorial large scale population study to sleep bruxism revealed highly stressful life as a significant risk factor [16]. A study by Van Selms et al demonstrated that daytime time clenching could significantly be explained by experienced stress, although experienced stress and anticipated stress were unrelated to sleep bruxism as recorded with ambulatory devices [17]. All these studies show possible relationship between bruxism and various psychosocial factors are growing but not conclusive.

Peripheral Factors

Several occlusal factors were suggested to be related to self reported bruxism in a study with children. Giffin in his article has mentioned that for an effective management of bruxism, establishment of harmony between maximum intercuspation and centric relation is required [18]. But most of studies published in the literature on this subject now agree that there is no or hardly any relationship between clinically established bruxism and occlusal factors in adults [19]. Manfredini et alin their review of literature have stated that there is still a lack of methodological sound studies to definitely refute the importance of occlusal factors in the etiology of bruxism. [20]

Clinical Diagnosis

Its diagnosis and clinical evaluation is complex, because both bruxers and normal individuals may show some nocturnal parafunctional activity. Diagnosis must be focused on identifying the signs and symptoms reported by the patient or by the dentist over the clinical examination [21]. Dental enamel is the first structure affected from the parafunctional load and abnormal wear of the teeth is the most common evidence of this condition. It may be restricted to a single tooth or the entire mouth [22]. Radiographic examination can show the loss of the

lamina dura, changes in the periodontal space, which can either disappear or be increased, root resorption, root fractures and pulp stones. The major lesions caused by bruxism can be gathered as: effects on teeth, periodontium, masticatory muscles and TMJ, headaches, behavioral and psychological effects. Other signs and symptoms are parafunctionalhyper mobility in the absence of periodontal disease, pulpitis, toothache (with normal pulp), partial crown fractures and teeth migration. Muscular symptoms include fatigue, increased tension in masticatory muscles, especially the lateral pterygoid muscle, mandibular elevator muscles, masseter and temporal [23]. The most common muscular symptom is fatigue, which is the inability to resist during a sustained effort without having apparent signs and symptoms of pain and discomfort [24]. Bruxism can also cause posture problems. In addition, it can affect masticatory muscles and postural muscles of the cervical spine, which may cause muscular pain and future chronic permanent changes. Bruxism harmful habit causes relevant changes in the stomatognathic system structure. It causes friction, inflammation, pulpal necrosis and teeth mobility. It may occur muscle pain and tenderness to palpation and TMJ pain and noises due to lack of coordination of the lateral pterygoid muscles or alter the articular heads, as well as the vertical dimension loss and mandibular displacement in maximal intercuspal position (MIP) [25].

Detection of Bruxism

Intraoral Appliance

Bruxism activity can be evaluated using the intra-oral appliance and is classified into two groups observation of wear facets of the intra-oral appliance and measurement of bite force loaded on the intra-oral appliance.

Wear Facets of Intra-oral Appliance

Holmgren et al. reported a repetitive wear pattern on the occlusal splint. They observed wear facets on full-arch acrylic resin splints, which reappeared in the same location with a similar pattern and direction, even after adjustment of the splints. Also, Korioth et al. reported that parafunctional nocturnal dental activity on full-arch occlusal stabilization splints resulted in wear, which was both asymmetric and uneven. Unfortunately, no confirmation of the reliability of these methods has been reported. [26]

Bruxcore Plate

The Bruxcore Bruxism-Monitoring Device (BBMD) is an intra-oral appliance that was introduced as a device for measuring sleep bruxism activity objectively [27] and the Bruxcore plate evaluates bruxism activity by counting the number of abraded microdots on its surface and by scoring the volumetric magnitude of abrasion. The BBMD is a 0.51-mm-thick polyvinyl chloride plate that consists of four layers with two alternating colours and a halftone dot screen on the topmost surface. The number of missing microdots is counted to assess the abraded area and the number of layers uncovered represents the depth parameter. Both parameters are combined to obtain an index for the amount of bruxism activity. The major disadvantage with this method is that it is difficult to count the number of missing dots with good precision. Pieree and Gale [27] in their study did not find any significant co relation between the duration of bruxism analyzed with the EMG data and that with the bruxcore plate scores. In this respect, the bruxism activity assessed by Bruxcore plates may not be the same as that measured with a portable EMG device. However, the relationship between wear and bruxism activity is still questionable [28].

Detection of Bite Force

Takeuchi et al developed a recording device for sleep bruxism, an intra-splint force detector (ISFD), which uses an intra-oral appliance to measure the force being produced by tooth contact onto the appliance. The force is detected using a thin, deformation-sensitive piezoelectric film, which is embedded 1–2 mm below the occlusal surface of the appliance. It was confirmed that the duration of bruxism events during simulated bruxism, i.e. clenching, grinding, tapping and rhythmic clenching, evaluated with the ISFD was correlated with that of the masseter EMG [29]. Even though the ISFD did not correctly capture force magnitudes during sustained clenching because of the characteristic of the piezoelectric film, i.e. this transducer is best at detecting rapid changes in force, not static forces. ISFD was not suitable for detecting the magnitude of force during steady-state clenching behaviour.

It is obvious; however, that the major problem of these methods is that subjects have to wear the intra-oral device and this may change the original bruxism activity. Well-designed comparative studies with polysomnographic recordings are required to evaluate the possible influences of the intra-oral device on the original bruxism activity [30].

Masticatory Muscle Electromyographic Recording

Among the various methods for the assessment of bruxism, the EMG recording has been commonly used to measure actual sleep bruxism activity directly. The principal advantage is that the occurrence of bruxism can be assessed without intra-oral devices, which may change natural bruxism activity.

Portable EMG Recording Device

Starting in the 1970s, sleep bruxism episodes were measured over an extended period in patients homes with the use of battery-operated EMG recording devices. The portable EMG recording system has become easy for subjects to operate and can measure masticatory muscle activity more minutely, i.e. the number, duration and magnitude of bruxism events can be evaluated with fair accuracy. Criteria for the detection of sleep bruxism with the portable EMG recording system have been suggested [31] but their validity in a large population has not yet been confirmed.

The detection power of sleep bruxism is generally considered inferior to that in a sleep laboratory because other confounding oro-facial activities (e.g. sight, coughing and talking) cannot be discriminated from sleep bruxism. Also, other sleep disorders cannot be ruled out or other physiological changes related to sleep bruxism (e.g. microarousal, tachycardia and sleep-stage shift) cannot be monitored. The implement for recording the heart rate was recommended as one of the compensatory measures for improving the accuracy of sleep bruxism recognition. Also, a surface EMG electrode with a built-in buffer-amplifier and a cordless type of EMG measurement system was developed to improve the reliability of recordings [32].

Miniature Self-Contained EMG Detector–Analyser

A miniature self-contained EMG detector–analyser (BiteStrip) was developed as a screening test for moderate to high level bruxers[33]. The device, which is comprised of EMG electrodes, an amplifier, a central processing unit (CPU) with software, a display which presents the outcome in the morning, a light emitting diode and a lithium battery records the number of masseter muscle activities above a preset threshold. The special feature of this device is that the number of bruxism events can be objectively estimated by simply attaching it to the skin over the masseter muscle. Minakuchi and Clark examined the sensitivity and specificity of the BiteStrip recording versus masseter EMG recordings during a polysomnogram in five suspected bruxers. Overall, there was good specificity for all subjects

but fair sensitivity for subjects that exhibit moderate to high levels of EMG determined bruxism. The device might be a cost-effective tool for screening moderate- to high level bruxism subjects. [34]

More recently, a miniature self-contained EMG detector–analyser with a biofeedback function (Grindcare) was developed as a detector and biofeedback device for sleep bruxism. It is comprised of EMG and stimulation electrodes, a microprocessor, a memory for data storage, a display for user interface, light-emitting diodes, a rechargeable battery, a plug-in USB connector for data connection to computer to a battery charger, and a strap for carrying the apparatus around the forehead. It enables the online recording of EMG activity of the anterior temporalis muscle, online processing of EMG signals to detect tooth grinding and clenching and also biofeedback stimulation for reducing sleep bruxism activities. Although scientific confirmation is needed for a large population, it is considered as one of the potent devices for detecting and also for managing sleep bruxism. The portable EMG recording system enables multiple-night recording in a natural environment for the subject with minimal expense.

Finally, a miniature self-contained EMG detector–analyser seems to be a potentially useful device for detecting sleep bruxism. Polysomnographic (sleep laboratory) recordings for sleep bruxism generally include electroencephalogram, EMG, electrocardiogram and thermally sensitive resistor (monitoring air flow) signals along with simultaneous audio–video recordings. Sleep bruxism activity is assessed based on EMG activity in the masticatory muscles (masseter and/or temporalis). Because the sleep laboratory setting offers a highly controlled recording environment, other sleep disorders (e.g. sleep apnoea and insomnia) can be ruled out and sleep bruxism can be discriminated from other orofacial activities (e.g. myclonus, swallowing and coughing) that occur during sleep [35]. Physiological changes related to sleep bruxism (e.g. microarousal, tachycardia and sleep-stage shift) can also be monitored. Hence, a polysomnographic study allows for multidimensional analyses of sleep-related physiological behaviours and studies on sleep laboratory EMG-based assessments are reported to be very reliable. One major limitation is that a change in the environment for sleep may influence the actual behaviour of bruxism. Another is the expense as multiple night recording is to be taken for the occurrence of sleep bruxism varies over a number of nights [36].

Management of Bruxism

Depending on the etiology, signs observed during the clinical examination and symptoms described by patients, the treatment changes. Furthermore, it is important to make a differential diagnosis [37]. Treatment demands a multidisciplinary approach, involving Psychology, Physiotherapy and Speech Therapy, having in consideration oral, medical and psychological aspects of the patient [38]. Treatment plan should attend the following objectives: physical and psychological stress reduction, treatment of signs and symptoms, reduce occlusal interferences and change the patient's usual neuromuscular pattern. The starting point for treatment aims to decrease psychological stress using relaxation exercises, massage and physiotherapy [39]. This treatment reduces the symptoms but not the cause. The habit may restart whenever the patient's tolerance regarding an occlusal change decreases [40]. Specific treatment for muscle pain is based on methods that disrupt mechanisms of pain cycle, as myofascial trigger point therapy (cool mist spray), anesthetic block in association with physiotherapy techniques such as exercise to restore function and deep heat massage. Occlusal therapy may include occlusal adjustment. Although occlusal condition exerts minor influence on the process, occlusal adjustment, irreversible therapeutic method, is suitable to minimize damages caused by teeth grinding but not for treating bruxism [37]. The use of interocclusal splints reduces symptoms, even if it has not stopped bruxism, because they act in TMJ, inducing the condyle to stand correctly in the condylar fossa. The distribution of masticatory forces is responsible for the relief of symptoms [40]. The splints may differ in material, rigid or resilient, and in structure, thickness and occlusal coverage extension. Thus, according to therapeutics indication, the splits may set different intermaxillary relations [38]. Depending on the complexity of the case, it is usually recommended its use at night, for 45 days, with weekly maintenance. Despite its etiology, occlusal therapy can always be suitable, because it promotes functional comfort, preventing further damage to the components of the masticatory apparatus. Biofeedback is based on the principle that bruxers can 'unlearn' their behaviour when a stimulus makes them aware of their adverse jaw muscle activities ('aversive conditioning'). This technique has been applied for bruxism during wakefulness as well as for sleep bruxism. While awake, patients can be trained to control their jaw muscle activities through auditory or visual feedback from a surface EMG. For sleep bruxism, auditory, electrical, vibratory and even taste stimuli can be used for feedback. Pharmacological treatment with drugs such as dopamine agonists, anxiolytics, buspirone,

nonbenzodiazepines hypnotics, antiepileptic and botulinum toxin, is appropriate when bruxism is intense[41].

Conclusion

Bruxism is a common parafunctional habit, occurring both during sleep and wakefulness, and sleep bruxism and awake bruxism should be differentiated. The etiology of bruxism is not well known can be agreed that it is multifactorial. There is no specific treatment available at this time to stop bruxism and hence we should focus on various methods to reduce the adverse effects of the habit. Early detection using various methods is of prime importance. The use of interocclusal appliances is the most common and accepted way to prevent wear of teeth and prosthodontic restorations in spite of lack of strong evidence for its efficacy. The role of bruxism in the multifactorial process of tooth wear is not clear, but it is in general not the major cause, as has been a frequently stated earlier literature. In extensive tooth wear, the decision to treat or not should be based on the patient's perceived need, the severity of the wear and risk of its progression with respect to the patient's age. When prosthetic intervention is indicated in a patient with bruxism, efforts should be made to reduce the effects of heavy occlusal loading on all the components that contribute to prosthetic structural integrity. Therefore, a multi disciplinary approach is steered towards attempting to prevent damage and to treat the pathological effects of bruxism on the structures of the stomatomasticatory system.

Corresponding Author

Madhulaxmi M

Reader, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Science

162 Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Email ID: madhulaxmi14@gmail.com

Telephone Number: 7373814000

References

1. Lavigne GJ. Bruxism physiology and pathology: an overview of clinicians. J Oral Rehab. 2008; 35:476–494.
2. Burton C Bruxism. Thesis, University of Sydney, 1983
3. Bader G, Lavigne G. Sleep bruxism; an overview of an oromandibular sleep movement disorder. Sleep Med Rev. 2000; 4:27–43.
4. AASM International classification of sleep disorders. American Academy of Sleep Medicine, Westchester, Second Edition 2005
5. Laat A, Macaluso GM. Sleep bruxism as a motor disorder. Mov Disord. 2002; 17:67–69.
6. Glaros AG. Incidence of diurnal and nocturnal bruxism. J Prosthet Dent. 1981;45:545–549
7. ICSD—International Classification of Sleep Disorders: Diagnostic and Coding manual. Diagnostic Classification Steering Committee, Thorpy, Chairman, Rochester, MN: American Sleep Disorders Association 1990
8. Sari S, Sonmez H. The relationship between occlusal factors and bruxism in permanent and mixed dentition in Turkish children. J Clin Pediatr Dent. 2001;25:191–194
9. Lavigne G, Montplaisir JV. Restless legs syndrome and sleep bruxism: prevalence and association among Canadians. Sleep. 1994; 17:739–743.
10. Okeson JP. Occlusion. In: Ramfjord S, Ash MM, editors. Management of Temporomandibular Disorders and Occlusion. 5th ed. Philadelphia: Saunders; 1966.
11. A Bader G, Lavigne G. Sleep bruxism; an overview of an oromandibular sleep movement disorder. Sleep Med Rev. 2000; 4:27–43.
12. Bader G, Lavigne G. Sleep bruxism; an overview of an oromandibular sleep movement disorder. Sleep Med Rev. 2000; 4:27–43.
13. Lobbezoo F, Soucy JP, Montplaster JY, Lavinge GJ. Striatal D2 receptor binding in sleep bruxism: a controlled study with iodine-123-iodobenzamide, single photon emission computed tomography. J Dent Res. 1996; 75:1804.

14. Lobbezoo F, Van Denderan RJ. Reports of SSRI-associated bruxism in the family physician office. J Orofac Pain. 2001; 15:340–346.
15. Ashroftt GW, Eccleston D, Waddell JL. Recognition of amphetamine addicts. Br Med J. 1965; 1:57.
16. Ohayon MM, Li KK, Guilleminault C. Risk factors for sleep bruxism in the general population. Chest. 2001;119:53–61
17. Selms MKA, Lobbezoo F, Wicks DJ, Hamburger HL, Naeije M. Craniomandibular pain, oral parafunction, and psychological stress in a longitudinal case study. J Oral Rehabil. 2004; 31:738–745.
18. Giffin KM. Mandibular adaptive reposturing: the aetiology of a common and multifaceted auto destructive syndrome. Gen Dent. 2003; 51:62–67.
19. Manfredini D, Landi N, Tognini F, Montagnani G, Bosco M. Occlusal features are not a reliable predictor of bruxism. Minerva Stomatol. 2004; 53:231–239.
20. Ashish. R. Jain, “Fp1 Prosthesis in Maxillary Ridge Defect and Fixed Partial Denture in Mandibular Ridge Defect - A Case Report.” International Journal of Dental Sciences and Research. 2014; 2:184-189.
21. Lobbezoo F, van der Zaag J, van Selms MK, Hamburger HL, Naeije M Principles for the management of bruxism. J Oral Rehabil 2008;35: 509–523.
22. Wahlund K, Nilsson IM, Larsson B Treating temporomandibular disorders in adolescents: a randomized, controlled, sequential comparison of relaxation training and occlusal appliance therapy. J Oral Facial Pain Headache. 2015;29: 41-50.
23. Muthukrishnan A, Sekar GS. Prevalence of temporomandibular disorders in Chennai population. J Indian Acad Oral Med Radiol 2015;27:508-515
24. Shukla D, Muthusekhar M R. Efficacy of low-level laser therapy in temporomandibular disorders: A systematic review. Natl J Maxillofac Surg 2016;7:62-6
25. Jain, A.R. & Sindhu, P. Full mouth rehabilitation of a bruxer with severely worn dentition using all ceramic zirconia crowns – A case report. Journal of Pharmaceutical Sciences and Research. 2017;9:2001-2003.
26. Badel T, Ćimić S, Munitić M, Zadravec D, Kes VB. Clinical view of the temporomandibular joint disorder. Acta Clin Croat 2014; 53: 462- 470
27. Baba K, Haketa T, Clark GT, Ohyama T. Does tooth wear status predict ongoing sleep bruxism in 30-year-old Japanese subjects? Int J Prosthodont. 2004; 17:39–44.

28. Ekfeldt A, Hugoson A, Bergendal T, Helkimo A. An individual tooth wear index and an analysis of factors correlated to incisal and occlusal wear in an adult Swedish population. Acta Odontol Scand. 1990; 48:343–349.
29. Johansson A, Johansson AK, Omer R, Carlsson GE. Rehabilitation of the worn dentition. J Oral Rehabil. 2008; 35:548–566.
30. Holmgren K, Sheikholeslam A, Riise C. Effect of a full-arch maxillary occlusal splint on parafunctional activity during sleep in patients with nocturnal bruxism and signs and symptoms of Craniomandibular disorders. J Prosthet Dent. 1993; 69:293–297.
31. Korioth TW, Bohlig KG, Anderson GC. Digital assessment of occlusal wear patterns on occlusal stabilization splints: a pilot study. J Prosthet Dent. 1998; 80:209–213.
32. Baba K, Clark GT, Watanabe T, Ohyama T. Bruxism force detection by a piezoelectric film-based recording device in sleeping humans. J Orofac Pain. 2003; 17:58–64.
33. Forgione. A Simple but effective method quantifying bruxing behavior. J Dent Res 53,1974
34. Pierce CJ, Gale EN. Methodological considerations concerning the use of bruxcore plates to evaluate nocturnal bruxism. J Dent Res. 1989;68:1110–1114
35. Lobbezoo F, Zaag J, Selms MKA, Hamburger HL, Naeije M. Principles for the management of bruxism. J Oral Rehabil. 2008;35:509–523
36. Takeuchi H, Ikeda T, Clark GT. A piezoelectric film-based intrasplint detection method for bruxism. J Prosthet Dent. 2001;86:195–202
37. Rugh JD, Solberg WK. Electromyography studies of bruxist behavior before and during treatment. J Calif Dent Assoc. 1975; 3:56–59.
38. Ikeda T, Nishigawa K. Criteria for the detection of sleep associated bruxism in humans. J Orofac Pain. 1996; 10:270–282.
39. Kato T, Thie NM, Huynh N, Miyawaki S, Lavigne GJ. Topical review: sleep bruxism and the role of peripheral sensory influences. J Orofac Pain. 2003; 17:191–213.
40. Lavigne GJ, Khoury S, Abe S, Yamaguchi T, Raphael K. Bruxism physiology and pathology: an overview for clinicians. J Oral Rehabil. 2008;35:476–494
41. Alonso-Navarro H, Martín-Prieto M, Ruiz-Ezquerro JJ, JiménezJiménez FBruxism possibly induced by venlafaxine. Clin Neuropharmacol2009; 32: 111-112.
42. Manfredini D, Ahlberg J, Winocur E, Lobbezoo Managements of sleep bruxism in adults: a qualitative systematic literature review. J Oral Rehabil, 2015

Burkitt's Lymphoma- A Review

Jayajanani.S [1], Jayashri.P [2]

Graduate Student [1],Senior Lecturer [2], Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Burkitt's lymphoma is a highly aggressive B-cell non-Hodgkin lymphoma and is the fastest growing human tumour. The disease is associated with Epstein-Barr virus and was one of the first tumours shown to have a chromosomal translocation that activates an oncogene (c-MYC). Burkitt's lymphoma is the most common childhood cancer in areas where malaria is holoendemic. The incidence is very high in immunosuppressed patients in non-endemic areas, especially when associated with HIV infection. Outcome with intensive chemotherapy has improved and is now excellent in children, but the prognosis is poor in elderly adults. The success of intensive treatment relies on good supportive care. The therapy offered in oncology units in low-income countries is not as aggressive as in centres in high-income countries and outcomes are less successful. Adjuvant monoclonal antibody therapy with rituximab shows promise for improved outcomes and reduced toxic effects in the future.

Key Words: Burkitt's lymphoma, Malaria, Histopathology, Prognostic Markers, Tumours

Introduction

Burkitt's lymphoma has had an important role in the understanding of tumorigenesis. It was the first human tumour to be associated with a virus,[1] one of the first tumours shown to have a chromosomal translocation that activates an oncogene,[2,3] and the first lymphoma reported to be associated with HIV infection[4]. Burkitt's lymphoma is the fastest growing human tumour, with a cell doubling time of 24–48 h, and was the first childhood tumour to respond to chemotherapy alone [5].It is the most common childhood cancer in areas where malaria is holoendemic—e.g., equatorial Africa, Brazil, and Papua New Guinea.6 The so-called Burkitt's lymphoma belt stretches across central Africa 15° either side of the equator where the climate is hot and wet (more than 50 cm annual rainfall). The epidemiological maps of malaria and Burkitt's lymphoma overlap [5–7].

Early in the 20th century, Sir Albert Cook, a missionary doctor in Uganda, and other medical staff working in west, east, and central Africa noted the high frequency of jaw tumours and childhood lymphomas [8–10]. In 1958, Denis Burkitt, an Irish surgeon working in Uganda, reported cases of children presenting with rapidly growing jaw or abdominal tumours [11]. Burkitt suggested that these tumours were round-cell sarcoma. However, in 1960 George O'Connor, a pathologist, concluded that the cancer was of lymphoma lineage [12]. In 1964, three virologists, Michael Anthony Epstein, Yvonne Barr, and Bert Achong identified viral particles in the tumour tissue; this virus became known as Epstein-Barr virus (EBV) [1]. Meanwhile, Burkitt travelled through eastern and central Africa to map the tumour spread and found records of affected children in all the malarial areas of the region [13].

Classification

The WHO classification of Burkitt's lymphoma de- scribes three clinical variants: endemic, sporadic (the predominant type found in non-malarial areas), and immunodeficiency-related [20]. These types are similar in morphology, immunophenotype, and genetic features.

The endemic variant is associated with malaria endemicity and EBV is found in almost all cases. The sporadic type occurs mainly throughout the rest of the world (predominantly North America and Europe), with no special climatic or geographical links, and is rarely associated with EBV infection. 1–2% of adult lymphomas and 30–40% of childhood non-Hodgkin lymphomas in Europe and North America are sporadic-type Burkitt's lymphoma [21]. The immunodeficiency-related type is seen most often in patients with HIV infection and less

than 40% of US and European cases are associated with EBV. Before the advent of antiretroviral therapy in North America the disorder was 1000 times more common in HIV-positive people than in uninfected individuals [22, 23]. Immunodeficiency-related Burkitt's lymphoma is more common when the CD4 T-cell count is greater than 200 per μL (early in the progression of HIV infection). The association of HIV with Burkitt's lymphoma is not as clear in the endemic form [24]. The risk of BL increases 4 to 5 years after organ transplantation, but this risk is much less than that associated with HIV infection [25].

Epidemiology

The distribution of endemic Burkitt's lymphoma across Africa and Papua New Guinea corresponds to areas of holoendemic malaria and the early acquisition of EBV [13,26–32]. The annual incidence has been estimated at 40–50 per million children younger than 18 years [33]. In these high-risk areas endemic Burkitt's lymphoma comprises about half of all childhood cancer diagnoses and up to 90% of lymphoma diagnoses. Incidence peaks at age 6 years and the disease is twice as common in boys as in girls. Sporadic Burkitt's lymphoma occurs most commonly in children aged 3–12 years (median 6–8 years) and is 3·5 times more common in boys than in girls [34,35].

Sporadic Burkitt's lymphoma is found in low-risk areas such as North America, northern and Eastern Europe, and East Asia at an annual incidence of 2 per million children younger than 18 years. Parts of South America, southern Europe, North Africa, and the Middle East are areas of intermediate risk [30]. Immunodeficiency-associated Burkitt's lymphoma occurs at an incidence of 22 per 100 000 person-years in the USA [36].

Cofactors

Epstein-Barr virus

Several observations suggest a direct causative role for EBV in endemic Burkitt's lymphoma. For example, EBV is consistently present in these tumours; [37] infection of malignant B cells precedes tumorigenesis; [38] EBV induces immortalisation of B cells in culture; and very high EBV antibody titres are recorded in children before development of the disease [39]. However, the underlying mechanism linking EBV infection of B cells to the emergence of malignancy remains undiscovered.

Although EBV encodes several latent proteins essential for viral immortalisation of B cells,[40–42] EBNA1 protein is the only EBV latent protein consistently expressed in endemic Burkitt's lymphoma tumours. Other EBV latent and lytic transcripts are also detected in some tumours (figure 2),[37,44] but only in a subset of cells. Tumours containing a deletion of the EBNA2 gene have been identified, which leads to expression of EBNA3A, EBNA3B, and EBNA3C genes [46]. Cell lines derived from these lymphomas are resistant to apoptosis, which suggests that loss of EBNA2 provides a survival advantage to the tumour [47]. One function of EBV in endemic Burkitt's lymphoma might be to block apoptosis in B cells with an MYC translocation through either the EBNA1 protein, the BHRF1 protein, EBER transcripts, or epigenetic modification and subsequent repression of the pro- apoptotic BIM protein by the latent transcript LMP1 [48,49]. EBV can also promote genomic instability, deregulate telomere functions, and induce DNA damage to infected cells.50 Viral microRNAs have been identified in EBV- positive endemic and AIDS-associated Burkitt's lymphomas,[45] and are thus also potential candidates for driving tumorigenesis.

The cell type of origin of the Burkitt's lymphoma cell is controversial; some have argued that the tumour arises from a germinal centre B cell, [51] whereas others believe that it originates from a memory B cell [52]. This question is also relevant to the role of EBV in endemic disease. EBV persists for the lifetime of the healthy host as a latent infection in peripheral memory B cells. If the malignant cell arises from a latently infected cell and cycling memory B cells express only EBNA1 (similar to endemic Burkitt's lymphoma), [53] the latently infected memory B cell could be the source of the malignancy.

EBV can be identified in almost all endemic Burkitt's lymphomas, but is reported less frequently in the other types, which raises the question of whether EBV is a requirement for pathogenesis. The absence of EBV in other types of Burkitt's lymphoma might result from loss of viral episomes from tumour cells after cell division. EBV-positive Burkitt's lymphoma has a higher frequency than EBV-negative disease of somatic mutations in the immunoglobulin variable heavy chain with evidence of antigen selection. A possible explanation for this findingis that EBV-positive Burkitt's lymphoma arises from memory cells whereas EBV-negative disease originates from an earlier germinal centre counterpart [54].

Malaria

In Africa, pronounced seasonal, temporal, and spatial variations in the incidence of Burkitt's lymphoma have long been linked to the prevalence of malaria,[31,55,56] and in 2008, direct evidence of a link between malaria, EBV, and endemic Burkitt's lymphoma emerged [57,58]. Two epidemic- logical studies showed that the risk of Burkitt's lymphoma was greatest in people with the highest titres of antibodies against both EBV and Plasmodium falciparum [19, 59].

Severalstudieshaveshownthatmalariacancausea profound dysregulation of EBV persistence and immunity in children [60–65]. These results suggest that malaria increases the risk of endemic Burkitt's lymphoma through interactions with EBV-infected Bcells.Forexample,thecysteine-richinterdomain1αofthe P falciparumerythrocytemembraneproteininduces reactivation of EBV.66 Additionally, P falciparum has a ligand for toll-like receptor 9 [67]. Signalling through toll- like receptor 9 was shown to induce the enzyme activation-induced cytidine deaminase in human B cells [68, 69]. Overexpression of this enzyme induces the immunoglobulin-MYC translocations [70].Characteristic of Burkitt's lymphoma. Normal B cells undergo apoptosis if MYC is overexpressed after an activation-induced cytidine deaminase-mediated translocation. However, EBV latent proteins are ant apoptotic, which might allow the B cells to tolerate the translocation and ultimately give rise to a malignant clone. In malaria-endemic regions, diminished EBV-specific cytotoxic T-cell responses were observed in children at peak age of Burkitt's lymphoma incidence [61]. Children with acute malaria also have transient loss of EBV-specific T-cell control [71]. Malaria probably increases the risk of endemic Burkitt's lymphoma by increasing the number of latently infected B cells through viral reactivation and reseeding of the latent pool; by causing loss of immune control of latently infected B cells; and by inducing MYC trans- location through a mechanism mediated by activation- induced cytidine deaminase.

HIV Infection

Burkitt's lymphoma occurs in HIV-infected patients with high CD4 T-cell numbers, which suggests that immunosuppression is not in itself the cause of the malignancy. Chronic antigenic stimulation of B cells, as in sustained P falciparum infection or chronic HIV infection, might be a common pathogenetic mechanism of endemic and HIV-associated Burkitt's lymphoma. HIV-infected patients with Burkitt's lymphoma have high serum

concentrations of soluble CD30 and CD23— markers of B-cell activation—before emergence of lymphoma [72–74]. Patients with chronic HIV viraemia, even if on antiretroviral therapy, have a higher risk of developing HIV-associated lymphomas than those with unmeasureable viral loads [75, 76]. The virus might affect B cells through dysregulation of activation-induced cytidine deaminase and chronic B-cell activation. The enzyme has been detected in peripheral lymphocytes in HIV-infected patients with lymphoma, but not in HIV- positive patients without the malignancy, nor in healthy controls [77]. Impaired immune surveillance and deregulated cytokine release could promote survival of B cells with chromosomal rearrangements induced by overexpression of the activation-induced cytidine deaminase.

The effect of HIV infection on the risk of endemic Burkitt's lymphoma is unclear. Different clinical presentations and tumour behaviour have been noted between HIV-infected and uninfected people [78, 79]. An association was first reported from a Ugandan study,24 but neither work in Côte d'Ivoire [80] nor in Zambia [81] confirmed this link. Preliminary data from Malawi identified an increased risk of endemic Burkitt's lymphoma in HIV-infected patients, 58 but updated analyses found no significant association [82].

Other Possible Cofactors

Arboviruses and schistosome parasites have both been suggested as causative cofactors of endemic Burkitt's lymphoma, although evidence is sparse [30]. The plants Euphorbiae tirucalli and Jatropha curcas are common in areas where the endemic type of the disease occurs. The milky sap of these plants contains dipterene esters that can activate latent EBV and induce rearrangements of chromosomes in about 10% of exposed EBV-infected B cells [83–85].

Clinical Presentation

The most common site of presentation in sporadic Burkitt's lymphoma is the abdomen (60–80%) [21]. Presenting symptoms include abdominal pain (25% of patients have ileocaecal disease—either a right lower quadrant mass or pain from intussusception), distension, nausea and vomiting, and gastrointestinal bleeding [86, 87]. The next most common site is the head and neck, including lymphadenopathy and involvement of the nasal or oropharynx, tonsils, or sinuses. The jaw is infrequently implicated. Bone marrow is infiltrated in roughly 20% of patients. Some cases are classified as Burkitt's leukaemia and are characterised by extensive

marrow infiltration (more than 25% blasts), with possible bone pain as a presenting feature. Rare presenting sites include the mediastinum, CNS, skin, testes, breasts, and thyroid gland.
Patients with endemic Burkitt's lymphoma most frequently present with jaw or per orbitalswellings, or abdominal involvement (of retroperitoneal tissue, gut, ovary, or kidney) [88]. 15% present with sudden paraplegia and incontinence. Infiltration of bone marrow is rare. Jaw involvement is common in young children (peak ages of incidence 3–7 years) [89]. In low- income countries, such as in sub-Saharan Africa, many children present with advanced disease. In a study of 84 Malawian children with Burkitt's lymphoma, [26] (31%) presented with facial disease only and [52] (62%) with abdominal disease; [58] (69%) had St Jude stage III or IV disease.88 Patients are commonly malnourished at diagnosis [90].

Histopathology and Immunocytochemistry

Burkitt's lymphoma is a highly aggressive B-cell non- Hodgkin lymphoma characterised by monomorphic medium-sized cells with a very high proliferation rate. The cells are intermediate in size and contain coarse chromatin and prominent basophilic nucleoli. Some plasmacytoid and atypical variants show more nuclear pleiomorphism. In tissue sections, typically the cells seem to be moulded and the cytoplasm is deeply basophilic with squared-off cytoplasmic margins. The proliferation index is almost 100%, with a high turnover shown by increased apoptosis. A "starry sky" appearance is due to scattered tingible-body-laden macrophages that contain apoptotic tumour cells [43]. The cells are always of B-cell lineage (CD20 positive and CD79a positive). CD10 and Bcl-6 are commonly co expressed, but the cells are generally negative for Bcl-2. There is a scarcity of T cells in the background.91 Epstein-Barr-encoded RNA can be identified by fluorescence in-situ hybridisation. Classification is difficult when the cells have the morphology of diffuse large B-cell lymphoma but the genetic and immunophenotype features of Burkitt's lymphoma. Some of these cases are now classified as "B-cell lymphoma, unclassifiable, with features between diffuse large B-cell lymphoma and BL [Burkitt's lymphoma]" [92]. However, distinct molecular changes in Burkitt's lymphoma could provide a more reliable diagnosis.

Diagnosis

High-Income Countries

Diagnosis of Burkitt's lymphoma should be confirmed by microscopy and immunocytological analysis. The recommended approach is to remove and examine the most accessible disease-containing tissue. This sample could be a superficial lymph node or

malignant pleural fluid. Excision biopsy of a lymph node is preferable to fine-needle aspiration, which does not provide sufficient tissue for all the investigations required. In some cases a laparotomy or laparoscopy is necessary to obtain tissue.

Several essential investigations should be done in patients with suspected Burkitt's lymphoma: full blood count, differential and film, ESR and urea and electrolyte measurements, liver function tests, a clotting screen (pro- thrombin time, partial thromboplastin time, D-dimers) to assess renal and hepatic involvement or dysfunction, serum lactate dehydrogenase and urate measurements (to assess tumour turnover), EBV status, and chest radio- graphy. The radiograph should be done before any anaesthetic is given, to look for mediastinal lymph nodes with or without pleural effusions. CTs of chest and abdomen show disease extent and can be done after tissue diagnosis unless airway obstruction is suspected. PET scanning is recommended, but is not essential. After confirmation of the diagnosis, bilateral bone-marrow aspirates, trephine cores, and cerebrospinal fluid should be examined for the presence of malignant cells [93].

Low-Income Countries

Diagnostic facilities in low-income countries are likely to be restricted. The most common diagnostic test is cytological examination of a fine-needle aspirate. Results are commonly not available at the time of clinical decision making. Ultrasonography is useful to detect intraabdominal masses. Examination of cerebrospinal fluid and bone- marrow aspirates will detect CNS and bone-marrow involvement. If possible radiography should be done, as well as basic blood tests (such as full blood count, urea and electrolyte measurements, and liver function tests).

Common co infections (e.g., malaria, helminth infections) should be identified and treated before chemotherapy begins. HIV infection should be noted so that antiretroviral therapy can be given after chemotherapy for Burkitt's lymphoma. Tuberculosis and Kaposi's sarcoma should be ruled out either clinically or histologically.

Prognostic Markers

Therapy is guided partly by clinical and histopathological staging with biological features beginning to inform therapeutic strategies. Clinically, prognosis is determined by staging, which includes extent of disease [94]. The St Jude/Murphy classification for Burkitt's

lymphoma is the most common staging system used (panel) [95]. Cytogenetic analysis is important in diagnosis to identify MYC deregulation and the presence of additional cytogenetic abnormalities, some of which have been shown to have prognostic significance.PET is helpful to assess residual or recurrent disease. Burkitt's lymphoma produces a very strong signal with fluorodeoxyglucose- PET. Disease can be monitored regularly throughout treatment. Because of the high fluorodeoxyglucose avidity of the lymphoma, a single scan can provide valuable confirmation of recurrent disease [96].

The role of minimal residual disease monitoring in Burkitt's lymphoma is not yet established. Some clinicians routinely monitor for it, although findings are not generally used to guide treatment. The presence of mini- mal residual disease in bone marrow (which is also a measure of minimal disseminated disease) is better applied to the prediction of high risk of treatment failure. The construction of primers for use in monitoring of minimal residual disease is generally based on the original tumour material: 5VH and 7DH family primers in com-bination with one JH consensus primer for immuno- globulin heavy chain rearrangements; [97]. BIOMED 1 primer set for kappa light chain deletions; and BIOMED 2 primer set for immunoglobulin kappa v–J [98]. Other strategies have been used such as patient-specific long-range PCR with primers related to the (8:14) translocation and primer pools from the immunoglobulin V [99]. However, the H1–H7 most accurate analysis uses primers determined by the original tumour material. Assessment of prognosis by retrospective molecular profiling (e.g., array-based com- parative genomic hybridisation and gene-expression profiling) does not seem to offer any advantages over standard morphology and immunocytochemistry [100]. In low-income countries, disease staging and response to treatment (including ultrasonography of the abdomen) might be the only prognostic guides available.

Management

High-Income Countries

Treatment of Burkitt's lymphoma in most centres is guided by the FAB LMB study (cooperative study between the Children's Cancer Group, the Societieś Française d'Oncologie Pédiatrique, and the UK Children's Cancer Study Group) [86,87] or Berlin–Frankfurt–Münster protocols. The former con- sists of initial cytoreduction with cyclophosphamide, prednisolone, and vincristine, followed by more intensive chemotherapy in varying combinations. The risk of pronounced tumour lysis is high in the first few days of

therapy, but the use of urate oxidase has reduced this danger substantially. Because of the toxic effects of these protocols, sophisticated supportive care is needed, which is not possible for most low-income countries.

Management of Burkitt's lymphoma can be divided into three broad groups of patients. Children with localised disease that has been completely removed surgically need only two cycles of moderately intensive chemotherapy such as cyclophosphamide, vincristine, prednisolone, and doxorubicin. Children with residual or stage III disease need at least four cycles of dose-intensive chemotherapy, such as two cycles of cyclophosphamide, vincristine, prednisolone, doxorubicin, and high-dose methotrexate, followed by two cycles of cytarabine and high-dose methotrexate with concurrent intrathecal treatment. Children with CNS or bone-marrow involvement are given similar treatment to the second group, but receive up to eight courses of dose-intensive treatment. This therapy typically involves two courses of cyclophosphamide, vincristine, prednisolone, doxorubicin, and high-dose methotrexate followed by two courses of high and low doses of cytarabine, and etoposide) and four courses of maintenance with varying combinations of vincristine, prednisolone, high-dose methotrexate, cyclophosphamide, doxorubicin, cytarabine, and etoposide. Intrathecal therapy is given alongside systemic chemotherapy.

The use of rituximab (anti-CD20) in primary therapy has been assessed, and some small single-centre studies report encouraging results. [101]. Data are awaited from a Children's Oncology Group pilot study on toxic effects (ANHL01P1) in which rituximab was given to patients with stage III and IV Burkitt's lymphoma. The next UK trial will randomise the use of rituximab for stage III and IV patients.

Low-Income Countries

Therapy needs to be modified in accordance with local conditions to avoid unacceptable treatment-related mortality. The intensity of treatment is determined by the amount of available supportive care, a child's tolerance of chemotherapy, and the extent of comorbidities. In Malawi, for example, the treatment for Burkitt's lymphoma of all stages is intravenous cyclophosphamide (40 mg/kg on day 1 and oral cyclophosphamide 60 mg/kg on days 8, 18, and 28). Intrathecal hydrocortisone (12·5 mg) and methotrexate (12·5 mg) are given with each treatment cycle. The cost of this 28-day treatment is less than US$50.88

Previous attempts to use intensive treatments with high-dose methotrexate resulted in unacceptably high treatment-related mortality (11 of 42 participants) [102].

In a French-African Paediatric Oncology Group study, two moderately intensive modified LMB 89 protocols (including high-dose methotrexate and cytarabine) were used in several French-speaking African countries.114 Of 306 patients, 71 (23·2%) died during treatments; 40 (13·1%) deaths were attributed to infection [103].Adequate and timely supportive care, even if not as intensive as in high-income countries, is essential, and should include measures to prevent and manage tumour lysis syndrome, nutritional support (malnutrition is associated with chemotherapy-related neutropenia90), antiemetics, transfusion support, and a local fever protocol. In low-income countries, many patients do not complete the full course of treatment [104-109]. Travel distances and expense, treatment costs, and poor knowledge of the disease all contribute to non-completion [110-113]. Ideally, medical treatment would be free to the patient and appropriate social support would be provided to enable treatment to be completed.

Conclusion

In low-income countries better diagnostic testing is needed. When only morphology is available, tumours are probably incorrectly classified as Burkitt's lymphoma.Additionally,ahighstandardofsupportive care and medical infrastructure is necessary to deliver the most effective therapy. New, effective, and inexpensive therapies are needed for low-income countries. One possibility is to use compounds with histone deacetylase inhibitor activity as adjuvant therapy.These agents stimulate tumour cells to differentiate and undergo apoptosis, and also induce virus lytic replication in EBV-positive tumours. Tumours with some viral replication have been shown to be more sensitive to chemotherapy than those without any replication.For example, sodium phenylbutyrate induces EBV lytic replication in susceptible B-lymphocyte cultures.

With improved molecular profiling and understanding of the cause of Burkitt's lymphoma, targeted therapy will be developed that still has excellent cure rates but has reduced toxic effects. Potential targets could include the MYC oncogene, DNA methyltransferase inhibitors, cyclin-dependent kinase inhibitors, and proteosome inhibitors. As further biological factors are identified, more targeted therapies will probably be developed.

In the 1980s Guy de Thé described Burkitt's lymphoma as "the Rosetta stone of cancer". This description remains true now. In attempting to understand Burkitt's lymphoma, much is still to be learnt about how all cancers develop, grow, and are treated.

Corresponding Author

P.Jayashri

Senior Lecturer,Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai-600077,Tamil Nadu, India.

Mobile:-+91-9841788803

Email id:-shrijaya2009@gmail.com

References

1. Epstein MA, Achong BG, Barr YM. Virus particles in cultured lymphoblasts from Burkitt's lymphoma. Lancet 1964; 283: 702–03.
2. Zech L, Haglund U, Nilsson K, Klein G. Characteristic chromosomal abnormalities in biopsies and lymphoid-cell lines from patients with Burkitt and non-Burkitt lymphomas. Int J Cancer 1976; 17: 47–56.
3. Manolov G, Manolova Y. Marker band in one chromosome 14 from Burkitt lymphomas. Nature 1972; 237: 33–34.
4. Schulz TF, Boshoff CH, Weiss RA. HIV infection and neoplasia. Lancet 1996; 348: 587–91.
5. Burkitt D. Etiology of Burkitt's lymphoma—an alternative hypothesis to a vectored virus. J Natl Cancer Inst 1969; 42:19–28.
6. Parkin DM, Hamdi-Cherif M, Sita F. Cancer in Africa: epidemiology and prevalence. Burkitt lymphoma.IARC Scientific Publications 2003; 153:324–28.
7. Kafuko GW, Burkitt DP. Burkitt's lymphoma and malaria.Int J Cancer 1970; 6: 1–9.
8. Davies JN. Pathology of central African natives; Mulago Hospital post-mortem studies. East Afr Med J 1948; 25 454–67.
9. Elmes BG, Baldwin RB. Malignant disease in Nigeria; an analysis of a thousand tumours. Ann Trop Med Parasitol 1947; 41:321–28.
10. Thijs A. Malignant tumors of Belgium Congo and Ruanda-Urundi natives; 2,536 case reports. Ann Soc Belg Med Trop (1920) 1957;37: 483–84.
11. Burkitt D. A sarcoma involving the jaws in African children. Br J Surg 1958; 46: 218–223.

12. O'Connor GT, Davies JN. Malignant tumors in African children. With special reference to malignant lymphoma. J Pediatr 1960; 56:526–35.
13. Burkitt D. A "tumour safari" in east and central Africa. Br J Cancer 1962; 16: 379–86.
14. Doll DC, List AF. Burkitt's lymphoma in a homosexual. Lancet 1982; 319: 1026–27.
15. Baer R, Bankier AT, Biggin MD. DNA sequence and expression of the B95-8 Epstein-Barr virus genome. Nature 1984; 310:207–11.
16. Kalter SP, Riggs SA, Cabanillas F. Aggressive non-Hodgkin's lymphomas in immunocompromised homosexual males. Blood 1985; 66:655–59.
17. Gerrard M, Cairo MS, Weston C. Excellent survival following two courses of COPAD chemotherapy in children and adolescents with resected localized B-cell non-Hodgkin's lymphoma: results of the FAB/LMB 96 international study. Br J Haematol 2008; 141: 840–47.
18. Robbiani DF, Bothmer A, Callen E. AID is required forthe chromosomal breaks in c-myc that lead to c-myc/IgH translocations. Cell 2008; 135: 1028–38.
19. Rochford R, Cannon MJ, Moormann AM. Endemic Burkitt's lymphoma: a polymicrobial disease? Nat Rev Microbiol 2005; 3: 182–87.
20. Jaffe ES. The 2008 WHO classification of lymphomas: implications for clinical practice and translational research.Hematology Am Soc Hematol Educ Program 2009; 1: 523–31.
21. Mbulaiteye SM, Biggar RJ, Bhatia K, Linet MS, Devesa SS. Sporadic childhood Burkitt lymphoma incidence in the United States during 1992–2005. Pediatr Blood Cancer 2009; 53: 366–70.
22. Knowles DM. Etiology and pathogenesis of AIDS-related non-Hodgkin's lymphoma. Hematol Oncol Clin North Am 2003;17: 785–820.
23. Martinez-Maza O, Breen EC. B-cell activation and lymphomain patients with HIV. Curr Opin Oncol 2002; 14: 528–32.
24. Newton R, Ziegler J, Beral V. A case-control study of human immunodeficiency virus infection and cancer in adults and children residing in Kampala, Uganda. Int J Cancer 2001; 92: 622–27.
25. Ferry JA. Burkitt's lymphoma: clinicopathologic features and differential diagnosis. Oncologist 2006; 11: 375–83.
26. Burkitt D. Determining the climatic limitations of a children's cancer common in Africa. BMJ 1962; 2: 1019–23.

27. Burkitt D. A children's cancer dependent on environment. In: Cumley RW, ed. Viruses, nucleic acids and cancer. A collection of papers presented at the 17th Annual Symposium on FundamentalCancer Research,1963. London: Bailliere, Tindall, and Cox, 1964: 615–629.
28. Burkitt D. A lymphoma syndrome dependent on environment. ii. epidemiological features. Basel: S Karger, 1964.
29. Cook-Mozaffari P, Newton R, Beral V, Burkitt DP. The geographical distribution of Kaposi's sarcoma and of lymphomas in Africa before the AIDS epidemic. Br J Cancer 1998; 78: 1521–28.
30. Cardy AH, Sharp L, Little J. Burkitt's lymphoma: a reviewof the epidemiology. Kuwait Med J 2001; 33: 293–306.
31. Rainey JJ, Mwanda WO, Wairiumu P, Moormann AM, Wilson ML, Rochford R. Spatial distribution of Burkitt's lymphoma in Kenya and association with malaria risk. Trop Med Int Health 2007; 12: 936–43.
32. De Thé G. Is Burkitt's lymphoma related to perinatal infection by Epstein-Barr virus? Lancet 1977; 309: 335–38.
33. Orem J, Mbidde EK, Lambert B, de Sanjose S, Weiderpass E. Burkitt's lymphoma in Africa, a review of the epidemiology and etiology. Afr Health Sci 2007; 7: 166–75.
34. Mwanda OW, Rochford R, Moormann AM, Macneil A, Whalen C, Wilson ML. Burkitt's lymphoma in Kenya: geographical, age, gender and ethnic distribution. East Afr Med J 2004; 8 (suppl): S68–77.
35. Burkitt DP. Epidemiology of Burkitt's lymphoma. Proc R Soc Med 1971; 64: 909–10.
36. Guech-Ongey M, Simard EP, Anderson. AIDS-related Burkitt lymphoma in the United States: what do age and CD4 lymphocyte patterns tell us about etiology and/or biology? Blood 2010; 116: 5600–04.
37. Niedobitek G, Agathanggelou A, Rowe M. Heterogeneous expression of Epstein-Barr virus latent proteins in endemic Burkitt's lymphoma. Blood 1995; 86: 659–65.
38. Neri A, Barriga F, Inghirami G. Epstein-Barr virus infection precedes clonal expansion in Burkitt's and acquired immunodeficiency syndrome-associated lymphoma. Blood 1991;77: 1092–95.
39. Geser A, de Thé G, Lenoir G, Day NE, Williams EH. Final case reporting from the Ugandan prospective study of the relationship between EBV and Burkitt's lymphoma. Int J Cancer 1982; 29: 397–400.

40. Cohen JI, Kieff E. An Epstein-Barr virus nuclear protein 2 domain essential for transformation is a direct transcriptional activator.J Virol 1991; 65: 5880–85.
41. Kaye KM, Izumi KM, Kieff E. Epstein-Barr virus latent membrane protein 1 is essential for B-lymphocyte growth transformation.Proc Natl Acad Sci USA 1993; 90: 9150–54.
42. Tomkinson B, Robertson E, Kieff E. Epstein-Barr virus nuclear proteins EBNA-3A and EBNA-3C are essential for B-lymphocyte growth transformation. J Virol 1993; 67: 2014–25.
43. Fujita S, Buziba N, Kumatori A, Senba M, Yamaguchi A,Toriyama K. Early stage of Epstein-Barr virus lytic infection leading to the "starry sky" pattern formation in endemic Burkitt lymphoma. Arch Pathol Lab Med 2004; 128: 549–52.
44. Xue SA, Labrecque LG, Lu QL. Promiscuous expression of Epstein-Barr virus genes in Burkitt's lymphoma from the central African country Malawi. Int J Cancer 2002; 99: 635–43.
45. Xia T, O'Hara A, Araujo I. EBV microRNAs in primary lymphomas and targeting of CXCL-11 by ebv-mir-BHRF1-3.Cancer Res 2008; 68: 1436–42.
46. Kelly G, Bell A, Rickinson A. Epstein-Barr virus-associated Burkitt lymphomagenesis selects for downregulation of the nuclear antigen EBNA2. Nat Med 2002; 8: 1098–104.
47. Kelly GL, Milner AE, Tierney RJ. Epstein-Barr virus nuclear antigen 2 (EBNA2) gene deletion is consistently linked with EBNA3A, -3B, and -3C expression in Burkitt's lymphoma cells and with increased resistance to apoptosis. J Virol 2005;79: 10709–17.
48. Rowe M, Kelly GL, Bell AI, Rickinson AB. Burkitt's lymphoma:the Rosetta Stone deciphering Epstein-Barr virus biology.Semin Cancer Biol 2009; 19: 377–88.
49. Paschos K, Smith P, Anderton E, Middeldorp JM, White RE,Allday MJ. Epstein-barr virus latency in B cells leads to epigenetic repression and CpG methylation of the tumour suppressor gene Bim. PLoS Pathog 2009; 5: e1000492.
50. Kamranvar SA, Gruhne B, Szeles A, Masucci MG. Epstein-Barr virus promotes genomic instability in Burkitt's lymphoma. Oncogene 2007; 26: 5115–23.
51. Kuppers R. B cells under influence: transformation of B cells by Epstein-Barr virus. Nat Rev Immunol 2003; 3: 801–12.
52. Thorley-Lawson DA, Gross A. Persistence of the Epstein-Barr virus and the origins of associated lymphomas. N Engl J Med 2004;350: 1328–37.

53. Hochberg D, Middeldorp JM, Catalina M, Sullivan JL, Luzuriaga K, Thorley-Lawson DA. Demonstration of the Burkitt's lymphoma Epstein-Barr virus phenotype in dividing latently infected memory cells in vivo. Proc Natl Acad Sci USA 2004; 101: 239–44.
54. Bellan C, Lazzi S, Hummel M. Immunoglobulin gene analysis reveals 2 distinct cells of origin for EBV-positive and EBV-negative Burkitt lymphomas. Blood 2005; 106: 1031–36.
55. Burkitt D, Wright D. Geographical and tribal distribution of the African lymphoma in Uganda. BMJ 1966; 1: 569–73.
56. Morrow RH. Burkitt's lymphoma. In: Schottenfield D, Fraumeni JF, eds. Cancer epidemiology and prevention. Philadelphia: WB Saunders, 1982: 779–94.
57. Carpenter LM, Newton R, Casabonne D. Antibodies against malaria and Epstein-Barr virus in childhood Burkitt lymphoma: a case-control study in Uganda. Int J Cancer 2008; 122: 1319–23.
58. Mutalima N, Molyneux E, Jaffe H. Associations between Burkitt lymphoma among children in Malawi and infection with HIV, EBV and malaria: results from a case-control study. PLoS One 2008; 3: e2505.
59. Chene A, Donati D, Orem J. Endemic Burkitt's lymphoma as a polymicrobial disease: new insights on the interaction between Plasmodium falciparum and Epstein-Barr virus. Semin Cancer Biol 2009; 19: 411–20.
60. Njie R, Bell AI, Jia H. The effects of acute malaria on Epstein-Barr virus (EBV) load and EBV-specific T cell immunity in Gambian children. J Infect Dis 2009; 199: 31–38.
61. Moormann AM, Chelimo K, Sumba OP. Exposure to holoendemic malaria results in elevated Epstein-Barr virus loads in children. J Infect Dis 2005; 191: 1233–38.
62. Donati D, Espmark E, Kironde F. Clearance of circulating Epstein-Barr virus DNA in children with acute malaria after antimalaria treatment. J Infect Dis 2006; 193: 971–77.
63. Rasti N, Falk KI, Donati D. Circulating Epstein-Barr virus in children living in malaria-endemic areas. Scand J Immunol 2005; 61: 461–65.
64. Yone CL, Kube D, Kremsner PG, Luty AJ. Persistent Epstein-Barr viral reactivation in young African children with a history of severe Plasmodium falciparum malaria. Trans R Soc Trop Med Hyg 2006; 100: 669–76.

65. Lam KM, Syed N, Whittle H, Crawford DH. Circulating Epstein-Barr virus-carrying B cells in acute malaria. Lancet 1991; 337: 876–78.
66. Donati D, Zhang LP, Chene A. Identification of a polyclonal B-cell activator in Plasmodium falciparum. Infect Immun 2004; 72: 5412–18.
67. Parroche P, Lauw FN, Goutagny N. Malaria hemozoin is immunologically inert but radically enhances innate responses by presenting malaria DNA to toll-like receptor 9. Proc Natl Acad Sci USA 2007; 104: 1919–24.
68. Capolunghi F, Cascioli S, Giorda E. CpG drives human transitional B cells to terminal differentiation and production of natural antibodies. J Immunol 2008; 180: 800–08.
69. Potup P, Kumsiri R, Kano S. Blood stage Plasmodium falciparum antigens induce immunoglobulin class switching in human enriched B cell culture. Southeast Asian J Trop Med Publ Health 2009; 40: 651–64.
70. Ramiro AR, Jankovic M, Callen E. Role of genomic instability and p53 in AID-induced c-myc-Igh translocations. Nature 2006; 440: 105–09.
71. Whittle HC, Brown J, Marsh K. T-cell control of Epstein-Barr virus-infected B cells is lost during P. falciparum malaria. Nature 1984; 312: 449–50.
72. Grulich AE, Wan X, Law MG. B-cell stimulation and prolonged immune deficiency are risk factors for non-Hodgkin's lymphoma in people with AIDS. AIDS 2000; 14: 133–40.
73. Breen EC, Fatahi S, Epeldegui M, Boscardin WJ, Detels R, Martinez-Maza O. Elevated serum soluble CD30 precedes the development of AIDS-associated non-Hodgkin's B cell lymphoma. Tumour Biol 2006; 27: 187–94.
74. Yawetz S, Cumberland WG, van der Meyden M, Martinez-Maza O. Elevated serum levels of soluble CD23 (sCD23) precede the appearance ofacquired immunodeficiency syndrome-associated non-Hodgkin's lymphoma. Blood 1995; 85: 1843
75. Engels EA, Pfeiffer RM, Landgren O, Moore RD. Immunologic and virologic predictors of AIDS-related non-hodgkin lymphoma in the highly active antiretroviral therapy era. J Acquir Immune Defic Syndr 2010; 54: 78–84.
76. Zoufaly A, Stellbrink HJ, Heiden MA. Cumulative HIV viremia during highly active antiretroviral therapy is a strong predictor of AIDS-related lymphoma. J Infect Dis 2009; 200: 79–87.

77. Epeldegui M, Breen EC, Hung YP, Boscardin WJ, Detels R, Martinez-Maza O. Elevated expression of activation induced cytidine deaminase in peripheral blood mononuclear cells precedes AIDS-NHL diagnosis. AIDS 2007; 21: 2265–70.
78. Sinfield RL, Molyneux EM, Banda K. Spectrum and presentation of pediatric malignancies in the HIV era: experience from Blantyre, Malawi, 1998–2003. Pediatr Blood Cancer 2007; 48: 515–20.
79. Orem J, Maganda A, Mbidde EK, Weiderpass E. Clinical characteristics and outcome of children with Burkitt lymphoma in Uganda according to HIV infection. Pediatr Blood Cancer 2009;52: 455–58.
80. Lucas SB, Diomande M, Hounnou A. HIV-associated lymphoma in Africa: an autopsy study in Côte d'Ivoire. Int J Cancer 1994; 59: 20–24.
81. Chintu C, Athale UH, Patil PS. Childhood cancers in Zambia before and after the HIV epidemic. Arch Dis Child 1995;73: 100–04; discussion 104–5.
82. Mutalima N, Molyneux EM, Johnston WT. Impact of infection with human immunodeficiency virus-1 (HIV) on the risk of cancer among children in Malawi—preliminary findings. Infect Agent Cancer 2010; 5: 5.
83. van den Bosch CA. Is endemic Burkitt's lymphoma an alliance between three infections and a tumour promoter? Lancet Oncol 2004; 5: 738–46.
84. Aya T, Kinoshita T, Imai S. Chromosome translocation and c-MYC activation by Epstein-Barr virus and Euphorbia tirucalli in B lymphocytes. Lancet 1991; 337: 1190.
85. MacNeil A, Sumba OP, Lutzke ML, Moormann A, Rochford R. Activation of the Epstein-Barr virus lytic cycle by the latex of the plant Euphorbia tirucalli. Br J Cancer 2003; 88: 1566–69.
86. Patte C, Philip T, Rodary C. High survival rate in advanced-stage B-cell lymphomas and leukemias without CNS involvement with a short intensive polychemotherapy: results from the French Pediatric Oncology Society of a randomized trial of 216 children. J Clin Oncol 1991; 9: 123–32.
87. Patte C, Auperin A, Michon J. The Societe Francaise d'Oncologie Pediatrique LMB89 protocol: highly effective multiagent chemotherapy tailored to the tumor burden and initial response in 561 unselected children with B-cell lymphomas and L3 leukemia. Blood 2001; 97: 3370–79.
88. Hesseling P, Molyneux E, Kamiza S, Israels T, Broadhead R. Endemic Burkitt lymphoma: a 28-day treatment schedule with cyclophosphamide and intrathecal methotrexate. Ann Trop Paediatr 2009; 29: 29–34.

89. Magrath IT. African Burkitt's lymphoma. History, biology, clinical features, and treatment. Am J Pediatr Hematol Oncol 1991; 13: 222–46.
90. Israels T, van de Wetering MD, Hesseling P, van Geloven N, Caron HN, Molyneux EM. Malnutrition and neutropenia in children treated for Burkitt lymphoma in Malawi. Pediatr Blood Cancer 2009; 53: 47–52.
91. Jaffe ES, Harris NL, Stein H, Vardiman JW. World Health Organization classification of tumours: pathology and genetics of tumours of haematopoietic and lymphoid tissues. Lyon: IARC Press, 2001.
92. Swerdlow SH, Campo E, Harris NL. WHO classification of tumours of haematopoietic and lymphoid tissues, 4th edn. Lyon: IARC Press, 2008.
93. Windebank K. Lymphoma. In: Bailey S, Skinner R. Paediatric haematology and oncology. Oxford: Oxford University Press, 2009 443–50.
94. Cairo MS, Krailo MD, and Morse M. Long-term follow-up ofshort intensive multiagent chemotherapy without high-dose methotrexate ('Orange') in children with advanced non-lymphoblastic non-Hodgkin's lymphoma: a children's cancer group report. Leukemia 2002; 16: 594–600.
95. Murphy SB. Classification, staging and end results of treatmentof childhood non-Hodgkin's lymphomas: dissimilarities from lymphomas in adults. Semin Oncol 1980; 7: 332–39.
96. Zeng W, Lechowicz MJ, Winton E, Cho SM, Galt JR, Halkar R. Spectrum of FDG PET/CT findings in Burkitt lymphoma.Clin Nucl Med 2009; 34: 355–58.
97. Szczepanski T, Willemse MJ, van Wering ER, van Weerden JF, Kamps WA, van Dongen JJ. Precursor-B-ALL with D(H)-J(H) gene rearrangements have an immature immunogenotype with a high frequency of oligoclonality and hyperdiploidy of chromosome 14. Leukemia 2001; 15: 1415–23.
98. Van Dongen JJ, Langerak AW, Bruggemann M. Design and standardization of PCR primers and protocols for detection of clonal immunoglobulin and T-cell receptor gene recombinations in suspect lymphoproliferations: report of the BIOMED-2 Concerted Action BMH4-CT98-3936. Leukemia 2003; 17: 2257–317.
100. Agsalda M, Kusao I, Troelstrup D, Shiramizu B. Screening for Residual Disease in Pediatric Burkitt Lymphoma Using Consensus Primer Pools. Adv Hematol 2009; 2009: 412163.

101.Klapper W, Szczepanowski M, Burkhardt B. Molecular profiling of pediatric mature B-cell lymphoma treated in population-based prospective clinical trials. Blood 2008; 112: 1374–81.

102.Griffin TC, Weitzman S, Weinstein H. A study of rituximab and ifosfamide, carboplatin, and etoposide chemotherapy in children with recurrent/refractory B-cell (CD20+) non-Hodgkin lymphoma and mature B-cell acute lymphoblastic leukemia: a report from the Children's Oncology Group. Pediatr Blood Cancer 2009; 52: 177–81.

103. Harif M, Barsaoui S, Benchekroun S. Treatment of B-cell lymphoma with LMB modified protocols in Africa—report of the French-African Pediatric Oncology Group (GFAOP).Pediatr Blood Cancer 2008; 50: 1138–42.

104.El Kababri MMF, Loumatine K, Harif M. Treatment of childhood Burkitt lymphoma in North Africa: a study of the French-African Pediatric Oncology Group (GFAOP). Hematol Meet Rep 2009; 3: 88.

105. Klumb CE, Apa AG, Kadma Carrico M. Long-term outcome of children with B-non-Hodgkin's lymphoma: results from Brazilian National Cancer Institute. Hematology Meeting Reports 2009; 3: 15.

106.Chantada GL Zubizaretta P, Felice MS. Results of a modified BFM strategy for the treatment of B-cell malignancies in Argentina. Hematol Meet Rep 2009; 3: 26.

107.Patte C, Auperin A, Michon. The Société Française d'Oncologie Pédiatrique LMB89 protocol: highly effective multiagent chemotherapy tailored to the tumor burden and initial response in 561 unselected children with B-cell lymphomas andL3 leukemia. Blood 2001; 97: 3370–79.

108.Hesseling P, Broadhead R, Mansvelt. The 2000 Burkitt lymphoma trial in Malawi. Pediatr Blood Cancer 2005; 44: 245–50. Arora RS, Eden T, Pizer B. The problem of treatment abandonment in children from developing countries with cancer.Pediatr Blood Cancer 2007; 49: 941–46.

109.Israels T, Chirambo C, Caron H, de Kraker J, Molyneux E, Reis R. The guardians' perspective on paediatric cancer treatment in Malawi and factors affecting adherence. Pediatr Blood Cancer 2008; 51: 639–42.

110. Meremikwu MM, Ehiri JE, Nkanga DG, Udoh EE, Ikpatt OF,Alaje EO. Socioeconomic constraints to effective management of Burkitt's lymphoma in south-eastern Nigeria. Trop Med Int Health 2005; 10: 92–98.

111. Hesseling PB, Molyneux E, Kamiza S, Broadhead R. Rescue chemotherapy for patients with resistant or relapsed endemic Burkitt’s lymphoma. Trans R Soc Trop Med Hyg 2008; 102: 602–07.
112. Adde M, Shad A, Venzon D. Additional chemotherapy agents improve treatment outcome for children and adults with advanced B-cell lymphomas. Semin Oncol 1998; 25: 33–39; discussion 45–48.
113. Nkrumah FK, Perkins IV. Burkitt’s lymphoma: a clinical study of 110 patients. Cancer 1976; 37: 671–76

Carcinogenesisin Squamous Cell Carcinoma of Head and Neck (SCCHN) by Evasion of Apoptosis- A Review of Literature

Hemani.K[1],Dhanalakshmi Ravikumar[2],
Graduate Student[1],Senior Lecturer[2],Department of Pedodontics and Preventive Dentistry,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, Iindia

Abstract

Apoptosis or a programmed cell death, is a physiological orchestration of molecular mechanism that is triggered by a noxious stimuli that has resulted in cellular and DNA damage. It is a mechanism to retain the integrity of the genome. It is regulated by proapoptotic and antiapoptotic molecules. Evasion of apoptosis ,one of the hallmarks of cancer results in abnormal growth of a mutated cell. The most intensely studied gene involved in carcinogenesis is a tumour suppressor gene TP53. Mutation in the TP53 gene can result in dysregulation of apoptosis which leads to genomic instability and thereby carcinogenesis.

This review aims to highlight the role of apoptosis in normal health ,and the molecular mechanisms of carcinogenesis by evasion of apoptosis , of Squamous cell carcinoma of head and neck (SCCHN).

To analyse the various cellular and molecular mechanismsthat interfere with the normal function of tumour suppressor gene- TP53 causing dysregulation and DNA mutation mediated evasion of apoptosis, a hallmark of cancer.

This review, will focus on Apoptosis and carcinogenesis of squamous cell carcinoma of the head and neck (SCCHN) by evasion of apoptosis . By determining these molecular pathways, basis for modulation in gene therapy for advanced and refractory SCCHN can be established

KeyWords: *Apoptosis, Squamous cell carcinoma of head and neck ,SCCHN, p53, TP53*

Introduction

Head and neck squamous cell carcinoma (HNSCC) a malignancy that presents in the head and neck region commonly involves the oral cavity, pharynx and larynx. It is by far the commonest subtype of cancer, and is the sixth most common site(1). The main risk factorsattributed are smoking , alcohol , and tobacco or a combination . Infection with the human papilloma virus (HPV) has emerged as a major risk factor, The associated complex genetic mutations complicates the management , rendering very low survival rates. The many molecular mechanisms caused by genetic and epigenetic factors, associated with malignancy have been reviewed by Park(2) and updated thereafter as hallmarks of cancer, involved in HNSCC .

In 2000, Hanahan and Weinberg (3)described six characteristics, necessary for a malignanttumour to develop as hallmarks of cancer,. These are (a) acquisition of sustained proliferative signalling (b) evasion of growth suppressor signals (c) resistance to cell death (evasion of apoptosis)(d) replicative immortality (immortalisation) (e) induction of angiogenesis and (f) activation of tissue invasion and metastasis. A later update in 2011 (4) added two emerging hallmarks(g) deregulation of cellular energy metabolism, and (h) evasion of immune destruction.The last two were classified as emerging hallmarks, as they may or may not prevail in all cancers.Twoenabling characteristics, genome instability and mutation,and tumour-promoting inflammation were then defined which could predispose to carcinogenesis.This review, will focus on Apoptosis and carcinogenesis of squamous cell carcinoma of the head and neck (SCCHN) by evasion of apoptosis.

Apoptosis

Apoptosis is a programmed cell death mechanism carried out by the cell’s own metabolism to establish development and homeostasis in metazoans(5,6). A cell that is subjected to a DNA damage,hypoxia or any other cellular abnormalities, activates this process through various pathways to retain its genetic stability(7). There is a perenial threat posed to the genetic stability in the form of and external and internal environmental pollutants ,irritants, like pollution ,food,chronic trauma ,stress, that act as cytotoxins, which trigger a sequence of reactions towards genotoxicity affecting a host of cells every day of life. In the attempt to challenge the insults, the body's innate defence mechanisms operates by initating a sequence of reactions for repair, but persistance of abberent stimuli results insenescence, or apoptosis to maintain integrity of the genome. The repair when it fails,orifabberant, results in genetic

mutations, and further cell proliferations with the mutant gene, needs to be arrested or expelled.

Ireversible cell cycle arrest or cellular senescence, of mutant cells acts as a protective barrier against any further proliferation of mutant cells, and is therefore a potent tumour suppressant providing a defence against malignant transformation of pre malignant leisons(8,9,10)

Elimination is performed by two major mechanisms necrosis and, apoptosis,Autophagy,lysosomal cell death, mitotic catastrophy(11,12) are the more recent discoveries in the mode of cell death . Necrosis involves plasmatic rupture and cell death associated with inflammation(13). Apoptosis happens by fragmentation of DNA that is phagocytosed by macrophages with no associated inflammation(14). Induction of apoptosos is either by promotion of proapoptoticfactors ,orsupression of anti proapoptotic factors in tumour cells or by infection of viral particles targeting specific transformed cells(15)

Default orretardation or delay in the initiation of apoptosis, retains the mutant gene in the body , and proliferation of such cells results ina tumour. The stimulus that initiated it is considered as a potent carcinogen.Preserving and conserving normal genetic material is therefore necessary forexistance and normal reproduction of life in this planet.

Defects in apoptosis may cause cancer or autoimmunity and enhanced apoptosis may lead to degenerative diseases(16), immunodeficiencies and infertility. Cancer cells inhibit apoptosis mainly by ignoring signals from extrinsic pathway and resetting the balance of intercellular pro and anti apoptotic molecules. This implies that apoptotic signals help in maintaining genomic integrity ,but dysregulation of apoptotic mechanism may lead to cancer development and also makes the cells resistant to cancer treatment (17). This evidently proves that evasion of apoptosis is a prominent hallmark of cancer (18).

Apoptosisor programmed cell death is initiated by three major mechanisms . An intrinsic mechanism which is receptor triggeredandinitated by ligation to death receptors inducing caspase-8 activation.Thesecond,an intrinsicormitochondrial pathway , that is triggered by cellular stress leading to activation of Caspase -9. The third is the granzyme B pathway where the cytotoxic cell proteases granzyme B is delivered to target cells . On a molecular basis all mechanisnms lead to caspase activation and DNA fragmentation by proteolytic activation of Caspase 3 , 7 from their inactive zymogens. Mitochondria plays an important role by releasing proapoptotic proteins into the cytosol in response to diverse stimuli which is regulated by proteins of Bcl-2 family (18)

TP53, a critical DDR(DNA damage response) gene is a known tumor suppressor gene, that encodes the p53 protein. p53 is considered to play a critical role in programming apoptosis to prevent cancer (19, 20). In 50% of cancers p53 is inactivated by mutations(21).Studies reveal that itmediates both cell autonomous and non cell autonomous DDRtoalleviate progress of cancer This review aims to explain the apoptotic pathways and how evasion of apoptosis promotes carcinogenesis in SCCHN.

Apoptotic Pathways

Extrinsic pathway-

Tumor Necrosis Factor (TNF) family receptors contain two major death domains (DD) containing receptors that use caspase activating signaling mechanisms , they are Fas (fibroblast associated antigen also known as Apo-1/CD95) and tumor necrosis factor receptor (TNF-R).Others includeDR3/Wsl-1/Tramp, DR4/TRAIL-R1, DR5/TRAIL-R2/TRICK2/Killer and DR6(22). When the death receptors are ligated it induces the formation of Death Inducing Signaling Complex (DISC), where the adaptor FADD/ TRADD binds to the death domain and induces the pathways that activate caspase 8 and 10. The activated caspase 8 cleaves procaspase 3 and leads to apoptosis. It also cleaves BH 3, the only proapoptotic protein that contains Bid and forms truncated Bid(tBid) which induces cytochrome c release from mitochondria to activate caspase 9 and 3 to start apoptosis(18) .

Intrinsic Pathway

Intrinsic pathway or mitochondrial pathway involves Bcl-2 family members. It contains anti apoptotic members(Bcl-2, Bcl XL) and pro apoptotic members (Bax, Bak). BH3 is the only protein that is pro apoptotic , it contains Bim and Bid to help in the actions of Bak/ Bax. When extra or intra cellular stresses activate the pathway, Bak/ Bax act on mitochondria through BH3 proteins and cause release of cytochrome into cytoskeleton. By homo oligomerisation of Bim and Bid , cytochrome c activates Apaf-1 to activate pro caspase 9 which in turn activates caspase 3,6,9 and causes apoptosis (23). Smac and Omi proteins neutralise the inhibitory effect of IAPs (inhibitor of apoptosis proteins)(24,25).

This pathway also includes the action of cytotoxic T cells and Natural killer cells (NK cells). They activate granzyme B (Gra B) which cleaves several caspase and leads to the activation of endonuclease and catabolism of cytoskeleton and it is followed by apoptosis.(26).

TP53-Tumor Suppressor Gene

TP53 is an important cell cycle regulator that plays a central role in the regulation of the cell cycle arrest at G1 phase and promotes cell death by activating apoptosis by various parallel pathways (27) that may or may not depend on transcriptional events to prevent damaged DNA from being passed on to daughter cells. Tumor cells inhibit apoptosis by dysregulating a key component that is important for upstream and downstream regulation of p53 mechanism for apoptosis. Wild type p53 tumours inactivate positive regulators of p53 such as p14ARF (28) or over activation of negative regulators of p53 activity such as Akt (29).

p53 Pathway for Apoptosis

The p53 gene was localized to chromosome 17p13 spanning 20 kb, consisting of 11 exons and encoding a 2.9-kb mRNA. The p53 protein contains 393 amino acids, is localized to the nucleus, and is expressed in all cells. Most mutations in the p53 gene are missense mutations occurring in the highly conserved central domain of the protein .p53 which is known as the 'guardian of the genome' follows a specific pathway to bind to DNA but tumor derived mutants lack this mechanism, thus disabling this mechanism causes tumorigenesis (30) . p53 senses a stress and activates the pathways through its transactivational capabilities. When a stress is felt, proteins like Ataxia Telangiectasia Mutated (ATM) and Chk2 phosphorylates p53 (31) . Similarly the oncogenes in mitochondria also activate p53 through p19ARF by inhibiting the action of Mdm2 mediated ubiquitination of p53 (32). p53 binds to Mdm2 gene and stimulates transcription of this gene into messenger RNA (mRNA) to translate it into protein (19). The level of p53 rise when Mdm2 binds to p53 and reduces transcription of Mdm2 gene and closes the feedback loop. The activated p53 then activates proapoptotic members (Bak/Bax) and suppresses the antiapoptotic members(Bcl-2,Bcl- XL) of Bcl2 family. The BH3 contains activates (Bim,tBid) that induce mitochondrial outer membrane permeability and sensitisers or derepressors (Bad,Bmf,Noxa,Puma) that neutralises antiapoptotic proteins . These proteins bind with Bax/Bak to act on mitochondria to start apoptosis (26).

As expression of both Bcl-2 and Bax is regulated by p53tumor suppressor gene(33),human B cell lymphoma has over expression of Bcl-2 (34) which proves failure of cell death which contributes to cancer. In mitochondria it transactivates the apoptotic effector mechanism and

induces Apaf-1 which is a co activator of caspase 9 and starts the caspase cascade. p53 being an up regulator of caspase 6, it creates chemo sensitivity (35) in cells and starts apoptosis.
Previous studies in cell lines,(36) revealed that introduction of p53 intohead and neck cancer cell lines induced growtharrest and apoptosis.Crowe(37) demonstrated that head andneck SCC lines express bcl2, bak, and bid but notbax and bad.(38)p53 inhibited bcl2 expression by twofold tothreefold in both SCC25 and SCC9 stable clones.p53 expression had no effect on bak and bid levels, nor did bax affect expression of any bcl2-related proteinA study(37) using wild-type cell linesp53 expression to three head and neck squamouscell carcinoma (SCC) lines containing well-characterizedmutations in this gene.(39) demonstrated that p53 did not induce growth arrest or celldeath in these lines but increased levels of DNAdamage–induced apoptosis that was dependenton bcl2 but not baxexpressionWild-type p53 wastranscriptionally active and inhibited cellularproliferation by slowing the G1 to S phase transition.suggestingthat p53 expression decreased the number of cellsentering S phase from G1.

In extrinsic pathway it sensitises death receptor ligand Fas/TNFR and induces apoptosis directly or enhanced cell death. Bid may facilitate crosstalk between extrinsic and intrinsic pathway (40).
p53 also transactivates other genes like PERP , p53AIP1 and genes that increase reactive oxygen species (ROS) (41).p53 can transactivate genes that short circuit antiapoptotic pathways, this is prominent in its ability to regulate PTEN, thus changes in PTEN can compromise p53 mediated apoptosis in some cell types (42). TP53 can produce changes in REDOX metabolism and increase the number of reactive oxygen species (ROS) and antioxidants which suppress p53 mediated cell death (30).
Carcinogenesisis induced by failure of the above mentioned pathways and other machanisms that are beyond the scope of this review article.Many molecular mechanisms are adopted bytumorcells to suppress apoptosis. Tumor cells can acquireresistance to apoptosis by the expression of antiapoptoticproteins such as Bcl-2 or by the downregulation or mutationofproapoptotic proteins such as BAX. Since the expressionof both Bcl-2 and BAX is regulated by the p53 tumorsuppressorgene(27)

Mechanisms of the Hallmarksof Cancer

Acquisition of sustained proliferative signalling - EGFR (epidermal growth factor receptor) is a key growth factor receptor involved in manycellular pathways that regulate growth and cell survival.EGFR is overexpressed in up to 90 % of HNSCC(43), andhigh expression of EGFR is associated with poor clinicalprognosis(44). Transforming growth factor-a (TGF-a), anEGFR ligand, is also overexpressed in cases of HNSCC,resulting in autocrine signalling (43). Radiation increases theexpression of EGFR in cancer cells, and blockade of EGFRsignalling sensitizes cells to the effects of radiation.

In addition to genetic mutations alterations in genetic control mechanisms by epigenetics should also be considered .The epigenetic processes include DNA methylation, histone acetylation, phosphorylation, ubiquitylation, and sumoylation. Such epigenetic changes can facilitate hallmark acquisition through altering the activity of normal genes.

The role of increased gene promoter methylation in HNSCC has been reported for several genes including CDKN2A, RARß2 and CDH1 (45). Silencing of RARß2 by promoter hypermethylation is an early event in head and neck carcinogenesis(46).

Hypermethylationcan also silence micro RNAs (miRNAs) with tumour suppressor functions,as a decrease in the levels of hsa-miR-205 and hsa-let-7d expression is a strong indicator of poor survival in patients with HNSCC (47). Target of hsa-let-7d is KRAS, which is asmallGTPase downstream of many growth factor receptors, whose overexpression could lead to sustained proliferativesignalling. KRAS gene is overexpressed in45 % of HNSCC (48).

Evasion of growth suppressor signals - The most important tumour suppressor protein is p53.p53 associates with the E3 ubiquitin ligaseMDM2, which targets p53 for proteolytic degradation, allowing it to accumulate in the cell.The TP53 gene is mutated in about 60 % of allHNSCC resulting in loss of tumour suppressor function(49). In the remaining 40 % of tumours, TP53 function isinhibited by an interaction with certain proteins.The E6oncoprotein produced by HPV-16 binds p53 and targets itfor degradation in about 20 % of HNSCC(50). HPV is amajor risk factor for squamous cell carcinoma of theoropharynx.

Retinoblastoma protein (Rb) is another major tumoursuppressor protein.Rb suppresses progression through the cell cycle by binding toand inhibiting the E2F transcription factor(51).The HPV oncoprotein E7 also affects thispathway by binding Rb and targeting it for degradation.Tp53 is not mutated in HPV-associated HNSCC expressingE7

Resistance to cell death - p53 is a key proteininvolved in the onset of apoptosis (36). It can cause thetranscription of the pro-apoptotic proteins Noxa, Puma andAip1 and repress the transcription of the anti-apoptoticBcl-2 protein.EGFR signalling affects not only sustained proliferationbut also evasion of apoptosis through its downstreameffector STAT3. STAT3 is a transcription factor with manyroles, including suppression of apoptosis. An increase inEGFRsignalling thereforecan inhibit apoptosis(52,53).

Phosphoinositide 3-kinase (PI3K), another downstreameffector of EGFR, is also commonly overexpressed inHNSCC. Presence of PIP3 in the plasma membrane results inrecruitment of PDK1 which recruits AKT(54). AKTphosphorylates many target proteins to promote cell survival. PI3K is overexpressed in 37 % of HNSCC.

Replicative immortality - In humans, telomerase is active inthe embryo but is lost later in life. Many cancersreactivate telomerase and so become immortal. This is thecase in HNSCC, where telomerase activation is seen in78 % of cancers, 85 % of precancerous tissue, and 53 % ofadjacent normal tissue (55).

Induction of angiogenesis- As the tumour increases in size , the prevailing hypoxia induces angiogenesis. Hypoxia causes increased production of proangiogenic factors, such as vascular endothelial growthfactor (VEGF), platelet-derived growth factor (PDGF),fibroblast growth factors (FGF 1 and 2) and interleukin(IL-8). andsupression of anti-angiogenicfactors such as interferons(56). VEGF is a pro-angiogenicfactor that binds receptors on endothelial cells, triggeringtheir proliferation and migration to facilitate angiogenesis.High VEGF expression has been documented in HNSCC(57). Expression of VEGF or its receptors is increased in50 % of premalignant and 75 % of malignant oral andlaryngeallesions(58)

Tissue Invasion and Metastasis

The tumour undergoes dysmorphic changes and invades the adjacent tissues, undergo epithelial to mesenchymal transition. E-cadherin and the cateninsa and b form a key protein complex at adherens junctions which link neighbouring cells. E-cadherin and the catenins are downregulated in 59% and 72 % respectivelyof HNSCC, and the downregulation is associated with lymphnode metastases(59).

SPARC is a glycoprotein associated with the ECM. It can increase cell permeability bycausing the production of enzymes which break down themembrane(60). This can facilitate tumour extravasationwhere cells migrate from the blood stream through

thevascularendothelium and into the tissue(61). In this way,SPARC overexpression may facilitate metastasis. SPARC/osteonectinare genetic markers for SCCHN

Reprogramming energy metabolism

Abnormality in tissue metabolism is an emerging hallmark of cancer. Alteration in oxygen consumption and altered aerobic glycolysis have in recent time been identified using nuclear magnetic resonance studies (62). Increased levels of lactate (awaste product of glycolysis) and monocarboxylate transporters (proteins for lactate export) were found in HNSCC cells.

Evading immune destruction - The immune response to HNSCC is also impaired by theapoptosis of CD8, lymphocytes at the tumour site, andreactive lymphocytes are also depleted in the circulation(63).

Enabling Characteristics

Genomic instability- TP53 mutations can increase genomic instability.An important function of p53 is in the DNA damageresponse, which is activated by the presence of damage andarrests the cell cycle to allow time for repair. If this processis disrupted, more mutations can be tolerated. A TP53mutation could also facilitate immortalization.(52)

The importance of functional p53 as a barrier to cancer is reflected in the progression to HNSCC, mutant TP53 was found in 29 % of hyperplastic lesions,46 % of dysplastic lesions, and 58 % of HNSCC(40,41).Thepresence of a TP53 mutation in a premalignant lesion is a very strong indicator of progression to carcinoma and has apositive predictive value of 86 % (64).

Tumour-Promoting Inflammation

Tumours normally invaded by a high density of immune cells including granulocytes, monocyte-macrophages, mast cells, dendritic cells, lymphocytes and fibroblasts, by chemokines(65,66). These immune cells enhance progression of tumour development with factors that facilitate acquisition of the hallmarks of cancer, such as growth factors and angiogenic factors.

Inflammatory cells also release reactiveoxygen species, which are highly mutagenic and canincrease genomic instability (67). COX-2, an enzymeinvolved in the production of inflammatory prostaglandins,and maintenance of blood flow, is overexpressed in 71 %of HNSCC, where it is associated with VEGF productionand perhaps with more aggressive

disease with lymph nodeinvolvement(68). Chronic infection in the oral cavity(periodontitis) is associated with more aggressive oralcancer, and bad dental hygiene may contribute to theassociation of cigarette smoking with HNSCC (69).HNSCC has a role in inflammation, because it expressesToll-like receptors (TLRs), which are a part of the ancient innate immune system. TLRs are usually expressed oninflammatory cells guarding sites such as mucosal membranes, where they trigger local inflammation in responseto binding particular molecules from pathogens such aslipopolysaccharide and double-strand RNA. TLRs onHNSCC probably enhance inflammation triggered bybacteria associated with the tumour. This may have someprotective function but at the same time can promotetumour progression by mechanisms such as induction ofproliferation, activation of NF-jB and resistance to naturalkiller lymphocytes (70).

Effect of Therapeutic Drugs on p53

An extensive and thorough understanding of the various molecular mechanisms have contributed largely towards therapeutics for cancer. Therapeutic drugs aim either at inducing proapoptotic proteins or in inhibiting the over expression of antiapoptotic proteins. Thus in case of a mutated p53, the pharmacokinetics involved , is aim at reviving apoptotic pathways in addition to other modes.

Action of 5 Fluorouracil(5 FU)

Fluoropyrimidine 5 Fluorouracil (5 FU) is acommonly used anti cancer drug but its resistance remains a problem. FU targets the tumor suppressor and triggers the cell cycle(26).5FU induced apoptosis is p53 dependent . Apoptosis can occur in p53 mutant cell lines by a mechanism still unknown(71,72).

Action of Doxorubicin on p53

Action of doxorubicin is more active against p53 wild type tumor xenografts than p53 mutant and null cells(73). Doxorubicin induces apoptosis that is dependent on the presence of wild type p53 (74).Negative clinical outcomes due to failure of the drugs to induce apoptosis in some tumor cases are also reported (75).

Action of Herbs

Anticarcinogenic and antioxidant effect of herbs are under study recently. Apoptotic potential of B. monnieri.against oral cancer has been evaluated (76) , but further studies are required to confirm on the role in apoptosis

Conclusion

The updated mechanisms that explains the hallmarks of cancer are a useful platform to understand the underlying molecular and cellular processes involved in the development of HNSCC. There may be more than one mechanism acting in each hallmark process. Some established molecular mechanisms such as mutation of TP53 and overactivity of EGFR are so frequent in HNSCC as to appear almost inevitable in carcinogenesis. The study of these innumerable and complex mutations could lead to better prognostic markers and the personalisation of therapy according to the characteristics of the particular tumour. The hope is that molecular-targeted therapies will play an increasingly successful role in the management of head and neck cancer

Corresponding Author

Dhanalakshmi Ravikumar,

Senior Lecturer,Department Of Pedodontics and Preventive Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077, Tamil Nadu, India.

Email id: dhana9677@gmail.com

Phone number - +91 9597011442

References

1. Rezende TM, de Souza Freire M, Franco OL ,Headandneck cancer: proteomic advances and biomarker achievements.Cancer, 2010; 116:4914–4925.
2. Park BJ, ChioseaSI, Grandis JR, Molecular changes in themultistage pathogenesis of head and neck cancer. Cancer Biomarker, 2010;9:325–339.
3. Hanahan D, Weinberg RA,The hallmarks of cancer. Cell,2000;100:57–70.

4. Hanahan D, Weinberg RA , Hallmarks of cancer: the nextgeneration. Cell 2011;144:646–674.
5. R. A. Lockshin and C. M. Williams, Programmed cell death Cytology of degeneration in the intersegmental muscles of the Pernyisilkmoth, Journal of Insect Physiology, 1965. 11(2), ;123–133.
6. T. G. Cotter, Apoptosis and cancer: the genesis of a research field, Nature Reviews Cancer, 2009; 9(7): 501–507.
7. S. Negrini, V. G. Gorgoulis, and T. D. Halazonetis, "Genomic instability an evolving hallmark of cancer," Nature Reviews Molecular Cell Biology, 2010; 11(3):220–228.
8. Bartkova J, Rezaei N, Liontos M, Karakaidos P, Kletsas D, Issaeva N, Vassiliou LV, Kolettas E, Niforou K, Zoumpourlis VC, Takaoka M, Nakagawa H, Tort F, Fugger K,Johansson F, Sehested M, Andersen CL, Dyrskjot L, Ørntoft T, Lukas J, Kittas C, Helleday T, Halazonetis TD, Bartek J, Gorgoulis VG. Oncogene-induced senescence is part of the tumorigenesis barrier imposed byDNA damage checkpoints. Nature. 2006; 444:633–637.
9. Di Micco R, Fumagalli M, Cicalese A, Piccinin S, Gasparini P, Luise C, Schurra C, Garre' M, Nuciforo PG, Bensimon A, Maestro R, Pelicci PG, d'AddadiFagagna F. Oncogene-induced senescence is a DNA damage response triggered by DNAhyper-replication. Nature.2006; 444:638–642.
10. Gorgoulis VG, Vassiliou LV, Karakaidos P, Zacharatos P, Kotsinas A, Liloglou T, Venere M, Ditullio RA Jr, Kastrinakis NG, Levy B, Kletsas D, Yoneta A, Herlyn M, Kittas C, Halazonetis TD. Activation of the DNA damage checkpoint and genomic instability in human precancerous lesions. Nature.2005; 434:907–913.
11. H. Okada and T. W. Mak, "Pathways of apoptotic and non-apoptotic death in tumour cells," Nature Reviews Cancer,2004; 4(8) 592–603 .
12. Y. Kondo, T. Kanzawa, R. Sawaya, and S. Kondo, "The role of autophagy in cancer development and response to therapy,"Nature Reviews Cancer,2005; 5(9),726–734.
13. Brown GD. Sensing necrosis with Mincle. Nat Immunol 2008;9:1099–100.
14. Lauber K, Blumenthal SG, Waibel M, Wesselborg S. Clearance of apoptotic cellsgetting rid of the corpses. Mol Cell 2004;14:277–87.
15. Ozben T. Oxidative stress and apoptosis: impact on cancer therapy. J Pharm Sci2007;96:2181–96.

16. Harper JW, Elledge SJ. The DNA damage response: ten years after. Mol Cell. 2007; 28:739–745
17. Ghavami S, Hashemi M, Ande SR, Yeganeh B, Xiao W, Eshraghi M, Bus CJ, Kadkhoda K, Wiechec E, Halayko AJ, Los M. Apoptosis and cancer: mutations within caspase genes. Journal of medical genetics. 2009 Aug 1;46(8):497-510.
18. B. Vogelstein, D. Lane, and A. J. Levine, Surfing the p53 network, Nature2000 ;408(6810) 307–310,
19. Jacks T, Tumor spectrum analysis in p53-mutant mice. Curr Biol. 1994; 4:1–7
20. Petitjean A, Impact of mutant p53 functional properties on TP53 mutation patterns and tumor phenotype: lessons from recent developments in the IARC TP53 database. Hum Mutat. 2007;28:622–629
21. Degterev A, Boyce M, Yuan J. A decade of caspases. Oncogene. 2003 Nov 24;22(53):8543-67.
22. Ghobrial IM, Witzig TE, Adjei AA." Targeting apoptosis pathways in cancer therapy". CA Cancer J Clin 2005;55:178–94.
23. Du C, Fang M, Li Y, Li L, Wang X. Smac, "a mitochodrial protein that promotes cytochrome c-dependent caspase activation by eliminating IAP inhibition". Cell 2000;102:33–42.
24. Li W, Srinivasula SM, Chai J, Li P, Wu JW, Zhang Z, Alnemri ES, Shi Y. "Structural insights into the pro-apoptotic function of mitochondrial serine protease HrtA2/Omi". Nat Strut Biol 2002;9:436–41.
25. Hassan, M.; Watari, H.; AbuAlmaaty, A.; Ohba, Y.; Sakuragi, N. Apoptosis and molecular targeting therapy in cancer. Biomed. Res. Int.2014, 1–23.
26. J. J. Fuster, S. M. Sanz-González, U. M. Moll, and V. Andrés, "Classic and novel roles of p53: prospects for anticancer therapy," Trends in Molecular Medicine, 2007; 13(5): 192–199,
27. T. Kamijo, J. D. Weber, G. Zambetti, F. Zindy, M. F. Roussel, and C. J. Sherr, "Functional and physical interactions of the ARF tumor suppressor with p53 and Mdm2," Proceedings of the National Academy of Sciences of the United States of America, 1998;. 95, (14): 8292–8297.
28. J. Feng, R. Tamaskovic, Z. Yang et al., "Stabilization of Mdm2 via decreased ubiquitination is mediated by protein kinase B/Akt-dependent phosphorylation," Journal of Biological Chemistry2004; 279(34): 35510–35517, .

29. Fridman, J. S. & Lowe, S. W. Control of apoptosis by p53. Oncogene 2003;22: 9030−9040
30. Johnstone RW, Ruefli AA, Lowe SW. Apoptosis: a link between cancer genetics and chemotherapy. Cell, 2002;108: 153–164.
31. Khanna, K.K., and Jackson, S.P. DNA double-strand breaks: signaling, repair and the cancer connections".Nat.Genet. 2001;27,247–254.
32. T. Miyashita, S. Krajewski, M. Krajewska et al., "Tumor suppressor p53 is a regulator of bcl-2 and bax gene expression in vitro and in vivo," Oncogene, 1994; 9(6): 1799–1805.
33. D. L. Vaux, "Immunopathology of apoptosis—introduction and overview," Springer Seminars in Immunopathology, 1998;19(3): 271–278, .
34. Penchala, S.C., Connelly, S., Wang, Y., Park, M.S., Zhao, L., Baranczak, A., Rappley, I., Vogel, H., Liedtke, M., Witteles, R.M., Powers, E.T., Reixach, N., Chan, W.K., Wilson, I.A., Kelly, J.W., Graef, I.A., Alhamadsheh, M.M.AG10 inhibits amyloidogenesis and cellular toxicity of the familial amyloid cardiomyopathy-associated V122I transthyretin.Proc.Natl.Acad.Sci.USA ,2013; 110: 9992-9997
35. Liu TJ, Zhang WW, Taylor DL, Roth JA, Goepfert H, Clayman GL. Growth suppression of human head and neck cancer cells by the introduction of a wild.type p53gene via a recombinant adenovirus. Cancer Res 1994; 54:3662–3667.
36. Crowe DL, Sinha UK. p53 apoptotic response to DNA damage dependent on bcl2 but not bax in head and neck squamous cell carcinoma lines. Head & neck. 2006 Jan 1;28(1):15-23.
37. Johnson SM, Grosshans H, Shingara J, Byrom M, Jarvis R,Cheng A, Labourier E, Reinert KL, Brown D, Slack FJ, RAS is regulated by the let-7 microRNA family. Cell,2005;120:635–647
38. Min BM, Baek JH, Shin KH, Gujuluva CN, CherrickHM,Park NH. Inactivation of the p53 gene by either mutationor HPV infection is extremely frequent in human oralsquamous cell carcinoma cell lines. Eur J Cancer B OralOncol 1994;30B:338–345

39. Sax JK, Fei P, Murphy ME, Bernhard E, Korsmeyer SJ and El-Deiry WS.BID regulation by p53 contributes to chemosensitivity.Nat. Cell Biol. 2002, 4, 842–849.

40. Hwang, P.M., Bunz, F., Yu, J., Rago, C., Chan, T.A., Murphy, M.P., Kelso, G.F., Smith, R.A., Kinzler, K.W., and Vogelstein, B. Ferredoxinreductase affects p53-dependent, 5-fluorouracil-induced apoptosis in colorectal cancer cells. Nat. Med. .2001; 7, 1111–1117.

41. Stambolic V, MacPherson D, Sas D, Lin Y, Snow B, Jang Y, Benchimol S and Mak TW. Regulation of PTEN transcription by p53.Mol. Cell, 2001; 8, 317–325.

42. Grandis JR, Chakraborty A, Zeng Q, Melhem MF, TweardyDJ,Downmodulation of TGF-alpha protein expression withantisense oligonucleotides inhibits proliferation of head and necksquamous carcinoma but not normal mucosal epithelialcells.JCell Biochem,1998;69:55–62

43. Mendelsohn J, Baselga J , Status of epidermal growthfactor receptor antagonists in the biology and treatment of cancer.J ClinOncol,2003; 21:2787–2799

44. Stephen JK, Chen KM, Havard S, Harris G, Worsham MJ ,Promoter methylation in head and neck tumorigenesis. MethodMol Biol,2012; 863:187–206

45. Youssef EM, Lotan D, Issa JP, Wakasa K, Fan YH, Mao L,Hassan K, Feng L, Lee JJ, Lippman SM, Hong WK, LotanR,Hypermethylation of the retinoic acid receptor-beta(2)gene in head and neck carcinogenesis. Clin Cancer Res,2004;10:1733–1742

46. Childs G, Fazzari M, Kung G, Kawachi N, Brandwein-GenslerM, McLemore M, Chen Q, Burk RD, Smith RV, PrystowskyMB,Belbin TJ, SchlechtNF,Low-level expression of microRNAs let-7d and miR-205 are prognostic markers of head andneck squamous cell carcinoma. Am J Pathol ; 2009; 174:736–745.

47. McDonald JS, Jones H, Pavelic ZP, Pavelic LJ, StambrookPJ,Gluckman JL ,Immunohistochemical detection of theH-ras, K-ras, and N-ras oncogenes in squamous cell carcinoma ofthe head and neck. J Oral Pathol Med , 1994;23:342–346.

48. Shin DM, Charuruks N, Lippman SM, Lee JJ, Ro JY, Hong WK,Hittelman WN , p53 protein accumulation and genomicinstability in head and neck multistep tumorigenesis. CancerEpidemiol Biomarker Prev,2001; 10:603–609.

49. Braakhuis BJ, Snijders PJ, Keune WJ, Meijer CJ, Ruijter-Schippers HJ, Leemans CR, BrakenhoffRH,Geneticpatterns in head and neck cancers that contain or lack transcriptionally active human papillomavirus. J Natl Cancer Inst,2004;96:998–1006.

50. Weinberg RA, The retinoblastoma protein and cell cyclecontrol. Cell,1995; 81:323–330.
51. Bernstein JM, Bernstein CR, West CM, Homer JJ. Molecular and cellular processes underlying the hallmarks of head and neck cancer. European archives of oto-rhino-laryngology. 2013;270(10):2585-93.
52. Grandis JR, Drenning SD, Zeng Q, Watkins SC, MelhemMF,Endo S, Johnson DE, Huang L, He Y, Kim JD ,Constitutive activation of Stat3 signaling abrogates apoptosis in squamouscell carcinogenesis in vivo. Proc NatlAcad Sci USA,2000;97:4227–4232.
53. Vanhaesebroeck B, Stephens L, Hawkins P , PI3 K signalling: the path to discovery and understanding. Nat Rev MolCell Biol,2012; 13:195–203.
54. Patel MM, Parekh LJ, Jha FP, Sainger RN, Patel JB, Patel DD,Shah PM, Patel PS, Clinical usefulness of telomeraseactivation and telomere length in head and neck cancer. HeadNeck,2002;24:1060–1067.
55. Bouck N, Stellmach V, Hsu SC How tumors becomeangiogenic. Adv Cancer Res 1996; 69:135–174.
56. Caponigro F, Formato R, Caraglia M, Normanno N, Iaffaioli RV, Monoclonal antibodies targeting epidermal growth factorreceptor and vascular endothelial growth factor with a focus onhead and neck tumors. CurrOpinOncol 2005;17:212–217.
57. Denhart BC, Guidi AJ, Tognazzi K, Dvorak HF, Brown LF, Vascular permeability factor/vascular endothelial growthfactor and its receptors in oral and laryngeal squamous cell carcinoma and dysplasia. Lab Invest,1997; 77:659–664.
58. Tanaka N, Odajima T, Ogi K, Ikeda T, Satoh M,Expression of E-cadherin, alpha-catenin, and beta-catenin in theprocess of lymph node metastasis in oral squamous cell carcinoma. Br J Cancer ,2003;89:557–563.
59. Tremble PM, Lane TF, Sage EH, WerbZ , SPARC, asecreted protein associated with morphogenesis and tissueremodeling, induces expression of metalloproteinases in fibroblasts through a novel extracellular matrix-dependent pathway.J Cell Biol,1993;121:1433–1444.
60. Goldblum SE, Ding X, Funk SE, Sage EH, SPARC(secreted protein acidic and rich in cysteine) regulates endothelialcell shape and barrier function. Proc NatlAcad Sci USA 1994;91:3448–3452.

61. Tripathi P, Kamarajan P, Somashekar BS, Mackinnon N, Chinnaiyan AM, Kapila YL, Rajendiran TM, Ramamoorthy A Delineating metabolic signatures of head and neck squamous cellcarcinoma: Phospholipase A(2), a potential therapeutic target. IntJ Biochem Cell Biol,2012; 44(11):1852–1861.
62. Hoffmann TK, Dworacki G, Tsukihiro T, MeidenbauerN,Gooding W, Johnson JT, Whiteside TL ,Spontaneousapoptosis of circulating T lymphocytes in patients with head andneck cancer and its clinical importance. Clin Cancer Res,2002;8:2553–2562.
63. Cruz IB, Snijders PJ, Meijer CJ, Braakhuis BJ, Snow GB, Waboomers JM, van der Waal I, p53 expression above thebasal cell layer in oral mucosa is an early event of malignanttransformation and has predictive value for developing oralsquamous cell carcinoma. J Pathol,1998; 184:360–368.
64. Dvorak HF,Tumors: wounds that do not heal. Similaritiesbetweentumorstroma generation and wound healing.N Engl JMed, 1986; 315:1650–1659.
65. Prince ME, Ailles LE, Cancer stem cells in head and necksquamous cell cancer. J ClinOncol, 2008; 26:2871–2875.
66. GrivennikovSI, Greten FR, Karin M,Immunity, inflammation, and cancer. Cell ,2010;140:883–899.
67. Gallo O, Masini E, Bianchi B, Bruschini L, Paglierani M, FranchiA,Prognostic significance of cyclooxygenase-2 pathwayand angiogenesis in head and neck squamous cell carcinoma.Hum Pathol,2002; 33:708–714.
68. Tezal M, Sullivan MA, Hyland A, Marshall JR, Stoler D, ReidME, Loree TR, Rigual NR, Merzianu M, Hauck L, LillisC,Wactawski-Wende J, Scannapieco FA, Chronic periodontitis and the incidence of head and neck squamous cellcarcinoma. Cancer Epidemiol Biomarkers Prev,2009;18:2406–2412.
69. Szczepanski MJ, Czystowska M, Szajnik M, HarasymczukM,Boyiadzis M, Kruk-Zagajewska A, Szyfter W, ZeromskiJ,WhitesideTL,Triggering of Toll-like receptor 4 expressedon human head and neck squamous cell carcinoma promotestumor development and protects the tumor from immune attack.Cancer Res,2009; 69:3105–3113.
70. I. Petak, D. M. Tillman, and J. A. Houghton, “p53 Dependence of Fas induction and acute apoptosis in response to 5-fluorouracil-leucovorin in human colon carcinoma cell lines,” Clinical Cancer Research,2000; 6 (11): 4432–4441, .

71. Bunz F, Hwang PM, Torrance C, Waldman T, Zhang Y, Dillehay L, Williams J, Lengauer C, Kinzler KW, Vogelstein B. Disruption of p53 in human cancer cells alters the responses to therapeutic agents, Journal of Clinical Investigation 1999; 104(3): 263–269.
72. A. Dart, S. M. Picksley, P. A. Cooper, J. A. Double, and M. C. Bibby, "The role of p53 in the chemotherapeutic responses to cisplatin, doxorubicin and 5-fluorouracil treatment," International Journal of Oncology2004;24(1): 115–125.

73. ViengthongLamJ.PeterMcPhersonLeonardoSalmenaJodiLeesWilliam Chu, Elizabeth Sexsmith, David W Hedley ,Melvin H FreedmanJohn C ReedDavidMalkinGerald J Goldenberg, "p53 gene status and chemosensitivity of childhood acute lymphoblastic leukemia cells to adriamycin," Leukemia Research,1999; 23(10): 871–880.

74. B. A. Goff, J. A. Ries, L. P. Els, M. D. Coltrera, and A. M. Gown, "Immunophenotype of ovarian cancer as predictor of clinical outcome: evaluation at primary surgery and second-look procedure," Gynecologic Oncology1998 ; 70(3): 378–385.

75. Keerthana.B, Gayathri R, Vishnu priyaV, Apoptotic induction potentials of bacopamoneri against oral cancer cell line, Asain Journal of Pharmaceutical and Clinical Research 2016; 9(5): 172-3.

Cardiovascular Risk Of Epinephrine Use In Hypertensive Dental Patient

Suvitha.S[1], BrigitVarghesenEapen[2], Balakrishna R N[3]
Graduated student[1] ,Senior Lecturer[2],Department Of Oral And Maxillofacial Surgery
Senior lecturer[3],Department of oral surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Epinephrine is widely used as an additive in local anaesthetics (typically in concentrations of 1:100,000) to improve the depth and duration of the anaesthesia, as well as to reduce bleeding in the operative field. Epinephrine counteracts the anaesthetic'slocalized vasodilator effects in subcutaneous and sub mucosal vessels, thereby reducing the risk of anesthetic toxicity by decreasing the rate of systemic absorption from the site of injection. Epinephrine is also impregnated in cotton cord that is inserted into the sulcus between a tooth and the surrounding gingiva, as a gingival retraction cord improving access for tooth preparation and allowing dental impression material to more readily flow into the sulcus to record details of teeth prepared for crowns. The epinephrine likewise tightens the blood supply to contiguous tissue, in this manner allowing the impression to be secured without Contamination by bleeding. Hypertension affects nearly 50 million people in the United States and underlies most cardio- vascular disease, its diagnosis and control should be of concern to all health care providers. Many people have undetected hypertension, and current levels of detection and control need to be improved. Clinical Implications.All health care providers, including dentists and members of the dental team, need to be involved in detection and management of this important public health problem. The dentist can play an important role in the detection and management of hypertension.

Key words: *epinephrine, hypertensive patient, cardiovascular disease, extraction, hemodynamic changes.*

Introduction

Epinephrine is commonly used in health care applications as life savers. Such as in management of anaphylaxis and cardiac arrest. The drug is produced in human body from adrenal gland above the kidney. Though it is produced in our system their requirements in emergency procedures are high, and always there is a confusion regarding dose limits for epinephrine. Especially when it is administered in patients with cardiovascular diseases.Epinephrine was first added to the local anaesthesia ester procaine over 100 years ago, which is not in current practices. It is one of the widely used vasoconstrictors in dentistry. However, the use of local anaesthetics containing epinephrine in cardiac compromised patient during dental procedures has been controversial. Epinephrine has been argued as a potential risk factor that can cause cardiovascular complications. Hence many studies have been conducted to evaluate the safety of epinephrine in dental treatment.

There are numerous physiological effects of epinephrine, as it is reason for the reaction “flight or fight” in all mammals. Some variations in this response depends on the predominant type of adregenic receptors in the target organ.[1]Epinephrine increases the heart rate and the force of ventricular contraction, which eventually increases cardiac output. This increasedcardiac workloadsimultaneously increases myocardial oxygen consumption. This should be concerned in an individual suffering from cardiac disease, particularly given that the beneficial coronary vasodilator effect of epinephrine is diminished or absent in the presence of coronary vessel atherosclerosis. Those risk to the cardiac patient is the potential of epinephrine to irritate cardiac pacemaker cells and results in dysarhythmias. Thus, the imprudent use of epinephrine can be harmful to a patient with cardiac disease.[2]

EpinephrineIn Dentistry

Epinephrine is a commonly used drug in cases of medical emergency situation. In case of dentistry, it has two uses. First, it is a constituent of local anesthetic solution commonly used in dentistry. Secondly, it is used as a haemostatic agents in gingival retraction cord[3].Though epinephrine have these use, which may not be as readily achievable through use of non-epinephrine preparations, the clinical impact of cardiovascular and hemodynamic changes caused by the introduction of exogenous epinephrine makes its use among hypertensive

individuals has some controversies in dentistry. The added risks produced by the use of epinephrine in hypertensive patients include:

- Through the action of epinephrine-greater probability of acute hypertensive crisis (seriously high blood pressure), angina pectoris and myocardial infarction, as well as cardiac arrthymias.
    - Risk of drug interaction of epinephrine and some antihypertensive medication can lead to acute hypertensive or hypotensive crisis.[4]

      Administrating epinephrine in patients should be done with care as a frequent monitoring of blood pressure is essential because of its pharmacological actions. Epinephrine produces two types of effects in patients. One effect is pronounced on heart due to its beta-1 effects and other effect is on skeletal muscle blood vessels due to its beta-2 effects. This results in increased blood pressure and heart rate. [5]

Hypertension

Hypertension is one of the most frequently encountered systemic diseases in patients visiting dental clinics, due to its high prevalenceworldwide. It is defined as the values greater than 140 mmHg SBP (Systolic Blood Pressure) and values greater than 90 mmHg DBP (Diastolic Blood Pressure).It is also called as “Silent Killer” because it often affects target organs such as brain, heart, kidneys, eyes etc before the appearance of clinical symptoms. A permanent high blood pressure affects blood vessels in heart, kidney and brain which increases the chances of renal ,coronary heart disease and stroke.[6]JNC 7 introduces a category called “prehypertensive” to describe people with SBP of 120 to 139 mm Hg or a DBP of 80 to 89 mm Hg. People with prehypertension are at increased risk of progressing to hypertension and require health- promoting lifestyle modifications to prevent CVD.[7] One of the reasons that the concept of pre- hypertension was developed is to provide a wake-up call for affected people to encourage them to make appropriate lifestyle choices. A critical aspect in the prevention and management of hypertension is the adoption of healthy lifestyles. Without lifestyle modifications, BP may not be controlled adequately despite sufficient doses of antihypertensive drugs or appropriate combinations of drugs. Recommended modifications are weight reduction for overweight patients, dietary sodium reduction, appropriate physical activity and moderation in consumption of alcohol. Also, the consumption of a diet rich in

fruits, vegetables and low-fat dairy products such as recommended in the Dietary Approaches to Stop Hypertension. [8, 9]

A greater of awareness on hypertension recorded in the present study could be due to the lifestyle and some other factors compared to the general population. The risk factors associated with hypertension are similar to that seen in other studies which were done in general population. So health-care providers should recognize the increased risk of prehypertension and should seek to identify and treat the modifiable risk factors in these persons especially among undergraduate students who are country's young generation. [10]

Etiology, Classification And Pathogenesis Of Hypertension

Hypertension is the most common primary diagnosis in the United States, affecting more than 50 millionsPopulations and also in developing countries like India and China. Etiological factors correlated with hypertension in adults have also been associated with blood pressure elevations in youth. Intrauterine malnutrition, family history of hypertension ,obesity, particularly excess abdominal fat, insulin resistance, high dietary sodium intakes, low dietary intakes of calcium, potassium and magnesium, physical inactivity, high alcohol intakes, tobacco use, drug use (e.g., cocaine, ecstasy, anabolic steroids), emotional stress, diet pill use, oral contraceptives are the factors associated with development of hypertension.[11]

Hypertension is classified as primary or essential hypertension and secondary hypertension. Primary hypertension is most common form of hypertension where the level of hypertension is medium to high for a long time without a known cause. [12]Secondary hypertension is the one which occurs with an organic cause which includes renal, endocrine, neurological and others such as drugs induced, pregnancy toxaemia etc.JNC 7 introduced in 2003 the category of prehypertension in which the patients with this condition are at increased risk of developing hypertension. [13]Pathogenesis of hypertension results from a complex interaction between genetic factors and the environment. Some of them are age, family history of premature cardiovascular disease, smoking, increased consumption of alcohol, cholesterol rich diet. [14]

Table: 1 Showing classification of Hypertention

Classification	SBP (mmHg)	DBP (mmHg)
Normal	<120	And <80
Prehypertension	120-139	Or 80-89
Stage I hypertension	140-159	Or 90-99
Stage II hypertension	>160	>100

HypertensionAnd Cardiovascular Disease

Hypertension is found to have alink in many cardiovascular diseases. Some of them are

Arterial hypertension is an important health problem as it causes risks of cardiovascular diseases such as chest pain,myocardial infarction and cerebrovascular event. [15] In such cases, patient's blood pressure is frequently monitored before dental treatment to make the blood pressure under control to manage this condition. Heart failure is defined as incapacity of heart to function properly, pumping insufficient blood towards the tissues and leading to fluid accumulation within the lungs, liver and peripheral tissues. It is of two types.

I. Acute heart failure: The most common causes of heart failure are severe and prolonged arterial hypertension, valve disease and ischemic heart disease. This typically manifests as acute lunge enema.[16]
II. Chronic heart failure: The most common cause of this type of heart failure is that this is also associated with arterial hypertension.

Hypertensive Emergency: It is defined as elevated BP with signs and symptoms of target organ damage. The blood pressure should be acutely lowered or else tissue perfusion in brain heart can occur which is regulated by MAP (meanarterial pressure). [17, 18, 19]

Epinephrine And Associated Risk Of Cardiovascular Disease

Various studies have revealed the fact that diastolic blood pressure decreased and heart rate increased in both normotensive and hypertensive patients during dental treatment with epinephrine containing local anesthetic. These changes were small and unlikely to be clinically significant. Systolic blood pressure rose slightly in hypertensive patients but not in

normotensive individuals when Epinephrine containing local anaesthetics were injected, this was proved by vet ale et al in 1960's [20].He stated that patients who received lidocaine with epinephrine and underwent subsequent tooth extraction presented clinical evidence of diminished postoperative bleeding from the socket wound. This was determined from the condition of the gauze sponges placed over the sockets. In addition, the patients so studied attested to the depth and duration of anaesthesia produced. It has been stated many times in the past that the use of local anesthetic solutions without epinephrine is unrealistic since the oral operations that follow are often associated with pain. This pain, in turn, will produce more epinephrine from natural secretion (adrenal medulla) than does the 1:50,000 or 1: 100,000 epinephrine normally used in local anesthetic solutions. It is true that depth of anaesthesia is desirable. However, as has been demonstrated in twenty-six. Patients, both normotensive and hypertensive, an anesthetic properly placed by infiltration or by the block method will produce satisfactory anaesthesia for routine dental procedures within five minutes. The pressor response related to fear of the injection and the impending dental surgery was so evident in this experiment, and also in those reported by Cheraskin and Prasertsuntarasai, that premeditation should become routine. When local anaesthesia for dental procedures involving trauma is anticipated for patients with hypertension, cardiovascular disease, and anxiety states. These patients are greater operative risks, and care should be taken to see that their physical abnormalities are recognized. The care of patients with this type of abnormalities needs a close cooperation between the physician and the dentist. Anxiety, pain, fear, or prolonged traumatic operation should be avoided. Adequate preoperative medication, usually with barbiturates as the drugs of choice is therefore important to ensure a smoother and safer surgical procedure.

Management

While managing a hypertensive patient in the dental office, the dentist must take efforts to perform the dental procedures with optimum pain control, reduced stress and anxiety. To perform a safe dental procedures in hypertensive patients, the operator must follow certain guidelines. Dentist must monitor the blood pressure carefully before and after the administration of local anaesthesia. Pre operative reassurance with the use of effective local anaesthesia (with or without epinephrine) may help in alleviating the related anxiety and reduced the chances of increased blood pressure. The risky patients should be advised to seek

medical attention, during the dental treatment. Epinephrine can be used in management of hypertension by giving two 1.8 ml cartridges of lignocaine containing 1:100,000 epinephrine which is considered as safer dose for these hypertensive patients. [21]

In 1964, a Working Conference of the American Dental Association and the American Heart Association 18stated that the concentrations of vasoconstrictors normally used in dental local anaestheticsolutions were not contraindicated in patients with cardiovascular disease when administered carefully and with preliminary aspiration.Malamed recommended a much smallermaximal dose of vasoconstrictor (no more than 40 µgof epinephrine at one appointment) for patients with severe cardiovascular disease. Little etepinephrine (18 to 36 µg of epinephrine) were of little clinical significance to most patients with hypertension or other cardiovascular diseases [22]. Kaneko21 recommended that the total dose of epinephrine should be limited to less than 40 µg in moderate cases or 20 µg in serious cases, with epinephrine concentrations of 1:100,000 or less. Current clinical practice has tacitly accepted the safety of epinephrine use in patients with cardiovascular disease. Therehave been several reports of the hemodynamic effects of epinephrine in patients with cardiovascular disease. Cintron et alreported that dental anaesthesia with 1.0 ml of 2% lidocaine with 1:100,000 epinephrine caused no significant changes in HR or blood pressure and was well tolerated by patients with recent myocardial infarction. Middlehurst and Coulthard reported that approximately 5 ml of 2% lidocaine with 1:50,000 epinephrine and 0.25 IU/ml of vasopressin did not cause ischemic changes in patients with heart disease. Davenport et al studied the hemodynamic effects of moderate doses of epinephrine during periodontal surgery in patients with stable cardiovascular disease and found no significant changes in HR or blood pressure, despite the elevation of the plasma epinephrine concentration. Whenexcessive concentration or dose of epinephrine is administered in patients with a normal heart, SV and cardiac output are further elevated because of the β1 effect of epinephrine, leading to a prominent rise in blood pressure. Consequently, rate pressure product, indexes of myocardial oxygen consumption, and left ventricular stroke work (the product of MAP and SV), representing the level of work by the left ventricle, also increase. Although a normal heart can apparently withstand these great demands, in failing or ischemic heart, cardiac functions may be impaired by these changes. The possibility of adverse interactions between epinephrine and nonselective β-blocking agents has been suggested. Epinephrine production may lead to an exaggerated increase in blood pressure in patients taking a nonspecific β-blocking agent such

as propranolol. As shown in Fig 1, the response to the low dose of epinephrine in patients taking nonselective β-blocking agents differed from that in patients not taking such medication. Little suggested, on the basis of clinical experience, that 1:100,000 epinephrine (no more than 0.036 mg of epinephrine) in a local anesthetic could safely be used for patients receiving nonselective βblocking agents. However, the possibility of a hypertensive interaction occurring with epinephrine should be considered when epinephrine is used in patients taking a nonspecific β-blocking agent. This study provides documentation that 1.8 ml of L-E is safe and has few, if any, hemodynamic consequences in patients with cardiovascular disease. We concluded that a low dose of epinephrine (22.5 µg) in local dental anaesthesia was well tolerated by cardiovascular patients. [23]

Conclusion

Dentistry has played a cardinal role in the detection of patients with hypertension. Patients suspected to have high blood pressure should be referred to medical diagnosis. In dental office medical emergencies do occur to which dentist has to be prepared to overcome those difficulties. An early detection of hypertension by the dentist can lead to prompt treatment in the dental office.

Corresponding Author

Balakrishna R N

Senior Lecturer,Department Of Oral And Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai-600077,Tamil Nadu

Email:balakrishdr8394@gmail.com

Mobile: 8939896729

Reference

1. Dr. Ben Davis, What dose of epinephrine contained in local anaesthesia can be safely administered to a patient with underlying cardiac disease during a dental procedure? J Can Dent Assoc 2010; 76:a36.

2. Fleisher LA, Beckman JA, Brown KA, Calkins H, Chaikof EL, Fleishmann KE, guidelines on preoperative cardiovascular evaluation and care for noncardiac surgery 2009;120(21):e169-276.

3. Popescu SM, Scrieciu M, Mercuţ V, Ţuculina M, Dascălu I. Hypertensive patients and their management in dentistry. ISRN Hypertension. 2013.
4. Bader JD, Bonito AJ, ShugarsDA.Cardiovascular Effects of Epinephrine in Hypertensive Dental Patients: Summary. 2002, 48.
5. Stephen Wilford Holm, DMD,Larry L. Cunningham, Jr, DDS, MD, Eric BensadounMD,and Matthew J. Madsen, HypertensionClassification, pathophysiology and management during outpatient sedation and local anaesthesia. J Oral MaxillofacSurg 2006; 64; 111-21.

6. Scheltens T, Bots ML, Numans ME, Grobbee DE, Hoes AW. Awareness, treatment and control of hypertension: the 'rule of halves' in an era of risk-based treatment of hypertension. Journal of human hypertension. 2007 Feb; 21(2):99.
7. Hajjar I, Kotchen TA. Trends in prevalence, awareness, treatment, and control of hypertension in the United States, JAMA 2003; 290(2):199-206.
8. Whelton SP, Chin A, Xin X. Effect of aerobic exercise on blood pressure: a meta-analysis of randomized, controlled trials. Ann Intern Med 2002;136:493-503.
9. XinX,Frontini MG, Ogden LG, Motsamai OI, Whelton PK. Effects of alcohol reduction on blood pressure: a meta-analysis of ran- domized controlled trials. Hypertension 2001;38:1112-7.
10. MohamadHaikal Bin Zakaria and Gheena, S. The awareness and knowledge of hypertension among Undergraduate students in Chennai BDS, Department of General Pathology, Saveetha Dental College, International Journal of Current Research. May 2017, Vol. 9, Issue, 05, pp.50380-50385,
11. Nandhini.S. Essential Hypertension J. Pharm. Sci. & Res. 2014 Vol. 6(9), 305-307
12. Kaplan NM. "Primary hypertension. From pathophysiology to prevention", Archives of Internal Medicine,1996, vol.156, no. 17, pp 1919-1920.
13. Vasan RS, Larson MG, Liep et al EP," Impact of high normal blood pressure on the risk of cardiovascular disease" The New England Journal of Medicine, 2001,vol 345, no.18,pp.1291-1297.

14. Kaplan NM. " Management of hypertensive patient with multiple cardiovascular risk factors", American Journal of Hypertension, 2001 vol.14,no.6,part 2, pp.221-224.
15. Herman WW, Konzelman JL, PrisantLM;Joint National committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. New national guidelines on hypertension: a summary of dentistry. J Am dent assoc. 2004; 135:576-84.
16. Margaix-Munoz M, Jimenez-Soriano Y, proveda- Roda R , Sarrion G. Cardiovascular disease in dental practice. Practical considerations. Med oral patrol Oral cirbucal. 2008;13: 296-302.
17. Bethesda (MD):National High Blood Pressure Education Program. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7).2004 Aug. Report No.: 04-5230.
18. Varon J, Marik PE. The diagnosis and management of hypertensive crises. 2000 Jul;118(1): 214-227.
19. Flanigan JS, Vitberg D. Hypertensive emergency and severe hypertension: what to treat, who to treat, and how to treat. Med Clin North Am. 2006 May;90(3):439-451.
20. Little JW, Falace DA, Miller CS, Rhodus NL. Little JW, Falace DS, Miller CS, RhodusNL,editors. Dental management of the medically compromised patient.5th ed. St Louis: Mosby; 1997. p. 176 92.
21. Holm SW, Cunningham LL, Bensadoun JE, Madsen MJ. Classification, pathophysiology, and management during outpatient sedation and local anaesthesia. J Oral MaxillofacSurg 2006; 64; 111 21.
22. Malamed SF. Angina pectoris. In: Handbook of medical emergencies in the dental office. 2nd ed. St Louis: Mosby; 1982. p. 327-8.
23. Charles A. Vernale, D.D.S., M.S., Waterbury, Conn. cardiovascular response To Local Dental anaesthesia with epinephrine in normotensive and hypertensive subjects. August 1960, 13(8); 943-951.

Cigarette Smoking, Endothelial Injury and Cardiovascular Disease

Benazir HussainM[1], Sivakumar M[2]

Graduate Student[1], Senior Lecturer[2], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

Abstract

Despite the fact that the epidemiological evidence linking cigarette smoking with cardiovascular disease is overwhelming, the precise components of cigarette smoke responsible for this relationship and the mechanisms by which they exert their effect have not yet been elucidated. There are however, some promising pointers as a result of recent developments and this review concentrates on new evidence since earlier reviews of this topic. It is now known that the endothelium has a vastly more important role than was ever thought to be the case a decade ago. Its role in health and disease is increasingly understood, as is the relationship between endothelial injury and the development of atherosclerosis. There is considerable evidence that cigarette smoking can result in both morphological and biochemical disturbances to the endothelium both *in vivo* and in cell culture systems. Cigarette smoke is a complex mixture and only a few components have been extensively studied. Nicotine and carbon monoxide are much less damaging than is whole smoke. However the free radical components of cigarette smoke have been shown to cause damage in model systems. Further work will be necessary to consolidate the evidence base but the data reported in this review suggest that the free radical components of cigarette smoke may be responsible for the morphological and functional damage to endothelium that has been observed in model systems. This reviews gives a detailed description on effect of smoking in heart and cardiovascular system

Key Words: *cigarette smoking, endothelium, injury, nicotine, carbon monoxide, free radicals, morphology*

Introduction

Death from coronary heart disease is more common in smokers than nonsmokers were published in the 1950s,[1] there have been many reports which convincingly demonstrate that cardiovascular disease is the most important cause of smoking-related premature death.[2] The prospective investigation of British Doctors commenced in 1951 showed that half the excess mortality from smoking results from cardiovascular disease with approximately one third due solely to coronary heart disease. Prospective studies on smokers have shown that the risk of death from coronary heart disease is roughly doubled for both men and women although there is evidence that the risk is greater for women estimated that cigarette smoking accounted for 25% of the overall mortality from ischaemic heart disease. More recently in the United States, has estimated that one fifth of all heart disease-related deaths are due to cigarette smoking and that smoking, as a single factor, doubles the risk of heart failure.[3] Cigarette smokers are also one and a half times more at risk of stroke than nonsmokers. Cigarette smoking is an independent risk factor in the development of atherosclerotic lesions in the internal pudendal and common penile arteries of young impotent men.[4] It is a significant predictor of the presence of intracranial internal carotid artery atherosclerosis. In the Edinburgh Artery Study, cigarette smoking was shown to have a direct effect on the risk of aortic aneurysm which was independent of atherosclerosis. This review also showed that smoking increases the risk of peripheral artery disease more than heart disease. Perhaps not surprisingly, cigarette smokers who regularly undertake physical activity have a reduced cardiovascular mortality rate than those who have a sedentary existence. Increased total exposure to tobacco in humans and a smoking pattern which maximizes nicotine yield are also associated with an increased risk of peripheral arterial disease.[5]

Endothelial Injury

Despite the strong epidemiological evidence that exists linking cigarette smoking and cardiovascular disease, the mechanisms by which cigarette smoking causes disease and the components of smoke responsible remain poorly understood. Endothelial injury is considered to be a key initiating event in the pathogenesis of atherosclerosis and it has therefore seemed reasonable to hypothesize that cigarette smoke, or some components of it, may exert its effects by damaging the endothelium.[6] Components of smoke that gain access to the circulation will come in contact with blood and with the vascular endothelial cells that form a monolayer lining the vessels. These cells are now known to have a crucial role in controlling

the blood circulation and to be highly active metabolically. Even minor disturbances to their normal functioning could have significant implications for the initiation and development of atherosclerosis. Endothelial functions, including an increase in permeability and decreased nitric oxide (NO) production along with increased expression of adhesion molecules and adherence of leukocytes to the vessel wall, have been shown to be impaired by risk factors for cardiovascular disease such as hypertension, hyperlipidaemia and hyperglycaemia.[7] This review examines the evidence that cigarette smoking can bring about vessel wall damage with particular reference to the vascular endothelium.

Damaging Effects of Cigarette Smoking on Human Arteries

Direct evidence that cigarette smoking can result in atherosclerosis continues to be obtained from human investigations. Using serial quantitative coronary arteriography, have shown that cigarette smoking accelerates coronary progression and new lesion formation.[8] Interestingly, Lovastatin was shown to slow the progression of coronary atherosclerosis and prevent the development of new coronary lesions in smokers. The relative thickness of the intima and media of an artery as measured by ultrasound can be used to provide an index of atherosclerosis. Carotid artery atherosclerosis determined by ultrasound determination of plaque thickness, a measure of atherosclerosis, is accelerated depending on the level of cigarette use and this is independent of age, hypertension and diabetes.[9] Cigarette smoking has an adverse effect on the intimal medial ratio and stenosis of the carotid artery as measured by ultrasound. Exposure to environmental tobacco smoke (passive smoking), and cigarette smoking have been shown using the intimal/medial ratio as an index to cause progression of atherosclerosis in the carotid artery. Cigarette smoking was shown to be an independent indicator of the severity of coronary and thoracic aortic atherosclerosis in men receiving coronary angiography and transoesophageal echocardiography.[10] In uraemic patients on chronic dialysis cigarette smoking is associated with carotid atherosclerosis as revealed using echo-colour-Doppler evaluation. Studies on the coronary arteries of trauma victims aged between 15 and 34 years of age suggest that intermediate atherosclerotic lesions progress rapidly into advanced lesions in smokers and that intima formerly having early lesions is replaced by intima with raised lesions.[11] This study also demonstrated that smoking was associated with more extensive fatty streaks and raised lesions in the abdominal aorta. Radiological observations on women aged between 45 and 64 years showed a direct association between the development of calcified deposits, which have been shown to represent atherosclerotic change, in the abdominal aorta and the number of cigarettes smoked

per day over a nine year period.[12] This study suggests that the rate of atherosclerotic change may be reduced by the cessation of smoking, but a residual effect appears to be present for a decade. Using ultrasonography to look at carotid intima and media thickness, the evidence indicates that atherosclerosis is mediated by components of tobacco smoke other than nicotine.[13]

Cigarette Smoking and Endothelial Modification

Direct evidence that cigarette smoking could result in endothelial injury was first obtained from morphological observations on the umbilical arteries from smoking mothers. These reports, along with others on the effects of cigarette smoking on endothelial morphology, have been previously reviewed by Pittilo (1988,1990). The morphological alterations, which included the endothelium having an irregular appearance along with membrane disturbances evidenced by the formation of blebs or microvillous-like projections, were also associated with functional changes to the endothelium. In laboratory rats exposed to cigarette smoke were able to demonstrate that the morphological alterations to the endothelium of the thoracic aorta were accompanied by reduced endothelial prostacyclin production as well as the adhesion of platelets to apparently intact cells.[14]

Cigarette Smoking and the Components of Smoke Responsible for Endothelial Injury

Cigarette smoke is a complex mixture and only a few components have been examined in isolation to assess their effects on endothelial morphology and function. The effects of nicotine on both the morphology and prostacyclin production of rabbit and cultured human endothelial cells has been reviewed by Bull (1988),Pittilo (1990) and Woolf *et al*. (1993), and although deleterious effects result from exposure, these are much more limited than those observed with whole smoke.[15] Clinical studies support these findings in that observations on pipe smokers and users of transdermal nicotine devices suggest that components of smoke other than nicotine are the most important causes of acute cardiovascular events. The effects of carbon monoxide exposure have also been reviewed and are conflicting, with some reports providing evidence of morphological or functional alterations and others reporting no effects.[16]

Rats exposed to nicotine show an increased frequency of endothelial cell death which results in enhanced transendothelial leakage of macromolecules including low density lipoproteins. Nicotine has been shown to enhance the release of platelet-derived growth factor by bovine

aortic smooth muscle cells and is associated with alterations in the cytoskeleton. Nicotine significantly stimulates DNA synthesis and endothelial cell proliferation in bovine pulmonary artery endothelial cells at concentrations lower than those obtained in the blood after smoking.[17] In rabbits, administration of nicotine is associated with an acceleration of intimal hyperplasia following endothelial removal.

Data derived from epidemiological and animal studies suggest that carbon monoxide is not atherogenic. However, exposure of cockerels to carbon monoxide does not have a discernible effect upon arteriosclerotic plaque development. Bovine pulmonary artery endothelial cells exposed to carbon monoxide release higher concentrations of nitric oxide and it has been suggested that carbon monoxide causes oxidative stress through competition for intracellular binding sites which increase steady state levels of nitric oxide and the generation of peroxynitrite by the endothelium.[18] Carbon monoxide suppresses the production of endothelin-1 and platelet-derived growth factor B by endothelial cells.

There is experimental evidence with cockerels to indicate that exposure to environmental tobacco smoke at levels equivalent to those routinely encountered by people in smoke-filled environments is sufficient to promote arteriosclerotic plaque development. In cholesterol-fed rabbits, passive smoking increases aortic and pulmonary artery atherosclerosis in a dose-responsive way and is independent of changes in serum lipids. One review of the literature concluded that passive smoking increases platelet activity, accelerates atherosclerotic lesion formation, and increases tissue damage following ischaemia or myocardial infarction.[19] However, it has been argued that the available experimental and epidemiological evidence to date does not support an association between environmental tobacco smoke exposure and an increased risk of heart disease.

Woolf *et al*. (1993) and Pittilo& Woolf (1993,1994) focused on the possible importance of free radicals as the mediator of cigarette smoke-induced endothelial damage for a number of reasons. Cigarette smoke has been known for many years to be a rich source of free radicals. The scanning electron microscope changes seen in laboratory rats following smoke exposure, or cultured cells following exposure to plasma from humans after smoking, were similar to the changes seen in cardiac myocytes following free radical-induced lipid peroxidation.[20] Finally, there is a strong association between lipid peroxidation and endothelial cell injury were reviewed some of the supportive evidence and considered that free radical-induced lipid peroxidation might eventually be shown to be the mechanism responsible for the

epidemiological relationship between cigarette smoking and atherosclerosis.[21] It has been suggested that the high levels of lipid peroxidation and increased formation of reactive oxygen species within the vascular wall in atherosclerosis can overwhelm cellular antioxidant defence mechanisms and human oxidatively modified LDL-induced expression of antioxidant stress proteins in vascular cells.

On the contrary, studies showing no differences in free radical markers between smokers and nonsmokers suggest that any free radical activity generated from smoking is adequately scavenged. No significant differences in free radical markers were found between young adult smokers and nonsmoking volunteers. Free radical activity due to cigarette smoking also appears to be adequately scavenged in young adults with diabetes who are free of significant macrovascular disease.[22] Furthermore, there is evidence to indicate that chemical modification of glutathione is a major damage mechanism of filtered cigarette smoke with free radical oxidations being less significant.[23]

Despite these findings, there remains strong evidence to implicate free radicals as an important cause of cigarette smoke-induced endothelial injury. The morphological alterations seen with cultured cells exposed to plasma obtained from volunteers after smoking are associated with functional changes to the endothelium including activation of the hexose-monophosphate shunt, a sharp increase in the total glutathione content of the culture medium, release of angiotensin-converting enzyme from the cells and a decrease in the ability of the cells to produce ATP, all of which indicate both oxidative stress and cell injury. Smoking is also a major determinant of increased plasma-free radical activity in dyspeptic subjects.[24]

Free Radicals and Cigarette Smoke

Cigarette smoke consists of two distinct populations of free radicals; the principal free radical in the tar phase is a quinone/hydroquinone (Q/QH_2) complex which is an active redox system that can reduce molecular oxygen to produce superoxide, and in the gas phase small oxygen-centred and carbon-centred radicals produced in a steady state by the oxidation of nitric oxide (NO) to nitrogen dioxide (NO_2) react with reactive species in smoke such as isoprene.[25] The radicals associated with the particulate or tar phase of cigarette smoke are long lived whereas the radicals associated with the gas or vapour phase have structures that predict a very short lifespan, although spin trap experiments demonstrate that these radicals may exist for periods in excess of five minutes. Aqueous extracts of cigarette tar auto-oxidize to

produce semiquinone, hydroxyl, and superoxide radicals in air-saturated buffered aqueous solutions.[26]

Direct-electron resonance measurements suggest that superoxide and hydroxl radicals are produced during the auto-oxidation of hydroquinone and catechol-related species in aqueous extracts of cigarette tar. Fresh cigarette smoke contains from 300 to 500 parts per million of nitric oxide. The nitric oxide radical is produced slowly from nitric oxide radical donors such as amine complexes, peroxinitrite and other reactants including nitrogen oxides. Nitric oxide can react with hydrogen peroxide to produce singlet oxygen which may also be important in mediating cigarette smoke-induced endothelial damage. Certainly there is evidence to suggest that nitric oxide in cigarette smoke is important as a mediator of oxidative damage and that DNA damage by cigarette smoke may invoke reactive nitrogen species as well as reactive oxygen species.[27] Analysis of cigarette smoke demonstrates that the main source of oxygen and hydrogen peroxide results from polyphenols in the particulate phase, and a synergistic effect is observed between these polyphenols and nicotine. The vapour phase contains a factor which produces reactive oxygen metabolites from hydrogen peroxide. Free radicals in the gas phase of cigarette smoke, identified using electron spin resonance spectrometry, were identified as mainly alkoxyl and alkyl free radicals with the latter making up approximately two thirds of the total spectral components.[28]

Haemoglobin-impregnated conventional cigarette filters are capable of withholding nitric oxide, nitrogen oxides, hydrogen peroxide, carbon monoxide, aldehydes, trace elements and carcinogenic nitrosocompounds from cigarette smoke.[29] This is interesting in view of the fact that the red blood cells of cigarette smokers contain more glutathione than those of nonsmokers and are more protective to endothelial cells in culture from hydrogen peroxide-mediated damage than are the red cells from nonsmokers.[30]

Cigarette Smoking and Endothelial Dysfunction

More recent studies that have not been the subject of previous reviews continue to provide evidence that cigarette smoke can alter vascular endothelium not only morphologically but functionally. In recent years, the discovery of nitric oxide has radically altered our understanding of vascular control.[31] Both nitric oxide and carbon monoxide generated within the blood vessel wall are important cellular messengers involved in the regulation of vascular smooth muscle tone. Carbon monoxide generated through haeme oxygenase inhibits mitogen-induced proliferation of vascular smooth muscle cells and there is evidence that

endogenous carbon monoxide serves as a protective factor limiting the excessive vascular smooth muscle cell proliferation associated with vascular disease.[32]

Cigarette smoking is associated with dose-related and potentially reversible impairment of endothelium-dependent arterial dilation in asymptomatic young adults, consistent with endothelial dysfunction. It augments endothelial-derived vaso relaxation but has no effect on endothelium-independent vaso relaxation.[33] It leads to a significant decrease in endothelium-dependent dilatation of the brachial artery. It causes immediate constriction of proxymal and distal epicardial coronary arteries and an increase in coronary vessel tone despite an increase in myocardial oxygen demand, and cigarette smokers show an impairment in basal but not stimulated nitric oxide mediated vaso dilation.[34]

Cigarette smoking has been shown to affect endothelial nitric oxide synthase activity and protein levels. It results in a decrease in exhaled nitric oxide in humans suggesting that it may inhibit the enzyme nitric oxide synthase. Cigarette smoke extracts cause an irreversible inhibition of nitric oxide synthase activity in pulmonary artery endothelial cells and a reduction in constitutive nitric oxide synthase activity in the rat in a dose-dependent manner.[35] Conversely, other studies in the rat have shown that cigarette smoking results in an increase in nitric oxide synthase gene expression and protein production. However, lower respiratory tract nitric oxide concentrations are increased following cigarette smoking although, in humans, plasma and urinary levels of nitrate, a metabolite of inhaled nitric oxide, have been shown to be unchanged suggesting that nitric oxide is not absorbed from the inhaled smoke.[36]

Inhibition of nitric oxide synthase activity may explain the impaired endothelium-independent vasodilation associated with cigarette smoking. The decrease in endothelial nitric oxide synthase activity may also partly explain the high risk of pulmonary and vascular disease in cigarette smokers. Cigarette smoking affects nitric oxide-mediated coagulation of coronary artery tone which is associated with a decrease in the bioactivity of nitric oxide.[37] It has been shown to increase isoprostane levels and reduce the generation of prostacyclin, l-arginine and l-citrulline in umbilical arteries and veins, and correlates with a direct vasoconstrictive effect. Chronic smoking in the rat leads to age-independent moderate hypertension and a decrease in penile nitric oxide synthase activity.[38]

It has been reported that exposure of cultured bovine pulmonary artery endothelial cells to carbon monoxide results in increased release of endothelial nitric oxide. The acute

cytotoxicity from carbon monoxide was owing to nitric oxide-derived oxidants. Incubation of isolated rabbit aortas with cigarette smoke extract inhibits endothelium-dependent relaxation in a dose-dependent manner.[39] If this incubation is carried out with free radical scavengers, attenuation of the inhibition occurs. This suggests that free radicals in cigarette smoke extract induce the impairment of endothelium-dependent relaxation and this may be partly because of the suppression of nitric oxide production. The same author has demonstrated, using cultured human endothelial cells, that cigarette smoke extract suppressed endothelial release of stable metabolites of nitric oxide and this was attenuated by free radical scavengers. The superficial femoral veins from rabbits exposed to cigarette smoke have a significant decrease in endothelium-dependent relaxation in response to acetylcholine without smooth muscle injury.[40] Ascorbic acid protects rabbit arteries from cigarette smoke-induced endothelial injury by reducing the impairment of endothelium-dependent acetylcholine relaxation caused by smoking, probably as a result of oxygen-free radicals.[41]

It has been suggested that the presence of hypoxia and exogenous nitric oxide, which lead to endothelial-dependent and independent vaso-relaxation secondary to cigarette smoking, may serve to explain the apparent augmentation of endothelial-derived relaxation in the rat. Cigarette smoking has been shown in the rat to increase aortic endothelial regeneration and serum levels of nitric oxide following balloon injury of the thoracic aorta.[42] Acute hypoxia causes pulmonary vessel constriction, and chronic hypoxia causes smooth muscle cell replication and extracellular matrix accumulation, resulting in vessel wall remodelling. Oxidized low-density lipoprotein, hypoxia and pro-inflammatory cytokines induce haem oxygenase expression and activity in vascular endothelial and smooth muscle cells.[43]

Cultured human umbilical endothelial cells from smokers convert significantly more LDL into an atherogenic form than do cells derived from nonsmokers. The LDL modifications were strongly thiol-dependent and the enhanced superoxide production seen in the cells derived from smokers was dependent on the presence of cysteine in the medium.[44] There is also evidence that cigarette smoking results in endothelial dysfunction in hypercholesterolemic patients by enhancing the oxidation of LDL. Cigarette smoke-modified low-density lipoprotein has been reported to impair endothelium-dependent relaxation in isolated rabbit arteries.[45]

Cells derived from smokers have higher levels of intracellular glutathione than those from nonsmokers and it may be that stimulation of cysteine uptake by the cells reflecting the

enhanced total glutathione content could account for the enhanced superoxide production, all of which may be relevant to the pathophysiology of smoking-related cardiovascular disease. It has already been noted that there is evidence to indicate that chemical modification of glutathione is a major damage mechanism of filtered cigarette smoke with free radical oxidations being less significant. Endothelial cell cGMP production is decreased in a dose-dependent manner following cigarette smoking and endothelial cell detachment is increased following smoking.[46] Externally added thiols protect endothelial cells from damage and it has been suggested that they may bind an unknown component of smoke to bring about this protection. In humans, the endothelial dysfunction observed following cigarette smoking lasts for about one hour and is not attenuated with repeat exposure.[47]

Cigarette smoking is associated with increased monocyte-endothelial cell adhesion in humans. Studies on hamsters using intravital microscopy and scanning electron microscopy demonstrate that cigarette smoking results in leucocytes adhering in clusters to the aortic endothelium and that aggregates of leukocytes and platelets are formed.[48] There is evidence that phagocytes may employ myeloperoxidase-generated reactive nitrogen intermediates as a physiological pathway for initiating lipid peroxidation as well as forming biologically active lipid and sterol oxidation productions *in vivo*.[49] Increased generation of oxygen free radicals by polymorphonuclear leucocytes may be responsible for the enhanced risk of certain smoking-related diseases. The promotion of neutrophil infiltration and free radical production in rats exposed to cigarette smoke has been shown to contribute significantly to the development of experimental inflammatory bowel disease.[50]

Cigarette smoking has been shown to increase re-endothelialization in the rat following large vessel injury and this is associated with an increase in serum nitric oxide levels. There is evidence that cigarette smoke-induced cell proliferation in the pulmonary arterial vessels is partly mediated through stimulation of endothelin-A receptors.[51] Conversely, cigarette smoke represses angiogenesis in the rat. Cigarette smoking, but not transdermal nicotine delivery, is associated with borderline increases in plasma endothelin-1 levels but these are restricted to the first 10 minutes after the onset of smoking.[52]

In a study of monozygotic twins discordant for smoking found that cigarette smoking was associated with an atherogenic lipid profile along with changes in platelets and white cells which might reflect endothelial cell damage. Cigarette smoking increases total serum cholesterol levels.[53] Smokers have mean serum levels that are higher than nonsmokers for

total and low-density lipoprotein cholesterol and triglyceride whereas high-density lipoprotein cholesterol levels are lower. There is evidence that cigarette smoking results in increased platelet consumption in human atherosclerotic vessels as well as the production of larger platelets which are more active.[54] Cigarette smoking causes marked inhibition of substance P-induced tissue plasminogen activator released *in vivo* in humans. Serum from cigarette smokers contains higher levels of von Willebrand factor and was more cytotoxic to endothelial cells *in vitro* than serum from nonsmokers.[55]

Conclusion

Since the publication of previous reviews on cigarette smoking, endothelial injury and cardiovascular disease, there have been new reports that demonstrate that cigarette smoking is associated with blood vessel wall damage including endothelial injury. Cigarette smoke is a complex mixture containing a range of individual components and only a small number of these have been examined in isolation. Whilst components such as nicotine and carbon monoxide have been the subject of a number of investigations, their damaging effects in model systems are less than those seen with whole smoke. Free radicals are an important component of cigarette smoke and our understanding of their chemistry and effects on biological systems has greatly increased. It seems likely that they will be further implicated as critical to the link between cigarette smoking and cardiovascular disease.

Corresponding Author

Sivakumar M

Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai-600077,Tamil Nadu, India.

Email: drsivaomfs@gmail.com

Telephone Number: +919443602627

References

1. Doll R, Hill AB. Lung cancer and other causes of death in relation to smoking. Brit. Med. J. 1956;4:1071–1081.

2. Fowler G. Smoking as a risk factor for cardiovascular disease. In: Poulter N, Sever P, Thom S, editors. Cardiovascular disease risk factors and intervention. Jcdr; 1993. pp. 161–169.
3. Manley AF. Cardiovascular implications of smoking: The Surgeon General's point of view. J. Health Care Poor Underserved. 1997;8:303–310.
4. Rosen MP, Greenfield AJ, Walker T. Cigarette smoking: an independent risk factor for atherosclerosis in the hypogastric-cavernous arterial bed of men with arteriogenic impotence. J. Urol. 1991;145:759–763.
5. Powell JT, Edwards RJ, Worrell PC, Franks PJ, Greenhalgh RM, Poulter NR. Risk factors associated with the development of peripheral arterial disease in smokers: a case-control study. Atherosclerosis. 1997;129:41–48.
6. Ross R. The pathogenesis of atherosclerosis: an update. N. Engl. J. Med. 1986;314:488–500.
7. Haller H. Risk factors for cardiovascular disease and the endothelium. J Pharm 2001;23:102-289
8. Waters D, Lesperance J, GLadstone P. Effects of cigarette smoking on the angiographic evolution of coronary atherosclerosis: a Canadian Coronary Atherosclerosis Intervention Trial (CCAIT) substudy. Circulation. 1996;94:614, 621.
9. Dempsey RJ, Moore RW. Amount of smoking independently predicts carotid artery atherosclerosis severity. Stroke. 1992;23:693–696.
10. Inoue T, Oku K, Kimoto K. Relationship of cigarette smoking to the severity of coronary and thoracic aortic atherosclerosis. Cardiology. 1995;86:374–379.
11. Zieske AW, Takei H, Fallon KB, Strong JP. Smoking and atherosclerosis in youth. Atherosclerosis. 1999;144:403–408.
12. Witteman JCM, Grobbee DE, Valkenburgh HA, Vanhermert AM, Stijnen T, Hofman A. Cigarette smoking and the development and progression of aortic atherosclerosis: a 9-year population-based follow-up-study in women. Circulation. 1993;88:2156–2162.
13. Bolinder G, Noren A, Defaire U, Wahren J. Smokeless tobacco use and atherosclerosis: an ultrasonographic investigation of carotid intima media thickness in healthy middle-aged men. Atherosclerosis. 1997;132:95–103.
14. Pittilo RM, Mackie IJ, Rowles PM, Machin SJ, Woolf N. Effects of cigarette smoking on the ultrastructure of rat thoracic aorta and its ability to produce prostacyclin. Throm. Haemostas. 1982;48:173–176.
15. Bull HA, Pittilo RM, BLow DJ. The effects of nicotine on PGI_2 production by rat aortic endothelium. Throm. Haemost. 1985;54:472–474.

16. Benowitz NL. The role of nicotine in smoking-related cardiovascular disease. Prev. Med. 1997;26:412–417.
17. Villablanca AC. Nicotine stimulates DNA synthesis and proliferation in vascular endothelial cells in vitro. J. Appl. Physiol. 1998;84:2089–2098
18. Thom SR, Xu YA, Ischiropoulos H. Vascular endothelial cells generate peroxynitrite in response to carbon monoxide exposure. Chem. Res. Toxicol. 1997;10:1023–1031.
19. GLantz SA, Parmley WW. Passive smoking and heart disease: mechanisms and risk. J. Am. Med. Assoc. 1995;273:1047–1053.
20. Noronha-Dutra AA, Steen EM. Lipid peroxidation as a mechanism of injury in cardiac myocytes. Lab. Invest. 1982;47:346–353.
21. Pittilo RM, Woolf N. Cigarette smoking as a risk factor for atherosclerosis. J. Smoking-Related Dis. 1994;5:43–47.
22. Leonard MB, Lawton K, Watson ID, Marfarlane I. Free-radical activity in young adult cigarette smokers. J. Clin. Pathol. 1995;48:385–387.
23. Maranzana A, Mehlhorn RJ. Loss of glutathione, ascorbate recycling, and free radical scavenging in human erythrocytes exposed to filtered cigarette smoke. Arch. Biochem. Biophys. 1998;350:169–182.
24. Phull PS, Price AB, Thorniley MS, Green CJ, Jacyna MR. Plasma free radical activity and antioxidant vitamin levels in dyspeptic patients: Correlation with smoking and Helicobacter pylori infection. Eur. J. Gastroenterol. Hepatol. 1998;10:573–578.
25. Church DF, Pryor WA. Free radical chemistry of cigarette smoke and its toxicological implications. Environ. Health Persp. 1985;64:111–126.
26. Zang LY, Stone K, Pryor WA. Detection of free radicals in aqueous extracts of cigarette tar by electron-spin resonance. Free Radic. Biol. Med. 1995;19:161–167.
27. Epperlein MM, Nouroozzadeh J, Noronhadutra AA, Woolf N. Nitric Oxide in cigarette smoke as a mediator of oxidative damage. Int. J. Experim. Path. 1996;77:197–200.
28. Zhao BL, Yan LJ, Hou JW, Xin WJ. ESR spin trapping studies on the free radicals in gas phase of cigarette smoke. Chin. Med. J. 1991;104:591–594.
29. Deliconstantinos G, Villiotou V, Stravrides JC. Scavenging effects of haemoglobin and related heme-containing compounds on nitric oxide, reactive oxidants and carcinogenic volatile nitrosocompounds of cigarette smoke: a new method for protection against the dangerous cigarette constituents. Jcdr.1994;14:2727–2726.

30. Toth KM, Berger EM, Beehler CJ, Repine JE. Erythrocytes from cigarette smokers contain more glutathione and catalase and protect endothelial cells from hydrogen peroxide better than do erythrocytes from non-smokers.Jcdr. 1986;134:281–284.
31. Vallance P. Exploring vascular nitric oxide in health and disease: The Goulstonian Lecture (1996) J. Roy. Coll. Physic. Lon. 1997;31:321–327.
32. Togane Y, Morita T, Suematsu M, Ishimura Y, Yamazaki J, Katayama S. Protective roles of endogenous-carbon monoxide in neointimal development elicited by arterial injury. Am. J. Physiol. Heart Circul. Physiol. 2000;278:623–632.
33. Nene S, Gelabert H, Moore W, Quinonesbaldrich W, Santibanezgallerani A, Ignarro L. Cigarette smoking increases endothelial-derived vasorelaxation in the rat carotid artery in a dose-dependent manner. J. Surg. Res. 1997;71:101–106.
34. McVeigh GE, Lemay L, Morgan D, Cohn JN. Effects of long-term cigarette smoking on endothelium-dependent responses in humans. Am. J. Cardiol. 1996;78:668–672.
35. Ma L, Chow JYC, Cho CH. Cigarette smoking delays ulcer healing: role of constitutive nitric oxide in synthase in rat stomach. Am. J. Physiol. Gastrointest. Liver. Physiol. 1999a;39:238–248.
36. Rangemark C, Wennmalm A. Smoke-derived nitric oxide and vascular prostacyclin are unable to counteract the platelet effect of increased thromoboxane formation in healthy female smokers. Clin. Physiol. 1996;16:301–315.
37. Kugiyama K, Yasue H, Ohgushi M. Deficiency in nitric oxide bioactivity in epicardial coronary arteries of cigarette smokers. J. Am. Coll. Cardiol. 1996;28:1161–1167.
38. Xie YN, Gargan H, Ng C, Rajfer J, Gonzalezcadavid NF. Effect of long-term passive smoking on erectile function and penile nitric oxide synthase in the rat. J. Urol. 1997;157:1121–1126.
39. Ota Y, Kugiyama K, Sugiyama S. Impairment of endothelium-dependent relaxation of rabbit aortas by cigarette smoke extract: Role of free radicals and attenuation by captopril. Atherosclerosis. 1997;131:195–202.
40. Freischlag JA, Johnson D, Farooq MM. Cigarette smoke impairs endothelium-dependent relaxation in rabbit superficial femoral veins. J. Surg. Res. 1999;81:77–800.
41. Mays BW, Freishlag JA, Eginton MT, Cambria RA, Seabrook GR, Towne JB. Ascorbic acid prevents cigarette smoke injury to endothelium-dependent arterial relaxation. J. Surg. Res. 1999;84:35–39.

42. Sarkar R, Gelabert HA, Mohiuddin KR, Thakor DK, Santibanez-Gallerani AS. Effect of cigarette smoke on endothelial regeneration in vivo and nitric oxide levels. J. Surg. Res. 1999;82:43–47.
43. Siow RCM, Sato H, Mann GE. Heme oxygenase carbon monoxide signalling pathway in atherosclerosis: anti-atherogenic actions of bilirubin and carbon monoxide. Cariovasc. Res. 1999;41:385–394.
44. PechAmsellemMA, Myara I, Storogenko. Enhanced modifications of low-density lipoproteins (LDL) by endothelial cells from smokers: a possible mechanism of smoking-related atherosclerosis. Cardiovasc. Res. 1996;31:975–983.
45. Kagota S, Yamaguchi Y, Shinozuka K, Kwon YM, Kunitomo M. Cigarette smoke-modified low density lipoprotein impairs endothelium-dependent relaxation in isolated rabbit arteries. Gen. Pharmacol. 1996;27:477–481.
46. Nagy J, Demaster EG, Wittmann I, Shultz P, Raij L. Induction of endothelial cell injury by cigarette smoke. Endothelium-NY. 1997;5:251–263.
47. Lekakis JP, Papamichael CM, Vemmos CJ, Stamatelopoulos K, Voutsas AA, Stamatelopoulos SF. Effects of acute cigarette smoking on endothelium-dependent arterial dilatation in normal subjects. Am. J. Cardio. 1998;81:1225.
48. Lehr HA, Frei B, Arfors KE. Vitamin C prevents cigarette smoke-induced leukocyte aggregation and adhesion to endothelium in-vivo. Proc. Natl. Acad. Sci. U.S.A. 1994;91:7688–7692.
49. Schmitt D, Shen ZZ, Zhang RL. Leukocytes utilise myeloperoxidase-generated nitrating intermediates as physiological catalysts for the generation of biologically active oxidised lipids and sterols in serum. Biochemistry. 1999;38:16904–16915.
50. Guo X, Wang WP, Ko JKS, Cho CH. Involvement of neutrophils and free radicals in the potentiating effects of passive cigarette smoking on inflammatory bowel disease in rats. Gastroenterology. 1999;117:884–892.
51. Dadmanesh F, Wright JL. Endothelin-A receptor antagonist BQ-610 blocks cigarette smoke-induced mitogenesis in rat airways and vessels. Am. J. Physiol. Lung Cell. Mol. Physiol. 1997;16:614–618.
52. Joseph John, Sri Sakthi. Effect of cigarette smoking and nicotine on plasma endothelin-1 levels. J. Cariovasc. Pharmacol. 1995;26:236–238.
53. Jorge PAR, Ozaki MR, ALmeida E**A.** Endothelial dysfunction in coronary vessels and thoracic aorta of rats exposed to cigarette smoke. Clin. Exp. Pharmacol. Physiol. 1995;22:410–413.

54. Harish Santhosh Pillai, Nithya. Tobacco potential threat to oral cavity. Clin. Labor. Haem. 1992;14:281–287.
55. BLann AD, McCollum CN. Adverse influence of cigarette smoking on the endothelium. Thromb. Haemost. 1993;70:707–711.

Cleft Lip and Cleft Palate

Vidhya.T.K[1],Yuvaraj Babu[2], Saravana Pandian[3],

Graduate Student[1],Reader[2], Department of Anatomy, Senior Lecturer[3], Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Cleft lip which is known as ancheiloschisis and cleft palate which has an other name of palatoschisis, which can also occur together as cleft lip and palate, are variations of a type of clefting congenital deformity caused by abnormal facial development during gestation. A cleft is a fissure or opening—a gap. It is the non-fusion of the body's natural structures that form before birth. In past, the condition was referred asan harelip, based on the similarity to the cleft in the lip of a hare, but that term is now generally considered to be offensive. Clefts can also affect other parts of the face, such as the eyes, ears, nose, cheeks, and forehead. Most of these craniofacial clefts are rare and are frequently described as Tessier clefts using the numerical locator which was coined byTessier. A cleft lip or palate can be successfully treated with surgery, especially so if conducted soon after birth or in early childhood.

Key Words: *Congenital deformity, velopharyngeal inadequacy, van der won syndrome,*

Introduction

Cleft lip is formed within the top of the lip as either small low gap or an indentation within the lip which is an partial or incomplete cleft when it continues into the nose it is known as an complete cleft. Lip cleft will occur as a 1 sided (unilateral) or 2 sided (bilateral). It's due to the failure of fusion of the jaw and medial nasal processes (formation of the first palate) congenital abnormality could be a condition during which the 2 plates of the bone that type the surface (roof of the mouth) don't seem to be fully joined. The soft palate is in these cases

cleft as well. In most cases, cleft lip is additionally present. congenital abnormality happens in concerning one in 700 live births worldwide

Cleft Lip

The cleft doesn't have an effect on the palate structure of the mouth it's named as birth defect. It is a kind of birth defect may be a microform cleft.[1] A microform cleft will seem as little as a bit dent in the red a part of the lip or appear as if a scar from the lip up to the nostril.[2] In some cases muscle tissue within the lip beneath the scar is affected and would possibly need surgery.[3] it's suggested to own newborn infants with a microform cleft checked with a craniofacial team as presently as attainable to work out the severity of the cleft.[4]

Cleft Palate

Palate cleft will occur as complete (soft and surface, presumably including a gap in the jaw) or incomplete (a 'hole' within the roof of the mouth, sometimes as a cleft soft palate). once birth defect happens, the flap is sometimes split. Cleft palate happens due to the failure of fusion of the lateral palatine processes, the septum, and/or the median palatine processes, that is failure in the formation of the secondary palate.The hole within the roof of the mouth caused by a cleft connects the mouth on to the cavum. AN open affiliation between the oral fissure and nasal cavity is named velopharyngeal inadequacy (VPI). due to the gap, air leaks into the cavum leading to a hypernasal voice resonance and nasal emissions whereas talking. possible treatment choices include therapy, medical specialty, augmentation of the posterior pharyngeal wall, lengthening of the surface, and surgical procedures.(5)

Cleft Lip and Palate

The primary palate has the following structures lip, alveolus, and anterior palate back to the incisive opening. The secondary palate consists of the exhausting and soft palates from the incisive opening back to the flap. The birth defect includes feeding difficulties, issues regarding to speech development, and therefore the chance of impaired facial growth. The breadth of a primary roof of the mouth cleft and therefore the degree of collapse square measure usually inflated within the presence of a cleft of the secondary roof of the mouth. The family is recommended concerning the anticipated inflated range of surgical operations which will be needed if a birth defect is present: primary birth defect repair with intravelarveloplasty; potential secondary surgery on the palatopharyngeal muscle sling, similar to a sphincteroplasty or tubular cavity flap; and potential orthognathic surgery at

skeletal maturity. The abnormal attachment of the muscles of the taste bud during a birth defect alters the strain on the tubular cavity voidance of the eustachian canal, increasing the incidence of ear infections. surgery and eyelet tube placement is performed within the majority of infants at the time of either the lip repair or the roof of the mouth repair to forestall the event of hearing abnormalities

Genetics of Cleft Lip and Palate

Cleft of the lip or palate is caused by several etiological factors. In massive of the causes they're caused by single mutant genes, some by chromosomal aberrations, some by environmental agents, and a few by interaction between the genetic and environmental variations. The first, second and third branchial arches play a vital role within the development of the face, mouth and tongue. The event of the face is well represented in terms of its formation and merging of varied processes or prominences. The frontonasal and jaw processes grow downward and forward however the mechanism of this interaction and its coordination is unclear. The secondary roof of the mouth could be a structure that separates the nasal passage from the oral fissure. The roof of the mouth correct develops from each primary and secondary elements. Sub-clinical phenotypes will embrace minor structural variants together with lip pits/prints, (6) dental anomalies,(7) defects of the orbicularis oris muscle,(8,9,10) facial image measure, brain variants as assessed by MRI(11,12)

Epidemiology and Etiopathogenesis

Among the cleft lip and surface population, the foremost common identification is cleft lip and surface at forty sixth, followed by isolated congenital anomaly at thirty third, then isolated cleft lip at twenty first. the bulk of bilateral cleft lips (86%) and unilateral cleft lips (68%) area unit related to a congenital anomaly. Unilateral clefts area unit nine times as common as bilateral clefts, and occur double as often on the left aspect than on the correct. Males area unit predominant within the harelip and surface population, whereas isolated congenital anomaly happens additional unremarkably in females. within the white population, cleft lip with or while not congenital anomaly happens in just about one in one,000 live births. These entities area unit double as common within the Asian population, and just about [*fr1] as common in African Americans. This racial nonuniformity isn't determined for isolated congenital anomaly, that has an overall incidence of zero.5 per 1,000 live births. each environmental teratogens and genetic factors area unit concerned within the genesis of cleft lip and surface. Intrauterine exposure to the medicament hydantoin is related to a 10-fold

increase within the incidence of cleft lip. Maternal smoking throughout physiological condition doubles the incidence of harelip. different teratogens, corresponding to alcohol, anticonvulsants, and retinoic acid, area unit related to malformation patterns that embody cleft lip and surface, however haven't been directly regarding isolated clefts. Genetic abnormalities may result in syndromes that embody clefts of the first or secondary palates among the organic process fields affected. quite four-hundredth of isolated cleft palates area unit a part of malformation syndromes, compared to below 15 August 1945 of harelip and surface cases.(13) The foremost common syndrome related to harelip and surface is van der Woude syndrome with or while not lower lip pits or blind sinuses. Microdeletions of body 22q leading to velocardiofacial, DiGeorge, or conotruncal anomaly syndromes area unit the foremost common diagnoses related to isolated congenital anomaly. though there's a recognized genetic part to nonsyndromic cleft lip and/or surface, it seems to be complex. Among different recent studies, a meta-analysis of thirteen order scans by Marazita et al. (2004) unconcealed multiple cleft lip/palate genes on sixteen body regions. oldsters with a toddler with a nonsyndromic cleft, or a case history of clefting, usually raise regarding their risk of clefts in ulterior pregnancies. the danger depends on whether or not the proband features a harelip aloneOvid: Grabb and Smith's cosmetic surgery, harelip with congenital anomaly (CLP), or a congenital anomaly alone (CP). If the family has one affected kid or parent with CLP, the danger of the kid of subsequent physiological condition having CLP is four. If 2 previous youngsters have CLP, the danger will increase to Sept. 11, and if one parent and one kid were antecedently affected, the danger to youngsters of ulterior pregnancies is Revolutionary Organization 17 November. For families with a history of CP, the danger of CP to youngsters of ulterior pregnancies is 2 hundredth if one antecedently affected kid, I Chronicles if 2 youngsters were antecedently affected, 6 June 1944 if one parent has CP, and 15 August 1945 if one parent and one previous kid have CP.(14)

In most cases, the reason for cleft lip and congenital anomaly is unknown. These conditions can not be prevented. Most scientists believe clefts area unit because of a mix of genetic and environmental factors. There seems to be a bigger likelihood of clefting during a newborn if a relative, parent, or relative has had the matter. Another potential cause could also be regarding a drugs a mother could have taken throughout her physiological condition. Some medication could cause harelip and congenital anomaly.(15) Among them: anti-seizure/anticonvulsant medications, disease of the skin medications containing Accutane, and antimetabolite, a drug unremarkably used for treating cancer, arthritis, and skin disorder.

cleftlip and congenital anomaly may additionally occur as a results of exposure to viruses or chemicals whereas the craniate is developing in uterus

Symptoms of Cleft Lip and Palate

Usually, a cleft — or split — within the lip or surface is instantly recognisable at birth. birth defect and {cleft surface|birthdefect|congenitalanomaly|congenitaldefect|congenitaldisorder|congenital abnormality} could seem as a split within the lip and roof of the mouth (palate) will|whichwill|that may} have an effect on one or each side of the face and a split within the lip which will seem as solely alittle notch within the lip or can extend from the lip through the higher gum and palate into the lowest of the nose then a split within the roof of the mouth (palate) that does not have an effect on the looks of the face. Less unremarkably, a cleft happens solely within the muscles of the palate (submucous cleft palate), that square measure at the rear of the mouth and lined by the mouth's lining. this kind of cleft typically goes forgotten at birth and will not be diagnosed till later once signs develop. Signs and symptoms of submucous congenital disorder could also be issue swallowing and nasal speaking voice and continual ear infections. (16)

Historical Perspective of Combined Presurgical and Surgical Treatments

Surgeons have long recognized the challenge of the bilateral cleft deformity. The most obstacles to the repair square measure the protrusive premaxilla and therefore the deficient columella. throughout the sixteenth, 17th, and eighteenth centuries, the surgical operation concerned excision of the premaxilla followed by a surgical union of the prolabium to the lateral lip segments. At a later age, prosthetic replacement of the anterior dentition was counseled to boost facial look. within the nineteenth century, surgeons finally accepted that excision of the premaxilla removed the higher incisors and underprivileged the lip of bony support, inflicting midface deficiency, jaw constriction, malocclusions, and a visible articulator prognathism.(17) The main focus became preservation and retraction of the premaxilla to attain best lip repair. 2 treatment philosophies evolved: surgical correction alone and surgical correction following presurgical orthopaedics.

Surgical Correction

Surgical choices for premaxillary retraction enclosed fracture of the bone, surgery of a part of the bone or septum, partial surgery of the anterior portion of the premaxilla, and a full-

thickness vertical incision of the septum, that allowed the proximal and distal segments to slip over one another. though these techniques achieved the first goal of retracting the premaxilla, they were related to important complications. each long-run clinical observations and animal studies incontestible that surgery of the septum made severe growth arrest of adjacent bones. The technique had alternative limitations, together with lingual inclination of higher incisors caused by a linguistically displaced premaxilla, nasal airway obstruction, and flat facies. Another surgical approach that tried to attain premaxillary retraction and lateral phase approximation was lip adhesion, that continues to be used at some centers. Johanson and Ohlsson (1961) delineated the employment of lip adhesion before primary bone attachment. (18)Millardrumored the employment of lip adhesion within the higher third of the congenital anomaly segments in preparation for the rotation-advancement technique of ultimate lip closure. Randall (1965), victimization short, broad, triangular flaps, claimed that closure of the soft tissues formed the underlying bony structures, reduced tension within the lip, and repositioned the alar base. Randall undermined the lateral lip if necessary once there was an outsized cleft gap. The disadvantages of lip adhesion embrace the risks of an extra surgery, scarring of the concerned tissue, loss of the native membrane flaps employed in some techniques for nasal lining repair, and potential organic phenomenon of the surgical web site. additionally, the stress of the surgically adhered lip over the 26/02/13 Ovid: Grabb and Smith's cosmetic surgery ovidsp.tx.ovid.com/sp-3.8.0b/ovidweb.cgi 8/44 alveolar segments is AN uncontrolled force that doesn't invariably align the segments in a perfect position, often inflicting collapse of the dental arches. Lip adhesion ought to be restricted to those cases within which the jaw segments ar swollen while not collapse. If the segments ar medially folded, lip adhesion is of no use and a way that gives alveolar growth is preferred.

several plastic surgeons were drawn to their surgical specialty when seeing their initial congenital anomaly repair. Cleft care stands out as a rare chance in cosmetic surgery to own an enormous impact on AN infant's future psychosocial well-being, and to follow these youngsters over the youth of their lives. The doc is reminded of the success, still as of the failure, of the surgeon's primary operations for years to return. trendy cleft surgical techniques, surgical orthodontia, and specialised multidisciplinary team care modify America to attain systematically favorable primary surgical results. Repair of secondary nasal deformities, however, remains a challenge, and continues to be best treated by preventative surgery at the time of the first repair.(19) Recent “inductive” techniques, admire nasoalveolarmolding and distraction osteogenesis, have improved cleft care over the past

decade, and as comparable advances in cosmetic surgery occur within the future, a toddler born with a cleft will forestall to fewer operations with higher aesthetic and useful results.

Timing of Surgery

The optimum temporal arrangement of congenital anomaly repair balances the good thing about traditional velopharyngeal operate to optimize speech development against the potential disadvantage of impaired facial growth secondary to early surgical trauma. Graber's description within the late Nineteen Forties of restricted jaw growth following early roof of the mouth closure was in the course of a recommendation to delay surgery till four to six years old-time. due to the harmful implications of this recommendation on speech development, the standard temporal arrangement for congenital anomaly repair was at random set at eighteen to twenty four months as a compromise between speech and facial growth. the present agreement, supported an accumulated understanding of speech development, is that congenital anomaly repair ought to be completed before eighteen months of age; but, there's no general agreement relating to however early the surgery may be performed. Since Graber's earlier work, there are variety of studies supporting the idea that impaired jaw growth in cleft patients is freelance of congenital anomaly repair, and might result from the lip repair alone or is additional a results of intrinsic restriction instead of early surgical trauma. Results from previous retrospective studies examining the impact of temporal arrangement of congenital anomaly repair on speech development area unit inconsistent and area unit compromised by little study numbers and potentialy unsupportive variables. Kirschner recently retrospectively reviewed Randall's and LaRossa's cases at Children's Hospital of city, exploitation 2 relativelly solid cohorts undergoing mouth repair either before or when seven months old-time, and located no vital good thing about early closure over later repair with regard to speech outcome. These authors emphasised the one factor that the surgical community agrees on: that long well-designed prospective studies area unit needed before the optimum temporal arrangement of congenital anomaly repair may be determined.(20) There area unit presently 2 common approaches to the temporal arrangement of congenital anomaly repair in North America: (a) two-stage repair, with the mouth repair and veloplasty performed at the time of lip adhesion or primary lip repair, and therefore the surface repaired before eighteen months, or delayed any with the utilization of Associate in Nursing prosthesis, and (b) single-stage repair round the age of eleven to twelve months. Our 2 centers apply the latter approach, delaying the surgery till the time once the kid starts to demonstrate the introduction of plosives (b, d, and g) in their speech. it's at now that they

need an intact velopharyngeal sphincter muscle to continue with traditional speech mechanics. In kids with airway problems, equivalent to that related to micrognathia of capital of South Dakota Robin sequence, the surgery may be delayed till age fourteen to eighteen months to permit any lower jawbone growth and to decrease the prospect of surgical airway compromise.

Conclusion

Because clefting causes very obvious physical changes, a congenital abnormality or birth defect is simple to diagnose. prenatal ultrasound will generally confirm if a cleft exists in AN unborn kid. If the clefting has not been detected in an ultrasound before the baby's birth, a physical examination of the mouth, nose, and surface confirms the presence of congenital abnormality or birth defect once a child's birth. generally diagnostic testing is also conducted to see or rule out the presence of alternative abnormalities. easy surgery is finished to treat cleft and surface once birth.

Corresponding Author

Saravana Pandian,

Senior Lecturer, Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id- sporthoresearch@gmail.com

Telephone Number: 9003101627

References

1. Kim EK, Khang SK, Lee TJ, Kim TG. Clinical features of the microform cleft lip and the ultrastructural characteristics of the orbicularis oris muscle. The Cleft Palate-Craniofacial Journal.2010 May;47(3):297-302.

2. Yuzuriha S, Mulliken JB. Minor-form, microform, and mini-microform cleft lip: anatomical features, operative techniques, and revisions. Plastic and reconstructive surgery. 2008 Nov 1;122(5):1485-93.

3. Tollefson TT, Humphrey CD, Larrabee WF, Adelson RT, Karimi K, Kriet JD. The spectrum of isolated congenital nasal deformities resembling the cleft lip nasal morphology.Archives of facial plastic surgery. 2011 May 16;13(3):152-60.

4. Sloan GM. Posterior pharyngeal flap and sphincter pharyngoplasty: the state of the art. The Cleft palate-craniofacial journal. 2000 Mar;37(2):112-22.

5.Neiswanger K, Chirigos KW, Klotz CM, Cooper ME, Bardi KM, Brandon CA, Weinberg SM, Vieira AR, Martin RA, Czeizel AE, Castilla EE. Whorl patterns on the lower lip are associated with nonsyndromic cleft lip with or without cleft palate. American Journal of Medical Genetics Part A. 2009 Dec 1;149(12):2673-9.

6. Neiswanger K, Walker K, Klotz CM, Cooper ME, Bardi KM, Brandon CA, Weinberg SM, Vieira AR, Martin RA, Czeizel AE, Castilla EE. “Whorl patterns on the lower lip are associated with nonsyndromic cleft lip with or without cleft palate” American Journal of Medical Genetics Part A, Volume 149A, Issue 12, pages 2673–2679, December 2009.

7. Vieira AR, McHenry TG, Daack-Hirsch S, Murray JC, Marazita ML. Candidate gene/loci studies in cleft lip/palate and dental anomalies finds novel susceptibility genes for clefts. Genetics in Medicine. 2008 Sep;10(9):668.

8. Neiswanger K, Weinberg SM, Rogers CR, Brandon CA, Cooper ME, Bardi KM, Deleyiannis FW, Resick JM, Bowen AD, Mooney MP, De Salamanca JE. Orbicularis oris muscle defects as an expanded phenotypic feature in nonsyndromic cleft lip with or without cleft palate. American Journal of Medical Genetics Part A. 2007 Jun 1;143(11):1143-9.

9. Weinberg SM, Neiswanger K, Richtsmeier JT, Maher BS, Mooney MP, Siegel MI, Marazita ML. Three- dimensional morphometric analysis of craniofacial shape in the unaffected relatives of individuals with nonsyndromic orofacial clefts: a possible marker for genetic susceptibility. American Journal of Medical Genetics Part A. 2008 Feb 15;146(4):409-20.

10. Nopoulos P, Berg S, Canady J, Richman L, Van Demark D, Andreasen NC. Structural brain abnormalities in adult males with clefts of the lip and/or palate.Genetics in Medicine. 2002 Jan;4(1):1.

11. Conrad AL, Dailey S, Richman L, Canady J, Karnell MP, Axelson E, Nopoulos P. Cerebellum structure differences and relationship to speech in boys and girls with nonsyndromic cleft of the lip and/or palate. The Cleft Palate-Craniofacial Journal. 2010 Sep;47(5):469-75.

12. Weinberg SM, Neiswanger K, Martin RA, Mooney MP, Kane AA, Wenger SL, Losee J, Deleyiannis F, Ma L, De Salamanca JE, Czeizel AE. The Pittsburgh Oral-Facial Cleft study: expanding the cleft phenotype. Background and justification.The Cleft palate-craniofacial journal. 2006 Jan;43(1):7-20.

13. Weinberg SM. Three-Dimensional morphometric analysis of the craniofacial complex in the unaffected relatives of individuals with nonsyndromic orofacial clefts (Doctoral dissertation, University of Pittsburgh).

14. Mitchell LE, Beaty TH, Lidral AC, Munger RG, Murray JC, Saal HM, Wyszynski DF. Guidelines for the design and analysis of studies on nonsyndromic cleft lip and cleft palate in humans: summary report from a Workshop of the International Consortium for Oral Clefts Genetics. The Cleft palate-craniofacial journal. 2002 Jan;39(1):93-100.

15. Adams MS, Niswander JD. Developmental 'Noise 'and a congenital malformation.Genetics Research. 1967 Dec;10(3):313-7.

16. Venkatesan A, Rajesh P. Epidemiology of cleft lip and cleft palate in south Indian population. Journal of Indian Association of Public Health Dentistry. 2003 Jan 1;3(3):6.

17. Nagappan N, John J. Periodontal status among patients with cleft lip (CL), cleft palate (CP) and cleft lip, alveolus and palate (CLAP) in Chennai, India. A comparative study. Journal of clinical and diagnostic research: JCDR. 2015 Mar;9(3):ZC53.

18. AlEmran SE, Fatani E, Hassanain JE. Craniofacial variability in parents of children with cleft lip and cleft palate.The Journal of clinical pediatric dentistry. 1999;23(4):337-41.

19. Anderson PJ, Moss AL. Dental findings in parents of children with cleft lip and palate. The Cleft palate-craniofacial journal. 1996 Sep;33(5):436-9.

20. Andres R, Bixler D, Shanks JC, Smith WL. Dominant inheritance of velopharyngeal incompetence.Clinical genetics. 1981 Jun 1;19(6):443-7.as

ClinicalSignificanceofHelicobacterPylorianditsRoleintheGastrointestinalTract

Hansini B. Ajantha[1],Pavithra Priyadarshoni S[2], Geetha RV[3].

Graduate Student[1],Tutor[2], Department of Public Health Dentistry, Assistant Professor[3],Department of Physiology.

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

H. pylori is a bacterial pathogen infecting the gastric antrum of half the population worldwide. It is found to be as the major cause of gastro duodenal pathologies, including gastric and duodenal ulcers, gastric cancer and gastric B cell lymphoma of mucosa associated lymphoid tissue. Although *H. Pylori* is disappearing worldwide, infection with *H. Pylori* remains one of the most common bacterial infections in all areas of the world infecting over 50% of the human race. This article describes about the relation of *Helicobacter pylori* with ulcers, gastritis and other upper abdominal infections and focuses on many aspects of the infection that are relevant to the clinician.

Key Words: *H. Pylori, Ulcers, Gastritis, Inflammation, Triple Therapy.*

Introduction

Dyspepsia refers to a constellation of upper gastrointestinal symptoms that occur mainly in adults and is one of the most common reasons for referral to any gastrointestinal unit. It has been known for a long period of time, that bacteria are present in the human stomach. These bacteria were thought to be contaminants from digested food rather than true gastric colonizers. Barry Marshall and Robin Warren described the successful isolation and culture of a spiral bacterial species, known as *Helicobacterpylori*, from the human stomach. The discovery of gastric microbiota and the first successful culture of *Helicobacter pylori* by Marshall and Warren opened a new chapter in human medicine (1). *Helicobacter pylori* is a small, curved, highly motile, gram negative bacillus that colonises only the mucus layer of the human stomach. Since the discovery of this bacteria in 1984, it has been recognised as the principal cause of peptic ulcer disease and as the main risk factor for the development of gastric cancer. The role of *H. pylori* infection in gastric cancers is increasingly recognized, and its role in other diseases of the upper gastrointestinal tract is being evaluated. Enormous progress has been achieved in determining the pathogenesis of this infection. Effective antimicrobial therapy is available, and indications for therapy continue to evolve. This review surveys scientific knowledge concerning *H. pylori* and focuses on the many aspects of the infection caused by this bacteria that are relevant to the clinician.

Morphology

Helicobacter pylori is a helix shaped, gram negative bacterium about 3 micrometres long with a diameter of about 0.5 micrometres. It is microphillic in nature; that is, it requires oxygen, but at lower concentration than found in the atmosphere. It contains hydrogenase which can be used to obtain energy by oxidizing molecular hydrogen produced by intestinal bacteria (2). It produces oxidase, catalase and urease. This bacteria has the ability to convert from spiral to a possibly non culturable coccoid form. (3) Like any other gram negative bacteria, the outer membrane of*H. pylori* consists of phospholipids and lipopolysaccharides. The organism has 2 to 6 unipolar, sheathed flagella of approximately 3 micrometre in length, which often carry a distinctive bulb at the end. The flagella confer motility and allow rapid movement in viscous solutions such as the mucus layer overlying the gastric epithelial cells (4). In contrast to many other pathogens of the gastrointestinal tract, it lacks fimbrialadhesins.

Cultivation

A key feature of *H. pylori* is its microaerophilicity, with optimal growth at oxygen levels of 2 to 5% and the additional need of 5 to 10% CO2 and high humidity. There is no need for hydrogen, although it is not detrimental to growth. Many laboratories utilize standard microaerobic conditions of 85% nitrogen, 10% carbon dioxide, and 5% oxygen for *H. pylori* culture. Growth occurs at 34 to 40°C, with an optimum of 37°C. Although its natural habitat is the acidic gastric mucosa, *H. pylori* is considered to be a neutralophile. The bacterium will survive brief exposure to pHs of 4.

But, the growth occurs only at the relative pH range of 5.5 to 8.0, with optimal growth at neutral pH (5). *H. pylori* is a fastidious microorganism and hence, it requires complex growth media. Often these growth media are supplemented with blood or serum. They may act as additional sources of nutrients and possibly also protect against the toxic effects of long-chain fatty acids. The most commonly used solid media for routine isolation and culture of *H. pylori* consist of Columbia or brucella agar supplemented with either lysed horse or sheep blood. For primary isolation and also for routine culture, selective antibiotic mixtures are available. Growth of *H. pylori* in chemically defined media has been reported (6), but these are not suitable for routine growth and isolation of *H. pylori*. Most of the commercially available synthetic media, such as tissue culture media, do not support the growth of *H. pylori* without the addition of serum. Isolation of *H. pylori* from gastric biopsy samples is difficult and not always successful.

Epidemiology

H pylori is one of the commonest bacterial pathogens in humans. The prevalence of infection varies but has less incidence in most developed countries. Seropositivity increases with age and low socioeconomic status. Retrospective studies have shown a cohort effect consistent with the hypothesis that infection is mainly acquired in early childhood. Until recently, however, it has been difficult to assess accurately the incidence or route of infection because of the inaccuracy and cost of detecting (non- invasively)*H pylori* in young children. Primary acquisition of the infection in adults and also secondary infection after successful eradication, does occur, but is less common. Sources regarding the mechanism by which, *H pylori* is usually acquired and its route of transmission are less. Since humans are the only known reservoir of infection, it is likely that, *H pylori* may also be picked up from siblings, other

children, or parents, predominantly through the gastro- oral route.*H pylori* infection is an occupational hazard for gastroenterologists and is associated with performing endoscopy (7).

Pathogenesis

The gastric mucosa is well protected against bacterial infections. *H. pylori* is highly adapted to this ecologic setting, with a unique array of features that permit entry into the mucus, swimming and spatial orientation in the mucus, attachment to epithelial cells, evasion of the immune response, and, as a result, persistent colonization and transmission. The *H. pylori* genome (1.65 million bp) codes for about 1500 proteins. (8,9) Among the most remarkable findings of two *H. pylori* genome-sequencing projects were the discovery of a large family of 32 related outer membrane proteins that includes most known *H. pylori* adhesins. The genome of *H. pylori* changes continuously during chronic colonization of an individual host by importing small pieces of foreign DNA from other *H. pylori* strains during persistent or transient mixed infections. (10, 11) After being ingested, the bacteria have to evade the enter the mucous layer. Urease production and motility are essential for this first step of infection. Urease hydrolyzes urea into carbon dioxide and ammonia, thereby permitting *H. pylori* to survive in an acidic environment. (12) Motility is essential for colonization, and *H. pylori* flagella have adapted to the gastric environment. *H. pylori* can bind tightly to epithelial cells by multiple bacterial surface components. The best-characterized adhesin, BabA, is a 78-kD outer-membrane protein that binds to the fucosylated Lewis B blood group antigen. (13) Accumulating evidence in animal models suggests that adhesion, particularly by BabA, is relevant in *H. pylori* associated disease (14) and may influence disease severity. The majority of *H. pylori* strains express the 95-kD vacuolatingcytotoxinVacA, a secreted exotoxin. (15) The toxin inserts itself into the epithelial-cell membrane and forms a hexameric anion-selective, voltage dependent channel through which bicarbonate and organic anions can be released. (16) VacA is also targeted to the mitochondrial membrane, where it causes release of cytochrome *c* and induces apoptosis. (17) Most strains of *H. pylori* possess the *cag* pathogenicity island (*cag*-PAI), a 37-kb genomic fragment containing 29 genes. (18) Several of these encode components of a predicted type IV secretion apparatus that translocates the 120-kD protein CagA into the host cell. (19) After entering the epithelial cell, CagA is phosphorylated and binds to SHP-2 tyrosine phosphatase, leading to a growth factor–like cellular response and cytokine production by the host cell. In order to avoid the acidic environment of the lumen of the stomach, *H. pylori* uses its flagella to burrow into the mucus

lining of the stomach to reach the epithelial cells lying underneath, where there is a more neutral pH (20). *H. pylori* is able to sense the pH variants in the mucus lining and move towards less acidic region. Hence, *H. Pylori* can perform chemotaxis. This also prevents the bacteria from being swept away into the lumen, as it is constantly moving from its site of origin at the epithelium to its dissolution at the lumen interface. In addition, *H. pylori* also neutralizes the acid in its environment. It does this by producing large amounts of urease, which breaks down the urea present in the stomach to carbon dioxide and ammonia, resulting in neutralisation of stomach acid.

Inflammation, GastritisandUlcers

H. pylori harms the stomach and duodenal linings by various mechanisms. The ammonia produced to regulate pH levels is toxic to epithelial cells. Similarly, biochemicals produced by *H. pylori* such as proteases, vacuolatingcytotoxin A (VacA) and certain phospholipases are also harmful (21). Cytotoxin associated gene [CagA] can also cause inflammation and is potentially a carcinogen (22). Colonization of the stomach by *H. pylori* can result in chronic gastritis, an inflammation of the stomach lining, at the site of infection. Chronic gastritis is likely to underlie *H. pylori*-related diseases (23). Ulcers in the stomach and duodenum result when the consequences of inflammation allow stomach acid and the digestive enzyme pepsin to overwhelm the mechanisms that protect the stomach and duodenal mucous membranes. The location of colonization of *H. pylori*, which affects the location of the ulcer, depends on the acidity of the stomach (24). In people producing huge amounts of acid, *H. pylori* colonizes near the pyloric antrum to avoid the acid-secreting parietal cells at the fundus (25). In people producing normal or reduced amounts of acid, *H. pylori* colonises the remaining area of the stomach. The inflammatory response caused by the bacteria colonising near the pyloric antrum induces G cells present there to secrete the hormone gastrin, which travels through the bloodstream to parietal cells in the fundus (26). Gastrin stimulates the parietal cells to secrete more acid into the stomach lumen, and also increases the number of parietal cells. The increased acid content damages the duodenum, which may eventually result in ulcer in the area of the duodenum.

GastricCarcinoma

Two related mechanisms by which *H. pylori* could promote cancer are under investigation. One mechanism involves the enhanced production of free radicals and an increased rate of host cell mutation. The other mechanism is known as perigenetic pathway (27), and involves enhancement of the transformed host cell phenotype by means of alterations in cell proteins, such as adhesion proteins. *H. pylori* has been proposed to induce inflammation at locally high levels of TNF-α and interleukin 6 (IL-6). According to the proposed perigenetic mechanism, inflammation associated signalling molecules, such as TNF-α and interleukin 6, can alter gastric epithelial cell adhesion and lead to the dispersion and migration of mutated epithelial cells without the need for additional mutations in tumor suppressor genes, such as genes that code for cell adhesion proteins (28).

Diagnosis

Various tests have been developed for the detection of *H. pylori*, and each one have their specific advantages and disadvantages. The available tests to detect *H. pylori infection* are divided into invasive tests, based on gastric specimens for histology, culture, or other methods, and noninvasive tests, based on peripheralsamples, such as blood, breath samples, stools, urine, or salivafor detection of antibodies, bacterial antigens, or urease activity.The choice of a specific test for an individual patient dependson local experience and the clinical setting. In research protocols, a combination of two methods is often applied. In daily clinicalpractice, use of a single test is generally adequate, and mosttests are sufficiently accurate to be used for this purpose. Forroutine diagnostic purposes, histology, urea breath testing, andculture are currently most often used. In hospital-based care, many patients undergo endoscopy, which is then combined with an invasive test for *H.pylori*. Otherwise, breath tests and serology are commonly used. For children, fecal antigen tests offer the opportunity to assess *H. pylori* status without the need for endoscopy. (29)

Invasive Tests

H. pylori can be detected by endoscopy, histology, culture, or urease tests. All these biopsy based methods for detecting *H pylori* are liable to sampling error. After partially effective eradication treatment, low levels of infection can easily be missed by endoscopic biopsy. Proton pump inhibitors may affect the pattern of *H. pylori* colonization in the stomach and compromise the accuracy of antral biopsy. Hence, multiple biopsies are taken from different

sites in the antrum for histology and for one other method, either culture or urease testing. (29)

Histology

Although *H pylori* may be recognised on sections stained with haematoxylin and eosin alone, supplementary stains such as Giemsa, Genta, Gimenez,Warthin- Starry silver, Creosyl violet are needed to detect low levels of infection and to show the characteristic morphology of *H pylori*. An important advantage of histology is that, sections from biopsies can be examined at any time, in addition to the historical record provided. Biopsy specimens from other parts of the stomach can be retained in formalin to be processed only if antral histology is inconclusive. (29)

Culture

Microbiological isolation and growth of the species is the theoretical gold standard test for identifying any bacterial infection, but culture of *H pylori* can be unreliable. Risks of overgrowth or contamination make it the least sensitive method of detection, and it is the least available test for use with endoscopy. The prevalence of multi resistant strains makes it increasing likely that culture and antibiotic sensitivity testing may become a prerequisite for patients with persistent infection after initial or repeated treatment failure. (29)

Urease tests

They are quick and simple tests for detecting *H pylori* infection but indicate only the presence or absence of infection. However, the sensitivity of urease tests is often higher than that of other biopsy based methods because the entire biopsy specimen is placed in the media, hence the additional sampling or processing error associated can be avoided with histology or culture. (29)

Non-Invasive Tests

Serology

H pylori infection can elicit a local mucosal and a systemic antibody response. Circulating IgG antibodies to *H pylori* can be detected by enzyme linked immuno sorbent assay (ELISA) or latex agglutination tests. These tests are generally simple, reproducible, and can be done on stored samples. Individuals may differ considerably in their antibody responses to *H pylori*

antigens, and no single antigen is recognised by sera from all subjects. The accuracy of serological tests therefore depends on the antigens used in the test. Antibody titres fall only slowly after successful eradication, so serology cannot be used to determine *H pylori* eradication or to measure secondary infection rates. An important advantage of serological methods over other testing methods for *H. pylori* infection has been the development of simple finger prick tests, to detect the presence of *H. pylori* immunoglobulins. (30)

Urea Breath Test

Non- invasive detection of*H. pylori* by the 13C- urea breath test isbased on the principle that a solution of urea labelled withcarbon- 13 will be rapidly hydrolysed by the urease enzyme of*H pylori*. The resulting carbon dioxide is absorbed across the gastricmucosa and hence, through the systemic circulation, excreted ascarbon dioxide in the expired breath. The 13C- urea breath test detectscurrent infection and is not radioactive. It can be used as ascreening test for *H pylori*, to assess eradication and to detectinfection in children. (31)

FaecalAntigen Test

In the stool antigen test a simple sandwich ELISA is used todetect the presence of *H pylori* antigens shed in the faeces.This test have sensitivities and specificities similar tothose of the 13C- urea breath test, and the technique hasthe potential to be developed as a near patient test. (31)

Treatment

Once *H. pylori* is detected in a person with a peptic ulcer after undergoing various test methods mentioned above, the normal procedure is to eradicate the infection and allow the ulcer to heal. The eradication of *H. pylori* is strongly recommended in all patients with peptic ulcer, including those with complications.It is advised that *H. pylori* eradication is considered to be an appropriate option in patients,with functional dyspepsia, as it leads to long term symptom improvement in a subset of patients. There was consensus that the eradication of H. pylori is not associated with the development of gastro-oesophageal reflux disease in most cases, and does not exacerbate existing gastro-oesophageal reflux disease. It was agreed that the eradication of *H. pylori* prior to the use of non-steroidal anti-inflammatory drugs reduces the incidence of peptic ulcer, but does not enhance the healing o gastric or duodenal ulcer in

patients. Treatment for *H. pylori* infection should be considered with first and second line eradication therapies together. First line therapy also known as triple therapy, is done using a proton pump inhibitor or ranitidine bismuth citrate, combined with clarithromycin and amoxicillin or metronidazole. Second line therapy should use quadruple therapy with a proton pump inhibitor, bismuth, metronidazole and tetracycline. Where bismuth is not available, second-line therapy should be with proton pump inhibitor-based triple therapy. (32)

Conclusion

H. pylori continues to be one of the most common bacterial infections in humans. Functional genomics may fill many of the gaps in our understanding of the pathogenesis of *H. pylori* infection and accelerate the development of novel therapies. Associations between bacterial characteristics and disease risks have not yet been defined sufficiently well to guide the clinician in treatment decisions. Prophylactic and therapeutic vaccination have been successful in animal models, but the translation to a human vaccine remains difficult. All of these developments will probably be needed to prevent and treat this infection in areas of the world where there is a high prevalence of chronic infection of *H. pylori.* Early diagnosis and eradication of *H. Pylori* not only improves symptoms but also help to prevent complications associated with *H. Pylori* infections.

Corresponding Author
S. PavithraPriyadarshoni,

Tutor, Department of Public Health Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id: drpavithraphd88@gmail.com

References

1. Chitra P, Chandrika C, Kannan AS, Srinath S, Srinivasan V, Jayanthi V. Clinical and life style variables in functional dysplasia and its subtypes. Trop gastro enteral. 2012 Jan-Mar; 33(1):33-8.
2. Olson JW, Maier RJ. "Molecular hydrogen as an energy source for Helicobacter pylori". Science. November 2002: 298 (5599): 1788–90.
3. Chan WY, Hui PK, Leung KM, Chow J, Kwok F, Ng CS. "Coccoid forms of Helicobacter pylori in the human stomach". Am J ClinPathol. October 1994:102 (4): 503–7.
4. O'Rourke JL, Solnick JV, Neilan BA, Seidel K, Hayter R, Hansen LM, Lee A. Description of 'Candidatus Helicobacter heilmannii'based on DNA sequence analysis of 16S rRNA and urease genes. International journal of systematic and evolutionary microbiology. 2004 Nov 1;54(6):2203-11.
5. Scott, D. R., E. A. Marcus, D. L. Weeks, and G. Sachs. Mechanisms of acid resistance due to the urease system of Helicobacter pylori. Gastroenterology. 2000: 123:187–195.
6. Reynolds, D. J., and C. W. Penn Characteristics of Helicobacter pylori growth in a defined medium and determination of its amino acid requirements. Microbiology 1994: 140:2649–2656.
7. Perez-Perez, G. I., D. Rothenbacher, and H. Brenner. Epidemiology of Helicobacter pylori infection. Helicobacter 2004: 9:1:1–6.
8. Tomb JF, White O, Kerlavage AR. The complete genome sequence of the gastric pathogen Helicobacter pylori. Nature 1997; 388:539-47.
9. Alm RA, Ling LS, Moir DT. Genomic-sequence comparison of two unrelated isolates of the human gastric pathogen Helicobacter pylori. Nature 1999; 397:176-80.
10. Falush D, Kraft C, Taylor NS. Recombination and mutation during long-term gastric colonization by Helicobacter pylori: estimates of clock rates, recombination size, and minimal age. Proc Natl AcadSci U S A 2001; 98:15056-61.
11. Suerbaum S, Smith JM, Bapumia K. Free recombination within Helicobacter pylori. Proc Natl AcadSci U S A 1998; 95:12619-24.
12. Mobley HLT. Helicobacter pylori urease. In: Achtman M, Suerbaum S, eds. Helicobacter pylori: molecular and cellular biology. Wymondham, United Kingdom: Horizon Scientific Press, 2001:155-70.

13. Ilver D, Arnqvist A, Ogren J. Helicobacter pylori adhesin binding fucosylatedhisto-blood group antigens revealed by retagging. Science 1998; 279:373-7.
14. Guruge JL, Falk PG, Lorenz RG. Epithelial attachment alters the outcome of Helicobacter pylori infection. Proc Natl AcadSci U S A 1998; 95:3925-30.
15. Montecucco C, Papini E, de Bernard M. Helicobacter pylori VacAvacuolatingcytotoxin and HP-Nap neutrophil activating protein. In: Achtman M, Suerbaum S, eds. Helicobacter pylori: molecular and cellular biology. Wymondham, United Kingdom: Horizon Scientific Press, 2001:245-63.
16. Szabo I, Brutsche S, Tombola F. Formation of anion-selective channels in the cell plasma membrane by the toxin VacA of Helicobacter pylori is required for its biological activity. EMBO J 1999; 18:5517-27.
17. Galmiche A, Rassow J, Doye Al. The N-terminal 34 kDa fragment of Helicobacter pylori vacuolatingcytotoxin targets mitochondria and induces cytochrome c release. EMBO J 2000; 19:6361-70.
18. Censini S, Lange C, Xiang Z. cag, A pathogenicity island of Helicobacter pylori, encodes type I-specific and disease-associated virulence factors. Proc Natl AcadSci U S A 1996; 93:14648-53.
19. Odenbreit S, Puls J, Sedlmaier B, Gerland E, Fischer W, Haas R. Translocation of Helicobacter pylori CagA into gastric epithelial cells by type IV secretion. Science 2000; 287:1497-500.
20. Harshini AK. Adhesion in Bacteria. International Journal of Science and Research. August 2017: 6 (8): 279-281.
21. Smoot DT. "How does Helicobacter pylori cause mucosal damage? Direct mechanisms". Gastroenterology. December 1997: 113 (6): S31–4; discussion S50.
22. Shiotani A, Graham DY. "Pathogenesis and therapy of gastric and duodenal ulcer disease". Med. Clin. North Am. November 2002: 86 (6): 1447–66,
23. Dixon MF. "Patterns of inflammation linked to ulcer disease". Baillieres Best Pract Res ClinGastroenterol. February 2000: 14 (1): 27–40.
24. Kusters JG, van Vliet AH, Kuipers EJ. "Pathogenesis of Helicobacter pylori Infection". ClinMicrobiol Rev. July 2006: 19 (3): 449–90.
25. Blaser MJ, Atherton JC. "Helicobacter pylori persistence: biology and disease". J. Clin. Invest. February 2004: 113 (3): 321–33.

26. Schubert ML, Peura DA. "Control of gastric acid secretion in health and disease". Gastroenterology. June 2008: 134 (7): 1842–60.
27. Tsuji S, Kawai N, Tsujii M, Kawano S, Hori M. "Review article: inflammation-related promotion of gastrointestinal carcinogenesis--a perigenetic pathway". Aliment. Pharmacol. Ther. July 2003: 18(1): 82–9.
28. Suganuma M, Yamaguchi K, Ono Y. "TNF-α-inducing protein, a carcinogenic factor secreted from H. pylori, enters gastric cancer cells". Int. J. Cancer. July 2008: 123 (1): 117–22.
29. Logan RP, Walker MM. ABC of the upper gastrointestinal tract: Epidemiology and diagnosis of Helicobacter pylori infection. BMJ: British Medical Journal. 2001 Oct 20; 323(7318):920.
30. Deepa T, Thirrunavukkarasu N. Saliva as a potential diagnostic tool. Indian journal of medical sciences. 2010 Jul 1; 64(7):293.
31. Stenström B, Mendis A, Marshall B. "Helicobacter pylori—The latest in diagnosis and treatment". AustFam Physician. August 2008: 37 (8): 608–12.
32. Malfertheiner P, Megraud F, O'Morain C. "Current concepts in the management of Helicobacter pylori infection: the Maastricht III Consensus Report". Gut. June 2007. 56 (6): 772–81.

Clotting Factors and Artificial Agents – A Review

Nivedha Chezhian [1], Gayathri Devi [2], S.Niveda [3]

Graduate Student [1],Assistant Professor [2],Department of Physiology

Tutor [3],Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

The variety of wound types has resulted in a wide range of wound dressings with new products frequently introduced to target different aspects of the wound healing process. The ideal dressing should achieve rapid healing at reasonable cost with minimal inconvenience to the patient. Coagulation involves the regulated sequence of proteolytic activation of a series of zymogens to achieve appropriate and timely haemostasis in an injured vessel, in an environment that overwhelmingly favours an anticoagulant state. In the non-pathological state, the inciting event involves exposure of circulating factor VII/VIIA to extravascularly expressed tissue factor, which brings into motion the series of steps which results in amplification of the initial stimulus, culminating in the conversion of fibrinogen to fibrin and clot formation.Excessive surgical bleeding causes hypovolaemia, hemodynamic instability, anaemia and reduced oxygen delivery to tissues, with a subsequent increase in postoperative morbidity and mortality. The role of anaesthetists in managing surgical blood loss has increased greatly in the last decade.

Key Words: *Anticoagulants, Blood, Haemostasis, Factor VIIa, Wound healing.*

Introduction

Wound dressings and agentsplay an important role in medical and pharmaceutical wound care worldwide. In the past, traditional dressings like natural or synthetic bandages, cotton wool, lint and gauzes all with varying degrees of absorbency were used for the management of wounds. Improper clotting may occur as result of defects in blood components such as platelets and/or clotting proteins, also called clotting factors [1-5].

Their primary function is to keep the wound dry allowing evaporation of wound exudates and preventing the entry of harmful bacteria into the wound. It has now been shown however, that having a warm moist wound environment achieves more rapid and successful wound healing. Effective wound healing depends on the number of different factors such as the type of wound being treated, the healing process, patient conditions in terms of health , environment and social setting, and the physical chemical properties of the available dressings.

Skilful surgery combined with blood saving methods and careful management of blood coagulation will all help reduce unnecessary blood loss and transfusion requirements. Excessive surgical bleeding causes hypovolaemia, hemodynamic instability, anaemia and reduced oxygen delivery to tissues with a subsequent increase in postoperative morbidity and mortality [1]. Dialysis membranes and lines are inherently pro-coagulant and activate both the intrinsic and extrinsic pathways of coagulation, as well as platelets and other circulating cellular elements. To provide safe and effective dialysis, appropriate anticoagulant measures must be applied. Haemodialysis, including anticoagulation, is prescribed by dialysis doctors but delivered by dialysis nurses [2]. The agents commonly used in clinical practice for anticoagulation during haemodialysis are unfractionated heparin (UF heparin) and low-molecular-weight heparin (LMWH). When bleeding is the consequence of a specific defect of haemostasis the goal of treatment is to correct the defect. A typical example is the replacement of factor viii by transfusion in patients with haemophilia. Specific treatment may be impossible. However, because bleeding may result from multiple defects or because no cause can be identified. In such situations non-transfusion drugs that help to stop bleeding are indicated [3, 4]. These drugs may also be indicated for patients to refuse blood transfusion or for those who undergo surgical procedures associated with large blood losses necessitating many transfusions of donated blood.

Coagulation Cascade

After injury to a vessel wall, tissue factor is exposed on the surface of the damaged endothelium. The interaction of tissue factor with plasma factor vii activates the coagulation cascade, producing thrombin by stepwise activation of a series of proenzymes (thrombin generation.). Thrombin is central in the clotting process: it converts soluble fibrinogen to fibrin; activates factors v, viii, and xi, which generates more thrombin; and stimulates platelets. The normal state of the circulatory system is that of a series of conduits through which blood flows in a liquid phase, until vascular injury occurs [5, 6]. At which time it is appropriate for haemostasis to temporarily seal the defect. In the process of resolution of the vascular injury, dissolution of the haemostatic plug should also concurrently take place. The different mechanisms involved are that of a system which is overwhelmingly tipped in favour of an anticoagulant state, and except in some pathological circumstances, only with appropriate stimulation is haemostasis able to occur [7]. The mechanisms in favour of blood flowing in a liquid phase include the following:

(I) The flow of blood itself diluting any activated or prothrombotic factors;

(II) The normal laminar flow of blood through normal vasculature which prevents platelets from directly contacting the endothelium by way of a thin layer of plasma;

(III) The expression of endothelial anti-platelet and anticoagulant factors in excess of procoagulant factors;

(IV) The need for adequate stimulation to produce activated platelets, requiring multiple stimuli for full activation;

(V) The state of the circulating coagulation factors as a series of inactive zymogens, with some factors also bound to other cells or inactivating complexes, requiring receptor-mediated proteolysis for activation; and

(VI) The multiple circulating proteases which serve to degrade any rogue factors that are activated in the presence of a sub maximal stimulus.

Wound Healing

The first stage of wound management should be a thorough assessment of the wound and the patient. The process begins with a diagnosis of the wound's aetiology and continues with optimizing the patient's medical condition, particularly blood flow to the wound area. Woundhealing is a specific biological process related to the general phenomenon of growth and tissue regeneration [6].This article relates to wound management and the choice of wound dressings. It is referred to the biological and physiological texts and literature for detailed scientific expositions [8]. Wound healing progresses through a series of interdependent and overlapping stages in which a variety of cellular and matrix components act together to re establish the integrity of damaged tissue and replacement of lost tissue[9][10]. Wound healing process has been reviewed and described by 'Schultz [8] as comprising five overlapping stages that involve complex biochemical and cellular processes. Thomashas described wound exudates as: 'a generic term given to liquid produced from chronic wounds, fistulae or other more acute injuries once haemostasis has been achieved'[11]. In certain conditions that include chronic wounds, there is excessive amount of exudates present which can lead to complications. Excess exudates results from oedema caused by inflammation, reduced mobility and venous or lymphatic insufficiency [12]. Increased exudates levels may also be the result of liquefying hard and Escher-like necrotic tissue to produce a wet and sloughy mass by a process known as autolysis debridement.

The migration phase involves the movement of epithelial cells and fibroblasts to the injured area to replace damaged and lost tissue. These cells regenerate from the margins, rapidly growing over the wound under the dried scab (clot) accompanied by epithelial thickening [13].

The proliferative phase occurs almost simultaneously or just after the migration phase (Day 3 onwards) and basal cell proliferation, which lasts for between 2 and 3 days. Granulation tissue is formed by the in-growth of capillaries and lymphatic vessels into the wound and collagen is synthesised by fibroblasts giving the skin strength and form. By the fifth day, maximum formation of blood vessels and granulation tissue[3, 4].

This phase known as remodelling phase involves the formation of cellular connective tissue and strengthening of the new epithelium which determines the nature of the final scar. Cellular granular tissue is changed to an acellular mass from several months up to about 2 years [13].

Haemostasis

Bleeding usually occurs when the skin is injured and serves to flush out bacteria and/or antigens from the wound. In addition, bleeding activates haemostasis which is initiated by exudates components such as clotting factors [12]. The currently accepted model of in vivo coagulation highlights the central importance of tissue factor as the main instigator of coagulation, while emphasising the rapid amplification of thrombin as an essential step in the development of a stable clot[14], and the interdependence of coagulation factors and cellular elements. Thrombin is obtained from bovine plasma. Thrombin therapy is restricted to local application in oozing of blood[15]. Haemostasis depends on a successful balance between the coagulation, complement and fibrinolytic pathways, with complex interactions between plasma proteins, platelets, blood flow and viscosity, and the endothelium. Formation of the primary haemostatic plug at the site of a damaged vessel is the first event in the control of bleeding. Von willebrand factor binds to the exposed subendothelium, exposing multiple intrinsic binding sites for a specific platelet membrane structure termed GPIB. This is followed by platelet activation, adhesion and generation of a platelet plug [16, 17].

Haemodialysis and Clotting

Haemodialysis involves the circulation of whole blood through a dialysis circuit and artificial kidney (dialyser) both of which have the tendency to activate coagulation pathways. Bleeding usually occurs when the skin is injured and serves to flush out bacteria and/or antigens from the wound. In addition, bleeding activates haemostasis which is initiated by exudates components such as clotting factors [18]. Fibrinogen in the exudates elicits the clotting mechanism resulting in coagulation of the exudates. The dialyser is generally constructed of synthetic microfibers with narrow lumen, lacking endothelial lining and experiencing disordered flow – including both shear and turbulence. Factors that determine the thrombogenicity of different dialysis membranes include chemical composition, charge, ability to adhere or activate circulating cellular elements (including platelets) and other

characteristics which activate thrombotic pathways[19]. The dialysis circuit also has a synthetic composition and artificial surfaces with dead spaces, stasis, turbulence and an air interface in the venous bubble trap. Clotting in the dialysis circuit is triggered by both the extrinsic and the intrinsic pathways at the same time but to different degrees depending on the composition of the dialysis membrane and design and composition of the lines. Once the blood flow is initiated, plasma proteins deposit on the dialyser surface, and factor xii and high-molecular-weight kallikrein accumulate and act as initiating factors for contact coagulation – the intrinsic pathway [20]. Peripheral blood leucocytes and monocytes, which contact the dialyser membrane, become adherent or activated and release blebs of surface membrane rich in tissue factor – activating the extrinsic pathway[21].

Wound Dressing

Dressings are classified in a number of ways basedon their function in the wound they are classified into debridement, antibacterial, occlusive, absorbent, adherence, [22] and based on type of material employed to produce the dressing it can be hydrocolloid, alginate, collagen and the physical form of the dressing as ointment, film, foam, gel. Dressings are also further classified into primary, secondary and island dressings [23, 24].

Hydrocolloid dressings are among the most widely used dressings. The role of hydrocolloid dressings, their properties, mechanism of action and the range of wounds for which they are useful have been reviewed[25, 26]. The term ‘hydrocolloid’ describes the family of wound management products obtained from colloidal (gel forming agents) materials combined with other materials such as elastomers and adhesives. Typical gel forming agents include carboxymethylcellulose (CMC), gelatine and pectin. Hydrocolloid dressings are used for light to moderately exuding wounds such as pressure sores, minor burns and traumatic injuries[27].

Alginate dressings are produced from the calcium and sodium salts of alginic acid, a polysaccharide comprising mannuronic and guluronic acid units. Alginate dressings occur either in the form of freeze-dried porous sheets (foams) or as flexible fibres, the latter indicated for packing cavity wounds [26]. The use of alginates as dressings stems primarily from their ability to form gels upon contact with wound exudates (high absorbency). The

high absorption occurs via strong hydrophilic gel formation, which limits wound secretions and minimises bacterial contamination [27, 28].

Biological dressings are made from biomaterials that play an active part in the wound healing process and sometimes referred to as ‘bioactive dressings’. Bioactive wound healing dressings also include tissue engineered products derived from natural tissues or artificial sources [29]. These technologies usually combine polymers such as collagen, hyaluronic acid, chitosan, alginates and elastin. Biomaterials have the advantage of forming part of the natural tissue matrix, are biodegradable and some play an active part in normal wound healing and new tissue formation[30, 31].

The purpose of applying antibiotics and other antibacterial is mainly to prevent or combat infections especially for diabetic foot ulcers[32] surgical and accident wounds where the incidence of infections can be high due to reduced resistance resulting from extreme trauma. In some cases, the delivery of certain antibiotics from paraffin based ointments such as bismuth subgallate are known to take active part in the wound healing process[33, 34]. Common antibiotics incorporated into available dressings for delivery to wounds include dialkylcarbamoylchloride[35].

Whilst antibacterial agents prevent or treat infections and can aid in wound healing, they do not necessarily take an active physiological part in the wound healing process. Growth factors are involved with cell division, migration, differentiation, protein expression and enzyme production [36]. The wound healing properties of growth factors are mediated through the stimulation of angiogenesis and cellular proliferation, which affects both the production and the degradation of the extracellular matrix and also plays a role in cell inflammation and fibroblast activity [37]. Growth factors therefore affect the inflammatory, proliferation and migratory phases of wound healing [38, 39]. A variety of growth factors have been reported which participate in the process of wound healing including, epidermal growth factor (EGF), platelet derived growth factor (PDGF), fibroblast growth factor (FGF), transforming growth factor (TGF-β1), insulin-like growth factor (IGF-1), human growth hormone and granulocyte-macrophage colony-stimulating factor [40].

Several factors apart from the choice of wound dressings need to be considered to ensure successful wound healing. In the case of chronic wounds, underlying factors such as disease,

drug therapy and patient circumstance must all be reviewed and addressed before a particular wound dressing is applied [41].

Debridement

It is important to remove necrotic tissue or foreign material from areas around the wound to increase the chances of wound healing and this process is known as wound debridement. Debridement of the wound area is important because the open wound bed cannot be observed and assessed effectively with necrotic tissue [42, 42]. The presence of necrotic tissue or foreign material in a wound also increases the risk of infection and sepsis and also prolongs the inflammatory phase, which inhibits wound healing. Several methods are employed for wound debridement including: surgical removal using scalpel and scissors, hydrotherapy or wound irrigation and autolytic removal by rehydration of necrotic tissue, for example using hydrogel dressings (see later), enzymatic removal using bacterial derived collagenases or preparations such as streptokinase[43,44]. The accumulation of devitalized tissue in the wound promotes bacterial colonization and impairs the body's ability to fight infection, thereby preventing complete repair of the wound. 3 The aim of debridement is to remove ischaemic and necrotic tissue, which presents potential for infection and contamination of the tissue by bacteria and foreign bodies [45].

Conclusion

Wound dressings and devices form an important segment of the medical and pharmaceutical wound care market worldwide. Wound healingis a biological process that relates to general phenomenon of growth and tissue regeneration. This review has considered many classes of wound dressings including topical pharmaceutical agents, traditional wound dressings and modern dressings such as hydrocolloids, alginates, hydrogel, polyurethane film and foam and novel biomaterials such as collagen, chitosan and hyaluronic acid used directly or as tissue engineered matrices for skin replacement. Haemodialysis involves the circulation of whole blood through a dialysis circuit and artificial kidney (dialyser) both of which have the tendency to activate coagulation pathways. Bleeding usually occurs when the skin is injured and serves to flush out bacteria and/or antigens from the wound. Any wounding damages the

tissue and affects the local environment. The host's response to wounding involves various processes of tissue healing that are triggered by tissue injury, and encompasses four continuous phases including coagulation and haemostasis, inflammation, proliferation and wound remodelling with scar tissue deposition. Correct clinical management may positively influence the wound healing course and reduce potential complications.

Corresponding Author

S.Niveda

Tutor,Department of Public Health Dentistry

Saveetha Dental College

Saveetha Institute of Medical and Technical Sciences,Saveetha University

162, Poonamalee High Road,Chennai 600077,TamilNadu, India

Email: drnivedasubash@gmail.com

Telephone Number: +919952979464

References

1. Gombotz H, Metzler H, List Wf. Methods of Reduction of Perioperative Bleeding.Br J Anaesth 1998; 81(1): 62–66

2. SchultzGs. Molecular Regulation of Wound Healing. In: Bryantra, Editor. Acute and Chronic Wounds: Nursing Management.2nd Edition. St. Louis, Mo: Mosby. 1999:413–429.

3. Hoffman R, Benz E, Shattil Sj, Furie Bruce, Cohen H (Eds). Hematology: Basic Principles and Practice, 4th Edn. Philadelphia, Pa: Elsevier, 2005.

4. Dahl backB. Blood Coagulation. Lancet; 355: 1627–32. Strodtbeck F. Physiology of Wound Healing. New INF Nurs Rev 2001; 1:43–52.

5.Russell L. UnderstandingPhysiology Of Wound Healing And How Dressings Help. Br J Nurs 1999; 9:10–21.

6.Mast Ba. The Skin. In: Choenik, Diegelmannrf, Lindbladw, Editors. Wound Healing: Biochemical And Clinical Aspects. Philadelphia, Pa: Saunders. 1999:344–355.

7.Schultz Gs. Molecular Regulation of Wound Healing. In: Bryantra, Editor. Acute and Chronic Wounds: Nursing Management.2nd Edition. St. Louis, Mo: Mosby. 1999:413–42.

8.Rothe M, Falanga V. Growth Factors, Their Biology and Promise in Dermatologic Disease and Tissue Repair. Arch Dermatol 1989; l125:1390–139.

9.Shakespeare P. Burn Wound Healing And Skin Substitutes. Burns2001; 27:517–522.

Thomas S. Assessment and Management of Wound Exudates. J Wound Care 1997; 6:327–330.

10.Armstrong S, Ruckley C. Use ofa Fibrous Dressing in Exuding Leg Ulcers. J Wound Care 1997; 7:322–324.

11.Perez Gomez F, Bover R. The New Coagulation Cascade and Its Possible Influence onthe Delicate Balance between Thrombosis and Hemorrhage. Rev. Esp. Cardiol. 2007; 60:1217–19.

12.Marlar Ra, Kleiss Aj, Griffin Jh. An Alternative Extrinsic Pathway of Human Blood Coagulation. Blood 1982; 60:1353–58.

13.Hofbauer R, Moser D, Frass M. Effect of Anticoagulation on Blood Membrane Interactions duringHaemodialysis. Kidney Int. 1999; 56:1578–83.

14.V.K.Vignesh, Dr.M.P.Santhosh Kumar., Management ofHaemorrhage inOral Surgery, JPSR, 2016; 8(7).

15. KirtanaGopalasamy AwarenessamongFinal Year andPostgraduate Dental Students on Bleeding Disorders a Questionnaire Based Study, InternationalJournal Of Science And Research (IJSR), E 2015: 78.96.

16.Purner SK, Babu M. Collagen based dressings—A review. Burns 2000. 26: 54–62.

17.Queen D, Orsted H, Sanada H, Sussman G. A dressing history. Int Wound J 2004; 1:59–77.

18.Falabella AF. Debridement and wound bed preparation. Dermatol There2006; 19:317–325.

19.van Rijswijk L. Ingredient-based wound dressing classification: A paradigm shift that is passé and in need of replacement. J Wound Care 2006; 15:11–14.

20.Fletcher J. Understanding wound dressings: Hydrocolloids. Nurs Times 2005. 101: 51.

21.Koksal C, Bozkurt AK. Combination of hydrocolloid dressing and medical compression stocking versus Unn's boot for the treatment of venous leg ulcers. Swiss Med Wkly 2003; 133:364–368.

22. Thomas S. Alginate dressings in surgery and wound management—Part 1. J Wound Care 2000; 9:56–60.

23. Kuo CK, Ma PX. Ionically cross linked alginate hydrogel as scaffolds for tissue engineering: Part 1. Structure, gelation rate and mechanical properties. Biomaterials2001; 22:511–521.

24. Barlett RH. Skin substitutes. J Trauma1981; 21:731.

25.Ramshaw JAM, Werkmeister JA, Glatteur V. Collagen based biomaterials. Biotechnology Rev. 1995; 13:336–382.

26.Doillon CJ, Silver FH. Collagen-based wound dressing: Effect of hyaluronic acid and fibronectin on wound healing. Biomaterials. 1986; **7**:3–8.

27. O'Meara S, Callum N, Majid M, Sheldon T. Systematic reviews of wound care management (3) antimicrobial agents for chronic wounds (4) diabetic foot ulceration. Health Techno Assess.2000; 4:1–237.

28.Nelson EA, O'Meara S, Craig D, Iglesias C, Golder S, Dalton J, Claxton K, Bell-Syer SE, Jude E, Dowson C, Gadsby R, O'Hare P, Powell J. A series of systematic reviews to inform a decision analysis for sampling treating infected diabetic foot ulcers. Health Techno Assess 2006; 10:1–221.

29.Harihara Y, Konishi T, Kobayashi H, Furushima K, Ito K, Noie T, Nara S, Tanimura K. Effects of applying povidone-iodine just before skin closure. Dermatology 2006; 212:53–57.

30.Russell L. Understanding physiology of wound healing and how dressings help. Br J Nurs. 1999; 9:10–21.

31. Mast BA. The skin. In: ChoenIK, Diegelmannrf, Lindbladw, editors. Wound healing: Biochemical and clinical aspects. Philadelphia, PA: Saunders. 1999:344–355.

32. Schultz GS. Molecular regulation of wound healing. In: BryantRA, editor. Acute and chronic wounds: Nursing management. 2nd edition. St. Louis, MO: Mosby.1999:413–429.

33.Musset JH, Winfield AJ. Wound management, stoma and incontinence products. In: WinfieldAJ, Richards ME, editors. Pharmacy practice. 2nd edition. UK: Churchill Livingstone.1998:176–187.

34.Gilliland EL. Bacterial colonisation of leg ulcers and its effect on success rate of skin grafting. Ann R Coll Surg 1988; 70:105–108.

Commercially Available Aesthetic Wires in Orthodontics

Priyadharshni S[1], Sumathi Felicita[2], E. Roja[3]

Graduate Student[1], Senior Lecturer[2], Department of Orthodontics,Tutor[3], Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

The development of an optimum, predictable and effective orthodontic force system is predicated on the knowledge of mechanics, biomaterials and their interaction. The evolution of manufacturing technology and the development of new orthodontic techniques have led to the search for better quality alloys, more biologically effective for the teeth and supporting tissues. Aesthetics areaimportant and integral part of orthodontic treatment. With the advent of revolutionary aesthetics brackets, the need for the aesthetic wires became very strong.Orthodontics has achieved the status of a recognised specialty of dentistry because of a long period of craftsmanship and professional expertise. The evaluation of wire manufacturing technology and the development of new orthodontic technique have led to the search for better quality alloys, more biologically effective for the teeth and supporting tissues. Esthetics has become today an important and integral part of the orthodontic treatment. With the invention of revolutionary esthetic brackets, the need for esthetic wires became very strong. With the development of new alloys it becomes necessary how the available energy of tooth movement varies with wire composition of equal diameter. Since there is now a variety of orthodontic wire alloy from which choose the clinician is now faced with more decision in regard to wire selection. The quality and performance of treatment can be improved by the application of lower forces and achieves a wider range of movements between sessions. During orthodontic treatment, orthodontic wires are used as Fixed appliances to apply forces to the teeth. They release the energy stored upon its placement by applying forces and torque to the teeth through appliances placed on them. Therefore, an orthodontist should have adequate knowledge of the biochemical behaviour and clinical applications of orthodontic wires to design the treatment plan.

Keywords*: Arch Wires, strength, aesthetics, orthodontic treatment,patients.*

Introduction

Aesthetic arch wire is highly desirable to complement aesthetic brackets in clinical orthodontics. Despite advancements in the aesthetics of clear orthodontic brackets, the appearance of orthodontic arch wires has changed little.While increasing attention has been given to the use of aesthetically pleasing orthodontic archwires to complement clear brackets, these wires have not been routinely incorporated in treatment due to a lack of evidence-based research and practitioner familiarity, as well as concerns regarding fragmentation of the aesthetic coating and increased frictional resistance.Currently, aesthetic archwires can be separated into coated metal and transparent non-metallic. Each undergoes a distinct manufacturing process, resulting in unique challenges when used in treatment.(1)

The orthodontic profession is constantly seeking to improve and optimise the aesthetics of orthodontic wires since the introduction of aesthetic brackets. Nickel-titanium (NiTi) wires since their introduction to orthodontics have been extensively researched *in vitro* and are used as an initial levelling and aligning arch wire because of its properties of spring back and super elasticity. Aesthetic wires are usually either coated NiTi wires or composite wires of reinforced polymers. Shape memory polymers have wide application in space technology and are being used currently in medicine and industrial applications These wires have enormous potential for clinical application in orthodontics, and polyphenylene, a self-reinforced polymer composite, is close to being introduced to orthodontic practice.However, these wires are still at the experimental stage. A fibre reinforced polymer is in clinical use that is manufactured using a pultrusion process with a photo-cured resin however, these wires may be more likely to crack during bending and have been shown to deliver less consistent forces compared with alloy wires.The coated wires, which are currently available, either have an epoxy resin, polytetrafluoroethylyene, or a low reflectivity rhodium coating applied to the surface. Atomized Teflon particles are used to coat the wire using clean compressed air as a transport medium, which is then further heat treated in a chamber furnace.The rhodium coating is applied by using a plasma-immersion ion implantation technique. The coated wires are found to be routinely damaged from mastication and activation of enzymes.(2)

Recently, orthodontic treatment has become more common in adult patients, and the demand for improvement in the aesthetics quality of braces has been increasing. Many individuals regard metallic braces as unsightly when placed in the mouth. Although aesthetic brackets have brought a dramatic improvement in the appearance of appliances, metal arch-wires are still visible. This limitation in the improvement in appearance has led many manufacturers

and researchers to attempt to produce durable aesthetic arch wires. These wires have to be visually unobtrusive and at the same time perform the essential function of aligning teeth.

With increasing number of adult patients seeking orthodontic treatment, the demand for esthetic orthodontic appliances has increased dramatically, creating a need for the so-called invisible orthodontic appliances like invisalign(3) and lingual braces (4). However, esthetics of fixed labial appliances has also evolved by inclusion of ceramic brackets aesthetic ligatures and tooth colored arch wires. Attractiveness evaluation reveals that sapphire brackets with esthetic arch wires are preferred just next to clear aligner trays to complement the esthetic brackets with invisible arch wires, esthetic arch wires have rapidly evolved in the last decade.(5).

Esthetic arch wire materials are basically a composite of two materials which can be broadly classified into two major groups. The ceramic – polymer composite prototype of arch wires are solid polymeric wires typically made from glass fiber spindles embedded in a polymeric matrix which are manufactured through a process called photo-pultrusion. Goldberg (1992) first reported the fabrication of fiber reinforced composites (FRC) using Bis-GMA resin and S2 glass fibers, but these wires were brittle and susceptible for intraoral breakage. Burstone (2011) introduced a self-reinforced polymer (SRP) polyphenylene thermoplastic arch wires which showed flexibility comparable to NiTi and beta titanium arch wires at thin cross sections without experiencing stress relaxation.

Coated esthetic arch wires have a core of a metallic wire coated with either tooth-colored polymer or inorganic materials to conceal the visibility of the underlying alloy and impart an enamel like hue to the arch wire. Coating improves aesthetics, but creates a modified surface, which can affect friction, corrosive(6, 7)

Different Types of Aesthetic Wires

Optiflex Wires

Optiflex is a non metallic orthodontic arch wire and it has got unique mechanical properties with a highly aesthetic appearance made of clear optical fibre. They are composed of a Silicon dioxide core which provides the force or resilience to the wire. The silicon resin forms the middle layer which adds strength to the wire and also protects the core from moisture. The outer nylon layer makes the wire stain resistant and also prevents it from damage. These wires provide light continuous forces and are used during the initial aligning phase of orthodontic treatment. To prevent permanent deformation, sharp bends should be

avoided during ligation to brackets. They are available in round as well as rectangular cross section.(8)

Fibre Reinforced Composite Wires

Fibre reinforced composites (FRC) can change the concept, mechanics and application of fixed appliances through the use of partially polymerised fibre matrix complex that fully polymerises in the clinical setting. Its greatest clinical potential lies in the active applications where they are used as adjuncts for active tooth movement. They have the potential to replace metals in orthodontics as they have good bonding characteristics not only to the tooth but also to the appliance itself. An FRC can be bonded to another and attachments added directly. Three configurations of FRC that is available are Rope type- 2 mm wide, round strips, Can be wrapped around corners of an arch and thus is useful in cuspid-to-cuspid retainers and Unidirectional parallel configurations have best mechanical properties for bending.FRC can be bonded by direct and indirect technique with good bond strength, as polymeric matrix is the same as the bonding adhesive. The advantage of indirect technique being shorter curing time needed intra-orally. If attachments such as brackets, tubes or hooks, they can be directly bonded on the FRC.(9)

Marsenol

MARSENOL is a tooth coloured elastomeric poly tetrafluroethyl emulsion (ETE) coated nickel titanium wire. The working characteristics of these wire similar to an uncoated super elastic Nickel titanium wire.

Coated Arch Wires

Metallic arch wires coated with tooth-colored resin materials, such as synthetic fluorine-containing resin or epoxy resin composed mainly of polytetrafluoroethylyene, are currently the existing solution to this esthetic problem. The coating is applied in a depository process that plates the base wire, and its thickness is approximately 0.002 inches, as reported by manufacturers. Thus, a strong adhesion is achieved between the coating and the wire.

There are different opinions in the literature concerning esthetic coated arch wires. An evaluation of sliding properties and adherence of the coating to the arch wires revealed that the plastic coating decreased friction between arch wires and brackets.Some authors have experienced difficulties with these coated arch wires, claiming that the color tends to change

with time and that the coating splits during use in the mouth, exposing the underlying metal.However, despite these problems, these wires continue to be marketed and used in clinical practice, mainly when an esthetic alternative for a rectangular cross-sectional arch wire is necessary.(10)

Teflon Coated

Coating on archwire material has been introduced to enhance aesthetics and decrease friction. These wires are designed to be aesthetically more acceptable by the patient. They are given a plastic tooth coloured coating so that it can blend with the tooth colour and also of ceramic brackets. Normally the coating is 0.002" thick. The coating frequently used is TEFLON. Teflon coating is applied in two coats by conventional air spray or electrostatic techniques. These are available in natural tooth shades or in blue, green and purple colours as Lee White Wire (Lee pharmaceuticals).(11)

Epoxy Coated

Epoxy coated archwire is tooth coloured wire and has superior wear resistance and colour stability of 6-8 weeks.It is available in nickel titanium and stainless steel in preformed arches of different sizes such as round 0.016" to 0.022" NiTi, rectangular 0.018" x 0.024" to 0.021" x 0.027" NiTi and round 0.014" to 0.018, rectangular – 0.18" x 0.024" to 0.021" x 0.027" stainless steel. Epoxy coated archwires are available under the trade name of Filafex (American Orthodontics), have high tensile stainless steel core and durable tooth coloured plastic coating. It is available in preformed round 0.018" arches.They are aesthetically coated high performance NiTi super-elastic archwires and blends exceptionally well with ceramic or plastic brackets and doesn't stain or discolour plus they resist cracking or chipping.(12)

NitaniumTooth Toned Archwire

It is a super elastic Ni-Ti wire with special plastic and friction reducing tooth colored coatings which blends with natural dentition, ceramic, plastic and composite brackets and maintains its original colour. The disadvantage of the coated white colored wires is that they have routinely succumbed to forces of mastication and enzyme activity of oral cavity. On the other hand, the uncoated transparent wires have poor mechanical properties that they function merely as placebo. Esthetics is important to the orthodontist but function is paramount and anything less is unacceptable. It is marketed by Ortho Organizers and is available in round

0.014”, 0.016”, 0.018” and rectangular 0.016” x 0.022” sizes. These wires deliver gentle force.

Transparent Non-Metallic Arches

Within the past 20 years, significant advancements have been made to create nonmetallic arches whose properties resemble metal alloys. Flexible nonmetallic arches are typically made from glass spindles embedded in a polymeric matrix. Some examples of nonmetallic arches include fiber-reinforced polymer or newer self-reinforced polymer (SRP).These arches allow for a few millimetres of deformation and may be suitable for levelling and aligning in patients with Class I malocclusions with mild to moderate crowding. More importantly, they display the translucency and transparency ideal for ceramic brackets. Nonmetallic arches are not readily available. The most popular commercially availablenonmetallic arch is made by BioMers Products LLC.

The manufacturing process for nonmetallic arches will vary depending on the type of polymer. Fiber-reinforced composite (FRC) orthodontic arches are made through a production process referred to as hot-drawing. Drawing is a working process that uses tensile forces to stretch a workpiece. Hot-drawing FRC arches entails: melting of glass pellets, spinning the glass into elastic and flexible fiber strands, coating the fibers with a silane-coupling agent, and coagulation of the glass fibers and methacrylate within an acetone solution.

Properties of Aesthetic Wires

Optiflex Wires

It is the most aesthetic arch wire, completely stain resistant and will not stain or loose its clear look even after several weeks in mouth. Optiflex is very flexible. It has an extremely wide range of actions. When indicated it can be tied with electrometric ligatures to severely mal-aligned teeth without fear of fracturing the arch wire. Due to superior properties optiflex can be used with any bracket system. Optiflex arch wires should be tied into brackets with elastomeric ligatures. Metal ligatures should never be used since they will fracture the glass core. Optiflex is a totally esthetic non-metallic labial orthodontic arch wire designed by Dr. Talass in 1992 and manufactured by Ormco. It is made of clear optical fibre with distinctive mechanical properties with highly esthetic appearance and entirely stain resistant. It consists of 3 layers. A)silicon dioxide core which provides the force for

tooth movement. B) A silicon resin middle layer which protects the core form moisture and adds strength. C) A strain resistant nylon outer layer which prevents wire damage and further increases strength orthodontically beneficial properties of optiflex arch wires includes efficient tooth movement with light continuous force, increased flexibility producing a wide range of action thus invariably permitting its use with various bracket systems. However sharp bends in the wire should not be attempted. Metal ligatures should not be used as they can fracture the glass core. When cutting the distal ends of the wire use the mini distal end cutter, which is designed to cut all the 3 layers of Optiflex. It is used inadult patients who are aesthetically concerned. Optiflex wire canbe used for initial alignment. It produces less force for the same amount of deflection when compared with coaxial wires. The optiflexarch wires are expensive and needs to be changed every 4-6 weeks.(13)

Coated Metal Wires

Coated metal archwires are nickel-titanium or stainless steel wires treated with a polytetra-fluoroethylene (PTFE), epoxy- resin, acrylene-polymer, or less commonly palladiumcovering to impart an enamel hue. Manufacturers vary with regard to the coating material, thickness of the coating, and steps within the application process to maximise aesthetics, flake resistance, and mechanical efficiency. PTFE is a synthetic polymer consisting wholly of carbon and fluorine. Due to the strength of the carbon-fluorine bonds, PTFE is nonreactive, heat resistant, and hydrophobic. Most importantly, it has the third lowest coefficient of friction of any known solid, making it ideal for use as a nonstick coating for cookware, gears, plain bearings, or where sliding action of parts is needed. PTFE coating is applied to an orthodontic wire by thermal spraying, a process in which finely heated materials are sprayed in a molten condition onto a surface to form a coating. Thermal spraying of PTFE onto an orthodontic archwire entails surface treatment of the wire by sandblasting to support coating adhesion and “masking” or covering with tape areas that are not be treated, air-spraying atomised PTFE particles with clean compressed air to coat the wire, baking in a chamber furnace to cure the coating onto the wire, and removal of the masking tape. The PTFE layer adds a minimal thickness (.0008 to .001 inch) to the archwire.Though PTFE has an extremely low coefficient of friction, it is used primarily for aesthetic purposes. (14)

Fibre Reinforced Composite Arch Wire

Fiber reinforced composite arch wire Fiber reinforced composite arch wires are fabricated using a procedure called pultrusion. Fiber bundles are pulled through an extruder, in which they are wetted with a monomer resin. Then the monomer is cured with heat and pressure resulting in polymerization. Circular or rectangular wires are formed during curing. This may be shaped into a different morphology by further curing, a process known as beta staging. For this, the monomer should initially only be partially cured. The composite arch wires have higher kinetic coefficients of friction than stainless steel but lower coefficients than either Nickel-titanium or Beta-Titanium. At high forces and angulations abrasive wear of the composite surface at the arch wire-bracket interface was observed. It can lead to release of glass fibers within the oral cavity, which is unacceptable Advantages of fiber reinforced composite wires over conventional metal wires are excellent combination of high elastic recovery, high tensile strength, low weight, excellent formability, excellent aesthetics because of their translucency, ability to form wires of different stiffness values for the same cross-section which would facilitate the practice of constant cross-section orthodontics. Attachments can be directly bonded to these wires, which eliminate the need for soldering and welding. These wires can also be directly bonded to teeth obviating the need for brackets, in certain situations, e.g. where anchorage from a large number of teeth is required. It is a safer choice for patients with nickel allergy.

Composite arch wires had higher kinetic coefficients of friction than stainless steel but lower than nickel-Titanium or beta titanium. The composite arch wire retained sufficient resilience to function during initial stage of orthodontic treatment and also during intermediate stages of orthodontic treatment.Burstone and Kuhlberg introduced a new fiber reinforced composite called "Splint-It" which has S2 glass fibers in a bis GMA matrix. Various configurations such as rope, woven strip and unidirectional strip are available. These materials are only partly polymerized during manufacture, which makes them flexible, adaptable and easily contourable over the teeth. Later they are completely polymerized and can be bonded directly to teeth. It can also be used for various purposes such as post treatment retention, as full arches or sectional arches, and to reinforce anchorage.(15)

Fibre reinforced composites (FRC) can change the concept, mechanics and application of fixed appliances through the use of 'pre-preg' or partially polymerized Fibermatrix complex that fully polymerizes in the clinical setting. Its greatest clinical potential lies in the active applications where they are used as adjuncts for active tooth movement. They have the potential to replace metals in orthodontics as they have good bonding characteristics not only to the tooth but also to the appliance itself. An FRC can be bonded to another and attachments added directly. Fibre reinforced composite are available as three configurations such as A)Rope type- 2 mm wide, round strip. B)Can be wrapped around corners of an arch and thus is useful in cuspid-to-cuspid retainers.C)Unidirectional parallel configurations have best mechanical properties for bending.

FRC can be bonded by direct and indirect technique with good bond strength, as polymeric matrixis the same as the bonding adhesive. The advantage of indirect technique being shorter curing time needed intra-orally. If attachments such as brackets tubes or hooks, they can be directly bonded on the FRC.

Advantages of FRC

i. Esthetic as the connecting bar is clear or translucent.

ii. Biocompatible and less hypersensitivity reported as compared to stainless steel and other metals.

iii. High modulus of elasticity in flexure (70% greater than highly filled dental composite), six times greater yield strength and 2 times greater resilience.

iv. Option to join pieces together with an adhesive to make a string structural unit.

v. Attachments can be added for inter-maxillary tooth movement without bands or brackets, making it simple to position hooks with ideal direction and point of force application respective to maxillary and mandibular centre of resistance.

vi. Vertical elastics can be applied directly to FRC bars, either on full arches or on segments for closure of an open-bite.

vii. Intra-arch movements such as space closure, with bonded tubes on FRC bars, which can be positioned to increase the inter-bracket distance.

viii. Correction of poorly erupted second molars after completion of orthodontic therapy can be done by uprighting the tooth using full-arch FRC as an anchorage unit. Active force applied by straight wire segment, T-loop or wire with differential bend.

ix. Ease of modification as more layers can be added if greater rigidity required. Attachments can be repositioned, repaired or replaced any time.

x. Passive applications such as bonded tooth-to-tooth retainer can be made with ideal esthetics as compared to metal wire bonded retainers.

FRC bars are strong and rigid in tension but less in bending mode and are weakest in shear and torsion. Unlike metals, they are not homogenous materials so shear loads need to be minimized. Sound bonding technique is required.FRC is available as 'Splint-it', a commercially available long-fiber-reinforced composite, uses strong S-glass fibers. The matrix is light cured thermoset bisGMA with the fibers correctly oriented gives excellent coupling. FRCs are produced in two steps. In the first step, "Amount, distribution and wetting of fibers by resin is closely controlled" and in the second step "Composite is formed into desired final shape".

Two important processes associated with fabrication of FRCs, i.e., Pultrusion and Beta staging.

Pultrusion: It is the process of manufacturing components having continuous lengths and a constant cross sectional shape such as in arch wire. In this bundle of continuous fibers are impregnated with polymeric resinand are then pulled through a Sizing Die that performs composite and establishes resin/fibre ratio. The bundles are then passed through Curing Die which imparts precise shape as it cures the resin.

Beta Staging: It is an intervening process in which partially cured resin and its bundles of continuous fibers are deformed into another form (e.g. preformed archwire) after which the curing is completed. Preformed arch wires and rectangular cross-section is possible by this process.(16)

FRC is Tooth coloured and they vary in stiffness from that of most flaccid multi-stranded wire to nearly that of β-Ti arch wire. These characteristics can be varied during manufacture without any change in wire slot engagement by pultrusion. Mechanical tests show that such arch wires are elastic until failure occurs and when failure does occur the wire loses its

stiffness but remains intact.When compared with NiTi, resilience and spring back are comparable. Specifics of other characteristics such as formability, weldability and frictional coefficients are unknown at this time. Low coefficients of friction and enhanced biocompatibility should be possible by modifying the surface chemistry of polymer. Like the advanced metal wires, their shape is very difficult to change once the manufacturing process is completed which leads to a number of practical problems for clinical applications.(17,18)

Clinical Implications

It is used in adult patients who wish that their braces not be really visible for reasons related to personal concerns or professional consideration. It can be used as initial arch wire in cases with moderate amounts of crowding in one or both arches. It should be used in cases to be treated without bicuspid extraction. Optiflex is not an ideal archwire for major bicuspid retraction. Optiflex can be used in pre-surgical stage in cases which require orthognathic intervention as a part of the treatment.(19)

Drawbacks

Coated metal arch wires with circumferential coating deliver statistically lower loading and unloading force levels than coated wires of the same nominal sizes. The lower force levels of coated wires may be attributed to the thick aesthetic coating having a negative effect on the load- deflection properties and a manufacturer’s use of a smaller- diameter wire to compensate for the thick coating, particularly for epoxy-coated arch wires. Additionally, the fragmentation of the coating adds increased frictional resistance and diminishes the aesthetic benefit. Meanwhile, nonmetallic arches are brittle and allow for only moderate deformation. Excess deformation or forceful grip with pliers can lead to permanent deformation and irreversible cracks, referred to as “craze lines.” These clear arches are restricted with regard to torque, detailing, and changes in arch width, and they are currently not suitable for patients requiring consolidation or anteroposterior correction(20).

Conclusion

Appearance is one of the patients' main concerns during orthodontic treatment. There is a growing demand for aesthetic orthodontic appliances and by the effect of this demand; most

of the companiesproduced many new aesthetic appliances.The new aesthetic appliances provided better appearance for the patients and acceptable results for clinicians but these appliances must be studied by the researchers more and also must be developed.

Corresponding Author

E. Roja,

Tutor,Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai – 600 077, Tamil Nadu, India.

Email Id: itsrojaa@gmail.com

Contact Number: +91 99941 74480.

References

1. DevinderPreetSingh Esthetic Archwires in Orthodontics- A Review, Oral Hyg Health.2016;4:1;133-141.
2. Abhishekagarwal, preetibhattacharya, siddharth.Newer orthodontic wires. A revolution in orthodontics-a Review- journal of dental sciences and oral rehabilitation.2011;1:30-33.
3. Masahiro Iijimaa; Takeshi Mugurumab; William A. Brantleyc; Han-CheolChoed; Susumu Nakagakie; Satish B. Alapatif; ItaruMizoguchig Effect of coating on properties of esthetic orthodontic nickel-titanium wires.Angle Orthodontist,2012;82(2):20-29
4. Aksakalli S, Malkoc S. Esthetic orthodontic archwires: Literature review. J Orthod Res, 2013;1:2-4
5. Dr. AparnaKhamatka, Ideal Properties of Orthodontic Wires and Their Clinical Implications -A Review,IOSR Journal of Dental and Medical Sciences (IOSR-JDMS),2015;1;14.
6. Cátia Cardoso AbdoQuintao, Ione Helena Vieira PortellaBrunharoOrthodontic wires: knowledge ensures clinical optimization Dental Press J. Orthod. 144 Maringá,2009:14(6):144-157.
7. Akın Mechanical Properties of Different Aesthetic Archwires.Turkish J Orthod.2014:27(2):177-189

8. Russell JS. Aesthetic orthodontic brackets. J Orthod. 2005; 32:146–163.
9. Elayyan F, Silikas N, Bearn D. Mechanical properties of coated superelasticarchwires in conventional and self- ligating orthodontic brackets. Am J Orthod Dentofacial Orthop. 2010;137:213–217.
10. Russell JS. Aesthetic orthodontic brackets. J Orthod. 2005; 32:146–163.
11. Ramadan AA. Removing hepatitis C virus from polytetraflu- oroethylene-coated orthodontic archwires and other dental instruments. East Mediterr Health J. 2003;9:274–278.
12. Clocheret K, Willems G, Carels C, Celis JP. Dynamic frictional behaviour of orthodontic archwires and brackets. Eur J Orthod. 2004;26:163–170.
13. Husmann P, Bourauel C, Wessinger M, Jager A. The frictional behavior of coated guiding archwires. J OrofacOrthop. 2002;63:199–211.
14. Silva DL, Mattos CT, Arau´jo MV, Ruellas ACO. Color stability and fluorescence of different orthodontic esthetic archwires. Angle Orthod. 2013;83:127–132.
15. Elayyan F, Silikas N, Bearn D. Ex vivo surface and mechanical properties of coated orthodontic archwires. Eur J Orthod. 2008;30:661–667.
16. Dhanraj Esthetic Management of Unilateral Cross Bite with Dento-vestibular Enhancement Prosthetic Appliance, J Indian Prosthodont Soc. 2011;11(2): 117–120.
17. Gayathri, M, Ravindra Kumar Jain, and A V Arun, Systematic Review on Initial Alignment Efficiency of Coaxial Wire. Research Journal of Pharmaceutical, Biological and Chemical Sciences, ISSN: 0975-8585. 2016 ;7(4):2961
18. Sharmila.R,Journal of pharmaceutical science and research /J. Pharm. Sci. & Res. V 2016;8(8): 895-897.
19. S.P. Saravana Dinesh, A.V. Arun, K.K. Shantha Sundari, Christine Samantha, K. Ambika, .AN INDIGENOUSLY DESIGNED APPARATUS FOR MEASURING ORTHODONTIC FORCE.Journal of Clinical and Diagnostic Research,2013 Nov:11 2623 –2626.

Comparison of Adverse Effects of Labial and Lingual Orthodontic Treatment-AReview

T. Nandhini[1], SaravanaPandian[2], ManishRanjan[3],

Graduate Student[1],Senior Lecturer[2], Department of Orthodontics[2],Reader[3], Department of Conservative Dentistry[3],

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

The aim of this review is to compare the adverse effects of lingual and labial orthodontics.To compare adverse effects between labial and lingual orthodontic treatments through a review of literature. Lingual orthodontic came into existence to eliminate the visibility of the appliance by sparing the labial surface and making use of lingual surface to fix the orthodontic attachments for aesthetic reasons on the demand of adults. Orthodontic treatment is often part of a comprehensive dental health care plan. With good care, including orthodontic treatment when necessary, teeth can last a lifetime. A variety of orthodontic "appliances" are available today. The purpose of this review is an attempt to present an overview of lingual orthodontic versus labial orthodontic.

Key Words*: labial orthodontic, lingual orthodontic, intrusion of anterior teeth, direct bonding, indirect bonding.*

Introduction

The goal of orthodontic treatment is a beautiful smile and a good bite meaning straight teeth that mesh well with the teeth in the opposite jaw and look great. Many people find a beautiful smile adds to self-esteem, self-confidence and leads toward career advancement. We live in a competitive world and a great smile can give you the edge.

Orthodontic treatment is often part of a comprehensive dental health care plan. With good care, including orthodontic treatment when necessary, teeth can last a lifetime. A variety of orthodontic "appliances" are available today, Options include traditional metal braces, tooth-coloured braces, braces that go behind the teeth, clear aligners and other devices, as appropriate.

Comfortable and efficient, today's orthodontic appliances also contribute to a stable, long-lasting result. While the appliances used in orthodontic treatment have come a long way since orthodontics became dentistry's first specialty in 1900, what endures is orthodontists' desire to help you achieve your healthiest bite possible.

Recent years have witness a noticeable increase in the demand for lingual orthodontic appliances among orthodontic patients looking for aesthetic improvement (1). Several seminal studies indicated that lingual appliances can afford treatment outcomes comparable to those achieved with labial appliances (2, 3). Lingual orthodontic appliances take pleasure in aesthetic advantages over conventional labial orthodontic appliances (4).Moreover, it has been claimed that lingual appliances put up with a lower risk of caries (5).Nevertheless, strong concerning the tongue soreness and difficulty in speech have arise regarding lingual orthodontic appliances (6-9).Natural products derived from plant source are found to be highly efficient in eradicating the dental caries/plaque found in fixed orthodontic appliances patients undergoing orthodontic treatment (10).

Specifically, it was recently reported by Khattab et al.(11) that more significant speech deteriorations were associated with lingual orthodontic treatment than labial appliances. However, to date, the reliability of this evidence has not been critically assessed. Therefore, a review that critically evaluates the reliability of evidence is necessary for relevant dental

practitioners. So this review is to compare adverse effects between lingual and labial orthodontic treatment.

Labial Orthodontic Treatment and Lingual Orthodontic Treatment

Fixed appliances are individuals which are fitted to the teeth and cannot be disconnected by the patient at will. A variety of complex tooth movements be finished possible with fixed appliance (12). Brackets and arch wires were located on the labial surface of the teeth for labial orthodontic treatment. Lingual braces are one of the many types of the fixed orthodontic treatment methods available. It involves attaching the orthodontic brackets on the inner sides of the teeth which is not easily visible from outside. The main advantage of lingual braces is its near invisibility compared to the standard braces which are trapped on the buccal sides of the tooth.Lingual braces were invented by Craven Kurz.

Brackets were initially bonded with a system known as Torque Angulation Referencing Guide (TARG) which allowed a clinician to place brackets on lingual surfaces of teeth by using the natural anatomy. Then another method called Custom Lingual Appliance Set-Up Service which allowed a clinician to set up brackets on a model first and then indirectly bond them on patient's teeth later on with a tray.

Lingual orthodontics began in 1970's when Fujita in Japan and Kurtz in the USA utilized lingual sections out of the blue. It made an amazing presentation as lingual sections were undetectable, and the quantity of started cases expanded exponentially. A couple of years after the fact, the quantity of lingual orthodontic cases diminished incredibly. The reason was clear; most specialists couldn't accomplish palatable outcomes with lingual orthodontics. Following this, underlying advancement and extension of lingual orthodontics in the 1990s, intrigue, especially in the United States, diminished, likely because of the poor consequences of finished cases(13).As time passed and critical thinking related with lingual orthodontics was enhanced, lingual orthodontics extended far and wide, particularly in European and Asiatic nations .

ApplianceDesign

Lingual section framework has developed from original Ormco lingual sections to PC supported plan/PC helped fabricating (CAD/CAM)- based totally modified lingual apparatus. Original Ormco lingual sections of 1970's were developed to seventh era in 1990's, the progressions were made in the profile of the section, expansion of snares, consolidation of rhomboidal shape chomp planes, and expanded mesiodistal width of the premolar sections for better rotational control.Mushroomarchwires have been being used since Fujita began lingual orthodontics which regularly requires vertical advance curves and insets amongst canine and first premolar and muddled wire twists. Scuzzo and Takemoto in 1995 presented lingual straight wire method and STb sections and showed that lingual straight wires can be utilized if the sections are repositioned gingivally since the distinction in thickness of the canines and premolars diminishes with the holding tallness. Weichmann in 2002 altered the idea of lingual orthodontics by presenting CAD/CAM-based modified lingual apparatus and automated wire twisting. In 2009, 3M Unitek assumed control over this modified apparatus and presented it as Incognito™ lingual appliance.Fillion in 2010, additionally built up a redid straight-wire procedure, utilizing the Orapix advanced framework to create lingual machines from a virtual setup. There are numerous modified lingual frameworks accessible today: WIN (DW Lingual Systems GmbH), HARMONY (American Orthodontics), and Indian tweaked lingual frameworks: Lingual Matrix and iLingual 3D. Scuzzo in 2011 presented first self-ligating lingual sections with square space (14). As indicated by their analyses, the square opening (0.018 inch × 0.018 inch) is better than rectangular space in rotational control with both round and square archwires.Kairalla in 2014 built up four lingual curve sizes: S, M, L, and XL and state of lingual curve frames was portrayed like a parabola, somewhat smoothed on its foremost area (15). Stop in 2015 additionally gave another arrangement of maxillary and mandibular lingual curve frames: Narrow, decreasing and ovoid as indicated by intercanine and intermolar widths and their best-fit bends on lingual surface of dentition(16).

Indirect Bonding and Laboratory Setup

The morphological varieties of lingual dental surfaces constrain the immediate holding of sections on lingual surface precisely and decisively. Consequently, backhanded holding procedure is essential for achievement in lingual orthodontics. There are distinctive research facility methods which have been created for aberrant situating and holding of lingual sections. Research center setup for circuitous holding of lingual apparatus can be separated into two classifications, one is manual setup which utilizes patient's dental models and

incorporates different strategies (BEST, CLASS, and HIRO, and so on.), the second is totally redone advanced lingual setup (Orapix, WIN, HARMONY, Incognito™ and Lingual Matrix, and iLingual III D,) individualized for every patient, made by utilizing patients examined model or three-dimensional (3D) picture and sections are composed and made by CAD/CAM innovation.

Disadvantages

Initial appliances formed in 1980s irritated patient's tongues and had higher breakage rate. However, different companies made the bracket profile smaller and smoother which allowed less frustration to soft tissues around the bracket. However, the same problems still persisted over the years and treatment move towards is to inform the patient that irritation and speech impairment will recover in 2–3 weeks after the bracket placement. Furthermore, initial brackets had bracket hooks which were long and caused gingival impingement. This was familiar in the later generations of brackets which had smaller hooks and did not interrupt the gingiva. Lingual braces are also related with extended chair time or an orthodontist which may or may not be beneficial for the source. Labial movement of teeth during orthodontic treatment may result in gingival recession in the upper or lower anteriors. In the absence of keratinized gingiva, orthodontic treatment only further deteriorates gingival health. In 28.6% of the cases undergoing tooth movement, gingival clefting occur (17).

Advantages

An advantage of the lingual brackets over the buccal brackets is the less decalcification marks on the buccal side of the teeth which is more visible to the naked eye. Patient's with poor oral hygiene can have increased white spot lesions which present themselves buccal and can stay there post- orthodontically if proper oral hygiene is not maintained.

Treatment Effects

Intrusion of Anterior Teeth

Lingual brackets are positioned more closer to the centre of resistance of a tooth then brackets which are placed on a buccal surface of a tooth. Thus when a patient bites down, the biting forces are focussed through the centre of resistance of those anterior teeth. As the mandibular teeth are biting on the upper brackets, it results in an anterior bite plane effect. This ultimately leads to the light, continuous intruding force that is being felt on the front

incisors. This bite plane effect may also induce slight opening in the posterior molar teeth and these teeth may extrude, leading to correction of deep bite. However, forces that are felt on]the anterior teeth seem to be minimal, in milligrams. An optimum force is essential to intrude teeth is 30-40g.

Changes in the Oral Environment after Placement of Lingual and Labial Orthodontic Appliance

The lingual orthodontic group could be explained by the fact that plaque deposit on the lingual gingival margin of the teeth are more difficult to remove with standard oral hygiene procedures with respect to the labial side; if maintained for a long time, they represent a bacterial injury for the gingiva and can cause gingival inflammation. According to many retrospective (18,19,20) and prospective studies (21,22) of the literature, wider lingual brackets cause a reduced inter bracket distance and make oral hygiene procedures very difficult with following risk for plaque accumulation and gingivitis.

In Speech

Lingual orthodontic patients are usually having some tongue discomfort and speech difficulty associated with the insertion of the appliance. However, the intensity and duration of the problems are not so far entirely clear and orthodontists are still doubtful of the patient's ability to adapt to the lingual bracket (23). Lingual appliances do cause more speech changes than the labial appliance. The insertion of fixed labial orthodontic appliances has a variable effect on speech sound production. In some patients, a transient negative change is seen. In others, a more persistent change is seen over a period of 2 months. In approximately 40%, no change is noted.Both sibilant and stopped sounds are affected by fixed labial appliance insertion. However, the sibi- lants, most notably /s/, are affected most often.The ability to accommodate sound production to the presence of fixed labial appliances, immediately and over time, depends to some extent on the severity of the malocclusion (24).

Biomechanically

Theoretically retraction forces used in the lingual technique is three-fold lower than that with labial technique. Controlling the position of anterior teeth is difficult in LiO, due to the unpredictability in the morphology of the palatal surface of the anterior teeth, the reduced

distance between the point of force application to the center of resistance of the tooth, the small inter bracket distance, influencing arch wire rigidity, and friction (25).

The sliding mechanics with lingual orthodontics also has the advantage of being effective in preventing transverse bowing effect leading to undesired buccal tipping of premolars and distolingual rotation of molar without using any auxiliary such as transpalatal arch. The lingual orthodontics shows expansive nature especially during alignment. This effect was first attributed to increase in tongue pressure as a result of placement of appliance on the lingual aspect but later it was realised to be a mechanical side effect/bowing effect, which can be controlled by use of stiff wires while retraction.

Agony

There has been a change in outlook inside orthodontics to the utilization of lingual settled machines instead of ordinary labial settled apparatuses in the treatment of malocclusions. Lingual machines can deliver an equivalent treatment result over comparative eras and are accordingly an elective treatment methodology for patients to consider. Albeit lingual machine treatment is frequently more costly than ordinary labial apparatus treatment, it has evident tasteful focal points amid the dynamic treatment stages for instance, even shading coordinated labial sections seem to offer no option in style contrasted and the lingual system .

Poor feel, be that as it may, are by all account not the only outcome of orthodontic treatment. Torment is one of the best aversions and a noteworthy dread of orthodontic treatment .

Besides, torment is a typical motivation behind why orthodontic treatment is hindered and even ended, influencing consistence and treatment times.Accordingly, it is basic that experience of agony is considered in the orthodontic treatment basic leadership process.

The experience of torment from traditional settled labial apparatuses has for some time been accounted for following the arrangement of orthodontic separators to archwire position and initiation, especially in the beginning periods of treatment. There is clashing confirmation in regards to torment experienced by those wearing removable orthodontic machines contrasted and those treated with regular labial apparatuses An ongoing report has recommended that Invisalign® causes less agony amid treatment than traditional labial machines (26). There is a

lack of data looking at the experience of agony among those treated with regular labial contrasted and those treated with lingual apparatuses.No huge contrasts in patients' worldwide evaluations of torment experienced amid treatment were seen between those treated with labial or lingual apparatuses.

Eating troubles

Ata-Ali in 2016, in their efficient audit, inferred that eating difficulties were not observed to be factually more typical with lingual than with buccal appliances(27).

White Spot Injuries

It is beneficial that lingual xed apparatuses are related with decreased rate of WSLs when contrasted with labial xed machine as detailed by different examinations including RCT and efficient surveys. A RCT led by van derVeen et al. in 2010, revealed that the quantity of new WSLs creating or advancing on sectioned buccal surfaces was 4.8 times higher than the quantity of new WSL creating or advancing on xed lingual surfaces (28).Wiechmann et al. in 2015, in their examination on totally tweaked xed lingual machine likewise discovered unmistakably diminished WSL (29).

Consideration Regarding Appliance Placement

Lingual orthodontics presents difficulty for appliance placement firstly, due to difficulty in direct accessibility to lingual surfaces and secondly, due to anatomic variations of the lingual surfaces and thereby, increasing the chances of inaccuracy of bracket positioning. Precise bracket positing is important for successful treatment. A change in bracket position on lingual side will have a greater effect on the final tooth position than the same change when applied on labial aspect because lingual brackets are bonded distally from the labial surface that has to be aligned. Precise bracket positioning is achieved either by direct bonding with lingual jigs (30), which also enables indirect bonding or by indirect bonding using TARG system(Torque Angulation Reference Guide), (31) the slot machine, the CLASS system (Customised Lingual Appliance Setup Service), the CRCS system (Convertible Resin Core System, the bending arch technique, the TOP system (Transfer Optimised Positioned). Direct bonding is less accurate, therefore indirect bonding is preffered.

Considerations Regarding Patient’s Acceptibility to the Appliance

Both labial and lingual patients feel discomfort at the beginning of the treatment and adaptation period for the patient to be comfortable with the appliance was the similar in the patients with either of the technique. Patients felt discomfort localised in lip and cheek areas with labial appliance, where as those with lingual appliance complained of tongue soreness as a common occurrence (32). No difference was observed in the eating and oral hygiene maintenance, but food sticking was also present .Speech was also found to be severely affected in patient with lingual appliance. Patient should be very cooperative and able to understand the discomfort caused by the appliance and need to be tolerant .Lingual orthodontics is associated with extended chair time in comparison to labial orthodontics. increasing in time, effort and support of a laboratory all lead to increase in the cost, which may discourage a patient from seeking lingual orthodontic treatment.

In labial orthodontics, white spots on the labial surfaces around the orthodontic attachments may be discovered after removal of appliance leading to anaesthetic appearance. In lingual orthodontics, such chances of white spot formation on labia surfaces can be eliminated by maintenance of proper oral hygiene as labial surface is spared of orthodontic attachments, this advantage is in favour of lingual orthodontics.

Considerations Regarding Selection of Appliance

The lingual appliance has a bite opening effect due to the position of lingual brackets on maxillary anterior teeth contacting incisal surfaces of lower incisors in deep bite cases as well as expansive nature. Therefore, the favourable cases are low angle, deep bite cases, mild crowding cases requiring expansion and class II cases requiring maxillary premolar extraction, high angle cases, anterior open bite cases and surgical cases. Unfavourable cases making contraindication to lingual orthodontics are cases having short clinical crowns less than 6mm, poor periodontal status, poor oral hygiene, patients having criticizing and restless tendency with a little hope of understanding the problems of discomfort, high angle cases, severe class II discrepancy and acute TMJ dysfunction (33,34).

The present confirmation on lingual orthodontics demonstrates that any case that can be treated with labial orthodontic apparatus can likewise be dealt with successfully with lingual orthodontic machine. As the quantity of grown-up patients looking for orthodontic treatment is expanding, the interest for stylish orthodontic machine is likewise expanding. Lingual

orthodontics is the main orthodontic apparatus which has preference of finish imperceptibility and 3D control of orthodontic tooth development.

Conclusion

This audit article has arrived at the accompanying conclusion,the confinements of manual setups, for example, repetitive research center strategies, error in section situating, visit debonding of sections, issue in rebonding of sections, troublesome and tedious control of the apparatus, powerlessness to express and control the torque in foremost portion successfully and poor standard of treatment result, now can be effectively tended to with the coming of totally modified lingual machine.

The main issue with the totally tweaked lingual machine today, is the cost of the machine which can be overwhelmed with the accessibility of cutting edge innovation like "metal" 3D printers which straightforwardly can make the metal sections without making the wax designs as in the ebb and flow strategy. Biomechanical standards of lingual orthodontics have been totally comprehended and set up today. The issue of torque control of front teeth can be tended to with the utilization of palatal smaller than normal screws and lever arms which influence the purpose of use of withdrawal to drive at the level of focal point of obstruction of upper foremost teeth amid as once huge mob withdrawal. Patients with lingual machines will probably report a view of enunciation change and shirking of a few sorts of discussion. The patient as a rule protestations of discourse issues identified with the lingual machine that may persevere until 1 month after beginning of the lingual treatment or even following 3 months in a few patients. There is no noteworthy contrast in torment experienced amid treatment between those treated with labial or lingual apparatuses. Ata-Ali et al., in their meta-examination, found a more prominent trade off in oral cleanliness levels with lingual machine It is favorable that lingual settled apparatuses are related with decreased rate of WSLs when contrasted with labial settled apparatus as detailed by different investigations including RCT and orderly audits. A deliberate survey has indicated empowering consequences of the clinical result related with the lingual orthodontic treatment, particularly concerning the accomplishment of individualized treatment objectives with the totally altered lingual apparatus.

Lingual appliance is a feasible option for the patients seeking fixed orthodontic treatment. It is a well – tolerated appliance. Speech problem and tongue soreness may require some more time for adaption to the appliance. Lingual technique due to poor accessibility, variations in anatomy of lingual surfaces, need of a laboratory and complicated mechanics, increased duration and cost, needs a much different consideration as compared to labial orthodontics. Hence, a good communication between the orthodontist and patient is required so that patient is able to understand the problems associated with lingual orthodontics and adopts positive attitude to accept the appliance with good cooperation.

Corresponding Author

Manish Ranjan,

Reader, Department of Conservative Dentistry and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University,

162, Poonamallee High Road, Chennai- 600017,Tamil Nadu, India.

Phone Number: 9543445029

Email Id:**manish.mds30@gmail.com**

Reference

1. Fritz U, Diedrich P, Wiechmann D. Lingual technique—Patients' characteristics, motivation and acceptance. Interpretation of a retrospective survey.JOrofacOrthop. 2002;63(9):227–233.
2. Gorman JC, Smith RJ. Comparison of treatment effects with labial and lingual fixed appliances. Am J OrthodDentofacialOrthop. 1991;99(7):202.
3. Gorman JC. Treatment of adults with lingual orthodontic appliances.Dent Clin North Am. 1988;32(1):589–620.
4. Fujita K. Development of lingual-bracket technique. (Esthetic and hygienic approach to orthodontic treatment) (Part 2) Manufacture and treatment [author's translation]. 1978;19(2):87–94.
5. VanderVeen MH, Attin R, Schwestka-Polly R, Wiechmann D. Caries outcomes after orthodontic treatment with fixed appliances: do lingual brackets make a difference? Eur J Oral Sci. 2010;118(8):298–303.

6. Miyawaki S, Yasuhara M, Koh Y. Discomfort caused by bonded lingual orthodontic appliances in adult patients as examined by retrospective questionnaire. Am J OrthodDentofacialOrthop. 1999;115(3):83–88.
7. Caniklioglu C, Ozturk Y. Patient discomfort: a comparison between lingual and labial fixed appliances. Angle Orthod. 2005;75(6):86–91.
8. Wu AK, McGrath C, Wong RW, Wiechmann D, Rabie AB. A comparison of pain experienced by patients treated with labial and lingual orthodontic appliances. Eur J Orthod. 2010;32(9):403–407.
9. Wu A, McGrath C, Wong RW, Wiechmann D, Rabie AB. Comparison of oral impacts experienced by patients treated with labial or customized lingual fixed orthodontic appliances. Am J OrthodDentofacialOrthop. 2011;139(4):784–790.
10. Aravind Kumar, Lakshmi.T.Efficacy of Natural Products in Fixed Orthodontic Appliances (FOA) treatment a dental note. International Journal of Drug Development &Research, 2011; 3(1):0975-9344.
11. Khattab TZ, Farah H, Al-Sabbagh R, Hajeer MY, Haj-Hamed Y. Speech performance and oral impairments with lingual and labial orthodontic appliances in the first stage of fixed treatment.Angle Orthod. 2012;43(5):58-62.
12. RitikaChhalani, Saravana Dinesh. S.P .Crestal Bone loss around Anchor Units due to Banding –A review.IOSR Journal of Dental and Medical Science. 2014;1(8):75-78.
13. Echarri P. Revisiting the history of lingual orthodontics: A basis for the future. SeminOrthod,2006;12(2):153-9.
14. Scuzzo G, Takemoto K. A new self-ligating lingual bracket with square slots. JClin Orthod, 2011;45(4):682-90
15. Kairalla SA, Scuzzo G. Determining shapes and dimensions of dental arches for the use of straight-wire arches in lingual technique. Dental Press J Orthod, 2014;19(5):116-22.
16. Park KH, BayomeM.New classification of lingual arch form in normal malocclusion using three dimensional virtual models. Korean J Orthod,2015;45(1):74-81.
17. S.Priyanka,SaravanaDinesh.Ortho-Perio Relationship: Is It A Nuptial Knot.Int J PharmBio Sci, 2014; 5(2):100 – 105.
18. Miyawaki S, Yasuhara M, Koh Y. Discomfort caused by bonded lingual orthodontic appliances in adult patients as examined by retrospective questionnaire. Am J Orthod,DentofacialOrthop,1999;114(5):83–8.

19. Sinclair PM, CannitoMF.Patient responses to lingual appliance.JClinOrthod,1986;20(6):396–404.
20. Artun JA.A post-treatment evaluation of multibonded lingual appliances in lingual orthodontics. Eur J Orthod,1987;9(2): 204–10.
21. Caniklioglu C, Ozturk Y. Patient discomfort: a comparison between lingual and labial fixed appliances.Angle Orthod, 2005; 75(8):86–91.
22. Stamm T, Hohoff A, Ehmer U. A subjective comparison of two lingual bracket systems.EurJOrthod,2005;27(3):420–6.
23. Caniklioglu C, Oztürk Y. Patient discomfort: A comparison between lingual and labial fixed appliances. Angle Orthod, 2005;75(1):86–91.
24. Jonathan S. Paleya.Effects of fixed labial orthodontic appliances on speech sound production .Angle Orthodontist,2016;86(3):56-58.
25. Rudolph DJ, Willes PM, Sameshima GT. A finite element model of apical force distribution from orthodontic tooth movement.Angle Orthod, 2001;71(1):127-31.
26. Miller K. A comparison of treatment impacts between Invisalign aligner and fixed appliance therapy during the first week of treatment.American Journal of Orthodontics and Dentofacial Orthopedics ,2007;131(2):302.
27. Attta-Ali F, Ata-Ali J.Adverse effects of lingual and buccal orthodontic techniques: A systematic review and meta-analysis. Am J OrthodDentofacialOrthop, 2016;149(7):820-9.
28. Van derVeen MH, Attin R. Caries outcomes after orthodontic treatment with fixed appliances: Do lingual brackets make a difference?.Eur J Oral Sci, 2010;118(4):298-303.
29. Wiechmann D, Klang E. Lingual appliances reduce the incidence of white spot lesions during orthodontic multibracket treatment. Am J OrthodDentofacialOrthop,2015;148 (3):414-22.
30. Geron S. The lingual bracket jig.J Clin Orthod, 1999;339(9):457-63.
31. FillonD.The resurgence of lingual orthodontics . Clin Impressions,1998;7(1):2-9.
32. CanikliogluC,OzturkY. Patient discomfort: A comparison between lingual and labial fixed appliances. Angle Orthod,2005;75(7):86-91.
33. Romano R. Lingual orthodontics. Decker and co,1998;98(4):34-36.
34. SmithJR,Gorman JC, Kurz C, DUNN rm. Keys to success in lingual therapy-Part I . JClin Orthod,1986;20(2):252-61.

Complications of Root Canal Irrigation - A Review

LeyaMeriam Mathew [1], Jayalakshmi Somasundaram[2], ShanthaSundari[3]

Graduate Student[1],Senior Lecturer[2],Department of Conservative and Endodontics

Professor and Head[2],Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Root canal irrigation plays an important role in the debridement and disinfection of the root canal. The most commonly used irrigants are sodium hypochlorite and hydrogen peroxide and have good benefits of tissue dissolving and disinfection capability. Sodium hypochlorite is an effective agent against a broad spectrum of bacteria and also dissolves the necrotic tissue. However, it also produces toxic effects on the vital tissues resulting in ulcer, necrosis etc. The aim of this review is to analyze the factors causing or affecting the Root canal irrigation during a root canal treatment.

Keywords *swelling, tissue necrosis, pain, haemorrhage, echymosis*

Introduction

The main aim of a root canal treatment is to ensure complete removal of connective tissue and destruction of residual microorganisms found in the infected root canals. To allow proper debridement and disinfection of root canal systems, root canal irritants like sodium hypochlorite and hydrogen peroxide play an important role during the procedure [1]. The complexity of root canal system, presence of numerous dentinal tubules in the roots, invasion of the tubules bymicroorganisms and preservation of healthy dentin after achieving the primary objectives of complete shaping and cleaning of root canal systems are done with the help of a proper irrigation [2]. However many mishaps can occur while cleaning and shaping the root canals with irrigating solutions ranging from damage to the patient's clothing, splashing the irrigant into the patients or operators eye, to allergic reactions. Some of the most commonly used irrigants are sodium hypochlorite, chlorhexidine,ethyelenediaminetetracetic acid (EDTA) and a mixture of tetracycline, an acid and a detergent (MTAD).Hence, the ideal properties of an irrigant [3] include, a potent tissue debris solvent ,low toxicity, low surface tension, lubricant, sterilizing agent, removal of smear layer, low cost and inactive endotoxin

Endodontic Irrigants

Non-Bactericidal Irrigants

Some general dental practitioners either use saline, local anaesthetics and/or distilled water. These have no antibacterial action and will not lessen bacterial significantly. These irrigants may be used regularly as they are easy to use and readily available. In the case of local anaestheticssolutions, they also come in sterile packaging and can be distributed easily through very small gauge needles. Extra contributing factors for their use may be safety. These irrigants should have no role in handling infected root canals. There are a number of better irrigating solutions existing which are more suitable for managing infected root canals.

Bacteriostatic/Bactericidal Irrigants

These include acollection of solutions which also kill bacteria or enable their death by allowing other irrigants to come into contact with the bacteria.

Sodium Hypochlorite (NaOCl)

Sodium hypochlorite was first recognized as an antibacterial agent in 1843 and used as a hand wash with low rates of infection transmission. Its advantages are two-fold; pulpal dissolution and antimicrobial effect. There is evidence to show that a decrease in microbial numbers is achievable when using NaOCl for endodontic treatment of teeth with apical periodontitis. Itwas used as an endodontic irrigant,withlow viscosity ,effective antimicrobial properties but low tissue dissolving capabilitiesacceptable shelf life , easily available and inexpensive [4]. The mechanism of actioninvolvesbreak down of protein by dissolution of amino acid content of vital and necrotic tissue by the available free chlorine in NaOCl

However, certain disadvantages of this irrigant are the toxicity to the vital tissues. The major disadvantage include cytoxicty when injected into periradicular tissue, foul smell and foul taste, fabric bleach on accidental spillage and corrosiveon metals [5]. Studies have also shown extrusion of irrignat the patient experienced a sharp, severe pain and an increased swelling.

Hydrogen Peroxide

Hydrogen peroxide (H_2O_2) is a colorless liquid and has been used in dentistry in concentrations varying from 1% to 30% [6]. H_2O_2 degrades to form water and oxygen. It is active against viruses, bacteria, bacterial spores and yeasts via the production of hydroxyl free radicals which attack proteins and DNA [7]. It has been shown that NaOCl, combined with H_2O_2, is no more effective against E. faecalis than NaOCl alone [8], however, CHX combined with H2O2 was a better antimicrobial agent than either one on their own.

Chlorhexidine

Chlorhexidine digluconate is widely used in disinfection because of its excellent antimicrobial activity. It is highly antimicrobial especially at pH 5.5-7.0 and is known for its longlasting effectiveness even after the removal of the solution but does not provide any tissue dissolving properties [9].It is a positively charged hydrophilic and lipophilic molecule which relates with phospholipids and lipopolysaccharides in cell membranes. Consequently, there is disruption of the cell membranes which allows CHX molecules to enter the cell to cause intracellular toxic effects, such as coagulation of the cytoplasm. Other advantages include available in acceptable flavor and not injurious to the surrounding tissues. Chlorhexidine has a unique property in that it has substantively, i.e. has a persistent residual antimicrobial action.It can theoretically prevent microbial colonization for a period of time after the actual medication period. Commonly, Chlorhexidine is used in conjunction with NaOCl as an irrigant as it raises the effectiveness of the irrigation protocol [10].

However, in spite of the advantages chlorhexidine cannot be considered as an ideal root canal irrigant because of its inability to dissolve necrotic tissue remnants [11]. But capable of dissolving the smear layer [12].Additionally studies have revealed the presence of desquamategingivitis, discoloration of teeth and tongue or dysgeusia associated with it .Laboratory findings showed chlorohexidine to be highly cytotoxic to human periodontal cells, fibroblast via inhibition of protein synthesis [13]. While it does not appear to cause any long term damage to host tissues, it may still cause an inflammatory response in these tissues if expressed beyond the root canal [14].

MTAD

MTAD is a mixture of 3% doxycycline, 4.25% citric acid and detergent developed by Torabinejad et.al. The irrigant has a combination of both chelating and antibacterial properties [15]. MTAD has been reported to be effective in removing the smear layer. The citric acid may serve to remove the smear layer, allowing doxycycline to enter the dentinal tubules and exert an antibacterial effect [16]. MTAD is considered to be more superior to Cholorhexidine in antimicrobial activity and is also7more biocompatible and enhances bond strength [17]. MTAD was seen to be less toxic than eugenol, 3% H2O2, CA (OH) 2 paste, 5.25% NaOCl, Peridex (a

CHX mouthwash with additives) and EDTA, however, was more lethal than NaOCl in absorptions of 2.63%, 1.33% and 0.66%. The procedure for clinical use of MTAD is 20 minutes with 1.3% NaOCl followed by 5 minutes of MTAD and is available from DENTSPLY as Bio Pure MTAD. This irrigant is based on a tetracycline isomer; there may be problems with staining, resistance and sensitivity.

EDTA

Ethylenediaminetetraacetic acid (EDTA) is a synthetic amino acid and the sodium salts of EDTA (Na2EDTA) are used in dentistry. It is often used as a chelating agent and is non-corrosive to instruments. EDTA is not bactericidal nor bacteriostatic but inhibits the growth of, and eventually kills, bacteria by starvation as metallic ions needed for growth are chelated thus are not available for use by micro-organisms . EDTA is relatively nontoxic but is slightly irritating in weak solution. EDTA at concentrations of 15–17% removes calcium from dentine leaving a softened matrix of dentine. It also emulsifies soft tissue and removes the smear layer with no deleterious effect to pulpal or periapical tissues. EDTA reacts with the calcium ions in dentine and forms soluble calcium chelates. It is considered to be superior to saline in reducingtheintracranialmicrobial flora and also helps to detach biofilms adhering to root canal walls [18]. However irrigation with 5%NaOCl or alternated with 17% EDTA, significantly increased the tooth strain .It reduces the chlorine in solution, rendering the sodium hypochlorite irrigant ineffective on bacteria and necrotic tissue [16]. A one minute application of 17% EDTA combined with ultrasonic is efficient for smear layer and debris removal in the apical region of the root canal [19]. EDTA is available in a liquid form for irrigation and a gel form for lubrication (Glide File Prep, Dentsply-Maillefer, and Ballaigues, Switzerland). A well-known alternative is Citric Acid; however, EDTA has been shown to be a faster chelating agent.

Other Irrigating Solutions

The other irrigating solutions are sterile water, physiologic saline, iodine compounds, ureaperoxide, etidronic acid, citricacid, maleic acid, tetraclean, chlorine dioxide etc [20].

HEBP is also known as etidronic acid or etidronate and has been proposed as an alternative potential alternative to EDTA or citric acid because it shows no reactivity with NaOCl [19]. It is nontoxic and has been also used to treat bone disease. However, thedemineralizationkineticswasslower than those of 17% EDTA.

Maleic acid is a mild organic acid used as acid conditioner in adhesive dentistry. Ballal et.al reported that the final irrigation with 7%maleic acid for 1minute was more efficient than 17% EDTA in the removal of smear layer [20].

Iodine Compounds are bactericidal, fungicidal, and virucidal. 2% iodine in 4% potassium iodide has been used in endodontic. It shows less toxicity and a decreased tendency to stain dentine. The main advantage of Iodine is that it is less irritating and toxic. However it is not the first choice as an irrigant. Despite its antimicrobial activity, iodine is a very potent allergen thereby causing a risk for allergy [21]

Curcuma longa (Turmeric): Curcumin, a member of a ginger family possesses anti-inflammatory,anti-oxidant,anti-microbial andanti-canceractivity In an in vitro study conducted by PrasannaNeelakantan, it has been shown that curcumin has significant anti-bacterial activity against E.faecalis and can be used as an alternative to sodium hypochlorite for root canal irrigation [22, 23].Thus this herb can be used especially in endodontics for root canal failure cases.

Complications during Root Canal Irrigation

Root canal irrigation is an integral part of the rootcanal treatment to ensure proper debridement and disinfection of the root canal system. The most frequently used irrigants are sodiumhypochlorite and hydrogen peroxide, or the combined use of both .Their benefits, goodtissue dissolving and disinfecting capability, have been demonstrated in several investigationsA review of these complications have been described briefly below, which necessitates the need to carry out effective techniques in order to avoid complications . However, several mishaps during root canal irrigation have been reported like damage to the

operators or patient's eye, allergic reactions etc. In the event of an accident, emergency treatment guidelines should be applied as may be relevant to each case.

Damage to Clothing

Sodium Hypochlorite, a common bleaching agent can cause a concern of damage. Accidental spillage of minute quantities can lead to rapid, irreparable bleaching [24, 25]. When using an ultrasonic device for root canal irrigation the aerosol may also cause damageto the clothing. These mishapsshould be prevented by proper shield of thepatients' clothing. When using hand irrigation, oneshouldreassure that the irrigation needle and syringe aretightly attached and will not get detached during transferor irrigation in order to prevent leakage over clothing.

Damage to the Eye

Mild burns with the alkali such as sodium hypochlorite can result in significant injury causing blurring of vision and patchy coloration of cornea [26].Irrigant in contact with patients or operators eyes can result in immediate pain, intense burning and erythema. Immediate ocular irrigation with large amounts of tap water or sterile saline should be performed by the dentist.

Damage to Oral mucosa and Skin

Skin injury with an alkaline substance requires an immediate irrigation with water as alkalis combine withproteins or fats in tissue to form soluble protein complexes or soaps which could further cause irritation to the mucosa. Accidental swallowing of irrigant by patient requires proper monitoring. It is possible that skin injury can result from secondary contamination [27, 28].

Allergic reactions

Various allergic reactions to Chlorhexidine have beendescribed. It is known to elicit allergic contact dermatitis, generally after repeated application. It can also contact urticarial,photosensitivity, fixed drug eruption and occupational asthma [29].

The allergic potential of sodium hypochlorite was first reported by Sulzberger when a 32 year old female reported a rapid onset of pain, swelling and difficulty in breathing [30]. A subsequent allergy skin scratch test performed two weeks later confirmed a positive result to sodium hypochlorite. Other symptoms include burning sensation, shortness of breath [31].

Complications Arising from the Irrigant Extrusion beyond Root Canal Apex

Chemical Burns and necrosis

When sodium hypochlorite is extruded beyond the root canal into the periradiculartissues, the effect is a chemical burn leading to localized or extensive tissue necrosis. This can further lead to tissue swelling both intra orally within the surrounding mucosa and extra orally within the skin and subcutaneous tissues. The swelling could be edematous or hemorrhagic which could later produce a sudden onset of pain, associatedbleeding, acute sinusitis, ecchymosis of the mucosa [32]. This reaction of the tissues may occur within minutes or may be delayed.

Neurological Complications

The major complications includeparathesia and anesthesia affecting the mental, inferior dental and infraorbital branches of the trigeminal nerve. Patients may take several months to regain the sensory and motor functions.

Facial nerve damage was first described by Witton et al. in 2005, where the buccal branch of the facial nerve was affected in 2 cases. Both patients exhibited a loss of the naso-labial groove and a down turning of the angle of the mouth. Both patients were reviewed and their motor function, was regained after several months [33].

Upper Airway Obstruction

Without adequate tooth isolation, sodium hypochlorite can lead to the leakage of the solution into the oral cavity and ingestion or inhalation by the patient. This could result in throat irritation and the upper airway could be compromised [34]. Ziegler presented a case of a 15-month-old girl who presented a complication of root canal irrigation with acute laryngotracheal bronchitis, stridor and profuse drooling from the mouth as a result of ingestion of a high concentration of household sodium hypochlorite.

Air Emphysema

Further accidents during endodontic therapy mayoccur when the root canal is dried with compressed air,which may be expressed through the apical constrictioninto the periapicaltissues. Studies show cases of emphysema which occurs when the root canal is dried with compressed air after injecting hydrogen peroxide into it. Symptoms and signs include a rapid swelling and erythema in the region of the treated tooth, emphysema of the face, the suborbital region, and neck. The mainsymptom is a crepitus of the swelling [35].

Flare up

An endodontic flare-up is a complication of endodontic treatment which is defined as an acute exacerbation of asymptomaticpulp, after the initiation or continuation of root canal treatment. Inadvertent extrusion of irrigants beyond the apical foramen will lead to acute reactions. Excessive pressure during irrigation will cause large amounts of irrigant to come in contact with the periapical tissues, thereby a possible etiological factor for Flare up [36].

Conclusion

Based on the various case reports and endodontic literature, special attention must be drawn to the potential risks associated with the use of root canal irrigant for root canal therapy. Thus, it is

important to carry out an effective technique in order to avoid complications. In the event of an accidental extrusion of any solution, treatment guidelines should be applied as may be relevant to each case.

Corresponding Author

ShanthaSundari

Professor and Head,Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University,

162, Ponammalle High Road,Chennai 600077,

Email id: shanthakkss@gmail.com

Telephone:+91 9444443640

References

1. Abraham S, Raj JD, Venugopal M. Endodontic irrigants: a comprehensive review. J Pharm Sci Res. 2015;7:5-9.
2. Torabinejad M, Handysides R, Khademi AA, Bakland LK. Clinical implications of the smear layer in endodontics: a reviewOralSurg Oral Med Oral Pathol Oral RadiolEndod. 2002 Dec;94(6):658-66.

3. Edgar Schafer. Irrigation of the root canal. ENDO 2007 ; 1[1]: 11-27
4. Spencer HR, Ike V, Brennan PA. the use of sodium hypochlorite in endodontics—potential complications and their management. BDJ. 2007 May;202(9):555.
5. Sena NT, Gomes BP, Vianna ME, Berber VB, Zaia AA, Ferraz CC, Souza-FilhoFJIn vitro antimicrobial activity of sodium hypochlorite and chlorhexidine against selected single-species biofilms.IntEndod J. 2006 Nov; 39(11):878-85.
6. Steinberg D, Heling I, Daniel I, Ginsburg. Antibacterial synergistic effect of chlorhexidine and hydrogen peroxide against Streptococcus sobrinus, Streptococcus faecalis and Staphylococcus aureus. J Oral Rehabil. 1999 Feb; 26(2):151-6.
7. Seal GJ, Ng YL, Spratt D, Bhatti M, GulabivalaK .An in vitro comparison of the Streptococcus intermedius biofilms in root canals. IntEndod J. 2002 Mar; 35(3):268-74
8. AzharIqbal, BDS, FCPS .Antimicrobial Irrigants in the Endodontic Therapy.Int J Health Sci (Qassim). 2012 Jun; 6(2): 186–19
9. Estrela Carlos, Silva Julio Almeida, Alencar Ana Helena Gonçalves de, Leles Claudio Rodrigues, Decurcio Daniel Almeida. Efficacy of sodium hypochlorite and chlorhexidine against Enterococcus faecalis: a systematic review. J. Appl. Oral Sci. 2008 Dec; 16(6): 364-368.
10. Naenni N, Thoma K, Zehnder M. Soft tissue dissolution capacity of currently used and potential endodontic irrigants.JEndod. 2004 Nov;30(11):785-7.
11. Mohammadi Z, Abbott PV. The properties and applications of chlorhexidine in endodontics. IntEndod J. 2009 Apr;42(4):288-302.
12. Torabinejad M, Khademi AA, Babagoli J, Cho Y, Johnson WB, Bozhilov K. A new solution for the removal of smear layer. J Endod. 2003;29:170–5.
13. DeivanayagamKandaswamy and Nagendrababu . Root canal irrigants J Conserv Dent. 2010 Oct-Dec; 13(4):256–264.
14. Babich H, Wurzburger BJ, Rubin YL, Sinensky MC, Blau An in vitro study on the cytotoxicity of chlorhexidine digluconate to human gingival c L ells. Cell BiolToxicol. 1995 Apr; 11(2):79-88.
15. Torabinejad M, Cho Y, Khademi AA, Bakland LK, Shabahang S The effect of various concentrations of sodium hypochlorite on the ability of MTAD to remove the smear layer. J Endod. 2003 Apr; 29(4):233-9.

16. Zehnder M, Schmidlin P, Sener B, WaltimoT,Chelation in root canal therapy reconsidered. J Endod. 2005 Nov; 31(11):817-20.
17. Zehnder M. Root canal irrigants. J Endod 2006; 32(5):389-8.
18. Von Der Fehr FR, NygaardÖstbyB. Effect of EDTAC and sulfuric acid on root canal dentine. Oral Surg Oral Med Oral Pathol. 1963;16:199–205.
19. De-Deus G, Zehnder M, Reis C, Fidel S, Fidel RA, Galan J Jr, PaciornikSLongitudinal co-site optical microscopy study on the chelating ability of etidronate and EDTA using a comparative single-tooth model. J Endod. 2008 Jan; 34(1):71-5
20. Ballal NV, Kandian S, Mala K, Bhat KS, Acharya SComparison of the efficacy of maleic acid and ethylenediaminetetraacetic acid in smear layer removal from instrumented human root canal: a scanning electron microscopic study. J Endod. 2009 Nov; 35(11):1573-6.
21. M. Hülsmann& W. Hahn Complications during root canal irrigation – literature review and case reports .IntEndod J. 2000 May;33(3):186-93
22. Neelakantan P, Subbarao C, Sharma S, Subbarao CV, Garcia-Godoy F, Gutmann JL. Effectiveness of curcumin against Enterococcus faecalis biofilm. ActaOdontologicaScandinavica. 2013 Nov 1;71(6):1453-7.
23. Pratishta Jain, Manish Ranjan . Role of herbs in root canal irrigation-A review IOSR Journal of Pharmacy and Biological Sciences 2014; 9(2):06-10.
24. Ingram T A. Response of the human to accidental exposure to sodium hypochlorite. J Endod 1990; 16:235–237.
25. Rutala W, Weber D J. Uses of inorganic hypochlorite (Bleach) in health-care facilities. Clinical Microbiology Reviews 1997; 10:597–610.
26. Marx, Hockberger, Wallis. Rosen's emergency medicine. 6th edn. pp 931–933. UK: Mosby Elsevier, 2006.
27. Krautheim AB, Jermann TH, Bircher AJ Chlorhexidine anaphylaxis: case report and review of the literature. Contact Dermatitis. 2004 Mar; 50(3):113-6
28. Serper A, Ozbek M, Calt S. Accidental sodium hypochlorite-induced skin injury during endodontic treatment. J Endod 2004; 30: 180–181
29. Caliskan M K, Turkun M, Alper S. Allergy to sodium hypochlorite during root canal therapy: a case report. IntEndod J 1994; 27: 163–167.

30. Mehra P, Clancy C, Wu J. Formation of a facial hematoma during endodontic therapy. J Am Dent Assoc 2000; 131: 67–71
31. Sulzberger MB. Dermatologic allergy; an introduction in the form of a series of lectures. The Indian Med Gazette. 1940 Dec; 75(12); 766-767
32. Gernhardt C R, Eppendorf K, Kozlowski A. Toxicity of sodium hypochlorite used as an endodontic irrigant. Int Dent J 2004; 37: 272–280.
33. Witton R, Henthorn K, Ethunandan M. Neurological complications following extrusion of sodium hypochlorite solution during root canal treatment. IntEndod J 2005; 38: 843–848.
34. Ziegler D S. Upper airway obstruction induced by a caustic substance found responsive to nebulised adrenalin. J Paed Child Health 2001; 37:524–525
35. Adnan Torgay, ErdinicAydin, UlkemCilasun, LaleDurmaz, GulnazArslan Subcutaneous emphysema after dental treatment: a case report 2004; 16(3):314- 317
36. Priyanka.S.R , Dr.Veronica, Saveetha Dental CollegeFlare-Ups in Endodontics – A Review IOSR- Journal of Dental and Medical Sciences, 2013; 9(4):26-31

Composite Gene Polymorphism in Periodontitis

S. Mohamed Jaqfer Sadiq[1], Kaarthikeyan G[2], Aarthi Vignesh[3],

Graduate Student[1],Reader[2], Department of Periodontitis,Senior Lecturer[3], Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Specific location on a chromosome is referred to as loci, and the variations in the nucleotide sequence at a locus are termed alleles. When different alleles of a given gene co-exist in the human population, it is called genetic polymorphism.The genetic polymorphisms may in some situations cause a change in the protein or its expression possibly resulting in alterations in innate and adaptive immunity and may thus be deterministic in disease outcome. Genetic polymorphisms may also be protective for a disease. Cytokines, such as Interleukine-1(IL-1), Interleukine-6 (IL-6) and Tumor necrosis factor (TNF-a) plays an important role in focal immunological role in periodontitis. Thus the genetic control on cytokine function affects the appearance and severity of periodontitis. IL-1 is composed of two distinct but functionally similar molecules, IL-1a and IL-1b. Both molecules are produced by macrophages, monocytes and dendritic cells.

Key Words*: Interleukin-A(889), Interleukin-B(3954), Human DNA, Composite Genotype, Polymorphism.*

Introduction

Specific location on a chromosome is referred to as loci, and the variations in the nucleotide sequence at a locus are termed alleles. When different alleles of a given gene co-exist in the human population, it is called genetic polymorphism[1]. The genetic polymorphisms may in some situations cause a change in the protein or its expression possibly resulting in alterations in innate and adaptive immunity and may thus be deterministic in disease outcome. Genetic polymorphisms may also be protective for a disease[2]. Cytokines, such as Interleukine-1(IL-1), Interleukine-6 (IL-6) and Tumor necrosis factor (TNF-a) plays an important role in focal immunological role in periodontitis. Thus the genetic control on cytokine function affects the appearance and severity of periodontitis. IL-1 is composed of two distinct but functionally similar molecules, IL-1a and IL-1b. Both molecules are produced by macrophages, monocytes and dendritic cells.

Association of IL-A-889 and IL-B-3954 in Periodontitis

IL-1a is largely a regulator of intracellular events and a mediator of local inflammation (Dinarello 1996). IL-1 single nucleotide gene polymorphisms (SNP) such as the C/T single basepair substitution in the IL-1A promoter (889) (which is more than 99% concordant with IL-1A G4845T at exon 5) and in the IL-1B13954 locus (formerly referred as IL-1B13953) have been early related to periodontitis and triggered the subsequent publication of numerous epidemiological studies[3].

IL-1 is a powerful and potent bone resorbing cytokine, it stimulates the proliferation of precursor cells of osteoblasts. It induces bone resorption through direct action on osteoclasts and independently of prostaglandin synthesis.

IL-1 compose of 2 distinct but functionally similar molecules, IL-1a and IL-1b. both these molecules are produced by Macrophages, Monocytes and Dentritic cells. Interleukin-1 (IL-1) gene cluster polymorphisms have been associated with an increased risk of developing diseases like osteoporosis, diabetic nephropathy, autoimmune disorders, periodontal disease etc[4].

The cluster of 3 IL-1 genes – IL-1A, IL-1B and IL-1RN that code for IL-1α, IL-1β and IL-ra (receptor antagonist) have been mapped to a 415-kb region on the long arm of chromosome 2 (2q13)[5,6].

IL-1 protein level in gingival fluid is one of the strongest and most consistent predictors of periodontitis severity and progression. IL-1b is a downstream regulator of osteoclastic bone resorption and matrix metalloproteinases 8, 9 and 13, which are implicated in the tissue destruction characteristic of periodontitis[7,8].

The first study of cytokine gene polymorphism was reported by Kornman. Polymorphisms of the IL-1 gene cluster have been implicated as risk or susceptibility factors for a number of diseases including adult periodontitis. This association involves a composite genotype with at least one copy of allele 2 (the rarer allele) of the IL-1A gene at nucleotide -899 located in the promoter region and one copy of allele 2 of the IL-1B gene at nucleotide +3954 located in the 5th exon[9].

Human DNA was extracted from the cellular part of the collected blood samples by means of the QIAamp pro- cedure (QIAamp Blood Kit, Kebo Lab, Go¨teborg, Sweden) and sent to the Lab- oratory of the Division of Molecular and Genetic Medicine, University of Sheffield, UK. Genotyping was per- formed without knowledge of clinical status. Allelic frequencies of single nu- cleotide variations were tested in the gene for IL-1a [IL-1A(π4845)] and in the gene for IL-1b [IL-1B(π3954)]. The common allele was designated as ‘‘1’’ and the rarer allele was designated ‘‘2’’ for each gene variant[10,11].

Composite genotype as stated by kornman refered to the presence of specific polymorphism, allele 2 at IL-1A-889 and IL-1B-3954 loci. [9,12] The prevelance of composite genotype IL-B-3954 was significantly higher in the chronic periodontitis patients(24%) than in aggressive and other types of periodontitis which was 3.3% and 7.4% respectively. This finding was in accordance with the studies that observed increased association of IL-B-3954 in chronic periodontitis in Australian and Brazilian populations [13].

Further functional studies on these SNPs have shown interesting results such as the association of the 889T variant with a fourfold increase in IL-1a levels in the gingival crevicular fluid (GCF)[14].

The polymorphism genotype directly influence the disease pathogenesis via an effect on cytokine synthesis. Allele C of IL-B-3954 is considered to be a important risk factor in chronic periodontitis[15].

This exacerbated expression of IL-B-3954 leads to incrased level of inflammation and tissue destruction. IL-1 α is about 15-fold less potent than IL-1 β which stresses the importance of IL-1 β in pathogenesis of periodontal disease than IL-1 α [16].

The composite genotype showed no association with increased GCF volume and bleeding on probing during development of experimental gingivitis; and on the clinical and radiographic regeneration results following guided tissue regeneration (GTR) therapy[17].

Polymorphisms in the IL4 and IL4RA Genes

Interleukine-4 (IL-4) is a pleiotropic cytokine, which is produced by the T helper 2 cell subpopulation and can rescue B lymphocytes from apoptosis and enhance their survival, thus promoting B-lymphocyte mediated immunity. IL-4 also downregulates macrophage function [23]. The gene for IL4 has been located on chromosome 5q31.1.

Gene polymorphisms studied in the IL4 gene are summarized in Table 6. An IL4 -590 promoter polymorphism and a 70-bp VNTR polymorphism are the most studied polymorphisms of IL4. Case–control studies have not shown any relationship between the IL4 gene polymorphims and susceptibility to CP in several different populations. However, a haplotype of IL4 polymorphisms (carriers of R-alleles in all three SNPs studied) has been associated with CP (17.0% in cases versus 11.0% in controls; OR 1.85) [23]. No association was found for the IL4RA polymorphims in a study on Caucasians [27].

Polymorphisms in the IL6 and IL6R Genes

Multiple roles have been identified for interleukine-6 (IL-6). It is released by different cell types and its secretion levels are determined by the cell type and the nature of the stimulus [25]. The IL6 gene was demonstrated to be localized on chromosome 7p21. IL6 polymorphisms affect the serum levels of circulating interleukin-6. The -174 was found to influence IL-6 expression and production. The 174 R-allele carrier individuals have decreased plasma levels of IL-6 and present lower IL6 gene transcriptional activity when compared with N/N individuals [26].

Therefore a genetically determined low IL-6 response (the 174 R-allele carriers) may hamper individual's defense against periodontal pathogens.The carriage rates of the IL6 174 R-allele varied in Caucasian populations from 44% to 54%, in Brazilian populations from 37% to 67%, and remarkably, the 174 as well as 190 and 597 loci were nonpolymorphic in a Japanese population (Table 7). Three out of six studies in Caucasian and one out of two studies in Brazilian populations have correlated the IL6 174 G>C polymorphism with susceptibility to CP. With regard to the other IL6 gene polymorphisms, the Czech study [20], suggested that the 572 polymorphism may be a protective factor to CP. Furthermore, for the other IL6 polymorphisms only single studies have been reported.

We conclude that the IL6 174 polymorphism may be associated with CP susceptibility. However, a meta-analysis of the IL6 174 polymorphisms did not show any association for this polymorphism with CP [29].

Polymorphisms in the IL10 Gene

Interleukine-10 (IL-10) is considered an antiinflammatory cytokine, downregulating the proinflammatory immune response of the monocytes and macrophages. However, the B lymphocyte stimulatory effect may also stimulate the production of autoantibodies [23]. As a matter of fact, auto-antibodies may play a role in periodontitis [24,26]. IL-10 is produced by monocytes, macrophages, and T cells and plays a role in the regulation of proinflammatory cytokines such as IL-1 and TNF-beta.

The gene encoding for IL-10 is mapped on chromosome 1q31-q32, in a cluster with closely related interleukin genes, including IL-19, IL-20, and IL-24. Several promoter polymorphisms have been described in the IL10 gene: 1087 (1082), 819 (824), 627, 592 (597), and 590. The IL-10 1082, 819, and 592 polymorphisms show strong linkage disequilibrium and form two common haplotypes. The haplotypes may be determined on basis of the IL10 592 polymorphism [15]. The R-allele of the 592 polymorphism has been associated with decreased synthesis of IL-10 in vitro and in vivo [27,28] and may lead to altered synthesis of IL-10 in response to inflammatory stimuli [29]. IL-10 has a protective role towards periodontal tissue destruction, inhibiting both matrix metalloprteinases (MMP) and receptor activator for nuclear factor-B

(RANK) systems [19,20]. Therefore the IL10 592 R-allele carriers may be less protected against bacterial challenge.

Frequency of Composite Genotype in Different Populations

The frequency of this composite genotype has been reported to be varied in different populations. In Caucasians, a prevalence of 30-40% has been reported. In the Chinese population only 2.3% were positive for composite genotype, in the Hispanics it was 23%; in a Thai population it was 1.6%.[18] Walker et al. reported a genotype-positive prevalence of 15% in an African[19].A prevalence of 11.66% of composite genotype has been found in asian population[20].

The 42.9% occurrence of the positive composite genotype found in this study is significantly higher than the 29.1% rate found in Northern European sub- jects of unknown periodontal status or the reported 36.3% prevalence in non- smoking periodontitis patients, but comparable to the 40.6% rate in Australians of essentially European heritage[11,21].

The frequencies of IL-1A-+4845 and IL-1B-+3954were significantly greater in severe periodontitis patients. The distribution of composite genotype (allele 2 of IL-1A+4845and allele 2 of IL-1B+3954) also correlated with the severity of periodontitis. Genotype-positive subjects had a higher mean bleeding index (%) when compared to genotype-negative patients[29].

A prevalence of 11.66% of composite genotype has been found in the Maharashtrian ethnic group, correlating with the severity of periodontitis. In a study evaluating the prevalence of only IL-1B polymorphism among the South Indian subjects, a higher percentage of genotype was observed among cases, but this observation did not reach statistical significant[30].

Association of IL-1 Gene Polymorphism with Coronary Artery Disease

Coronary artery disease (CAD), resulting from atherosclerosis (AS), has become the leading cause of disability and death globally.[22] Evidence suggests that inflammation and immunity play a key role in the pathogenesis of AS and CAD [23].

Patients with pro-inflammatory IL-1(þ) genotypes were at a continuum of risk for the presence of CAD, whereas patients with IL-1(–) genotypes seemed to be insensitive to the risk for CAD.The

genes encoding the pro-inflammatory cytokines IL-1a and IL-1b are among the first to be activated in the course of an inflammatory response[24]. Plasma levels of IL-1a and IL-1b show reproducible interindividual differences and oxidation of phospolipds(OxPL) And elevated level of Lp. Vascular wall cells, such as endothelial cells and smooth muscle cells, as well as macrophages and monocytes, can produce IL-1b and IL-1Ra, and these cytokines are also present in human atherosclerotic lesions[25].

Periodontitis Susceptibility Test

Single genes may contribute to susceptibility, but since we have many interactions at the gene-gene and the gene-environmental levels, the real contribution to disease outcome might not be decisive.It detects the simultaneous presence of a specific form of two interleukin genes—allele 2 at the IL1A+4845 and IL1B+3954 loci. Patients are referred to as being “genotype-positive” if both of these alleles are present. A fundamental premise of the test is that a combination of these alleles is responsible for increased secretion of IL-β, which results in a hyperinflammatory response to a bacterial challenge, thereby predisposing a person to develop severe chronic periodontitis. It is possible to perform genetic testing for several syndromic forms of periodontitis, but there is no evidence that mutations in the genes responsible for these conditions are responsible for the more prevalent forms of non-syndromic aggressive or chronic periodontitis[26,27,28].

Corresponding Author

Aarthi Vignesh,

Senior Lecturer, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077, Tamil Nadu, India.

Email Id: draarthiomfs@gmail.com

Phone Number: +91 8056985855

References

1. Page RC, Offenbacher, S, Schroeder H.E, Seymour, G.J. & Kornnman, KS. Advances in the pathogenesis of periodontitis: Summary of developments, clinical implications and future directions. Periodontology . 1997;14:216-248.
2. Norderyd O. Risk for periodontal disease in a Swedish adult population. Cross-sectional and longitudinal studies over two decades. Swedish Dental Journal Supplement. 1998;132: 1-67.
3. Kornman KS, Crane A, Wang HY, diGiovineFS , Newman MG, pirk FW. The interluiken 1 as a severity of adult periodontitis. J Clin periodontal 1997;24:72-7.
4. Offenbacher S. Periodontal diseases: Pathogenesis. Ann Periodontol 1996;1:821-878.
5. Nicklin MJ, Weith A, Duff GW. Physical map of region encompassing the human interleukin 1a, interleukin-1b and interleukin 1-receptor antagonist genes. Genomics 1994;19:382-4.
6. Nothwang HG, Strahm B, Denish D, Kübler M, Schwabe J, Gingrich JC, et al. Molecular cloning of the IL-1 gene cluster: Construction of an integrated YAC/PAC contig and a partial transcriptional map in the region of chromosome 2q13. Genomics 1997;41:370-8.
7. Kaarthikeyan G, Jayakumar ND, Padmalatha O, Sheeja V, Sankari M, Anandan B. Analysis of the association between interleukin-1beta (+3954) gene polymorphism and chronic periodontitis in a sample of the south Indian population. Indian J Dent Res 2009;20:37-40.
8. Liu D, Xu JK, Figliomeni L. Expression of RANKL and OPG mRNA in periodontal disease: possible involvement in bone destruction. Int J Mol Med 2003;11:17–21.
9. Kornman KS, Crane A, Wang HY, di Giovine FS , Newman MG, pirk FW, et. Al .the interluiken 1 as a severity of adult periodontitis. J Clin periodontal 1997;24:72-7
10. Socransky, S. S., Haffajee, A. D. & Smith, C. (2000) Microbiological parameters associated with IL-1 gene polymorphisms in periodontitis patients. Journal of periodontology. 2000;13:322-343.

11. Papapanou, P. N., Neiderud, A.-M., Papadimitriou, A., Sandros, J. &Dahle´n, G. ‘‘Checkerboard’’ assessments of periodontal microbiota and serum antibody responses: A case-control study. Journal of Periodontology 71,2000;71: 885–897.
12. Kornman KS, di Giovine FS. Genetic variation in cytokine expression. A risk factor of adult periodontitis .ann periodontal 1998;3:327-38.
13. Moreira PR, de Sa AR, Xavier GM, Costa JE, Gomez RS, Gollob KJ, et al .a function interleukine 1 B gene polymoprphism is associated with chronic periodontitis in a sample of brazilian individuals. J periodontal Res 2005;40:306-11.
14. Engebretson, S. P., Lamster, I. B., HerreraAbreu, M., Celenti, R., Timms, J. M., Chaudhary, A. G. A., Di Giovine, F. S. & Kornman, K. S. The influence of interleukin gene polymorphism on expression of interleukin-1b and tumor necrosis factor alpha in periodontal tissue and gingival crevicular fluid. Journal of Periodontology. 1999;70:567–573.
15. SheteAR, Joseph R, Vijayan NN, Srinivas L, Banerjee M. Association of single nucleotide gene polymorphism at interleukin-1beta+3954, −511, and −31 in chronic periodontitis and aggressive periodontitis in Dravidian ethnicity. J Periodontol 2010;81:62-9.
16. Stashenko P, Fujiyoshi P, Obernesser MS, Prostak L, Haffajee AD, Socransky SS. Levels of interleukin 1 beta in tissue from sites of active periodontal disease. J Clin Periodontol 1991;18:548-54.
17. Jepsen S, Eberhard J, Fricke D, Hedderich J, Siebert R, Acil Y. Interleukin-1 gene polymorphisms and experimental gingivitis. J Clin Periodontol 2003;30:102-6.
18. Shete AR, Joseph R, Vijayan NN, Srinivas L, Banerjee M. Association of single nucleotide gene polymorphism at interleukin-1beta +3954, -511, and -31 in chronic periodontitis and aggressive periodontitis in Dravidian ethnicity. J Periodontol 2010;81:62-9.
19. Christgau M, Aslanidis C, Felden A, Hiller KA, Schmitz G, Schmalz G. Influence of interleukin-1 gene polymorphism on periodontal regeneration in intrabony defects. J Periodont Res 2003;38:20-7.

20. Walker SJ, Van Dyke TE, Rich S, Kornman KS, di Giovine FS, Hart TC. Genetic polymorphisms of the IL-1a and IL-1b genes in African– American LJP patients and an African–American control population. J Periodontol 2000;71:723-8.
21. Cullinan, M. P., Westerman, B., Palmer, J. E., Faddy, M. J. & Seymour, G. J.IL-1 genotype and progression of periodontal disease in an Australian population. Journal of Dental Research 79. 2000;220:16-18.
22. Murray CJL, Vos T, Lozano R, Naghavi M, Flaxman AD Disability adjusted life years DALYs for 291 diseases and injuries in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study. Lancet 1990–2010;380:2197–2223.
23. Ross R. Atherosclerosis-an inflammatory disease. The New England Journal of Medicine. 1999;340:115–126.
24. Dinarello CA, Simon A, van der Meer JW. Treating inflammation by blocking interleukin-1 in a broad spectrum of diseases. Nat Rev Drug Discov 2012;11:633–52.
25. Fearon WF, Fearon DT. Inflammation and cardiovascular disease: role of the interleukin-1 receptor antagonist. Circulation 2008;117:2577–9.
26. Research, Science and Therapy Committee of the American Academy of Periodontology. Informational paper: implications of genetic technology for the management of periodontal diseases. J Periodontol. 2005;76(5):850-7.
27. Hassell TM, Harris EL. Genetic influences in caries and periodontal diseases. Crit Rev Oral Biol Med. 1995;6(4):319-42.
28. Michalowicz BS. Genetic and inheritance considerations in periodontal disease. Curr Opin Periodontol. 1993;4:11-17.
29. Agrawal AA, Kapley A, Yeltiwar RK, Purohit HJ. Assessment of single nucleotide polymorphism at IL-1A+4845 and IL-1B+3954 as genetic susceptibility test for chronic periodontitis in Maharashtrian Ethnicity. J Periodontol. 2006;77:1515–21.
30. Kaarthikeyan G, Jayakumar ND, Padmalatha O, Sheeja V, Sankari M, Anandan B. Analysis of the association between interleukin -1β (+3954) gene polymorphism and chronic periodontitis in a sample of the south Indian population. Indian J Dent Res. 2009;20:37–40.

Computerised Tomography in Endodontics- A Review

K.Pavithra[1], Jayalakshmi[2], M.S. Nivedhitha[3],
Graduate Student[1],Senior Lecturer[2], Department of Conservative and Endodontics,
Professor and Head[3], Department of Conservative and Endodontics
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

.

Abstract

This review provides an overview of digital radiography as it exists, including advanced imaging such as computed tomography (CT), cone beam-CT and micro-CT as relevant to the practice of Endodontics.New image reconstruction techniques have been introduced that provide information three-dimensionally to the clinician for routine endodontic and surgical treatment planning. In addition, carious lesions of a certain size and expansions of the maxillary sinus and proximity to the roots were observed. The 3-dimensional image analysis was interesting, but detailed observations could not be made with this technique.CT presents several advantages in comparison with other methods, but otherwise has some limitations. Scanning electron microscopy, stereomicroscopy and confocal laser microscopy can be used for superficial analysis but do not provide 3D images without the requisite of sectioning the samples. Others advantages of CT are the possibility of repeated scanning and the manipulation of image using specific software. On the other hand, a limitation of CT is the impossibility of using for in vivo studies due to the radiation level of exposure.The success of endodontic therapy is directly related to the identification of all root canals for its adequate cleaning.The analysis of internal anatomy is important for knowledge of the complexity of root canal system and planning the treatment. Instrumentation techniques and instruments are evaluated to improve the removal of contamination from root canal. Root canal filling techniques and sealers should be appropriately adapted into root canal walls and the quality of obturation is commonly studied. Microorganisms are the main cause of persistent apical periodontitis and the retreatment is indicated in some cases to decontaminate the root canal system. The aim of this work is to describe the applications of X-ray microtomography in endodontic research

Key Words: *CT scan,CBCT, Root canal treatment, Advantages in dentistry*.

Introduction

Successful management of endodontic problems is reliant on diagnostic imaging techniques to provide critical information about the teeth under investigation, and their surrounding anatomy.[1,2]Since its inception, conventional radiography has remained the mainstay of imaging in Endodontics.[3]In recent decades, however, advances in medical imaging have been applied, with varying success, to the various dental disciplines[4].Among the specific imaging techniques, which have been researched as potential diagnostic and treatment planning tools in Endodontics, are digital subtraction radiology (DSR), tuned aperture computed tomography (TACT), ultrasound (US), magnetic resonance imaging (MRI) and computed tomography (CT) [5]. These imaging techniques have been slow to gain acceptance in Endodontics, for an array of different reasons. As such, conventional radiography, despite its inherent limitations, remains the default imaging system in the field[6].However, the development of cone beam computed tomography (CT) has highlighted the inadequacies of conventional radiography when assessing the unique anatomy of the maxillofacial skeleton [7].

Conventional Computed Tomography (CT) Scan

Computed tomography (CT) of the body uses special x-ray equipment to help detect a variety of diseases and conditions[8].CT scanning is fast, painless, noninvasive and accurate. In emergency cases, it can reveal internal injuries and bleeding quickly enough to help save lives.Computed tomography, more commonly known as a CT or CAT scan, is a diagnostic medical test that, like traditional x-rays, produces multiple images or pictures of the inside of the body.The cross-sectional images generated during a CT scan can be reformatted in multiple planes, and can even generate three-dimensional images[9].These images can be viewed on a computer monitor, printed on film.CT images of internal organs, bones, soft tissue and blood vessels typically provide greater detail than traditional x-rays, particularly of soft tissues and blood vessels.

Working Mechanism of CT

X-ray slice data is generated using an X-ray source that rotates around the object; X-ray sensors are positioned on the opposite side of the circle from the X-ray source. The earliest sensors were scintillation detectors, with photomultiplier tubes excited by caesium iodide

crystals[10]. Caesium iodide was replaced by ion chambers containing high-pressure xenon gas. Initial machines would rotate the X-ray source and detectors around a stationary object. Following a complete rotation, the object would be moved along its axis, and the next rotation started(10). Newer machines permitted continuous rotation with the object to be imaged slowly and smoothly slid through the X-ray ring. These are called helical or spiral CT machines. Instead of a single row of detectors, multiple rows of detectors are used to effectively capture multiple cross-sections simultaneously. [11]

In conventional CT machines, an X-ray tube and detector are physically rotated behind a circular shroud (see the image above right). An alternative, short lived design, known as electron beam tomography (EBT), used electromagnetic deflection of an electron beam within a very large conical X-ray tube and a stationary array of detectors to achieve very high temporal resolution, for imaging of rapidly moving structures, for example the coronary arteries. Cone-beam CT functionality is also an increasingly common function found in medical fluoroscopy equipment; by rotating the fluoroscope around the patient, a geometry similar to CT can be obtained, and by treating the 2D X-ray detector.[11]

Cone Beam Computed Tomography (CBCT)

Cone beam computed tomography (CBCT) is a relatively new method to visualize an individual tooth or dentition in relation to surrounding skeletal tissues and to create three-dimensional images of the area to be examined. The use of CBCT in Endodontics is rapidly increasing worldwide.[13] Compared with traditional radiographic methods, which reproduce the three-dimensional anatomy as a two-dimensional image, CBCT is a three-dimensional imaging method that offers the possibility to view an individual tooth or teeth in any view, rather than predetermined ‘default’ views. [13]Therefore, CBCT can be a powerful tool in endodontic diagnosis, treatment planning and follow-up.Currently, CBCT is used most commonly in the assessment of bony and dental pathologic conditions, including fracture; structural maxillofacial deformity and fracture recognition; preoperative assessment of impacted teeth; and temporomandibular joint imaging; and in the analysis of available bone for implant placement. [12]In orthodontics, CBCT imaging is now being directed toward 3D cephalometry. CBCT diagnosis and 3D simulations with virtual surgery and computer-assisted design and manufacture. Image guidance is an exciting advance that will

undoubtedly have a substantial impact on dentistry.Radiation dose of CBCT is minimal when compared toconventional CT scan.

Working Mechanism of CBCT

CBCT is a recent technology. Imaging is accomplished by using a rotating gantry to which an x-ray source and detector are fixed. A divergent pyramidal-or cone-shaped source of ionizing radiation is directed through the middle of the area of interest onto an area x-ray detector on the opposite side.[14] The x-ray source and detector rotate around a rotation fulcrum fixed within the centre of the region of interest. During the rotation, multiple sequential planar projection images of the field of view (FOV) are acquired in a complete, or sometimes partial, arc. This procedure varies from a traditional medical CT, which uses a fan-shaped x-ray beam in a helical progression to acquire individual image slices of the FOV and then stacks the slices to obtain a 3D representation. [15]Each slice requires a separate scan and separate 2D reconstruction.

Advantage of Cone Beam Ct over Conventional CT

CBCT is well suited for imaging the craniofacial area. It provides clear images of highly contrasted structures and is extremely useful for evaluating bone. [16]The use of CBCT technology in clinical practice provides a number of potential advantages for maxillofacial imaging compared with conventional CT:

1. **X-Ray Beam Limitation**

Reducing the size of the irradiated area by collimation of the primary x-ray beam to the area of interest minimises the radiation dose. [16]Most Cone Beam CT units can be adjusted to scan small regions for specific diagnostic tasks. In dentistry CBCT contains short and long film,short film used for particular area which reduces the radiation dose for the patients whereas long film capable of scanning the entire craniofacial complex when necessary.

2. **Image Accuracy**

The volumetric data set comprises a 3D block of smaller cuboid structures, known as voxels, each representing a specific degree of x-ray absorption. The size of these voxels determines the resolution of the image. [17]In conventional CT, the voxels are anisotropic rectangular

cubes where the longest dimension of the voxel is the axial slice thickness and is determined by slice pitch.

3. Rapid Scan Time

Because Cone Beam CT acquires all basis images in a single rotation, scan time is rapid (10–70 seconds) and comparable with that of medical spiral CT systems. [17]Although faster scanning time usually means fewer basis images from which to reconstruct the volumetric data set, motion artifacts due to subject movement are reduced.

4. Display Modes Unique To Maxillofacial Imaging

Access and interaction with conventional CT data are not possible as workstations are required. Although such data can be converted and imported into proprietary programs for use on personal computers, this process is expensive and requires an intermediary stage that can extend the diagnostic phase. Reconstruction of CBCT data is performed natively by a personal computer.[18] Because the CBCT volumetric data set is isotropic, the entire volume can be reoriented so that the patient's anatomic features are realigned[19]. In addition, cursor-driven measurement algorithms allow the clinician to do real-time dimensional assessment.

5. Reduced Image Artifact

With manufacturers' artifact suppression algorithms and increasing number of projections, our clinical experience has shown that Cone Beam CT images can result in a low level of metal artifact, particularly in secondary reconstructions designed for viewing the teeth and jaws.

Use of CT scanin Dentistry

1.A computed tomography scan is a non-invasive medical test that uses special X-ray equipment to produce multiple images or pictures of the inside of the body and a computer to join them together in cross-sectional views of the area being studied.[20]

2.Traditional panoramic X-rays performed by dentists provide only a limited two-dimensional view. While they can show the height and contour of the jaw bone, they give no indication of the bone width and density and may distort the location of the alveolar nerve.

3.Three-dimensional dental CT imaging takes the guesswork out of implants. This quick and safe diagnostic imaging exam produces life-like spatial views of the mouth that let the

surgeon determine pre-surgically if a patient is an implant candidate[20]. With 3D imaging, a surgeon can proceed with confidence, knowing the amount of bone a patient has, the distance to the alveolar nerve and the exact angles to situate the implant. Dental CT imaging is used when patients are being fitted for implants[21].

4.The more information a surgeon has about the anatomy of the patient's mouth before a dental implant, the better the outcome. Important measurements for the surgeon to know include the width and density of the jawbone ridge in order to assess implant feasibility and the exact placement of the alveolar nerve in order to prevent painful nerve damage.

5.Dental CT imaging can also help visualize nerve location prior to wisdom tooth extraction.[22]

6. In endodontics used to detect apical periodontitis,root canal anatomy and complex morphology.

Uses of Ct in Endodontics

Assessment of Periapical Periodontitis

Apical periodontitis (AP) is the principal disease associated with infection of the root canal system. Currently, the accepted reference standard for the radiological detection of AP is periapicalradiography[23] .However, in the early stages of AP, periapical bone destruction may be minimal or be masked by adjacent anatomy, such that its presence is not manifested on conventional radiographs. Periapical radiographs identified the presence of periapical lesions in 3% of the roots whilst CBCT could demonstrate the presence of AP in 14% of the paired roots. The current evidence suggests that CBCT does have a higher sensitivity compared with periapical radiography for the detection of periapical lesions. The specificity of both types of imaging systems is similar;CBCT should not be used for routine assessment of periapical disease prior to Endodontic treatment. However, it may be indicated to aid the diagnosis of (non)odontogenic pain when clinical examination and conventional radiographic assessment is not clear;CBCT should not be used for the routine assessment of the outcome of root canal treatment[24].

Assessment of Root Canal Anatomy

Investigated to compare charged-couple device and photostimulable phosphor plate digital radiography systems with CBCT to detect the number of root canals in 72 extracted mandibular incisors, first premolars and maxillary first molar teeth. They found that with

digital radiographs, regardless of the system used, Endodontists despite taking parallax radiographs failed to identify at least one root canal in 40% of teeth when compared with CBCT[25].assessed the prevalence of disto-lingual roots in mandibular first molar teeth assessed with conventional radiographs and CBCT; they found that the prevalence of disto-lingual canals was 21% and 33%, respectively, with radiographs and CBCT respectively. Cone beam computed tomography is a useful addition to the endodontists armamentarium for identifying root canals however, it should only be reserved for cases where root canal anatomy cannot be fully appreciated with existing aids, such as parallax radiographs and the dental operating microscope[25].

Pre Surgical Assessment

In addition to revealing radiographic signs of periapicalpathosis and root canal anatomy, CBCT scans accurately determine the relationship of adjacent anatomical structures to teeth with endodontic problems[26]. This clinically relevant information may be useful for treatment planning and the subsequent management of the tooth in question. Cone beam computed tomography should only be considered when existing radiographic techniques do not yield adequate diagnostic information, such as the proximity of the root apices (and associated periapicalpathosis) to neighbouring anatomical structures (e.g. the maxillary sinus and inferior dental canal)[27].

Applications of Dental Trauma Assessment

Radiographic assessment is essential for establishing a differential diagnosis of my traumatic dental injuries.Intra-oral views can help to identify the location, type and severity of the traumatic dental injuries[28] .In addition, periapical radiographs can help to assess: stage of root development in young permanent teeth, periapicalpathosis and the correlation of displaced primary teeth with developing successors and proximity of pulp tissue with tooth fractures[28].

Diagnosis and Management of Root Resorption

Due to its often quiescent onset, varying clinical presentation and potential to affect any part of the root’s external surface or canal wall, the detection of root resorption is often challenging. The definitive diagnosis of root resorption is ultimately dependent on the

radiographic demonstration of the disease process, which in turn is limited by the diagnostic potential of the imaging device used to determine its presence[29].

Advantages of CT

1.There are several advantages that CT has over traditional 2D medical radiography. First, CT completely eliminates the superimposition of images of structures outside the area of interest. Second, because of the inherent high-contrast resolution of CT, differences between tissues that differ in physical density by less than 1% can be distinguished[30].

2.Finally, data from a single CT imaging procedure consisting of either multiple contiguous or one helical scan can be viewed as images in the axial, coronal, or sagittal planes, depending on the diagnostic task. This is referred to asmultiplanar reformatted imagin[30].CT is used in medicine as a diagnostic tool and as a guide for interventional procedures.

3.Sometimes contrast materials such as intravenous iodinated contrast are used. This is useful to highlight structures such as blood vessels that otherwise would be difficult to delineate from their surroundings. Using contrast material can also help to obtain functional information about tissues[31].

4.The software necessary for CT data reconstruction can be run on personal computers, potentiating its use as a chairside diagnostic and treatment planning tool. In addition, multiple slices can be scrolled through in real time producing dynamic images. Cursor driven measurements allow dimensional assessments to be made, again in real-time.

5. In addition, basic image manipulations are possible[32].Window levels can be adjusted, specific areas can be magnified and annotations can be added[32].Surface rendering software is also available.

6.The scan times achievable with CT are short and comparable with panoramic radiography. This is beneficial in that the likelihood of patient movement during the scan is less. In addition, the CT hardware is much large and more expensive than CBCT machines. Therefore, CT is well suited to use in dental practice[33].

Limitations of CT

1. Computed tomography imaging is sometimes affected by radiographic artifacts related to the X-ray beam.

2. When the CT X-ray beam encounters an object of very high density, such as enamel or metallic restorations, lower energy photons in the beam are absorbed by the structure, in preference to higher energy photons.

3. The result is that the mean energy of the X-ray beam increases[34].This is called 'beam hardening' and the phenomenon produces two types of artifact: distortion of metallic structures, called 'cupping artifact', and the appearance of streaks and dark bands between two dense structures.

4. These artifacts can reduce the diagnostic yield of the images.Furthermore, patient movement during the scan can adversely affect the sharpness of the final image[34].

Technical Consideration

CT radiation dose and digital image quality:

The effective dose of CT scans is higher than periapical and panoramic radiography. The effective dose varies between scanners[34]. It is also dependent on the region of the jaw being scanned, exposure settings of the CT scanner, the size of the field of view (FOV), exposure time (s), tube current (mA) and the energy/potential (kV)[28]. An image always contains a degree of ‘noise’, that is, false grey scale level of a single pixel, which may influence thequality of the image generate [35].Too low mA causes noise in the image, conversely a higher mA decreases the amount of noise; however this comes at an increased radiation dose to the patient. Endodontic CT imaging should offer not only 3D assessment of the region of interest, but also generate the images with an adequate spatial resolution to allow detailed assessment of the tooth and the surrounding alveolar anatomy[36].CT systems have significantly lower spatial resolution than intra-oral radiographs[29].

Dose Reduction and Optimisation

To ensure patient safety, personnel who use a CT scanner must have appropriate training and knowledge of patient radiation doses related to the specific CT scanner they are using. For endodontic purposes, the FOV should be limited to the region of interest, that is, the FOV should encompass the tooth (or teeth) under investigation and its surrounding structures. This is an effective way to reduce the patient dose[37].The tube current (mA) selected should be as low as possible, so that the image produced is of sufficient diagnostic yield even though there may be a degree of noise[38].The effective dose is also dependent on the region of the oral cavity being scanned[39].

Conclusion

The decision to expose a patient to a CT investigation must be carried out on a case-by-case selection. The potential benefits of the CT scan should outweigh the potential risks. Radiology is constantly evolving[40].clinicians must regularly update their core knowledge in CT. Postgraduate endodontic programmes should incorporate the use of CT into their curriculum to ensure graduates are competent[41].The impact of CT on decision-making in endodontic treatment planning requires further investigation[42].

Corresponding Author

M.S. Nivedhitha,

Professor and Head,Department of Conservative and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077,Tamil Nadu, India.

Contact Number: 9840912367

E-mail: nivea16@hotmail.com

References

1. Patel S, Dawood A, Whaites E, Pitt Ford T. New dimensions in endodontic imaging: part 1. Conventional and alternative radiographic systems. International endodontic journal. 2009; 42(6):447-462.
2. Cotti E, Campisi G. Advanced radiographic techniques for the detection of lesions in bone. Endodontic Topics. 2004;7(1):52-72.
3. Durack C, Patel S, Davies J, Wilson R, Mannocci F. Diagnostic accuracy of small volume cone beam computed tomography and intraoral periapical radiography for the detection of simulated external inflammatory root resorption. International endodontic journal. 2011;1; 44(2):136-47.
4. Silva JA, Alencar AH, Rocha SS, Lopes LG, Estrela C. Three-dimensional image contribution for evaluation of operative procedural errors in endodontic therapy and dental implants. Brazilian dental journal. 2012;23(2):127-34.
5. Webber RL, Messura JK. An in vivo comparison of diagnostic information obtained from tuned-aperture computed tomography and conventional dental radiographic

imaging modalities. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 1999;88(2):239-47.

6. Nance R, Tyndall D, Levin LG, Trope M. Identification of root canals in molars by tuned-aperture computed tomography. International endodontic journal. 2000;33(4):392-6.
7. Brynolf I. A histological and roentgenological study of the periapical region of human upper incisors. Almqvist&Wiksell; 1967;66(2):224-46.
8. Forsberg J. Radiographic reproduction of endodontic “working length” comparing the paralleling and the bisecting-angle techniques. Oral Surgery, Oral Medicine, Oral Pathology. 1987;64(3):353-60.
9. Forsberg J. A comparison of the paralleling and bisecting-angle radiographic techniques in endodontics. International endodontic journal. 1987;20(4):177-82.
10. Forsberg J. Estimation of the root filling length with the paralleling and bisecting-angle techniques performed by undergraduate students. International endodontic journal. 1987;20(6):282-6.
11. .Whaites E, Drage N. Essentials of dental radiography and radiology. Elsevier Health Sciences; 2013;56(4):544-57.
12. Burger CL, Mork TO, Hutter JW, Nicoll B. Direct digital radiography versus conventional radiography for estimation of canal length in curved canals. Journal of endodontics. 1999;25(4):260-3.
13. Revesz G, Kundel HL, Graber MA. The influence of structured noise on the detection of radiologic abnormalities. Investigative Radiology. 1974;9(6):479-86.
14. Kundel HL, Revesz G. Lesion conspicuity, structured noise, and film reader error. American Journal of Roentgenology. 1976;126(6):1233-8.
15. Bender IB, Seltzer S, Soltanoff W. Endodontic success—A reappraisal of criteria: Part I. Oral Surgery, Oral Medicine, Oral Pathology. 1966;22(6):780-9.
16. Bender IB, Seltzer S, Soltanoff W. Endodontic success—A reappraisal of criteria: Part II. Oral Surgery, Oral Medicine, Oral Pathology. 1966;22(6):790-802.
17. Schwartz SF, Foster JK. Roentgenographic interpretation of experimentally produced bony lesions. Part I. Oral Surgery, Oral Medicine, Oral Pathology. 1971;32(4):606-12.
18. .Andreasen JO. Experimental dental traumatology: development of a model for external root resorption. Dental Traumatology. 1987;3(6):269-87.

19. .Chapnick L, Endo D. External root resorption: an experimental radiographic evaluation. Oral Surgery, Oral Medicine, Oral Pathology. 1989;67(5):578-82.
20. Webber RL, Messura JK. An in vivo comparison of diagnostic information obtained from tuned-aperture computed tomography and conventional dental radiographic imaging modalities. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 1999;31;88(2):239-47.
21. .Rudolph DJ, White SC. Film-holding instruments for intraoral subtraction radiography. Oral Surgery, Oral Medicine, Oral Pathology. 1988;65(6):767-72.
22. .Mozzo P, Procacci C, Tacconi A, Martini PT, Andreis IB. A new volumetric CT machine for dental imaging based on the cone-beam technique: preliminary results. European radiology. 1998;8(9):1558-64.
23. .Nance R, Tyndall D, Levin LG, Trope M. Identification of root canals in molars by tuned-aperture computed tomography. International endodontic journal. 2000;33(4):392-6.
24. .Kristensen K. Radiation protection. Recommendations of the International Commission on Radiological Protection. Fra Sundhedsstyrelsen. 1966;4(12):189-92.
25. .Loubele M, Bogaerts R, Van Dijck E, Pauwels R, Vanheusden S, Suetens P, Marchal G, Sanderink G, Jacobs R. Comparison between effective radiation dose of CBCT and MSCT scanners for dentomaxillofacial applications. European journal of radiology. 2009;71(3):461-8.
26. .Gijbels F, Jacobs R, Sanderink G, De Smet E, Nowak B, Van Dam J, Van Steenberghe D. A comparison of the effective dose from scanography with periapical radiography. Dentomaxillofacial Radiology. 2002 ;31(3):159-63.
27. .Lennon S, Patel S, Foschi F, Wilson R, Davies J, Mannocci F. Diagnostic accuracy of limited-volume cone-beam computed tomography in the detection of periapical bone loss: 360° scans versus 180° scans. International endodontic journal. 2011;44(12):1118-27.
28. .Scarfe WC, Farman AG. What is cone-beam CT and how does it work?. Dental Clinics of North America. 2008;52(4):707-30.
29. .Scarfe WC, Farman AG, Sukovic P. Clinical applications of cone-beam computed tomography in dental practice. Journal-Canadian Dental Association. 2006;72(1):75.

30. .Webber RL, Messura JK. An in vivo comparison of diagnostic information obtained from tuned-aperture computed tomography and conventional dental radiographic imaging modalities. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 1999;88(2):239-47.
31. .Ning R, Chen B. Cone beam volume CT mammographic imaging: feasibility study. Medical imaging. 2001;75:655-64.
32. .Feldkamp LA, Davis LC, Kress JW. Practical cone-beam algorithm. JOSA A. 1984;1(6):612-619.
33. .Wischmann HA, Luijendijk HA, Meulenbrugge HJ, Overdick M, Schmidt R, Kiani K. Correction of amplifier nonlinearity, offset, gain, temporal artifacts, and defects for flat-panel digital imaging devices. Medical imaging. 2002;2:427-37.
34. .Grangeat P. Mathematical framework of cone beam 3D reconstruction via the first derivative of the Radon transform. InMathematical methods in tomography 1991 (pp. 66-97).
35. .Huan LI, Rui QI, Yuxi PA, Junli LI. Development of lymphatic nodes in the Chinese reference adult male voxel model (CRAM) with applications to radionuclide therapy. Journal of Tsinghua University (Science and Technology). 2016;20;56(12):1290-6.
36. .Ludlow JB, Davies-Ludlow LE, Brooks SL. Dosimetry of two extraoral direct digital imaging devices: NewTom cone beam CT and Orthophos Plus DS panoramic unit. Dentomaxillofacial Radiology. 2003;32(4):229-34.
37. .Ludlow JB, Davies-Ludlow LE, Brooks SL, Howerton WB. Dosimetry of 3 CBCT devices for oral and maxillofacial radiology: CB Mercuray, NewTom 3G and i-CAT. Dentomaxillofacial Radiology. 2006;35(4):219-26.
38. .Brynolf I. A histological and roentgenological study of the periapical region of human upper incisors. Almqvist&Wiksell; 1967;45(65):243-35.
39. .Schulze D, Heiland M, Thurmann H, Adam G. Radiation exposure during midfacial imaging using 4-and 16-slice computed tomography, cone beam computed tomography systems and conventional radiography. Dentomaxillofacial Radiology. 2004;33(2):83-6.
40. .Scaf G, Lurie AG, Mosier KM, Kantor ML, Ramsby GR, Freedman ML. Dosimetry and cost of imaging osseointegrated implants with film-based and computed

tomography. OralSurgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 1997;83(1):41-8.

Cone Beam Computed Tomography Imagining In Orthodontics -A Review

Anitha.D.R [1], Saravana Pandian.K [2], Sri Sakthi.D [3]

Graduate Student [1],Senior Lecturer [2], Department of Orthodontics,

Reader [3], Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

A review to identify the current status and the usage of Cone BeamComputed Tomography(CBCT) imagining in orthodontics.Cone beam computed tomography (CBCT) has been a vital tool for imaging diagnostic tool in orthodontics. The utilisation of CBCT for diagnosis and treatment planning has been facilitated by the relative advantages of three-dimensional (3D) over two-dimensional radiography.The current diagnostic and treatment planning indications for CBCT include TMJ morphology and pathology contributing to malocclusion, impacted teeth, cleft lip and palate and skeletal discrepancies requiring surgical intervention,Other situations such as rootresorption, supernumerary teeth, temporomandibular joint (TMJ) pathology, asymmetries.The purpose of this review is to highlight the clinical use of CBCT in orthodontics.Although the history and clinical examination are of prime importance, the use and evolution of three-dimensional (3D) CBCT for imaging is more accurate for diagnosis and treatment planning.

Key Words: *Cone beam computed tomography, Malocclusion, Orthodontic treatment, TMJ, Cleft lip and Cleft palate.*

Introduction

Comprehensive visualisation and records of the craniofacial complex have always beenimportant goals in orthodontic imaging. These tasks have routinely been performed by means of plaster models, photographs and radiographs. Current imaging techniques in the dental office are essentially two-dimensional (2D) representations of three- dimensional (3D) objects. These 2D projections, suffer from several limitations like magnification, distortion, superimposition and misrepresentation of structures[1]. However, cone-beam computed tomography(CBCT) has gained considerable acclaim worldwide in recent years as a viable 3D imaging modality.

CBCT in dentistry is high resolution, low distortion, digital imaging of the hard tissues of the head. Instead of pixels, the resolution is measured in voxels and often is sharper than a conventional CT. Cone-beam refers to the cone shape of the X-ray beam, unlike conventionalCT, which uses a fan-shaped beam to create multiple thin slices[2]. CBCT produces panorex and cephalometric projections, which become 3D when the data is reformatted in a volume. With CBCT technology all possible radiographs can be taken in under 1 minute. The orthodontist now has the diagnostic quality of periapical, panoramic, cephalograms, occlusal radiographs, and TMJ series at their disposal, along with views that cannot be produced by regular radiographic machines like axial views, and separate cephalograms for the right and left sides.

Malocclusion is a three-dimensional problem resulting from vertical, transverse and anterior-posterior discrepancies in the teeth, maxilla or mandible. It comes as little surprise that orthodontists immediately welcomed the 3-D rendering capacity of cone beam computed tomography (CBCT) as a means to optimise diagnosis and treatment planning of malocclusion[3]. CBCT imaging provides accurate measurements, improves localisation of impacted teeth, provides visualisation of airway abnormalities, it identifies and quantifies asymmetry, it can be used to assess periodontal structures, to identify endodontic problems, to plan placement sites for temporary skeletal anchorage devices, and to view condylar positions and temporomandibular joint (TMJ) bony structures of orthodontic diagnosis[4].This review will cover CBCT use in orthodontics.

Three-dimensional CBCT based hard and soft tissue simulations, photographic integrations and superimpositions have ushered in a new era of dynamic CBCT imaging. Future developments in this field offer promises of even greater benefits in orthodontic diagnosis and treatment. The long awaited incorporation of the 3D to our radiographic records is soon becoming a reality. CBCT is the future of orthodontics and the applications in orthodontics seem almost limitless.

Types of Computed Tomography

Computed tomography can be divided into 2 categories based on acquisition x-ray beam geometry; namely: fan beam and cone beam.

In fan-beam scanners, an x-ray source and solid-state detector are mounted on a rotating gantry. Data are acquired using a narrow fan-shaped x-ray beam transmitted through the patient. The patient is imaged slice-by-slice, usually in the axial plane, and interpretation of the images is achieved by stacking the slices to obtain multiple 2D representations. The linear array of detector elements used in conventional helical fan-beam CT scanners is actually a multi-detector array[5]. This configuration allows multi-detector CT (MDCT) scanners to acquire up to 64 slices simultaneously, considerably reducing the scanning time compared with single-slice systems and allowing generation of 3D images at substantially lower doses of radiation than single detector fan-beam CT arrays[1].

CBCT Working Principle

A CBCT scan with a single revolution of the radiation source is sufficient to scan the entire maxillofacial region. CBCT technology is based on the use of a cone-shaped X-ray beam that is directed through the patient and the remnant beam is captured on a flat two-dimensional (2D) detector[6]. The X-ray source and detector are able to revolve about a patient's head, and a sequence of two-dimensional (2D) images is generated. These 2D images are then converted into a 3D image using computer software. The rapid movement of the X-ray tube and digital detector through 180°, or more frequently through 360°, produces essentially instantaneous and precise 2D and 3D radiographic images of an anatomical structure[7].

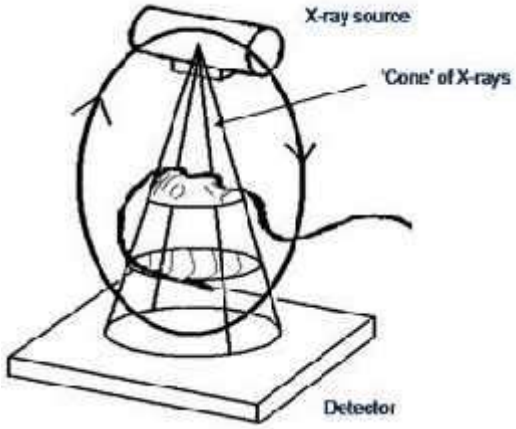


Figure 1: Working principle of CBCT

CBCT Orthodontic Application

Since the introduction of CBCT in the late 1990s, it has become well established as an effective radiographic tool for oral and maxillofacial diagnosis. CBCT is being utilised for many of the same applications CT has been used for in the past. However, with its improved characteristics, such as lower radiation and improved accessibility and affordability, it is being employed to a much greater degree in orthodontic diagnosis and treatment planning. The applications of CBCT in orthodontics include assessment of palatal bone thickness,skeletal growth pattern, severity of tooth impaction, and upper airway evaluation for possible obstructions[8]. CBCT is helpful in treatment planning of orthodontic cases which need buccal tooth movement and arch expansion.In general, orthodontics has relied on 2D X-rays to assess 3D structures. However, CBCT provides a 3D visualisation of the craniofacial skeleton, and this has applications in various orthodontic situations.

Table 1: CBCT in Diagnosis

Diagnosis	Assessment of skeletal structures and dental structures
	Skeletal jaw relation

	Symmetry/asymmetry
	3D evaluation of impacted tooth position and anatomy
	Growth assessment
	Pharyngeal airway analysis
	Assessment of TMJ complex in three dimension
	Cleft palate assessment

Treatment planning	Orthognathic surgery treatment planning in true 1:1 imaging
	Planning for placement of temporary anchorage devices (TADs)
	Accurate estimation to space requirement for unerupted/impacted teeth
	Used in association with CAD/CAM techno logy for construction of custom appliances.

Radiation Dose Considerations

The decision for any radiographic imaging procedure should be performed following the as-low-as-reasonably-achievable (ALARA) principle[9]. CBCT can be justified only if the anticipated information does have the potential to change a patient's treatment modalities or

outcome. Radiation risk has most often been estimated by calculating the effective dose of a CBCT scan and comparing this value to the following: 1) measurements obtained from comparable imaging modalities, 2) background equivalent radiation time or 3) radiation detriment. Often, the base unit of these comparisons (typical panoramic dose, background radiation, weighted probabilities of fatal and nonfatal cancers) is variable and not absolute [10]. This implies that depending on the panoramic image dose used for the comparison, the risk of CBCT may be reported either conservatively or liberally compared with the risk of panoramic radiography. Because CBCT exposes patients to ionising radiation that may pose elevated risks to some high-risk patients (pregnant or younger patients), elaborate explanations and disclosure to patients about radiation exposure risks, benefits, and imaging modality alternatives are crucial and should be documented in the patient records[11].

Application in Orthodontic Diagnosis

Impacted Teeth Evaluation

CBCT is commonly used to assess an impacted tooth and its position.Research has shown that enhanced precision in the localisation of canine teeth and improved estimations of the space conditions in the arch can be obtained with CBCT, and this can greatly affect diagnosis and treatment planning to facilitate a more clinically-orientated approach [12]. Small volume CBCT is also justified as a supplement to routine panoramic X-rays in the following cases: when canine inclination in the panoramic X-ray exceeds 30°, when root resorption of adjacent teeth is suspected, and/or when the canine apex is not clearly discernible in the panoramic X-ray, implying dilaceration of the canine root[13]. When comparing conventional radiography and CBCT, found that CBCT provides more information regarding the location of pathology, the presence of root resorption, and treatment planning. However, the benefits of CBCT imaging must be weighed against the radiation risk to paediatric patients and the complexity of the pathology involved.Ectopic cuspids are a relatively common occurrence the orthodontist must address. The ectopic or impacted incidences of maxillary cuspids is only second to that of the third molar; and has been reported by various authors in ranges that fall between 0.92% and 3%[7,8]. Impactions are twice as common in females as in males.Maxillary impactions are most often located palatally (85%) and of the patients with maxillary impactions, approximately 8% are bilateral[14]. The prevalence of mandibular impactions (0.35%) is much lower than that of maxillary impactions.Surgical

removal of impacted teeth demands precise knowledge of the tooth location in the jaw and its relation to other teeth and surrounding anatomical structures[15].

Bone Quality and Quantity Assessments

With the widespread use of temporary anchorage devices (TADs), the determination of bone volume, bone quality, and the location of adjacent structures have become important in providing orthodontic treatment. It has been shown that a location 4 mm palatal to the incisive foramen provides excellent bone volume for palatal bone screws[16]. A technique using high-resolution CBCT scans and rapid prototyping to fabricate surgical guides has also been described for placing TADs on the buccal aspect of the jaws.

Tooth-Bone Relationships

For orthodontic treatment purposes, the boundary conditions may be defined as the amount (depth and height) and morphology of the alveolar bone relative to tooth root dimensions, angulation, and spatial position[17]. The complex anatomical boundary conditions may limit or dictate the planned or potential tooth movement as well as the final desired spatial position and angulation of the tooth. Patients with alveolar bone phenotypes that clinically appear too narrow to accommodate significant labiolingual or buccolingual displacements or angulations of teeth, patients with compromised periodontium or gingival anatomy or both, and patients in whom the movement of the tooth or teeth may entail translocation past another tooth or obstruction may benefit from CBCT scans for assessing tooth-bone relationships [10,11].

Orthodontic Implant Placement

The knowledge of the root positioning can greatly enhance the opportunity for proper placement and success of orthodontic implants. CBCT images allow more accurate and dependable views of the inter-radicular relationships than panoramic radiographs. CBCT data can be used to construct placement guides foe positioning mini-implants between the roots of adjacent teeth in anatomically difficult sites[18]. The volume and quality of the bone in the proposed placement sites can be evaluated before insertion of the mini-implants.

Orthognathic Surgery

Several applications of CBCT in orthognathic surgery treatment simulation, guidance and outcome assessment have been developed. CBCT 3D surface reconstructions of the jawbones are used for preoperative surgical planning and simulation in patients with traumas and skeletal malformations. Coupled with dedicated software tools, simulations of virtual repositioning of the jaws, osteotomies, distraction osteogenesis and other interventions can now be successfully implemented[19].

Root Morphology and Resorption

Root length, root form and root resorption have traditionally been assessed via periapical radiographs. CBCT imaging becomes a preferred method for diagnosis by orthodontists because of its three dimensional rendering capability. Root position and morphology are critical issues for an orthodontist as it may affect the final occlusion[20]. Root anatomy, such as short or dilacerated, is a determinant factor for the amount and direction of a tooth movement. Furthermore, because of the concerns about external root resorption, orthodontistsneed to get precise measurement of root angulation and length before treatment. Using CBCTimages also provide detailed information about dysmorphic roots. Root positioning and morphology might be indicators of a disease[21]. Of course, all root anomalies are not identical, but when supported with genetic testing, CBCT imaging will be helpful in interpreting anomalous root morphology in syndromic cases.

CBCT could provide enhanced visualisation of roots, making it a valuable tool for assessing pre-orthodontic or post orthodontic root resorption. The mean difference between direct and radiographic CBCT measurements of root length has been shown to be 0.05 mm(SD ¡e. 0.75)[22]. Although these differences were statistically significant for some teeth, the statistical analysis appears not to have corrected for multiple comparisons performed in the study that would have diminished the number of significant differences found. Also determination of measurement error of in vivo root lengths showed an error of approximately 0.2 mm in the pre-and post-treatment measurements[23]. Although these finding some value in the use of CBCTin determining root resorption and morphology, additional studies assessing the efficacy and sensitivity of CBCT in detecting root resorption and evaluating root morphology are recommended.

Supernumerary Teeth

A supernumerary tooth may closely resemble the teeth of the group to which it belongs. In supernumerary cases, radiographic examination aims to determine the localisation and the morphology of the supernumerary teeth. As it is critical to decide which teeth to be extracted and which teeth to be retained, CBCT helps to precisely evaluate the position and morphology of these teeth. It is also possible to detect any contact between the supernumerary teeth and adjacent teeth and to evaluate their relation with other anatomical structures[24]. The information obtained from CBCT images also facilitates the determinationof the optimal surgical access to these teeth in order to minimize harm to adjacent teeth and to surrounding tissue.

Location of Anatomical Structures

Anatomic structures, such as the mental foramen, inferior alveolar nerve, maxillary sinus, and adjacent roots are easily visible using CBCT. CBCT images also allow precise measurement of distance, area and volume which helps the clinicians in treatment planning for sinus lifts, ridge augmentations, extractions and implant placements[25].

Fractured Root

To view root fractures radiographically, it may be difficult if the fracture is in an oblique direction. With CBCT, the tooth of interest can be viewed in all the three planes of space making it easier to determine the site of root fracture and degree of displacement.

TMJ Assessment

The temporomandibular joint (TMJ) is a complex entity with hard and soft tissue components. TMJ disorders (TMDs) are common but widely variable. MRI has sustained its position as the gold standard imaging modality for diagnosing TMDs since it provides excellent visibility of the disk and the associated joint muscles[26]. CBCT para-sagittal and coronal slices show clear images of the condylar head and the glenoid fossa.Additionally, provides images from different orientations and different reconstruction views thus providing axial, coronal and para-sagittal imaging of the condylar head. CBCT is more accurate than panoramic radiography and conventional tomography for detecting TMDs. CBCT exam was also recommended before image-guided puncture operation of the superior compartment of the joint space[18].

Asymmetry Evaluation

Three-dimensional visualisation of the patient allows for a more accurate evaluation of both dental and skeletal asymmetries. Presence of truly unilateral crossbite vs one subsequent to a shift of the mandible into centric occlusion can be determined more easily by viewing and measuring the maxillary and mandibular bones in three dimensions.

Cleft Lip and Cleft Palate

CBCT for patients with cleft lip and palate is useful for both preoperative and therapeutic evaluations.In cleft lip and palate patients, information regarding the number and orientation of teeth, dental and skeletal age, the amount and quality of available bone and bone graft in the cleft region are considered vital for the clinical management.CBCT imaging of the TMJ complex with panoramic radiography and linear tomographic views, and found that the CBCT images were more accurate and showed superior reliability in diagnosing condylar morphology disturbances and erosion[12]. For a complete bilateral TMJ exam, an average of four tomographic cuts in both the lateral and frontal planes are needed for each TMJ. In addition, scout images preceding the actual tomography are needed. In comparison, a CBCT examination requires less time, it includes image data for both the right and left TMJs from a single 360° rotation scan around the patient's head, and it simplifies patient positioning.

Additional advantages include a potentially lower radiation dose and the possibility of views and image manipulation in the form of rotated views. Medical CT is typically used to quantify the amount of bone present. Yet, the young age of cleft patients makes the routine use of medical CT problematic due to the relatively high radiation dose involved[14]. CBCT is rapidly replacing medical CT for this task since it provides excellent 3D visualisation of the palate at the pre-maxilla region at a lower patient dose. CBCT is used to determine dental age and when a large scan field of view FoV selection is available, 3D reconstructions of the cervical vertebra can be made and employed to determine skeletal age. Additionally, CBCT has been used to show any deformities in the piriform margin in the nasal platform and the antero- posterior depression of the nasal alar base.Three-dimensional CBCT reconstructions of the skin surface of the face and nose for cleft lip assessment are also possible[16,17].

Pathologies of Jaw

Presence of radiopaque lesions near the apexes of teeth, such as exostosis, condensing osteitis, dense bone island and focal apical osteoporosis are not readily visualised on

panoramic radiographs. They appear to have no causative factors but can prevent tooth movement. Such lesions can be viewed easily using CBCT images.

Airway Analysis

The CBCT technology provides a major improvement in the airway analysis, allowing for its three- dimensional and volumetric analysis. Airway analysis has conventionally been carried out by using lateral cephalograms[27]. Three-dimensional airway analysis will be useful in understanding the reasons why clinical conditions like sleep apnoea and enlarged adenoids affect the way clinicians manage these complex conditions and alveolar height, especially when multiple units are proposed. This has improved the clinical success of these prostheses, and led to more accurate and aesthetic outcomes in oral rehabilitation.

The introduction of CBCT technology means that both the cost and effective radiation dose can be reduced, suggesting that its frequency of use may increase. The CBCT has already been in use in implant therapy and may be exploited in orthodontics for the clinical assessment of bone graft quality following alveolar surgery in patients with cleft lip and palate[27]. The images produced resulted in greater precision in the evaluation of bone sites and, therefore, gave the clinician a better chance of restoring the site with implants and also influenced the decision-making process of whether to move teeth orthodontically into the repaired alveolus.

Dentofacial Deformities and Craniofacial Anomalies

Evaluation of changes in the craniofacial region during growth and with treatment using lateral cephalograms makes a great contribution to the science of orthodontics. However, in recent years, researches discussed the validity of evaluating a 3D craniofacial structure in a 2D plan. CBCT imaging can provide valuable information about dentofacial deformities and craniofacial anomalies, like facial asymmetry which affects three dimensions of the face, and it can be used to simulate virtual treatment plans for orthopaedic corrections, orthognathic surgeries and distraction osteogenesis[28]. By capturing images and analysing the craniofacial hard and soft tissues and by generating virtual patient models, CBCT imaging permits the clinicians to reposition and reconstruct craniofacial structures.

Application in Orthodontic Treatment Planning

Many of the applications of CBCT in conventional orthodontics also apply in combined orthosurgery treatments. In fact, 3D CT has already been applied to a much greater extent in maxillofacial surgery than in orthodontics[29]. Several applications of CBCT in orthognathic surgery treatment simulation, guidance and outcome assessment have been developed. CBCT3D surface reconstructions of the jawbones are used for preoperative surgical planning and simulation in patients with traumas and skeletal malformations. Coupled with dedicated software tools, simulations of virtual re-positioning of the jaws, osteotomies, distraction osteogenesis and other interventions can now be successfully implemented. Pre and post-operative 3D CBCT skull models can also be registered (i.e. superimposed on each other) to assess the amount and position of alterations in the mandibular rami and condylar head following orthognathic surgery of the maxilla and the mandible 3D reconstructions of the jawbones from CBCT are of sufficient quality for clinical work[27,28].In addition, databases may be interfaced with the anatomical models to provide characteristics of the displayed tissues to reproduce tissue reactions to development, treatment, and function.

Estimation of Space Requirement of Unerupted or Impacted Teeth

CBCT scans enable the accurate localisation of impacted and/or transposed teeth, and this helps determine the best method for surgical access and bond placement. It also helps delineate the ideal and most efficient path for extrusion into the oral cavity to circumvent or decrease collateral damage [18]. Furthermore, CBCT scans provide the orthodontist with valuable information regarding the teeth neighbouring the impacted teeth in terms of root proximity. This information can then be used to place adjacent teeth and their roots away from the traction path of the impacted tooth so as to avoid untoward changes in these teeth[30]. Another advantage of CBCT over conventional radiographs is its capacity to obtain precise dimensions of an impacted tooth, which aids in estimating and creating the necessary space to accommodate the tooth within the arch.

Superimposition

The introduction of CBCT allows clinicians to perform superimpositions in three-dimensions and has eliminated some of the errors that occur with traditional lateral cephalometric superimposition. These 3D superimpositions help in better assessment of treatment outcomes.

Conclusion

CBCT is a valuable tool, and it is particularly important in cases where conventional radiography cannot provide adequate diagnostic information. The use of CBCT imaging is justified when it benefits to the diagnosis and/or treatment plan outweigh the potential risks of exposure to radiation in the presence of individual anamnesis, clinical inspection, and available radiographs. The decision for any radiographic imaging procedure should be performed following the ALARA principle. CBCT can be justified only if the anticipated information has the potential to change a patient's treatment modalities or outcome. CBCT can be used when there is a controversy between the adequacy of conventional radiographs and 3D imaging. These cases include cleft palate patients, assessment of unerupted tooth position, and identification of root resorption caused by unerupted teeth, and planning orthognathic surgery. The later includes cases of cleft palate, craniofacial syndromes, supernumerary teeth, assessment of multiple impacted teeth, and identification of root resorption caused by impacted teeth, and planning for orthognathic surgery. CBCT imaging may also be applied to other types of cases in which it is likely to provide valuable diagnostic information following verification of a positive benefit.

Corresponding author

D.Sri Sakthi

Reader,Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Email: drsrisakthiphd@gmail.com

Reference

1. M.A. Albuquerque, B.F. Gaia, M.G. Cavalcanti. Comparison between multi slice and cone-beam computerized tomography in the volumetric assessment of cleft palate.Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 2011;112: 249–257.
2. W.C. Scarfe, A.G. Farman, P. Sukovic. Clinical applications of cone-beam computed tomographin dental practice. J. Can. Dent. Assoc. 2006; 72:75–80.

3. S.H. Kim, H.G. Yoon, Y.S. Choi, E.H. Hwang, Y.A. Kook, G. Nelson. Evaluation of interdental space of the maxillary posterior area for orthodontic mini-implants with cone-beam computed tomography Am. J. Orthod. Dentofacial Orthop.2009;5:635–64.
4. B.E. Larson. Cone-beam computed tomography is the imaging technique of choice for comprehensive orthodontic assessment Am. J. Orthod. Dentofacial Orthop.2012;141:402–411.
5. E.D. Schneiderman, H. Xu, K.E. Salyer. Characterization of the maxillary complex in unilateral cleft lip and palate using cone beam computed tomography: a preliminary study.J. Craniofac. Surg. 2009; 20:1699–1710.
6. J.K. Mah, R.A. Danforth, A. Bumann, D. Hatcher. Radiation absorbed in maxillofacial imaging with a new dental computed tomography device.Oral Surg. Oral Med. Oral Pathol. Oral Radiol. Endod. 2003;96:508–513.
7. O.J. Van Vlijmen, G.R. Swinnen, E.M. Bronkhorst, C. Katsaros, A.M. KuijpersJagtman
8. Comparison of cephalometric radiographs obtained from cone-beam computed tomography scans and conventional radiographs. J. Oral Maxillofac. Surg. 2009;67:92-97.
9. D.J.Halazonetis. From 2-dimensional cephalograms to 3-dimensional computed tomography scansAm. J. Orthod. Dentofacial Orthop. 2005;127:627-637.
10. A.F. Ferreira, J.C. Henriques, G.A. Almeida, A.R. Machado, N.Machado, A.J. FernandesNeto Comparative analysis between mandibular positions in centric relation and maximum intercuspation by cone beam computed tomography (CONE-BEAM). J. Appl. Oral Sci. 2009;17:27-34.
11. S. Kapila, R.S. Conley, W.E. Harrell Jr. The current status of cone beam computed tomography imaging in orthodontics. Dentomaxillofac. Radiol. 2011;40:24-34.
12. Chaushu S, Chaushu G, Becker A. The role of digital volume tomography in the imaging of impacted teeth. World J Orthod 2004; 5:120–32.
13. Ericson S, Kurol PJ. Resorption of incisors after ectopic eruption of maxillary canines:a CT study. Angle Orthod. 2000;70(6):415- 23.
14. Walker L, Enciso R, Mah J. Three-dimensional localization of maxillary canines with cone-beam computed tomography. Am J Orthod Dentofacial Orthop. 2005;128(4):418-23.

15. Kau CH, Olim S. The future of orthodontic diagnostic records. Semin Orthod. 2009;15(17):39-45.
16. James K, Huang JC. Practical applications of CBCT in orthodontics. Jam Dent Assoc 2010;141.(3):7S-13S.
17. Arai Y, Tammisalo E, Iwai K. Development of a compact computed tomographic apparatus for dental use. Dentomaxillofac Radiol.1999; 28:245.
18. Kook YA, Kim Y. Evaluation of facial asymmetry with three-dimensional cone-beam computed tomography. J Clin Orthod 2011;45:112-5.
19. Lagravore MO, Hansen L, Harzer W, Major PW. Plane orientation for standardizationin 3-D. Cephalometric analysis with computerized tomography imaging. Am J Orthod Dentofacial Orthop. 2006 May;129(5):601-4.
20. Palomo JM, Kau CH, Palomo LB, Hans MG. Three-dimensional cone beam computerized tomography in dentistry. DentToday.2006;25(11):130, 132-5.
21. Tamimi D, ElSaid K. Cone beam computed tomography in the assessment of dental impactions. Semin Orthod 2009;15:57-62.
22. Honey OB, Scarfe WC, Hilgers MJ, Klueber K, Silveira AM, Haskell BS.Accuracy of cone- beam computed tomography imaging of the temporomandibular joint: comparisons with panoramic radiology and linear tomography. American Journal of Orthodontics and Dentofacial Orthopedics.2007;132(4):429- 438.
23. Tuncer BB, Ataç MS, Yuksel S. A case report comparing 3- D evaluation in the diagnosis and treatment planning of hemimandibular hyperplasia with conventional radiography. Journal of CranioMaxillo- Facial Surgery. 2009;37(6):312- 319.
24. Alhammadi MS, Fayed MS, Labib A. Three- dimensional assessment of condylar position and joint spaces after maxillary premolar extraction in skeletal Class II malocclusion. Orthodontics & Craniofacial Research. 2017;6(8):4-6.
25. Subhasri Raman, Jayanth kumar. Cone beam tomography study of prevalence of two or more canal in mandibular anterior in chennai population. J Adv pharm edu res 2017; 7(2): 92-95.
26. Jang HI, Kim SC, Chae JM, Kang KH, Cho JW, Chang NY, Lee KY, Cho JH. Relationship between maturation indices and morphology of the midpalatal suture obtained using cone- beam computed tomography images. The Korean Journal of Orthodontics. 2016;46 (6):345- 355.

27. Roshene, Jayanth kumar.Awareness of CBCT among final years and interns. Jmscr. 2016; 5(4); 10375-10380.
28. Schendel SA, Lane C, Harrell WE Jr. 3D Orthognathic surgery simulation using image fusion. Seminars in Orthodontics. 2009;15:48- 56.
29. Menezes CC, Janson G, Cambiaghi L, Massaro C, Garib DG. Reproducibility of bone plate thickness measurements with cone- beam computed tomography using different image acquisition protocols. Dental Press Journal of Orthodontics. 2010;15(5):143- 149.
30. Wriedt S, Jaklin J, Al- Nawas B, Wehrbein H. Impacted upper canines: Examination and treatment proposal based on 3D versus 2D diagnosis. Journal of Orofacial Orthopedics. 2012;73(1):28- 40.
31. Baumrind S. The road to three- dimensional imaging in orthodontics. Seminars in Orthodontics. 2011;17(1):2- 12.

DeleteriousEffectsofSmokingonOralHealth andOralConditionsAssociatedWithSmoking

Hansini B. Ajantha[1],S. Pavithra Priyadarshoni[2], Abilasha Ramasubramanian[3].

Graduate Student[1],Tutor[2], Department of Public Health Dentistry,Senior Lecturer[3],Department of Oral Pathology.

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

It is well established that smoking contributes to the development of lung disease and cardiovascular disease, and there is evidence that it has a considerable influence on oral health. Smoking has many negative effects on the mouth, including staining of teeth and dental restorations, reduction of the ability to smell and taste, and the development of oral diseases such as smoker's palate, smoker's melanosis, oral candidiasis and dental caries, periodontal disease, implant failure, oral pre cancer and cancer, in which the latter is obviously the most serious tobacco-related effect in the mouth. However, importance has been attached to periodontitis, which affects a large proportion of the population, and during recent years more attention has been given to implant survival rates. As tobacco accounts for such a high proportion of these diseases, comprehensive tobacco control policies are required to make progress in reducing the burden of tobacco-related oral diseases. Effective treatments to prevent tobacco use and increase cessation are available and need greater implementation. Dental practices must provide a uniquely effective setting for tobacco prevention and cessation.

Key Words: *Smoking, Tobacco, Pre-Cancerous Lesions, Periodontitis, Dental Caries*

Introduction

Tobacco is one of the major toxic agents in our civilization. Tobacco comes from a plant that is native to America, around Peru and Ecuador(1). Tobacco was introduced to Europe from America in the fifteenth century, first being used in medicinal purposes. Later, it came to be burnt in pipes for pleasure on a large scale for nearly 100 years in Europe as well as the rest of the world. Pipe smoking gave way to the use of tobacco as snuff and in time to cigars and cigarettes varying from country to country, until cigarette smoking became the dominant form in most of the developed countries between the two world wars(2).Cigarette smoke is highly dynamic and has a complex matrix consisting of a gas phase and a particulate phase with more than 3800 compounds. Among these compounds,60 of them are well-established carcinogens in animals and 15 of them are carcinogens in humans. Some of the carcinogens found in cigarette smoke include polycyclic aromatic hydrocarbons, aldehydes, arsenic, nickel and cadmium(3). Smoking not only harms smokers but also harms the people around them.

Forms of Tobacco and its Pattern of Use

Tobacco is used in both smoke and smokeless forms. Smoking forms are more common in western countries compared to the Middle East and eastern countries. Smoking form includes the use of beedis and cigarattes predominantly with various aids like hooka, hookli, chutta, dhumthi, chillum. However the higher pricing of these products compared to other forms makes this more common amongst the middle and upper socioeconomic classes of population. Beedi is a smoking stick made by rolling a dried piece of rectangular temburni leaf (Diospyrosmelanaxylon) with sun-dried, flaked tobacco arranged in a conical shape. The roll is then secured with the thread. Chutta is a kind of reverse smoking wherein the burnt end is kept inside the mouth. It is made up of coarsely prepared roll of tobacco and prevalent in South-eastern parts of India. Dhumthi is another type of smoking forms made by rolling the tobacco leaves inside the leaf of the jackfruit tree. Hooka is made using metallic or wooden pipes and used for smoking tobacco. This device consists of a spherical receptacle containing water with aromatic substances. The tobacco products pass through the water into the smoking pipe. Hookli is another form of smoking tobacco device which employs a short clay pipe like device. Chillum is a conical clay pipe used to smoke tobacco and tobacco with ganja in northern parts of India. The narrow end of this device is put inside the mouth and wrapped in a wet cloth to act as a filter(4). The smokeless

forms of tobacco includes betel quid, khaini, mawa, snuff, gutka and pan masala. Betel quid consists of areca nut, slaked lime, tobacco mixed and folded in a betel leaf and placed either in the buccal or labial vestibule. Khaini is prepared by mixing roasted tobacco flakes and slaked lime and the mixture is placed in the lower buccal or labial sulcus. Mawa is a mixture of areca nut, tobacco and slaked lime. Snuff is a blackish brown powder obtained from tobacco through roasting and pulverisation. It is generally used through nasal insufflations or applied on the gums using a finger. Gutka is a mixture of areca nut, tobacco and few other condiments and marketed in different flavors n colorful pouches. Pan masala may or may not contain tobacco, with areca nut and few other condiments(5).

This article further gives a short review about the effects and oral conditions associated with smoking and tobacco intervention in dental practice.

Oral Conditions Associated With Smoking

Oral PrecancerousLesion

Leukoplakia

Smokeless palate and smoker’s melanosis

Oral candidiasis and hairy tongue

Oral Cancers

Squamous cell carcinomas of the

• Tongue

• Floor of the mouth

• Lip

Verrucous carcinomas of the

• Buccal mucosa

• Gingiva

• Alveolar ridge

Periodontal Diseases

• Increased plaque and calculus depositions

• Ischaemia

- Gingival inflammation
- Periodontal pockets
- Gingival recession
- Alveolar bone loss

Dental Caries and Saliva

Peri-Implantitis

Aesthetics, Halitosis and Taste Derangement

Leukoplakia

Leukoplakia is believed to be a premalignant lesion associated with development of oral cancer (6). Tobacco smoking is the most important known etiological factor in development of oral leukoplakia. Cross-sectional studies show a higher prevalence rate of leukoplakia among smokers, with a dose-response relationship between tobacco use and oral leukoplakia, and intervention studies show a regression of the lesion after the cessation of smoking(7). Leukoplakia of the floor of mouth appeared to be significantly more often present in smokers than in non-smokers(8). Smokers have a six-fold increase in the risk of developing leukoplakia of the oral mucosa regard to non-smokers.

Smoker's Palate and Smoker's Melanosis

Palatal leukokeratosis**(**smoker's palate**)** is an asymptomatic lesion associated with heavy pipe and cigar smoking usually appearing as white changes in hard palate, often combined with multiple red dots located centrally in small elevated nodule. It may disappear after smoking cessation(9),(10). It does reveal premalignant potential. Melanin pigmentation of the oral mucosa is normally seen in coloured races. Among Caucasian heavy smokers, 30% prevalence in pigmentation is seen, mostly on the attached gingiva(10). A recent study in the Indian population showed that smokers were more likely to develop smoker's melanosis compared to other lesions(11)

OralCandidiasisandHairyTongue

Cigarette smoking is associated with a variety of changes in the oral cavity and it has an effect on oral commensal bacteria and fungi, mainly Candida species, which causes oral candidiasis. How cigarette smoke affects oral Candida is still controversial(12). Further studies and research need to find the exact etiology of smoking and oral candidiasis. It has been seen in the clinical experience that some Candida infections disappear following smoking cessation alone. Another oral lesion, “hairy tongue” or “black hairy tongue” is a benign condition characterized by hypertrophy of the filiform papillae that give the dorsum of the tongue a furry appearance associated sometimes with heavy smoking (13), but its etiology remains unclear.

OralCancer

Cigarette smoking and use of other types of tobacco products cause oral cancer (14). Oral cancer affects mostly middle aged or elderly people and is more common in men than in women. It constitutes 2-3 % of all cancers worldwide. Cigarette smokers have two to five times higher risk of oral cancer than that of non-smoker, the risk increasing with the number of cigarettes and years smoked. Tobacco-specific N-nitrosamines, aromatic amines, and polycyclic aromatic hydrocarbons presented in mainstream tobacco smoke are considered the major carcinogens contributing to the risk of oral cancer from smoked tobacco products (15). Although the underlying mechanisms are not known in detail, it is said that smoking could lead to cancer because carcinogens in tobacco smoke can induce changes in DNA. In recent years much attention has been given to smoking related mutations in a tumor suppressor gene coding for p53 protein. This protein is important in regulating cell proliferation and has a role in the repair of DNA damage (16). Smoking-related mutations in the gene may lead to an accumulation of DNA damage in the cells, which may play an important role in the development of cancer. It has been estimated that between 75 % and 90 % of all cases of oral cancer can be explained by the combined effect of smoking and alcohol use. This could be because alcohol dissolves certain carcinogenic compounds in tobacco smoke and also alcohol increases the permeability of the oral epithelium. Smoking and excessive alcohol intake increases the risk of the development of oral cancer.

Periodontitis

Periodontal diseases, including gingivitis and periodontitis, are common human bacterial infections that affect the gingiva, periodontal tissues and bone supporting the teeth. Gingivitis is a form of inflammation limited to the marginal gingival tissues, and is usually caused by the accumulation of plaque due to inadequate oral hygiene. Periodontitis refers to the destructive inflammation that results in irreversible loss of periodontal attachment and tooth-supporting alveolar bone(17). Gingival recession may result from periodontal destruction and exposure of part of the root surfaces of teeth to the oral environment. The exposed root surfaces are at risk of developing root surface caries. Acute necrotizing gingivitis is also strongly correlated with tobacco use(18). Although the precise cause of this disease remains unknown, it tends to occur most frequently in teenagers and young adults. Tobacco smoking may exert a masking effect on gingival symptoms of inflammation, which might give the smoker a false sense of assurance of gingival health(19). Smoking upregulates the expression of pro-inflammatory cytokines, such asinterleukin-1, which contributes to increased tissue damage and alveolar bone resorption(20). Interleukin-1 genotype positive smokers are more susceptible to severe adult periodontitis(21).

DentalCariesandSaliva

Few studies have shown a relationship between smoking and a higher incidence of dental caries (22),(23). There is no evidence of any direct etiological relationship, but the findings of higher counts of lactobacillus and, although various results are reported, presence of Streptococcus mutans in smokers (24)may explain this relationship.

Tobacco usage stimulates salivary flow, but there isno long term effect on saliva flow rates. Over longer time periods smokers may have slightly reduced pH and buffering power compared to nonsmokers. A recent study showed that smoking is associated with lower salivary cystatin activity and output of cystatin C during gingival inflammation (25).

Peri-Implantitis

Peri-Implantitis is the formation of deep mucosal pockets around dental implants, inflammation of the peri-implant tissues, and increased resorption of implant surrounding bone. Chronic peri-implantitis may lead to implant failure,if left untreated(26). Tobacco use may directly compromise the osseointegration of root-form dental implants(27). The combination of smoking and inflammationinduced by local deposits like plaque and calculus significantly promotes bone loss around the implants(28), whereas occlusal loading has only a minor role.

Aesthetics, HalitosisandTasteDegeneration

Discoloration of teeth, dental restorations and dentures are very frequent among smokers and is more severe than that caused by tea and coffee consumption. Smoking is found to be associated with halitosis(29). It has been seen that smoking influences the decreased function of smell and it is also associated with worsening of taste perception(30).

TobaccoInterventioninDentalPractices

The primary goal is to ensure that every patient who uses tobacco is identified and offered at least a brief intervention at each clinical visit. Tobacco intervention can be either by behavioral or pharmacological management.

BehavioralManagement

Following is the summary of the suggested guidelines(31)

Ask

Askpatients about smoking. A system should be implemented that ensures that every patient at every visit is asked about tobacco use, and the answer documented in the patient’s record.

Advise

Adviseall smokers to stop the habit. The health care professional or the dentist should provide the patients with information and advice, reinforcing the patient's own motivation when possible and emphasizing the benefits of stopping. Dentists should demonstrate the oral effects of tobacco

if present, or inform patients about the increased risk of poor response or healing after dental procedures relevant to the patient (31).

Assess

Assessthe patient's willingness to stop. If the patient is willing to make an attempt to quit, dentists should assist the patient. Dentists should ensure that the patient is aware of the staff's willingness to help, for instance by providing the patient with written information or/and asking the patient in a subsequent visit to reconsider his or her decision (31).

Assist

Assist the patient in stopping the habit. If a patient has a desire to stop, the dentist should help the patient set a realistic quitting date which should be soon but not immediately, so that the patient has time to prepare. If consultation time is limited, self-help materials that provide the patient with necessary information about smoking cessation can be provided. Nicotine replacement therapy (nicotine gum, inhaler, nasal spray, or skin patch) can be very helpful(32). Whatever the approach, the dentist should see to it that the patient leaves the office with a concrete plan for stopping and information about how to prepare for the quitting date and how to successfully stop, keeping in mind that most smokers relapse three to five times before succeeding in stopping.

Arrange

Arrange follow-up contact. Follow-up contacts are very important as the chances of a successful outcome are improved when patients know their progress will be reviewed. The dentist should confirm the quitting date, show continuing support, and follow through if the patient was successful or encourage another try if unsuccessful. If possible the dentist should arrange to see the patients within one or two weeks after the quitting date and consider a second follow-up one or two months later (32).

It is important that the entire dental team is aware of the relationship between smoking and oral conditions associated with smoking. The clinical staff should be familiar with current facts and encouraged to actively participate in tobacco intervention routines.

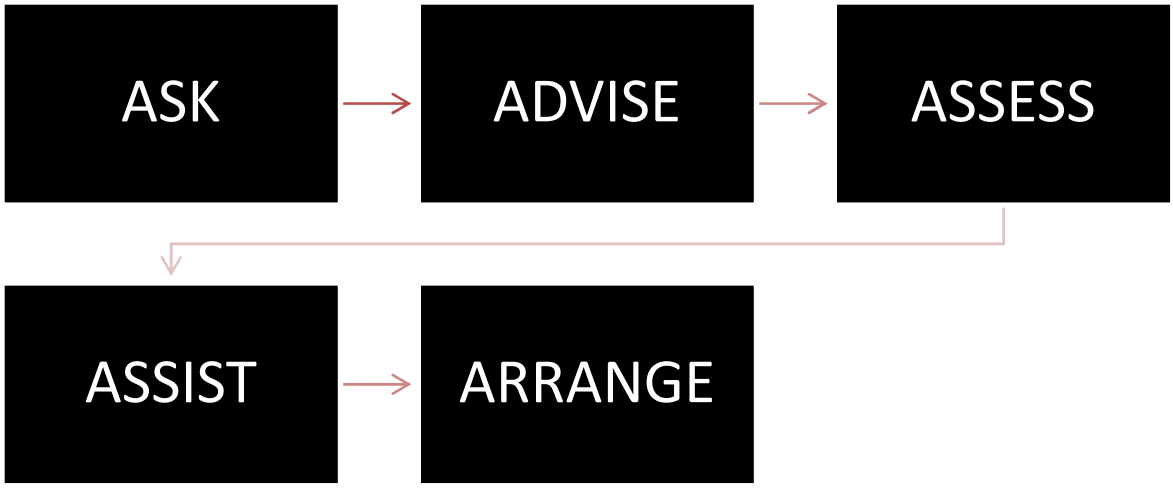


Figure1: The five A's in behavioral tobacco interception

PharmacologicalManagement

The Food and Drug Administration (FDA) has approved five nicotine replacement therapy products for smoking-cessation: Sustained-release bupropion and four nicotine-replacement products such as a gum, a transdermal patch, a nasal spray and a vapor inhaler.

Nicotine ReplacementTherapy

Nicotine replacement therapy provides an alternative form of nicotine to relieve symptoms of withdrawal in a tobacco smoker who is abstaining from tobacco usage. The patch provides a stable, fixed dose of nicotine over a period of 16 to 24 hours. The other nicotine replacement products have a more rapid onset and a shorter duration of action, allowing the user to adjust the dose of nicotine. Blood nicotine levels peak 5-10 minutes after the administration of nicotine nasal spray, 20 minutes after chewing nicotine gum or using a vapor inhaler and 2 to 4 hours after applying a transdermal nicotine patch. The nicotine gum and the inhaler have similar pharmacokinetic properties since, both are absorbed through the oral mucosa. For heavy smokers, the nicotine gum containing 4 mg of nicotine per piece is more effective than that

containing 2 mg per piece (33). The use of all nicotine-replacement products increases the long-term rates of smoking-cessation and relieves cravings for nicotine and symptoms of nicotine withdrawal.

Non NicotineTherapy

Bupropion, an antidepressant with dopaminergic and noradrenergic activity, was efficacious for smoking-cessation when combined with counseling. Other medications like nortryptyline, Selective Serotonin Reuptake Inhibitors like fluoxetine, paroxetine, sertraline, citalopram and Monoamine oxidase inhibitors like moclobemide, selegiline, lazabemide and also antidepressants like tryptophan, imipramine, doxepin, venlafaxine are also used in tobacco cessation (34).

Conclusion

Periodontal diseases, oral cancers, and precancerous development are linked closely with the tobacco usage. Advising patients to quit tobacco use is a dental professional responsibility, and the dental professionals must take an active role in nicotine replacement counseling. Smoking cessation should be incorporated as an integral teaching component of the undergraduate dental curriculum, particularly with respect to the prevention and diagnosis of tobacco-induced oral lesions and complications. Close collaboration of both dentists and physicians with smoking cessation programs must be advocated in the treatment of tobacco-smoking patients.

Corresponding Author

S. Pavithra Priyadarshoni

Tutor, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id: drpavithraphd88@gmail.com

References

1. Musk AW, de Klerk NH...History of tobacco and health.Respirology. 2003; 8(3):286–90.

2. Doll R. Uncovering the effects of smoking: historical perspective. Stat MethodsMed Res. 1998; 7(2):87–117.

3. Adam T, Baker RR, Zimmermann R. Investigation, by single photon ionisation (SPI)-Time-of-flight mass spectrometry (TOFMS), of the effect of different cigarette-lighting devices on the chemical composition of the first cigarette puff. AnalBioanal Chem. 2007; 2:575–84.

4. Pillai HS, Jagannathan N. Tobacco- A potential threat to the oral cavity. Int J Pharm Pharm Sci, 2014, Vol 6, Issue 1, 38-40.

5. Aysha S, Maheswari TU. Occurence of Oral Mucosal Changes among the Smokeless Tobacco Users in Patients Visiting Saveetha Dental College.Journal of Oral Medicine, Oral Surgery, Oral Pathology and Oral Radiology. 2015; 1(3):117-20.

6. Ma N, Tagawa T, Hiraku Y, Murata M, Ding X, Kawanishi S. 8-Nitroguanineformation in oral leukoplakia, a premalignant lesion. Nitric Oxide. 2006; 14(2):137–43.

7. Banoczy J, Gintner Z, Dombi C. Tobacco use and oral leukoplakia. J Dent Educ.2001; 65(4):322–7.

8. Schepman KP, Bezemer PD, van derMeij EH, Smeele LE, van derWaalI.Tobacco usage in relation to the anatomical site of oral leukoplakia. Oral Dis.2001; 7(1):25–7

9. Johnson NW, Bain CA. Tobacco and oral disease. EU-Working Group onTobacco and Oral Health. Br Dent J. 2000; 189(4):200–6.

10. Reibel J. Tobacco and oral diseases. Update on the evidence, with recommendations. MedPrincPract. 2003; 12 Suppl 1:22–32.

11. Saraswathi TR, Ranganathan K, Shanmugam S, Sowmya R, Narasimhan PD, Gunaseelan R. Prevalence of oral lesions in relation to habits: Cross-sectionalstudy in South India. Indian J Dent Res. 2006; 17(3):121–5.

12. Soysa NS, Ellepola AN. The impact of cigarette/tobacco smoking on oral candidiasis:an overview. Oral Dis. 2005; 11 (5):268–73

13. Yuca K, Calka O, Kiroglu AF, Akdeniz N, Cankaya H. Hairy tongue: a case report.ActaOtorhinolaryngolBelg. 2004; 58(4):161–3.

14. Williams SA, Kwan SY, Parsons S. Parental smoking practices and caries experiencein pre-school children. Caries Res. 2000; 34(2):117–22.

15. Winn DM. Tobacco use and oral disease. J Dent Educ. 2001; 65(4):306–12.

16. Nylander K, Dabelsteen E, Hall PA. The p53 molecule and its prognostic role insquamous cell carcinomas of the head and neck. J Oral Pathol Med. 2000; 29(9):
413–25.

17. Ravald N, Birkhed D, Hamp SE. Root caries susceptibility in periodontally treated patients. Results after 12 years. J ClinPeriodontol1993; 20:124-9.

18. Johnson BD, Engel D. Acute necrotizing ulcerative gingivitis. A review of diagnosis, etiology and treatment.J Periodontol 1986; 57:141–50.

19. Bergstrom J, Preber H. Tobacco use as a risk factor. J Periodontol 1994; 65(5 Suppl):545S-550S.

20. Travis J, Pike R, Imamura T, Potempa J. The role of proteolytic enzymes in the development of pulmonary emphysema and periodontal disease. Am J Respir Crit Care Med 1994; 150:143S-146S.

21. Meisel P, Siegemund A, Dombrowa S, Sawaf H, Fanghaenel J, KocherT. Smoking and polymorphisms of the interleukin-1 gene cluster (IL-1alpha, IL1beta, and IL-1RN) in patients with periodontal disease. J Periodontol 2002; 73:27-32.

22. Axelsson P, Paulander J, Lindhe J: Relationshipbetween smoking and dental status in 35-, 50-, 65-, and 75-year-old individuals. J ClinPeriodontol 1998; 25:297–305.

23. Hirsch JM, Livian G, Edward S, Noren JG:Tobacco habits among teenagers in the city ofGoteborg, Sweden, and possible associationwith dental caries. Swed Dent J 1991; 15:117–123.

24. Jette AM, Feldman HA, Tennstedt SL: Tobaccouse: A modifiable risk factor for dental disease among the elderly. Am J Public Health1993; 83:1271–1276.

25. Lie MA, Loos BG, Henskens YM, TimmermanMF, Veerman EC, van derVelden U, van derWeijden GA: Salivary cystatin activity and cystatinC in natural and experimental gingivitis in smokers and non-smokers. J ClinPeriodontol2001; 28:979–984.

26. Grossi SG, Skrepcinski FB, DeCaro T, Zambon JJ, Cummins D, GencoRJ. Response to periodontal therapy in diabetics and smokers. J Periodontol 1996; 67(10 Suppl):1094S-1102S.

27. Wallace RH. The relationship between cigarette smoking and dentalimplant failure. Eur J Prosthodont Restor Dent 2000; 8:103-6.

28. Lindquist LW, Carlsson GE, Jemt T. Association between marginal bone loss around osseointegrated mandibular implants andsmoking habits: a 10-year follow-up study. J Dent Res 1997; 76:1667-74.

29. Hoffmann D, Hoffmann I. The changing cigarette, 1950–1995. J Toxicol Environ Health. 1997; 50(4):307–64.

30. Sato K, Endo S, Tomita H. Sensitivity of three loci on the tongue and soft palate to four basic tastes in smokers and non-smokers. ActaOtolaryngolSuppl.2002 ;(546):74–82.

31. Fiore MC, Bailey WC, Cohen SJ: Treating Tobacco Use and Dependence. Rockville, US Department of Health & Human Services, Public Health Service, 2000.

32. Fiore MC, Bailey WC, Cohen SJ: A clinical practice guideline for treating tobacco use and dependence: A US Public Health Service Report. JAMA 2000; 283:3244–3254.

33. Kaushiksaha, Smoking cessation: How to achieve;The journal of association of chest physicians, 2013, Volume: 1, Issue: 2, 27-31.

34. Kolawole S. Okuyemi, Nicole L. Nollen, Jasjit S. Ahluwalia:Interventions to Facilitate Smoking Cessation, American Family Physician: July 15, 2006 Volume 74, Number 2: 262-271

Dental Anomalies in Pre-Term Children: A Review

Ruksana Sheik[1], Gifrina Jayaraj [2],Jayanth Kumar V[3]

Graduate Student[1], Senior Lecturer[2], Department of Oral Pathology, Reader[3], Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Preterm and low birth weight children comprise approximately 6% of live births. Apart from medical problems they also have dental anomalies. Studies have shown a high prevalence of generalized enamel hypoplasia in the primary dentition of around 40-70 per cent in preterm children which is likely to be associated with low bone mineral stores. Other dental defects observed in preterm children are localized enamel hypoplasia, crown dilacerations, and palatal distortions which are usually associated with traumatic laryngoscopy. The current review would analyse the dental anomalies in preterm children so as to be aware of the possible anomalies they might develop the children can also be monitored and treatment intervention can be done at an early stage.

Key Words:*dental defects, low birth weight, preterm, tooth anomalies, tooth eruption*

Introduction

Prematurity

Preterm births which occur prior to 37 weeks gestation comprise approximately 6 per cent of all live births in developed Western nations. The birth weight of preterm children is often well below that of the average birth weight of 3.3 kg, with the lowest birth weights being experienced by those with the shortest gestational ages. The survival rates of preterm children range from >95 per cent for those with birth weights 2000-2500 g to around 50 per cent for those with birth

weights of 700-800 g. With increasing sophistication of neonatal care in recent years, the survival rates of the most preterm children have improved dramatically, even neonates with birth weights of around 500 g having a 10 per cent chance of survival. [1,2] A number of etiological factors for premature birth exist, many of which are associated with maternal and fetal diseases, but often the cause remains obscure (Yu, 1989), and inborn factors of the child may be of importance. The immaturity of many organs makes a prematurely born infant more prone to neonatal complications and systemic derangements, and there is also interference with the developing teeth, especially those that are at a critical stage at the time of the insult.[3] Many studies about the growth and development of preterm infants with low birth weight indicate that, although some complications may be present on the physical growth some time after birth, they are usually corrected by catch up growth (the growth that occurs at a higher rate than estimated for the age and gender of the child in response to nutritional rehabilitation).[4,5]

Complications of Prematurity

Premature infants are poorly prepared for extrauterine life. Especially, very early and early preterm infants require many weeks of intensive neonatal care. Many serious complications may occur in nearly all the major organ systems. The brain, the lungs, and the eyes are the most susceptible organs. Furthermore, the infants are at increased risk of infections either at or shortly after birth or at a later age. (6,7) Because premature infants often have respiratory distress syndrome resulting from immature lung tissues and a deficiency of surfactant (the mixture of lipoproteins excreted by the alveolar epithelium that lowers surface tension), they have to be treated with oxygen ventilation through an oro-oronasotracheal tube in combination with surfactant administration. Oxygen can also be supplemented with continuous positive airway pressure. (7) Infants aged 35–36 gestational weeks are mature enough to suck and swallow milk. Less mature infants will need to be fed with breast milk supplemented with proteins, calories, and minerals through an oro-or nasogastric tube. (7)

The early and long-term effects of birth prematurity on the physical and psychological growth and development of the child are subjects of considerable current interest. Most studies have indicated that in early childhood preterm children show significant delay in many areas of

physical and psychological growth and development. Although ‘catch-up’ growth has been reported in later childhood, some studies have indicated that long-term delays into adolescence may occur. [3]

Pre term children, especially early preterm children, were shorter, had lower weight, and had a smaller head circumference than full-term controls. (8)Also, less muscle mass has been reported among pre term children. (9)Behavioral symptoms observed in pre term children included increased rate of hyperactivity, difficulties in concentration, and below-grade-level performance at school. (8,10)

In a study conducted by Benjamin et al, 100 (5.55%) pregnancies resulted in Low birth weight neonates out of total 1800 pregnancies. Out of 100 low birth weight infants, 93 survived beyond four weeks of life. (11)

Oral Defects

Like other tissues and organs of the body, the facial bones and dentition can be affected by premature birth. Most studies on oral defects have shown that premature birth can cause enamel defects, classified as quantitative loss of enamel (hypoplasia), qualitative change in the translucence (opacity) of the enamel, or a combination of both. These effects are usually located on the primary teeth, which are undergoing mineralization around the time of the premature birth, although even permanent teeth can be affected.(12)
The pathogenesis is considered multifactorial, the most important factor being calcium disturbances in the neonatal period. However, contributing causes of the enamel defects include local trauma from laryngoscopic and endotracheal intubation, which abuts against the maxillary anterior alveolar ridge.(13)

Other defects, such as notching of the alveolar ridge, palatal grooving, high arched palate, dental crossbite, and palatal asymmetry, have also been reported with higher frequencies when compared with full-term controls. Moreover, delayed eruption and developmental defects of both the primary and permanent dentitions have also been noted.(12)

Many of the studies considering altered morphology have also highlighted that pressure from the oro-or nasotracheal tube or direct trauma from the laryngoscope when the tube is placed might account for the defects.(14)
Thus, the presence of the tube on the palate can conceivably inhibit a normal growth process, and it has also been discussed whether altered morphology of the alveolar ridge and palate can be eliminated by compensating remodeling and growth.(13)
Conceivably, the altered palatal morphology can lead to an increase in malocclusions such as crossbite, resulting in an increasing need for orthodontic treatment. Moreover, changes in the path of eruption of teeth, which can influence the occlusion and tooth spacing, can also contribute to an increasing need for orthodontic treatment.

Oral Complications

Studies suggest that birth is the stimulus for tooth eruption and the eruption time is the same in preterm infants as for full-term infants. (15)
Delayed dental eruption and dental caries are more significant in preterm children as compared to the full term infants. This increased risk is due to local trauma and systematic factors including neonatal asphyxia, respiratory distress syndrome, hyperbilirubinemia and neonatal infection. Premature loss of teeth and malformation of the oral cavity can lead to speech and appearance problems.(16) The prevalence of dental defects is significantly higher in prematurely born children than in their full term counterparts, including enamel hypoplasia, which are reported to exist in both the primary and permanent dentitionand are thought to be associated with a number of systemic derangements typical of pre-term birth, notably hypocalcaemia, metabolic and nutritional disorders, neonatal infections and respiratory distress syndrome.(3)
Prematurely born children show an increased prevalence of developmental defects in enamel in the primary dentition The incidence can be as high as 96%. It has been shown that children born preterm also have an increased frequency of enamel defects in the permanent dentition. However, few studies have been made concerning enamel defects in the permanent dentition of children born preterm and none has prospectively studied both primary and permanent dentitions in the same children.(3)

Primary Dentition

In a study conducted, the mean mesiodistal dimensions of the teeth appeared to be directly related to birthweight, with the very low birthweight groups showing the smallest dimensions and the normal birthweight groups showed the largest dimensions. The low birthweight groups showed measurements in between those of the very low birthweight and normal birthweight groups.(17)

In case of maxillary central incisors, in the very low birthweight group, the mean mesiodistal dimension was 9% less as compared to the normal birthweight group children. In case of maxillary lateral incisors, In the very low birthweight group, the mean mesiodistal dimension was 11% less as compared to the normal birthweight group. (18)

The mandibular central and lateral incisors of the very low lowbirthweight children has the smallest dimensions as compared to the normal birthweight children. (18)

In another study conducted, all 15 children showed dental defects of the primary dentition. Three children (20%) had only dental opacities. In 7 other children, the opacities and hypoplasia were present in different teeth. These opacities, which consisted of white or yellow brownpatches, were located mainly on the buccal and occlusal surfaces. 12 children (80%) showed hypoplasia of at least one tooth. The teeth most commonly showing hypoplasia were the maxillary primary central and lateral incisors followed by the maxillary and mandibular canines. On the other hand, the mandibular first primary molar and canine were the teeth most commonly showing opacities followed by the mandibular second primary molar. (19)

In a study comparing three birthweight groups including a control group of normal birth weight (NBW) children, Seow and co-workers reported a direct relationship of birthweight and gestational age with the prevalence of enamel defects, that is, the VLBW group showed a very high prevalence of enamel defects of over 70 per cent, compared with 50 and 20 per cent in the LBW andNBW gr o u p s respectively. (18) .This high prevalence of enamel defects in the primary dentition of the VLBW group had been confirmed in two more recent studies. (20,21)

Permanent Dentition

In another study conducted 13 (33%) VLBW children showed enamel opacity of at least one permanent tooth, and another four (10%) had enamel hypoplasia of at least one permanent tooth,

giving a total prevalence of 17 (43%) children affected with enamel defects. In contrast, only six (15%) of the NBW children had enamel opacity, and another four (10%) had enamel hypoplasia of at least one permanent tooth,giving a total prevalence of 10 (25%) children with enamel defects. Overall, the prevalence of enamel defects in the VLBW group was significantly higher than the NBW group. (22)

In the VLBW group, four (3%) of the 141 permanent first molars examined showed enamel hypoplasia, and 24 (17%) showed enamel opacity, yielding a total of 30 (20%) teeth affected with enamel defects. Of the NBW children, identical numbers of first permanent molars with enamel hypoplasia compared to the VLBW group were found. Only 10 (8%) teeth were affected with enamel opacity, giving a totalof 14 (11%) teeth affected with enamel defects. The difference in total prevalence of enamel defects in the permanent first molars between the two groups was statistically significant. In contrast, in the case of the permanent central incisors, although the VLBW group showed a slightly higher percentage of enamel defects (5%) than the NBW group (2%), the difference was not significant. However, in the case of the permanent lateral incisors,there was a prevalence of enamel defects of 12% in the VLBW compared with 0% in the NBW group. (22)

One other report,which included a group of 38 premature children with at least one maxillary incisor, gave a prevalence of enamel hypoplasia in the permanent central incisors of 58%, but other permanent teeth were not examined. (23)

Conclusion

Preterm children have a higher prevalence of enamel defects than normal birth weight children. They also have a higher prevalence to enamel hypoplasia. Also the tooth eruption and development is significantly delayed in preterm infants as compared to normal birth weight children. Further research has to be done to get a better insight about pre term children and the dental anomalies associated with them.

Corresponding Author

Jayanth Kumar V,

Reader, Department of Oral Medicine and Radiology,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai -600077, Tamil Nadu, India

Mobile Number: +91 8056135807,

Email: doctorjayanth@gmail.com

References

1. Yu VYH. P r e m at u ri t y. In: Thong YH, ed. Textbook of paediatric practice. Sydney: Butterworths, 1989:357-60.

2.Usher RH. The special problems of the premature infant.In:Avery GB, ed. Neonatology-pathophysiology and management of the newborn. Philadelphia: Lippincourt, 1981:23-261.

3. VirpiHarila-Kaera, TuomoHeikkinen and LassiAlvesaloDepartment of Oral Development and Orthodontics, Institute of Dentistry, University of Oulu, Finland.The eruption of permanent incisors and first molars in prematurely born children.European Journal of Orthodontics.2003;25:293-9.

4.- Fadavi S, Punwani IC; Adeni S; Vidysagar D. Eruption pattern in the primary dentition of premature low-birth-weight children. ASDCJ Dent Child. 1992;59:120-2.

5.-Loevy HT, Raval DS, Aneybuno M, Goldberg AF. Oraldevelopment in children with very low birth weight.J Dent Res.1989;68:942-5.

6. Usher RH. The special problems of the premature infant. In: Avery GB, ed. Neonatalogy—Pathophysiology and Management of the Newborn. Philadelphia, Pa: Lippincott; 1981:23–261.

7.Lissauer T, Claydon G. Neonatal medicine. In: Lissauer T, Claydon G, eds. Illustrated Textbook of Paediatrics. London, UK: Mosby International Limited; 1997:83–100.

8. Stjernqvist K, Svenningsen NW. Ten-year follow-up of children born before 29 gestational weeks: health, cognitive development, behaviour and school achievement. Acta Paediatr. 1999;88:557-562.

9. Hediger ML, Overpeck MD, Ruan WJ, Troendle JF. Birthweight and gestational age effects on motor and social development. Paediatr Perinat Epidemiol. 2002;16:33–46.

10. Stjernqvist K, Svenningsen NW. Extremely low-birth-weight infants less than 901g: development and behaviour after 4 years of life. Acta Paediatr. 1995;84:500–506.

11. Sagayaraj MB, Nair LD, Kumar R, Sharma N. Magnifying Glass on Why Newborns Die: Low Birth Weight and Maternal Factors.Indian Journal of Neonatal Medicine & Research.2014;2(1):7-10

12.Seow WK. Effects of preterm birth on oral growth and development. Aust Dent J. 1997;42:85–91.

13. Seow WK. Oral complications of premature birth. Aust Dent J. 1986;31:23–29.

14. Angelos GM, Smith DR, Jorgenson R, Sweeney EA. Oral complications associated with neonatal oral tracheal intubation: a critical review. Pediatr Dent. 1989;11:133–140

15. 26- Seow WK, Humphrys C, Mahanonda R, Tudehope DI. Dental eruption in low birth-weight prematurely born children: a controlled study. Pediatr Dent. 1988;10(1):39-42.

16.Margo Anne Pritchard Dental health in children born preterm: Review of screening and prevention Neonatal, Paediatric And Child Health Nursing 24vol.7 No.2 July 2004

17. Seow WK, Humphrys C, Tudehope DI. Increased prevalence of developmental dental defects in low-birth-weight children: A controlled study. Pediatr Dent 1987;9:221-5.

18.W.K. Seow, A. Wan. A controlled study of the morphometric changes in the primary dentition of the preterm ,very low birthweight children. J Dent Res 79(1) 2000.

19. W. Kim Seow, John P. Brown, David A. Tudehope, Michael O'Callaghan, Dental defects in the deciduous dentition of prematureinfants with low birth weight and neonatal rickets. American academy of Pedodontics, 1984 vol 6, no. 2

20. Lai PY, Seow WK, Tudehope DI, Rogers Y. Enamel hypoplasia and dental caries in very low birthweight children: a case controlled study. Pediatr Dent (in press).

21. Fearne JM, Bryan EM, Elliman AM, Brook AH, Williams DM. Enamel defects in the primary dentition of children born weighing less than 2000 g. Br Dent J 1990;168:433-7.

22. WK. A controlled study of the development of the permanent dentition in very-low birthweight children. Pediatr Dent 1996;18:379-84.

23. Pimlott JFL, Howley TP, Nikiforuk G, Fitzhardinge PM: Enamel defects in prematurely born, low birthweight infants. Pediatr Dent 7:218-23, 1985.

Dental Aspects of Grape Seed Extract-A Dental Note

Jayajanani.S [1], Lakshmi.T [2], Jayashri.P [3]
Graduate Student [1],Reader [2], Department of Pharmacology,
Senior Lecturer [3],Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Dental diseases are the most prevalent chronic diseases worldwide, and a costly burden to health care services.The treatment of dental diseases is expensive, accounting for between 5% and 10% of total health care expenditures in industrialized countries. In most developing low-income countries, the prevalence rate of dental caries is high and more than 90% of caries is untreated. An estimated 5 billion people worldwide suffer from dental caries (tooth decay).Dental diseases include dental caries, developmental defects of enamel, dental erosion and periodontal disease. In low-income countries, the cost of restorative treatment of dental disease is disproportionately expensive. In order to overcome this, many researches have been conducted to study the uses of plant extracts for treating dental diseases. Grape seed extract(GSE) known as Vitisvinifera.L.These natural extracts are observed to possess various therapeutic uses.This is attributed to the presence of proanthocyanins. Grapes are consumed all over the world and their seeds are rich in phenolic compounds, with an ability to damage microbial cells by exerting an influence on the selective permeability of the plasma membrane, which results in the leakage of vital intercellular substances. GSE has exhibited promise as a source for the manufacture of new generations of antibacterial agents for dental uses without exerting an influence on the biological equilibrium in the oral cavity. Various researches have been conducted to assess their medicinal uses across various fields of Health sciences.This review focuses on the healing and therapeutic uses of GSE in dental treatments.

Key Words:GSE,dental,antibacterial,biofilm.

Introduction

Oral diseases and conditions, including dental caries, periodontal disease, orofacial disorders, and tooth loss, affect more persons than any other disease [1].Oral diseases and/or disorders can affect a person's overall health[2]. Dental caries is a multi-factorial infectious disease that depends on diet and nutrition, microbial infection, and host response. Although the introduction of fluoride has resulted in the reduction of dental caries, the latter is still the most common infectious disease in humans and is especially prevalent in children and people with xerostomia (dry mouth) [3]. In adults, the incidence of root caries was found to increase dramatically with age. Thus, control of caries is of major importance in dentistry and will continue to be for the foreseeable future. Besides dental caries, gingivitis and periodontal disease affect most of the adult population, with the prevalence of severe disease increasing with age [4]. Recent research has shown that oral bacteria may contribute to increased risk of heart attacks, strokes, and lung disease and may be associated with premature childbirth in some women [5,6].Root caries is especially prevalent among the elderly population due to gingival recession and the exposure of susceptible root surface[7].Proanthocyanidin (PA) is a naturally occurring plant metabolite widely available in fruits, vegetables, nuts, seeds, flowers, and bark[8].Commonly used as natural antioxidants and free-radical scavengers, PA has been proven to be safe in various clinical applications and as dietary supplements [9,10]. Grape seed extract (GSE) is a rich source of PA, which has been reported to strengthen collagen-based tissues by increasing collagen cross-links [11]. PA from cranberries inhibited the surface-adsorbed glucosyltransferases and acid production by *S. mutans* [12]. Studies have also shown that PA increased collagen synthesis and accelerated the conversion of soluble collagen to insoluble collagen during development [13,14]. PA-treated collagen matrices were demonstrated to be nontoxic and resisted enzyme digestion in vitro and in vivo [15]. Xie et al. [16] performed a study to examine the effect of GSE on the remineralization and demineralization of the collagen-rich root tissue of human teeth.The following summarizes some of the studies related to grape seed extract and their potential oral health benefits.

Grape Seed Extract and Dental Uses

Addition of Grape seed extract renders phosphoric acid a collagen-stabilizing etchant.

Studies found that grape seed extract (GSE), which is rich in proanthocyanidins, could protect demineralized dentin collagen from collagenolyticactivities following clinically

relevant treatment. Because of proanthocyanidin's adverse interference to resin polymerization, it was believed that GSE should be applied and then rinsed off in a separate step, which in effect increases the complexity of the bonding procedure. The present study aimed to investigate the feasibility of combining GSE treatment with phosphoric acid etching to address the issue. It is also the first attempt to formulate collagen-cross-linking dental etchants. Based on Fourier-transformed infrared spectroscopy and digestion assay, it was established that in the presence of 20% to 5% phosphoric acid, 30 sec of GSE treatment rendered demineralized dentin collagen inert to bacterial collagenase digestion. Based on this positive result, the simultaneous dentin etching and collagen protecting of GSE-containing phosphoric acid was evaluated on the premise of a 30-second etch- ing time. According to micro-Raman spectroscopy, the formulation containing 20% phosphoric acid was found to lead to overetching. Based on scanning and transmission electronic microscopy, this same formulation exhibited unsynchronized phosphoric acid and GSE penetration. Therefore, addition of GSE did render phosphoric acid a collagen-stabilizing etchant, but the preferable phosphoric acid concentration should be <20% [17].

Anti-Arthritic Activity of Grape Seed Ethanolic Extract.

Inflammation is a complex biological process and is an initial response to tissue injury.it is mediated by the release of autacoids and usually precedes the development of the immune response. Rheumatoid arthritis is an auto immune disorder which affects the adult population worldwide. Grape seed extract commonly known as vitisvinifera. It contains proanthocyanidins, a polymer of Catechin molecule. It has antioxidant and free radical scavenging, Anti microbial, Anti diabetic, Immuno modulatory, Anti cariogenic, Hepato protective activity. Grape seed extract exhibits significant anti arthritic activity. The ethanolic seed of Grape extract shows an inhibitory activity at 200-1000mg/ml by inhibiting denaturion of protein and its effect was compared with standard drug diclofenac sodium. Auto antigen production in rheumatoid arthritis is due to denaturation of protein. From the results of the present study it can be stated that ethanolic extract of Grape seed is capable of managing the production of auto antigen and inhibiting the protein denaturation in rheumatoid arthritis [18].

Caries

Dental caries is an infectious microbiological disease of the teeth that results in localized dissolution and destruction of the calcified tissues. It is the second most common cause of

tooth loss and is found universally, irrespective of age, sex, caste, creed or geographic location. It is considered to be a disease of civilized society, related to lifestyle factors, but heredity also plays a role. In the late stages, it causes severe pain, is expensive to treat and leads to loss of precious man-hours. However, it is preventable to a certain extent. The prevalence of dental caries in India is 50%–60%.Dental caries is especially prevalent among the elderly population due to gingival recession and the exposure of susceptible root surface [5].Proanthocyanidin (PA) is a naturally occurring plant metabolite widely available in fruits, vegetables, nuts, seeds, flowers, and bark [7].PA has been proven to be safe in various clinical applications and as dietary supplements [8,9]. Grape seed extract (GSE) is a rich source of PA, which has been reported to strengthen collagen-based tissues by increasing collagen cross-links [19].

Remineralisation

The high consumption of energy drinks can lead to tooth erosion [20].Malinauskas et al. showed that energy drink consumption levels were high in university students, 51% of them routinely drinking energy drinks to improve their ability to exercise and function in daily life [21].Rabobank's research in 2011 indicated that energy drink sales growth will increase by 15%in the next 5 years in Indonesia, confirming a high level of energy drink consumption. This consumption was influenced by a city lifestyle and daily business [22]. Energy drinks have a low pH, which can cause tooth erosion. However, the acidity level alone is not sufficient to predict the erosive potential of energy drinks. The erosive potential is further influenced by the low degree of hydroxyapatite and fluorapatite saturation and the presence of cirate, a substance that can eliminate calcium from saliva and teeth [23].

Demineralization can be prevented by restoring tooth mineral components through the process known as remineralization. Common remineralization agents include fluoride, casein-phosphopeptide– amorphous calcium phosphate (CPP-ACP), and beta-tricalcium phosphate (β-TCP) proven to prevent caries [24,25,26]. Remineralization can also be achieved with natural ingredients that have been used for a long time as medications. One example is grapeseed extract, a supplement that is often used as an antioxidant and is known to contain proanthocyanidin (PA); this extract is an effective remineralization agent [27]. In 2008, Qian Xie et al. showed that application of a 6.5% grapeseed extract could contribute in remineralization of dentin in permanent tooth roots [28]. Research concuted by Mahkameh et

al. In 2013 also showed that a 12.5% grapeseed extract had a positive effect on the remineralization process of primary tooth enamel surfaces [29].Grapeseed extract applied to the enamel surface acted as a remineralization agent by depositing minerals onto lesion surfaces by forming an insoluable complex when mixed with phosphate buffer [27].

Biofilm Inhibition

The inhibitory effects of GSE on biofilm formation are dose dependent. Higher concentrations in the extracts led to a fall of its effectiveness, principally caused by its poor dissolution in water.But it is also observed that these inhibitory effects are less effective than those of chlorhexidine. Thus their clinical efficiency has been evaluated to further improvise their actions that can exceed artificial equivalents [28,29].

Uses of Grape Seed Extract in Inflammatory Diseases.

Periodontal diseases (gingivitis and periodontitis) are chronic inflammatory diseases, which are generally caused by gram-negative bacteria, and feature gingival inflammation. Lipopolysaccharide is a cell wall component of gram-negative bacteria, which inhabit in almost all the subgingival tissues, and acts as pathogenic and exacerbating factors for periodontal diseases through inflammatory response [30,31,32]. One of the main targets of LPS is human gingival fibroblasts (hGFs) that play a pivotal role in inducing periodontal tissues injury through cytokine production such as IL-6 and IL-8 [33,34,35].

Thepretreatment of human gingival fibroblasts (hGFs) with grape seed extract (GSE) containing proanthocyanidin for 1 min elicited cytoprotective effects upon hGFs exposed to harsh environmental conditions; short-term exposure of hGFs in the mitotic phase to pure water or physiologic saline resulted in the low recovery of viable cells [36]. GSE pretreatment improved the recovery of cells exposed to pure water or physiologic saline. In addition, hGFs exposed to GSE for 1 min were proliferous, even after culture in a serum-free medium. In that study, it was also shown that intracellular formation of reactive oxygen species (ROS) induced by culture in serum-free medium was inhibited in cells pretreated with GSE for 1 min. Those results suggested that, because of its cytoprotective effects, GSE could be a novel prophylactic and/or therapeutic agent for oral injury. Several studies have shown that polyphenols (including proanthocyanidin) can protect cells. For instance, the reduced cell viability and oxidative stress in HepG2 cells induced by tert-butyl hydroperoxide can be

mitigated by treatment with proanthocyanidin for 6 h [37].Also, ellagic acid has been shown to ameliorate the cytotoxic effect of paraquat (1,1′-dimethyl-4,4′-bipyridinium dichloride) on human alveolar A549 cells *via*its antioxidant action [38].In addition, epigallocatechin-3-gallate shows a cytoprotective effect on mycotoxin-induced cytotoxicity in the human colon adenocarcinoma cell line HT29 through anti-oxidative and anti-inflammatory mechanisms [39]. Also, lemon grass (*Cymbopogoncitratus*Stapf) polyphenols can protect human umbilical vein endothelial cells from oxidative damage induced by high glucose, hydrogen peroxide (H_2O_2), and oxidized low-density lipoprotein [40]. Such studies suggest that cytoprotective effects are exerted *via* the antioxidant action of those polyphenols.

Grape Seed Extract and Oral Squamous Cell Carcinoma.

Oral squamous cell carcinoma (OSCC) poses an important health concern all over the world. Standard treatment plan for oral squamous cell carcinoma depends on the stage of the disease. Early-stage tumors are treated primarily by surgery or radiotherapy, with both modalities resulting in similar local control and survival rates. More advanced carcinomas often require multi-modality therapy with surgery, radiation and chemotherapy, which can result in very high morbidity [41]. Treatment modalities of oral SCC have numerous side effects. Radical surgery can result in disfigurement and functional impairment. Common side effects of radiotherapy include mucositis, oral candidiasis, loss of taste and xerostomia, which may be permanent due to the detrimental effect of radiation on salivary glands [42]. Osteoradionecrosis of bones within the radiation field (most commonly the mandible) may occur as a result of damage to the bone vasculature and osteocytes and is one of the most seriouscomplications of radiotherapy [43]. Current chemotherapeutic drugs exert many cytotoxic effects on normal cells, resulting in adverse systemic and cytotoxic effects and development of resistance to therapy in OSCC patients. Despite continuing research and advances in treatment, the clinical outcomes for HNSCC have not improved significantly over the last several decades, with the overall 5-year survival rate as low as 50% [44,45,46];therefore, development of new treatment modalities is crucial for prevention and to reduce mortality. Herbal medicines (plant-derived compounds), in combination with routinely implemented treatment modalities, such as radiotherapy and surgery, can reduce morbidity and mortality in OSCC patients. Recent studies focus on herbal medicines as potent anti-cancer drug candidates. Previous studies have revealed the anticancer activities of herbal extracts against oral squamous cell carcinoma cell lines. These include Tamoxifen in

combination with Cisplatin, 5-Fluorouracil, Cordycepin, Scutellariabaicalensis, Quercetin and Artemisinin. In a study by Ghashmet et al, time-and dose-dependent inhibitory effect of anti-proliferative activity of local honey (Tualang) on OSCC and HOS cell lines was investigated by MTT assay, light and fluorescent microscope, and Annexin V-FITC Apoptosis Detection Kit [47].In a study by Hseu et al, ethanol (70%) extracts of *Alpiniapricei* rhizome could induce G2/M phase of the cell cycle arrest and apoptosis in KB cells [48].Grapes (*Vitisvinifera*) are rich in flavonoids, polyphenols, anthocyanin, proanthocyanidins, trans- resveratrol, and procyanidins. Recent studies have revealed that GSE has antioxidant and free radical scavenging activities, and anti-diabetic, cardioprotective, hepatoprotective, anti-carcinogenic, antimicrobial, and anti-viral effects. Dinicola et al showed that GSE has anti-proliferative and apoptotic effects on CaCO2 and HCT-8 colon cancer cell lines [49] Sun et al [50] indicated that GSEs have the ability to inhibit HNSCC cell invasion by targeting the expression of EGFR and activation of NF-κ B as well as inhibiting the epithelial-to-mesenchymal transition In the present study, for the first time the cytotoxic effects of the grape seed extract was investigated and their possible effects on cell death properties in KB cell line were determined. Furthermore, the probable side effects of the grape seed extract on HUVEC were evaluated. The results showed that grape seed extract inhibited the growth of oral squamous cell carcinoma through the induction of apoptosis. The IC50 values strongly indicated that the GSE had a potent cytotoxic effect on KB cells. The crucial results of our study addressed to selectivity of GSE effects on KB vs. HUVEC cells (effect was observed on KB, but HUVEC cells) indicate possibility for its promising use for its promising use for the treatment oral squamous cell carcinoma. The different sensitivities of human oral squamous cell carcinoma and normal cells to GSE suggested that used GSE may be used as a natural chemotherapeutic drug. Many chemotherapeutic agents can trigger the apoptosis of cancer cells. Morphological changes in apoptosis are chromatin condensation and nuclear fragmentation. Apoptotic DNA fragmentation is a key feature of apoptosis, characterized by the activation of endogenous endonucleases with subsequent cleavage of chromatin DNA into internucleosomal fragments of approximately 180-200 base pairs (bp) [51,52]. The results of our study showed induction of apoptosis in the cancer cells as a result of treatment with the GSE. Cell death assay, TUNEL and DNA fragmentation analyses were used to reveal if induction of apoptosis was achieved by GSE. It was shown that DNA fragmentation happened in the late stage of apoptosis. *In vitro* study have shown an anticancer potential of the GSE and selectivity to

inhibit growth and induce apoptosis in KB, unlike HUVEC cells. However, additional experiments should be done to test grape seed extracts effect on other cancer cell lines *in vitro* and *in vivo* (animal studies) conditions to establish their therapeutic ability as well as its adverse effects.The results show that polyphenol-containing extracts inhibited S. mutans. Both red and green grape extracts had higher activity than the other fruits tested, and extracts of grape seed were more active than those from grape skins. The highest activity was P70 extract from red grape seeds. The flavan-3-ol and procyanidin oligomer content of grapes can vary with the variety, degree of maturity and part of the fruit studied. It is known that the degree of con- densation of polyphenols differs in different parts of ber- ries. The skins of red and white grapes contain higher con- centrations of catechin and epicatechin whilst grape seeds contain higher concentrations of oligomericprocyanidins [53]. This is consistent with the results obtained here.

GSE against S.mutans.

Organisms present in biofilms such as dental plaque are also more resistant to antimicrobial agents [54].However, the extracts also inhibited adhesion of S. mutans, suggesting that they inhibited glucosyltransferaseactivity and inhibit glycan formation as has been shown for specific polyphenols from oolong tea [55]. The double action of inhibiting growth and adhesion may help in preventing plaque formation and inhibition of acid production may reduce damage to teeth by pre-formed plaque. The extracts of plants containing high levels of polyphenols inhibited growth of S. mutans and other bacteria. Inhibition of S. mutans occurred in the presence of both sucrose and glucose, two of the main sugar com- ponents used in confectionery and food products and acid production was inhibited. Adhesion of S. mutans was also inhibited, suggesting that glycan synthesis was also inhibited. The extracts may have a role in caries prevention.

GSE and Bleaching

In this study, the grape seed extract (proanthocyanidin) group demonstrated the highest bond strength which makes it a viable alternative to sodium ascorbate. Proanthocyanidins are high molecular weight polymers that comprise the monomeric flavan-3-ol catechin and epicatechin. They are found in natural sources such as grape seed extract, pine bark extract, cranberries, lemon tree bark and hazel nut tree leaves in high concentrations. It has been proven to be safe for use in dietary supplements since it is a naturally occurring plant

metabolite. The reversal in reduction of bond strength by proanthocyanidins has been attributed to their specificity for hydroxyl free radicals, the presence of multiple donor sites that trap superoxide radicals and esterification of epicatechin by gallic acid which enhances the free radical scavenging ability [56,57].ARI scores indicated that the most common site of bond failure was at the bracket/adhesive interface or within the 9 adhesive in all the groups except the immediate bonding group, which showed bond failure closer to the enamel/ adhesive interface and the difference was statistically significant. This was consistent with the lower bond strength values found in this group. The bond failure at the bracket/adhesive interface and within the adhesive seen in the ceramic brackets was similar to that found by Oztas et al. [58] and indicated that risk of enamel damage was reduced. Bleaching with 35% hydrogen peroxide immediately before bonding reduces the bond strength of ceramic brackets. A delay in the bonding procedure by one week reverses the reduction in bond strength. Treating the bleached enamel surface with 10% sodium ascorbate solution or grape seed extract (5% proanthocyanidin solution) also reverses the reduction in bond strength and can be used as an alternative to delayed bonding.

Reactive Active Species Inhibition

Periodontitis is initiated by the sub-gingival biofilm but it's progression is attributed to the presence of reactive oxygen species[59]. Excess of reactive oxygen species release is implicated in the inflammatory process[60]. Thus the antioxidant activity of GSE inhibits ROS which ultimately leads to inhibition of periodontitis[61].

Antibacterial Effect of GSE

GSE used in this work showed antibacterial activity against Campylobacter spp., being the MIC lower than 20 mg/l and the MBC 60 mg/l. Phenolic acids and catechins, were the main responsible of the growth inhibitory effect of GSE. As MIC and MBC values of the GSE are in the range between 10 and 100 mg/l, this extract could be considered as a promising antibacterial agent (62), potentially useful for the control of Campylobacter in foods. Identification and quantification of the individual phenolic compounds of GSE could be useful to standardize the production process to obtain an enriched extract to inhibit Campylobacter growth. The fact that grape seed is sold commercially as dietary supplement and also has Generally Recognized as Safe (GRAS) status approved by Food and Drug Administration

(FDA), could facilitate its potential use in different points of the food chain to fight against this food- borne pathogen.

Anti-Microbial Activity of GSE

In this study the effect of GSE (Vitisvinifera) has been evaluated against two most important bacterial strains in dental pathologies. A. actinomycetemcomitans was first identified as a possible periodontal pathogen in 1975 in localized aggressive periodontitis (LAP) and it's highly association with periodontal disease in adolescents has been established (63). On the other hand S. mutans is the primary causal agent for dental caries specially in the initiation and development stages (64), this microbe was first described in 1924 (65). Grape (Vitisvinifera) seeds are considered rich sources of polyphenolic compounds, mainly monomeric catechin and epicatechin, gallic acid and polymeric and oligomericprocyanidins (66). Grape phenolics are simple molecules, such as hydroquinone, pyrocatechol, caffeic acid, ferulic acid, p-coumaric acid, gallic acid, ellagic acid and resveratrol [10]. Furthermore, GSE is a rich source of diverse bioflavonoids, collectively known as grape seedproanthocyanidins extract (67). Polyphenols are well documented to have microbicidal activities against a huge number of pathogenic bacteria (68,69). The mechanism of polyphenols toxicity against microbes may be related to inhibition of hydrolytic enzymes (proteases and carbohydrolases) or other interactions to inactive microbial adhesions, cell envelope transport proteins and non specific interactions with carbohydrates (68). It means that phenolic antibacterial activity is due to enzyme inhibition by the oxidized compounds possibly through a reaction with sulfhydryl groups or through more non specific interactions with the proteins (68,70), further more flavonoids and tannins can bind or form precipitates with various proteins (71).At all with consideration of inhibitory effect of GSE on A. actinomycetemcomitans, it may have potential for further development as natural agent in prevention of periodontal disease. Further studies with different species of S.mutans and different concentration of GSE are recommended. Grape seed extract as a natural antimicrobial compound derived from Vitisvinifera has inhibition effect against A. actinomycetemcomitans ATCC 43718. There was not any bactericidal or bacteriostatic effect observed against S. mutans PTCC 1683.

Conclusion

Thereby this review article compiles all works done using grape seed extract that is potentially beneficial in application during dental procedures. Hence, this helps other investigators to further develop other methodologies using grape seed extract that is more effective than the present artificial products available that in the long run may affect the oral cavity.

Corresponding Author

Jayashri.P,

Senior Lecturer, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai-600077,Tamil Nadu, India

Mobile:-+91-9841788803

Email id:-shrijaya2009@gmail.com

References

1. Gift HC, Redford M. Oral health and the quality of life. Clin Geriatr Med. 1992;8:673–83

2. U.S. Department of Health and Human Services. Oral health in America: a report of the Surgeon General. Rockville (MD): U.S. Department of Health and Human Services, National Institute of Dental and Craniofacial Research, NIH; 2000.
3. National Institute of Dental and Craniofacial Research. Snack smart for healthy teeth. NIH publication No. 01–1680. Bethesda (MD): NIH; 2001
4. U.S. Department of Health and Human Services. Oral health in America: a report of the Surgeon General. Rockville (MD): U.S. Department of Health and Human Services, National Institute of Dental and Craniofacial Research, NIH; 2000.
5. Beck JD, Slade G, Offenbacher S. Oral disease, cardiovascular disease and systemic inflammation. Periodontol. 2000;23:110–20.
6. Xiong X, Buekens P, Fraser WD, Beck J, Offenbacher S. Periodontal disease and adverse pregnancy outcomes: a systematic review. BJOG. 2006;113:135–43.

7. Sarni-Manchado P, Cheynier V, Moutounet M. Interactions of grape seed tannins with salivary proteins. J Agric Food Chem. 1999; 47:42–7
8. Fujii H, Sun B, Nishioka H, Hirose A, Aruoma OI. Evaluation of the safety and toxicity of the oligomerized polyphenol Oligonol. Food Chem Toxicol. 2007;45:378–87.
9. Yamakoshi J, Saito M, Kataoka S, Kikuchi M. Safety evaluation of proanthocyanidin-rich extract from grape seeds. Food Chem Toxicol. 2002;40:599–607.
10. Bedran-Russo AK, Pereira PN, Duarte WR, Drummond JL, Yamauchi M. Application of crosslinkers to dentin collagen enhances the ultimate tensile strength. J Biomed Mater Res B Appl Biomater. 2007;80:268–72.
11. Duarte S, Gregoire S, Singh AP, Vorsa N, Schaich K, Bowen WH, Koo H. Inhibitory effects of cranberry polyphenols on formation and acidogenicity of Streptococcus mutans biofilms. FEMS Microbiol Lett.2006;257:50–6.
12. Rao CN, Rao VH, Steinmann B. Bioflavonoid-mediated stabilization of collagen in adjuvant-induced arthritis. Scand J Rheumatol. 1983;12:39–42.
13. Cetta G, Pallavicini G, Tenni R, Bisi C. Influence of flavonoid-copper complexes on cross linking in elastin. Ital J Biochem. 1977;26:317–27.
14. Han B, Jaurequi J, Tang BW, Nimni ME. Proanthocyanidin: a natural crosslinking reagent for stabilizing collagen matrices. J Biomed Mater Res A.2003;65:118–24.
15. Xie Q, Bedran-Russo AK, Wu CD. In vitroremineralization effects of grape seed extract on artificial root caries. J Dent. 2008;36:900–6.
16. 1Banting DW.Epidemiology of root caries. Gerodontology. 1986;5:5–11.
17. Y. Liu, V. Dusevich, and Y. Wang. Addition of Grape seed extract renders phosphoric acid a collagen stabilizing etchant. J Dent Res. 2014; 93(8):821- 827
18. Anandadeepan, Karthic Anand, Lakshmi. T. Invitro Anti-arthritic activity of grape seed ethanolic extract. International journal of Pharmacognosy and Phytochemical Research 2015; 7(5):977-979
19. Bedran-Russo AK, Pereira PN, Duarte WR, Drummond JL, Yamauchi M. Application of crosslinkers to dentin collagen enhances the ultimate tensile strength. J Biomed Mater Res B Appl Biomater. 2007;80:268–72.
20. Ceneppele TMF, Jeronymo RD, Di Nicolo R, de Araujo MA and Soares LE. In Vitro Assessment of dentin erosion after immersion in acidic beverages: surface profile

analysis and energy-dispertive X-ray fluorescence spectrometrystudyBraz. Dent. J.2012; 23273-8.

21. Malinauskas BM, Aeby VG, Overton RF, Carpenter-Aeby T and Barber-Heidal K. A survey of Energy Drink Consumption Pattern Among College StudentNutr. J.2007; 635.
22. Meryana E 2011 PenjualanTigaProdukIni Akan TumbuhPesat. 2014
23. Ehlen LA and Marshall TA . Acidic beverages increase the risk of in vitro tooth erosionNutr. Res.2008;(28): 299-303.
24. Mount GJ and Hume WR.Preservation and Restoration of Tooth Structure.(Knowledge Book and Software).2005:21-8.
25. Azarpazhooh A and Limeback H . Clinical efficiacy of casein derivates: a systematic review of literature J. Am. Dent. Assoc.2008:139915-24.
26. Karlinsey RL, Mackay AC, Walker ER and Frederick KE.Preparation, characterization, and in vitro efficiacy of an acid modified beta-TCP Material for dental hard tissue remineralizationActa Biomaterialla.2010:3969-78.
27. Castellan CS, Pereira PNR, Viana G, Chen S, Pauli GF and Bedran-Russo AK. Solubility Study of Phytochemical Cross-Linking Agents on Dentin Stiffness. J. Dent.2010:58451-46.
28. Shapiro, S., Giertsen, E., & Guggenheim, B. An in vitro oral biofilm model for comparing the efficacy of antimicrobial mouth rinses. Caries Research.2002;36:93–100.
29. Guggenheim, B., Giertsen, W., Schupbach, P., & Shapiro, S.Validation of an in vitro biofilm model of supragingival plaque. Journal of Dental Research. 2001;80:363–370.
30. Padilla C, Lobos O, Hubert E, Gonzalez C, Matus S, Pereira M. Periodontal pathogens in atheromatous plaques isolated from patients with chronic periodontitis. J Periodontal Res. 2006;41: 350–353.
31. Park HJ, Jeong SK. Kim SR, Bae SK, Kim WS, Jin SD. Resveratrol inhibits Porphyrombas gingivalis lipopolysaccharide induced endothelial adhesion molecule expression by suppressing NF- kappaB activation. Arch Pharm Res.2009; 32:583-591.
32. Sun Y, Shu R, Li CL, Zhang MZ .Gram negative periodontal bacteria induce the activation of Toll-like receptors 2 and 4, and cytokine production in human periodontal ligament cells. J Periodontal.2010; 81: 1488-1496.

33. Graves D. Cytokines that promote periodontal tissue destruction. J Periodontol. 2008;79: 1585–1591.
34. Herath TD, Darveau RP, Seneviratne CJ, Wang CY, Wang Y, Jin L. Tetra- and penta-acylated lipid A structures of Porphyromonas gingivalis LPS differentially activate TLR4-mediated NF-kappaB signal transduction cascade and immuno-inflammatory response in human gingival fibroblasts.2013.
35. Wei C, Tan CK, Xiaoping H, Junqiang J.Acanthoic acid inhibits LPS-induced inflammatory response in human gingival fibroblasts. Inflammation. 2015;38: 896–901.
36. Kurauchi M, Niwano Y, Shirato M, Kanno T, Nakamura K, Egusa H. Cytoprotective effect of short-term pretreatment with proanthocyanidin on human gingival fibroblasts exposed to harsh environmental conditions. 2014.
37. Kim Y, Choi Y, Ham H, Jeong HS, Lee J. Protective effects of oligomeric and polymeric procyanidin fractions from defatted grape seeds on tert-butyl hydroperoxide-induced oxidative damage in HepG2 cells. Food Chem. 2013;137: 136–141.
38. Kim YS, Zerin T, Song HY. Antioxidant action of ellagic acid ameliorates paraquat-induced A549 cytotoxicity. Biol Pharm Bull.2013; 36: 609–615.
39. Kalaiselvi P, Rajashree K, Bharathi Priya L, Padma VV.Cytoprotective effect of epigallocatechin-3-gallate against deoxynivalenol-induced toxicity through anti-oxidative and anti-inflammatory mechanisms in HT-29 cells. Food Chem Toxicol. 2013;56: 110–118.
40. Campos J, Schmeda-Hirschmann G, Leiva E, Guzman L, Orrego R, Fernandez P. Lemon grass (Cymbopogoncitratus (D.C) Stapf) polyphenols protect human umbilical vein endothelial cell (HUVECs) from oxidative damage induced by high glucose, hydrogen peroxide and oxidised low-density lipoprotein. Food Chem.2014;151: 175–181.
41. Posner MR. Integrating systemic agents into multimodality treatment of locally advanced head and neck cancer. Ann Oncol. 2010;(Suppl 7):246–251.
42. Vissink A, Jansma J, Spigkervet FKL, Spijkervet FK, Burlage FR, Coppes RP. Oral sequelae of head and neck radiotherapy. Crit Rev Oral Biol Med. 2003;14:199–212. Firoozamand LM, Brandao JVP, Fialho MPN. Influence of microhybrid resin and

etching times on bleached enamel for the bonding of ceramic brackets. Braz Oral Res 2013;27(2):142-8.

43. Thorn JJ, Hansen HS, Specht L, Bastholt L. Osteoradionecrosis of the jaws: clinical characteristics and relation to field of irradiation. J Oral Maxillofac Surg. 2000;58(10):1088–1093.
44. Stell PM. Survival times in end-stage head and neck cancer. Eur J Surg Oncol. 1989;15(5):407–410.
45. Vokes EE, Weichselbaum RR, Lippman SM, Hong WK. Head and neck cancer. N Engl J Med. 1993;328:184–194.
46. Argiris A, Brockstein BE, Haraf DJ, Stenson KM, Mittal BB,Kies MS. Competing causes of death and second primary tumors in patients with locoregionally advanced head and neck cancer treated with chemoradiotherapy. Clin Cancer Res. 2004;10:1956–1962.
47. Ghashm A, Othman N, Khattak M, Ismail N, Saini R. Antiproliferative effect of Tualang honey on oral squamous cell carcinoma and osteosarcoma cell lines. BMC Complement Altern Med. 2010;10:49.
48. Hseu YC, Chen CS, Wang SY. Alpiniapricei rhizome extracts induce cell cycle arrest in human squamous carcinoma KB cells and suppress tumor growth in nude mice. Evid Based Complement Alternat Med. 2011:123815.
49. Dinicola S, Cucina A, Pasqualato A, D'Anselmi F, Proietti S, LisiE. Antiproliferative and Apoptotic Effects Triggered by Grape Seed Extract (GSE) versus Epigallocatechin and Procyanidins on Colon Cancer Cell Lines. Int J Mol Sci. 2012;13(1):651–664.
50. Sun Q, Prasad R, Rosenthal E, Katiyar SK. Grape seed proanthocy-anidins inhibit the invasiveness of human HNSCC Cells by targeting targeting EGFR and reversing the epithelial-to-Mesenchymal Transition. PLoS ONE. 2012;7(1):e31093.
51. Johnstone RW, Ruefli AA, Lowe SW. Apoptosis: a link between cancer genetics and chemotherapy. Cell. 2002;108:153–64.
52. Brown JM, Attardi LD. The role of apoptosis in cancer development and treatment response. Nat Rev Cancer. 2005;5:231–7
53. Macheix JJ, Fleuriet A, Billot J: Fruit Phenolics. Boca Raton, CRC Press, 1990.
54. Fux CA, Costerton JW, Stewart P, Stoodley P: Survival strategies of infectious biofilms. Trends Microbiol 2005;13:34–40.

55. Matsumoto M, Hamada S, Ooshima T: Molecu- lar analysis of the inhibitory effects of oolong tea polyphenols on glucan binding domains of recombinant glucosyltransferase from Streptococcus mutans. FEMS Microbiol Lett 2003;228:73–80.
56. Vidhya S, Srinivasulu S, Sujatha M, Mahalaxmi S. Effect of grape seed extract on the bond strength of bleached enamel. Oper Dent. 2011;36(4):433-8. 16.
57. Fine AM. OligomericProanthocyanidin Complexes: History, Structure and Phytopharmaceutical applications. Altern Med Rev 2000;5:144-51. 17.
58. Oztas E, Bagdelen G, Kilicoglu H, Ulukapi H, Aydin I. The effect of enamel bleaching on the shear bond strengths of metal and ceramic brackets. Eur J Orthod 2012;34:232
59. Page, R. C., &Kornman, K. S. The pathogenesis of human periodontitis: An introduction. Periodontology, 2000;14: 9–11
60. Gustafsson, A., &Asman, B. Increased release of free oxygen radicals from peripheral neutrophils in adult periodontitis after Fc delta-receptor stimulation. Journal of Clinical Periodontology.1996; 23:38–44
61. Chapple, I. L., & Matthews, J. B. The role of reactive oxygen and antioxidant species in periodontal tissue destruction. Periodontology, 2000:43:160–232.
62. Ríos, J. L., &Recio, M. C. Medicinal plants and antimicrobial activity. Journal of Ethnopharmacology. 2005;100:80-84.
63. Fine DH, Markowitz K, Furgang D. Aggregatibacteractinomycetemcomitans and its relationship to initiation of localized aggressive periodontitis: Longitudinal cohort study of initially healthy adolescents. J Clin Microbiol 2007; 45(12): 3859-3869.
64. Roeters FJ, van der Hoeven JS, Burgersdijk RC and Schaeken MJ. Lactobacilli, mutants streptococci and dental caries: A longitudinal study in 2-year-old children up to the age of 5 years. Caries Res 1995; 29(4): 272-279.
65. Clarke JK. On the bacterial factor in the etiology of dental caries. Brit J Exp Pathol 1924; 5: 141-147.
66. Monagas M, Gomez-Cordoves C, Bartolome B. Monomeric, oligomeric, and polymeric flavan-3-ol composition of wines and grapes from Vitisvinifera L. Cv. Graciano, Tempranillo, and Cabernet Sauvignon. J Agric Food Chem 2003; 51(22): 6475-6481. 10.

67. Jayaprakasha GK, Selvi T, Sakariah KK. Antibacterial and antioxidant activities of grape (Vitisvinifera) seed extracts. Food Res Int 2003; 36: 117-122.
68. Nakamura Y, Tsuji S, Tonogai Y. Analysis of proanthocyanidins in grape seed extracts, health foods and grape seed oils. J Health Sci 2003; 49(1): 45-54. 12
69. Cowan MM. Plant products as antimicrobial agents. Clin Microbiol Rev 1999; 12(4): 564-582.
70. Karou D, Dicko MH, Simpore J and Traore AS. Antioxidant and antibacterial activities of polyphenols from ethnomedicinal plants of Burkina Faso. Afr J Biotechnol 2005; 4(8): 823-828.
71. Song JH, Kim SK, Chang K. In vitro inhibitory effects of polygonumcuspidatum on bacterial viability and virulence factors of streptococcus mutans and streptococcus sobrinus. Arch Oral Biol 2006; 51(12): 1131-1140.

Dental Crowding In Orthodontics

N.S.Harini[1], Sumathi[2], Sankari M[3]

Graduate Student[1], Reader[2], Department of Orthodontics, Professor[3], Department of Periodontics.

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

The most common cause of dental crowding is the presence of an arch-length--tooth-size discrepancy. Conventional methods of gaining space in orthodontics involve the extraction of teeth, often premolars. However, there are a number of clinical situations in which the extraction of permanent molars might be considered. The main aim of this review article is to know in detail about dental crowding.The objective of this article is to assess dental crowding in all aspects and to know the opinions of different authors.

The present study was to know about dental crowding in detail. Various articles from different authors were collected and summed up as a review article so that dental crowding is knownelaborately in all aspects. Dental crowding is defined as a discrepancy between tooth size and jaw size that results in a mal alignment of the toot. Some reasons for dental crowding could be excessively large teeth, small jaws, and a combination of both. The main causes for dental crowding are Mesio distal width of the teeth is directly related to crowding in the arch. Arch length and arch width is inversely related to crowding. In isolation these parameters may sometimes not show a significant correlation. Together arch length and tooth size are the major cause of crowding in dental arches. Orthodontic treatment of dental irregularity and crowding is achieved by either expanding the dental arches or creating space through tooth extraction or enamel stripping or a combination of any of these alignment with orthodontic appliances.

Key Words: *Dental crowding, primary crowding, secondary crowding, tertiary crowding, relapse, supracrestalfibrotomy.*

Introduction

Dental crowding is defined as a discrepancy between tooth size and jaw size that results in a mal alignment of the toot. Some reasons for dental crowding could be excessively large teeth, small jaws, and a combination of both when teeth become crowded it is due to lack of space for all of the teeth to properly fit. Crowding of teeth occurs when the tooth and jaw size do not have a harmonious relationship. Early or late loss of deciduous teeth and improper tooth eruption can also cause crowding. [(1)]

Dental crowding is distinguished into 3 types:

1) Primary crowding
2) Secondary crowding
3) Tertiary crowding

Primary crowding refers to the crowding caused by the genetically determined size discrepancy between the teeth and the jaw that contains them.

Secondary crowding is the shortage of space that can manifest after extraction of deciduous teeth through migration of neighbouring teeth in to the open space. It also includes crowding caused by deviating functional conditions.

Tertiary crowding refers to irregularities developing in the mandibular and sometimes slightly in the maxillary anterior region during early adulthood and later[(2)].

The accurate assessment of crowding is an essential part of orthodontic diagnosis and subsequent treatment planning and may be affected by a number of factors. The principal factors are the mesio-distal tooth widths and arch length. Other factors can include arch form, arch symmetry, and curves of Spee, the correction of which may require, or sometimes create space and therefore affect the assessment of crowding [(1)].

In an attempt to more accurately quantify the effects of these different factors, a number of formal space analyses have been developed [(2-7)],the most popular probably being the Royal London Space analysis [(8,9)]. The purported benefits of such analyses include consistency in treatment planning and as an aid for trainee orthodontists when assessing space requirements.The

most common cause of dental crowding is the presence of an arch-length--tooth-size discrepancy. Conventional methods of gaining space in orthodontics involve the extraction of teeth, often premolars.

However, there are a number of clinical situations in which the extraction of permanent molars might be considered. One of the greatest writer in Spanish language and one of the world's greatest novelists Miguel de Cervantes Saavedra, said that "Every tooth in a man's head is more valuable than a diamond". The statement is quite true because teeth are the most enriching or most beautifying segment of the face and yearning for teeth, which give in any event the presence of perfection. Any irregularity or imperfection such as dental crowding in the teeth can change one's beauty, which is undesirable. In this regard the orthodontic treatment plays an important role.

Dental crowding, also referred as swarming, can be characterised as an inconsistency between tooth size and arch dimension which results in malocclusion. Dental crowding occurs because of the absence of coordination between the tooth size and arch dimensions and results in positioning the teeth on each other. When there is shortage of space for the arrangement of teeth in the dental arch, teeth experience the ill effects of rotation dislocated eruption etc., [(28)]

Crowded teeth are very difficult to clean properly and thoroughly, which results in poor mouth cleanliness and further dental and medical problems. Mesiodistal crown Diameter (MD) which is also known as tooth size, tooth crown size, or tooth width, in human populations has been used in various studies due to its role in human advancement and biological problems as well in forensic investigations and clinical dentistry.

Various factors are responsible for dental crowding, including the effect of the environmental as well as genetic factors, on dental arch dimensions such as arch width, arch length, arch perimeter etc. Mesiodistal tooth width is additionally influenced by hereditary qualities, race, and sex [(28)]. However, the reasons for overcrowding have not completely been comprehended till now [(29)]. The factors that may contribute to teeth crowding are broad teeth, bony bases and also the developmental pattern towards a decreased facial skeletal size without a relating diminish in tooth size [(30)]. Crowding and spacing can be depicted as a declaration of an adjusted proportion

between tooth size and dental arch dimensions, both of which are affected by inherent and ecological components [31]. The arrangement of teeth inside the upper and lower arch, for the most part, relies on the relationship between tooth size and jaw size. Any irregularity amongst dental parameters, for example, tooths size and arch dimension have been studied by several investigators [32].

The advanced medical imaging like Computed Tomography (CT) scan provides cross-sectional images of internal body structures [33]. A 3D CT can generate the volume data with high-quality images, permits estimation or measurement of structures to be made on a picture helping analysis and diagnosis [34].

Multi-Detector Computed Tomography (MDCT) can give more geometric accuracy than other devices such as cone beam computed tomography and Vernier's calliper [35]. Measurement of orthodontic outcomes only in the sagittal view, as recorded in 2D lateral cephalograms or profile photographs may not be sufficiently informative, but the 3D CT gives better frontal and three-quarter profile data for diagnosis, treatment planning, and measurement purposes [35].

The main aim of this review article is to know in detail about dental crowding.The objective of this article is to assess dental crowding in all aspects and to know the opinions of different authors.

Materials and Methods

The present study was to know about dental crowding in detail. Various articles from different authors were collected and summed up as a review article so that dental crowding is known elaborately in all aspects. The articles were collected by manual searching of the references of the relevant retrieved articles, peer-reviewed orthodontic journals, and gray literature. Search terms included dental crowding, orthodontics, and aesthetics. Non-English articles were excluded from the review in the study-selection stage. Data extraction and evaluation of primary studies were performed independently by 2 reviewers.

Discussion

Crowding of teeth is considered as the most common type of malocclusion. Dental crowding can be defined as a disparity in the relationship between the tooth size and jaw size which result in

the imbrications and rotation of teeth. Tooth size-arch length discrepancy (TSALD) is a well defined means of assessing dental crowding[16]

The face is said to be the most important physical characteristic in the development of self-image and self-esteem, as positive social interactions have been shown to result in better interpersonal relationships and more self-confidence[39,40]. Any significant deviations from the norm may result in feelings of insecurity related to appearance, inhibition in social contacts, and comparison of self with others considered to be 'superior', all of which may negatively affect the quality of life of the individual[41].

Malocclusion is a causative problem in a lot of dental diseases so its documentation will help create awareness among the public [42]. The perception of aesthetics varies from person-to-person and is influenced by personal experiences and social environment [43]. The primary dentition is an important indicator of the permanent dentition. Absence of spaces in the primary dentition indicates that some malocclusion might appear in the future permanent dentition [44]. Third molar eruption is a very important clinical issue because retention of this tooth might be beneficial for orthodontic anchorage, prosthetic abutment or transplantation [45].

Ever since Gregor Johan Mendel proposed the law of inheritance, genetics has transcended the field of health and has entered all walks of life in its application. Thus, the gene is the pivoting factor for all happenings revolving around it [46]. This indicates that gene acts as a significant factor in dental crowding.

Relapse is the loss of any correction achieved by orthodontic treatment. The technique to prevent relapse requires the use of fixed retainers in both arches. It also seems important the use multi stranded wires, coaxial type, made of stainless steel with 0.021mm in diameter due to the excellent stability and surface roughness which allows the wire to hold the elastomeric solid thread that will generate the necessary force to move the teeth. [10].

Nance[11] described dental crowding as the difference between the space needed in the dental arch and the space available in that arch, that is the space discrepancy. Thus, crowding or spacing can be described as an expression of an altered tooth tissue ratio or as a dentoalveolardisproportion. Many investigators have examined the relationship of arch size, and tooth size with crowding. The observations made in these studies are quite conflicting.

Lundstorm[12]and Fastlicht[13]found on one hand, a significant relationship of dental crowding with tooth size, whereas Mills[14] and Howe [15] found a more significant relationship of dental crowding with arch dimensions.

Although, previous studies have compared the tooth size between dental arches with or without crowding in some specific ethnic group, so far no such studies have been done on the population. One of the most common malocclusions facing the orthodontist is the crowding of the teeth before as well as after the completion of orthodontic treatment. The current emphasis on non extraction therapies has result in significant reduction in number of teeth extracted for the orthodontic treatment.

The relationship between dental crowding and different dental parameters such as tooth size and arch dimension has been studied in great detail by investigators such as LundstromA -1951and Mills -1964[17-20]. The finding of this study partially agreed to Fastlischt J -1970 finding showed a significant relationship between crowding and mesiodistal width of incisor. The difference in the mean values collective of mesiodistal tooth between the crowded and the non crowded group was found to be not statistically significant in agreement with Waheed -Ul- Hameed -2005[21-25]

Gilmore CA, Little RM -1984 findings showed that there is a weak correlation between mesiodistal width of incisors and irregular alignment of teeth over long term study but previous authors have suggested that well aligned mandibular incisor are narrower mesiodistally than incisors which are crowded[26,27].

Stability of Crowding

It is also called the “stabilisation” or “retention period”. The orthodontic retention phase also called orthodontic contention is aimed at minimising unwanted dental movements and maintaining the corrections obtained during the initial period following the removal of brackets or other appliances used for correction[20]. Even if the most critical period is the first few months and year following the end of treatment, it is often indicated to stabilise or retain the teeth for a much longer period. Certain practitioners even advocate “lifetime” retention.

Circumferential Supra Crestal Fibrotomy

Supra crestal fiberotomy is the procedure done to prevent relapse of the rotated tooth after the orthodontic procedure. It severes the attachment of supra-alveolar and transseptal fibers around the tooth[36].Reitan, in 1969, showed that the relapse of the rotated tooth will occur 5 hours after removal of the appliance[37]. So, the circumferential supracrestalfiberotomy (CSF) procedure should be carried out toward the end of finishing stage of active orthodontic treatment. The continuous use of CSF in orthodontic forced eruption for crown lengthening prevents the coronal displacement of gingiva and the attachment apparatus. This prevents the additional need for gingival recontouring after orthodontic treatment. Repositioning of the rotated teeth is often easier during orthodontic procedures but poses a problem in retaining its new position in the arch. The reorganization of the periodontal fiber complexes, transseptal fibers, and the supra-alveolar fibers occur after orthodontic tooth movement.

Transseptal fibers stretch elastically during orthodontic treatment and tend to pull the teeth back toward their original position. Campbell, in 1975, stated that the supra-alveolar fibers are non-elastic by nature and more stable with a slower rate of turnover. These fibers do not adapt to new tooth positions and are in part responsible for relapse after the active orthodontic treatment procedures. Transseptal fibers, sharpey's fibers of the newly formed bone may rearrange even after a retention period of 4-6 months[37].

Therefore, the retention period should continue for a 12-month period[38]. The fibrotomy or the pericision in the orthodontic surgical procedure designed to sever the gingival fibres around the tooth. It usually reduces the tendency to relapse of tooth rotations corrected by dental braces or other treatments. The most frequently encountered post orthodontic problem is the retention of re-established tooth position. Relapse (drifting of the tooth back to its position prior to orthodontic correction) may occur anywhere, but it is often associated with teeth that have undergone rotation (twisting) as a part of the orthodontic therapy[21,22].

It involves the detachment of fibres that attach he tooth to the bone via the gum. It acts much like a twisted rubber bands and releasing the tension between the fivers and the tooth and reduces the forces that attempt to pull the tooth back to its original position. It is shown to be effective in

preventing the relapse of the teeth. It is a procedure that is painless as long as the patient takes an analgesic after the numbing has worn off.

Plaque Check

Dental crowding is not just a cosmetic flaw. Dental crowding is sure to give gingival and other periodontal tissue problems. Crowded and misplaced teeth are particularly vulnerable to decay due to constant plaque accumulation. This is a vicious circle where plaque causes tooth decay and inflammation of the gums due to dental crowding[(24)]. This circle can be broken by plaque check and orthodontic treatment which usually involves braces.

Treatment

Orthodontic treatment of dental irregularity and crowding is achieved by either expanding the dental arches or creating space through tooth extraction or enamel stripping or a combination of any of these alignments with orthodontic appliances.

Conclusion

Dental crowding is defined as a discrepancy between tooth size and jaw size that results in a mal alignment of the toot. Some reasons for dental crowding could be excessively large teeth, small jaws, and a combination of both. The main causes for dental crowding are Mesio distal width of the teeth is directly related to crowding in the arch. Arch length and arch width is inversely related to crowding. In isolation these parameters may sometimes not show a significant correlation. Together arch length and tooth size are the major cause of crowding in dental arches. Orthodontic treatment of dental irregularity and crowding is achieved by either expanding the dental arches or creating space through tooth extraction or enamel stripping or a combination of any of these alignments with orthodontic appliances.

Corresponding Author

M.Sankari,

Professor, Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai-600077, Tamil Nadu,India.

Phone Number: +91 9840285905

Email Id: msankari@gmail.com

References

1. Baldridge, D.W.Leveling the curve of Spee: its effect on mandibular arch length. The Journal of Practical Orthodontics, 1969; 3: 26–41.
2. Herren, P., Schmoker, R. and Jordi, T. Arch shape and space balance determined by arcogramme technique. Transactions of the European Orthodontic Society, 1973;4: 61–73.
3. Rudge, S.J. A computer program for the analysis of study models. European Journal of Orthodontics,1982; 4, 269–273.
4. Bhatia, S.N. and Harrison, V.E. (Operational performance of the travelling microscope in the measurement of dental casts. British Journal of Orthodontics, 1987; 14:147–153.
5. Harris, E.F., Vaden, J.L. and Williams, R.A. Lower incisor space analysis: a contrast of methods. American Journal of Orthodontics and Dentofacial Orthopedics, 1987; 92:375–380.
6. Richmond, S. () Recording the dental cast in three dimensions. American Journal of Orthodontics and Dentofacial Orthopedics,1987; 92:199–206.
7. Schirmer, U.R. and Wiltshire, W.A. Manual and computer-aided space analysis: a comparative study. American Journal of Orthodontics and Dentofacial Orthopedics,1997; 112: 676–680.
8. Kirschen, R.H., O'Higgins, E.A. and Lee, R.T. The Royal London Space Planning: an integration of space analysis and treatment planning: part I: assessing the space required to meet treatment objectives. American Journal of Orthodontics and Dentofacial Orthopedics, 2000;118:448–455.

9. Kirschen, R.H., O'Higgins, E.A. and Lee, R.T.The Royal London Space Planning: an integration of space analysis and treatment planning: part II: the effect of other treatment procedures on space. American Journal of Orthodontics and Dentofacial Orthopedics, 2000;118, 456–461.
10. Am J Orthod dentofacial Orthop,2004;125(6):716-25
11. Nance HN; The limitations of orthodontic treatment; Am J Orthod Oral Surg 1947; 33:177-223.
12. Lundstrom.A; The aetiology of crowding of the teeth (based on studies of twins and on morphological investigations) and its bearing on orthodontic treatment. Trans Eur Orthod Soc 1951: 176-89.
13. Fastlicht J. Crowding of mandibular incisors; Am J Orthod 1970; 58:156-63.
14. Mills LF. Arch width, arch length and tooth size in young males. Angle Orthod 1964;34:124-9.
15. Howe RP; An examination of dental crowding and its relationship to tooth size and arch dimension; Am J Orthod 1983; 83:363-73.
16. Warren JJ, Bishara SE, YonezuT;Tooth size —arch length relationships in the deciduous dentition: a comparison between contemporary and historical samples. Am J Orthod Dentofacial Orthop 2003;123;614-9.
17. Norderval K, Wisth PJ, BoeOE;Mandibular anterior crowding in relation to tooth size and craniofacial morphology. Scand J Dent Res. 1975; 83:267–273.
18. Doris JM, Bernard BW, Kuftinec MM, Stom D; A biometric study of tooth size and dental crowding. Am J Orthod. 1981; 79:326–336.
19. Adams CP; A comparison of 15 year old children with excellent occlusion and with crowding of the teeth, Angle Class I malocclusion, in respect of face size and shape and tooth size.Swed Dent J Suppl. 1982; 15:11–26.
20. Smith RJ, Davidson WM, Gipe DP; Incisor shape and incisor crowding: a re-evaluation of the Peck and Peck ratio. Am J Orthod. 1982; 82:231–235.
21. Chang HF, Shiau YY, Chen KC;The relationship of dental crowding to tooth size, dental arch width, and arch depth. Proc .Natl Sci CouncRepub China B. 1986; 10:229–235.

22. Yoshihara T, Matsumoto Y, Suzuki J, Sato N, OguchiH;Effect of serial extraction alone on crowding: relationships between tooth width, arch length, and crowding. Am J Orthod Dentofacial Orthop. 1999; 116:691–696.
23. Gaan SM, Lewis AB, WalengaAJ;Maximum confidence values for the human mesiodistal crown dimension of human teeth. Arch Oral Biol 1968;13:841-4.
24. Seipel CM; Variation of tooth position, a metric study of variations and adaption in the deciduous and permanent dentition; 1982; 82:231–235.
25. Moorrees CFA; The dentition of the growing child; a longitudinal study of dental development between 3 and 18 years of age. Cambridge, Mass:Harvard University Press, 1959:230.
26. Lundstrom A; Inter-maxilla tooth width ratio and tooth alignment and occlusion. Acta Odont Scand 1954;12:265-92.
27. Beresford JS;Tooth size and class distinction. Dent Practit 1969;20:113-2.
28. Sadeghian S, Esnaashari N, ghoreishiN;Relationship of dental crowding with mesiodistal crown diameters and arch dimension; Iranian Journal of Orthodontics. 2014;1:33–38.
29. Hussain SS, Ashraf B, Khan S;Relationship of dental crowding to tooth size and arch dimensions in class I normal & class I malocclusion sample. Pakistan Oral & Dental Journal. 2014;34(4):660–64.
30. Waheed-Ul-Hamid M, RahbarM;Dental crowding and its relationship to tooth size and dimensions. Pakistan Oral & Dental Journal. 2005;25(1):47–52.
31. Abdul-QadirMY;Dental arch and mesiodistal crown dimensions in normal, crowded, and spaced samples. Al – Rafidain Dent Journal. 2011;11(2):211–18.
32. Plavcan JM, Daejling DJ; Interspecific and intraspecific relationships between tooth size and jaw size in primates. Journal of Human Evolution. 2006;51(2):171–84.
33. Kalender WA;X-ray computed tomography. Phys Med Biol. 2006;51:29–43.
34. Markose E, Vikraman B, VeerabahuM;Three-dimensional CT reconstruction: A comparison between 2D, 3D CT and original anatomic structures. Journal of Maxillofac Oral Surg. 2009;8(1):8–12.
35. Bourzgui F; Orthodontics – Basic Aspects and Clinical Considerations. ISBN. 2015;51(7):456 p.

36. Edwards JG; A long-term prospective evaluation of the circumferential supracrestal fi berotomy in alleviating orthodontic relapse. Am J Orthod Dentofacial Orthop 1988;93:380-7.
37. ReitanK;Principles of retention and avoidance of posttreatment relapse. Am J Orthod 1969;55:776-90.
38. Proffit WR, Fields HW. Contemporary Orthodontics..Louis:CV Mosby; 1993;2:530-533.
39. Bos A, Hoogstraten J, Prahl-Andersen B;Expectations of treatment and satisfaction with dentofacial appearance in orthodontic patients.Am J Orthod Dentofacial Orthop 2003;123:127-132.
40. Baron RA; Social thought and social behaviour. In: Essentials of psychology.. Massachusetts: Allyn& Bacon; 1999;2:535-66.
41. Shilpa.R; Self Perceived Orthodontic Treatment Need in Dental Students; Int. J. Pharm. Sci. Rev. Res.,2017; 46(2):39-44.
42. Cinthura, C., Dr. Dhanraj and Dr. Sangeetha;Status of malocclusion among adults in urban areas; International Journal of Information Research and Review ,2017;4(9): 4502-4504.
43. SwethaSridharan, Christine Samantha; Comparison of perception of smile by orthodontists and other specialty dentists: A questionnaire study; International Journal of Orthodontic Rehabilitation; 2018 ; 27(62): 202-216.
44. SamrithiYuvaraj& Dr. Ganesh Jeevanandan; Prevalence of spaced and closed dentition and its relation to malocclusion in primary and permanent dentition among children of south Indian population; International Journal of Multidisciplinary Research and Modern Education; 2017; 3(1):114-119
45. Sivaramakrishnan MS, Ramani P Study on the Prevalence of Eruption Status of Third Molars in South Indian Population. Biol Med (Aligarh),2015; 7: 245.
46. Krishnamurthy Anuthama,Harikrishnan Prasad, Pratibha Ramani, Priya Premkumar, Anuja Natesan, and Herald J. Sherlin; Genetic alterations in syndromes with oral manifestations;Dent Res J (Isfahan). 2013;10(6): 713–722.

Implications of Dental Stem Cells in Life

Swetha.S[1],Deepa Gurunathan[2],Jayanth Kumar.V[3],
Graduate Student[1],Professor[2],Department of Pedodontics,Reader[3], Department of Oral Medicine and Radiology,
Saveetha Dental College, Saveetha Institute Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Stem cells have the potential to self-renew and to give rise to a variety of cell types that ensure tissue repair and regeneration throughout life. Dental pulp stem cells (DPSCs) are having a MSCs phenotype and they are differentiated intoneurone,cardiomyocytes,chondrocytes, osteoblasts and adipocytes. These cells have been isolated from dental and periodontal tissues, characterised, and tested for their potential applications in regenerative dentistry.It is done to educate the dentists and patient regarding the importance of stem cells in regenerative therapy.

Key Words: *Dental stem cells, differentiation, proliferation, scaffold,regeneration*

Introduction

Stem cells are immature, unspecialised cells that have the potential to develop into many different cell lineages via differentiation.There are two primary sources of stem cells:embryonic stem cells and adult stem cells. While there has been a debate on ethical issues surrounding the use of embryonic stem cells , adult stem cells are free of this controversy and only adult stem cells ,to date have been used to treat people(1,2).The most common source of adult stem cell is bone marrow, which contains both hematopoietic stem cells(also found in cord blood) and mesenchymal stem cells(also found in teeth).Bone marrow is a rich source of MSCs, which plays an important role in modern regenerative medicine(3). It is isolated in high number from cultures of bone marrow by selecting the cells that are adhered to tissue culture plastic and proliferates rapidly(4).

It is known that adult stem cells taken from one area of body can be transplanted into another area and grown into a completely different type of tissue. Dental stem cells are being studied for wide range of diseases due to their ability to form connective tissue , neural , bone ,muscle and dental tissues. Dental stem cells are present in various dental tissues including both deciduous and permanent teeth. The various sources of tissue in the oral cavity from where stem cells can be isolated are periodontal ligament, apical papilla, alveolar bone, dental pulp tissue, maxillary sinus, periodontal ligament granulation tissue and gingiva(5).

Regenerative capacity of the dental pulp is well-known and has been recently attributed to function of dental stem cells. Dental stem cells offer a very promising therapeutic approach to restore structural defects.Periodontal ligament- derived stem cells are able to generate periodontal ligament and cementum. Extracted third molars; exfoliating/extracted deciduous teeth; and teeth extracted for orthodontic treatment, trauma or periodontal disease are all sources of dental stem cells from the dental pulp. The dental pulp provides a scaffold of stem cells postnatallywhich is available, with a less invasive process thatmay results in minimal trauma(6,7).

Stem cells from deciduous teeth have been found to grow more rapidly than those from other sources, and it is believed that this is because they may be less mature than other stem cells found in the body(8).Additional advantages of sourcing stem cells from exfoliating deciduous teeth are that the cells are readily available, provided they are stored until they may be needed later in life; the process does not require a patient to sacrifice a tooth to source the stem cells; and there is little or no trauma(9).

Types of Dental Stem Cells

During formation of tooth,interactions between dental papilla and epithelial cells induce tooth morphogenesis by promoting the subpopulation of mesenchymal cells to differentiate into odontoblasts,that results in formation of primary dentin(10,11).Theseodontoblast are believed to come from proliferation and differentiation of earlier population, which is present within pulp tissue.

The various groups of postnatal mesenchymal stem cells have been realised to produced from odontoblast like cells:-

- Dental pulp stem cells (DPSC)
- Stem cells of apical papilla (SCAP)
- Dental follicle progenitor cell (DFPC)
- Stem cells of human exfoliated deciduous teeth (SHED)

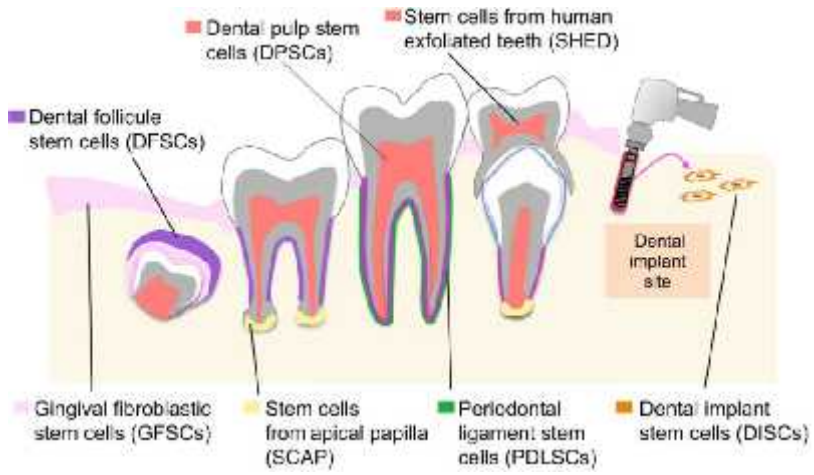


Figure 1: Types of Dental Stem Cells

Dental Pulp Stem Cells

Regenerative capacity of the dental pulp is well-known and has been recently attributed to function of dental stem cells. Dental stem cells offer a very promising therapeutic approach to restore structural defects.Periodontal ligament- derived stem cells are able to generate periodontal ligament and cementum.Extracted third molars; exfoliating/extracted deciduous

teeth; and teeth extracted for orthodontic treatment, trauma or periodontal disease are all sources of dental stem cells from the dental pulp. The dental pulp provides a scaffold of stem cells postnatallywhich is available, with a less invasive process that results in minimal trauma(12).

Johnsen in 1985 proved that DPSCs are capable of producing dental tissues in vivo including dentin , pulp and crown like structures(13).Whereas other investigations has shown that these stem cells can bring about formation of bone liketissues.

Stem Cells of Human Exfoliated Deciduous Teeth

They contain multi-potentstem cells from human exfoliated deciduous teeth(SHED) . They were identified to be a population of highly proliferative, clonogenic cells capable of differentiating into variety of cell types including neural cells, adipocytes and odontoblasts(14).Thus, the exfoliated deciduous teeth is aunique resource for stem cell therapies including autologous stem cell transplantation and tissue engineering.

Stem Cells of Apical Papilla

A unique population of dental stem cells known as stem cells from apical papilla is located at the tip of growing tooth roots. The apical papilla is present only during root formation before the he tooth erupts into oral cavity. They have the ability to differentiate into odontoblasts and adipocytes. They contain larger number of adult stem cells than mature dental pulp. Thus, have a larger potential for regenerating dentin than dental pulp stem cells(15).

Dental Follicle Progenitor Cells

The dental follicle is an ectomesenchymeoriginated from connective tissue sac surrounding the enamel organ and dental papilla of the developing tooth germ before eruption. It is believed to contain precursors or cementoblasts, periodontal ligament and osteoblasts(15).Dental follicle cells form periodontal ligament by differentiating into periodontal ligament fibroblasts that secretes collagen and interact with fibres on the surface of adjacent bone and cementum.

Therapeutic Potential

- **Dental Tissue Repair**

Stem cells have the potency to become an important tool of tissue engineering and regenerative medicine(16).Tissue engineering is utilising the principles of engineering and bioscience aiming to develop biological substances for the restoration, conservation and/or improvement of the tissue function(17). These include obvious uses of cells to repair damaged tooth tissues such as dentin, periodontal ligament and dental pulp. The tissue engineering using enamel and dental stem cell has been recommended as source of cells to facilitate repair of non-dental tissues such as bones and nerves(18).

- **Periodontal Regeneration**

The periodontium is a set of specialised tissues that surround and support to maintain them in the jaw. Periodontitis is an inflammatory disease that affects the periodontium and results in irreversible loss of connective tissue attachment and supporting alveolar bone(18).The challenge for cell based replacement of a functional periodontium is therefore to form a new ligament and bone, and to form ensure appropriate connections are made between these tissues as well as between bone and tooth root(19,20,21).The aim of current research is to use various populations of dental stem cells to replicate events in periodontal tooth development both temporarily and spatially, so that the healing can occur in a sequential manner to regenerate periodontium.

- **Regeneration Of Dental Pulp In Immature Tooth**

Dental pulp needs to be removed when it becomes infected, and this is complicated for root pulp that requires root canal treatment. The restoration of tooth pulp is thus a much sought after goal in dentistrybecause the current practice of replacing infected pulp with inorganic materials results in devitalised tooth. A recent studies have evaluated the de novo regeneration of dental pulp in emptied root canal space using dental stem cells(22,23).

- **Craniofacial Reconstruction**

Stem cells have been used in the tissue engineering of a human-shaped temporomandibular joint. Studies used MSC-derived cells encapsulated in a poly {ethylene glycol} diacrylate hydrogel that was moulded into an adult human mandibular condyle in stratified yet integrated layers of cartilage and bone(24).The osteochondral grafts, in the shape of human TMJs, were implanted in immunodeficient mice for up to 12 weeks. Upon harvest, the tissue-engineered mandibular joint condyles retained their shape and dimensions.Reconstruction of

craniofacial and dental defects using MSC avoids many of the limitations of both auto- and allografting techniques(25,26).Clinical studies are being conducted using stem cells for alveolar ridge augmentation and long-bone defects. Vascularised bone grafts are also used in development using stem cells, and reconstruction of a patient's resected mandible has been carried out using this technique.

- **Use Of Non-Dental Stem Cells**

The method for growing teeth from stem cells obtained in urine were identified. In this study, pluripotent stem cells derived from human urine were induced to generate tooth-like structures in a group of mice with a success rates of up to 30%(27). The generated teeth had features resembling that of normal human teeth except hardness (about one-third less in hardness of human teeth). The advantages were being noninvasive technique, low cost, and use of somatic cells (instead of embryonic) that are wasted anyways(28).The urine-derived stem cells doesnotnotproducetumourswhen it gets transplanted in the body unlike other stem cells; the autologous source of these cells reduces the likelihood of rejection.

- **Uses of Dental Stem Cells in Medicine**

Dental stem cells have the potential to be utilised for medical applications like heart therapies,regenerating brain tissue,for muscular dystrophy therapies and for bone regeneration(28).SHED can be used to generate cartilage as well as adipose tissue.In 2008 first advanced animal study for bone grafting was announced resulting in reconstruction of large size cranial bone defects in rats with human DPSCs(29).

Advantages

- Hematopoietic stem cells save thousands and thousands of patients each and every year. They are patients with cancer and several rare blood diseases.
- Stem cell research provides development in currently untreatable diseases like Parkinson's, Alzheimer's, cancer, osteoarthritis etc(30).
- Most scientists believe that stem cells are the key to one day grow tissues, organs and perhaps limbs. Researchers have already grown and transplanted simple organs, using a combination of stem cells and scaffolds.

- Stem cells have shown some very promising results in people with spinal cord injuries.
- Not all stem cells require the destruction (or killing if you prefer) of a human embryo. Adult and induced pluripotent stem cell lines can be developed using a piece skin or a bone marrow sample.
- Unlike embryonic, adult and induced pluripotent stem cells are autologous which means those that that developed from the patient himself. So, there is no risk of rejection for stem cell based treatments.
- The knowledge gained from stem cells providesus a better understanding of ageing.

Disadvantages

- The use of human embryonic stem cells requires the destruction of laboratory-fertilised human eggs. Many view the blastocysts destroyed during the process as just a mass of cells, while believe that life begins at conception and thus consider the whole process to be immoral and unacceptable.
- The embryonic and induced pluripotent stem cells have a potency to form tumours. The adult stem cells don't seem to have this problem, due to their limited differentiation nature.
- We must wait for years, perhaps decades before seeing widespread clinical application of stem cell therapies, with the exception of course hematopoietic stem cell transplantation.
- Having an experimental, none FDA approved treatment is dangerous and may prove to be deadly. This of course applies to many of the experimental, not FDA approved stem cell therapies provided all over the world by clinics and individual physician.

Conclusion

Stem cell-based therapies represent the most promising alternative for successful regeneration of damaged or pathological dental tissues or even the entire tooth following tooth loss. It is therefore essential to understand the mechanisms underlying the potential of the various dental stem cell populations as well as their behaviour after transplantation in ectopic sites.

Innervation and vascularisation play fundamental roles in the regulation of stem cell niches homeostasis, thus affecting the fate and behaviour of stem cells. Stem cell-based approaches are beginning to emerge in dentistry, and huge challenges and problems such the enamel and entire tooth regeneration should be overcome. Nevertheless, stem cell-based regenerative approaches are the future of dentistry that will benefit millions of patients worldwide.

Corresponding Author

Jayanth Kumar.V,

Reader, Department of Oral Medicine and Radiology,

Saveetha Dental College,Saveetha Institute Medical and Technical Sciences,

Saveetha University,Chennai, India

162,Poonamallee High Road, Chennai 600077,Tamil Nadu, India

Email Id:doctorjayanth@gmail.com

Mobile Number: +91 8056135807

Reference

1. Mao JJ. Stem cell and future of dental care. NY State Dent J. 2008;74:20–4.
2. Nedel F, André Dde A, de Oliveira IO, Cordeiro MM, Casagrande L, Tarquinio SB, Stem cells: Therapeutic potential in dentistry. J Contemp Dent Pract. 2009;10:90–6.
3. Saravana Kumar Sampath Kumar, SaraswathiPerumal, and VijayaraghavanRajagopalan, "Therapeutic effect of bone marrow mesenchymal stem cells on cold stress induced changes in the hippocampus of rats" Neural Regen Res. 2014; 9(19): 1740–1744.
4. Prockop DJ. Marrow stromal cells as stem cells for nonhematopoietic tissues. Science. 1997;276:71–74.
5. KavarthapuAvinash, Sankari Malaippanand Jayakumar Nadathur Dooraisamy, "Methods of Isolation and Characterization of Stem Cells from Different Regions of Oral Cavity Using Markers: A Systematic Review" Int J Stem Cells. 2017; 10(1): 12–20.
6. Pompilio G, Cannata A, Peccaori F, Bertolini F, Nascimbene A, Capogrossi MC, Autologous peripheral blood stem cell transplantation for myocardial regeneration. A novel strategy for cell collection and surgical injection. Ann Thorax Surg. 2004;78:1808–12.

7. De Ugarte DA, Morizona K, Elbarbary A, Alfonso Z, Zuk PA, Comparison of multi-lineage cells from human adipose tissue and bone marrow. Cells Tissue Organ. 2003;174:101–9.
8. Laughlin MJ. Umbilical cord blood for allogenic transplantation in children and adults. Bone Marrow Transplant. 2001;27:1–6.
9. Gang EJ, Jeong JA, Hong SH, Hwang SH, Kim SW, Yang IH,Skeletal myogenic differentiation of mesenchymal stem cells isolated from human umbilical cord. Stem cells. 2004;22:617–24.
10. Miura M, Gronthos S, Zhao M, Lu B, Fisher LW, Robey PG, Shi S. SHED: Stem cells from human exfoliated deciduous teeth. Proc Natl Acad Sci USA. 2003;100:5807–12.
11. KimS,HonmouO,KatoK,NonakaT,HoukinK,Neuraldiorientationpotentialofperipheral blood- and bone-marrow-derived precursor cells. Brain Res. 2006;1123:27–33.
12. NosratIV,WidenfalkJ,OlsonL,NosratCA.Dental pulp cells produce neurotrophicfactors ,interact with trigeminal neurons in vitro, and rescue motoneurons after spinal cord injury. Dev Biol. 2001;238:120–132.
13. D. C. Johnsen, “Innervation of teeth: qualitative, quantitative, and developmental assessment,” Journal of Dental Research, 1985;84: 555–563,
14. D. Ricketts, “Management of the deep carious lesion and the vital pulp dentine complex,” British Dental Journal,.2001;191(11):606–610,.
15. P. D. Potdar and Y. D. Jethmalani, “Human dental pulp stem cells: applications in future regenerative medicine,” World Journal of Stem Cells,2015;7(5): 839–851.
16. S.Lymphani, "Dental Stem Cells and their Applications in Dental Tissue Engineering" Open Dent J. 2013; 7: 76–81.
17. J. J. Mao, P. G. Robey, and D. J. Prockop, “Stem cells in the face: tooth regeneration and beyond,” Cell Stem Cell, 2012; 11(3) 291–301.
18. Zuk PA, Zhu M, Ashjian P, De Ugarte DA, Huang JI, Mizuno H, Alfonso ZC, Fraser JK, Benhaim P, Hedrick MH. Human adipose tissue is a source of multipotent stem cells. Mol Biol Cell. 2002;13:4279–4295.
19. Gronthos S, Mankani M, Brahim J, Robey PG, Shi S. Postnatal human dental pulp stem cells (DPSCs) in vitro and in vivo. Proc Natl Acad Sci USA. 2000;97:13625–13630.

20. Beltrão-Braga PC, Pignatari GC, Maiorka PC, Oliveira NA, Lizier NF, Wenceslau CV, Miglino MA, Muotri AR, Kerkis I. Feeder-free derivation of induced pluripotent stem cells from human immature dental pulp stem cells. Cell Transplant. 2011;20:1707–1719.
21. Verma K, Bains R, Bains VK, Rawtiya M, Loomba K, Srivastava SC. Therapeutic potential of dental pulp stem cells in regenerative medicine: An overview. Dent Res J (Isfahan) 2014;11:302–308.
22. Kadar K, Kiraly M, Porcsalmy B, Molnar B, Racz GZ, Blazsek J, Kallo K, Szabo EL, Gera I, Gerber G, et al. Differentiation potential of stem cells from human dental origin - promise for tissue engineering. J Physiol Pharmacol. 2009;60(7):167–175.
23. Shoi K, Aoki K, Ohya K, Takagi Y, Shimokawa H. Characterisation of pulp and follicle stem cells from impacted supernumerary maxillary incisors. Pediatr Dent. 2014;36:79–84.
24. Tziafas D, Kodonas K. Differentiation potential of dental papilla, dental pulp, and apical papilla progenitor cells. J Endod. 2010;36:781–789.
25. Lei M, Li K, Li B, Gao LN, Chen FM, Jin Y. Mesenchymal stem cell characteristics of dental pulp and periodontal ligament stem cells after in vivo transplantation. Biomaterials. 2014;35:6332–6343.
26. Yamada Y, Ito K, Nakamura S, Ueda M, Nagasaka T. Promising cell-based therapy for bone regeneration using stem cells from deciduous teeth, dental pulp, and bone marrow. Cell Transplant. 2011;20:1003–1013.
27. Choi YA, Seol MY, Shin HI, Park EK. Bobby Sox homology regulates odontoblast differentiation of human dental pulp stem cells/progenitors. Cell Common Signal. 2014;12:35.
28. Nakashima M, Mizunuma K, Murakami T, Akamine A. Induction of dental pulp stem cell differentiation into odontoblasts by electroporation-mediated gene delivery of growth/differentiation factor 11 (Gdf11) Gene Ther. 2002;9:814–818.
29. Racz GZ, Kadar K, Foldes A, Kallo K, Perczel-Kovach K, Keremi B, Nagy A, Varga G. Immunomodulatory and potential therapeutic role of mesenchymal stem cells in periodontitis. J Physiol Pharmacol. 2014;65:327–339.

Development and Growth of Maxillary Sinus

Neeraj Surathu[1], Saravana Kumar[2], S.Niveda[3]

Graduate student[1], Senior Lecturer[2], Department of Anatomy,

Tutor[3], Department of Public health dentistry

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

Abstract

Para nasal sinuses comprises of four paired air-filled spaces which surrounds the nasal cavity, maxillary sinuses, above the eyes, frontal sinuses, between the eyes, ethmoidal sinuses, and behind the ethmoids, sphenoid sinuses. The sinuses are named based on the front facial bones in which they are located. The maxillary sinus is pyramid shaped and is the largest of the four paranasal sinuses. The article talks about the development of maxillary sinus and the dental surgeries that it is involved in.

Key Words: *Maxillary sinus, maxillary sinus grafting, maxillary sinusitis*

Introduction

The maxillary sinus was described firstand properly shown by Leonardo da Vinci, but the earliest significant discovery was by Nathaniel Highmore, the British surgeon and anatomist who had discussed the maxillary sinus in detail in 1651. It is found to be present during birth as minor air spaces and develops throughout life.

The paranasal sinuses develop within the bones of the viscerocranium. The maxillary sinus is the first paranasal sinus to form [1]. The development of the maxillary sinus has been documented as early as the 17th week of the prenatal period [2], however it is after birth that the majority of growth occurs. In new born children the viscerocranium is relatively undeveloped. It grows simultaneously with other bones and reaches 25% of its final size by the end of the 2 years and up to the 50% by the end of the 8th year of age. It was reported, that the growth of the viscerocranium is most extensive in the vertical direction, slower in the anterior andposterior direction, and slowest in the horizontal direction [3]. As the paranasal sinuses are mostly enclosed within the bones of the viscerocranium, the dynamics of the development of the skull should also refer to their growth. Interestingly, Scuderi et al[1].

stated, that at birth, the rudimentary aerated maxillary sinus is 6–8 cm^3 in volume with its maximal dimension in the anterior andposterior direction. Bone expansion during the formation of the paranasal sinuses although continuous, is not uniform and can take place at different times and at different speeds within the developmental process. Therefore, the shape and size of the paranasal sinuses are probably the most variable of all the anatomical structures of the body. Due to extended aeration, the walls of the paranasal sinuses may turn into thin bony laminae. Undoubtedly the shape and size of the maxillary sinus is determined but not limited by the boundaries of surrounding bony structures. A close link between the size of the facial skeleton and the maxillary sinus volume is suggested, even when severe facial pathologies are present [3]. As the shape and size of the maxillary sinus reflect the development of bony structures, it may be associated with determining the shape of the middle face, moreover, it is suggested that the maxillary sinus might play a crucial role in the formation of facial contours [4]. Despite many proposed theories, the real function of the maxillary sinuses has not been clarified [5]. The most often believed is that the sinuses support the respiratory function and resonation, but, it cannot be excluded, that they arose as an aid to the skull structure or are residual remnants with yet unknown purpose [6]. According to Kawarai et al, [7] the fact, that the paranasal sinuses constitute the major cavity of the skull indicates thatthe particularity ofthe sinus is their air space. Nonetheless,the aeration of the sinuses must play an important role if the maxillary sinus is already filled with air in newborns [8,9]. Therefore, it is suggested that the volume of the air cavity is the simplest and most important index for sinus evaluation

The maxillary sinus is a variable anatomical structure as show in a study done on the 3D reconstruction and simulation of the maxillary sinus showed that its morphology and size are variable. Also, the developmental pattern of the paranasal sinuses varies widely according to the individual and the age [11].

Anatomical and developmental descriptions of the maxillary sinus may be of great clinical importance. The pneumatization of children’s sinuses and the pattern of normal development may serve as a reference for evaluating normal or abnormal development of the maxillary sinus. Normal values and clinical indexes can be used in diagnostic, preoperative evaluation, and treatment planning. More studies on the dimensional changes in the maxillary sinus during growth and volume of the normal maxillary sinus may help in radiological interpretation of the status of the maxillary sinus and its possible pathologies.

Sinus means “pocket” in Latin. Human beings have airfilled spaces that are found surrounding the nasal region known as the paranasal sinuses. These paranasal sinuses are paired and they are four in numberincluding the front sinus, ethmoidal sinus, sphenoidal sinus and the maxillary sinus. The largest of the four paranasal sinuses is the maxillary sinus. Although insufficient literature is available some studies show that the roles of these sinuses include decreasing the relative weight of skull, increasing voice resonance, providing a buffer against blows to face, insulating structures, and humidifying/heating inhaled air[12].

The membrane that lines the walls of the maxillary sinus is called Schneiderian membrane. It is multi-layered and 0.13 mm to 0.5 mm in thickness. The condition of the maxillary sinus is self-maintainingby postural drainage and with the help of the ciliated epithelial lining, which places the bacteria towards that of the ostium. This ostium is described as a non-physiologic drainage port high on the medial wall that opens into the nasal cavity between the middle and lower nasal conchae (hiatus semilunaris).

Sinus lifting procedures may help in the improvement of the symptoms of sinusitis and congestion by relocation of the sinus lining closer to the drainage port, and this does not affect the normal physiological function of the maxillary sinus [12].

Traditionally, the maxillary sinus has been an area that has been avoided in most dental procedures. In the past general practitioners and oral maxillofacial surgeons have avoided entering the maxillary sinus from the oral cavity unless they considered it to be absolutely necessary[13]. The maxillary sinus is pyramidal in shape. The base of the pyramid is the medial wall of the sinus that is also the lateral wall of the nasal cavity, and its apex is pointed toward the zygomatic bone. The roof of the sinus is also the floor of the orbit. The average volume of a sinus is about 15 ml (range between 4.5 and 35.2 ml). The maxillary sinus does not fluctuate in its size and maintains itself while the posterior teeth remain in contact as the size increases with age. This is a process commonly known as pneumatisationand is usually occurring due to atrophy caused from reduced occlusal function[14]. The pneumatisation of this sinus is variable and ranging from agenesis to hyperpneumatization.

Until the age of 8 the maxillary sinus grows 2 mm a year vertically and 3 mm a year in an anteriorposterior direction. There are spurts of maxillary sinus growth from birth to 2 years, from 7 and a half to 10 years, and from 10 to 12 years of age. There is slower steady growth

until 14 to 18 years of age. From 7 months to birth, the sinus averages 7 mm to 16 mm anteriorposterior and 2 to 13 mm superiorinferior and 1 mm to 7 mm [15].

The blood supply to the maxillary sinus is derived primarily from the maxillary artery. The posterior superior alveolar and infraorbital arteries anastomose in the bony lateral wall. On average, the anastomoses intraosseously arises mostly in 100% of the cases and usually from a height of 19mm from the crest of the alveolar ride, however anastomoses extraosseously arises mostly in about 40% of the cases and this is seen to be at a height of 23 mm from the crest of the alveolar ridge. More than one septa can be involved in division of the maxillary sinuses, known as Underwood's septa [16,18].It is observed in about 30% of cases and most dominant in anterior region (commonly seen in between the 2^{nd}premolar and 1^{st}molar) observed in 77% of the cases. The mean height of the septae is 7.9 mm (range 0 to 17 mm). [16,18.]

Contents

Development

The maxillary sinuses are the only pair of sinuses of the paranasal sinuses which are visible during the time of birth. During the time of birth they have the size of a small lima bean measuring about 8x4 mm, and are situated with their longer dimension directed anteriorly and posteriorly.They develop at the third month of intrauterine life, in the place existing between the oral cavity and the floor of the orbit.They develop as evagination of the mucous membrane of the lateral wall of the nasal cavity at the level of the middle nasal meatus forming a minute space that enlargesin the inferior directioninto the maxillary sinus region [15].The maxillary sinus enlarges variably and greatly by pneumatisation (the formation of air cavities in tissues) until it reaches the adult size by the eruption of the permanent teeth [15].Growth of the sinus slows down with decline of facial growth during puberty but continues throughout life.

Function of Maxillary Sinus

- Lightening the weight of the skull.
- Resonance of voice.

- Regulations of the Olfactory and respiratory modulations through the air pressure within the sinus during respiration.
- Inspired air conditioning.
- Craniofacial protection against mechanical trauma.
- Bactericidal enzyme production (lysozyme) has the significance in protection against bacterial infection of the nasal mucosa.

Anatomy of Maxillary Sinus

There are not only variations in size, shape and position among different individuals, but also there is difference in different sides of the same individual.which is pyramidal in shape that has a base, an apex and four walls.

- The base: lateral wall of the nasal cavity.
- The apex is directed laterally towards the zygomatic process of the maxilla.
- The four walls:

  Anterior wall: Frontal surface of the maxilla.

Posterior wall: Infratemporal surface of the maxilla.

Roof: Floor of the orbit.

Floor: Alveolar process of the maxilla.

- Maxillary sinus is seen to have a direct communication with the middle meatus and this commonly described as ostiummaxillaewhich is about 36 mm in its diameter.
- The sinus has septa that partially divide it into intercommunicating compartments with separate ostia.
- The average capacity of the maxillary sinus is about 15 ml. and its average dimensions in centimetres are transversely 2.3cm, anteroposteriorly 3.4cm, vertically 3.35cm.

Development Anomalies of Maxillary Sinus

Agenesis (imperfect development or complete absence), Aplasia and Hypoplasia (altered development or under development) of the sinus occurs either alone or in association with other anomalies like:

- Chronal atresia
- Cleft palate
- Septal deformity
- Absence of concha
- Malformation of external nose

- Maxillary sinus hypoplasia is the name given by dentists to define the underdevelopment of the maxillary sinus. It can be detected radiologically and can be related to maxillary sinusitis.
- Maxillary agenesis refers to the complete absence of the maxillary sinus surrounding the nasal cavities.
- Maxillary aplasia is the failure in the development of the maxillary sinus.

Occurrence of any of these three scenarios could lead to the anomalies mentioned above.

Maxillary Sinusitis

Sinusitis is a common disease seen to affect approximately 35 million people per year in the region of North America. An approximate amount of two to three billion dollars is paid for health treatment per year. Nevertheless, maxillary sinus infections are on the most missed out diagnosis seen in clinical practice and it is a real problem. The dental practitioner must be fully aware of the possible symptoms and representation of maxillary sinusitis in order to diagnose it correctly. In dental practice sinusitis of both acute and chronic type may represent itself as a chronic orofacial pain or atypical odontalgia and will be in need for proper medical care. Odontogenic origin for sinusitis is generally a well-recognizedwith an incidence of around ten percent. These cases of sinusitis of odontogenic originwhich appearsthat are usually resistant to conventional treatment methods should be prescribed proper medications

and referral. To ensure thatproper care for a patient presenting with maxillary sinusitis the dental practitioner must be well versed in the knowledge of maxillary sinusitis.

Maxillary sinusitis is inflammation of the maxillary sinuses. Themost commonly observed signs and symptoms of maxillary sinusitis are mild headache, usually near the region of the maxillary sinus and foul-smelling discharge, possibly with some systemic signs of infection such as fever and weakness. The skin over the involved sinus can be tender, hot, and even reddened due to the inflammatory process in the area. On radiographs, there is opacification (or cloudiness) of the usually translucent sinus due to retained mucus[19].

Maxillary sinusitis is common due to the close anatomic relation of the frontal sinus, anterior ethmoidal sinus and the maxillary teeth, allowing for easy spread of infection.The maxillary sinus may lead into the mouth through an abnormal opening, an oroantral fistula[14,15].

Microbiology of Maxillary Sinusitis

In health the paranasal sinuses were initially thought to be sterile. This is highly controversial as many studies have documented the presence of normal flora in these air-filled cavities[20]. Most cases of sinusitis are thought to be of bacterial etiology. The microbiology of sinusitis of both acute and chronic type is usually different can vary depending upon which sinus is involved. In sinusitis of odontogenic origin, it isoften the polymicrobial typewhich can be a mixedaerobic/anaerobic type in nature.

In sinusitis of acute type, Streptococcus pneumonia and Haemophilus influenzae have been identified as primary etiologic agents. A large number of organisms, including coagulase positive and negative Staphylococci and Escherichia coli have found to cause about 5% of maxillary sinusitis cases.

In sinusitis of chronic type, the etiologicorganisms can vary from type to type, with anaerobic organisms isolated generally with an increased frequency. Studies show that it has been reported that 88% of culture positive cases contained anaerobes, and 32% were part of a mixed infection [20]. Of the anaerobes approximately 50% exhibited beta lactamase activity. The predominant organisms include Bacteroides species, anaerobic cocci, Pepto streptococcus anacrobius, P. niger and Clostridium species.

In sinusitis caused by odontogenic origin, most were considered to be mixed aerobicand anaerobic infections with the most common organisms including anaerobic streptococci, Bacteroides, Proteus and coliform bacilli[21,22].Patients with cystic fibrosis and immotile cilia syndrome are predisposed to Pseudomonas aeruginosa and S. aureus infections.

Sinusitis Treatment

Traditionally the treatment of acute maxillary sinusitis is usually prescription of a broad-spectrum cephalosporin antibiotic resistant to betalactamase, administered for 10 days.

There areadvantages in the treatments that primarily focus on the underlying issue and probably inflict damage to the nasal membrane causing inflammation, instead of the secondary bacterial infection that has been the primary target of past treatments for the disease. Direct removal of mucus that is loaded with toxins from the inflammatory cells is the surgical procedure for the treatment of sinus with chronic infections. Leaving the mucus behind might predispose early recurrence of the chronic sinus infection.

Maxillary Sinus Floor Life Augmentation

Sinus lift procedure is a surgery increases the bone height in the upper jaw in the area of the teeth present within the maxillary sinus region. It is commonly also known as sinus augmentation. The bone is formulated between the jaw and the maxillary sinuses that is present on the either side of your nose. The sinus membrane has to be moved upward or lifted inorder to create room for the bone[23,24]. Maxillary sinus has great potential for healing and bone formation and indicates that bone grafts or bone substitutes may not be needed to achieve augmentation of the maxillary sinus floor[15].

A sinus lift usually is done by a specialist. This could be either an oral and maxillofacial surgeon or a periodontist. A sinus lift procedure is carried out when there is not enough bone height in the upper jaw, or the sinuses are too close to the jaw, for dental implants to be placed[23].

The gum tissue is raised, exposing the bone. A small oval window is opened in the bone. The sinus and the jaw are separated by a membrane lining the sinus on the other side of the window.This membrane is gently pushed up and away from your jaw[24].

Surgical Methods for Enhancement of the Alveolar Bone Height in the Posterior Maxilla

Maxillary Sinus Floor Augmentation Applying the Lateral Window Technique with a Grafting Material and Simultaneous or Delayed Implant Installation

Surgical intervention is one of the most commonlyemployed method to increases the vertical height of the crest of the alveolar ridge before or during implant placement to ensure success of the implant supported prosthesis and many cases have been reported and documented such as reviews and meta-analysis[20-25].

Surgical Technique

One of the methods to carry out maxillary sinus augmentation is the lateral window techniqueand it is usually performed under local anaesthesia or conscious sedation. Through the oral mucosa, access is gained to the maxillary sinus usually in the region of the anterior as well as the lateral wall of the maxillary sinus. A mid crestal incision is made along with vertical cuts in the anterior and posterior direction. Once this is done,a mucoperiosteal flap with a base shaped in a trapezoidal pattern is elevated allowing access to the lateral wall of the maxillary sinus[23]. An osteotomy cut is then performed on the lateral wall of the maxillary sinus with the use of burs and a high rpm handpiece avoiding any injury to the Schneiderian membrane. The osteotomy window and the Schneiderian membrane is cautiously improved from the floor of the maxillary sinus in addition to the lateral and medial sinus wall and displaced dorsocranially with blunt dissector to create a compartment for placement of the graft fabric. Implants are inserted simultaneously with the augmentation system if the height of the residual alveolar bone gives enough number one implant balance. An implant mattress is successively organized with burrs before the implant are inserted inside the residual alveolar bone with the implant tip exposed in the created compartment within the maxillary sinus. The graft cloth is densely packed across the exposed implant floor to facilitate de novo bone formation. The lateral window to the maxillary sinus is commonly protected by using a resorbable collagen membrane to prevent ingrowth of fibrous tissue earlierthan the mucoperiosteal is readapted and sutured. Once the stability is confirmed the procedure is carried out one year after the augmentation and the prosthetic replacement is initiated 3-6 months after the successful placement of the implant [23-26].

Surgical Complications

Perforation of the Schneiderian membrane is one of the most common surgical complication that can occur during the procedure of sinus floor augmentation.Presence of septa a residual crestal bone height of less than 3.5 mm increases the risk for a sinus membrane perforation. However, perforation of the Schneiderian membrane seems not to influence the final treatment outcome, but a higher prevalence for sinusitis is reported in cases of membrane perforation. Other complications include bleeding, migration of dental implants into the maxillary sinus, postoperative infection, sinusitis, exposure of the graft, graft loss, oedema, seroma formation, benign paroxysmal positional vertigo and exposure of the collagen membrane. Smokers generally exhibited greater chances for complications and a newly published systematic review concluded that smoking seems to be associated with increased risk of wound dehiscence and infection after maxillary sinus floor augmentation.

Conclusion

The dental practitioner must be well aware of the literature on the anatomy of the maxillary sinus in order to perform sinus procedures and successfully diagnose and treat maxillary sinusitis.

Corresponding Author

Niveda S,

Tutor, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077, Tamil Nadu, India

Email: nive6393@gmail.com

Telephone number: 9952979464

References

1. Scuderi AJ, Harnsberger HR, Boyer RS. Pneumatization of the paranasal sinuses: normal features of importance to the accurate interpretation of CT scans and MR images. AJR. American journal of roentgenology. 1993 May;160(5):11014.
2. Farah G, Nafis AF. Morphometric analysis of developing maxillary sinuses in human foetuses. Int J Morphol. 2006;24(3):3038.
3. Kulczyk T, Przystańska A, Rewekant A, TurskaMalińska R, CzajkaJakubowska A. Maxillary sinuses and midface in patients with cleidocranial dysostosis. Annals of AnatomyAnatomischer Anzeiger. 2018 Jan 1; 215:7882.
4. Song SY, Hong JW, Roh TS, Kim YO, Kim DW, Park BY. Volume and distances of the maxillary sinus in craniofacial deformities with midfacial hypoplasia. Otolaryngology Head and Neck Surgery. 2009 Nov;141(5):61420.
5. Jun BC, Song SW, Park CS, Lee DH, Cho KJ, Cho JH. The analysis of maxillary sinus aeration according to aging process; volume assessment by 3-dimensional reconstruction by highresolutional CT scanning. Otolaryngology—Head and Neck Surgery. 2005 Mar;132(3):42934.
6. Keir J. Why do we have paranasal sinuses? The Journal of Laryngology & Otology. 2009 Jan;123(1):48.
7. Kawarai, Kkunihiro Fukushima, Teruhiro Ogawa, Kazunori Nishizaki, Mehmet Gunduz, Masaaki Fujimoto, Yu Masuda Y. Volume quantification of healthy paranasal cavity by three-dimensional CT imaging. Acta OtoLaryngologica. 1999 Jan 1;119(540):459.
8. Barghouth G, Prior J, Lepori D, Duvoisin B, Schnyder P, Gudinchet F. Paranasal sinuses in children: size evaluation of maxillary, sphenoid, and frontal sinuses by magnetic resonance imaging and proposal of volume index percentile curves. European radiology. 2002 Jun 1;12(6):14518.
9. Weiglein A, Anderhuber W, Wolf G. Radiologic anatomy of the paranasal sinuses in the child. Surgical and radiologic anatomy. 1992 Dec 1;14(4):3359.
10. SahlstrandJohnson P, Jannert M, Strömbeck A, AbulKasim K. Computed tomography measurements of different dimensions of maxillary and frontal sinuses. BMC medical imaging. 2011 Dec;11(1):8.
11. Kim HJ, Yoon HR, Kim KD, Kang MK, Kwak HH, Park HD, Han SH, Park CS. Personalcomputerbased three-dimensional reconstruction and simulation of maxillary sinus. Surgical and Radiologic Anatomy. 2002 Dec 1;24(6):3928.

12. Lindhe J, Lang N, Karring T. Ch. 50: Elevation of the maxillary sinus floor. Clinical Periodontology and Implant Dentistry, Fifth Edition 2008.
13. Meyers GS. The Sinus Bone Graft. Journal of Prosthodontics. 2007 Sep 1;16(5):409-10.
14. Solar P, Geyerhofer U, Traxler H, Windisch A, Ulm C, Watzek G. Blood supply to the maxillary sinus relevant to sinus floor elevation procedures. Clinical Oral Implants Research. 1999 Feb 1;10(1):3444.
15. Rontal M, Zinreich SJ. Anatomy of the paranasal sinuses. Thieme Medical Publishers; 1996.
16. Underwood AS. An inquiry into the anatomy and pathology of the maxillary sinus. Journal of anatomy and physiology. 1910 Jul;44(Pt 4):354.
17. Ulm CW, Solar P, Gselimann B, Matejka M, Watzek G. The edentulous maxillary alveolar process in the region of the maxillary sinus—a study of physical dimension. International journal of oral and maxillofacial surgery. 1995 Aug 1;24(4):27982.
18. Kim MJ, Jung UW, Kim CS, Kim KD, Choi SH, Kim CK, Cho KS. Maxillary sinus septa: prevalence, height, location, and morphology. A reformatted computed tomography scan analysis. Journal of periodontology. 2006 May;77(5):9038.
19. Nair UP, Nair MK. Maxillary sinusitis of odontogenic origin: conebeam volumetric computerized tomography–aided diagnosis. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics. 2010 Dec 1;110(6): e537.
20. Brook I. Bacteriologic features of chronic sinusitis in children. Jama. 1981 Aug 28;246(9):9679.
21. Laurier C, Lachaine J, Ducharme M. Economic evaluation of antibacterials in the treatment of acute sinusitis. Pharmacoeconomics. 1999 Jan 1;15(1):97113.
22. Topazian RG, Goldberg MH, Hupp JR. Oral and maxillofacial infections. Elsevier Health Sciences; 2002.
23. Agnihotri A, Agnihotri D. Maxillary sinus lift up: an indirect approach for implant placement in posterior maxilla. International Journal of Oral Implantology and Clinical Research. 2012 May 1;3(2):1014.
24. Zorzano LA, Tojo MJ, Urízar JM. Maxillary sinus lift with intraoral autologous bone and B–Tricalcium Phosphate: histological and histomorphometric clinical study. Medicina oral, patología oral y cirugía bucal. Ed. inglesa. 2007;12(7):12.

25. Dr.D. Shiva kumar*, Dr.N.D. Jayakumar, Dr.G.Karthikeyan, Dr.M.Sankari, Dr. Sheeja.S.Varghese. Saveetha Dental College and Hospitals, Maxillary sinus augmentation without any graft material. A case Report, JPR, 2013,1(8),798 802
26. Vidhya G, Nesappan T. A piezoelectric surgery for direct sinus lift with immediate implant placement. Journal of Dental Implants. 2016 Jul 1;6(2):79.

Developmental Enamel Defects - A Review

Tamizh Paavai.Tha [1], Nithya Jaganathan[2], Iffat Nasim [3]
Graduate student [1], Reader [2],Department of Oral Pathology
Reader [3],Department of Conservative Dentistry and Endodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Enamel defects occur primarily as developmental defects due to systemic problems that occur during pre and postnatal periods. The extent of the defect shows the passage of early developmental events or stresses. Early detection of these conditions during pregnancy stage helps in preventing the deleterious defects of the disease. A Pedodontists should be better informed as the affected children will visit them at an early age due to defect present and the severe morbidity can lead to improper dentition at an early age, also the complexity of the disease will affect the treatment plan for that individual.

Key Words: *Amelogenesisimperfecta, Developmental,Enamel defect, Hypoplasia, Primary, Pathogenesis*

Introduction

Amelogenesis or enamel formation is a programmed genetic mechanism, which can be affected due to environmental disturbances. The developmental defects once established cannot be repaired. Enamel formation occurs through the pre and postnatal life of a child and thus act as a storehouse of the environmental insults received during this process [1] [2]. This helps us detect the timing of the systemic insult [3]. These defects can be classified based on the type of dentition affected, the causative agent or the histopathology of the defect [4].

Pre term birth is associated with Hypoplasia of the primary dentition. Primary tooth Hypoplasia leads to various other deformities like increased caries risk, esthetic deformities and accelerated attrition of teeth, which results in loss of vertical dimension. The aim of this study is to educate the clinicians about this defect, which will help in better diagnosis and treatment of the individual [5].

Etio-pathogenesis

Various systemic and local factors are attributed for the developmental defects of enamel. Systemic factors include nutrition, hypoglycemia, hypocalcaemia, metabolic disorders, respiratory distress, sepsis and neurological disorders. The non-specific appearance of the defect makes it difficult to point the etiologic factor [6-9]. The local factors include injury to the taste buds; shorter period of breast feeding, early bottle-feeding and the general factors are socioeconomic status, low weight and height for the age [10][11].Neonatal lines are used as reference to estimate approximate timing and position of insult [12].

Clinical Features

The clinical presentation of the defect can vary from simple demarcated opacity to complete absence of enamel tissue. The primary feature of this defect is that they are multiple, symmetric and chronological in nature [13] [14]. The clinical manifestation can vary with type of etiologic agent, duration, calcification level and the metabolic activity of the individual [15].

The enamel defects also help the clinician to assess the presence of any other systemic disorder present in the patient such as brain injury [16], intelligence quotient, neurologic deficits [17] and renal disease [18].

The dental problems encountered due to this defect include poor esthetics, malocclusion and increased risk of dental caries. The presence of enamel Hypoplasia in primary teeth is also observed to be a predisposing factor for the occurrence of the defect in permanent dentition [19] [20]. The increased caries incidence can be due to more porosity in hypo calcified teeth, increased biofilm adherence that leads to higher number of Streptococcus mutans [21][22].

Treatment

The extent and severity of the lesions decide the ease of treatment required. The treatment aims at maintaining healthy teeth in oral cavity until the permanent teeth erupt. For esthetic corrections, adhesive restorations, polycarbide crown, strip crown and stainless steel crown are used. The unrestorable teeth are extracted and space maintainers are placed [23-26].

Amelogenesis Imperfecta

Amelogenesis imperfect is a developmental disorder that is genomic in origin, which produces biochemical and morphologic changes in the tooth structure. It can be expressed as autosomal recessive, autosomal dominant, sex linked, and sporadic cases or associated with another syndrome [27].

Classification and Etiology

Initially, they were classified as hypo plastic and hypo calcified types but with time they have been periodically classified under various other categories such as their appearance, mode of inheritance, genome and their biochemistry. However, the inheritance pattern is considered to be a more useful classification than the other factors considered [28] [29].

The formation of enamel is a very complex and organized process that occurs by the interaction of various organic molecules such as enamelin, amelogenin, ameloblastin, tuftelin, amelotin, dentin sialophosphoprotein and enzymes such as kallikrein 4 and matrix metalloprotease 20 [27]. The pattern of inheritance, which influences the phenotype of the disease, is seen in [Table 1].

Table 1: Type of AI Caused by Various Mutations

Genomic Site	Type Of Disease
AMELX, AIH I	X-Linked disease [30]
AIH3	X-Linked disease [31]
AIH2, ENAM 4q21	Autosomal dominant and recessive disease [32]
AMELOTIN 4q13	Can cause any type of disease [33]

Many authors under different categories have classified the condition. Some of these classifications are given below [27].**Weinmann et al, in 1954** Two types based on phenotype: hypo plastic and hypo calcified**Wit kop, 1988** Four major categories based on phenotype (hypo plastic, hypo maturation, hypo calcified, hypomaturation-hypoplastic with taurodontism) subdivided into 15 subtypes by phenotype and secondarily by mode of inheritance

Type l. Hypo plastic
Type lAHypo plastic, pitted autosomal dominant
Type lB Hypo plastic, local autosomal dominant
Type lCHypo plastic, pitted autosomal recessive
Type lDHypo plastic, smooth autosomal dominant
Type lEHypo plastic, smooth X-linked dominant
Type lFHypo plastic, rough autosomal dominant
Type lG Enamel agenesis, autosomal recessive
Type ll. Hypo maturation
Type llA Hypo maturation, pigmented autosomal recessive
Type llBHypo maturation, X-linked recessive
Type llCHypo maturation, snow- capped teeth, X-linked
Type llDHypo maturation, snow- capped teeth, autosomal dominant?
Type lllA Autosomal dominant
Type lllB Autosomal recessive
Type lV Hypomaturation-hypoplastic with taurodontism
Type lVA Hypomaturation-hypoplastic with taurodontism, autosomal dominant
Type lVB Hypomaturation-hypoplastic with taurodontism, autosomal recessive

Hart et al, 2002 Proposed a molecular defect sub classification of the AMELX condition

1.1 Genomic DNA sequence

1.2 cDNA sequence

1.3 Amino acid sequence

1.4 Nucleotide and amino-acid sequence

1.5 AMELX mutations described to date

Aldred et al, 2003

Classification based on:

Mode of inheritance

Phenotype- Clinical and Radiographic

Molecular defect (when known)

Biochemical result (when known)

X-Linked forms of Amelogenesis Imperfecta

This is also known as Amelogenesis Imperfecta 1or hypo plastic type. It is observed that heterozygous females pass on the mutant gene to children of both sexes with the risk of 50%. However, the affected males and females express the condition differently. Males exhibit only a thin layer of enamel of normal color and translucency; it can also be normal thickness enamel but have poor mineralization which gives rise to loss of translucency and a yellow-brown discoloration. Some families also exhibit a combination of Hypoplasia and abnormal mineralization occurring together. The affected individual may also exhibit sensitivity to thermal and osmotic stimuli along with the predominant phenotype of Hypoplasia. In case of affected females, they show vertical markings on the enamel due to X chromosome inactivation, which is also known as Lysonisation. Therefore they are observed to have teeth with vertical ridges or grooves due to Hypoplasia or also exhibit vertical bands of alternating normal and discolored enamel [30].

Genetic mapping have suggested that the X-linked disease to Xp22 region of the short arm of the X chromosome and these subsequently result in mutation of AMELX gene [30].

Amelogenin gene (OMOM*300391)

This gene codes for the amelogenin protein. It has seven exons that span over nine kilo bases. The mutations reported on this gene include deletions on some parts of the gene, single base mutations and premature stop codons as well. They play a critical role in controlling the enamel thickness and enamel mineralization. The studies conducted show no evidences of pre-mutations in this gene. Also there are no evidences of mutation in the amelogenin gene AMELY (Yp11) on the Y chromosome or their role in enamel formation. But it has been suggested that the Y site contributed to the tooth size [30].

Autosomal forms of Amelogenesis Imperfecta

The positional cloning done in some families suggest that the autosomal dominant Amelogenesis Imperfecta maps in 4q11-q21. The genes involved include albumin ALB, ameloblastin AMBN and enamelin ENAM. These genes map the same region and are considered as candidate genes for autosomal dominant Amelogenesis Imperfecta. Another protein named Tuftelin, which maps the 1 q21, has also been named as a cause for other cases of autosomal Amelogenesis Imperfecta. Studies have also shown that genes that code for enamel proteases, which are necessary to degrade enamel proteins, will also cause Amelogenesis Imperfecta [27].

Autosomal Dominant Amelogenesis Imperfecta

This form of the disease is observed to affect one or more individuals in each generation of the family. The clinical signs may be consistent for each affected member or may vary from person to person, even if the affected individuals may belong to the same family.

This type of Amelogenesis Imperfecta is predominantly hypo plastic in some families and can be characterized by thin enamel and spacing between teeth. Other individuals may also present with rough, irregular or randomly pitted enamel. The affected may present with hard enamel, which are normally translucent and not affected by significant attrition if the primary defect is in the production of adequate amount of enamel matrix. It is also observed that some families have an exclusive or predominant hypomineralised phenotype of the disease. For ease of classification, these defects have been named as demineralization to explain the spectrum of defects of mineralization in Amelogenesis Imperfecta. The most severe form of

demineralization is observed to have “cheesy” enamel that is easily lost which leads to reduction in tooth size and increased sensitivity [27].

Some cases show a combination of both enamel Hypoplasia and demineralization which is expressed as teeth with reduced size ab initio that have lesser enamel, small crowns with spacing and can also exhibit yellowish hue due to mineralization defect [27].

ENAM 4q21

The enamelin gene (ENAM) is mapped to chromosome 4q to the same region that is mapped for Amelogenesis Imperfecta 2. A study has reported an extensive family where the causative gene was mapped to 4q11-q21. Another study performed in Japan suggested that the extended family had a similar phenotype wherein the condition was linked to a single G-deletion. Another study performed by Mardh et al, traced a local, hypo plastic form of the autosomal dominant inherited Amelogenesis Imperfecta that was caused due to a nonsense mutation in the enamelin gene. This resulted in the localized Hypoplasia in contrast to a generalized form that occurs due to a “splice-site” mutation [34]. Another study by Kim et al, helps to relate phenotype to genotype and helps in identification of a mid-crown, horizontal form of Hypoplasia [35].

Chromosome 8q24.3

This chromosome has been found as an additional locus for autosomal dominant Amelogenesis Imperfecta [36].

Autosomal Recessive Amelogenesis Imperfecta

This form of the disease is most often encountered in ethnic or family groups where intermarriage between relatives is more common such as Amelogenesis Imperfecta that is caused due to cone rod dystrophy, which is a syndromic condition. Autosomal recessive Amelogenesis Imperfecta is also prevalent in cases there is a high frequency of mutant gene in the population as seen in some of the Polynesian communities [27].

ENAM 4q21

These genes exhibit Amelogenesis Imperfecta, which is hypo plastic in type and is also observed to show open bite malocclusion with hypo plastic enamel, which can be seen as localized enamel pitting in certain cases. Studies suggested that in

homozygous individuals the ENAM mutation in chromosome 4 showed enamel pitting and an anterior open bite while the heterozygous individuals presented with only enamel pitting. This variation has been attributed to “dose dependency” wherein generalized hypo plastic Amelogenesis Imperfecta is a recessive trait and localized enamel pitting is seen as a dominant trait [29].

Other studies done in the same center evaluated nine Jordanian families affected with autosomal recessive Amelogenesis Imperfecta on the basis of clinical, familial, radiographic, and microscopic and protein levels. This resulted in finding a wide spectrum of phenotypes, which supported the earlier inheritance pattern suggested but also stated four different sub-types of autosomal recessive Amelogenesis Imperfecta [37]. The gene KLK4 that maps chromosome 19q13.4 has been associated with the formation of autosomal recessive hypo maturation Amelogenesis Imperfecta [29].

MMP-20

The mutation in the region of 11q22.3-q23 of the matrix metalloproteinase 20 gene has been identified to cause autosomal recessive pigmented hypo maturation Amelogenesis Imperfecta [29].

Sporadic Cases of AmelogenesisImperfecta

The sporadic cases of Amelogenesis Imperfecta can be caused due to a number of reasons. They can be representatives of autosomal recessive Amelogenesis Imperfecta, may be caused due to a new mutation or they may be an example of variable expression with or without incomplete penetrance of a dominant gene. This aspect emphasizes on the fact that proper clinical examination of other family members is important to avoid wrongly classified Amelogenesis Imperfecta case. Also, the tooth abnormality may be caused by non-genetic reasons such as fluorosis and tetracycline staining. When studying other genetic disorders we can observe the passage of new mutations as a autosomal dominant trait but this has not been conclusively proven in the case of Amelogenesis Imperfecta.

In the recent years, a condition known as Molar-Incisor Hypomineralisation (MIH) has been identified. The etiological condition of this disease is unknown but is observed to affect one or more first permanent molars and the anterior teeth in an

unrelated pattern. However, this condition is not yet classified under Amelogenesis Imperfecta yet.

Association of AmelogenesisImperfecta with Syndromes

Earlier the strict definition of Amelogenesis Imperfecta suggested it is an enamel defect that does not include other structures. But such narrow restrictions cannot be applied to a condition that varies in development and progression among different families. Therefore, it has been suggested that Amelogenesis Imperfecta is a genetic condition that may occur in combination with other syndromes or as a separate entity. It has also been explained that the condition may occur due to a single gene defect or from a microsomal or chromosomal defect. The association of Amelogenesis Imperfecta with cone-rod dystrophy is an example [27].

AmelogenesisImperfecta as a Syndrome

In about 50% of the cased affected with this condition, either an X-linked or autosomal inheritance, an anterior open bite is present. This association may be considered as a syndrome but no such mention has been made in any classification yet, it is still a topic to be elucidated.

The phenotypic classifications of Amelogenesis Imperfecta have included taurodontism as an intrinsic feature in one of the variants- AI with taurodontism (AIT). This classification is reasonable as taurodontism is an ectodermal trait. The Hertwig's epithelial root sheath (HERS), which maps the shape of the tooth and root is derived from the enamel organ and helps I n the differentiation of inner enamel epithelium cells into ameloblasts that later form the enamel.

AmelogenesisImperfecta in Syndromes

Recently there have been similarities found between AIT and the tricho-dento-osseous (TDO) defect that includes additional features such as bony defects like bone sclerosis and hair feature of "curly hair". The hair changes and the enamel defect are explained as ectodermal changes but the bony defects occur by a different pathway, which is primarily of mesoderm origin. It is observed that TDO occurs due to a mutation in DLX3 gene. One study reported that Amelogenesis Imperfecta and TDO are genetically distinct, while another study that was conducted later suggests that

TDO and Amelogenesis Imperfecta hypoplastic-hypomaturation with taurodontism (AIHHT) are allelic for DLX3 [27].

There ismany other AI like changes which are associated with other whole body findings but are not included in the diagnosis of Amelogenesis Imperfecta. If it is to be accepted that Amelogenesis Imperfecta can occur as a insolated trait as well as in association with other syndromes, ten many conditions have to be considered in the differential diagnosis of enamel defects. Some syndromes to present with enamel defects include Kohlschutter syndrome, Platypondyly with Amelogenesis Imperfecta, Amelogenesis Imperfecta and nephrocalcinosis, cone-rod dystrophy and Amelogenesis Imperfecta.

The correlation between hypo plastic Amelogenesis Imperfecta and nephrocalcinosis emphasizes on renal examination in patients diagnosed with Amelogenesis Imperfecta [38].

Enamel Defects other than AmelogenesisImperfecta in Syndromes

The meticulous differential diagnosis of causes of the enamel defect in a patient is necessary for therapeutic, professional and patient personal reasons. Many alternative causes can be attributed to an enamel defect. For example, regional odontoplasia is not a primary differential diagnosis in a case of a localized defect of central incisor, which is coupled with the non-eruption of an adjacent tooth. This case can be mainly diagnosed to have occurred due to a childhood trauma that had affected the eruption of the unerupted tooth. Many families are affected by such conditions and thus it is necessary for careful diagnosis, mainly in relation to inheritance of such defects [27].

Clinical Description

Amelogenesis imperfect affects the enamel of all teeth irrespective of inheritance pattern and chronology of the teeth and at occasions, it can be associated with syndromes as well. When an individual is affected with this condition, the enamel can be hypo plastic, hypomineralised or a combination of both. The affected teeth are observed to be discolored, sensitive and prone to disintegration either in pre-eruptive period or in the post – eruptive period [27]. It either occurs due to a single defect or due to micro deletion and chromosomal defect as seen in the case of Cone-red

dystrophy associated with Amelogenesis imperfect, that shows linkage of 2q11[39][40].

Diagnostic Methods

Clinically obtaining a family history, clinical observation, preparing a pedigree plotting is essential to diagnose the condition like any other inherited disease. Radiographic examination of these individuals may help us detect unerupted and resorbing teeth present. In combination with the clinical observation, these records help us identify the extent of enamel Hypoplasia that has occurred in the patient. However, laboratory genetic diagnosis is still under research [27].

Differential Diagnosis

The extrinsic disorders of tooth formation, chronological disorders of tooth formation and localized disorders of tooth formation are primary conditions to be considered during differential diagnosis.

The most common differential diagnosis includes dental fluorosis. This condition may vary from mild white flecking of the enamel to dense areas of discoloration with randomized staining and Hypoplasia. Fluorosis may present with horizontal banding that occurs due to intense fluoride intake that may have spared the premolars and second permanent molars to be spread, a chronological distribution is observed. The latter case can be mainly due to reasons such as local water supply or habits like eating toothpaste in childhood.

Similar findings can also be observed can be due to various causes at the time of tooth formation such as gastrointestinal upset of a prolonged nature as seen in celiac disease and anti- leukemic therapy. These conditions can be found from the history and the chronological distributions of the markings seen. [27]

Treatment

The treatment planning for individuals affected is more radical and stats at a very early age. It is observed that the disease has a profound effect on the emotional status of the patient as many cases have been reported where the patient becomes withdrawn, reclusive and even threatens suicide due to their deformed teeth [41]. Patients are known to have covered their teeth with pieces of paper, chewing gum or other materials to mimic a normal appearance of their teeth. Many people affected

with Amelogenesis Imperfecta have been observed to request to remove their teeth and the fitting of dentures in a society where one's own teeth are the preferred option. The treatment process is described to go through a temporary phase to reach a transitory phase. The primary dentition is protected by the use of metal crowns in the posterior teeth and, composite restorations and polycarbonate crowns are used in the anterior teeth [42]. In case of permanent dentition, if the patient has less hypomineralised and more hypo plastic teeth then usage of preformed crows to maintain the oral hygiene is a viable option. The malocclusion that is also observed to occur in these patients must be corrected by the combined efforts of the clinician and an orthodontist [43].

Conclusion

Due to advancement in pre and postnatal care of premature children, the reported cases of developmental enamel defects are on the rise. Therefore it is necessary that the clinician is well informed about the initiation and progression of the condition to systematically treat the same.

Corresponding Author

Iffat Nasim

Reader,Department of Conservative Dentistry and Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

E-Mail:nasimiffat@gmail.com

Telephone number: +9940063567

References

1. Lunardelli SE, Peres MA. Prevalence and distribution of developmental enamel defects in the primary dentition of preschool children. Braz Oral Res 2005; 19:144–9.
2. Levine RS, Turner EP, Dobbing J. Deciduous teeth contain histories of developmental disturbances. Early Hum Dev. 1979; 3(2):211-20
3. Asl Aminabadi N, Ghettos Oskouei S, Pouralibaba F, Jamali Z, Pakdel F. J

Dent Res Dent Clin Dent Prospects. 2009; 3(4):110-6.

4. Murray JJ, Shaw L. Classification and prevalence of enamel opacities in the human deciduous and permanent dentitions. Arch Oral Biol. 1979; 24(1):7-13.
5. Jayam C, Bandlapalli A, Mishra G, Puttashamachari Y. Developmental Enamel Defects of Primary Teeth: A Review. Global Journal of Medical Research. 2014,4;1:43-6
6. Seow WK. Oral complications of premature birth. Aust Dent J 1986; 31:23-9.
7. Jorgenson RJ, Yost C. Etiology of enamel dysplasia. J Pedod. 1982; 6(4):315-29.
8. Takaoka LA, Goulart AL, Kopelman BI, Weiler RM. Enamel defects in the complete primary dentition of children born at term and preterm.Pediatr Dent. 201; 33(2):171-6.
9. Franco KM, Line SR, de Moura-Ribeiro MV. Prenatal and neonatal variables associated with enamel Hypoplasia in deciduous teeth in low birth weight preterm infants. J Appl Oral Sci; 15(6):518-23
10. Vignarajah S, Williams GA. Prevalence of dental caries and enamel defects in the primary dentition of Antiguan pre-school children aged 3-4 years including an assessment of their habits. Community Dent Health. 1992; 9(4):349-60.
11. Li Y, Navia JM, Bian JY. Caries experience in deciduous dentition of rural Chinese children 3-5 years old in relation to the presence or absence of enamel Hypoplasia. Caries Res. 1996; 30(1):8-15.
12. Levine RS, Turner EP, Dobbing J. Deciduous teeth contain histories of development disturbances. Early Hum Dev 1979; 3:211–20.
13. Sabel N, Klingberg G, Dietz W, Nietzsche S, NorÃ©n JG. .Polarized light and scanning electron microscopic investigation of enamel Hypoplasia in primary teeth. Int J Paediatr Dent. 2010 Jan; 20(1):31-6.
14. Suckling GW. Developmental defects of enamel-- historical and present-day

perspectives of their pathogenesis. Adv Dent Res. 1989; 3(2):87-94.

15. Zheng S, Deng H, Bao Y. The study on the clinical manifestation of developmental enamel defects in primary dentition. Hua Xi Kou Qiang Yi Xue Za Zhi. 2003; 21(3):200-1, 204.

16. Herman, S.C., 1961. Enamel hypoplasia in cerebral palsied children (Doctoral dissertation).
17. Cohen HJ, Diner H. the significance of dental enamel developmental defects in neurological diagnosis. Paediatrics 1970; 46: 737-747.
18. Oliver WJ, Owings CL. Hypo plastic enamel associated with nephrotic syndrome. Paediatrics 1963; 32: 399-406.

19. Elfrink ME, ten Cate JM, Jaddoe VW, Hofman A, Moll HA, Veerkamp JS. Deciduous molar hypomineralization and molar incisor hypomineralization.J Dent Res. 2012 Jun; 91(6):551- 5.

20. Casanova-Rosado AJ, Medina-Solís CE, Casanova- Rosado JF, Vallejos-Sánchez AA, Martinez-Mier EA, Loyola-Rodriguez JP, Islas-Marquez AJ, Maupomé G. Association between developmental enamel defects in the primary and permanent dentitions. Eur J Paediatr Dent. 2011 Sep; 12(3):155-8.

21. Zheng S, Deng H, Gao X: Studies on developmental enamel defects in the primary dentition of children with histories of low birth weight and prematurity and their susceptibility to dental caries. Zhonghua Kou Qiang Yi Xue Za Zhi 1998; 33: 270–272.
22. Seow WK, Young W, Tsang AK, Daley T. A study of primary dental enamel from preterm and full -term children using light and scanning electron microscopy. Pediatr Dent 2005; 27:374-9.
23. Seale NS. The use of stainless steel crowns. Pediatr Dent. 2002; 24(5):501-5.
24. Kind élan SA, Day P, Nichol R, Wilmot N, Fayle SA. UK National Clinical Guidelines in Paediatric Dentistry: stainless steel preformed crowns for primary molars. Int J Paediatr Dent. 2008 18(1):8.
25. Mahoney EK. The treatment of localised hypo plastic and hypomineralised

defects in first permanent molars. N Z Dent J. 2001; 97(429):101-5.

26. Ramya Chellammal, M & Dharman, Sreedevi. (2016). Assessment of enamel defects in patients visiting Saveetha dental college, Chennai: A pilot study. 7. 995-1001.
27. Crawford PJ, Aldred M, Bloch-Zupan A. Amelogenesis Imperfecta. Orphaned journal of rare diseases. 2007 Apr 4; 2(1):17.
28. Aldred MJ, Crawford PJM: Amelogenesis Imperfecta – towards a new classification. Oral Diseases 1995, 1:2-5.
29. Hart PS, Hart TC, Simmer JP, and Wright JT: A nomenclature for X- linked AmelogenesisImperfecta. Arch Oral Biol 2002, 47:255-260
30. Gibson CW, Yuan ZA, Hall B, Longenecker G, Chen E, Thyagarajan T, Sreenath T, Wright JT, Decker S, Piddington R, Harrison G, and Kulkarni AB: Amelogenin-deficient mice display an amelogen- esis Imperfecta phenotype. J Biol Chem 2001, 276:31871-31875.
31. Aldred MJ, Crawford PJ, Roberts E, Gillespie CM, Thomas NS, Fenton I, Sandkuijl LA, Harper PS: Genetic heterogeneity in X-linked AmelogenesisImperfecta. Genomics 1992, 14:567-573.
32. Hart TC, Hart PS, Gory MC, Michael MD, Ryu OH, Uygur C, Ozdemir D, Firatli S, Aren G, Firatli E: Novel ENAM mutation responsible for autosomal recessive Amelogenesis imper fecta and localised enamel defects. J Med Genet 2003, 40:900-906
33. Santos MC, Hart PS, Ramaswami M, Kanno CM, Hart TC, Line SR: Exclusion of known gene for enamel development in two Brazilian families with AmelogenesisImperfecta. Head Face Med 2007, 3:8.
34. Mardh CK, Backman B, Holmgren G, Hu JC, and Simmer JP, Forsman- Semb K: A nonsense mutation in the enamelin gene causes local hypo plastic autosomal dominant Amelogenesis imper- fecta (AIH2). Hum Mol Genet 2002, 11:1069-1074.
35. Kim JW, Seymen F, Lin BP, Kiziltan B, Gencay K, Simmer JP, Hu JC: ENAM mutations in autosomal-dominant AmelogenesisImperfectaJ Dent Res 2005, 84:278-282.

36. Mendoza G, Pemberton TJ, Lee K, Scarel-Caminaga R, Mehrian-Shai R, Gonzalez-Quevedo C, Ninis V, Hartiala J, Allayee H, Snead ML, Leal SM, Line SR, Patel PI: A new locus for autosomal dominant AmelogenesisImperfecta on chromosome 8q24.3. Hum Genet 2007, 120:653-662.

37. Nusier M, Yassin O, Hart TC, Samimi A, Wright JT: Phenotypic diversity and revision of the nomenclature for autosomal recessive AmelogenesisImperfecta. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004, 97:220-230.

38. Hunter L, Addy LD, and Knox J, Drage N: Is AmelogenesisImperfecta an indication for renal examination? Int J Paediatr Dent 2007, 17:62-65.

39. Jalili IK, Smith NJD: A progressive cone-rod dystrophy and AmelogenesisImperfecta: a new syndrome. J Med Genet 1988, 25:738-740.

40. Downey LM, Keen TJ, Jalili IK, McHale J, Aldred MJ, Robertson SP, Mighell A, Fayle S, Kissinger B, Ingle earn CF: Identification of a locus on chromosome 2q11 at which recessive Amelogenesis Imperfecta and cone-rod dystrophy co segregate. Eur J Hum Genet 2002, 10:865-869.

41. Aldred MJ, Crawford PJM, and Savarirayan R, Savulescu J: It's only teeth – is there a limit to genetic testing? Clinical Genet 2003, 63:333-339.

42. Bouvier D, Duprez JP, Bois D: Rehabilitation of young patients with AmelogenesisImperfecta: a report of two cases. ASDC J Dent Child 1996, 63:443-447.

43. Neelakantan P, John S, Subbarao CV. Restoring function and esthetics in a pair of twins with amelogenesis Imperfecta. The Journal of Indian Prosthodontic Society. 2009 Apr 1; 9(2):105.

Diabetes and Cardiovascular Diseases – A Review

Savitha P [1], Naufal Rizwan [2], S.Pradeep [3]

Graduate Student [1],Senior Lecturer [2],Department of General Medicine

Senior Lecturer [3],Department of Conservative Dentistry and Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Cardiovascular diseases are a group of disorders that involve the heart and/or blood vessels. CVD are a spectrum of diseases that includes coronary heart disease, hypertensive heart disease, rheumatic heart disease, cardiomyopathy and cardiac arrhythmias etc. Cardiovascular involvement in diabetes is a common occurrence and in fact, CVD is the leading cause of early death among people with diabetes—about 65 percent of people with diabetes die from heart disease and stroke. High blood glucose levels damage nerves and blood vessels, leading to complications such as heart disease and stroke, the leading causes of death among people with diabetes. Uncontrolled diabetes can eventually lead to other health problems as well, such as vision loss, kidney failure, and amputations.

Key Words: *Glucose levels, Stroke, Health, Kidney, Uncontrolled diabetes*

Introduction

Cardiovascular disease is a class of diseases that occur in heart or blood vessels. CVD includes angina, myocardial infarction, hypertensive heart disease, rheumatic heart disease, cardiomyopathy, atrial fibrillation, endocarditis and venous thrombosis. CVD is a major complication of diabetes and the leading cause of early death among people with diabetes—about 65 percent of people with diabetes die from heart disease and stroke [1]. High blood glucose levels damage nerves and blood vessels, leading to complications such as heart disease and stroke, the leading causes of death among people with diabetes. Uncontrolled diabetes can eventually lead to other health problems as well, such as vision loss, kidney failure, and limb amputations. Cardiovascular diseases (CVD), comprising coronary heart (CHD) and cerebrovascular diseases, are currently the leading cause of death globally, accounting for 21.9 per cent of total deaths, and are projected to increase to 26.3 per cent by 2030 [2]. The number of people with diabetes mellitus is alarmingly increasing due to the growing prevalence of obesity, genetic susceptibility, urbanization, and aging [3].

Diabetes mellitus is a chronic disease which has been described as a state of raised blood glucose associated with premature mortality. Diabetes is an important chronic disease which incidence is globally increasing and though considered as an epidemic [4]. It arises when the pancreas fails to produce enough insulin (type 1 diabetes), or when the body cannot effectively make use of the insulin produced (type 2 diabetes). Failure of insulin secretion, action or both leads to raised blood glucose and other metabolic changes which, if uncontrolled, can cause serious complications [5]. The most important of these are retinopathy (affecting the eyes), nephropathy (affecting the kidneys), neuropathy (affecting the nerves) and cardiovascular disease (affecting the circulatory system) [6]. Diabetes is fast becoming a world pandemic. Although there is no evidence that type 1 diabetes is preventable, it is clear that modifiable factors exist for type 2 diabetes. If action is not taken to stem the tide of type 2 diabetes, the prospects for world health are bleak [7].

Diabetes

Diabetes mellitus is a chronic disease which has been described as a state of raised blood glucose associated with premature mortality. Itarises when the pancreas fails to produce

enough insulin (type 1 diabetes), or when the body cannot effectively make use of the insulin produced (type 2 diabetes). Failure of insulin secretion, action or both leads to raised blood glucose and other metabolic changes which, if uncontrolled, can cause serious complications [5]. The most important of these are retinopathy (affecting the eyes), nephropathy (affecting the kidneys), neuropathy (affecting the nerves) and cardiovascular disease (affecting the circulatory system) [6].Diabetes is fast becoming a world pandemic. Although there is no evidence that type 1 diabetes is preventable, it is clear that modifiable factors exist for type 2 diabetes. If action is not taken to stem the tide of type 2 diabetes, the prospects for world health are bleak [7]. A regular energy source is a prerequisite for every cell to function in the human body. Glucose is the body's primary energy source, which circulates in the blood as a mobilizable fuel source for cells [8,9,10]. Insulin is a pancreatic hormone responsible for blood glucoseregulation. The hormone binds to its receptor sites on peripheral side of the cell membranes. It affords entry of glucose into respiring cells and tissues via requisite channels. Insulin stimulates catabolism on glucose into pyruvate through glycolysis. It also upregulates glycogenesis from excessive cytosolic glucose and lipogenesis from excessive cytosolic acetyl-COA. These metabolic events are antagonistic to metabolic events triggered by the hormone glucagon. When glucose levels are at or below threshold, glucose stays in the blood instead of entering the cells [11]. The prevalence of diabetes increases with age in all race/ethnicity groups [12].

The body attempts to arrest hyperglycemia, by drawing water out of the cells and into the bloodstream. The excess sugar is excreted in the urine. This is why diabetics present with constant thirst, drinking large amounts of water, and polyuria as the cells try to get rid of the extra glucose. This subsequently leads to glucosuria[8].

Classification

One problem over the years has been the classification of diabetes into different categories. Most recently, a World Health Organization (WHO) Consultation and the American Diabetes Association (ADA) Expert Committee have divided diabetes into four main types. What was

previously known as insulin dependent diabetes mellitus (IDDM) has become type 1 diabetes under this now,

Table 1: Types of Diabetes

Type 1 diabetes	Insulin required for survival- due to a lack of insulinproduced by the body as a result of beta cell destruction.
Type 2 diabetes	• Characterized by disorders of both insulin action or secretion, either of which may predominate, but both of which are usually present. Usually controlled by diet, exercise and oral hypoglycemic agents. Insulin may be required for metabolic control.
Other specific types of diabetes	Other types of diabetes where the cause is known (eg: genetic defects in beta cell function or insulin action, diseases of the pancreas, certain other hormonal disorders,or drug induced disorders).

Risk Factors of Type 2 Diabetes

- Ethnicity
- Family history
- Obesity (particularly central)
- Physical inactivity
- Urbanization and mechanization
- Westernized diet
- Age

Cardiovascular Diseases

Globally, myocardial infarction is an important cause of premature death. Among its well-established modifiable risk factors are hypertension, dyslipidemia, smoking, and diabetes.[13]. Cardiovascular diseases are diseases that affect the heart and circulatory system. In developing

countries,the most common cause ofcardiovascular disease used to be infection ofthe heart valves. However, in recent years therehas been a shift away from infectious causes inmany developing nations. Today the mostwidespread form of cardiovascular diseasearound the world is that which starts with damage to the blood vessels[14]. Atherosclerosis is an immuno-in ammatory disease which occurs mainly in the large and medium sized arteries [15].

The two main processes by which the blood vessels become damaged are atherosclerosis and hypertension:

1. Atherosclerosis leads to the formation of plaques of atheroma which narrow the diameter of the large and medium-sized arteries. This narrowing of the arteries impairs blood flow. Plaques are also prone to rupture or to ulcerate and then act as a site for blood clot formation. The resulting blood clots, which can block the affected vessel completely, are usually responsible for the more severe clinical manifestations of cardiovascular disease such as heartattack and stroke.

2. Hypertension damages the smaller vessels in the circulatory system. Over time they become scarred, hardened, narrowed and less elastic. Hypertension can also both predispose to and accelerate the development of atherosclerosis [16].

Clinical Manifestation

The major clinical manifestations of cardiovascular disease can be divided into three groups:

1. Those affecting heart and coronary circulation (coronary heart disease).
2. those affecting the brain and cerebral circulation (cerebro-vascular disease); and
3. those affecting the lower limbs (peripheral vascular disease).[16,17]

Risk Factors of Cardiovascular Diseases

- Advancing age
- Diabetes and other high blood glucose conditions
- Dyslipidemia
- Genetic background
- High alcohol consumption
- Hypertension

- Insulin resistance
- Male gender
- Menopause
- Obesity
- Sedentary lifestyle
- Smoking

Pathophysiology

The pathophysiology of the link between diabetes and cardiovascular disease (CVD) is complex and multifactorial. Understanding these profound mechanisms of disease can help clinicians identify and treat CVD in patients with diabetes, as well as help patients prevent these potentially devastating complications. The endothelium, the innermost layer of blood vessels, is an active organ that regulates multiple vascular functions. It not only acts as a barrier but also regulates vascular growth, platelet function, and coagulation. When the endothelium is healthy, it releases nitric oxide (NO) and maintains a balance between various functions such as dilatation/constriction, growth inhibition/promotion, antithrombosis/prothrombosis, and anti-inflammation/proinflammation[18]. This article reviews the biological basis of the link between diabetes and CVD, from defects in the vasculature to the cellular and molecular mechanisms specific to insulin-resistant states and hyperglycemia. Insulin resistance and several other factors related to central adiposity have been implicated in endothelial dysfunction. Hyperglycemia induces free radical production and increases superoxide production and oxidative stress, which contributes to atherogenesis. Hyperglycemia also augments the expression of adipokines, further inducing adipokine-related endothelial dysfunction. Additionally, impaired insulin action in adipose tissue results in elevated rates of lipolysis and free fatty acid (FFA) release [19].

Macrovascular

Atherosclerosis is the major threat to the macrovasculature for patients with and without diabetes. Clinically, dyslipidemia is highly correlated with atherosclerosis, and up to 97% of patients with diabetes are dyslipidemic[20]. In addition to the characteristic pattern of increased triglycerides and decreased HDL cholesterol found in the plasma of patients with diabetes, abnormalities are seen in the structure of the lipoprotein particles. In diabetes, the predominant

form of LDL cholesterol is the small, dense form. Small LDL particles are more atherogenic than large LDL particles because they can more easily penetrate and form stronger attachments to the arterial wall, and they are more susceptible to oxidation. Because less cholesterol is carried in the core of small LDL particles than in the core of large particles, subjects with predominantly small LDL particles have higher numbers of particles at comparable LDL cholesterol levels [21].

Moreover, diabetic blood is more likely to be high in triglycerides. Dyslipidemia is only one mechanism by which diabetes promotes atherosclerosis; endothelial dysfunction often contributes. Healthy endothelium regulates blood vessel tone, platelet activation, leukocyte adhesion, thrombogenesis, and inflammation. The net effect of healthy endothelium is vasodilatory, anti-atherogenic, and anti-inflammatory [22].

When these mechanisms are defective, the process of atherosclerosis is accelerated. Therefore, both insulin deficiency and insulin resistance promote dyslipidemia accompanied by increased oxidation, glycosylation, and triglyceride enrichment of lipoproteins. In addition, endothelial dysfunction is present, and all of these factors contribute to the increase in atherogenicity, and thus macrovascular disease, found in patients with diabetes[23].

- Macrovascular
    1. Peripheral vascular disease
    2. Ischaemic heart disease
    3. Stroke
- Microvascular
    1. Nephropathy
    2. Neuropathy
    3. Retinopathy

Microvascular

Microvascular disease" associated with diabetes, we think of retinopathy, nephropathy, and neuropathy. In addition, however, small vessels throughout the body are affected by diabetes,

including those in the brain, heart, and peripheral vasculature. This small vessel damage is typically not related to atherosclerosis and is not predicted by lipid levels. [24].

Table 2: Prevalence of cardiovascular risk factors in people with diabetes compared to people without diabetes.

Hypertension	Prevalence is at least double in people with type 2 diabetes
High blood cholesterol	Prevalence is similar in people with diabetes.
High triglycerides with low HDL	Prevalence is higher in people with diabetes
Left ventricular hypertrophy	Most commonly seen in people with long-standing high blood pressure, but is also seen in the absence of elevated blood pressure in people with diabetes
Obesity	Prevalence is stronger in people with diabetes. Weight distribution is also usually different, with more central obesity which is linked with a tendency to develop coronary heart disease
Smoking	People with diabetes smoke less (presumably due to medical advice)

Table 3: Targets for common cardiovascular risk factors in people with diabetes

RISK FACTORS	**TARGETS**
Dyslipidaemia	• Decrease LDL cholesterollevels (<115mg/dl or 3 mmol/l*) • Raise HDL cholesterol levels (>46 mg/dl or 1.2 mmol/l*) • Lower triglycerides (<150 mg/dl or 1.7 mmol/l*)

Hypertension	• Lower blood pressure (<135/85 mm Hg).
Hyperglycaemia	• Reduce hyperglycaemia(HbA1c <7%)

Diabetes and its Relation to Cardiovascular Diseases

Diabetics have an increased risk of cardiovascular morbidity and mortality[25]. They particularly have an increased risk of congestive failure[26]. Diabetics in less affluent societies have fewer cardiovascular complications compared with non-diabetics in the same area and to diabetics inaffluent societies [27].

This suggeststhatnotall diabetesisthesame orthatother factorsmarkedlyinfluence itsimpacton health.Diabetesmellituswasoriginallyconsidered a simple consequence of a pancreasunable to produceenoughinsulin,causingtheblood sugar tobecomeunduly elevatedwhich,inturn, produced a series of disastrous metabolicalterationsculminatingincoma anddeath. Current concepts are more complex.Diabetesbeginning in childhood is usually due to severe insulin deficiency; when it firstappears inadultlife,itisoften associatedwithnormalor increasedinsulin levels.In the latter case, something interfering with insulin utilizationseems more likely. Theelevatedbloodpressure ofdiabetics had reported previously [28]. Incomparisonsoflipidvalues ofdiabetics andnon-diabetics,diabeticshavegenerallybeenfoundtohave elevated cholesterolvalues[29,30,31]. Other studies have indicated that diabetics who develop atheroscleroticcomplications have higher cholesterol values than thosewhodonot, [32] buttheredoesnot appear to have been any previous analysisofpossibleinteraction between cholesterol and diabetes.

Treatment

People with stable angina are usually treated by tight control of cardiovascular risk factorsand by prescribing drugs (eg: aspirin, beta blockers, nitrates and long-acting calcium channel blockers) [33]. When unstable angina develops, the risk of heart attack is very high and rapid preventive treatment in an intensive or coronary care unit may be required. People with diabetes who have experienced an acute heart attack benefit to the same degree, and in some cases even more, from therapeutic interventions that are used in people without diabetes [34].

Table 4: Effectiveness of medication in people with Diabetes

TREATMENT	EFFECTIVENESS IN PEOPLE WITH DIABETES
Aspirin •	Equally effective
Beta-blockers	• Equally effective
ACE inhibitors	• Particularly advantageous if started within 24 hours
Statins	• Equally effective
Clot dissolving	Useful within the first few hours only

Conclusion

Atherosclerosis is a major contributor to the morbidity and mortality observed in DM. The development of atherosclerosis is not only a result of hyperglycemia, but also from the secondary insulin resistance, dyslipidemia, hypercoagulability, altered secretion and function of local regulatory substances, and impaired response to injury. Strict glucose control alone is insufficient and a multifaceted approach targeting all mechanisms is required.

Corresponding Author

S. Pradeep

Senior Lecturer,Department of Conservative Dentistry and Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India

Telephone Number: +91 9710404482

Email Id: pandu.pradeep@gmail.com

Reference

1. Gupta R, Joshi P, Mohan V, Reddy KS, Yusuf S. Epidemiology and causation of coronary heart disease and stroke in India. Heart 2008; 94 : 16-26. サ
2. World Health Organization. World Health Statistics. Department of Measurement & Health Information Systems of the Information, Evidence and Research Cluster. Geneva: WHO Press; 2008:29-31.
3. DeFronzo RA, Ferrannini E. Insulin resistance. A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and atherosclerotic cardiovascular disease. Diabetes Care 1991;14:173-194.
4. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030," Diabetes Care, vol. 27, no. 5, pp. 1047–1053.
5. Wei M, Gaskill SP, Haffner SM, Stern MP. Effects of diabetes and level of glycemia on all-cause and cardiovascular mortality. The San Antonio Heart Study. Diabetes Care 1998;21:1167-1172.
6. Rutter MK, Nesto RW. Blood pressure, lipids and glucose in type 2 diabetes: How low should we go? Re-discovering personalized care. Eur Heart J 2011;32:2247-2255.
7. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care2003;26:5-20.
8. Piero MN. 2006. Hypoglycemic effects of some Kenyan plants traditionally used in management of diabetes mellitus in eastern province, Msc thesis, KenyattaUniversity.
9. Kibiti CM. Hypoglycaemic potential of some Kenyan plants used in traditional medicine in Rift valley, Nairobi and Eastern provinces, Msc thesis, Kenyatta University 2006.
10. Njagi JM. Hypoglycemic effects of some Kenyan plants used traditionally in the management of diabetes mellitus in Gachoka division, Mbeere district, Msc thesis, Kenyatta University, Kenya, 2006.
11. Belinda R. 2004. Gale Encyclopaedia of Alternative Medicine. pp 2603-2605.

12. JaseeraBanu.K, Naufal Rizwan.T.A. Epidemiology of Stroke Associate with Diabetes Mellitus. JMSCR Volume 04 Issue 10 2016
13. Yusuf S, Reddy S, Ôunpuu S. Global burden of cardiovascular diseases, part I: general considerations, the epidemiologic transition, risk factors, and impact of urbanization. Circulation 2001;104:2746–2753.
14. Eshwège E, Richard JL, Thibult N, Warnet JM, Claude JR, Rosselin GE. Coronary heart disease mortality in relation with diabetes, blood glucose andplasma insulin levels: the Paris Prospective Study, ten years later. Hormone and MetabolicResearch 1985;153:41-46.
15. Kritika Jangid, Jayakumar Nadathur Doraiswamy, Sheeja Varghese and Sankari Malaiappan. Periodontitis as a Risk Factor for Cardiovascular Diseases- Hype or a Fact?. Journal of Cardiovascular Disease Research Vol 6 Issue;Jan-Mar 2015.
16. Goldberg RA. Cardiovascular disease in diabetic patients. Med Clin North Am 2000;84:81-93.
17. Grundy SM, Benjamin IJ, Burke GL, Chait A, Eckel RH, Howard BV, Mitch W, Smith SC, Sowers JR. Diabetes and cardiovascular disease. A statement forhealthcare professionals from the American Heart Association. Circulation 1999 Sep 7;100(10):1134-1146.
18. Eckel RH. Natural history of macrovascular disease and classic risk factors foratherosclerosis. Diabetes Care 1999 Apr 1;22:C21
19. Huang PL. Unraveling the links between diabetes, obesity, and cardiovas- cular disease. Circ Res. 2005;96:1129-31.
20. Lau DC, Dhillon B, Yan H, Szmitko PE, Verma S. Adipokines: molecular links between obesity and atherosclerosis. Am J Physiol Heart Circ Physiol. 2005;288:2031-41.
21. Fagot-Campagna A, Rolka DB, Beckles GL, Gregg EW, Narayan KM: Prevalence of lipid ablormalities, awareness, and treatment in US adults with diabetes. Diabetes 49: A78, 2000.
22. Rosenson RS: Clinical role of LDL and HDL subclasses and apolipoprotein measurement. ACC Curr J Rev May, 2004, p. 33–37.
23. Celermejer D: Endothelial dysfunction: does it matter? J Am Coll Cardiol 30:325 – 333, 1997.

24. Lewis DH: The effect of trauma on the regulation of the microcirculation. Pathophysiol 5: 191, 1998
25. Smith SE, Smith SA, Brown PM: Cardiac autonomic dysfunction in patients with diabetic retinopathy. Diabetologia 1981;21:525 –528.
26. Stamler J,BerksonDM,LindbergHA: RiskFactors:theirrole intheetiologyandpathogenesisoftheatherosclerotic diseases. In The Pathogenesis of the Atherosclerotic Diseases, edited by Wissler RW, Geer JC. Baltimore, Williams and Wilkins Co, 1972: 67-69
27. Kannel WB, Hjortland M, Castelli WP: Role of diabetes in congestive heartfailure: theFraminghamstudy.AmJCardiol1974;34: 29.
28. Gordon T, Garcia-Palmieri MR, Kagan A, Kannel WB, Schiffman J: Differences in coronary heart disease in Framingham, Honolulu and Puerto Rico. J Chron Dis 1974; 27: 329-344.
29. Garcia MJ, McNamara PM, Gordon T, Kannel WB: Mor- bidity and mortality in diabetics in the Framingham population sixteen year followup study. Diabetes 1974; 23: 105.
30. Jarret RJ, Keen H: Diabetes and atherosclerosis. In Complications of Diabetes, edited by Keen H, Jarret RJ. Lon- don, Edward Arnold Co, 1975;179-203
31. Kaufmann RL, Assal J, Soeldner JS, Wilmhurst EG, Lemaira JR, Gleason RE, White P: Plasma lipid levels in diabetic children. Effect of diet restricted in cholesterol and saturated fats. Diabetes 1975;24: 672.
32. Kissebah AH, Siddig YK, Kohner EM, Lowy C, Lewis B, Fraser TR: Plasma lipids and glucose insuline relationship in non-insulin-requiring diabetics with and without retinopathy. Lancet 1975;1: 1104.
33. Lowy AD, Barach JH: Predictive value of lipoprotein ana cholesterol determinations in diabetic patients who developed cardiovascular complications. Circulation 1958;18: 14.
34. Tooke JE: Microcirculation and diabetes. British Med Bull 1989;4 : 206–223.
35. Gao Y, Lu B, Sun ML, Hou ZH, Fang FY, Cao HL, Chen Y, Yang YJ, Jiang SL, Budoff MJ. Comparison of atherosclerotic plaque by computed tomography angiography in patients with and without diabetes mellitus and with known or suspected coronary artery disease. Am J Cardiol. 2011 Sep 15;108 (6): 809–13.

Diabetic Peripheral Neuropathy – A Review

Karthic.A [1], Remmiya Mary Varghese [2]

Graduate Student [1], Senior Lecturer [2], Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Peripheral neuropathy is a micro vascular complication of diabetes and is a frequent cause of morbidity and disability. The various risk factors associated with Diabetic peripheral neuropathy (DPN) includes smoking, heavy alcohol use, dyslipidemia, duration of diabetes and genetic profile. DPN is usually symmetrical and affects distal extremities and hence it is also referred as distal symmetric polyneuropathy. Patients with DPN may be asymptomatic or present with symptoms such as paraesthesia, numbness, tingling sensation and loss of ankle jerk. Nerve conduction study will detect DPN even if patient is asymptomatic.

Key Words*: Diabetic peripheral neuropathy, morbidity, disability.*

Introduction

Diabetic peripheral neuropathy (DPN), likewise alluded to as distal symmetric polyneuropathy (DSP), is the nearness of side effects and additionally indications of fringe nerve brokenness in patients with diabetes after different reasons for brokenness have been excluded.[1]Diabetes mellitus is a critical reason for fringe neuropathy, representing half of all cases. The frequency of DPN is high in patients with diabetes, Another investigation, which characterized neuropathy as reciprocal nonappearance of lower leg jerks as well as vibratory misfortune, detailed a predominance of 70% in patients who have had type 2 diabetes for a long time or progressively [2,3] Recent examinations have evaluated that the pervasiveness of difficult DPN among diabetic patients is 20%.[4]Many of these patients and additionally the greater part of those with easy DPN are uninformed of their neuropathy. Easy neuropathy can be especially hazardous in light of the fact that patients will most likely be unable to distinguish damage to the foot (eg, get scorched from a hot shower, venture on a sharp question, or build up a rankle from a tight shoe). If not perceived and treated forcefully and rapidly, these wounds may bring about foot ulcers, contamination and even amputation.

Diabetes has turned out to be one of the biggest worldwide social insurance issues of the 21 st century. The quantity of individuals with diabetes worldwide is anticipated to twofold in the vicinity of 2000 and 2030, achieving a pandemic level of 366 million individuals. Diabetic polyneuropathy (DPN), which has a lifetime predominance of around half, is the most well-known diabetic difficulty. DPN is a main source for handicap because of foot ulceration and removal, walk unsettling influence, and fall-related damage. Roughly 20 to 30% of patients with DPN experience the ill effects of neuropathic torment. DPN fundamentally brings down personal satisfaction and considerably expands wellbeing costs related with diabetes.

Excruciating diabetic neuropathy (PDN) side effects display a symmetrical "stocking and gloves" appropriation and are o en related with nighttime compounding. It can be displayed from a mellow sticks and needle sensation to wounding, consuming, unremitting, or even upsetting electric stun sensation. Ere can be allodynia as cutaneous hypersensitivity prompting intense trouble on contact with an outer jolt, for example, apparel. The torment is regularly more terrible during the evening and frequently irritates rest, causing tiredness amid the day. A few patients give upsetting allodynia and serious agony in the legs. Is might be painful to the point that it

keeps them from playing out their day by day exercises, subsequently affecting their work and social life. E steady, unremitting torment and withdrawal from social life often result in discouragement. In outrageous cases, patients lose their craving and experience significant weight reduction, which which is reported in the literature as “diabetic neuropathic cachexia”

Physiology of Pain

Pain is the body's impression of real or potential harm to the nerve or tissue by poisonous boosts. tangible an afferent nerves convey sensations from the skin, joints, and viscera by means of huge and little fibres. Expansivefibres, for example, an alpha, are in charge of appendage proprioception and A-beta fibres convey vibes of appendage proprioception, weight, and vibration. Vast A-delta myelinatedfibresand little C unmyelinatedfibresare mostly in charge of conveying nociceptive sensations. Superficial torment is o en a sharp or pricking sensation and is transmitted by A-delta fibres. A profound situated, consuming, tingling, hurting sort of torment is o en went with hyperalgesia and allodynia and is transmitted by means of moderate, unmyelinated C fibres. Tissue harm brings about the arrival of inflammatory chemicals, for example, prostaglandins, bradykinins, and histamines, at the site of inflammation, which triggers the depolarization of nociceptors, along these lines producing an activity potential. e activity potential transmits the nociceptive sensation, by means of the dorsal root ganglion (DRG), to the dorsal horn of the spinal line. e arrival of glutamate and substance P brings about the hand-off of nociceptive sensations to the spinothalamic tract, thalamus, and, hence, the cortex, where torment is translated and seen.

Nociceptive agony is the typical reaction to toxic affront or damage of tissues, for example, skin, muscles, instinctive organs, and joints. Nociceptive torment more often than not dies down upon the mending of the tissue damage. Then again, neuropathic torment emerges as an immediate result of an injury or illness an etching the somatosensory framework with no poisonous jolts. Neuropathic torment is caused by harm or neurotic change and is portrayed by the actuation of anomalous pathways of agony at the fringe nerves and back roots (fringe neuropathic torment) or spinal string and mind (focal torment). Neuropathic torment appearance can be central,

multifocal, or summed up contingent upon the association of fringe or focal root and reason for the sickness.

Pathophysiology

Neuropathic Pain is a perplexing, ceaseless pain express that more often than not is joined by tissue damage. Usually in clinical practice and exhibits a test to patients and clinicians alike. With neuropathic torment, the nerve strands are harmed may end up useless or harmed. Neuropathic Agony is the aftereffect of ailment or damage to the fringe or focal sensory system (spinal or supra spinal sensory system) and the injury may happen anytime. These harmed nerve strands incorporate an adjustment in nerve work both at the site of the damage and territories around the damage and they send off base signs to other agony focuses. The effect of a nerve fiber damage incorporates an adjustment in nerve work—both at the site of the damage and zones around the damage. Clinical signs of Neuropathic Agony normally incorporate positive tangible marvels, for example, unconstrained torment, paraesthesia, allodynia, and hyperalgesia. Highlights that separate Neuropathic Agony from different kinds of torment incorporate torment and tactile manifestations enduring past the recuperating time frame.

The speculations with respect to the pathophysiology of Neuropathic Agony are numerous. Before going into subtle elements, a short audit with respect to torment circuit is required. There are two kinds of agony circuits: facilitatory and inhibitory circuits' facilitatory circuit in charge of torment sensation, and is especially interceded by glutamate (excitatory neurotransmitter) intervened receptors. The inhibitory circuits cause torment concealment for the most part intervened by GABA (gamma amino butyric corrosive), which is an inhibitory neurotransmitter. The system basic neuropathic torment, notwithstanding, isn't as clear. A few creature considers have demonstrated that numerous components might be included. To begin with arrange neurons may expand their terminating on the off chance that they are somewhat harmed because of increment in the quantity of sodium channels and therefore upgraded depolarization prompts unconstrained torment and development related agony. Impedance of Inhibitory circuits at the level of the dorsal horn or cerebrum stem permits uninhibited transmission of torment driving forces. Likewise, there are a few changes in the focal preparing of agony where there is

expanded the affectability of spinal neurons and abatement in actuation edges bringing about focal refinement marvel working in patients of diabetic neuropathy. Raja, et al. in an intriguing examination portrayed that barricade of α - adrenergic receptor work with intravenous imbuement of the rival phentolamine additionally prompts relief from discomfort. In this way, there likewise can be the connection of the thoughtful sensory system towards neuropathic torment.

The correct instrument of nerve harm in diabetic neuropathy is as yet misty, metabolic and vascular/hypoxic factors seem, by all accounts, to be included. Propel Glycosylation Finished results (AGEP) may harm vessels; repress axonal transport prompting axonal degeneration. Increment generation of sorbitol because of actuation of polyol pathway causes basic nerve harm. Additionally, expanded endoneurial vascular protection from hyperglycemic blood can prompt nerve ischemia.

Etiology

Diabetic neuropathy is a type of nerve damage that can occur who have diabetes. High blood sugar can injure nerve fibers throughout your body, but diabetic neuropathy most often damages nerves in legs and feet.[5] Depending on the affected nerves, symptoms of diabetic neuropathy can range from pain and numbness in extremities to problems with digestive system, urinary tract, blood vessels and heart. For some people, these symptoms are mild; for others, diabetic neuropathy can be painful, disabling and even fatal. Diabetic neuropathy is a common serious complication of diabetes. Prolonged exposure to high blood sugar can damage delicate nerve fibers, causing diabetic neuropathy. [6]Why this happens isn't completely clear, but a combination of factors likely plays a role, including the complex interaction between nerves and blood vessels. High blood sugar interferes with the ability of the nerves to transmit signals. It also weakens the walls of the small blood vessels (capillaries) that supply the nerves with oxygen and nutrients.[7] Other factors that may contribute to diabetic neuropathy include:

- **Inflammation in the nerves** caused by an autoimmune response. This occurs when immune system mistakenly attacks part of body as if it were a foreign organism.

- **Genetic factors** unrelated to diabetes that make some people more susceptible to nerve damage.
- **Smoking and alcohol abuse,** which damage both nerves and blood vessels and significantly increase the risk of infections.

Symptoms

There are four main types of diabetic neuropathy. The signs and symptoms of diabetic neuropathy vary, depending on the type of neuropathy and which nerves are affected.

Peripheral neuropathy

Peripheral neuropathy is the most common form of diabetic neuropathy. Feet and legs are often affected first, followed by hands and arms. [8]Signs and symptoms of peripheral neuropathy are often worse at night, and may include:

- Numbness or reduced ability to feel pain or temperature changes
- A tingling or burning sensation
- Sharp pains or cramps
- Increased sensitivity to touch
- Muscle weakness
- Loss of reflexes, especially in the ankle
- Loss of balance and coordination
- Serious foot problems, such as ulcers, infections, deformities, and bone and joint pain

Autonomic neuropathy The autonomic nervous system controls heart, bladder, lungs, stomach, intestines, sex organs and eyes. [9, 8]Diabetes can affect the nerves in any of these areas, possibly causing:

- A lack of awareness that blood sugar levels are low (hypoglycemia unawareness)
- Bladder problems, including urinary tract infections or urinary retention or incontinence
- Constipation, uncontrolled diarrhea or a combination of the two
- Slow stomach emptying (gastroparesis), leading to nausea, vomiting, bloating and loss of appetite
- Difficulty swallowing
- Erectile dysfunction in men
- Increased or decreased sweating
- Inability of body to adjust blood pressure and heart rate, leading to sharp drops in blood pressure after sitting or standing that may cause you to faint or feel lightheaded
- Problems regulating your body temperature
- Changes in the way your eyes adjust from light to dark

RadiculoplexusNeuropathy (Diabetic Amyotrophy)

Radiculoplexus neuropathy affects nerves in the thighs, hips, buttocks or legs. Also called diabetic amyotrophy, femoral neuropathy or proximal neuropathy, this condition is more common in people with type 2 diabetes and older adults. Symptoms are usually on one side of the body, though in some cases symptoms may spread to the other side. Most people improve at least partially over time, though symptoms may worsen before they get better. This condition is often marked by:

- Sudden, severe pain in your hip and thigh or buttock
- Eventual weak and atrophied thigh muscles

- Difficulty rising from a sitting position
- Abdominal swelling, if the abdomen is affected
- Weight loss

Mononeuropathy

Mononeuropathy involves damage to a specific nerve. The nerve may be in the face, torso or leg. Mononeuropathy, also called focal neuropathy, often comes on suddenly [11, 12] It's most common in older adults. Although mononeuropathy can cause severe pain, it usually doesn't cause any long term problems. Symptoms usually diminish and disappear on their own over a few weeks or months. Signs and symptoms depend on which nerve is involved and may include:

- Difficulty focusing your eyes, double vision or aching behind one eye
- Paralysis on one side of your face (Bell's palsy)
- Pain in shin or foot
- Pain in lower back or pelvis
- Pain in the front of your thigh
- Pain in chest or abdomen

Sometimes mononeuropathy occurs when a nerve is compressed. Carpal tunnel syndrome is a common type of compression neuropathy in people with diabetes.

Signs and symptoms of carpal tunnel syndrome include:

- Numbness or tingling infingers or hand, especially in thumb, index finger, middle finger and ring finger
- A sense of weakness in hand and a tendency to drop things.

Complications

Loss of a limb Because nerve damage can cause a lack of feeling in feet, cuts and sores may go unnoticed and eventually become severely infected or ulcerated — a condition in which the skin and soft tissues break down. The risk of infection is high because diabetes reduces blood flow to feet. Infections that spread to the bone and cause tissue death (gangrene) may be impossible to treat and require amputation of a toe, foot or even the lower leg.[13]

Charcot joint This occurs when a joint, usually in the foot, deteriorates because of nerve damage. Charcot joint is marked by loss of sensation, as well as swelling, instability and sometimes deformity in the joint itself. Early treatment can promote healing and prevent further damage.[14]

Urinary tract infections and urinary incontinence Damage to the nerves that control bladder can prevent it from emptying completely. This allows bacteria to multiply in bladder and kidneys, leading to urinary tract infections. Nerve damage can also affect ability to feel when need to urinate or to control the muscles that release urine.

Hypoglycemia unawareness Normally, when blood sugar drops too low — below 70 milligrams per deciliter (mg/dL), or 3.9 millimoles per liter (mmol/L) — develop symptoms such as shakiness, sweating and a fast heartbeat. Autonomic neuropathy can interfere with ability to notice these symptoms.[15,16]

- **Low blood pressure** Damage to the nerves that control circulation can affect body's ability to adjust blood pressure. This can cause a sharp drop in pressure when stand after sitting (orthostatic hypotension), which may lead to dizziness and fainting.
- **Digestive problems** Nerve damage in the digestive system can cause constipation or diarrhea — or alternating bouts of constipation and diarrhea — as well as nausea, vomiting, bloating and loss of appetite. It can also cause gastroparesis, a condition in which the stomach empties too slowly or not at all. This can interfere with digestion and cause nausea, vomiting and bloating, and severely affect blood sugar levels and nutrition.

- **Sexual dysfunction** Autonomic neuropathy often damages the nerves that affect the sex organs, leading to erectile dysfunction in men and problems with lubrication and arousal in women.
- **Increased or decreased sweating**When the sweat glands don't function normally, body isn't able to regulate its temperature properly. A reduced or complete lack of perspiration (anhidrosis) can be life-threatening. Autonomic neuropathy may also cause excessive sweating, particularly at night or while eating.
-

Diagnosis

Diabetic neuropathy is usually diagnosed based on symptoms, medical history and a physical exam. **Filament test.** Sensitivity to touch may be tested using a soft nylon fiber called a monofilament.[17]

- **Nerve conduction studies.** This test measures how quickly the nerves in arms and legs conduct electrical signals. It's often used to diagnose carpal tunnel syndrome.
- **Electromyography (EMG).** Often performed along with nerve conduction studies, electromyography measures the electrical discharges produced in muscles.
- **Quantitative sensory testing.** This noninvasive test is used to assess how nerves respond to vibration and changes in temperature.
- **Autonomic testing.**If have symptoms of autonomic neuropathy, your doctor may request special tests to look at blood pressure in different positions and assess ability to sweat

Prevention

Blood sugar controlkeeping your blood sugar tightly controlled requires continuous monitoring and, if patient take insulin, frequent doses of medication. But keeping blood sugar consistently within target range is the best way to help prevent neuropathy and other complications of diabetes [18] Consistency is important because shifts in blood sugar levels produce damage

Foot care

- Foot problems, including sores that don't heal, ulcers and even amputation, are a common complication of diabetic neuropathy[19,20] But can prevent many of these problems by having a comprehensive foot exam at least once a year, having your doctor check your feet at each office visit and taking good care of feet at home. **Check your feet every day.** Look for blisters, cuts, bruises, cracked and peeling skin, redness and swelling. Use a mirror or ask a friend or family member to help examine parts of feet that are hard to see.
- **Keep your feet clean and dry.** Wash feet every day with lukewarm water and mild soap. Avoid soaking feet. Dry feet and between your toes carefully by blotting or patting with a soft towel.

  Moisturize feet thoroughly to prevent cracking. Avoid getting lotion between toes, however, as this can encourage fungal growth.

- **Trim your toenails carefully.** Cut toenails straight across, and file the edges carefully so there are no sharp edges.
- **Wear clean, dry socks.** Look for socks made of cotton or moisture-wicking fibers that don't have tight bands or thick seams.
- **Wear cushioned shoes that fit well.** Always wear shoes or slippers to protect your feet from injury.

Treatment

Diabetic neuropathy has no known cure. Treatment for diabetic neuropathy focuses on:

- Slowing progression of the disease
- Relieving pain
- Managing complications and restoring function.

Conclusion

This review comprises the importance of understanding the causes,symptoms,complications and management of Diabetic Peripheral Neuropathy (DPN)

Corresponding author

Remmiya Mary Varghese

Senior Lecturer, Department of Orthodontics

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Science

162 Poonamallee High Road

Chennai-600077

Tamil Nadu, India

Email ID-remmiya@gmail.com

REFERENCES

1. Boulton AJ, Gries FA, Jervell JA.Guidelines for the diagnosis and outpatient.Management of diabetic peripheral neuropathy.Diabet Med 1998; 15:508–14.

2. Dyck PJ, Karnes JL, O'Brien PC. The Rochester Diabetic Neuropathy Study: reassessment of tests and criteria for diagnosis and staged severity. Neurology 1992; 42:1164–70.

3. Mohan V, Vijayaprabha R, Rema M. Vascular complications in long-term. Southern Indian NIDDM of over 25 year's duration. Diabetes Res Clin Pract
1996; 31:133–40.

4. Schmader KE. Epidemiology and impact on quality of life of postherpetic.Neuralgia and painful diabetic neuropathy. Clin J Pain 2002; 18:350–4.

5. Tanenberg RJ, Pfeifer MA. Neuropathy: the "forgotten" complication. Diabetes
Forecast 2000; 53:56–60.

6. Tasfaye S, Chaturvedi N, Eaton S, et al. Vascular risk factors and diabetic neuropathy.N Engl J Med 2005; 352:341–50.

7. Vinik AI, Mehrabyan A. Diabetic neuropathies. Med Clin N Am 2004 88:947–99.

8. Tanenberg RJ, Donofrio PD. Neuropathic problems of the lower extremities. In diabetic patients. In: Bower JH, Pfeifer MA, editors. Levin and O'Neal's.The diabetic foot, 7th ed. St. Louis: Mosby; 2008.

9. International Association for the Study of Pain. IASP pain terminology.Available at www.iasp-pain.org/AM/Template.cfm?Section=Pain_Definitions.
Accessed 3 Nov 2009.

10. Treede RD, Jensen TS, Campbell JN, .Neuropathic pain: redefinition and a grading system for clinical and research purposes. Neurology 2008;70:1630–5.

11. Duby JJ, Campbell RK, Setter SM. Diabetic neuropathy: An intensive review. Am J Hosp Pharm. 2004; 61:160–173.

12. American Diabetes Association Standards of Medical Care in Diabetes 2007. Diabetes Care. 2007; 30(Supp 1):S4–S41.

13. Tseng CH. Prevalence of lower-extremity amputation among patients with diabetes mellitus: Is height a factor? Can Med Assoc J 2006311743319–323.323.

14. Head KA. Peripheral neuropathy: Pathogenic mechanisms and alternative therapies. Altern Med Rev. 2006; 11(4):294–329.

15. Max MB, Culnane M, Schafer SC. Amitriptyline relieves diabetic neuropathy pain in patients with normal or depressed mood. Neurology.1987; 37:589–596.

16. Argoff CE, Backonja MM, Belgrade MJ, Consensus guidelines: Treatment planning and options. Mayo Clin Proc. 2006; 81(4 Suppl):S12–S25.

17. Joss JD. Tricyclic antidepressant use in diabetic neuropathy. Ann Pharmacother. 1999; 33:996–1000.

18. McQuay HJ, Framer M, Carroll ND, A systematic review of antidepressants in neuropathic pain.Pain.1996; 68:217–227.

19. Sindrup SH, Ejlertsen B, Froland A,. Imipramine treatment in diabetic neuropathy: Relief of subjective symptoms without changes in peripheral and autonomic nerve function. Eur J Clin Pharmacol. 1989; 37:151–153.

20. Kvinesdal B, Molin J, Froland A, Imipramine treatment of painful diabetic neuropathy. JAMA.1984; 251:1727–1730.

Diagnosing Aids of Oral Cancer – A Review

Anandadeeban K B S [1], Balakrishna R.N [2]

Graduate Student [1], Senior Lecturer [2], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Prevention and early detection are major objectives in the control of the oral cancer burden worldwide. At the present time, screening of oral cancer and its pre-invasive intra-epithelial stages, as well as its early detection, is still largely based on visual examination of the mouth. There is strong available evidence to suggest that visual inspection of the oral mucosa is effective in reducing mortality from oral cancer in individuals exposed to risk factors.However is well known to be limited by subjective interpretation and by the potential, albeit rare, occurrence of dysplasia and early OSCC within areas of normal-looking oral mucosa. As a consequence, adjunctive techniques have been suggested to increase our ability to differentiate between benign abnormalities and dysplastic/malignant changes as well as to identify areas of dysplasia/early OSCC that are not visible to naked eye. These include the use of toluidine blue, brush biopsy, chemiluminescence and tissue autofluorescence. We conclude that available studies have shown promising results, but strong evidence to support the use of oral cancer diagnostic aids is still lacking.

Key Words: Oral cancer, Biopsy, Premalignant lesion

Introduction

The incidence of oral cancer is rising in most countries, especially in developing countries. In the general male population, oral cancer is the sixth most frequent cancer [1]. Squamous cell carcinoma (SCC) accounts for 95% of oral cancers, and it is associated with avoidable etiological risk factors. Familial history of oral cancer is also documented as a risk factor for SCC of the head and neck. It affects the tissue lining of the mouth which is perfectly visible to dentists during routine examination. Dentists being the first to encounter such changes in the oral cavity, have the responsibility to counsel, diagnose and treat the disease at initial stage. Most of the oral cancer arises from premalignant lesion and is usually Asymptomatic,routine dental screening is essential for early diagnosis. cancer lesions with immediate referral to a head and neck surgeon or cancer treatment canter for biopsy and treatment of cancer is the best way to manage such cases and proper screening for evaluation of metastasis to or in some cases from other sites like breast,pancreas etc.

Cancer of the head and neck (H&N cancer), including all oral, laryngeal and pharyngeal sites, is the sixth most common cancer, accounting for about 643 000 new cases annually [2]. About 40% of head and neck malignancies are known to be squamous cell carcinomas arising in the oral cavity . Oral cancer is largely related to lifestyle, with major risk factors being tobacco and alcohol misuse. In addition to smoking, the use of smokeless tobacco has been strongly linked to oral cancer. Carcinoma of oral cavity occur as a primary lesion which originates in any of the tissues in the oral cavity,either by metastasis from a distant site of origin, or by extending from a neighbouring anatomic structure, such as the nasal cavity.The oral cancers may originate in any of the tissues of the mouth, and may be of varied histologic types: teratoma, adenocarcinoma which are derived from a major or minor salivary gland, lymphoma from tonsillar or other lymphoid tissue, or melanoma from the pigment-producing cells of the oral mucosa. Oral or mouth cancer most commonly involves the tongue. It may also occur on the floor of the mouth, cheek lining, gingival(gums), lips, or palate (roof of the mouth). Less commonly other types of oral cancer occur, such as Kaposi's sarcoma[3]. Diagnostic tools and aids to diagnosis are discussed, as are treatment modalities. It is imperative that all dental professionals perform a simple head and neck examination in addition to an oral examination during each new patient visit and each six-month recall appointment. Early detection saves lives.This article reviews on various diagnostic aids for detection of oralcancer.In this review article we will be discussing about the various diagnostic aids present for detection of the oral cancer

Detection of Precancerous Stage

Early diagnosis of oral cancer patients would decrease mortality and help to improve treatment. Oral surgeons and dentists are the early diagnoses that diagnose these patients in early stages. Persons who deal with oral cancer such as healthcare providers, dental surgeons, and oral oncologist should have high knowledge and awareness that would help them to provide better diagnosis for oral cancer patients. The clinical significance of oral precancerous lesions lies in its association with malignant transformation into OSCC[4,5]. Only by histology the oral cancerous lesion can be diagnosed, although present as a spectrum of epithelial changes, rather than distinct categories. The World Health Organization in 2005 graded precancerous changes into mild, moderate, severe and carcinoma in situation.Precancerous lesion occur in oral cavity in three types.They are white,red and mix of white and red lesions.These conditions are known as leukoplakia which are white lesion or erythroplakia which are red lesions[6]. There are other pathological conditions that are considered precancerous including oral lichen planus and oral submucous fibrosis. The malignant potential of the above mentioned oral lesions cannot be accurately predicted solely on the basis of their clinical characteristics, histological evaluation is essential for all suspicious lesions. Unfortunately, histological findings only indicate that a given lesion may have malignant potential (dysplasia), and cannot be used for the prediction of malignant changes. Thus, the presence of dysplasia only indicates that an oral lesion may have an increased risk of malignant transformation[7]. Molecular biomarkers capable of identifying the subset of lesions likely to progress to cancer are being widely investigated including genetic and epigenetic alterations observed in oral mucosal precancerous lesions.

Investigations

Chest x-ray, CT or MRI scans, and tissue biopsy are some special investigations that are done during diagnosing oral cancer. There are a different types of screening devices that may help dentists in diagnosing oral cancer, including the Velscope, Vizilite Plus and the identafi 3000. There is no evidence that routine use of these devices in general dental practice saves lives. The most traditional type of biopsy is incisional. This is simply taking a small portion of the cancerous tissue for microscopic examnation. In an incisional biopsy, the doctor will remove part or all of the lesion depending on its size and his ability to define the extent of it at this early stage. In diagnosing the larger areas such as an enlarged lymph node, fine needle aspiration cytology (fine needle biopsy or FNB) has found an increasing role in

diagnosis[8,9] The technique is reliable and relatively inexpensive. In it, a syringe attached with a small needle is inserted into the questionable mass, and cells are aspirated, or pulled out into the syringe as the doctor draws back the piston of the syringe. Depending onplacement of the needle , as with all biopsies, the skill and experience of the pathologist who will be diagnosing the cells,the success will be determined . It is likely that the dentist will insert the needle and draw out cellular material from several different locations in the mass to ensure that a thorough and representative sample has been taken.

Non-Invasive Methods

Recent advancements in oral cancer research have led to the development of potentially useful diagnostic tools at the clinical and molecular level for the early detection of oral cancer. A non-invasive brush biopsy (BrushTest) can be performed to rule out the presence of dysplasia (pre-cancer) and cancer on areas of the mouth that exhibit an unexplained colour variation or lesion. While this has some usefulness in preliminary evaluation of a suspect area, it is not a standalone procedure, and if a positive find returns, this must be confirmed by a conventional incisional biopsy[10].

Other ways to determine the presence or extent of oral cancer exist. For instance, radiographs, also referred to as x-rays, can assist in determining the potential growth of a tumour into bone. While oral cancers unlike many other malignancies can usually be seen with the naked eye, some cancers are located internally in the body, making their detection difficult. Different scanning options, some of which assist in determining the presence of tumours or growths, and some of which can even detect malignancy, are necessary in these instances. CT O CAT and Magnetic Resonance Imaging (MRI), is helpful in providing accurate views of the affected area. Because cancer cells are dividing rapidly, they break down glucose much faster than normal cells. The increased activity will show up on a PET scan, and can indicate both primary and metastatic tumours. All these types of scans are still used largely for confirmation or measuring extent. Saliva from patients has been used in a novel way to provide molecular biomarkers for oral cancer detection. Saliva is a mirror of the body, reflecting virtually the entire spectrum of normal and disease states and its use as a diagnostic fluid meets the demands for an inexpensive, non-invasive and accessible diagnostic tool. Discovery of analytes in saliva of normal and diseased subjects suggests a very promising function of saliva as a local and systematic diagnostic tool. Toluidine blue (TB) is a member of the thiazine group of metachromatic dyes, which binds to DNA and is partially soluble both in water and in alcohol. Theoretically, dysplastic and malignant cells

have higher nucleic acid content than normal, and thus, staining of suspicious lesions with this dye can aid recognition of mucosal changes[11] TB has been used as a vital stain to highlight potentially malignant oral lesions[12].since the early 1980s. A positive staining of TB may appear as a dark royal blue. Recently, molecular studies on TB stained lesions reported a link between carcinoma and loss of heterozygosity at 3p and 17p, while dysplasia resulted in loss of heterozygosity at 9p. The presence of loss of heterozygosity has also been reported in high frequency of TB-stained lesions without or with low grade dysplasia[13]

Brush Biopsy

The oral brush biopsy, also known as OralCDx Brush Test system, consists of a method of collecting a trans-epithelial sample of cells from a mucosal lesion with representation of the superficial, intermediate and parabasal/basal layers of the epithelium[16]. This test was specifically designed to investigate mucosal abnormalities that would otherwise not be subjected to biopsy because of low-risk clinical features [17].A specially designed brush is the non-lacerational device used for epithelial cell collection and samples are eventually fixed onto a glass slide, stained with a modified Papanicolaou test and analyzed microscopically via a computer-based imaging system. Results are reported as "positive" or "atypical" when cellular morphology is highly suspicious for epithelial dysplasia or carcinoma or when abnormal epithelial changes are of uncertain diagnostic significance respectively. Results are defined as negative when no abnormalities can be found. The test is considered an intermediate diagnostic step as a scalpel biopsy must follow when an abnormal result is reported (atypical or positive).

Several studies have been performed in an attempt to test the sensitivity and specificity of brush biopsy in detecting dysplasia or OSCC. However, inconsistencies and potential bias of these studies have been reported by several authors[18].In the majority of studies for example, scalpel biopsy was performed after brush biopsy of lesions with high-risk clinical features, but not after brush biopsy of innocuously-looking lesions [18]. This is believed to alter results regarding sensitivity and specificity of the test in the clinical context where accuracy is much needed (diagnosis of those lesions that appear innocuous and would otherwise not be biopsied) and supports the criticism an intermediate non-diagnostic test would be superfluous when clinical features are highly suspicious for dysplasia/OSCC and a biopsy has to be performed anyway.

Indeedcurrent data show that OralCDx's cytologic test is highly sensitive and specific in detecting dysplastic changes in high-risk mucosal lesions (due to clinical findings suggestive of malignancy), but when used in a low-risk population with benign-appearing oral epithelial lesions, the accuracy is reduced and the rate of false-positive findings increases.
Further rigorous studies are needed to investigate the sensitivity and specificity of brush biopsy in detecting in low-risk populations with clinically innocuous lesions.

Toluidine Blue Staining

Toluidine blue (TB), also known as tolonium chloride, is a vital dye that is believed to stain nucleic acids. Hence, it has been used for many years as an aid to the identification of clinically occult mucosal abnormalities and as a useful way of demarcating the extent of a potentially malignant lesion prior to excision [19] Several studies on TB have been performed in the past years but the majority of them present significant limitations and methodological biases [18,19]. These have been reviewed in detail by Lingen and coworkers and include (i) absence of randomized controlled trials, (ii) absence of histological diagnosis as a gold standard, (iii) and variability in methods of application [20].

Analysis of current evidence suggests that TB is good at detecting carcinomas, but its sensitivity in detecting dysplasias is significantly lower. Furthermore, there remain a high percentage of false positive stains which impairs its use in primary care settings as a valid screening mean. In addition, controversy exists regarding the subjective interpretation of mucosal staining and criteria for positive results (e.g. dark royal blue versus pale blue staining) .

At present, TB is best used by experienced clinicians as an adjunct to clinical examination in the evaluation of the biologic potential of potentially malignant oral lesions. A recent study [21] showed that TB preferentially stained visible lesions with high risk molecular patterns and predicted risk and outcome in cases where little to no microscopic evidence of dysplasia was present. To date, however, research has not been extended to determine whether TB can identify and predict the risk of progression for epithelial abnormalities that cannot be seen with the naked eye.

Chemiluminescence

Clinical inspection of oral mucosa with the aid of chemiluminescent blue/white light was recently suggested to improve the identification of mucosal abnormalities with respect to the use of normal incandescent light[22].

The relevant technology (ViziLite system – Zila Pharmaceuticals, Phoenix, AZ), involves the use of an oral rinse with a 1% acetic acid solution for 1 minute followed by the examination of the oral mucosa under diffuse chemiluminescent blue/white light (wavelength of 490 to 510 nm). The theory behind this technique is that the acetic acid removes the glycoprotein barrier and slightly desiccates the oral mucosa, the abnormal cells of the mucosa then absorbing and reflecting the blue/white light in a different way with respect to normal cells. Hence normal mucosa appears blue, whereas abnormal mucosal areas reflect the light (due to higher nuclear/cytoplasmic ratio of epithelial cells) and appear more aceto-white with brighter, sharper and more distinct margins [23]

More recently, the ViziLite system was modified in order to include the use of TB and a new chemiluminescence device (MicroLux DL) was introduced.

Several studies have been performed with the Vizilite system with the attempt to demonstrate its efficacy in to enhance the identification of mucosal abnormalities. It should be highlighted that no study has demonstrated that the chemiluminescence can help in differentiating dysplasia/carcinoma from benign lesions. Hence, the majority of studies have investigated how chemiluminescence enhances subjective clinical evaluation of intra-oral lesions including brightness, sharpness and texture with respect to routine clinical examination .As these parameters are highly subjective, it is not surprising that results have been contradictory . Whilst some authors report that this technique can improve the detection of intra-oral abnormalities (regardless their nature), other reported that the overall detection rate was not significantly improved and the chemiluminescent light produced reflections that made visualization even more difficult than with incandescent light . Furthermore, the majority of the studies are limited by methodological flaws such as lack of histopathological diagnosis or clear objectives (screening device versus case-finding device) . Some studies suggest that chemiluminescence may help identifying occult lesions that cannot be seen with incandescent light but this, however, is not supported by any strong evidence [24].

Tissue Fluorescence Imaging

Tissue autofluorescence has been used in the screening and diagnosis of precancers and early cancer of the lung, uterine cervix, skin and, more recently, of the oral cavity. The concept behind tissue autoflorescence is that changes in the structure (e.g., hyperkeratosis, hyperchromatin and increased cellular/nuclear pleomorphism) and metabolism (e.g. concentration of flavin adenine dinucleotide [FAD] and nicotinamide adenine dinucleotide [NADH]) of the epithelium, as well as changes of the sub-epithelial stroma (e.g. composition of collagen matrix and elastin), alter their interaction with light [25] Specifically, these epithelial and stromal changes can alter the distribution of tissue fluorophores and as a consequence the way they emit fluorescence after stimulation with intense blue excitation (400 to 460 nm) light, a process defined autoflorescence. The autoflorescence signal is finally visualized directly by a human observer. With regards to the oral cavity, normal oral mucosa emits a pale green autofluorescence when viewed through the instrument handpiece whilst abnormal tissue exhibits decreased autofluorescence and appears darker with respect to the surrounding healthy tissue. Autoflorescence technology for inspection of the oral mucosa has been developed by LED Medical Diagnostics Inc.

Tissue Flouresence Spectroscopy

In addition to visual autofluorescence, a technique called autofluorescence spectroscopy has been recently tested in oral oncology research[26].The autofluorescence spectroscopy system consists of a small optical fiber that produces various excitation wavelengths and a spectrograph that receives and records on a computer and analyzes, via a dedicated software, the spectra of reflected fluorescence from the tissue . This technique has the clear advantage of eliminating the subjective interpretation of tissue fluorescence changes. However, the downside is that more variables (e.g. combination of wavelengths, methodology of fluorescence analysis etc) have to be tested and considered and this has led to controversial and often unclear results. Overall, autofluorescence spectroscopy seems to be very accurate for distinguishing lesions from healthy oral mucosa, with high sensitivity and specificity, especially when malignant tumors are compared to healthy mucosa. However, the ability of the technique to distinguish and classify different types of lesion has been reported to be low.Moreover autofluorescence spectroscopy is for practical reasons not suitable to detect new lesions or to demarcate large lesions as the optical fiber can sample only a small mucosal area. This limits the use of spectroscopy to the evaluation of a well defined small mucosal lesion that has been already identified through visual inspection, with the attempt to clarify its

benign or (pre)malignant nature. Further research is needed to support this clinical application of autofluorescence spectroscopy

Conclusion

The World Health Organization has clearly indentified prevention and early detection as the major targets in the battle to control the oral cancer burden worldwide (26) Prevention and early detection of Oral cancer and its pre-invasive intra-epithelial stages is still largely based on visual examination of the mouth, although a variety of molecular techniques have been tested and are likely to represent the ultimate goal of oral cancer research [27].Toluidine Blue has been used by clinicians for many years, yet a clear demonstration of TB indications, limitations, as well as strong evidence from methodologically sound clinical trials is still lacking. Brush biopsy is another example of promising novel diagnostic technique that unfortunately has not been supported by robust evidence. At present, the utilization of these techniques in clinical practice is largely anedoctal and is principally directed to help experienced clinicians at improving their ability to detect dysplasia and early Oral cancer in high-risk individuals attending secondary and tertiary centers.

Corresponding Author

Balakrishna R.N
Senior Lecturer,Department of Oral and Maxillofacial Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162,Poonamallee High Road,Chennai 600077,Tamil Nadu,India
Phone Number: +91 8939896729
Email id: balakrishdr8394@gmail.com

References

1. 1.Harshitha C, Laliytha KB. Effect of Radiotherapy on the Oral Cavity. Journal of Pharmaceutical Sciences and Research. 2017 Dec 1;9(12):2332-4.
2. Nazar SA, Uma Maheshwari TN. Awareness Of Early Detection And Prevention Of Oral Cancer Among Dentists-A Review. IOSR-JDMS.;13(1):10-2.

3. Ries LA, Harkins D, Krapcho M, Mariotto A, Miller BA, Feuer EJ, Clegg LX, Eisner MP, Horner MJ, Howlader N, Hayat M. SEER cancer statistics review, 1975-2003.
4. Mignogna MD, Fedele S, Russo LL, Ruoppo E, Muzio LL. Costs and effectiveness in the care of patients with oral and pharyngeal cancer: analysis of a paradox. European journal of cancer prevention. 2002 Jun 1;11(3):205-8.
5. 5.Mashberg A. Diagnosis of early oral and oropharyngeal squamous carcinoma: obstacles and their amelioration. Oral oncology. 2000 May 1;36(3):253-5.
6. 6.Mignogna MD, Fedele S, Russo LL, Ruoppo E, Muzio LL. Oral and pharyngeal cancer: lack of prevention and early detection by health care providers. European Journal of Cancer Prevention. 2001 Aug 1;10(4):381-3.
7. 7.Mignogna MD, Fedele S, Russo LL, Muzio LL, Bucci E. Immune activation and chronic inflammation as the cause of malignancy in oral lichen planus: is there any evidence?. Oral oncology. 2004 Feb 1;40(2):120-30.
8. Thomson PJ. Field change and oral cancer: new evidence for widespread carcinogenesis?. International journal of oral and maxillofacial surgery. 2002 Jun 1;31(3):262-6.
9. Derk CT, Rasheed M, Artlett CM, Jimenez SA. A cohort study of cancer incidence in systemic sclerosis. The Journal of rheumatology. 2006 Jun 1;33(6):1113-6.
10. Rajendran R. Oral submucous fibrosis: etiology, pathogenesis, and future research. Bulletin of the World Health Organization. 1994;72(6):985.
11. Lingen MW, Kalmar JR, Karrison T, Speight PM. Critical evaluation of diagnostic aids for the detection of oral cancer. Oral oncology. 2008 Jan 1;44(1):10-22.
12. Eaton L. World cancer rates set to double by 2020. BMJ: British Medical Journal. 2003 Apr 5;326(7392):728.
13. World Health Organization. Cancer control: knowledge into action: WHO guide for effective programmes. World Health Organization; 2006.
14. Allison P, Locker D, Feine JS: The role of diagnostic delays in the prognosis of oral cancer: a review of the literature. Oral Oncol. 1998, 34: 161-170. 10.1016/S1368-8375(97)00071-7.
15. Smart CR. Screening for cancer of the aerodigestive tract. Cancer. 1993 Aug 1;72(S3):1061-5.
16. Eisen D. Brush biopsy'saves lives'. Journal of the American Dental Association (1939). 2002 Jun;133(6):688-90.

17. Sciubba JJ. Improving detection of precancerous and cancerous oral lesions: computer-assisted analysis of the oral brush biopsy. The Journal of the American Dental Association. 1999 Oct 1;130(10):1445-57.
18. Potter TJ, Summerlin DJ, Campbell JH. Oral malignancies associated with negative transepithelial brush biopsy. Journal of oral and maxillofacial surgery. 2003 Jun 1;61(6):674-7
19. Mashberg A. Final evaluation of tolonium chloride rinse for screening of high-risk patients with asymptomatic squamous carcinoma. The Journal of the American Dental Association. 1983 Mar 1;106(3):319-23.
20. Lingen MW, Kalmar JR, Karrison T, Speight PM. Critical evaluation of diagnostic aids for the detection of oral cancer. Oral oncology. 2008 Jan 1;44(1):10-22.
21. Zhang L, Williams M, Poh CF, Laronde D, Epstein JB, Durham S, Nakamura H, Berean K, Hovan A, Le ND, Hislop G. Toluidine blue staining identifies high-risk primary oral premalignant lesions with poor outcome. Cancer research. 2005 Sep 1;65(17):8017-21.
22. Ram S, Siar CH. Chemiluminescence as a diagnostic aid in the detection of oral cancer and potentially malignant epithelial lesions. International journal of oral and maxillofacial surgery. 2005 Jul 1;34(5):521-7.
23. Farah CS, McCullough MJ. A pilot case control study on the efficacy of acetic acid wash and chemiluminescent illumination (ViziLite™) in the visualisation of oral mucosal white lesions. Oral oncology. 2007 Sep 1;43(8):820-4.
24. Schantz SP, Kolli V, Savage HE, Yu G, Shah JP, Harris DE, Katz A, Alfano RR, Huvos AG. In vivo native cellular fluorescence and histological characteristics of head and neck cancer. Clinical Cancer Research. 1998 May 1;4(5):1177-82.
25. .Patton LL, Epstein JB, Kerr AR. Adjunctive techniques for oral cancer examination and lesion diagnosis: a systematic review of the literature. The Journal of the American Dental Association. 2008 Jul 1;139(7):896-905.
26. Allison P, Locker D, Feine JS. The role of diagnostic delays in the prognosis of oral cancer: a review of the literature. Oral oncology. 1998 May 1;34(3):161-70.
27. Smart CR. Screening for cancer of the aerodigestive tract. Cancer. 1993 Aug 1;72(S3):1061-5.

Diagnosis and Treatment of Antibiotic Associated Diarrhoea

NeerajSurathu[1,]Gopinath P[2],S.Niveda[3]
Graduate Student[1], Assistant Professor [2],Department of Microbiology,
Tutor[3], Department of Public Health Dentistry
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

The term antibiotic associated diarrhoea (AAD) refers to a benign, self-limited diarrhoea following the use of antimicrobials. The prolonged use of multiple antibiotics, especially broad-spectrum group of antibiotics with poor intestinal absorption or high biliary excretion, induces a change in the composition and function of the intestinal flora thus resulting in AAD. Almost all antibiotics can result in the occurrence of AAD but few have a greater chance in doing so for example, Vancomycin, erythromycin and cephalosporins. The spectrum of manifestations in AAD ranges from normal diarrhoea to severe colitis. The main cause for AAD is the proliferation of Clostridium Difficile in intestinal flora. Apart from these bacteria, there are other bacteria which could cause AAD such as Staphylococcus aureus or Clostridium Perfringens. The occurrence of AAD is influenced by factors like nosocomial outbreaks, individual susceptibility and the pattern of prescription of the antibiotics. For the diagnosis of Clostridium Difficile associated diarrhoea stool samples can be taken for testing, colonoscopies can be taken to check for the presence of the bacteria and a blood test can be taken to test for leukocytosis which occurs during severe AAD.Diarrhoea is usually a symptom of a bowel infection [47]. Consumption of probiotics containing beneficial bacteria such as yeast, lactobacillus can help in curing AAD as they restore the healthy balance of the intestinal tract.

Key Words*:* *Antibiotic, Diarrhoea, Clostridium Difficile, Prophylaxis, Bacteria*

Introduction

The human body has a diverse assemblage of bacteria. Their symbiotic interaction have now been recognised as important predictors of human health. Abnormal communities are associated with complex diseases like obesity, irritable bowel syndrome. Administration of antibiotics disrupts the normal flora leading to diseases. Diarrhoea is a common adverse effect of antibiotic therapy and is characterised by mushy or watery stools. American Heart Association (AHA) and other committees of experts have proposed different guidelines and antibiotic prophylaxis regimens.

Organisms Which Cause AAD

There are various microorganisms which cause AAD like clostridium difficile, clostridium Perfringens, Staphylococcus Aureus, Klebsiellaoxytoca, salmonella species and candida species[2]. The most common organism being clostridium difficile. Infection with difficile is treatable.

Clostridium Difficile

Cl. difficile is an anaerobic, gram-positive spore forming bacillus first isolated in 1935 from faecal flora of healthy neonates. Spores of Cl. difficile may persist for many months in hospital wards and are particularly resistant to oxygen, desiccation and many disinfectants[3]. Pathogenic strains of Cl. difficile produce two major toxins: toxin A, an enterotoxin (308 kDa protein) and toxin B, a potent cytotoxin approximately 270 kDa protein. Both toxins are involved in the pathogenesis of Cl. difficile disease in humans. This toxigenic gram-positive bacillus is asymptomatically carried by 1%–3% of the human population, yet more prevalent in infants[4]. C.difficle is detected in 5% of stools or more healthy adults and upto 63% of patients without diarrhoea in hospital setup [5]. Asymptomatic carriers very rarely develop CDAD but they can contaminate the hospital environment [6,7]. This pathogen affects the non-hospitalisedpatientsfor whom the risk factors are antibiotic therapy, proton pump inhibitors, and also the use of receptor antagonists [8].

In the rectum and in the distal colon pseudomembranous colitis is fatal for 6%-30% cases [9]. These pseudo-membranes appear as yellow plaques which are easily visualized by colonoscopy. Almost all the antibiotics including vancomycin, Metronidazole and cancer therapy has been linked with CDAD and colitis [10, 11].

C.difficle releases two pathogenic strains with two major toxins: an Enterotoxin, toxin A and toxin B, a potent cytotoxin approximately with 270 kDA protein. Both these toxins are causative agents for C.difficle associated infections. Variety of genes in close proximity in bacterial genome encodes these toxins and they show only about 49% of structural homology[12]. Both the toxins activate cell-signaling molecules leading to production and also to the release of proinflammatory cytokines including interleukin-1, TNF- and interleukin-8 [13]. After internalization inside the host cell, toxin mediated glucosylation inactivates cellular rho proteins, that are essential for maintaining the actin cytoskeleton. This leads to the disorganization of colonocyte structure, disruption of protein synthesis, cell rounding and cell death [14].

Infants, mostly newborns, are colonized with C.difficle at the rate of 25-80% still most of them remain asymptomatic in spite the presence of toxin[15]. This is mostly due to immaturity of enterocytes with absence of toxin receptors expression[16]. Clinical presentation varies from asymptomatic carrier, diarrhoea, and colitis to PMC. Cl. difficile diarrhoea is associated with the passage of frequent loose stools along with mucus. Presence of visible blood is not much common. Leucocytosis is evident in stool. Anegative cytotoxicity test maybe performed, however, it does not completely rule out Cl. difficile because the cause of diarrhoea as 30% of patients may be missed, if toxin detection alone is employed [17]. Tests must be performed on fresh sample, because storage at ambient temperatures results in possible denaturation of toxin [18].

The standardised diagnostic test for Cl. difficile toxin in stool is the tissue culture cytotoxicity assay. It is 67 to 100% sensitive and 85-100% specific if it is performed correctly [19]. It can detect very little, like 10 pg of toxin B [15]. A variety of cell lines have been used by various workers, which include Vero, CHO, HEp2, MRC 526 etc. The main disadvantage is that it needs requires cell culture facility and requires incubation for 24-48 hrs. Counter current immuno electrophoresis has also been used in the past for direct detection of toxin B in stool [20]. Several commercial kits (EIAs) are available for detection of toxin A or both A and B in stool specimens.

Clostridium Perfringens

C. perfringens causes food poisoning and a different genotype has been implicated in causing AAD [21]. Clostridium perfringens type A is associated with 5-20% cases of antibiotic-associated diarrhoea (AAD) even though Clostridium difficile is implicated in the most severe cases[22]. Borriello et al, were the first to suggest that the association of C. perfringens with antibiotic treatment is causal rather than fortuitous [23]. They reported the prevalence of C. perfringens and its enterotoxin in 1.6% patients with Cl. difficile toxin positivity in 14.4% of their patients and 2/11 of them excreting both the organisms. Evidence supporting a causal role for C. perfringens in non-food borne diarrheal disease includes the presence of enterotoxin in the faeces of patients with AAD. Clostridium perfringens AAD is a distinct entity though it is unclear if antibiotic exposure primarily permits the proliferation of small numbers of resident C. perfringens strains or allows theiracquisition [24]. Clostridium difficile may account for only approximately 20% of all cases of AAD [24] and C. perfringens up to 15% [25].

Staphylococcus Aureus

Staphylococcus aureus was once thought to be a main cause of AAD, and was misdiagnosed for the clostridium difficile AAD infections [26].Certain studies provide evidence that enterotoxin-producing strains of methicillin-resistant S. aureus may cause nosocomial antibiotic-associated diarrhoea [27].

Klebsiellaoxytoca

K.oxytoca is considered an opportunistic pathogen and is now recognized as a clinically significant pathogen associated with nosocomial infections in hospitalized patients, including children and neonates [28][29][30].K. oxytoca also is purported to be an etiological agent of antibiotic-associated hemorrhagic colitis (AAHC) in adults and adolescents. AAHC patients develop clinical signs following antibiotic and/or anti-inflammatory therapy; these typically include bloody diarrhoea, severe abdominal cramping, and segmental hemorrhagic colitis as visualized by colonoscopy, most commonly in the ascending colon and the cecum [29][32][33].

Salmonella Species

S.newport was found which was multi drug resistant producing resistance to Ampicillin, Carbenecillin, and Methicillin etc was recently reported as a cause for AAD[34]. The patients who are taking antibiotic initially took this S.newport before taking the antibiotic and this S.newport selected the pathogenic resistance. Previously patients had even taking fluoroquinolone that has led to fluoroquinolone resistance enteric disease caused by Salmonella[35].Salmonella might even cause pseudo membranous colitis[36].

Candida Species

In patients those who are negative for Clostridium difficle, overgrowth of Candida species has been identified as the cause of AAD. Almost all of these patients respond to oral nystatin. [37].Lactose intolerance in rabbits is caused by candida which depresses the lactase activity in the rabbit's intestine, the net secretion of water, sodium; potassium in the ileum of rats is done by an endotoxin [38].

Diagnosis of the Main Causative Agents

Clostridium difficile causes mainly nosocomial enteric diseases that range from antibiotic-associated diarrhoea to pseudomembranous colitis (PMC) [39]. Various laboratory methods may be used to diagnose C. difficile associated diarrhoea and colitis (CADC), but the two main approaches currently available are based on detection of toxin A, toxin B, or both in stool specimens and detection of toxigenic strains after stool culture (toxigenic culture). For toxigenic culture, such an approach has been realized until now in only a few studies using both a toxin A enzyme immunoassay (EIA) and a cytotoxin assay [40-42].

The cytotoxin assay is considered the most sensitive method for detecting toxin B in stools, but it requires the use of cell culture and antitoxin and is not well standardized. This limits its use in many clinical laboratories. Commercially available rapid EIAs that detect both toxins have been shown to accurately detect toxins in stools and may represent a more practical method among diagnostic strategies that combine detection of toxins in stools with toxigenic culture[43].

An enzyme-linked immunoassay (ELISA) can be carried out for the detection of clostridium perfringens. Other non pathogenic species like Staphylococcus aureus species, Candida sp.

and Klebsiellapneumoniae can also be detected by ELISA. Pulsed-field gel electrophoresis (PFGE) can be used to detect S. aureus [44].

Treatment of AAD

Discontinuation of antibiotic therapy withdraws the offending agent but is often not appropriate if the indication for such therapy was correct. An alternative is to change the antibiotics to a low risk group for the induction of AAD (parenteral aminoglycosides, trimethoprim, rifampicin or a quinolone) [45].

The major form of intestinal disorders is the pseudomembranous colitis associated with Clostridium difficile which occurs in 10-20% of all AAD. In most cases of AAD discontinuation or replacement of the inciting antibiotic by another drug with lower AAD risk can be effective. For more severe cases involving C. difficile, the treatment of diarrhoea requires an antibiotic treatment, with glycopeptides (vancomycin) or metronidazole. Another approach to AAD treatment or prevention is based on the use of non-pathogenic living organisms, capable of re-establishing the equilibrium of the intestinal ecosystem. Several organisms have been used in treatment or prophylaxis of AAD such as selected strains of Lactobacillus acidophilus, L. bulgaricus, Bifidobacteriumlongum, and Enterococcus faecium[46].

Conclusion

A common complication of antibiotic use is the development of gastrointestinal disease. Comparative studies that use microbial ecology techniques to analyze temporally sampled patients and control individuals are promising approaches against AAD. Traditional culture-based methods continue to be the gold standard for disease diagnostics, but this approach can only detect organisms that are easy to isolate and have simple metabolic requirements. Since the vast majority of the human gut microbiota is currently non-cultivable, a nonculture-based approach may be more useful for the diagnosis and prediction of clinical outcomes. The key to addressing AAD is prompt diagnosis followed by effective treatment and institution of control measures. The prevalence of AAD in children is lower than that in adults. Majority suffer from mild illness. Further researches should be made on clinical treatments of AAD as it has been showing an increasing scale in the present scenario.

Corresponding Author

S.Niveda, Tutor, Department ofPublic Health Dentistry

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email: drnivedasubash@gmail.com, Telephone Number: +919952979464

References

1. Seth T. Walk and Vincent B. Young Emerging Insights into Antibiotic-Associated Diarrhoea and Clostridium difficile Infection through the Lens of Microbial Ecology. Interdisciplinary Perspectives on Infectious Diseases. 2008:7;56-58.
2. Fekety R, Kim KH, Brown D, Batts DH, Cudmore M, Silva J Jr.Epidemiology of antibiotic associated colitis: Isolation of Clostridium difficile from the hospital environment. Am J Med 1981; 70:906-908.
3. Penders J, Thijs C, Vink C, Stelma FF, Snijders B, Kummeling I, Van den Brandt PA, Stobberingh EE.Factors influencing the composition of the intestinal microbiota in early infancy. Pediatrics, 2006; 118(2):511–521.
4. Fekety R. Guidelines for the diagnosis and management of Clostridium difficile - associated diarrhoea and colitis. Am J Gastroenterol 1997; 92:739-750.
5. McFarland LV, Mulligan ME, Kwok RYY, Stamm WE. Nosocomial acquisition of Clostridium difficile infection. N Engl J Med 1989; 320:204-210.
6. Rabatsky-Ehr, K. Purviance, D. Mlynarski, P. Mshar, J. Hadler, and L. Sosa, Surveillance for community-associated Clostridium difficile—Connecticut. Morbidity and Mortality Weekly Report. 2006; 7(13):340–343.
7. J. Halsey.Current and future treatment modalities for Clostridium difficile-associated disease. American Journal of Health-System Pharmacy. 2008; 65(8):705–715.
8. E. J. Kuijper, B. Coignard, and P. Tüll. Emergence of Clostridium difficile-associated disease in North America and Europe. Clinical Microbiology and Infection. 2006; 12:2–18.
9. Bignardi GE, Risk factors for clostridial infection. J Hosp Infect. 1998; 40:1-15.
10. Hecht JR, Olinger EJ. Clostridium difficile colitis secondary to intravenous vancomycin. Dig Dis Sci 1989; 34:148-149.

11. VonEichel-Streiber C, Laufenberg-Feldmann R, Sartingen S, Schulze J, Sauerborn M. Cloning of Clostridium difficile toxin B gene and demonstration of N-terminal homology between toxin A and B. Med MicrobiolImmunol 1990;179:271-279.
12. Pathoulakis C. Pathogenesis of Clostridium difficile-associated diarrhoea. Eur J GastroenterolHepatol 1996; 8:1041-1047.
13. Pathoulakis C, Barone LM, Ely R, Faris B, Clark ME, Franzblau C, KaMont JT. Purification and properties of Clostridium difficile cytotoxin B. J BiolChem 1986;26:1316-1321.
14. Kelly CP, Pothoulakis, C, LaMont JT. Clostridium difficile colitis. N Engl J Med 1994; 330:257-262.
15. Eglow R, Pothoulakis C, Itzkowitz S, Israel EJ, O'Keane CJ, Gong D, Gao N, XuYL, Walker WA, La Mount JT. Diminished Clostridium difficile toxin A sensitivity in newborn rabbit ileum is associated with decreased toxin A receptor. J Clin Invest 1992; 90:822-829.
16. Peterson LR, Kelly PJ, Nordbrock HA. Role of culture and toxin detection in laboratory testing for diagnosis of Clostridium difficile-associated diarrhoea. Eur J ClinMicrobiol Infect Dis 1996; 15:330-336.
17. Brazier JS. The diagnosis of Clostridium difficile-associated disease. J AntimicrobChemother 1998; 41:29-40.
18. Gerding DN, Johnson S, Peterson LR, Mulligan ME, Silva J Jr. Clostridium difficile-associated diarrhoea and colitis. Infect Control HospEpidemiol 1995;16:459-477.
19. Ayyagari A, Sharma P, Mehta VS, Agarwal KC. Prevalence of Clostridium difficile in pseudomembranous colitis and antibiotic-associated colitis in North India. J Diarrhoel Dis Res 1986; 4:157-160.
20. Sparks SG, Carman RJ, Sarker MR, McClane BA. Genotyping of enterotoxigenic Clostridium perfringens fecal isolates associated with antibiotic associated diarrhoea and food poisoning in North America. J ClinMicrobiol 2001; 39:883-888.
21. Clostridium perfringens enterotoxin in antibiotic-associated diarrhea.Vaishnavi C1, Kaur S. Indian J PatholMicrobiol. 2008 Apr-Jun; 51(2):198-9.
22. Borriello SP, Larson HE, Welch AR, Barday F, Stringer MF, Bartholomew BA. Enterotoxigenic Clostridium perfringens: A possible cause of antibiotic associated diarrhoea. Lancet 1984; 1:305-7.

23. Modi N, Wilcox MH. Evidence for antibiotic induced Clostridium perfringens diarrhoea. J ClinPathol 2001; 54:748-51.
24. Asha NJ, Wilcox MH. Laboratory diagnosis of Clostridium perfringens antibiotic associated diarrhoea. J Med Microbiol 2002; 51:891-4.
25. Barlett JG. Antibiotic-associated diarrhoea. N Engl J Med 2002; 346:334-339.
26. Nosocomial antibiotic-associated diarrhea associated with enterotoxin-producing strains of methicillin-resistant Staphylococcus aureus.Boyce JM1, Havill NL. Am J Gastroenterol. 2005 Aug; 100(8):1828-34.
27. Savino F, Cordisco L, Tarasco V, Calabrese R, Palumeri E. Molecular identification of coliform bacteria from colicky breastfed infants. ActaPaediatr. 2009; 98:1582–1588.1651-2227.
28. Hoffmann KM, Deutschmann A, Weitzer C, Joainig M, Zechner E Antibiotic-associated hemorrhagic colitis caused by cytotoxin-producing Klebsiella oxytoca. Pediatrics. 2010; 125:960–963
29. Savino F, Cordisco L, Tarasco V, Locatelli E, Di Gioia D. Antagonistic effect of Lactobacillus strains against gas-producing coliforms isolated from colicky infants. BMC Microbiol. 2011; 157:1471-2180-11-157
30. Murphy MS Management of bloody diarrhea in children in primary care. BMJ 2008; 336:1010–1015.
31. Hogenauer C, Langner C, Beubler E, Lippe IT, Schicho RKlebsiella oxytoca as a causative organism of antibiotic-associated hemorrhagic colitis. N Engl J Med. 2006; 355:2418–2426.
32. Zollner-Schwetz I, Hogenauer C, Joainig M, Weberhofer P, Gorkiewicz G. Role of Klebsiella oxytoca in antibiotic-associated diarrhea. Clinical infectious diseases: an official publication of the Infectious Diseases Society of America2008; 47:74–78.
33. Holmberg SD, Osterholm MT, Senger KA, Cohen ML. Drug resistant Salmonella from animal fed antimicrobials. N Engl J Med 1984; 311:617-622.
34. Olsen SJ, DeBess EE, McGivern TE, Marano N, Eby T, Mauvais S, Balan VK, Zirnsterin G, Cieslak PR, Angulo FJ. A nosocomial outbreak of fluoroquinolone-resistant Salmonella infection. N Engl J Med 2001;344:1572-1579.
35. Hovius SE, Rietra PJ. Salmonella colitis clincially presenting as a pseudomembranous colitis. Neth J Surg 1982; 34:81-82.

36. Danna PL, Urban C, Belin E, Rahal JJ. Role of Candida in pathogenesis of antibiotic associated diarrhoea in elderly inpatients. Lancet 1991; 337:511-514.
37. Gupta TP, Ehrinpreis MN. Candida-associated diarrhoea in hospitalised patients. Gastroenterology 1990; 98:780-785.
38. Johnson S, Gerding D N. Clostridium difficile-associated diarrhea. Clin Infect Dis. 1998; 26:1027–1036.
39. al-Barrak A, Embil J, Dyck B, Olekson K, Nicoll D, Alfa M, Kabani A. An outbreak of toxin A negative, toxin B positive Clostridium difficile-associated diarrhea in a Canadian tertiary-care hospital. Can Commun Dis Rep. 1999;25:65–69
40. Cohen S H, Tang Y J, Hansen B, Silva J., Jr Isolation of a toxin B-deficient mutant strain ofClostridium difficile in a case of recurrent C. difficile-associated diarrhea. Clin Infect Dis. 1998; 26:410–412.
41. Limaye A P, Turgeon D K, Cookson B T, Fritsche T R. Pseudomembranous colitis caused by a toxin A− B+ strain of Clostridium difficile. J ClinMicrobiol. 2000; 38:1696–1697.
42. Arrow S A, Croese L, Bowman R A, Riley T V. Evaluation of three commercial enzyme immunoassay kits for detecting faecal Clostridium difficile toxins. J ClinPathol. 1994; 47:954–956.
43. Prévost G, Jaulhac B, Piémont Y. DNA fingerprinting by pulsed-field gel electrophoresis is more effective than ribotyping in distinguishing among methicillin-resistant Staphylococcus aureus isolates. J ClinMicrobiol. 1992; 30:967–973.
44. Spencer RC. The role of antimicrobial agents in the aetiology of Clostridium difficile associated disease. J AntimicrobChemother 1998; 41:21-27.
45. Santhosh kumar, Sneha. Knowledge and awareness regarding antibiotic prophylaxis for infective endocarditis among undergraduate dental students. Asian J. Of Pharmaceutical and clinical Res. 2016; 9(2)
46. Dhanalakshmi, GayatriDevi.RIndia Effect of Black Tea on Diarrhoea – A Research, Int. J. Pharm. Sci. Rev. Res. 44(1), May - June 2017; 15:56-58.

Diagnostic Aids Used in Caries Detection – A Review

Keerthika. S[1], M. N. Jayalakshmi Somasundaram[2], L. Leelavathi[3]

Graduate Student[1], Senior Lecturer[2], Department of Conservative Dentistry and Endodontics, Senior Lecturer[3], Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Dental caries, a progressive bacterial damage to teeth, is one of the most commondiseases that affects 95% of the population and is still a major cause of tooth loss. Caries diagnosis - as an intellectual process - is the determination of the presence and extent of a caries lesion. Furthermore the judgement of its activity is an integral part of diagnosis.The most common method of caries detection is visual-tactile. Other non-invasive techniques for detection of early caries have been developed and investigated such as Quantitative Light-induced Fluorescence (QLF), DIAGNOdent (DD), Fibre-optic Transillumination (FOTI) and Electrical Conductance (EC). Based on previous systematic reviews, the diagnosis of NCCLs might be more accurately achieved in combination of the visual method and the use of other methods such as electrical methods and QLF for monitoring purposes.The use of technologies as adjunct to clinical visual examination for caries diagnosis will facilitate preventive care in dentistry to lower treatment cost as well as reduce the cost and time for testing potential anticaries agents. It also helps to know about the state of the art methodologies for the detection and assessment of early carious lesions. Therefore, this articles is provides an overview of various diagnostic aids used in modern dentistry.

Key Words:*diagnostic methods, early detection, caries, Biofilm, treatment*

Introduction

Mostprevalentdiseaseofmankind isdental carieswhich manifests from a range of microscopic to macroscopic changes that affects the surrounding vicinity of the tooth surface. Conventional examination of caries detection is based on visual examination and tactile

sensation aided byradiograph.Dental caries is a dynamic disease process, where early lesions undergo many demineralizationandremineralisation cyclesbefore being identifiedclinically.[1]Moreover , remaining caries after cavity preparation continues to be a great concern for most of the clinicians , as most of them are not sure about complete removal of residual caries . Infact , success of restorative procedure depends on complete removal of unhealthy tooth tissues. During the early stages of the disease , caries progression is reversible and converted from a active to an inactive state . Therefore ,accurate diagnosis before cavitation aids in preventive treatment such asfluoride application, pit and fissure sealants, significantly improves dental healthand reducing the need for extensive drilling and filling.[2] Conventionaldiagnosis of caries with subjective interpretation, visual examination and tactile sensation , aided by radiographs allows clinician to make a dichotomous decision . Many studies based on these conventional methods often shows that these methods has low sensitivity and high specificity, that is a large number of lesions may be missed out.So it apparent that conventional method of caries detection does not fullfill the criteria of an ideal caries detection methods. This criteria includes detection of caries at an earlier age,provide valid prospective caries risk assessment for different age groups and determine present caries activity and monitor lesions behaviour over time.The shortcomings with conventional method, facilitates the need for newer and supplementary caries detection aids.[3]

Mechanism of Caries

Increased intake of sucrose, in the presence of a biofilm creates an acidic environment in the immediate vicinity of the tooth structure. This is followed by the sequence of random biochemical reactions resulting in demineralization and remineralization of the tooth. However, as long as the dynamic equilibrium of mineral content of the tooth is maintained, the entire sequence remains within boundary of a physiological process. In the presence of factors disturbing the haemostasis the process of deminerization persists and prevents the reminerization process. In due of course of time this shift in the balance leads to more changes in the structure of tooth structure. Thus physiological caries process bomes pathological caries disease.

Examination of Caries Lesion

An diagnostic tool should have the ability to detect the presence or absence of caries It should be sensitive and specific enough to produce maximum number of true negative results than false neagtives and false positives to the minimum. Accurate interpretation of caries depends on many factors including attributes of the clinician, observation, knowledge, discipline etc. A diagnostic aid should be designed in such a way, so that it culminates in reliable, reproducible results, with minimal inter/ intra observer variation. It should also provide same accuracy, in detecting signs of varying severity on various surfaces such as occlusal, proximal and root surface. Also, a scale of measurement of diagnostic threshold should be present in an ideal diagnostic tool .However detection of carious lesion, scale of measurement differs with clinician, researches, gadgets and techniques. Therefore the current trend in caries diagnosis if focused on

a) Formulating diagnostic criteria that are realistic and useful for the treatment decision.
b) Evolution of technical gadgets which aims at lowering the diagnostic threshold in order to detect the earliest mineral loss.
c) Quantification of test results should be reduced , so that inter and intra observer variation associated with qualitative assessment
d) Dichotomous, nominal, ordinal scale used for caries detection should be shifted to a numerical scale therby monitoring the progress of the lesion over time.

Visual and Tactile Examination

It is accomplished using mouth mirror, sharp probe, 3in 1 syringe. It also requires good lightening, clean and dry tooth surface.This examination is primarily based on subjective interpretation of surface characteristics such as integrity, texture, translucency,location and colour[3].Presence of plaque, debris and other local factors should be checked to assess the texture of the enamel. Gingival and periodontal status is also assessed by the presence or absence of bleeding.[4] The enamel decalcification index was also used to determine the number of white spot lesions. But this method of examination is not as effective as advanced methods because of the spread of caries from one site to other site which leads to further spread of the disease. This method is widely used in clinics for detecting carious lesions on the entire surface. [5] But this method is not effective in detecting caries in the relatively advanced stage,involving one third or more of the thickness.[6] Major drawback in this

conventional method of caries detection is use of various diagnostic criteria used by various authors. There have also been controversies whether to probe or not. However from many studies it has been resurged as an valuable adjunt in the visual examination of caries. However probing is limited to removal of plaque from the surface of an incipient lesion, thereby enhancing visibility. Probing is also used to assess the surface texture of a lesion. In this method blunt probe is manipulated at an anle of 20- 40 degree angle to the surface of the tooth. Blunt probing is recommended against a sharp probr acting perpendicular to the tooth surface

Nyvad's System

Reliable system for assessment of cavitated and noncavitated lesion is Nyvaad system. In this method, accumulation of biofilm is used as an indicator of caries activity and also sharp dental probe is used to assess surface roughness .In modern dentistry, Nyvaad system is modified with the adoption of ball ended probe , which is gently run across the surface in order to assess its texture and also to remove biofilm . However important aspect of caries detection is that surface must be dry because saliva can mask differences in the reflection of light between carious and healthy tooth structure , therby hindering the observation of changes in color and brightness on the enamel surface.[7]

Radiograph Techniques

Radiographic techniques are based on the fact that as caries progress there will be decrease in the mineral content of enamel and dentine , results in decrease in the attenuation of X ray beam as it passess through the teeth . This feature is being recorded in the image receptor as increase in radiographic density.[7]

Conventional Radiography

Bitewing radiography came into practise in 1925, play a role in clinical examination of proximal and occlusal caries lesion in dentine. This method has more sensitivity and provides enhanced estimation of lesion depth than the visual inspection.This method is preferred over periapical radiograph mainly because of the superior imaging geometry for visualizing caries. Bite wing requires 40 -60% of tooth calcification is required to produce the radiographic

image.Bitewing radiograph has a tendency to make false positive results due to Mack band effect. Mach band effect is a phenomenon in which there is enhancement of contrast dark and relatively light area. This ultimately results in dark band being sharply demarcated radiolucency in the dentino enamel junction.Another effect called cervical burnout is wrongly interpreted as cervical caries, when a collar or wedge shaped radiolucency occurs between bone height and cement enamel junction, there occurs an optical illusion phenomenon.[7] Due to this, there is variability in the penetration of X- rays at the cervical region of the tooth and also in the regions above and below it .This results in the dark shadow in the radiograph due to lower absorption of photons in the cervical region of tooth. This necessitates the fact that the radiographic images should be interrupted with caution, requiring constant retraining, updating, experience and also should be accompanied other supplemental caries detection aids .[8]

Digital Radiography

Digital radiography has more advantages than conventional radiography.This includes image acquisition,since the image is displayed immediately after exposure and no processing has to performed.Reduction in radiation dose is another advantage of this method.It is comprised of number of pixels and produces instant and constant images. Each pixel has a value in the range betwee 0 and 255 , with 0 representing back and 255 representing white . The values in between represents shades of grey .Concerning assessment of small proximal caries digital radiography has low specificity and sensitivity compared to conventional methods .No dark room needed.Reduced dose of radiation.Eliminates hazards of film development. Capable of tele transmission.Major disadvantages include high cost and reduced life expectancy of chip.Digital radiography has replacing traditional analogue or screen film systems. It uses the principle of conversion of x ray energy into digital signals using scanning laser stimulating illuminescence.[9]

Digital Image Enhancement

It is a more advanced image analysis tools.It is used to detect small differences between subsequent radiograph that would have remaind unobserved , because of over projection of anatomical structures[7].This procedure is based on the principle that the two radiographic

images obtained at different time intervals, with the same projection geometry, which is alligned by using specific software.Resolution of unedged radiograph is lower when compared to enhanced radiograph .Contrast of the image can be enhanced by a mathematical rule which is decided by algorithm/ filter.They are not practically used because they are very time-consuming.[10]

Digital Substraction Radiography

In this method ,a digital bitewing radiograph is taken and later a second radiograph of exactly the same region is produced with identical exposure time, tube current, and voltage. By subtracting,gray values for each coordinate of the firstradiograph from equivalent coordinate of second, a subtraction image is obtained .If there is no changes , the result of subtraction is zero .Nonzero result will be obtained in case of onset or progression of demineralization. It has been used in the assessment of progression , arrest ,or regression of of caries lesion. Due to difficulty of image visualisation, this method is not routinely applied in clinical detection of caries.[11]

Tuned Aperture Computed Tomography

This method is used to visualize radiographic section through teeth. Then the sectioned slices can be viewed for the presence of radiolucency.It is used to detect small caries and even secondary caries.But this method is technique sensitive.Once the exposure begins , the tube and film move in opposite direction through a mechanical linkage.Image of objects in the focal plane remain in fixed positions on radiographic films and are clearly imaged[12] .Images of objects located outside focal plane are continuouslybeing changing their positions on the film .As a result , images of these objects are blurred beyond recognition by motion unsharpness.Advantage of this system over other system is that,images produced less superimposition of anatomic noise over the area of interest.Radiation of Tuned Aperture computer Tomography is 1-2 times less than that of a conventional periapical xrayfilm . So that the total exposure dose is divided amongst the series of images taken . It is most suitable for detecting vertical root fractures .

Enhanced Visible Techniques

i) Fibre Optic Transillumination (FOTI)

The differential transmission of light through healthy tooth structure as compared to carious tooth structure can be detected by using quantitative fibre optic transmission and digital image fibre optic trans illumination .Operator is able to use a more focused and higher intensity light beam instead of an operatory light , which increases the potential to detect smaller carious lesions .[11] This system has a built-in CCD camera to allow for image capture of the tooth . By illuminating teeth with this system . Areas of demineralized enamel or dentin scatter light and incipient caries appear darker in the resultant image . In this method photon energy is converted to electric energy by using receptors with photon cells .[12] This is then transmitted to a video processor and converted into color value and displayed on video monitor .In early days , this method is used for the detection of approximal lesions in the anterior teeth but later its use was extended to detect proximal lesions in the posterior teeth as well . [13]

I) Digital Imaging Fiber Optic Transillumination

Short coming perceived in FOTI lead to the development of (DIFOTI) . In this method images of the teeth are obtained by using visible light via fiber optic transillumination . These images are acquired by digital electronic CCD camera . This method is used to enhance the contrast between sound and carious tissues and to quantify features of incipient , frank, and secondary lesions on occlusal , approximal and smooth surface and also changes in the coronal tooth structure such as tooth fractures and fluorosis. It has high specificity when compared to radiography .[14]

Quantitative Laser Induced Fluorescence

It provides a fluorescent image of a tooth surface within yellow- green spectrum of visible light that is used to quantify mineral loss and size of the lesion . It is also used to detect enamel caries in visually inaccessible areas . This system consists of a micro video ccd camera ,measurement probe, control unit , and computer fitted with a frame grabber .The control unit consists of an illumination device and imaging electronics . Light source is a special arc lamp based on xenon technology .[15] Fluorescent image is recorded with yellow

transmitting filter positioned in front of the CCD sensor.Appearence of red fluorescence indicates leaking around restorations and sealants . It is emitted by porphyrins metabolised by bacteria dental bio film , calculus, or an infective carious lesion . In this method first lesion is detected by the examiner and subsequent capturing the image of the lesion .Quantitative analysis of the image is done . Finally long term monitoring of the lesion is done . In a study done by Beerens MW et al , to compare discrimatory power of caries assessment using QLF and digital radiography after de bonding of orthodontic appliance ,it was shown that standardized oral photographs were equally useful to detect changes in caries severity and outperformed ICDAS.[16] In a study done by Mariana et al to review the application of QLF for quantitative assessment of early enamel lesions showed that , because of this property associated with high reliability values and a video repositioning software , which facilitates the acquisition of identical images on different occasion ; this device is an excellent method for monitoring enamel caries lesions to assess whether preventive measures are able to arrest or remineralize the lesion.[17] Advantage of this method is that image can be stored and can be used to motivate patients to seek healthcare and also to prevent dental disease through routine preventive care . A study done by Eggertsson et an demonstrates various applications of quantitative laser fluorescence which includes dectection of primary, secondary , root caries on smooth and occlusal surface in both primary and permanent teeth; detection of remineriliza tion around orthodontic appliance ; monitoring demineriliation and remineriliation caries lesion ; quantifying dental plaque ; erosion and fluorosis ; detection of extrinsic stains .[18]

Laser Induced Fluorescence

i) **Diagnodent**:

Diagnosis of occlusal caries in adjunct to visual and radiographic examination .DIAGNOdent system is a part of exciting new generation of dental equipment . It uses infrared laser fluorescence f 655 nm for the detection of occlusal and smooth surface caries . It uses a simple laser diode to compare the reflection wavelength against a well-known healthy baseline to uncover decay. Carious tooth structure exhibits fluoresce proportionate to the degree of caries , resulting in elevated scale readings on the display . [19] The unit has a fiber- optic cable that transmits light source to a handpiece that contains a fiber- optic eye in the tip . First , the laser diode is aimed at the healthy enamel tooth structure to obtain a

benchmark reading . After calibration , it is moved to inspect all the surfaces of the teeth , shining the laser at 2.5 mm into all suspected areas . As the laser pulses into grooves , fissures , and crackes, it redirects fluorescence light with particular wavelength . This is because light is absorbed by the organic and inorganic components of the tooth which induce infrared fluorescence . This fluorescence is collected at the top of handpiece and transmitted back to the DIAGNOdentunit . Light is measured by receptors , converted into an acoustic signal , and evaluated electronically to reveal values between 0 and 99.The intensity of the fluorescence depends upon the wavelength of the light as well as the structure and condition of hard dentinal tissues . Factors such as the presence of bacterial plaque , dental prophylactic pastes , fissure sealants , and composite resin materials give false positive readings with this instrument . In a study done by, Ava valisichani et al to evaluate diagnostic value of diagnodent in detecting caries under composite restoration it was reported that diagnodent has higher positive and negative prediction value compared to radiograph . These findings along with higher sensitivity and specificity has proven that diagnodent is most accurate in detecting secondary caries under primary molar restoration, compared to radiograph.[20]Diagnodent pen is the recent modification of diagnodent which has the tip of 0.4mm therby facilitating easy placement in approximal areas for the detection of incipient proximal caries . Diagnodent may show errors in the presence of stains , biofilm and filligs but still has advantage in monitoring the progression or regression of incipint lesion over time .

Ultra Sound Caries Detection

Use of ultrasound to detect dental caries has been proposed for the past 30 years , but the technique has received renewed interest particularly in the past 10 years. It was introduced for detecting early carious lesions on smooth surface .Demirelizationof natural enamel is assessed by ultrasound pulse- echo technique .[21] It is observed that there is a definite correlation between the mineral content of the body of the lesion and the relative echo amplitude changes . Ultrasound makes the use of sound waves with frequency . They are longitudinal or pressure waves which travel through gasses , liquids , and solids . Ultrasound interacts differently with different tissues .They have a frequency of > 20,000 Hz and have all the properties of waves , in that they may be scattered, refracted , or absorbed . The

relative ability of a medium to reflecting sounds on its mechanical properties such as elasticity , density , and wavelength of sound . Amount of sound detected provides information about the structure of reflecting interface , whereas the time taken for sound to be reflecting provides information about the position of the reflecting interface . Sound waves produced as a result of minute changes in crystal dimension may be omitted continually , as burst of waves or as a single pulse .

Mechanism of Action

For sound waves to reach the tooth , they have to travel through a coupling medium or an agent which has acoustic impedance . Various acoustic coupling agents have been used such as mercury ,aluminum rods , water , and glycerine .An ultrasonic probe is used which sends and receives longitudinal waves to and from the surface of the tooth . [22] Initial white spot lesions produce no or weak surface echoes , whereas sites with visible cavitation produce echoes with substantially higher amplitude .[23]This method if improved can be a realistic alternative to radiographic diagnosis of caries on approximal surfaces . It is also more sensitive than visual-tactile method .

Conclusion

The devices vary in their modes of action as well as their capability as caries diagnostic aids . Difference in caries presentation and behaviour in different anatomical sites make it unlikely that any one of the diagnostic aids will have adequate sensitivity and specificity of detection of carious lesions for all sites ; a combination of diagnostic tools will help us diagnose lesions earlier and detect failing restorations as early as possible .

Corresponding Author

L. Leelavathi,

Senior Lecturer, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai - 600 077, Tamil Nadu, India

Mobile Number: 9443702882,

Email id: karleela81@gmail.com

Reference

1. R. Effects of dental probing on occlusal surfaces: A scanning electron microscopy evaluation. Caries Res 2007;41:43-8
2. NyvadB ,Machiulskienev,reliability of caries diagnostic system in differentiating between active and inactive caries , caries res 1999; 33;252-60.
3. van der Stelt, P.F. (2008). Better imaging: the advantages of digital radiography. Journal of the American Dental Association, Vol.139, No. Suppl, (June), pp. 7S-13S, ISSN 0002-8177
4. Varma, S.; Banerjee, A. & Bartlett, D. (2008). An in vivo investigation of associations between saliva properties, caries prevalence and potential lesion activity in an adult UK.
5. Wenzel, A. Bitewing and digital bitewing radiography for detection of caries lesions. Journal of Dental Research. 2004;Vol.83, No. Spec No C, pp. C72-C75, ISSN 0022-0345.
6. Hekmatian, E.; Sharif, S. &Khodaian, N. Literature review: digital subtraction radiography in dentistry. Dental Research Journal. 2005;Vol.2, No.2, pp. 1-8, ISSN 1735-3327.
7. Neuhaus, K.W.; Rodrigues, J.A.; Hug, I.; Stich, H. & Lussi, A. (2010). Performance of laser fluorescence devices, visual and radiographic examination for the detection of occlusal caries in primary molars. Clinical Oral Investigations, May 27 (Epub ahead of print).
8. Baelum V, Hintze H, Wenzel A, Danielsen B, Nyvad B: Implications of caries ;diagnostic strategies for clinical management decisions. Community;Dentistry and Oral Epidemiology 2012, 40(3):257-266.
9. Ricketts DN, Kidd EA, Liepins PJ, Wilson RF: Histological validation of ;electrical resistance measurements in the diagnosis of occlusal caries.;Caries research 1996, 30(2):148-155.

10. Longbottom C, Huysmans MC: Electrical measurements for use in caries ;clinical trials. Journal of Dental Research 2004, 83(Spec No C):C76-79.

11. de Josselin de Jong E, Sundstrom F, Westerling H, Tranaeus S, ten Bosch JJ, Angmar-Mansson B. A new method for in vivo quantification of changes in initial enamel caries with laser fluorescence. Caries Research 1995;29(1):2–7.

12. 19. Ando M, Hall AF, Eckert GJ, Schemehorn BR, Analoui M, Stookey GK. Relative ability of laser fluorescence techniques to quantitate early mineral loss in vitro. Caries Research 1997;31(2):125–31.

13. van der Veen MH, de Josselin de Jong E. Application of quantitative light-induced fluorescence for assessing early caries lesions. Monography in Oral Science 2000;17: 144–62.

14. 21. Angmar-Mansson B, ten Bosch JJ. Quantitative light-induced fluorescence (QLF): a method for assessment of incipient caries lesions. Dentomaxillofac Radiology 2001;30(6):298–307.

15. van der Veen MH, de Josselin de Jong E. Application of quantitative light-induced fluorescence for assessing early caries lesions. Monography in Oral Science 2000;17: 144–62.

16. 21. Angmar-Mansson B, ten Bosch JJ. Quantitative light-induced fluorescence (QLF): a method for assessment of incipient caries lesions. Dentomaxillofac Radiology 2001;30(6):298–307.

17. Hall A, Girkin JM. A review of potential new diagnostic modalities for carious lesions. J Dent Res 2004;83:C89-94.

18. KunishJ ,inter/ intra reproducibility of the new DIAGNOdent Pen on occlusal sites. J Dent

19. Zandona AF, Zero DT. Diagnostic tools for early caries detection. J Am Dent Assoc 2006;137:1675-84.

20. Hall A, Girkin JM. A review of potential new diagnostic modalities for carious Lesions. J Dent Res 2004;83:C89-94.

21. Lussi A, Angmar-Mansson B. Additional diagnostic measures. In: Fejerskocv O, Kidd E, Nyvad B, Baelum V, editors. Dental caries. The disease and its clinical management. 2nd ed. Blackwell Munksgaard Ltd; 2008. p. 90-101.

22)Ando M, Analoui M, Schemehorn BR, Eggertsson H, Stookey GK. In vitro comparison of three caries diagnosis techniques. Caries Res 1999; 33(3):298.

23)Twetman S, Fontana M. Patient caries risk assessment. In: Pitts NB, (editor). Detection, assessment, diagnosis and monitoring of caries. Monogr Oral Sci. Basel, Krager; 2009. p. 91-101.

Distalization Method in Correction of Class II Malocclusion-A Review

Jaseera Banu.K [1], Saravana Pandian[2], Padma Ariga[3]
Graduate Student[1] ,Reader[2], Department of orthodontics
Professor[3], Department of Prosthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Class II malocclusion is one of the most difficult malocclusions to treat, and stationary anchorage is one of the main factors determining the success of the treatment. Maxillary molar distalization, a common treatment modality used to correct Class II malocclusions, is particularly indicated when maxillary skeletal or dentoalveolar protrusion is present. Using intraoral appliances, maxillary molars can routinely be moved distally with little or no patient cooperation. One factor that influences the movement rate is the type of movement and another factor is the timing of treatment.The correction of Class II malocclusions has been hampered by the use of appliances which require the patient to co-operate with headgear, elastics, or the wearing of a removable appliance. 'Non-compliance therapy' involves the use of appliances which minimize the need for such co-operation and attempt to maximize the predictability of results. The treatment effects of extra oral forces in the correction of Class II malocclusions are well known. Although headgear use is usually inevitable in subjects with maxillary excess, co-operation problems seem to be a challenge for orthodontist.The present article explains about different modality of distalization methods in correction of class II malocclusion.

Key Words:*Malocclusion, distalization, removable appliance, head gear, anchorage.*

Introduction

Orthodontic problems can be classified either as skeletal problems or dental problems. Skeletal problems can be originated from skeletal deviations and treated with orthopaedic appliances, fixed mechanics, or surgery. Dental problems are classified as dental class I malocclusion, dental class II malocclusion, and dental class III malocclusion [1]. Dental class II malocclusion is generally caused by the early loss of the upper deciduous second molar. After early tooth loss, upper first permanent molars erupt more mesially than their normal locations or drift mesially [2]. The traditional approach to distalize molars, especially in the maxilla, is extra oral traction[3].

Patient's cooperation is a determinant factor for successful orthodontic treatment, thus, protocols that require minimal collaboration are of great value in orthodontic practice. Intraoral distalizersfulfil this function, i.e., they correct Class II malocclusion without entirely depending on the patient to achieve satisfactory results by the end of treatment [4]. The success of molar distalization has been reported to depend on two main factors: a] the type of movement and [b] the timing of treatment. It has been noted that distalization of the first molar occurs by tipping rather than by bodily movement [5].

Class II malocclusion is one of the most common problems encountered in orthodontic practice today. Advances in mechanotherapy and changes in treatment concepts and philosophies now minimize the need for extractions in mild to moderate discrepancies [6]. Non-extraction treatment of class II malocclusions, which are characterized by the absence of skeletal involvement, frequently requires distal driving of maxillary molars into a class I relationship. Several intraoral distalizing appliances have been introduced to eliminate the need for patient cooperation. The distal jet is a palatally placed, intra-arch maxillary molar distalization appliance [7].

Nonextraction treatment of Angle Class II malocclusion usually requires distalization of maxillary molars. Beginning in the 1980s, intraoral appliances, such as repelling magnets, 1superelastic NiTi coil springs, pendulum, Jones jig and distal jet, have been introduced to distalize molars with minimal patient compliance. Intraoral distalization appliances have been designed to deliver a continuous reciprocal force on the maxillary first molars. Any action to move molars distally produces a mesial reaction force on the anchoring teeth. As a consequence, if the premolars or incisors [or both] are the anchoring teeth, they move mesially, the incisors protrude, and overjet increases [8].However, this effect is in

contradiction with the main objective of Class II treatment. Furthermore, distalized molars are questionable anchors for the retraction of premolars and incisors, despite attempts [headgears, Nance appliance etc] that have been made to maintain them in their new positions.

The creation and use of intramaxillary intraoral appliances for molar distalization in Class II malocclusion have been made possible through advancements in biomechanics and technology and materials that have allowed the delivery of light and constant forces over a wide range of deactivation. The application of force in these appliances can occur from the buccal region, the palatal region, or both [9]. The forces applied can involve friction [sliding mechanics], as with the use of nickel-titanium [NiTi] coil springs, or they can be friction free, as when the pendulum appliance is used. When the efficiency of these different categories of appliances is compared, it is not clear which ones are associated with the largest molar distalization and which produce the smallest quantity of undesirable adverse effects, such as loss of anchorage and molar tipping [10].The aim of the study is to review the distalization method in correction of class II malocclusion.

Anchorage

Anchorage, defined as a resistance to unwanted tooth movement, is a prerequisite for the orthodontic treatment of dental and skeletal malocclusions. Its role in orthodontic treatment was appreciated early on, as prominent orthodontists such as Gunnell, Desirabode, and Angle realized the limitations of moving teeth against other teeth used for anchorage, introducing ideas such as the use of occipital, stationary, and occlusal anchorage [11].Controlling anchorage helps to avoid undesirable tooth movements. However, even a small reactive force can cause undesirable movements; it is important to have absolute anchorage to avoid them. Absolute or infinite anchorage is defined as no movement of the anchorage unit [zero anchorage loss] as a consequence to the reaction forces applied to move teeth [12]. Such an anchorage can only be obtained by using ankylosed teeth or dental implants as anchors, both relying on bone to inhibit movement. Anchorage provided by devices, such as implants or miniscrew implants fixed to bone, may be obtained by enhancing the support to the reactive unit [indirect anchorage] or by fixing the anchor units [direct anchorage], thus facilitating skeletal anchorage [13].

Types of Anchorage

Skeletal anchorage devices can be classified into 2 main categories, based on their origin.The first category has its origins in osseointegrated dental implants and includes the orthodontic mini-implants, the retromolar implants, and the palatal implants. The second category finds its origin in the surgical mini-implants, such as the one used by Creekmore and Eklund and those described later by Kanomiand Costa et al [14].The main differences between the 2 categories are that devices of the second category are smaller in diameter, have smooth surfaces, and are designed to be loaded shortly after insertion. In a similar manner, Cope classified the current available methods of skeletal anchorage as either biocompatible or biologic in nature [15]. The biologic group included ankylosed and dilacerated teeth, whereas the biocompatible group included temporary anchorage devices. He further subclassified both groups based on the manner in which they are attached to bone into biochemical [osseointegrated] or mechanical [16].

Various Methods of Distalization

Distalization procedures are aimed at moving the molars in a distal direction so as to gain space. Distal movement of maxillary molars in individuals with class2 malocclusion and moderate space deficiency in upper arch can be achieved by extra oral traction or by a combination of extra oral traction and removable appliance [17].It can be brought about by either extra oral or intra oral methods.

Extra oral methods include usage of head gears whereas intra oral methods includes open coil springs,intra oral magnets,Sagittal appliance and pendulum appliance. The treatment of Class II malocclusion without extractions usually requires patient compliance, either for bodily distalization of molars [headgear] or for distalization of molar roots after use of intraoral distalizing appliances [pendulum].Intraoral distalization through intraoral fixed appliances is only the first phase of treatment that will be finalized with fixed corrective mechanics [18].

In recent years, clinical interest has focused in particular on the different pendulum appliances. The aim of molar distalization should be maximum bodily tooth movement, on the one hand to minimize the risk of root resorption and on the other to permit subsequent bodily retraction of the anterior dentition. The standard pendulum appliance introduced by Hilgers. However, gives rise to the appliance-specific fundamental problem that once the

activated pendulum springs have been inserted into the palatal sheaths of the molar bands, the molars are moved in an arc on pendulum like radii. The potential consequences are palatal movements of the molars and tipping of the dental crowns [19].

HEAD GEAR-as an Extra Oral Distalizer

Extra oral headgear traction, which has been used since the 1800s, is the oldest and most commonly used method to correct a Class II buccal segment relationship by restricting forward growth of the maxilla and/or distalizing the maxillary molars [20]. Despite its efficacy, headgear treatment is dependent on patient co-operation. Furthermore, the difficulty of use and aesthetic concerns make patient co-operation difficult to maintain throughout treatment [21]. In comparison with headgear, intraoral molar distalization methods are easier to use, more socially acceptable for patients, and the distalization time is shorter because they apply continuous forces. However, they have several disadvantages such as mesialization of the maxillary premolars, protrusion of the maxillary incisors, an increase in overjet, and anchorage loss [22, 23].

Fig 1 :Headgear

Pendulum Appliance

Distalization of maxillary teeth with the pendulum and presumably increasing mandibulargrowthwiththeHerbstappliance are two treatment options for correction of class II malocclusion[24].The investigators, however, found no differences in mandibular growth between groups, although they noted slightly greater increases in lower anterior facial height

[LAFH] in the pendulum group. Treatment success with both appliances occurred primarily from dentoalveolarchanges [25].In 2008, Angelieri et al proposed that the pendulum appliance produced only dentoalveolar effects, while the CHG appliance, which restricted maxillary forward displacement, improved the skeletal maxillomandibular relationship. [26]

The pendulum appliance consists of an anterior Nance portion with an expansion screw and two posteriorly extending TMA coil springs that were recurred at the end where they fit into lingual sheath. Using a composite resin and auxiliary wires, the appliance was bonded to the maxillary premolars or molars, avoiding occlusal interferences [27].The pendulum springs were activated 45 degree in the centre of the helices on the Sagittal plane with an initial force of 200-250 g. Depending on the molar movement required, activation was repeated intra orally once or twice during the treatment [28].

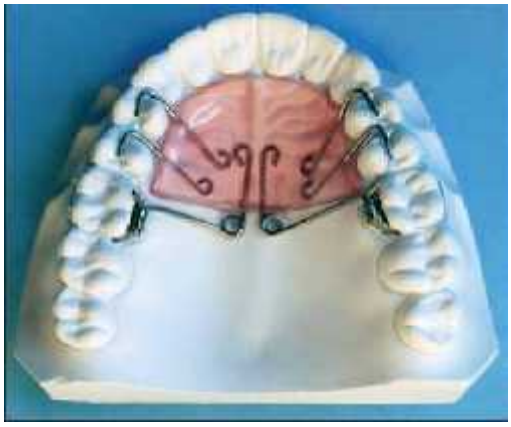


Fig 2:Pendulum Appliance

Nickel-Titanium Coil Springs

Nickel titanium coil springs have been used in conjugate with various non compliance appliances to produce rapid maxillary molar movements, because it was found that they have greater spring back and super elastic properties than stainless steel coils. Furthermore, the most important reason for their implementation in noncompliance distalization devices is their ability to exert a very long range of constant, light and continuous force [29].

Open coil springs are commonly used in orthodontic practice; but there have been few experimental studies on their clinical effects. Chaconas et al. [1984] investigated the effects of wire thickness, length and radius of open coil springs on the forces produced. It was found that more linear force was produced in open coil springs with a large lumen and in order to obtain an optimum force, open coil springs had to be compressed by about a third of its original length, producing a force value of between 270 and 540 g [30].

In 1988, Miura et al. [1988] compared the mechanical properties of Japanese nickel-titanium and stainless steel coil springs, in both closed and open types. They found that Japanese nickel-titanium coil springs exhibited superior spring-back and super elastic properties. Additionally, it was shown that the load value of super elastic activity could be effectively controlled by changing the diameter of the wire, the size of the lumen, the Martensitic transformation temperature, and the pitch of the open coil spring. The most important characteristic of Japanese nickel-titanium alloy coil springs was the ability to exert a very long range of constant, light, and continuous force [31]. Gianelly et al. [1991], obtained an average of 1–1·5 mm molar distalization in one month by 8–10 mm activation of super elastic nickel-titanium coil springs. To maintain anchorage, a modified Nance appliance was cemented to the upper first premolars [32]. An additional means of anchorage reinforcement involved the incorporation of uprighting springs to tip the crowns of these teeth distally and as a result, effective molar distalization was obtained. In cases with second molars in total occlusion, the use of Class II elastics was recommended to avoid anchorage loss [33]. Fraunhofer et al. [1993] compared nickel-titanium coil springs with stainless steel coil springs. These authors concluded that the light and continuous forces necessary for optimum tooth movement could be produced by means of nickel-titanium coil springs [34].

Fig 3: Nickel Titanium coil spring

Distal Jet Appliance

The distal jet is one of the more commonly used “noncompliance appliances” for molar distalization. It is a fixed lingual appliance that can produce bilateral or unilateral molar distalization in permanent as well as mixed dentition. The appliance consists of a bilateral piston and tube system with the tube embedded in an acrylic button in the palate, supported by attachments on the maxillary second premolars [35]. A distal jet appliance consists of an

acrylic Nance button and stainless steel wires. The appliance can easily be converted to a Nance appliance when the distalization is complete. A study done by karamanet al, concluded that the implant supported modified distal jet appliance is an effective and reliable method for distalizing maxillary molars, and this treatment requires minimal patient compliance [36].

Among the aforementioned appliances, the distal jet, a lingual distalization appliance, is said to feature several distinct advantages. The maxillary molars are distalized with less distal tipping and without the lingual movement that occurs with the pendulum and the distal jet can be easily converted into a Nance holding arch to maintain the distalized molar position [37].

The distal jet with full fixed brackets produces less molar tipping than other intraoral alternatives such as the pendulum, Jones jig, Greenfield molar distalizing appliance and the sagittal appliance. These studies also suggest that mild increases in lower anterior facial height are related to the amount of molar tipping generated during distalization. Perhaps appliances with better control of molar inclination [eg, distal jet] might diminish the risk, however small, of clinically significant vertical change [38].

To reduce anchorage loss, Gianelly et al recommended that molar distalization be performed before the eruption of the maxillary second molars. Several investigators, however, have found no difference in distalization and anchor- age loss for the pendulum, Jones jig, and distal jet when second molars were unerupted or erupted [39].Bussick and McNamara,Huerter, and Chiu discovered a mild increase in the mandibular plane angle during distalization when second molars were erupted. In contrast, Huerter described less anchorage loss when the second molars were completely erupted. The effect of the eruption status of second molars on distalization is inconclusive [40].

Fig 4: Distal Jet Appliance

CarriereDistalizer

The CarriereDistalizer is a simple-to-use device that is inconspicuous enough to promote patient acceptance and compliance. Loss of anchorage has not been a significant problem, although several techniques, including mini screws, are available for anchoring the Class II elastics[41].The CarriereDistalizer is designed to create a Class I molar and canine relationship. This appliance can be used as a first phase treatment in Class II cases with fully erupted first molars [42].

Fig 5: Carriere Distalizer

First Class Appliance

The first class appliance [FCA; Leone, Firenze, Italy] was proposed as an intraoral device that allows rapid distal movement of the maxillary molars [43]. A study done by Fortini et al proved that FCA produced rapid molar distalization: bilateral Class II molar relationship was corrected in 2.4 months on average. He also suggest that no significant changes in sagittal or vertical skeletal relationships were observed [44].Another study done by Papadopoulos et al, suggested that even though it shows a distalization without distal rotation and extrusion of the molars and with minimal, non significant proclination of the incisors it shows side effects similar to those produced with other noncompliance distalization appliances [45].

Fig 6: First Class Appliance

Keles Slider

In 2000, Keles and Sayinsu developed the intraoral bodily molar distalizer [IBMD] for molar distalization. Their results showed that the molars distalized without tipping; however, anchorage loss also occurred [46].In 2002 Keles et al, in his study provedthat theKeles Slider is an effective appliance to bodilydistalize molars. Class I molar relationships can be established in a short period of time, and there is little anchorage loss in comparison with the other intraoral distalization mechanics. The appliance is effective in deep bite correction. TheKeles Slider can also be used for correction of unilateral Class II molar relationships [47].

Fig 7: Keles Slider

Skeletal Anchorage System

Introduction of skeletal anchorage in orthodontics not only has allowed the simplification of many procedures conventionally employed for the control of anchorage, but also has reduced the undesirable effects of many appliances [48]. Moreover, miniscrews present many advantages, including low cost, low invasive insertion procedures, and great versatility: many authors have demonstrated that they can be used as a successful source of anchorage during orthodontic therapy [49]. In addition, miniscrews can be used in children, in adolescents, and in adults for different orthodontic procedures such as distalization, retraction of maxillary anterior teeth, intrusion, and protraction of maxillary posterior teeth and remain almost stationary throughout orthodontic loading, if they have been correctly positioned.

A skeletal anchorage system [SAS] has been developed that uses pure titanium anchor plates and screws as absolute orthodontic anchorage units[50].The anchor plates are mono cortically placed at the piriform opening rim, the zygomatic buttresses, and any regions of the mandibular cortical bone. SAS enables the rigid anchorage that results from the osseointegration effects in both the anchor plates and screws [51].Sugawara et al, used a

specially designed skeletal anchorage system [SAS] for correction of anterior open bite by intruding the mandibular molars in humans [52].

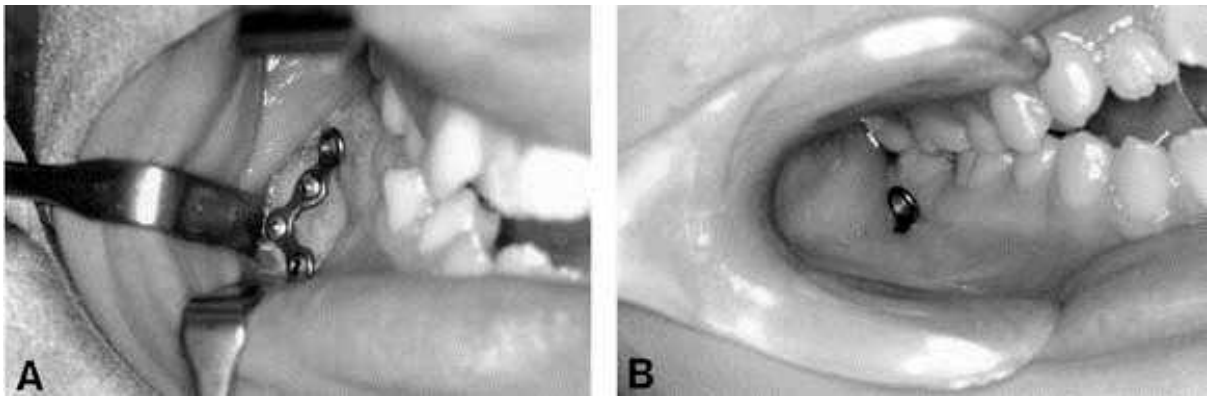


Fig 8: Intraoral Skeletal Anchorage System

Frozat Appliance

A modification of the Frozat [fixed Crozat] appliance initially developed by Mayes, the unilateral Frozat appliance consists of two molar bands soldered to a .038" Blue Elgiloyor .040" stainless steel wire [53]. Kinzinger et al, in his study suggested that unilateral Frozat appliance allows a controlled uprighting and distalization of the lower molars. Intramaxillaryanchorage is achieved by simultaneously inserting a rigid and passive buccal wire segment [54].

Fig 9: Frozat Appliance

Repelling Magnets

Magnets were introduced as a force system a few years before the introduction of super elastic coils. Ithas been shown that approximately one-half of the distal movement of molars treated with magnets are related to tipping, with the obvious risk of relapse[55].Bondemark et al, suggested that the super elastic nickel titanium open coils are more effective than the repelling samarium-cobalt magnets for simultaneous distal movement of maxillary first and second molars. He also says that the force applied by coils are more constant when compare to magnets [56].

Due to the increasing tendency towards non-extraction treatment, much work has been put into developing new intra-oral molar distalization procedures, including the use of magnetic forces and nickel-titanium coil springs. With their excellent properties, such as high energy product values and high coercive forces, rare earth magnets have been used in orthodontic tooth movements [57]. An important disadvantage of rare earth magnets is their low corrosion resistance, but by means of stainless steel coatings, the resistance against corrosion is increased.

Various investigators have studied the biological effects of static magnetic fields on living tissues. Changes in osteoblastic and osteoclastic activities, a decrease in epithelial thickness and an increase in blood flow rate have been accepted by several investigators as the biological effects of magnetic fields [58]. However, there is no agreement as to whether the effects reported were produced by the magnetic alloy or the magnetic field. In two different studies, Gianelly et al. achieved effective molar distalization using repelling magnets. They suggested weekly activation of the magnets in order to maximize the force and stated that in cases with second molars in complete occlusion, distalization took longer. Anchorage loss in this study was calculated as 20 per cent [59].

In another study by Itoh et al, also have used repelling magnets for molar distalization; they recommended activation of the magnets at intervals of 2 weeks and reported an anchorage loss of about 30 per cent. Using repelling magnets, Bondemark and Kurol [1992], reported effective molar distalization, together with distobuccal rotation [60].

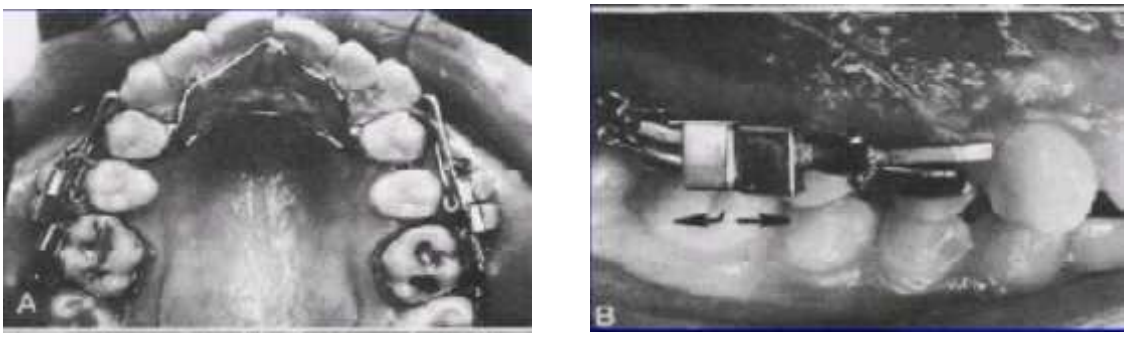


Fig 10: Intraoral Repelling Magnets

Frog Appliance

One of the important goals of molar distalizing therapy is to obtain bodily tooth movement of the molars with minimal rotation and distal inclination. TheFrog appliance was positioned approximately 10 to 12 mm apically to the occlusal surface of the maxillary molar with parallel orientation to the occlusal plane in our case. In this manner, a vector of effective force passing through the centre of resistance of the first molar was obtained [61].

In 2006, Walde developed an intraoral molar distalizing appliance named the Frog appliance [62].The Frog appliance consists of a special distalizing expansion screw with an anterior activation head that can be easily reached intraorally; additionally, a wired spring is attached to the expansion screw and is inserted into the palatal tubes of the molars [63].Mehmet et al,suggested that the Frog appliance is an effective and reliable method for the distalization of maxillary molars. Unfortunately, reciprocal anchorage loss in the premolars and incisors occurred during distalization [64].

Fig 11: Frog Appliance

Essix Based Molar Distalization

A new thermoplastic appliance called EssixH [RaintreeEssix, Inc., 4001 Division St, Metairie, LA 70002, USA] was first introduced as a retainer by Sheridan and colleagues in 1993 [65]. Thermoformed Essix appliances have multiple uses in general dentistry and orthodontics, which greatly expand the many treatment options currently available. Essix appliances are clear plastic; the result is aesthetically pleasing to the eye [66].Essix retainer proved to be sufficient for maintaining orthodontic treatment results [67]. The expected increase of occlusal contacts was not observed at the end of the retention period with Essix thermoplastic retainers as these cover the occlusal surfaces of teeth [68].

Fig 12: Essix Retainer

Mini Implants

Terms such as mini-implants, miniscrews, micro implants, and micro screws have been used to describe devices of skeletal anchorage. Although the group of these terms describes devices smaller than conventional dental implants that provide skeletal anchorage which is discontinued after treatment, they should not be used interchangeably. Implants and mini-implants refer to systems, which by definition imply that osseointegration sets in prior to loading, whereas screws to self-tapping devices that may be used without the condition of osseointegration. However, since 2004 it was agreed on that the word mini-implant should be applied both to palatal implants, to mini-implants, to miniscrews, and to micro screws [69].

Conclusion

Distalization represents an important constituent of non-extraction treatment in orthodontics. Molardistalization is recommended for class II malocclusion with skeletal and dentoalveolar maxillary protrusion or when the dental extraction is not recommended for

the maxillary. Distalization is the first phase treatment in correction of class II malocclusion. Molardistalization can be made unilaterally as well, in order to correct a unilateral class II relation.

Corresponding Author

Padma Ariga

Professor, Department of Prosthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 poonamelle high road,Chennai 600 077-Tamil Nadu, India

Mail Id:padmanest@gmail.com

Telephone Number:9489594048

References

1. NurhatOzkalayci and Mehmet Yetme.A New Orthodontic Appliance with a Mini Screw for Upper Molar Distalization.Applied Bionics and Biomechanics Volume 2016, Article ID 5728382.

2.A.Prakash,R.Patil,O.P.Mehta,G.Singh,andP.K.Jayaprakash.Postdistalization—anchoringmolars.Indian Journal of Dental Sciences.2013; vol. 5, pp. 120–121.

3.Cangialosi TJ, Meistrell ME Jr, Leung MA, Ko JY. A cephalometric appraisal of edgewise Class II non extraction treatment with extra oral force. Am J Orthod Dentofacial Orthop .1988; 93:315-24.

4. Fortini A, Lupoli M, Giuntoli F, Franchi L. Dentoskeletal effects induced by rapid molar distalization with the first class appliance. Am J Orthod Dentofacial Orthop. 2004;125[6]:697-704.

5. Brickman CD, Sinha PK, Nanda RS. Evaluation of the Jones jig appliance for distal molar movement.Am J Orthod Dentofacial Orthop. 2000;118[5]:526-34

6.Carano A, Testa M**.**The distal jet appliance for upper molardistalization.J Clin Orthod. 1996;30:374–380.

7.ProffittWR.Contemporary Orthodontics. 2nd ed. St Louis,Mo: CV Mosby; 1993:306–307.

8.Gianelly AA. Distal movement of the maxillary molars.Am J Orthod. 1998;114:66–72.

9.Sfondrini MF, Cacciafesta V, Sfondrini G. Upper molar distalization: a critical analysis. Orthod Craniofac Res. 2002;5: 114–126.

10.Gianelly AA, Bednar J, Dietz VS.Japanese Ni-Ti coils used to move molars distally. AmJ Orthod Dentofacial Orthop.1991;99:564–566.

11.WeinbergerBW.Orthodontics: an historical review of its origin and evolution.St Louis: CV Mosby Co.; 1926.

12.Weinstein S, Haak DC, Morris LY, Snyder BB, Attaway HE**.**On equilibrium theory of tooth position.Angle Orthod 1963; 33:1-26.

13.Pilon JJ, Kuijpers-Jagtman AM, Maltha JC. Magnitude of orthodontic forces and rate of bodily tooth movement.An experimental study. Am J Orthod Dentofacial Orthop 1996;110:16-23.

14.Creekmore TD, Eklund MK. The possibility of skeletal anchorage. J Clin Orthod1983;17:266-9.

15. Douglass JB, Killiany DM**.** Dental implants used as orthodontic anchorage. J Oral Implant 1987;13:28-38.

16.Roberts WE, Helm FR, Marshall KJ, GongloffRK.Rigidendosseous implants for orthodontic and orthopedic anchorage. Angle Orthod 1989;59:247-56.

17.Hilgers JJ. The pendulum appliance for Class II non-compliance therapy.J Clin Orthod. 1992;26:706–714.

18.Snodgrass DJ. A fixed appliance for maxillary expansion, molar rotation, and molar distalization.J Clin Orthod.1996; 30:156–159.

19. Ghosh J, Nanda RS. Evaluation of an intraoral maxillary molar distalization technique.Am J Orthod Dentofacial Orthop. 1996;110:639–646.

20. Gianelly AA, Vaitas AS, Thomas WM, Berger OG. Distalization of molars with repelling magnets.J Clin Orthod.1988; 22:40–44.

21.JeckelN,RakosiT.MolarDistalizationby intraoral force application.Eur J Orthod 1991;13:43-6.

22.Renato Rodrigues de Almeida, Marcio Rodrigues de Almeida, and James A. McNamara, Jre.Effects of the pendulum appliance, cervical headgear, and 2 premolar extractions followed by fixed appliances in patients with Class II malocclusion.American journal of orthod Dentofacial Orthop.2009;136[6]:833-42.

23.Weinberger B W. Orthodontics: a historical review of its origin and evolution . Mosby, St Louis.1926

24. Burçak Kaya, AyçaArman, SinaUçkan and Ay s¸ e Canan Yazıcı .Comparison of the zygoma anchorage system with cervical headgear in buccal segment distalization. European Journal of Orthodontics.2009, 31:417–424

25. Wilson W L,Wilson R C.Modular 3D appliances problem solving in edgewise, straight wire and light wire treatment.Journal of Clinical Orthodontics.1984;18:272 – 281

26. Ngantung V ,Nanda R S ,Bowman S J.Post treatment evaluation of the distal jet appliance.American Journal of Orthodontics and Dentofacial Orthopedics.2001; 120: 178–185.

27.Burkhardt DR, McNamara JA Jr, Baccetti T. Maxillary molar distalization or mandibular enhancement: a cephalometric comparison of comprehensive orthodontic treatment including the pendulum and the Herbst appliances. Am J Orthod Dentofacial Orthop 2003;123:108-16.

28.Fernanda Angelieri, Renato Rodrigues de Almeida, GuilhermeJanson, José Fernando CastanhaHenriques and ArnaldoPinzan.Comparison of the effects produced by headgear and pendulum appliances followed by fixed orthodontic treatment.European Journal of Orthodontics.2008;30: 572–579.

29.Fraunhofer, J. A., Bonds, P. W. and Johnson, B. E.Force generation by orthodontic coil springs, Angle Orthodontics, 1993;63, 145–148.

30.Gianelly, A. A., Vaitas, A. S. and Thomas, W. M.Distalization of molars with repelling magnets, Journal of Clinical Orthodontics. 1988;22, 40–44.

31.Gianelly, A. A., Vaitas, A. S. and Thomas, W. M.The use of magnets to move molars distally.American Journal of Orthodontics and Dentofacial Orthopedics,1989;96, 161–167.

33.Pancherz H. The mechanism of Class II correction in Herbst appliance treatment.A cephalometric investigation.Am J Orthod. 1982;82:104–113.

34.DahlbergG**.**Statistical Methods for Medical and Biological Students. London, United Kingdom: Allen and Unwin; 1940: 122–132.

35.Houston WBJ. The analysis of errors in orthodontic measurements.Am J Orthod. 1983;83:382–390.

36.Bondemark L, Kurol J. Distalization of maxillary first and second molars simultaneously with repelling magnets. Eur J Orthod.1992;14:264–272.

37.Muse DS, Fillman MJ, Emmerson J, Mitchell RD. Molar and incisor changes with Wilson rapid molar distalization. Am J Orthod Dentofacial Orthop. 1993;104:556–565.

38.ReinerTJ.Modifiednance appliance for unilateral molar distalization.J Clin Orthod 1992;26:402-04

39.Bourauel C, Drescher D ,Ebling J, Broome D, KanarachosA.Super elastic nickel-titanium alloy.RetractionSprings.An experimental investigation of force system.Eur J Orthod 1997;19:491-500

40. Arfan-UlHaq, Mwaheed-Ul-Hamid,Naseer Ahmad Chaudhry, RaabiaLatif**.**Effects Of Distal Jet Appliance In Class-II Molar Correction,Pakistan Oral & Dental Journal.*2010;* Vol 30, No. 1.

41. A. I. Karaman, DDS, PhD; F. A. Basciftci, DDS, MS; O. Polat, DDS.Unilateral Distal Molar Movement With an Implant-Supported Distal Jet Appliance. Angle Orthodontist,2002;Vol 72, No 2.

42. Hector LuisRodríguez.Unilateral application of the CarriereDistalizer Journal of clinical orthodontics;JCO· March2011.

43. Luis Carrière, Dds, Msd.A New Class II Distalizer. JCO, Inc,2004.

44. Fortini A, Lupoli M, Parri M. The first class appliance for rapid molar distalization. J Clin Orthod 1999;33:322-8.

45. A. Gracco, A. Cirignaco, M. Cozzani, A. Boccaccio, C. Pappalettere, and G. Vitale. "Numerical/experimental analysis of the stress eld around miniscrews for orthodontic anchorage," European Journal of Orthodontics, 2009;vol. 31, no. 1, pp. 12–20.

46.G. S. M. Kinzinger, N. Gulden, F. Yildizhan, and P. R. Diedrich. "Efficiency of a skeletonized distal jet appliance supported by miniscrew anchorage for noncompliance maxillary molar distalization,".American Journal of Orthodontics and Dento- facial Orthopedics,2009;vol. 136, no. 4, pp. 578–586.

47.Arturo Fortini, MD, DDS,a Massimo Lupoli, MD, DDS,a Fabio Giuntoli, DDS,a and Lorenzo Franchi, DDS, PhD .Dentoskeletal effects induced by rapid molar distalization with the first class appliance. American Journal of Orthodontics and Dentofacial Orthopedics, 2004, 125[6]:697-704.

48. Moschos A. Papadopoulos, Aristides B. Melkos andAthanasios E. Athanasiou.Noncompliance maxillary molar distalization with the First Class Appliance: A randomized controlled trial.American Journal of Orthodontics and Dentofacial Orthopedics2010.

49. Keles A, Sayinsu K. A new approach in maxillary molar distalization: Intraoral bodily molar distalizer. Am J Orthod Dentofacial Orthop 2000; 117:39–48.

50.Ahmet Keles, DDS, DMSc/BinnurPamukcu, DDS/EbruCetinkayaTokmak, DDS.Bilateral Maxillary Molar Distalization with Sliding Mechanics: KelesSlider.world journal of orthodontics, 2002, vol 3.

51.Nagasaka H, Sugawara J, Kawamura H, Kasahara T, Umemori M, Mitani H. A clinical evaluation on the efficacy of titanium miniplates as an orthodontic anchorage.Jpn J Orthod Soc1999; 58:136-47.

52. Junjisugawara, Takayoshi Daimaruya, MikakoUmemori, Hiroshi.Distal movement of mandibular molars in adult patients with the skeletal anchorage system. American Journal of Orthodontics and Dentofacial Orthopedics Volume 125, Number 2.

53.Sugawara J, Baik UB, Umemori M, Takahashi I, Nagasaka H, Kawamura H, Mitani H. Treatment and post treatment dentoalveolar changes following intrusion of mandibular molars with application of a skeletal anchorage system [SAS] for open bite correction. Int J Adult Orthod Orthognath Surgery. 2002; 17:243245.

54.Blechman, A. M. and Smiley, H**.**Magnetic force in orthodontics, American Journal of Orthodontics, 1978; 74, 435–443.

55.Aronson, A. L., Lindskog, S. and Rygh, P.Orthodontic magnets: effects on gingival epithelium and alveolar bone in monkeys,European Journal of Orthodontics, 1992;14, 255–263.

56.Bondemark, L. and Kurol, J.Distalization of maxillary first and second molars simultaneously with repelling magnets,European Journal of Orthodontics, 1992; 14, 264–272.

57.Papadopoulos M A , Mavropoulos A , Karamouzos A. Cephalometric changes followingsimultaneous first and second maxillary molar distalization using a non-compliance intraoralappliance . Journal of Orofacial Orthopedics,2004; 65 : 123 – 136

58.Kinzinger, G.; Fritz, U.; and Diedrich, P.: Various anchorage approaches in unilateral mandibular molar distalization using a fixed lingual arch appliance, J. Orofac. Orthop.2004;65:137-149.

59. G. S. Antonarakis and S. Kiliaridis, “Maxillary molar distalization with noncompliance intramaxillary appliances in class II malocclusion: a systematic review,” Angle Orthodontist,2008;vol.78, no. 6, pp. 1133–1140.

60.Bondemark L, KurolJ.Distalization of maxillary first and second molars simultaneously with repelling magnets. Eur J Orthod 1992;14:264-72.

61.Lars Bondemark, DDS;Jurikurol, DDS, PHD;MatsBernholdDDS**.**Repelling magnets versus super elastic nickel titanium coils in simultaneous distal movement of maxillary first and second molars. The Angle orthodontist,1994;vol.64,number 3.

62.Ludwig B, Glasl B, Kinzinger GS, Walde KC, Lisson JA. The skeletal frog appliance for maxillary molar distalization.J Clin Orthod 2011;45:77-84.

63. Ahmad SharafeddinBurhan .Combined treatment with headgear and the Frog appliance for maxillary molar distalization:a randomized controlled trial;Korean J Orthod 2013;43[2]:101-109.

64. Mehmet Bayram, DDS, PhD,MetinNur, DDS, PhD, DoganKilkis, DDS**.**The frog appliance for upper molar distalization: a case report. Korean J Orthod,2010;Vol. 40, No. 1.

65.H. Babacan and C. Doruk.Essix H-based molar distalization appliance., Journal of Orthodontics,2005;Vol. 32, 229–234.

66.Kamlesh Kumar Garg, Ashish Mahendra, SonahitaAgarwal.Essix retainer as a modified esthetic anchorage device. APOS Trends Orthod 2014;4:48-9.

67. Jaderberg S, Feldman I, EngstromC**.**Removable thermoplastic appliances as orthodontic retainers—a prospective study of different wear regimens. European Journal of Orthodontics 2011.

68.Müfi de Dinçer and BelmaIsikAslan**.**Effects of thermoplastic retainers on occlusal contacts. European Journal of Orthodontics.2010;32:6–10.

69.Carano A, Melsen B. Implants in orthodontics. Prog Orthod 2005;6:62-9.

DNA Replication In Bacteria, Eukaryotes And Archaea

Anitha.D.R [1], Anitha. M [2], Sri Sakthi.D [3]
Graduate Student [1], [2]Department of Bio chemistry
Reader [3], Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical And Technical Science, Saveetha University, Chennai, India

Abstract

Replication of DNA – deoxyribonucleic acid – happens before a cell divides to ensure that both cells receive an exact copy of the parent's genetic material. While there are many similarities in how prokaryotic and eukaryotic cells replicate their DNA, there are several distinctions between them, due to the different size and complexity of the molecules, including the time it takes to complete the process.Prokaryotic cells are quite simple in structure. They have no nucleus, no organelles and a small amount of DNA in the form of a single, circular chromosome. Eukaryotic cells on the other hand, have a nucleus, multiple organelles and more DNA arranged in multiple, linear chromosomes.Replication of DNA is essential for the propagation of life. It is somewhat surprising then that, despite the vital nature of this process, cellular organisms show a great deal of variety in the mechanisms that they employ to ensure appropriate genome duplication. This diversity is manifested along classical evolutionary lines, with distinct combinations of replicon architecture and replication proteins being found in the three domains of life: the Bacteria, the Eukarya and the Archaea. Furthermore, although there are mechanistic parallels, even within a given domain of life, the way origins of replication are defined shows remarkable variation.

Keywords: DNA, Replication, Bacteria, Eukarya, Archaea.

Introduction

DNA replication is the way toward creating two indistinguishable reproductions from one unique DNA particle. This natural procedure happens in every single living creature and is the reason for organic legacy. DNA is comprised of two strands and each strand of the first DNA particle fills in as layout for the creation of the correlative strand, a procedure alluded to as semiconservative replication[1]. Cell editing and mistake checking systems guarantee close ideal constancy for DNA replication in a cell, DNA replication starts at particular areas, or inceptions of replication, in the genome. Loosening up of DNA at the inception and union of new strands brings about replication forks becoming bidirectional from the birthplace. Various proteins are related with the replication fork which helps as far as the start and continuation of DNA amalgamation. Most unmistakably, DNA polymerase incorporates the new DNA by adding reciprocal nucleotides to the layout strand. DNA replication can likewise be performed in vitro (falsely, outside a cell). DNA polymerases disconnected from cells and fake DNA preliminaries can be utilized to start DNA union at known successions in a format DNA atom. The polymerase chain response (PCR), a typical research facility method, consistently applies such simulated blend to open up a particular target DNA section from a pool of DNA.

DNA or deoxyribonucleic corrosive is a kind of particle known as a nucleic corrosive. It comprises of a 5-carbon deoxyribose sugar, a phosphate, and a nitrogenous base. Twofold stranded DNA comprises of two winding nucleic corrosive chains that are contorted into a twofold helix shape. This turning enables DNA to be more reduced. Keeping in mind the end goal to fit inside the core, DNA is pressed into firmly curled structures called chromatin. Chromatin gathers to shape chromosomes amid cell division. Preceding DNA replication, the chromatin extricates giving cell replication apparatus access to the DNA strands[2].

Replication of DNA – deoxyribonucleic corrosive – occurs before a cell partitions to guarantee that the two cells get a precise of the parent's hereditary material. While there are numerous similitudes in how prokaryotic and eukaryotic cells repeat their DNA, there are a few qualifications between them, because of the distinctive size and intricacy of the particles, including the time it takes to finish the process[3]. Prokaryotic cells are very straightforward in structure. They have no core, no organelles and a little measure of DNA as a solitary,

roundabout chromosome. Eukaryotic cells then again, have a core, various organelles and more DNA masterminded in different, straight chromosomes.

Arrangement of DNA replication

Stage 1: Replication Fork Formation

Before DNA can be repeated, the twofold stranded particle must be "unfastened" into two single strands. DNA has four bases called adenine (A), thymine (T), cytosine (C) and guanine (G) that shape combines between the two strands. Adenine just combines with thymine and cytosine just ties with guanine. Keeping in mind the end goal to loosen up DNA, these associations between base sets must be broken. This is performed by a compound known as DNA helicase. DNA helicase disturbs the hydrogen holding between base sets to isolate the strands into a Y shape known as the replication fork. This territory will be the layout for replication to begin[4].

DNA is directional in the two strands, connoted by a 5' and 3' end. This documentation connotes which side gathering is appended the DNA spine. The 5' end has a phosphate (P) amass joined, while the 3' end has a hydroxyl (OH) gather appended. This directionality is vital for replication as it just advances in the 5' to 3' course. In any case, the replication fork is bi-directional; one strand is situated in the 3' to 5' course (driving strand) while the other is arranged 5' to 3' (slacking strand). The two sides are along these lines recreated with two unique procedures to oblige the directional contrast.

Stage 2: Primer Binding

The main strand is the least difficult to repeat. Once the DNA strands have been isolated, a short bit of RNA called a groundwork ties to the 3' end of the strand. The groundwork dependably ties as the beginning stage for replication. Preliminaries are produced by the protein DNA primase.

Stage 3: Elongation

Proteins known as DNA polymerases are capable making the new strand by a procedure called extension. There are five diverse known sorts of DNA polymerases in microscopic organisms and human cells. In microorganisms, for example, E. coli, polymerase III is the primary replication compound, while polymerase I, II, IV and V are in charge of mistake checking and repair. DNA polymerase III ties to the strand at the site of the groundwork and starts adding new base sets corresponding to the strand amid replication[5]. In eukaryotic cells, polymerases alpha, delta, and epsilon are the essential polymerases associated with DNA replication. Since replication continues in the 5' to 3' bearing on the main strand, the recently shaped strand is consistent.

The slacking strand starts replication by authoritative with different preliminaries. Every preliminary is just a few bases separated. DNA polymerase at that point includes bits of DNA, called Okazaki sections, to the strand between preliminaries. This procedure of replication is broken as the recently made sections are disconnected.

Stage 4: Termination

Once both the nonstop and irregular strands are framed, a catalyst called exonuclease expels all RNA preliminaries from the first strands. These preliminaries are then supplanted with proper bases. Another exonuclease "edits" the recently framed DNA to check, evacuate and supplant any errors[6]. Another protein assembled DNA ligase joins Okazaki sections framing a solitary brought together strand. The finishes of the straight DNA introduce an issue as DNA polymerase can just add nucleotides in the 5′ to 3′ heading. The finishes of the parent strands comprise of rehashed DNA successions called telomeres. Telomeres go about as defensive tops toward the finish of chromosomes to keep close-by chromosomes from intertwining. A unique kind of DNA polymerase chemical called telomerase catalyzes the union of telomere arrangements at the closures of the DNA.Once completed, the parent strand and its complementary DNA strand coils into the familiar double helix shape. In the end, replication produces two DNA molecules, each with one strand from the parent molecule and one new strand.

Steps in DNA replication

DNA replication starts at a particular spot on the DNA atom called the birthplace of replication. At the starting point, catalysts loosen up the twofold helix making its segments open for replication. Each strand of the helix at that point isolates from the other, uncovering the now unpaired bases to fill in as layouts for new strands. A little section of RNA – ribonucleic corrosive – is included as a preliminary, at that point new nucleotide bases that supplement the unpaired bases can be gathered to shape two girl strands by each parent strand. This get together is expert with catalysts called DNA polymerases[7]. At the point when the procedure is finished, two DNA atoms have been framed indistinguishable to each other and to the parent particle.

Replication starts at specific positions in chromosomes called "causes" where assigned initiator proteins tie to DNA to begin the procedure of replication. There are imperative contrasts among microbes, archaea, and eukaryotes in this procedure, however there are likewise numerous striking similitudes that recommend the procedure goes back to the last all inclusive cell ancestor[8]. Microscopic organisms regularly contain just on chromosome with one beginning at which two replication forks gather and move inverse way. In spite of the fact that not all microorganisms take after this worldview, this is the situation for the Escherichia coli round 4.4 Mb genome, shaping a solitary replicon or unit of replication from a solitary source. At a rate of 1 Kb/s for each fork, this genome is reproduced inside 30 min. Interestingly, eukaryotes ordinarily have numerous direct chromosomes, each with numerous birthplaces. Numerous causes are a need for eukaryotes as they have substantially bigger genomes than microscopic organisms and eukaryotic replication forks move around 20 times more gradually than bacterial replication forks. For instance, the biggest human (chromosome #1) is 250 Mb and on the off chance that it had just a single starting point, it would require over 50 days to imitate contrasted with the regular 24 h division time of an eukaryotic cell and roughly 8 h for duplicating DNA in S stage. Start at every birthplace produces two disparate DNA replication forks along the chromosome to make a replicon that is copied just once per cell division. The duplication of numerous replicons in the long run yields two girl chromosomes assembled sister chromatids that are fastened until the point that they isolate amid mitosis. Albeit few archaea species have been described, they seem, by all accounts, to be transformative cross breeds amongst microbes and eukaryotes, since a few species have a solitary chromosome with a solitary cause, though different species have numerous starting points per chromosome. Besides, the ploidy of the genome in archaea shifts impressively,

with a few species having a 1C– 2C dispersion all through their cell cycle, while others have up to 25 duplicates of their genome in multiplying cells. The rate of DNA replication fork movement likewise has all the earmarks of being in the middle of that in microscopic organisms and eukaryotes, at around 20 kB/min, ∼10 times quicker than that in eukaryotes. Albeit a few microbes like E. coli duplicate their genomes significantly quicker, others, for example, Caulobacter crescentus reproduce at generally an indistinguishable rate from some archaea, for example, Pyrococcus abyssi.

Bacterial starting points are very much characterized groupings to which the replication initiator proteins tie. Conversely, eukaryotic starting points are not regularly characterized at the level of DNA arrangement (with the vital special case of the sprouting yeast Saccharomyces cerevisiae). Noteworthy late advance has demonstrated that eukaryotic beginnings are characterized less by DNA grouping than by chromatin association, with numerous roots relating to locales of DNA with transcriptional action or different highlights that enable access to starting point restricting proteins. Numerous human cell sources happen in successions that are developmentally moderated among vertebrates, recommending that they are a long way from arbitrary[9] . In many eukaryotes, a little subset of potential sources is utilized as a part of an average cell cycle in singular cells, however birthplace use can be extraordinarily expanded to encourage incredibly fast cell division, as found in the treated eggs of numerous creatures. Regardless of whether a birthplace is utilized or not is a stochastic procedure that relies upon the chromatin setting and now and again the formative condition of cells in multicellular life forms. Moreover the numerous beginnings in eukaryotic chromosomes are sorted out into bunches that are initiated at particular circumstances amid S period of the cell cycle, and the transient designing shifts again with formative designing of cells.

To start the way toward actuating a birthplace for replication, bacterial, archaeal, and eukaryotic cells utilize source restricting proteins made out of AAA+ family subunit(s). AAA+ proteins for the most part work as multimeric machines. For instance in microbes, various duplicates of the DnaA birthplace restricting protein shape a helical fiber that ties the beginning. The DnaA fiber ties ATP to loosen up an A/T-rich locale of the starting point, bringing about a solitary strand DNA (ssDNA) "bubble" onto which the replicative helicase loads[10].

Eukaryotes contain a six subunit beginning restricting protein alluded to as ORC (birthplace acknowledgment complex). Five of the ORC subunits are identified with AAA+ proteins and together with another AAA+ protein called Cdc6 that is profoundly related in grouping to the biggest ORC subunit, Orc1, they frame a ring-molded hexamer that ties DNA . Be that as it may, not at all like bacterial DnaA, ORC does not loosen up DNA at districts to which it ties. Archaeal cells additionally utilise AAA+proteins that are identified with the biggest subunit of ORC, Orc1 and to Cdc6, yet the quantity of these subunits changes relying upon the specific sort of archaeal cell. Both DnaA and ORC are utilized as a part of different procedures other than replication.

Replication mechanism

Replication is performed by a multiprotein replication machine that incorporates both little girl duplexes all the while. Replication machines have a similar centre parts in all cells: DNA polymerases, round sliding clips, a pentameric clasp loader, helicase, primase, and SSB (single-strand restricting protein). The manner by which these proteins are masterminded and interface with each other contrasts among cell types[11,12]. The replication machine is regularly alluded to as a "replisome." The bacterial replisome, is sorted out by the clasp loader, which contains three indistinguishable τ subunits that predicament three C-family DNA polymerase III (Pol III) polymerases. The τ subunit additionally ties the homohexameric helicase (i.e., E. coli DnaB). As primase shapes RNA groundworks, the cinch loader over and over burdens new roundabout β braces onto the preliminary/layout for use by the slacking strand Pol III(s). This primase/polymerase switch is encouraged by SSB, which ties ssDNA and empowers the cinch loader to unstick primase from the prepared site. SSB shields the ssDNA from nucleases and encourages prolongation by Pol III-β on ssDNA. The bacterial replisome is exceedingly processive, implying that in E. coli it can incorporate ~86 kb on the main strand without separating from the format. In any case, high processivity is a weakness on the slacking strand, which is blended as various short ~1000 nt Okazaki parts and requires the polymerase to separate from the format DNA and reassociate at another prepared site for each Okazaki section. To conquer this "processivity obstruction," particular systems have advanced that pry the polymerase from the cinch to discharge an Okazaki piece, on which the polymerase reassociates with another clasp at the following RNA prepared site.

The three Pol IIIs in the bacterial replisome help the generation of different Okazaki fragments[13].

Albeit eukaryotic and archaeal replisomes have comparative parts, the associations among the segments are very not the same as bacterial replisomes. The hexameric MCM2-7 circles the main strand, not the slacking strand and needs the Cdc45 and GINS proteins for it to work in loosening up DNA[14]. Additionally, the eukaryotic replisome contains two diverse B-family polymerases that capacity independently for the main and slacking strands, Pol ε and Pol δ, individually, notwithstanding Pol α/primase, which begins each Okazaki section. The slacking strand is covered by replication protein A (RPA), a heterotrimer SSB that is fundamentally and practically comparable to the bacterial SSB tetramer. As in microbes, a primase/polymerase switch is intervened by RPA. Both Pol ε and Pol δ work with a ring-molded multiplying cell atomic antigen (PCNA) clasp of comparable structure to E. coli β. PCNA is collected on DNA by the pentameric replication factor C (RFC) brace loader, made out of subunits with succession homology and comparative structure to the bacterial cinch loader. Dissimilar to microbes, RFC does not seem to contact the polymerases or the helicase, and the associations among replisome segments stay indistinct. There are various competitors among the numerous proteins known to be required for proficient eukaryotic replication that have no homolog in microorganisms. For instance, the GINS heterotetramer and the Cdc45 protein that shape a complex with the MCM2-7 helicase to yield the CMG complex may tie Pol α/primase. GINS and Cdc45 are required for proficient DNA loosening up movement and may tie different factors notwithstanding the MCM2-7 complex to compose the replisome. In growing yeast, a replication movement complex has been distinguished containing potential connections between the CMG and Pol α/primase by means of Ctf4/AND-1, and amongst CMG and Pol ε through Mrc1/Claspin. Point by point confirm for these and other conceivable associations, the elements of closely resembling proteins in different eukaryotes, and their significance for fork movement can be found.

The oligomeric structures of sliding cinches empower different proteins to tie one clasp at the same time, alluded to as the "toolbelt" hypothesis[15]. An extraordinary case is an archaeal PCNA heterotrimer in which each unique subunit ties an alternate accomplice protein (i.e., polymerase, ligase, and Fen1) that is associated with the amalgamation and development of

Okazaki pieces. These highlights of sliding cinches are talked about , however one angle will be said here as it has critical ramifications for replisome structure and capacity. Specifically, all phones contain an assortment of low-loyalty DNA polymerases that can sidestep injuries in the DNA, alluded to as "TLS" Pols (translesion blend polymerases). Distinctive Pols can tie the clip all the while and exchange places with each other, making the replisome a considerably more unique machine than initially thought , E. coli contains three "translesion" DNA polymerases (TLS Pols), which are instigated on DNA harm. Studies have demonstrated that at high fixations, for example, those instigated on DNA harm, the TLS Pols tie the β clip and exchange places with Pol III at a moving fork, yet hold the helicase to shape a "TLS replisome." TLS replisomes move significantly more gradually than Pol III and manage the speed of the helicase. A conspicuous preferred standpoint of shaping TLS replisomes on DNA harm is to moderate the replication fork, giving time for DNA repair before a replication fork experiences a sore. In the occasion a sore is experienced, it can be circumvent by the TLS polymerase.

Bacteria

In bacteria the origin of replication is named oriC, and commonly a solitary beginning exists for every bacterial chromosome. In Escherichia coli, oriC is situated between the gldA and mioC qualities. The % 250 bp oriC locale contains numerous rehashed groupings containing a nine base match accord component named the DnaA box[5,6]. Other microbes additionally have single starting points of replication with different DnaA boxes albeit both the exact number and conveyance of these crates change between species. Strikingly, in numerous microorganisms the cause of replication is discovered contiguous the quality for DnaA itself, recommending a system for the organize control of birthplace action and levels of initiator proteins. An individual accord DnaA box is bound by a monomer of the DnaA protein and this association actuates a sharp twist in the coupling site. In any case, in characteristic bacterial starting points there are various DnaA boxes and these arrange complex coopererative restricting occasions to DnaA boxes with changing degrees of adjustment to the agreement sequence[16,17]. An especially intriguing consequence of this is a DnaA box with poor preservation to the accord will most likely be unable to tie DnaA all alone. In any case, authoritative to this 'feeble' site can be encouraged by official of DnaA to a nearby high

partiality agreement site. Bacterial starting points of replication additionally have a moment monitored component, an exceedingly AT rich area. The loosening up of this naturally meltable DNA is a key advance in replication start at roots. Under very characterized in vitro conditions, DnaA is equipped for intervening halfway loosening up of this locale all alone. It shows up thusly, that the blend of DNA bowing initiated by DnaA and the agreeable connections between DnaA monomers on DNA result in nearby topological pressure that shows itself by loosening up of this naturally less steady district of duplex.

Eukaryotic sources

The recognisable proof of start destinations in eukaryotic creatures has been a burdensome errand. As opposed to the single, plainly characterized locales of bacterial replication, eukaryotic DNA union begins from hundreds or even a huge number of birthplaces, which once in a while contain evident arrangement themes, and are regularly hard to characterize[17] . This many-sided quality is intensified by the way that eukaryotic birthplace actuation is non concurrent. What's more, start site use shows significant adaptability under changing development conditions, or all through various phases of advancement. In later years, it has turned out to be progressively evident that epigenetic factors represent the direction of eukaryotic source activity[18]. These tweaks give the versatility important to composed start from different locales.

Archaea

Rather than the abundance of sub-atomic, hereditary and biochemical detail that is currently thought about roots of replication and their association with initiators in microorganisms and eukaryotes, next to no is thought about the sub-atomic premise of replication start in the third space of life, the archaea. It is entrenched that archaea have a captivating mix of bacterial and eukaryotic highlights and in addition viewpoints that are special to this space of life[19,20,21]. Archaeal chromosomes look like those of most microscopic organisms, being little, round and having polycistronic interpretation units. What's more, archaea are probably going to have coupled interpretation and interpretation. Notwithstanding, it has turned out to be evident that the centre data preparing apparatuses of the archaea are in a general sense

identified with those of eukaryotes. Accordingly, the interpretation and DNA replication apparatuses of archaea are firmly identified with, however altogether less difficult than, their eukaryotic partners and particular from those of microscopic organisms. There-fore, archaea introduce themselves as a conceivably straightforward model framework to comprehend the rationed occasions in DNA replication. Various investigations have portrayed the biochemical properties of archaeal DNA replication proteins[19]. It is likewise of impressive enthusiasm to see how the straightforward bacterial-like chromosomes of the archaea are repeated by an eukaryotic-type replication contraption, to clarify the idea of the archaeal replicon association , and to build up the instruments by which archaeal replication birthplaces are characterized.

Conclusion

Replication of DNA is basic for the spread of life. It is fairly shocking then that, regardless of the indispensable idea of this procedure, cell living beings demonstrate a lot of assortment in the systems that they utilise to guarantee fitting genome duplication. This assorted variety is showed along established transformative lines, with unmistakable blends of replicon engineering and replication proteins being found in the three spaces of life: the Bacteria, the Eukarya and the Archaea. Moreover, despite the fact that there are unthinking parallels, even inside a given area of life, the path causes of replication are characterized demonstrates noteworthy variety.

In prokaryotic cells, there is just a single purpose of source, replication happens in two restricting ways in the meantime, and happens in the cell cytoplasm. Eukaryotic cells then again, have various purposes of cause, and utilise unidirectional replication inside the core of the cell. Prokaryotic cells have maybe a couple sorts of polymerases, while eukaryotes have at least four.

Replication likewise occurs at a substantially speedier rate in prokaryotic cells, than in eukaryotes. A few microorganisms take just 40 minutes, while creature cells, for example, people may take up to 400 hours. What's more, eukaryotes likewise have an unmistakable procedure for duplicating the telomeres at the closures of their chromosomes. With their

roundabout chromosomes, prokaryotes have no finishes to incorporate. Ultimately, the short replication in prokaryotes happens constantly, yet eukaryotic cells just experience DNA replication amid the S-period of the cell cycle.

Corresponding author

Sri Sakthi. D

Reader,Department of Public Health Surgery

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University,Chennai,India

162, Poonamallee High Road,Chennai – 600077,Tamil Nadu, India.

Mobile Number: +91 – 9962787531

Email ID: drsrisakthiphd@gmail.com

Reference

1. Araki H. Cyclin-dependent kinase-dependent initiation of chromosomal DNA replication. Curr Opin Cell Biol 2010;22: 766–771 .
2. Barry ER, Bell SD. DNA replication in the archaea. Microbiol Mol Biol Rev.2006;70: 876–887.
3. Gilbert DM, Takebayashi SI, Ryba T, Lu J, Pope BD, Wilson KA, Hiratani I. Space and time in the nucleus: Developmental control of replication timing and chromosome architecture. Cold Spring Harb Symp Quant Biol.2010;75: 143–153.
4. Frick DN, Richardson CC. DNA primases. Annu Rev Biochem. 2001; 70: 39–80.
5. Lehmann AR. DNA polymerases and repair synthesis in NER in human cells. DNA Repair (Amst). 2011;10: 730–733.
6. Kill IR, Bridger JM, Campbell KH, Maldonado-Codina G, Hutchison CJ. The timing of the formation and usage of replicase clusters in S-phase nuclei of human diploid fibroblasts. J Cell Sci. 1991;100: 869–87.
7. Rhind N. DNA replication timing: Random thoughts about origin firing. Nat Cell Biol. 2006; 8: 1313–1316.

8. O'Keefe RT, Henderson SC, Spector DL. Dynamic organization of DNA replication in mammalian cell nuclei: Spatially and temporally defined replication of chromosome-specific α-satellite DNA sequences. J Cell Biol. 1992;116: 1095–1110 .
9. Masai H, Matsumoto S, You Z, Yoshizawa-Sugata N, Oda M. Eukaryotic chromosome DNA replication: Where, when, and how? Annu Rev Biochem. 2010;79: 89–130.
10. Toro E, Shapiro L. Bacterial chromosome organization and segregation. Cold Spring Harb Perspect Biol. 2010; 2: a000349.
11. van Deursen F, Sengupta S, De Piccoli G, Sanchez-Diaz A, Labib K. Mcm10 associates with the loaded DNA helicase at replication origins and defines a novel step in its activation. Embo J. 2012;31: 2195–2206 .
12. Stillman B. Origin recognition and the chromosome cycle. FEBS Lett. 2005;579: 877–884 .
13. Sun J, Kawakami H, Zech J, Speck C, Stillman B, Li H. Cdc6-induced conformational changes in ORC bound to origin DNA revealed by cryo-electron microscopy. Structure. 2012;20: 534–544.
14. Tanaka S, Araki H. Regulation of the initiation step of DNA replication by cyclin-dependent kinases. Chromosomal. 2010;19: 565–574
15. Labib K. How do Cdc7 and cyclin-dependent kinases trigger the initiation of chromosome replication in eukaryotic cells? Genes Dev. 2010; 24: 1208–1219
16. Langston LD, Indiani C, O'Donnell M. Whither the replisome: Emerging perspectives on the dynamic nature of the DNA replication machinery. Cell Cycle. 2009; 8: 2686–2691
17. Schaarschmidt D, Baltin J, Stehle IM, Lipps HJ & Knippers R .An episomal mammalian replicon: sequence-independent binding of the origin recognition complex. EMBO J.2004;23, 191–201.
18. Kelman LM & Kelman Z Archaea: an archetype for replication initiation studies? Mol Microbiol. 2003; 48, 605– 616.
19. Lundgren M, Andersson A, Chen LM, Nilsson P & Bernander R .Three replication origins in Sulfo- lobus species: Synchronous initiation of chromosome replication and asynchronous termination. Proc Natl Acad Sci USA.2004; 101, 7046–7051.

20. Ramya chellammal muthusamy. DNA repair.J. Pharm. Sci. & Res. 2013:5(11);213 – 215.
21. Gheeta, Anitha roy, Vishnu priya, Lakshmi. Mitochondrial DNA and Inherited disease.Drug Invention Today. 2011:3(9);221-226 221.

Drug Eluting Stents-A Review

Sai Chaitanya Raj. B[1] , Anitha Roy [2] , Meenakshi Krishnan [3]
Graduate Student [1], Professor and Head [2], Department of Pharmacology,
Senior Lecturer [3],Department of Oral Medicine and Radiology
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Drug eluting stents were first developed in an attempt to prevent or address the issue of restenosis. Drug eluting stents consists of three parts namely, a stent platform, a coating of polymer that binds the drug to the stent and releases the drug and the stent itself.

The basic mechanism of drug delivery from a drug eluting scaffold involves encapsulating a drug in a polymer that either allows the drug to diffuse outward from it or that undergoes degradation in order to release the drug directly. For long term effects, non -biodegradable stents are the most commonly used stents. There are a wide variety of stents available and they are used for different purposes and procedures in the field of medicine. Vascular and biliary stents, expandable coronary stents and there are also simple plastic stents which is used to allow the flow of urine between kidney and the bladder. Drugs such as dexamethasone, betamethasone, heparin, hirudin, tocopherol, angiopeptin are delivered via stents.

Key Words: *Drug delivery, drug eluting stents, eluting agents, scaffold, non-biodegradable,*

Introduction

In general, the term stent is used to refer to a metal or a plastic tube which is inserted into the lumen of an anatomic vessel or a duct to keep the passage open during which the procedure is done. The term 'stenting' is used to refer to the procedure which involves the placement of these stents. Drug eluting stents consists of three parts namely, a stent platform, a coating of polymer that binds the drug to the stent and releases the drug and finally, the stent itself[1].The first procedure to treat blocked coronary arteries was coronary artery bypass graft surgery (CABG), in which a section of vein or artery from anywhere else in the body is used to bypass the diseased segment of coronary artery. In 1977, Andreas Grüntzig introduced PTCA which stands for percutaneous transluminal coronary angioplasty which is also called as balloon angioplasty, in which a catheter tube has been introduced through an artery, preferably a peripheral artery in the body and a balloon is expanded to bring the dilated narrowed segment of the artery back to normal size [2].As the equipment and techniques were improved, the use of PTCA increased by many folds, and by the mid-1980s, PTCA and CABG were both being performed at almost comparable rates and frequencies throughout the world[3].Balloon angioplasty was generally effective and safe, but restenosis which means a chance for the vessel to dilate once again was very much frequent, occurring in about 30– 40% of cases, which usually occurs within the first year after the process of dilation. In almost around 4 to 5% of balloon angioplasty cases, the dilation process may fail as a whole and acute or threatened closure of the coronary artery which can often be because of the dissection which has been prompted by an emergency CABGs [4].Two medical professionals namely, Dotter and Melvin Judkins suggested using prosthetic devices inside the arteries in the legs to maintain proper blood flow after the dilation process as early as the 1960s[5].In the year 1986, two medical professionals namely, Duel and Sigwart made a great breakthrough in the field of prosthetic stent placement by implanting the first ever coronary artery stent in human trials. This was done in the early 1990s and it showed the superiority of stent placement over theballoon angioplasty process. Restenosis was greatly reduced because of the fact that the stent acted as a scaffold to hold open the dilated segment of artery; acute closure of the coronary artery and also the requirement for emergency CABG was greatly reduced, because the stent repaired dissections of the arterial wall.

There are a wide variety of stents available and they are used for different purposes and procedures in the field of medicine. Vascular and biliary stents, expandable coronary stents and there are also simple plastic stents which is used to allow the flow of urine between

kidney and the bladder. These types are the most frequently used types of stents in the field of medicine.

- **Coronary Stents**

  Coronary stents are made of metal and will consist of three parts, an inflated balloon with a drug coated stent, a stent delivery catheter and a location marker. Coronary stents are used during an angioplasty procedure. It is also called as a percutaneous coronary intervention. Coronary stents are most commonly placed inside a coronary artery. Inside these coronary arteries, three types of stents can be placed. It can either be a drug eluting stent, bio absorbable stent or a dual therapy stent or it can also be a covered stent.

- **Vascular Stents**

  Vascular stents are placed as a part of a peripheral stent angioplasty. Common sites which are treated with peripheral artery stents include carotid, femoral and iliac arteries. Because of the compressive and the mechanical forces which are applied at this point, the stents which are associated with these regions must be flexible.

  Materials like nitinol are used for these locations.

- **Ureteral Stents**

  Ureteral stents are used to open up and ensure the patency of a ureter which might be compromised. This is usually done as a temporary measure to prevent damage to a blocked kidney until the kidney stone which is causing the blockage can be removed.

- **Oesophageal Stents**

  Oesophageal stents are generally made of metal. Oesophageal stents are important tools for palliative treatment of inoperable oesophageal malignancies [6].Oesophageal stent is a flexible mesh and is about 2cm wide. These stents are used to allow the free movement of food from the oral cavity to the stomach via the oesophagus.

- **Biliary Stents**

  Biliary stents provide the drainage of bile juice from the bile ducts, pancreas and the gall bladder to the duodenum in conditions such as ascending cholangitis due to the obstruction of the passage way by the gall stones [7].It is usually made up of plastic or metal.

- **Prostatic Stents**

  These stents are placed from the bladder through the penile urethra and prostatic urethra to allow the drainage of a bladder through the penis. This is sometimes

required in benign prostatic hypertrophy. Prostatic stents are generally made up of plastic.

- **Duodenal Stents**

  An alternative to a stomach bypass operation is the insertion of a stent, which holds the sides of the duodenum open in the same way that a stent might relieve a blocked bile duct. The aim of putting in a stent is to allow food and other substances to pass through the stomach and reduce nausea and vomiting. Usually made up of metal or plastic.

Recent Advances in Stents

Glaucoma drainage stents have been very recently introduced and they are a very recent advancement which is still awaiting approval in several countries of the world. These stents are used to reduce the intraocular pressure in the eyes by providing a drainage channel for the fluid in the eye which gets collected during glaucoma. Some of the other types of stents are colon stents, duodenal stents and pancreatic stents. These stents are also a relatively recent development in the field of stents. Different types of stents are constantly being developed.

Drug Eluting Stents

A drug eluting stent is a metal or a ceramic tube which is placed in a blocked artery to block cell proliferation by slowly releasing drugs into the blood circulation. Drugs such as glucocorticoids (dexamethasone and betamethasone), heparin, hirudin, tocopherol, angiopeptin are some of the drugs that are delivered via stents. A drug-eluting stent which is also called DES is a peripheral scaffold which can also be placed in internal organs like the heart which is placed into narrowed and diseased peripheral or coronary arteries that work by slowly releasing a drug or several drugs to block the cell proliferation. Drug-eluting stents which are in current clinical use were approved by the Food And Drug Administration after several clinical trials showed that they were statistically and mechanically superior to baremetal stents for the treatment of coronary artery narrowing, and also having lower rates of major or also minor adverse cardiac events[8]. By the late 1990s, stents were used in almost

82 to 85% of percutaneous coronary interventions. Coronary interventions represent those that are done via a catheter and not by open chest surgery [9]. Early difficulties were also present with coronary stents which included a risk of early thrombosis which was resulting in the occlusion or blockage of the stent [10]. Coating stainless steel stents with other rare earth

metal substances such as platinum or gold did not eliminate this problem of early thrombosis whatsoever. High-pressure balloon expansion of the stent to ensure its full apposition and approximation to the walls of the arteries, combined with drug therapy using aspirin and another inhibitors of platelet aggregation almost completely eliminated the risk of early stent thrombosis. Though it occurred less frequently than with balloon angioplasty or other techniques, stents still remained highly vulnerable to restenosis, caused almost exclusively by the neo-intimal tissue growth. To overcome and prevent this issue of restenosis and occlusion, developers of drug-eluting stents used the devices themselves as a tool for delivering medication directly into the arterial wall or walls. While initial efforts were not very successful, the release of drugs with certain specific physicochemical properties from the stent was shown in the early 2000s to achieve high concentrations of the drug locally and directly at the target lesion, with minimal to nil systemic side effects. As currently used in clinical practice, "drug-eluting" stents refers to the metal stents that elute or deliver a drug or several drugs that are designed to limit the growth of neo-intimal scar tissue or tissue[s], thus reducing the likelihood of stent restenosis greatly. The first successful trials were of sirolimus-eluting stents were done in the early 2000s.

A clinical trial in the early 2000s led to an approval of the sirolimus eluting or releasing Cypher stent in Europe in 2002 by the Food and Drug Administration. After a larger pivotal trial that was published in 2003, the device received FDA approval and was released in the United States of America in 2003.Soon after their initial release, a series of trials of paclitaxel-eluting stents led to Food And Drug Administration approval of the Taxus stents in early 2004.The first stents that were classified or grouped as resorbable stent that was tested in humans was developed by the Igaki Medical Planning Company in Japan and was constructed from a form of polylactic acid. They published their early results in 2000. The German company named Biotronik later developed a magnesium absorbable stent and published their clinical results in 2007.The first company to bring a bio-resorbable stent to market was a company that goes by the name of Abbott Vascular which received a European marketing approval in September of 2012; the second was Elixir which received its Clinical excellence mark in May 2013

Dexamethasone releasing stents is one of the first ever generations of drug eluting stents that were used for local drug delivery to prevent or to address the issue of restenosis. Other glucocorticoids including betamethasone are also delivered via drug releasing stents to address restenosis. Drug eluting stents were first developed in an attempt to prevent or

address the issue of restenosis. Restenosis is the closure of a peripheral or coronary artery following a trauma to that artery or surrounding the areas supplied by the artery [12]. The basic mechanism of drug delivery from several of the drug eluting scaffolds involves encapsulating or coating a drug in a polymer or a combination of different polymers that can either allow the drug to diffuse outward from it or that it undergoes degradation in order to release the drug directly into the desired site. Polymers can also be subdivided into bioerodable or non-bio-erodable categories. The bio-erodable polymers can be further subdivided into two categories that are either bulk or surface erosion [13]. For long term effects, the non-bio-erodable stents are the most commonly used stents for this purpose. There are various drugs that are delivered through stents and these drugs can also be used to prevent restenosis. These drugs fall under four major categories [14].They are antineoplastics, immunosuppressive, migration inhibitors and also enhanced healing factors [15]. Anti-neoplastic drugs like Sirolimus that is currently undergoing research as CYPHER, tacrolimus, everolimus, leflunomide, M-prednisolone, cyclosporin are all some of the examples of the anti-neoplastic drugs that are delivered through stents[16].

Anti-proliferative compounds or preparations include drugs like paclitaxel, QP-2, actinomycin, statins and many other drugs. Paclitaxel was originally used to inhibit the tumour growth by assembling or aggregating microtubules that have been found to prevent cells from multiplying. It has also recently been noted that they can attenuate neo-intimal growth [17].

Immunosupressives: Taxol, actinomycin, methotrexate, angiopeptin are delivered through stents [18]. Immunosupressive drugs are generally drugs that are used to prevent the immune rejection of allogenic organ transplants. The general mechanism of action of most of these drugs is to stop cell cycle progression, by inhibiting the DNA synthesis of these cells. Everolimus, sirolimus, tacrolimus, ABT-578, interferon, dexamethasone, and cyclosporine are all examples of drugs working on such mechanisms of action. The sirolimus derived compounds appear highly reasonable in their ability to bring down and eliminate the neointimal thickening or thickenings [19].

Migration inhibitors: Batimistat, probucol are examples of migration inhibitors [20].These compounds are aimed at preventing endothelial cell migration to the inside of the stent. Once the smooth muscle cells migrate to the other side that is, the luminal side of the stent or stents in question, they can produce extracellular matrices and can start to occlude the blood flow greatly. Therefore by inhibiting their migration, they can have great therapeutic or medicinal applications for prevention of stent restenosis. Examples of these compounds are batimastat

and halofuginone. Batimastat, for example, is a potent inhibitor of matrix metalloproteinase enzymes. It can prevent the matrix degradation that is necessary for cells to free themselves to move. If the cells cannot move, they cannot invade the stent area [20].

Enhanced Healing Factors: VEGF is an example of an enhanced healing factor [21].Vascular endothelial growth factor (VEGF) promotes healing of the vasculature. In the context of stents, this would heal the implantation site and reduce platelet sequestration due to injury related chemotaxis. Nitrous oxide donor compounds may also replicate this effect. Healing of the vessel wall seems to be the gentlest approach to preventing ISR, but healing factors are still in the early stages of development for this application [22].The release of the drugs from the stents is principally based upon the process of diffusion [23]. Most of the drug eluting stents will operate through the principle of diffusion with the exception of a few stents undergoing clinical trials like CYPHER and TAXUS. The Cypher drug eluting stent manufactured by Cordis, a Johnson and Johnson company, utilized the drug Sirolimus to combat restenosis. The Cypher stent comes in a variety of lengths and diameters. These range from 8 to 33 mm in length and 2.5 to 3.5 mm in diameter. These coronary stents are constructed out of 316L stainless steel (low-magnetic, low-carbon) and are coated with a mixture of two polymers, parylene C, and the Sirolimus drug. The two non-erodible polymers (polyethylene-co-vinyl acetate (PEVA) and poly n-butyl methacrylate (PBMA)) are combined in a 67/33 percent ratio respectively and then applied to a parylene C coated stent. A drug free coat of PBMA is also applied to the stent surface to control drug release [24]. RAVEL and SIRIUS were two clinical trials which were conducted to test the efficacy of the CYPHER system [25]. Following up on the success of the Sirolimus-eluting Cypher stent manufactured by Cordis, Scientific designed their own drug-eluting stent in hopes of creating some competition in the drug-eluting stent in hope of taking some of Cordis's monopoly. The Boston Scientific stent, called Taxus, utilizes the drug Paclitaxel. Paclitaxel is in a class of drugs called taxanes. Its main uses are to prevent the growth of cancer cells in the body, treat metastatic breast cancer, metastatic ovarian cancer, and Kaposi's Sarcoma [26]. Paclitaxel works by promoting the assembly of microtubules and stabilizes them by preventing depolymerization. This stability results in an inhibition of the reorganization of the microtubule network during the cell mitotic process – thus preventing the accumulation of anti-inflammatory cells at the site of the injury. The Taxus stent comes in lengths of eight to 28 mm and diameters of 2.5 to 3.75mm. The stent is constructed out of 316L stainless steel and is coated with the Translute polymer [poly(styrene-b-isobutylene-b-styrene)]. This polymer functions similarly to the PEVA/PBMA copolymer used in the CYPHER stent. This

polymer is also notable for its excellent vascular compatibility, which is extremely important in a system designed for long-term implementation. The pharmacokinetics of the paclitaxel release are slightly different from the CYPHER stent: Burst release in the first 48 hours [27]. Paclitaxel and CYPHER stents are polymer based stents. Metallic stents are deployed in over 1.5 million patients in a year. Polymer-based coronary stents eluting sirolimus or paclitaxel substantially reduce the need for repeat percutaneous intervention compared with bare-metal stents, and drug-eluting stents are rapidly replacing bare-metal stents. [28]. Stent thrombosis is quite common in drug eluting stents. Stent thrombosis usually results in ST-segment elevation myocardial infarction or death. Angio- graphically documented late, greater than 6 months stent thrombosis is extremely rare with bare-metal stents except after intracoronary irradiation, which can delay the vascular healing.

Conclusion

A stent is still a foreign substance that has been introduced into the body, and the body will respond to the presence of the stent in a variety of ways. Macrophagesaccumulate around the stent and there is proliferation of the smooth muscle cells nearby. These changes, can lead to restenosis, and they are limited by the drugs that can be released by the stent, but these drugs will also limit formation of an endothelial layer over the new stent that has been introduced into the body to inhibit the formation of a blood clot. Endothelialization is an important feature of the vascular healing and is very important for the prevention of the thrombus formation. Lack of healing caused by certain drugs can also make the newly inserted stent an exposed surface on to which a clot, sometimes life-threatening, can be formed. For drugeluting stents, the presence of a clot formation within the stent may persist longer than usual when compared to others. It can maybe also last for as long as five years after the treatment has ended. Drug-eluting stents have been associated with a highly delayed arterial healing process. There is also evidence that drug-eluting stents might also be susceptible to thrombosis related to delayed endothelialisation of stent struts. Most probably, late thrombosis. [29]. Studies based on animals have also raised concerns regarding the fact that late thrombosis is very common among drug delivery through drug eluting stents but this can vary among humans and evidence so far, hasn’t been suggested. Evidence from animal models suggests that the Cypher sirolimus- eluting stent does not impede endothelialisation [30]. Despite overall improvements were made in the field of antiplatelet therapy, thrombotic events remain to be a very important cause of death after a percutaneous coronary intervention surgery. Sirolimus-releasing or delivering stents and the paclitaxel releasing or

delivering stents that are a polymer based group of stents have been proven to reduce neointimal hyperplasia and also other risks of restenosis while decreasing the risk of stent thrombosis by many folds.[31] Operators are now using drug-eluting stents as the primary choice of drug delivery for a wide variety of clinical and also for a wide variety of anatomic situations, many of which have not been evaluated in several of the randomized studies that have been published.

Corresponding Author

Meenakshi Krishnan,

Senior Lecturer, Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha University,Saveetha Institute of Medical and Technical Sciences,

162, Poonamallee High Road, Chennai -600077 Tamil Nadu, India.

E-mail: drmeenakshisdc@gmail.com

Telephone Number: 8754415200

Reference

1. Nikam N, Steinberg TB, Steinberg DH. Advances in stent technologies and their effect on clinical efficacy and safety. Medical Devices (Auckland, NZ). 2014;7:165.
2. Tamburino C. Left Main Coronary Artery Disease: A Practical Guide for the Interventional Cardiologist. Springer Science & Business Media; 2009 Aug 29.
3. Bharti ML. Recent advancement in development of drug eluting stents delivery system. International Journal of Therapeutic Applications. 2012;4:1-9.
4. Dotter CT, Judkins MP. Transluminaltreatment of arteriosclerotic obstruction: description of a new technic and a preliminary report of its application. Circulation. 1964 Nov 1;30(5):654-70.
5. Woods TC, Marks AR. Drug-eluting stents. Annu. Rev. Med.. 2004 Feb 18;55:169-78.
6. Dinh TQ, Tuch RJ, Schwartz RS, inventors; Medtronic Inc, assignee. Drug eluting stent. United States patent US 5,697,967. 1997 Dec 16.
7. BhartiML. Recent advancement in development of drug eluting stents delivery system. International Journal of Therapeutic Applications. 2012;4:1-9.

8. Grüntzig AR, Senning Å, Siegenthaler WE. Nonoperative dilatation of coronaryartery stenosis: percutaneous transluminal coronary angioplasty. New England Journal of Medicine. 1979 Jul 12;301(2):61-8.
9. Serruys PW, Kutryk MJ, Ong AT (2006). "Coronary-artery stents". N. Engl. J. Med. 354 (5): 483–95.
10. Baim, Donald S. (2005) [1958]. "Percutaneous Coronary Revascularization". In Dennis L. Kasper, Anthony S. Fauci, Dan L. Longo, Eugene Braunwald, Stephen L. Hauser,& J. Larry Jameson. Harrison's Principles of Internal Medicine (16th ed.). New York: McGraw-Hill. pp. 1459–1462.
11. Libby P, Schwartz D, Brogi E, Tanaka H, Clinton SK. A cascade model for restenosis. A special case of atherosclerosis progression. Circulation. 1992 Dec;86(6 Suppl):III47-52.
12. Honari G, Ellis SG, Wilkoff BL, Aronica MA, Svensson LG, Taylor JS. Hypersensitivity reactions associated with endovascular devices. Contact dermatitis. 2008 Jul 1;59(1):7-22.
13. Honda Y. Drug-eluting stents. Circulation Journal. 2009;73(8):1371-80.
14. Mok TS, Wu YL, Thongprasert S, Yang CH, Chu DT, Saijo N, Sunpaweravong P, Han B, Margono B, Ichinose Y, Nishiwaki Y. Gefitinib or carboplatin–paclitaxel in pulmonary adenocarcinoma. New England Journal of Medicine. 2009 Sep 3;361(10):947-57.
15. Ranade SV, Miller KM, Richard RE, Chan AK, Allen MJ, Helmus MN. Physical characterization of controlled release of paclitaxel from the TAXUS™ Express2™ drug‐eluting stent. Journal of Biomedical Materials Research Part A. 2004 Dec 15;71(4):625-34.
16. Parmar M, Ledermann JA, Colombo N, Du Bois A, Delaloye JF, Kristensen GB, Wheeler S, Swart AM, Qian W, Torri V, Floriani I. Paclitaxel plus platinum-based chemotherapy versus conventional platinum-based chemotherapy in women with relapsed ovarian cancer: the ICON4/AGO-OVAR-2.2 trial. Lancet (London, England). 2003 Jun;361(9375):2099-106.
17. Tanabe K, Regar E, Lee CH, Hoye A, Giessen WJ, Serruys PW. Local drug delivery using coated stents: new developments and future perspectives. Current pharmaceutical design. 2004 Feb 1;10(4):357-67.

18. García-García HM, Vaina S, Tsuchida K, Serruys PW. Drug-eluting stents. Archivos de cardiologia de Mexico. 2006 Sep;76(3):297.
19. Hofma SH, Serruys PW. Recent developments in coated stents. Current interventional cardiology reports. 2001 Feb;3(1):28-36.
20. Fattori R, Piva T. Drug-eluting stents in vascular intervention. The Lancet. 2003 Jan 18;361(9353):247-9.
21. Maisel WH, Laskey WK. Drug-eluting stents. Circulation. 2007 May 1;115(17):e426-7.
22. Ozols RF, Bundy BN, Greer BE, Fowler JM, Clarke-Pearson D, Burger RA, Mannel RS, DeGeest K, Hartenbach EM, Baergen R. Phase III trial of carboplatin and paclitaxel compared with cisplatin and paclitaxel in patients with optimally resected stage III ovarian cancer: a Gynecologic Oncology Group study. Journal of Clinical Oncology. 2003 Sep 1;21(17):3194-200.
23. Shulze JE, Betts RE, Savage DR, inventors; Biosensors International Group Ltd, assignee. Drug-delivery endovascular stent and method for treating restenosis. United States patent US 7,682,387. 2010 Mar 23.
24. Chaudhry S, Cypher R, Ekman M, Karlsson M, Landin A, Yip S, Zeffer H, Tremblay M. Rock: A high-performance sparccmt processor. IEEE micro. 2009 Mar;29(2).
25. Kelly K, Crowley J, Bunn Jr PA, Presant CA, Grevstad PK, Moinpour CM, Ramsey SD, Wozniak AJ, Weiss GR, Moore DF, Israel VK. Randomized phase III trial of paclitaxel plus carboplatin versus vinorelbine plus cisplatin in the treatment of patients with advanced non–small-cell lung cancer: a Southwest Oncology Group trial. Journal of Clinical Oncology. 2001 Jul 1;19(13):3210-8.
26. Muskopf A. Cardiovascular stents. Journal of Clinical Engineering. 2007 Jan 1;32(1):34-7.
27. McFadden EP, Stabile E, Regar E, Cheneau E, Ong AT, Kinnaird T, Suddath WO, Weissman NJ, Torguson R, Kent KM, Pichard AD. Late thrombosis in drug-eluting coronary stents after discontinuation of antiplatelet therapy. The Lancet. 2004 Oct 23;364(9444):1519-21.
28. Kałuza GL, Joseph J, Lee JR, Raizner ME, Raizner AE. Catastrophic outcomes of noncardiac surgery soon after coronary stenting. Journal of the American College of Cardiology. 2000 Apr 1;35(5):1288-94.

29. Wilson SH, Fasseas P, Orford JL, Lennon RJ, Horlocker T, Charnoff NE, Melby S, Berger PB. Clinical outcome of patients undergoing non-cardiac surgery in the two months following coronary stenting. Journal of the American College of Cardiology. 2003 Jul 16;42(2):234-40.
30. Colombo A, Drzewiecki J, Banning A, Grube E, Hauptmann K, Silber S, Dudek D, Fort S, Schiele F, Zmudka K, Guagliumi G. Randomized study to assess the effectiveness of slow-and moderate-release polymer-based paclitaxel-eluting stents for coronary artery lesions. Circulation. 2003 Aug 19;108(7):788-94.
31. Iakovou I, Schmidt T, Bonizzoni E, Ge L, Sangiorgi GM, Stankovic G, Airoldi F, Chieffo A, Montorfano M, Carlino M, Michev I. Incidence, predictors, and outcome of thrombosis after successful implantation of drug-eluting stents. Jama. 2005 May 4;293(17):2126-30.

Drug Influencing Tooth Movement in Orthodontic Patient-A Review

Preethi Pavithra.R [1], Anitha Roy [2] , Asha Ramesh [3]

Graduate Student [1], Senior Lecturer [2],Department of Pharmacology

Senior Lecturer [3],Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Orthodontic treatment is based on the premise that when force is delivered to a tooth and thereby transmitted to the adjacent investing tissues, certain mechanical, chemical, and cellular events take place within these tissues, which allow for structural alterations and contribute to the movement of that tooth. Molecules present in drugs and nutrients consumed regularly by patients can reach the mechanically stressed paradental tissues through the circulation and interact with local target cells. The combined effect of mechanical forces and one or more of these agents may be inhibitory, additive, or synergistic. Current orthodontic research aims to develop methods of increasing the tissue concentration of molecules promoting tooth movement, while simultaneously decreasing the concentration of unwanted elements which can produce harmful side effects.The purpose of this review is to summarize the effects of some commonly used drugs on tissue remodeling and orthodontic tooth movement. A thorough knowledge about the drugs is therefore mandatory for the orthodontist dealing with patients under orthodontic treatment.

Key Words: Hormones, immunomodulatory drugs, immunosuppressant drugs, NSAIDs, orthodontic tooth

Introduction

Tooth movement is the key principle behind any orthodontic treatment. Orthodontic tooth movement is mainly a biological response towards mechanical force. It is induced by the prolonged application of controlled mechanical force on the tooth[1]. Molecules present in drugs and nutrients consumed regularly by patients can reach the mechanically stressed paradental tissues through the circulation and interact with local target cells. The combined effect of mechanical forces and one or more of these agents may be inhibitory, additive, or synergistic.[2] It is induced by the prolonged application of controlled mechanical force on the tooth, which eventually causes remodeling of the tooth socket by creating pressure and tension zones in the alveolar bone and periodontal ligament[1-2]. Strains of periodontal cells, bone-related cells, and extracellular matrix play a key role in orthodontic tooth movement (OTM)[3]. This strain forms changes in gene expression in the cells by interactions between the cells and the extracellular matrix, whereby integrins play an important role[4]. Various cell-signaling pathways are activated, ultimately leading to stimulation of periodontal ligament metabolism, and localized bone resorption and bone deposition.These interactions are regulated by local factors such as cytokines (IL-1), and growth factors, as well as by systemic factors such as parathyroid hormone, vitamin D, estrogen, or caicitonin

Rate of orthodontic tooth movementdepends on the amount of force applied, type of force appliedand the presence or absence of biologic modifiers thataffect bone metabolism. Different pharmacological agents aretried as biologic modifiers to alter the rate of orthodontic tooth movement. These interactions are regulated by local factors such as cytokines (IL-1), and growth factors, as well as by systemic factors such as parathyroid hormone, vitamin D, estrogen, or caicitonin. Drugs that alter or interfere with the inflammatory process will therefore have an effect on the tooth movement. Any pharmacologic agent or supplement consumed by a patient can reach the periodontal tissues through circulation and thus interacts with and influence a cell's response to orthodontic forces.[5]. These drugs may have dual effect on orthodontic tooth movement. They may either potentiate or inhibit tooth movement as well as exacerbate or reduce tooth resorptionThese drugs can be classified as drugs that potentiate or increase tooth movement

and drugs that inhibit or decrease tooth movement. As a clinician, this information is very important because orthodontic treatment does involve postoperative pain and the form of pain management is very important because not all drugs favours tooth movement. Many patients use over the counter medications for immediate pain relief, which may interfere with the treatment plan. Therefore, practitioners should have a proper knowledge of the drugs being prescribed and the patient should be well informed. In this study it is discussed about the effect of short and long term administration of medication on orthodontic tooth movement.[6]

Methods of Accelerating Tooth Movement

There are three phases of tooth movement: the initialphase, which is characterized by rapid movement afterthe application of force; followed by a lag period, wherelittle or no movement, and the last phase, where gradualor sudden increase of movement occurs.The early phase of tooth movement involves acute inflammatoryresponses characterized by leucocytes migratingout of blood capillaries and producing cytokines,which stimulates the excretion of prostaglandins andgrowth factors . The acute phase is followed by the chronic phase that involves the proliferation of fibroblast,endothelial cells, osteoblasts, and alveolar bonemarrow cells remodeling process.[7]

Drugs Affecting Tooth Movement

According to WHO (1966), drug is any substance or product that is used to modify or explore physiological systems or pathological states for the benefit of the recipient. During orthodontic treatment, drugs are prescribed to manage pain from force application to biological tissues, manage temporomandibular joint (TMJ) problems and tackle some infectionthroughout the course of treatment. Some of the drugs that have effect are on orthodontic tooth movement are Bisphosnates,Vitamin D,Fluoride,NSAID,Immunosuppressant Drugs,Anticancer Drugs And Hormones Like Estrogen Thyroid,Prostaglandin,Echistatin And RGD Peptide,Parathyroid,Corticosteroids.[8]

Bisphosphonates

Bisphosphonates (BPNs) have strong chemical affinity to the solid-phase surface of calcium phosphate; this causes inhibition of hydroxyapatite aggregation, dissolution, and crystal formationBisphosphonates are synthetic class of pyrophosphate analogues and they are powerful inhibitors of bone resorption.They act by inhibiting the osteoclastic activity and decreasing the number of osteoclasts.This leads to inhibition of orthodontic tooth movement and hence delays orthodontic treatment.The Topical application of bisphosphateis known to be very useful in anchoring and retaining teeth under orthodontic treatment. Bisphosphonates cause a rise in intracellular calcium levels in osteoclastic-like cell line, reduction of osteoclastic activity, prevention of osteoclastic development from hematopoietic precursors and production of an osteoclast inhibitory factor.[9]

Vitamin D3

Vitamin D3 is an important regulator of calcium homeostasis. 1,25 dihydroxycholecalciferol (1,25[OH]2D3) is the active metabolite of vitamin D3. Together with parathyroid harmones and calcitonin help in regulating the calcium and phosphate serum levels in the body. It also promotes the calcium and phosphate absorption in the intestine and reabsorption in the kidneys. Recent studies have proven that Vitamin D3 is very effective in treating osteoporosis and this explains how Vitamin D3 is considered as an active suppressor drug. It has been proven that it increases the bone mass, thus reduces fractures in osteoporotic patients[10].But some authors consider vitamin D3 to be a resorption promoting agent because it has stimulatory effects on osteoclasts23. Collins in 1988[11], demonstrated that local application of vitamin D3 improves the rate of tooth movement in rats. This result was due to the well-balanced bone turnover induced by vitamin D3. So more studies have to be conducted in determining the exact role of Vitamin D3 in orthodontic tooth movement.

Fluoride

Fluoride is one of the trace elements having an effect on tissue metabolism. Fluoride increases bone mass and mineral density, and because of these skeletal actions, it has been used in the treatment of metabolic bone disease, osteoporosis.active caries treatment by application of

sodium fluoride during treatment course may delay orthodontic tooth movement and increase the time of orthodontic treatment Sodium fluoride has been shown to inhibit the osteoclastic activity and reduce the number of active osteoclasts.[12]

NSAID

Most commonly used medications in orthodontics are for control of pain following mechanical force application to tooth. Nonsteroidal Anti-Inflammatory Drugs or generally called as NSAIDS is a very common drug in pain control.They have analgesic, antipyretic, and anti-inflammatory effects, and are prescribed for many conditions, such as rheumatoid arthritis, osteoarthritis, gout, dysmenorrhea, headache, migraine, and postoperative pain, as well as for the prevention of cardiovascular diseases and colorectal cancer. In cases of pain and headache, NSAIDs are taken incidentally because it is freely available over the counter.[13]

Acetylsalicylic acid and the related compounds, and their action result from inhibition of COX activity, which converts unsaturated fatty acids in the cell membrane to PGs Clinical experience shows that orthodontic tooth movement is very slow in patients undergoing long-term acetylsalicylic therapy.

Salicylate therapy decreases bone resorption by inhibition of PGs' synthesis and may effect differentiation of osteoclasts from their precursors. Therefore, it is recommended that patients undergoing orthodontic treatment should not be advised to take aspirin and related compounds for longer period during orthodontic treatment.[14]

Paracetamol has become one of the most popular antipyretic and analgesic drugs worldwide, and it is often also used in combination with other drugs which is a weak COX-1 and COX-2 inhibitor. The difference between NSAIDs and paracetamol is, NSAIDs acts by blocking COX-1 and COX-2, whereas paracetamol acts on a third isoform, COX-3 , which is expressed only in the brain and the spinal cord. As a result, paracetamol has minimal effects on prostaglandin synthesis In 1997, Roche JJ et al. reported that acetaminophen showed no effect on tooth

movement when tested on rats. paracetamoldoes'nt affect the orthodontic tooth movement, so it's safe to use as a choice of pain management in orthodontic treatment.[15]

Immunosuppressant Drugs

Patients with chronic renal failure or kidney transplants and on immunosuppressant drugs can encounter some difficulty during orthodontic treatment. Drug consumed for prevention of graft rejection (cyclosporine A) produce severe gingival hyperplasia, making orthodontic treatment and maintenance of oral hygiene difficult. Treatment should be started or resumed after surgical removal of excessive gingival tissues once there is good oral hygiene. Whenever possible, fixed appliances should be kept to a minimum period with brackets and avoiding the use of cemented bands. Removable appliances in these cases are not recommended due to improper fit.[16]

Anticancer Drugs

These are used for the treatment of childhood cancers. There is every chance of observing disturbances in dental as well as general body growth and development due to the adverse effects of the chemotherapeutic agents.Patients who had undergone chemotherapy with busulfan/cyclophosphamide belong to the risk group for orthodontic treatment.These drugs are known to produce damage to precursor cells involved in bone remodeling process, thereby complicating tooth movement.[17]

Phenytoin

Phenytoin results in gingival hyperplasia due to increased amountof gingival collagen fibers, which involve the interdental papilla, making application of orthodontic mechanics and maintaining oral hygiene difficult. If used during pregnancy, it can produce fetalhydantoin syndrome characterized by hypoplastic phalanges, cleft palate, hare lip, and microcephaly.[18] Valproic acid has a potential to induce gingival bleeding even with minor trauma, making orthodontic maneuvers difficult. Gabapentin of phenytoin group produces xerostomia, making oral hygiene maintenance difficult during orthodontic treatment.

Alcohol Abuse

Alcohol crosses the placental barrier and can stunt fetal growth or weight, create distinctive facial stigmata, damage neurons and brain structures, which can result in psychological or behavioral problems, and cause other physical damage (Fetal Alcohol Syndrome or FAS). The three FAS facial features are a smooth philtrum, thin vermilion, and small palpebral fissures. Chronic ingestion of large amounts on a daily basis may have devastating effects on a number of tissue systems, including skeletal system. Circulating ethanol inhibits the hydroxylation of vitamin D3 in liver, thus impending calcium homeostasis. In such cases, the synthesis of PTH is increased, tipping the balance of cellular function toward the enhanced resorption of mineralized tissues, including root resorption, in order to maintain normal levels of calcium in blood.[19] Davidovitch . have found that chronic alcoholics receiving orthodontic treatment are at high risk of developing severe root resorption during the course of orthodontic treatment.[20]

Hormones

Prostaglandins

The Prostaglandins are a chemical messengers called eicosanoids. It acts by regulating the synthesis of cyclic AMP in many tissues. Cyclic AMP is responsible in controlling the action of various hormones. This allows prostaglandin to affect a wide range of cellular and tissue functions. Prostaglandins are responsible in stimulating contraction of the smooth muscles of the uterus, affects blood flow, sleepcycle and also response to hormones such as adrenaline and glucagon. It also plays a role in elevating body temperature, which leads to inflammation and pain. According to Klein and Dowsett, prostaglandins play an significant role in promoting bone resorption. It is believed that, prostaglandins promote resorption by stimulating cells to produce cyclic AMP, which is a very important chemical messenger for bone resorption. In 1973, Goldhaber, reported that there is an increase in level of prostaglandins in periodontal diseases. In Orthodontics, Yamasaki and his teamswere the first to introduce the use of prostaglandins in controlling the rate of tooth movement. Results have showed that the local administration of PGE1 or PGE2 in the gingiva near the distal area of canines to be retracted, caused double the rate of tooth movement compared to the opposite, control side. . Studies on humans were

conducted in 1984, where Yamasaki studied the effects of PGE1 administration on orthodontic tooth movement. In the result the amount of tooth movement was twice compared to control sides. Lee[22] in a later study, compared lidocaine and PGE 1and reported that PGE 1 was more effective in bone resorption but it had certain side effects such as local irritation and phlebitis. Investigation in humans by Spielmann in 1989 with a split-mouth design showed significant increases in the rate of palatal premolar movement after multiple local injections of PGE1 at a dosage of 10 g. In india, Bhalajhi and Shetty conducted a study on the effect of exogenous administration of PGE2 in young rabbits. At the end there was a major increase in the amount of tooth movement clinically, and microscopically there were increase in the number of osteoclasts and resorption of lacunae. But there was a frequent need of administration of the drug as PGE2 gets metabolised rapidly in the lungs.

Estrogen

Estrogens do not appear to have any anabolic effects on bone tissue but there are studies indicating that estrogen directly stimulate the bone-forming activity of osteoblasts.They act by inhibiting interleukin-1(IL-1), tumour necrosis factor-a (TNF-a), and interleukin 6 (IL-6} which appears to be involved in bone resorption by stimulating osteoclastic activity.Oral contraceptive pills contains estrogen, when taken by younger woman for a long span, it can influence the rate of tooth movement.Therefore, it is extremely important for the dentist to consider this factor during history taking and treatment planning in females.[23]

Leukotrienes

Leukotrienes are a type of eicosanoid which is a product of arachidonic acid conversion and are the only eicosanoids that are formed independently from cyclooxygenase (COX). They are produced when arachidonic acid is metabolised by lipoxygenase enzymes[24].Leukotrienes also play an important role in Inflammation, allergies, and diseases such as asthma. These conditions can be cured by using leukotriene inhibitors which block leukotriene receptors hence counteracts their effects. Examples of medication are montelukast and zafirlukast. According to Mohammed AH 1989, leukotrienes causes

increase in orthodontic tooth movement, through bone remodeling whereas, leukotrine inhibitors work the other way round.Therefore, the use of leukotriene inhibitors can delay orthodontic treatment, leukotrienes can be used in future clinical applications that could result in increasing tooth movement.

Parathyroid Hormones

The function of parathyroid is to maintain a normal level of diffuse calcium and phosphorus in the blood plasma and to keep constant the ratio of these minerals to each other. The act as a check on the thyroid gland parathyroids are important organs in ca metabolism and play a leading role in calcification of teeth.After the complete development of tooth, there is no evidence of clinical case found with calcium withdrawal from teeth due to parathyroid disturbances. The parathyroid's are important in regulating blood ca level, but have little or no direct effect on growth or tooth eruption. PTH affects osteoblasts' cellular metabolic activity, gene transcriptional activity, and multiple protease secretion. Its effects on osteoclasts occur through the production of RANK-L Receptor activator of nuclear factor kappa -B legend), a protein playing a crucial role in osteoclasts' formation and activity[25]. In 1970s, animal studies demonstrated the Parathyroid hormone affects both bone resorption and formation process PTH could induce an increase in bone turnover that would accelerate orthodontic tooth movement. These results indicate that orthodontists should take note of patients being treated with PTH.[26]

Corticosteroids

Evidence indicates that the main effect of corticosteroid on bone tissue is direct inhibition of osteoblastic function and thus decreases total bone formation. Decrease in bone formation is due to elevated PTH levels caused by inhibition of intestinal calcium absorption which is induced by corticosteroids. Corticosteroids provokes the level of tooth movement, and since new bone formation becomes tough in a Patients who undergone treatment , they reduce the stability of tooth movement and stability of orthodontic treatment in general.When they are used for longer periods of time, the main side effect is osteoporosis is greater, but tooth movement is less stable since little bone is present and there is no indication of bone formation. These drugs are used as anti inflammatory and immunosuppressive agent in treatment of a wide range of chronic medical

conditions.[27] A low dose(1mg/kg body weight)decreases orthodontic tooth movement by suppressing osteoclastic activity. At an higher dose(15mg/kg body weight)it provokes the bone resorption activity thus results in more rapid orthodontic tooth movement and subsequent relapse The main effect of corticosteroid on bone tissue is direct inhibition of osteoblastic function and thus the decrease of total bone formation.[28] Decrease in bone formation is due to elevated parathyroid hormone levels caused by inhibition of intestinal calcium absorption which are induced by corticosteroids. [29]

Echistatin and RGD peptides

Echistatin is a strong inhibitor of tooth resorption.Therapeutic administration of eicosanoids resulted in increased tooth movement, whereas their blocking led to a decrease.olce et al made the first attempt in this aspect and reported that ELVAX-40 (a non-biodegradable,non-inflammatory, sustained release polymer) could be used to deliver integrin inhibitors like echistatinand RGD peptide agents (known to perturb bone remodeling), to decrease tooth movement at a particular level.[30]Recent research has even demonstrated decrease in root resorption following orthodontic force application after administration of echistatin.[31]

Thyroid Hormone

Thyroid hormones are recommended for the treatment of hypothyroidism and used after thyroidectomy in substitutive theraphy. Thyroxin administration lead to increased bone remodeling, increased bone resorptive activity and reduced bone density.Administration of thyroxine will lead to increase in bone remodeling, increase in bone resorption activity and reduces bone density.[32] Thyroxin produces interleukin 1 (IL-IB),a type of cytokine which involves in bone formation through osteoclastic reaction.Studies on rat have been conducted to determine the relationship between exogenous thyroxine and tooth movement. Results show that there was a significant increase in orthodontic movement compared to the control[33].

The thyroid hormone increases the speed of orthodontic tooth movement in patients undergoing such medication. Low dosage and short-term thyroxine administration are reported to lower the frequency of “force-induced” root resorption[34]. Reduced osteoclastic activity can may be

compared to a change in bone remodeling process and a reinforcement of the protection of the cementum and dentin to “force-induced” osteoclastic resorption.[35]

Discussion

This systematic review of literature summarizes the effects of medications, such as anti-inflammatory and anti-asthmatic , antiarthritics, analgesics, corticosteroids, estrogens and other hormones, and calcium regulators in orthodontic tooth movement. As described by Krishnan V and Davidovitch Z these groups of drug has an effect on OTM. Some of these drugs are promoter drugs where it promotes orthodontic tooth movement, but others have an inhibitory effect. Eicosanoids such as prostaglandins and leukotrienes are group of drugs, which increases the rate of OTM.They act by stimulating bone resorption. Eicosanoid inhibitors on the other hand acts in preventing OTM.[36] Example of eicosanoid inhibitors are NSAIDs where it inhibits the synthesis of prostanoids which is an important mediator of bone resorption. So, it is important that the patient does not take NSAIDs such as aspirin or other related compounds for long periods of time during orthodontic treatment[37]. The alternative that can be suggested to patients is paracetamols. Paracetamol also known as acetaminophen is a type of analgesic, which does not have any deleterious effect on OTM. Role of vitamin D3 and Corticosteroids in OTM still remains unclear. Some author have reported that it promotes tooth movement but there are also studies that demonstrate bone formation after application of these drugs. So, it is important to have more clinical trials on determining the exact role of Vitamin D3 and Corticosteroids in orthodontic tooth movement.[38] Bisphosphonates are drugs which also effect the calcium homeostasis. It is known for its role in inhibiting tooth movement. They are used in cases of prevention of orthodontic relapse but it should be used with great caution because of its severe side effects on long term use. Hormones also play an important role in tooth movement.[39] Hormones such as thyroxin is known to increase the rate of tooth movement by directly stimulating the action of osteoclast.Calcitonin and estrogen have the opposite effect on tooth movement. It is very important to know if the patient is under any oral contraceptive pills. It containsestrogens, which inhibits tooth movement.[40]

Conclusion

This review of literature summarizes the effects of medications, such as, analgesics, corticosteroids, estrogens and other hormones, and calcium regulators in orthodontic tooth movement. Some of these drugs are promoter drugs where it promotes orthodontic tooth movement, but others have an inhibitory effect. Hormones also play an important role in tooth movement. Hormones such as thyroxin is known to increase the rate of tooth movement by directly stimulating the action of osteoclast.Calcitonin and estrogen have the opposite effect on tooth movement. It is very important to know if the patient is under any oral contraceptive pills. It contains estrogens, which inhibits tooth movement.

Orthodontists have long observed that teeth move at different rates and individuals differ in their response to treatment. Some of the differences are caused by change in bone remodeling induced by drugs and systemic factors. All the drugs reviewed have the therapeutic effects as well as side effects that influence the cells targeted by orthodontic forces. The value of a thorough medical history is increasingly significant as young and old alike are exposed to a greater range of therapeutic agents. Therefore, it is imperative that the orthodontists need to pay attention to drug consumption and history of each and every patient, before and during the course of orthodontic treatment, so that the best treatment can be selected for each case.

Corresponding author

Asha Ramesh

Senior Lecturer,Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University

Chennai-600077

Mobile: +91 9841722113.

Email: doc.ash4587@gmail.com.

References

1. Batmaraj R, Umashankar K. The effects of commonly used drugs on orthodontic tooth movement: A systematic review. Asian J Pharm Clin Res. 2014;7(1):10-4.
2. Diravidamani K, Sivalingam SK, Agarwal V. Drugs influencing orthodontic tooth movement: An overall review. Journal of pharmacy &bioallied sciences. 2012 Aug;4(2):S299.
3. Nimeri G, Kau CH, Abou-Kheir NS, Corona R. Acceleration of tooth movement during orthodontic treatment-a frontier in orthodontics. Progress in orthodontics. 2013 Dec;14(1):42.
4. Collins MK, Sinclair PM. The local use of vitamin D to increase the rate of orthodontic tooth movement. American Journal of Orthodontics and Dentofacial Orthopedics. 1988 Oct 1;94(4):278-84
5. Tyrovola JB, Spyropoulos MN. Effects of drugs and systemic factors on orthodontic treatment. Quintessence international. 2001 May 1;32(5).
6. Kale S, Kocadereli I, Atilla P, Aşan E. Comparison of the effects of 1, 25 dihydroxycholecalciferol and prostaglandin E2 on orthodontic tooth movement. American journal of orthodontics and dentofacial orthopedics. 2004 May 1;125(5):607-14
7. Masella RS, Meister M. Current concepts in the biology of orthodontic tooth movement. American Journal of Orthodontics and Dentofacial Orthopedics. 2006 Apr 1;129(4):458-68
8. Arias OR, Marquez-Orozco MC. Aspirin, acetaminophen, and ibuprofen: their effects on orthodontic tooth movement. American Journal of Orthodontics and Dentofacial Orthopedics. 2006 Sep 1;130(3):364-70.
9. Vayda P, Loveless J, Miller R, Theroux K. The effect or short term analgesic usage on the rate of orthodontic tooth movement. Indian Journal of Dental Research 2000 79;1:614-18.
10. Roche JJ, Cisneros GJ, Acs G. The effect of acetaminophen on tooth movement in rabbits. The Angle Orthodontist. 1997 Jun;67(3):231-6.

11. Shdayfat NA. Effects of drugs on periodontal tissue remodeling and clinical responses to orthodontic mechanotherapy. Pakistan Oral & Dental Journal. 2011 Dec 1;31(2):45-48
12. Krishnan V, Davidovitch Z. The effect of drugs on orthodontic tooth movement. Orthodontics & craniofacial research. 2006 Nov 1;9(4):163-71
13. Boivin G, Vedi S, Purdie DW, Compston JE, Meunier PJ. Influence of estrogen therapy at conventional and high doses on the degree of mineralization of iliac bone tissue: a quantitative microradiographic analysis in postmenopausal women. Bone. 2005 Mar 1;36(3):562-7
14. Sabuncuoglu FA, Esenlik E. Influence of drugs on orthodontic tooth movement. Pakistan Oral & Dental Journal. 2010 Dec 1;30(2):45-49
15. Soma S, Iwamoto M, Higuchi Y, Kurisu K. Effects of continuous infusion of PTH on experimental tooth movement in rats. J Bone Miner Res 1999;14:546-54.
16. Kalia S, Melsen B, Verna C. Tissue reaction to orthodontic tooth movement in acute and chronic corticosteroid treatment. Orthod Craniofac Res 2004;7:26-34.
17. Reitan, K. Some factors determining the evaluation of forces in orthodontics. Am J Orthod 1957;43:32-45.
18. Nimeri, G., Kau, C. H., Abou-Kheir, N. S., Corona, R. Acceleration of tooth movement during orthodontic treatment-a frontier in Orthodontics. Progress in orthodontics. 2013;14(1):42.
19. Dolce C, Vakani A, Archer L, Morris-Wiman JA, Holliday LS. Effects of echistatin and an RGD peptide on orthodontic tooth movement. J Dent Res 2003;82:682-6.
20. Shirazi M, Dehpour AR, Jafari F. The effect of thyroid hormone on orthodontic tooth movement in rats. J Clin Pediatr Dent 1999;23: 259-64.
21. Schoutens A, Laurent E, Markowicz E, Lisart J, De-Maertelaer V. Serum triiodothyronine, bone turnover and bone mass changes in euthyroid pre and postmenopausal women. Caleif Tissue Int 1991;49:95-100.
22. Roodman GD. Role of cytokines in the regulation of bone resorption. Calcified tissue international. 1993 Feb 1;53(1):S94-8.
23. Shirazi M, Dehpour AR, Jafari F. The effect of thyroid hormone on orthodontic tooth movement in rats. The Journal of clinical pediatric dentistry. 1999;23(3):259-64.

24. Klaushofer K, Hoffmann O, Gleispach H, Leis HJ, Czerwenka E, Koller K, Peterlik M. Bone- resorbing activity of thyroid hormones is related to prostaglandin production in cultured neonatal mouse calvaria. Journal of bone and mineral research. 1989 Jun 1;4(3):305-12.
25. KLEIN DC, RAISZ LG. Prostaglandins: stimulation of bone resorption in tissue culture. Endocrinology. 1970 Jun 1;86(6):1436-40.
26. Dowsett M, Eastman AR, Easty DM, Easty GC, Powles TJ, Neville AM. Prostaglandin mediation of collagenase-induced bone resorption. Nature. 1976 Sep;263(5572):72.
27. Goldhaber P, Rabadjija L, Beyer WR, Kornhauser A. Bone resorption in tissue culture and its relevance to periodontal disease. The Journal of the American Dental Association. 1973 Oct 1;87(5):1027-33.
28. Yamasaki K, Shibata Y, Fukuhara T. The effect of prostaglandins on experimental tooth movement in monkeys (Macacafuscata). Journal of dental research. 1982 Dec;61(12):1444-6.
29. Singh VP, Roychodhury S, Nigam P. Drug Induced Orthodontic Tooth Movement-A Review. Journal of Advanced Medical and Dental Sciences Research. 2015 Jan 1;3(1):191.
30. Watted N, Proff P, Péter B, Muhamad AH. Influence of drugs on orthodontic tooth movement. Journal of Research in Medical and Dental Science. 2017 Mar 25;2(4):9-16.
31. Shirazi M, Dehpour AR, Jafari F. The effect of thyroid hormone on orthodontic tooth movement in rats. The Journal of clinical pediatric dentistry. 1999;23(3):259-64.
32. Krishnan V, Davidovitch Z. The effect of drugs on orthodontic tooth movement. Orthodontics & craniofacial research. 2006 Nov 1;9(4):163-71
33. Brandi ML, Crescioli C, Tanini A, Frediani U, Agnusdei D, Gennari C. Bone endothelial cells as estrogen targets. Calcified tissue international. 1993 Nov 1;53(5):312-7.
34. Liu L, Igarashi K, Haruyama N, Saeki S, Shinoda H, Mitani H. Effects of local administration of clodronate on orthodontic tooth movement and root resorption in rats. Eur J Orthod 2004;26:469-473.

35. . Igarashi K, Mitani H, Adachi H, Shinoda H. Anchorage and retentive effects of a bisphosphonate (AHBuBP) on tooth movements in rats. Am J Orthod Dentofacial Orthop 1994;106:279-289
36. Zahrowski JJ. Bisphosphonate treatment: an orthodontic concern calling for a proactive approach. Am J Orthod Dentofacial Orthop 2007;131:311-320
37. Chumbley AB, Tuncay OC. The effect of indometacin (an aspirin-like drug) on the rate of orthodontic tooth movement.Am J Orthod 1986;89:312-400.
38. Komm BS, Terpening CM, Benz DJ, Graeme KA, Gallegos A, Korc M,. Estrogen binding, receptor mRNA and biologic response in osteoblast-like ostcosarcoma cells. Science 1988;241:81-84
39. Eriksen EF. Colvard DS, ßerg NJ, Graham ML, Mann KG, Spelsberg TG. Evidence of estrogen receptors in normal human osteoblast-Iike cells. Science 1988:241:84-86.
40. Gray TK, Flynn TC, Gray KM. Nabell LM. 17 beta-estradiol acts directly on the clonal osteoblastic cell line UMR106. Proc Natl Acad Sci U S A 1987; 84:6267-6271.

Drug Metabolism of NSAID's

Priyadharshni S[1], Anitha[2], E. Roja[3]

Graduate Student[1], Reader[2], Department of Pharmacology,Tutor[3], Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Drug metabolism is the biochemical modification of pharmaceutical substances or xenobiotics respectively by living organisms, usually through specialized enzymatic systems. Drug metabolism often converts lipophilic chemical compounds into more readily excreted hydrophilic products. Non steroidal anti inflammatory medicines(NSAIMs) are a class of drugs that provides analgesic and antipyretic effects, and, in higher doses, anti inflammatory effects. NSAIDs are metabolised primarily in the liver. They vary in half-lives and bio availability. They are classified as mild analgesics. NSAIDs inhibit the activity of both cyclooxygenase-1 (COX-1) and cyclooxygenase-2 (COX-2), and thereby, the synthesis of prostagladins and thromboxane. NSAIDs are usually used for the treatment of acute or chronic conditions where pain and inflammation are present. The widespread use of NSAIDs has meant that the adverse effects of these drugs have become increasingly prevalent. NSAIDs, like all drugs, may interact with other medications.NSAIDs are mostly excreted as phase-II glucouronides and in a few cases as sulfate conjugates. Drug metabolism has an important role in the determination of the pharmacokinetic parameters like oral bioavailability, clearance and the half-life of the entity within the cell. The drug metabolic studies help to screen the compounds based on their metabolic rate. The various metabolic pathways of NSAIDs are reviewed.

Key Words:*Drugs, anti-inflammatory, metabolism, enzymes, liver*

Introduction

NSAIDs are non-steroidal anti-inflammatory drugs, also known as NAIDs, non-steroidal anti-inflammatory agents/analgesics (NSAIAs) or non-steroidal anti-inflammatory medicines (NSAIMs). They are medications with analgesic, antipyretic effects.In higher doses they also have anti-inflammatory effects - they reduce inflammation. Non-steroidal distinguishes NSAIDs from other drugs which contain steroids, which are also anti-inflammatory. NSAIDs are non-narcotic (they do not induce stupor). All NSAIDs are rapidly absorbed from the gastrointestinal tract, and thus almost all NSAIDs are available as oral preparations. NSAIDs are metabolised by the liver and excreted through urine. NSAIDs are usually used for the treatment of acute or chronic conditions wherepain and inflammation are present. The most prominent members of this group of drugs,aspirin, ibuprofen,diclofenac and naproxen are all available over the counter in most countries. Paracetamol (acetaminophen) is generally not considered an NSAID because it has only little anti-inflammatory activity. It treats pain mainly by blocking COX-2 mostly in the central nervous system, but not much in the rest of the body. This article reviews the metabolic pathways of various NSAIDs. It is generally accepted that non steroidal anti- inflammatory drugs (NSAIDs) act by inhibiting the synthesis of prostaglandins (PGs); however, this hypothesis may not account for all of the effects of NSAIDs at all dosages. For example, higher doses of NSAIDs are needed to suppress inflammation than are required to inhibit PG synthesis; moreover, sodium salicylate is an ineffective inhibitor of cyclooxygenase at concentrations used clinically. Alternative hypotheses have therefore been proposed in an attempt to explain fully the anti-inflammatory effects of NSAIDs, including their capacity at higher concentrations to blunt the functions of such inflammatory cells as the neutrophil. NSAIDs have also been found to disrupt a variety of processes at the plasmalemma that regulate the generation of twin signals. These effects, most likely due to the capacity of planar lipophilic NSAIDs to insert into the lipid bilayer, result in the disruption of signal transduction through the plasma membrane. Indeed, NSAIDs even inhibit activation of the primitive cells of marine sponges that neither respond to stable PGs nor contain a cyclooxygenase.

Whereas the inhibition of PG synthesis by NSAIDs is a satisfactory explanation for many of the pharmacologic actions and toxicities of NSAIDs, further studies are needed to clarify the entire spectrum of their anti-inflammatory activities.

Chronic pain remains a significant source of dis- tress and disability, particularly in the elderly population. It is estimated that 20% of individuals suffer from various forms of chronic pain worldwide [1]. Among this population, over 30 million people utilisenon-steroidal anti-inflammatory drugs (NSAIDs) for treat- ment of their discomfort. Approximately half of these patients are over the age of 65 years and use NSAIDs on a daily basis [2,3]. It is these patients that have the most comorbidity, which places them at highest risk of side effects. As the population ages, it is anticipated that the use of chronic pain medications for degenerative joint disease and other chronic ailments will only continue to increase. The most common pain medications used in these patients are NSAIDs as an alternative to opioid analgesics. Although initially viewed as a safer alternative in this particular population, emerging evidence suggests that the adverse effects on a variety of end organ systems may sometimes outweigh the analgesic benefits. Additionally, treatment costs of NSAID-induced cardiovascular (CV) and gastrointestinal (GI) side effects may negate the cost–effectiveness of oral administration of this class of medications when compared with topical preparations as measured by quality-adjusted life years [4].

Metabolism

The process by which the body brings about chemical changes in drug molecule is variously referred to as Drug metabolism or Bio transformation. Liver is the main organ involved in the process of bio-transformation. The enzymes involved in the biotransformation of drugs are distinct from those involved in carbohydrate, protein, and fat metabolism. These enzymes are called microsomal or nonmicrosomal. Microsomal enzymes are present in the smooth endoplasmic reticulum of the liver, kidneys, and gastrointestinal tract. Other enzymes are hydroxylase, dehydrogenase, reductase, glucuronly transferase, GSH-S-transferase, etc. Microsomal enzymes are responsible for glucuronide and some glutathione conjugations, most of the oxidation reactions, some reduction and hydrolysis reaction. These enzymes accept primarily the substrates of high lipid/ water partition coefficient. The activity of these enzymes can be stimulated (induced) or inhibited by prior or concomitant treatment with drugs. Nonmicrosomal enzymes are present in the cytoplasm, mitochondria and extracellular spaces of different organs. High concentrations are found in the liver, plasma, kidneys and other tissues. Other enzymes are esterase, amidase, hydrolase, sulfotransferase, GSH-S transferase, etc. They catalyze all conjugation reactions (except glucuronide and some of the glutathione conjugation), hydrolysis, and some oxidation and reduction reactions. The

substrate of high lipid/water partition coefficient. Most of these enzymes are not inducible a bit, they can be inhibited by drugs.[1]

Aspirin

Aspirin (acetylsalicylic acid) is a prodrug which is metabolically converted to salicyclic acid by the action of carboxylesterases. Aspirin is absorbed rapidly from the stomach and intestine by passive diffusion. Aspirin is transformed into active metabolite, salicylate in the stomach, in the intestinal mucosa, in the blood and mainly in the liver. Salicylate is the active metabolite responsible for most anti-inflammatory and analgesic effects. Therefore, aspirin has a very short half-life. Salicylate, in turn, is mainly metabolized by the liver. This metabolism occurs primarily by hepatic conjugation with glycin or glucuronic acid, each involving different metabolic pathways. The predominant pathway is the conjugation with glycin, which is saturable. With low doses of aspirin approximately 90% of salicylate is metabolized through this pathway. As the maximum capacity of this major pathway is reached, the other pathways with a lower clearance become more important. Therefore, the half-life of salicylate depends on the major metabolic pathway used at a given concentration and becomes longer with increasing dosage[2]. Salicylate is said to follow nonlinear kinetics at the upper limit of the dosing range.Urinary excretion of unchanged salicylate accounts for 10% of the total elimination of salicylate. Excretion of salicylate results of glomerular filtration, active proximal tubular secretion through the organic acid transporters and passive tubular reabsorption. Salicylate metabolites are also excreted in the urine.

Acetaminophen

Acetaminophen is a non-narcotic, analgesic and antipyretic drug, widely used as a pain relief medicine. APAP is rapidly absorbed from the gastrointestinal (GI) tract with peak concentrations achieved within 90 minutes of a therapeutic dose. The presence of food in the stomach may delay the peak but not the extent of absorption. Distribution is rapid with a volume of distribution (VD) of about 0.9 L/kg and minimal protein binding at thera- peutic concentrations. The half-life of APAP is 2.0 to 2.5 hours. Acetaminophen is metabolized in the liver by enzyme cytochrome P 450 to a highly reactive metabolite – N-acetyl-p-benzoquinoneimine (NAPQI), which can cause acute hepatic necrosis if not followed by conjugation with glutathione (GSH). The other known metabolic pathways of acetaminophen are via glucuronidation and sulfation. Metabolites separated include 2-methoxy-

acetaminophen, its glucuronide and sulfate conjugates, conjugated AAP, the sulfate conjugate of 2-hydroxyacetaminophen, glucuronide and sulfate conjugates of acetaminophen, S-(5-acetamido-2-hydroxyphenyl) cysteine, and S-(5-acetamido-2-glucuronosidophenyl)cysteine. Approximately 85% of a therapeutic dose undergoes phase II conjugation to sulfated and glucuronidated metabolites that are renally eliminated proportions of APAP are eliminated unchanged in the urine and by ring oxida tion to a catechol derivative.[3].

Indomethacin

Indomethacin is an anti-inflammatory antipyretic drug commonly used for symptomatic relief of pain and stiffness in rheumatic diseases.The drug has a biological half-life of about 5 to 10 hours and a plasma clearance of 1 to 2.5ml/kg/min. Indomethacin is distributed into the synovial fluid, is excreted in human breast milk and crosses the placenta in significant amounts. It is metabolised to O-desmethylindomethacin, N-deschlorobenzoylindomethacin and O-desmethy-N-deschlorobenzoylindomethacin, which are devoid of anti-inflammatory activity and are present in significant amount in the plasma. About 60% of an oral dose is excreted in the urine predominantly in glucuronidated form, while about 40% is excreted in the faeces after biliary secretion.[4]. A large amount of the dose undergoes biliary recycling. The biotransformation is independent of the route of administration. The drug has a biological half-life of about 5 to 10 hours and a plasma clearance of 1 to 2.5ml/kg/min.

Ketorolac

Ketorolac or ketorolac tromethamine is a non-steroidal anti-inflammatory drug (NSAID) in the family of heterocyclicacetic acidderivatives, used as an analgesic. Ketorolac acts by inhibiting the bodily synthesis of prostaglandins. Ketorolac tromethamine is largely metabolized in the liver. The metabolic products are hydroxylated and conjugated forms of the parent drug. The products of metabolism, and some unchanged drug, are excreted in the urine.[5].The principal route of elimination of ketorolac and its metabolites is renal. About 92% of a given dose is found in the urine, approximately 40% as metabolites and 60% as unchanged ketorolac. Approximately 6% of a dose is excreted in the faeces.

Mefenamic Acid

Mefenamic acid is a member of fenamate group of non-steroidal anti-inflammatory drug.

Mefenamic acid and its metabolites are firmly bound to plasma proteins. Mefenamic acid metabolism is predominantly mediated via cytochrome P450 CYP 2C9 in the liver. Two distinct metabolic products, one a hydroxymethyl derivative and the other a carboxy derivative, have been identified in both plasma and urine. Mefenamic acid and its two metabolic derivatives become conjugated with glucuronic acid through an ester linkage which is alkali labile and are excreted principally in the urine, but also to some extent in the bile and faeces.Following a single dose, 67% of the total dose is excreted in the urine as unchanged drug or as one of the two metabolites. 20% to 25% of the dose is excreted in the faeces during the first three days.[6].

Diclofenac

Diclofenac, a 2-arylacetic acid, nonsteroidal anti-inflammatory drug.The metabolism of diclofenac to its 5-hydroxylated derivative in humans is catalyzed by cytochrome P450.The biotransformation of diclofenac partly involves glucuronidation of the intact molecule, but mainly single and multiple hydroxylation followed by glucuronidation.About 60% of the administered dose is excreted in the urine in the form of metabolites from one of these two processes; less than 1% is excreted as unchanged substance. The remainder of the dose is eliminated as metabolites through the bile in the faeces.Diclofenac is completely absorbed from the enteric-coated tablets after their passage through the stomach. Following ingestion of one tablet with or after a meal, its passage through the stomach is slower than when it is taken before a meal, but the amount of active substance absorbed remains the same. In fasting subjects, the mean peak plasma concentration is attained on average 2 hours after ingestion of one 50 mg tablet. The plasma concentrations, as measured by the area under the time-concentration curve, are in linear relation to the size of the dose.Topical diclofenac is as effective as oral diclofenac in relieving joint stiffness and pain.[7]

Ibuprofen

Ibuprofen was developed by Boots (UK) in the 1950–1960's and after establishing its favourable safety profile at dose ranges for analgesic and anti-pyretic efficacy (up to 1200mg daily) it was the first NSAID (other than aspirin) to be approved for non-prescription (over-the-counter or OTC sale) use in the UK (in 1963), then the USA (in1964) and later in many other countries worldwide (Rainsford, 1999). Just after ibuprofen was developed, a large number of pharmaceutical companies undertook the discovery and development of NSAIDs

with a range of chemical and biological properties (Evans & Williamson, 1987; Otterness, 1995; Rainsford, 1999, 2004a; 2005a). Their anti-inflammatory, analgesic and anti- pyretic properties were discovered using animal models with some supportive properties being established in some biochemical systems which were known also to be important in inflammation (e.g. mitochondrial oxidative, intermediary and connective tissue collagen and proteoglycan metabolism; stability of albumin; and later oxyradicals).

Ibuprofen (IBU) is anon-steroidal anti-inflammatory drug (NSAID) widely used in the treatment of mild to moderate pain and inflammation.Ibuprofen is suitable for self medication with regards to its relatively wide spectrum of indication, good tolerance and safety.[8] This may be as a short term over-the-counter treatment for headaches, muscle aches or fever reduction or long-term, often prescription use, for arthritis and other chronic conditions. IBU inhibits the cyclooxygenase enzymes COX1 and COX2 coded for by PTGS1 and PTGS2, preventing the formation of various prostaglandins. The primary metabolism of IBU is oxidative and involves the cytochrome P450 enzymes. Ibuprofen is extensively bound to plasma proteins (99%) and extensively metabolised in the liver to two major inactive metabolites that are rapidly and completely excreted by the kidneys.[9].Like aspirin and indometacin, ibuprofen is a nonselective COX inhibitor, in that it inhibits two isoforms of cyclooxygenase, COX-1 and COX-2.However, the role of the individual COX isoforms in the analgesic, anti-inflammatory, and gastric damage effects of NSAIDs is uncertain and different compounds cause different degrees of analgesia and gastric damage.

Naproxen

Naproxen is a nonsteroidal anti-inflammatory drug. Administration of naproxen as the sodium salt (Anaprox), however, permits more rapid absorption from the gastrointestinal tract. In either form, the drug is essentially completely absorbed. Its metabolic half-life averages 13 hours. Naproxen is rapidly and completely absorbed from the gastrointestinal tract with an in vivo bioavailability of 95%. The rapidity, but not the extent, of absorption is influenced by the presence of food in the stomach. After administration of naproxen and naproxen sodium tablets, peak plasma levels are attained in 2 to 4 hours and 1 to 2 hours, respectively.[10] The metabolism of naproxen is quite simple: it is excreted almost entirely in the urine as the native molecule, its oxidative 6-desmethyl metabolite and their respective conjugates. Naproxen is an acidic drug that is highly bound to

plasma albumin. It may thus be expected to displace and transiently increase the tissue availability of other protein-bound drugs. In practice, however, potential interactions with both warfarin and tolbutamide have been evaluated and do not appear to be of clinical significance. Naproxen has a high therapeutic index and a shallow dose-response curve, so the effect of other drugs on its pharmacokinetics is not likely to have a large clinical impact.

Ketoprofen

Ketoprofen is a white or off-white, odorless, non-hygroscopic, fine to granular powder, melting at about 95°C. It is freely soluble in ethanol, chloroform, acetone, ether and soluble in benzene and strong alkali, but practically insoluble in water at 20°C. Ketoprofen is rapidly and well-absorbed, with peak plasma levels occurring within 0.5 to 2 hours.When Ketoprofen is administered with food, its total bioavailability (AUC) is not altered; however, the rate of absorption is slowed.The metabolic fate of Ketoprofen is glucuronide conjugation to form an unstable acyl-glucuronide. The glucuronic acid moiety can be converted back to the parent compound. Thus, the metabolite serves as a potential reservoir for parent drug, and this may be important in persons with renal insufficiency, whereby the conjugate may accumulate in the serum and undergo deconjugation back to the parent drug (see Special Populations, Renally Impaired). The conjugates are reported to appear only in trace amounts in plasma in healthy adults, but are higher in elderly subjects-presumably because of reduced renal clearance [11]. There are no known active metabolites of Ketoprofen. Ketoprofen has been shown not to induce drug-metabolizing enzymes.

Piroxicam

Piroxicam, an oxicam, is a non-steroidal anti-inflammatory drug (NSAID) which is structurally related to tenoxicam, another NSAID.The plasma half-life in humans is approximately 36 to 45 hours and stable plasma concentrations are maintained throughout the day on once daily dosage. Following repeated administration, plasma concentrations increase for five to seven days, by which time a steady state is reached which is not exceeded following further constant daily drug administration. Piroxicam is extensively metabolised, with less than 5% of the daily dose excreted unchanged in urine and faeces. One important metabolic pathway is hydroxylation of the pyridyl ring of the piroxicam side chain, followed by conjugation with glucuronic acid and urinary elimination. Approximately 5% of the dose is metabolised to and excreted as saccharin.[12]

Meloxicam

Meloxicam is a non-steroidal anti-inflammatory drug (NSAID). Meloxicam is eliminated almost entirely by hepatic metabolism: two thirds by cytochrome (CYP) P450 enzymes (CYP2C9 two thirds and CYP3A4 one third) and one third by other pathways, such as peroxidase oxidation. Meloxicam is almost completely metabolised to four pharmacologically inactive metabolites. The major metabolite, 5'-carboxymeloxicam (60% of dose), from CYP2C9 mediated metabolism, is formed by oxidation of an intermediate metabolite 5'-hydroxymethylmeloxicam, which is also excreted to a lesser extent (9% of dose). In vitro studies suggest that CYP2C9 plays an important role in this metabolic pathway, with a minor contribution from the CYP3A4 isoenzyme[13]. The patient's peroxidase activity is probably responsible for the other two metabolites which account for 16 and 4% of the administered dose respectively.

Celecoxib

Celecoxib is a nonsteroidal anti-inflammatory drug (NSAID) with anti-inflammatory, analgestic, antipyretic properties. It is approved for the treatment of osteoarthritis, rheumatoid arthritis, ankylosing spondylitis, and acute pain.After oral administration, celecoxib is rapidly absorbed and achieves peak serum concentration in about 3 hours. It is extensively metabolized in the liver with very little drug (<3%) being eliminated unchanged.The major routes of excretion for celecoxib are feces and urine. Celecoxib is metabolized primarily through methyl hydroxylation to form hydroxycelecoxib. This reaction is largely catalyzed by CYP2C9, although CYP3A4 also plays a minor (<25%) role. Hydroxycelecoxib is further oxidized to form carboxycelecoxib via cytosolic alcohol dehydrogenases ADH1 and ADH2, then conjugated with glucuronic acid via UDP glucuronosyltransferases (UGTs) to form the 1-O-glucuronide. None of the metabolites are pharmacologically active.Since celecoxib metabolism is predominantly mediated via CYP2C9, polymorphisms in CYP2C9 are likely to have a direct impact on celecoxib pharmacokinetics and variability in drug responses.[14]

Combination of Paracetamol and Ibuprofen

There are an increasing number of products being marketed to the public that contain both paracetamol and ibuprofen. It is uncertain whether the concomitant use of paracetamol and ibuprofen significantly improves analgesia compared to the use of NSAIDs alone. Studies

have produced mixed results and outcomes may be influenced by the cause of the pain being studied. It is also not clear whether the combined use of paracetamol and ibuprofen increases the risk of adverse effects.(15)

A Cochrane review of the analgesic efficacy of paracetamol and ibuprofen in the treatment of post-operative pain, concluded that combinations of paracetamol plus ibuprofen provided better analgesia than either medicine alone.(16) It was also concluded that the combination treatment reduced the need for additional analgesia to be administered and reduced the risk of adverse events occurring.A study of approximately 900 patients using paracetamol or ibuprofen, or a combination of the two, for the treatment of osteoarthritis of the knee found significantly more patients achieved pain control at ten days and at 13 weeks with the combination treatment compared to paracetamol alone, but there was not a statistically significant difference compared to using ibuprofen alone.(17) In contrast, a small study of 90 patients randomised to one of three treatment groups in an emergency department setting found that combination treatment with paracetamol and ibuprofen did not provide more effective pain relief following musculoskeletal injury compared to either medicine alone.

A large British study funded by a pharmaceutical company reported that compared to the use of the paracetamol and ibuprofen alone, the combined use of the two medicines did not increase the number of adverse effects. However, in the treatment of osteoarthritis of the knee a trend towards increased dyspepsia, diarrhoea and blood loss was reported in patients using a combination product.(18)

The lack of a demonstrated strong synergistic analgesic effect between paracetamol and ibuprofen, suggests that the two medicines may have similar modes of actions and their effect may not be additive. The lack of clear evidence of improved analgesia has led some experts to question the value of combination products containing paracetamol and ibuprofen.

The combination of paracetamol with NSAIDs may provide more effective analgesia for some patients, e.g. for post- surgical pain, than either medicine alone. This combination treatment may allow the dose of NSAID required to achieve analgesia to be reduced (compared to NSAID treatment alone) therefore reducing the amount NSAID-related risk the patientis exposed to. However, this approach does not appear to be effective for all conditions. If a combination of paracetamol and NSAIDs is used to treat pain, consider titrating the NSAID dose downwards as pain becomes more manageable, while continuing treatment with paracetamol at the same dose. The NSAID can then be withdrawn, before

paracetamol, and treatment with paracetamol continued, as required. If the risk of NSAID-related adverse events is high, it may be appropriate to consider adding codeine to paracetamol, in preference to NSAID treatment. For example, an older patient with osteoarthritis, diabetes and chronic kidney disease (CKD) may be particularly susceptible to the nephrotoxic effects of NSAIDs.(19).

An appropriate starting dose of codeine in combination with paracetamol for mild to moderate pain in adults is 15 mg, every four hours, as required. Codeine can be given in doses up to 60 mg, if required, but the total dose should not exceed 240 mg per day. The main adverse effects of codeine are gastrointestinal disturbance and potential respiratory depression The effective of codeine may vary between individuals due to genetic differences in metabolism, and it may not be an appropriate choice for all patients.(20)

Conclusion

The non-steroidal antiinflammatory drugs (NSAIDs) are widely used for the treatment of pain and for the management of edema and tissue damage resulting from inflammatory joint disease (arthritis).Drug metabolism has an important role in the determination of the pharmacokinetic parameters like oral bioavailability, clearance and the half-life of the entity within the cell. The drug metabolic studies help to screen the compounds based on their metabolic rate. Drug metabolism is very essential in the toxicity studies too. The persistence of the compounds in the systemic circulation for long period causes toxicity and the nature of the metabolites and the reaction of the metabolites within the body, must be studied thoroughly before the compounds progress to the next stage of screening in drug discovery process. In this way, it is seen that drug metabolic studies form an integral part in drug discovery. Since NSAIDs are commonly used drug, it is important to know the metabolism of various NSAIDs and excretion for its proper use in particular disease condition .

Corresponding Author

E. Roja,

Tutor,Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai – 600 077, Tamil Nadu, India

Email Id: itsrojaa@gmail.com

Contact Number: 91 99941 74480.

References

1. M Coleman, Human Drug Metabolism: An Introduction, John Wiley & Sons, UK, 2010;1:56-67.
2. Shibasaki,J.,Konishi,R.,Takeda,V.,andKoizumi,T.,Drugabsorption,metabolism,andexcretion.VII.Pharmacokineticsonformation and excretion of theconjugatesofN-acetyl-p-amino-phenolinrabbits.Chem.Pharm.Bull.1971;19:1800.
3. J.E.Mrochek,S.Katz,W.H.ChristieandS.R.DinsmoreAcetaminophen Metabolism in man,as Determined by high-Resolution liquid Chromatography .CLIN.CHEM.1974; 20(8):1086-1096.
4. Zhou Y, Hancock JF, Lichtenberger LM The Nonsteroidal Anti-Inflammatory Drug Indomethacin Induces Heterogeneity in Lipid Membranes: Potential Implication for Its Diverse Biological Action. PLoS ONE 2010: 5(1): 88-111.
5. Carlo P. Campobasso, PhD, MD, Rossella Procacci, MD,and Marina Caligara, PhD Fatal Adverse Reaction to Ketorolac Tromethamine in Asthmatic Patient Am J Forensic Med Pathol 2008; 29: 358–363.
6. Shikandar D. Bukkitgara, Nagaraj P. Shettia, Deepti S. Nayaka, Gangadhar B. Bagehallia and Sharanappa T. Nandibewoor.Electrochemical sensor for the detection of mefenamic acid in pharmaceutical sample and human urine at glassy carbon electrode.Scholars Research Library Der Pharma Chemica, 2014, 6(2):258-268.
7. Niklas Schuelert Fiona A Russell Jason J McDougall Department of Physiology and Pharmacology, University of Calgary, Calgary, AB, Canada.Topical diclofenac in the treatment of osteoarthritis of the knee.orthopedic research and reviews2011:3 1–8.
8. Rabia Bushra1 and Nousheen Aslam. An Overview of Clinical Pharmacology of Ibuprofen.Oman Medical Journal, 2010; 25(3) .155-161.
9. Trevor Tanner, Sue Aspley, Andrew Munnand Tracy Thomas. The pharmacokinetic profile of a novel fixed-dose combination tablet of ibuprofen and paracetamol.Tanner BMC Clinical Pharmacology,2010;10:10.

10. Setiawati E, Deniati SH, Yunaidi DA, Handayani LR, Harinanto G, Sntoso ID, Purnomo Sari A, Rimainar Bioequivalence Study with Two Naproxen Sodium Tablet Formulations in Healthy Subjects. J BioequivAvailab, 2009;1:028-033.
11. European Review for Medical and Pharmacological Sciences Ketoprofen 2.5% gel: a clinical overview.2011;15:943-949.
12. N. Ravi Kiran, S. Palanichamy, M.Rajesh, T.Godwin Rajadhas, V.Anusha, N.Parasakthi and A. Thanga Thirupathi. Formulation and Evaluation of OrodispersiblePiroxicamTablets.RaviKiran /J. Pharm. Sci. & Res. 2010; 2 (10):615-621.
13. Jashanjit Singh and Rajmeet Singh,Optimization and Formulation of Orodispersible Tablets of Meloxicam.Tropical Journal of Pharmaceutical Research, 2009; 8 (2): 153-159.
14. Stephen E. Kimmel, MD, MSCE; Jesse A. Berlin, ScD; Muredach Reilly, MB; Jane Jaskowiak, BSN, RN; Lori Kishel, MS; Jesse Chittams, MS; and Brian L. Strom, MD, MPH.Patients Exposed to Rofecoxib and Celecoxib Have Different Odds of Nonfatal Myocardial Infarction.Ann Intern Med. 2005;142.(3):157-64.
15. Visha.M.G, Selective Cox-2 Inhibitor, International Journal of Pharmaceutical Science Invention,2013;3(2)28-33.
16. Anitha Roy, Novel approaches in Insulin Drug Delivery – A Review, Int. J. Drug Dev. & Res.2013, 5 (4): 25-29.
17. SamrithiYuvaraj, A. JothiPriya, NirishaSriram,Effect of Non Steroidal Anti Inflammatory Drugs in Pregnancy: A Systematic Review.RJPT:2015;8(6):0974-1618.
18. Savitha Palanisamy and Jaiganesh Ramamurthy.NSAIDS as Host Modulating Agent-A Review.International Journal of Current Research and Academic Review.Int.J.Curr.Res.Aca.Rev.2017; 5(5): 97-100.
19. TrophimusGnanabagyanJayakaran, The Effect of Drugs in the Oral Cavity - A Review,J. Pharm. Sci. & Res.2014;6(2):89-96.
20. Doherty M, Hawkey C, Goulder M, A randomised controlled trial of ibuprofen, paracetamol or a combination tablet of ibuprofen/ paracetamol in community-derived people with knee pain. Ann Rheum Dis. 2011;70(9):1534–1541.

Dysmenorrhea in Women

N.S.Harini[1], Gowri Sethu[2], Sankari M[3]

Graduate Student[1], Professor[2], Department of Physiology[2], Professor[3],Department of Periodontics.

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Dysmenorrhea is a common menstrual complaint with a major impact on women's quality of life, work productivity, and health-care utilization. The prevalence of dysmenorrhea varies between 16% and 91% in women of reproductive age, with severe pain in 2%–29% of the women studied. Women's age, parity, and use of oral contraceptives were inversely associated with dysmenorrhea, and high stress increased the risk of dysmenorrhea. Inconclusive evidence was found for modifiable factors such as cigarette smoking, diet, obesity, depression, and abuse. Dysmenorrhea is a significant symptom for a large proportion of women of reproductive age; however, severe pain limiting daily activities is less common.The main aim of this review article is to know in detail about Dysmenorrhea in Women.The objective of this article is to know about dysmenorrhea in women, what is the cause, what are the consequences and why is it happening and also to know about dysmenorrhea in all aspects and to know the opinions from different authors.

The present study was to know about Dysmenorrhea in women. Various articles from different authors were collected and summed up as a review article so that Dysmenorrhea in women is known elaborately in all aspects. In conclusion, this review shows that dysmenorrhea is a significant symptom for a large proportion of women throughout the reproductive years. Severe dysmenorrhea limiting daily activities is much less common. Improvement of the symptom over time has been observed, although many women also experience unchanged or worse symptoms. Dysmenorrhea is inversely related to age, parity or number of livebirths, and oral contraception use. Dysmenorrhea is positively associated with stress related to both work and general life, as well as with family history of dysmenorrhea.

Key Words: *Dysmenorrhea, women, menstruation, painful periods, abdomen.*

Introduction

Dysmenorrhea or painful menstruation is defined as a severe, painful, cramping sensation in the lower abdomen that is often accompanied by other symptoms, such as sweating, headaches, nausea, vomiting, diarrhea, and tremulousness, all occurring just before or during the menses [(1).] There are 2 types of dysmenorrhea: Primary dysmenorrhea refers to pain with no obvious pathological pelvic disease and almost always first occurs in women 20 years or younger after their ovulatory cycles become established [(1).] Secondary dysmenorrhea is caused by underlying pelvic conditions or pathology and is more common in women older than 20 years [(1, 2).]

Dysmenorrhea is considered the most common symptom of all menstrual complaints and poses a greater burden of disease than any other gynecological complaint in developing countries [(3).] Among women of reproductive age worldwide, dysmenorrhea is more prevalent than the other 2 common types of chronic pelvic pain, namely, dyspareunia and noncyclical chronic pelvic pain [(4).] Being a debilitating condition for many women, it has a major impact on health-related quality of life, work productivity, and health-care utilization [(5–9).] As a result, dysmenorrhea is responsible for considerable economic losses due to the costs of medications, medical care, and decreased productivity [(1).] The prevalence of dysmenorrhea reported in the literature varies substantially. A greater prevalence was generally observed in young women, with estimates ranging from 67% to 90% for those aged 17–24 years [(10, 11).]

A recent large Australian study of senior high school girls found that a higher proportion, 93%, of teenagers reported menstrual pain [(12).] The studies in adult women are less consistent in reporting prevalence of dysmenorrhea and often focus on a specific group, with rates varying from 15% to 75% [(10).] Severe pain sufficient to limit daily activities is considerably less common, affecting approximately 7%–15% of women [(10),] although a study of adolescents and young adults aged 26 years or less reported that 41% of the participants had limitations in their daily activities due to dysmenorrhea [(7).]

A range of risk factors for dysmenorrhea have been identified in the literature, although mixed results have been observed for many of these factors. In general, increased severity of dysmenorrhea has been suggested to relate to age [13–15], smoking [14, 16, 17], higher body mass index [18], earlier age at menarche [15, 18], nulliparity [15, 19], longer and heavier menstrual flow [15, 18], and family history of dysmenorrhea [20]. Women using oral contraceptives generally report less severe dysmenorrhea [14, 15, 21]. Depression and stress have also been shown to increase the risk of dysmenorrhea [9, 22]. Other common factors, such as education [23], marital status [14], employment [19], alcohol consumption [17, 18], and physical activity [15, 18], show largely negative or inconclusive results.

Materials and Methods

The present study was to know about Dysmenorrhea in detail. Various articles from different authors were collected and summed up as a review article so that dental crowding is known elaborately in all aspects. The articles were collected by manual searching of the references of the relevant retrieved articles, peer-reviewed physiologic journals, and gray literature. Search terms included dysmenorrhea, menstruation, women,painful periods. Non-English articles were excluded from the review in the study-selection stage. Data extraction and evaluation of primary studies were performed independently by 2 reviewers.

Discussion

Knowledge of physiology of reproduction is important for teenage girls to practice menstrual hygiene [47]. Dysmenorrhoea is a common distressing disorder in women that manifests during menstrual phase especially in young females [51]. Primary dysmenorrhea is a medical condition of pain which is worst during first day of menstruation that interferes with daily activities. It is not associated with any pathological changes.[48]. Dysmenorrhea is defined as painful menstrual cramps of uterine origin. It is the common gynecological condition that can affect as many as 50% of women[49].

Onset of menstruation, menarche is a part of many adolescence changes, is a very important emotional land mark for every girl. It may be associated with some menstrual problems like dysmenorrhea. This condition is commonly observed between the ages of 15 and 25

years.Dysmenorrhea is classified in to two types, Primary, which usually begins shortly after the onset of menstruation and secondary dysmenorrhea, which develops later in life and mostly is associated with pelvic abnormalities.

Its prevalence varies from 40% to 90% according to different studies. The exact etiology of dysmenorrhea is unknown but it is thought to be caused by release of prostaglandins, which causes uterine contractions and pain. In some studies it is mentioned that it results from hypoxia and ischemia even though vasopressin may also play a role in increasing uterine contractility, causing ischemic pain and elevated levels of vasopressin evaluated in women with primary dysmenorrhea. Where most of the females experience some degree of pain and discomfort in their menstruation but approximately 10-15% of the women have severe dysmenorrhea which significantly affects the quality of life and could have impacts on daily activities, social and academic performances at their workplace or at home.

Dysmenorrhea is found to be the leading cause of short term school absenteeism from 38 to 45.6% mentioned in different studies, 58.9% reported decreased daily activity & socialization 46%. Risk factors for dysmenorrhea include nuliparity, heavy menstrual flow, smoking and depression. There are certain risk factors which are seemed to be associated with more severe dysmenorrhea like earlier age at menarche, long menstrual periods, heavy menstrual flow, smoking and positive family history. Somehow women differ considerably in their individual risks of dysmenorrhea for reasons that are largely unknown.

Still limited data is available in this regard which can avert the relationship of positive family history in occurrence of dysmenorrhea and severity of condition. Therefore present study was carried out to evaluate familial predisposition of primary dysmenorrhea among the medical students with positive family history.

It has been estimated that 7–15% of women suffer from severe pain which causes disturbances in their performance for 1–3 days in a month, and 14–52% of female students miss school because of complaining dysmenorrhea. This issue is associated with decline in career and educational function and results in disturbed quality of life and emerging economic problems.

Various reasons have been proposed for primary dysmenorrhea such as progesterone reduction in the final stages of luteal phase that causes lissom rupture and subsequent release of A2 phospholipase of the endometrium. Origin of increasing phospholipase is prostaglandin that causes retraction of uterine muscles vessels which finally result in ischemia and pain. However, mild physical activities, proper diet, relaxation, massage, biofeedback techniques, some Yoga exercises, contraceptive pills, and nonsteroidal anti-inflammatory drugssuch as mefenamic acid and ibuprofen are recommended for decreasing the pain of primary dysmenorrhea.

Since most of the drugs utilized for controlling the pain of dysmenorrhea have various side-effects and also are expensive, most of the nonpharmacological methods are in the category of complementary medicine for this disorder. The use of nonpharmacological methods such as aromatherapy, massage, acupuncture, and acupressure are also having been popular in the recent years. Traditionally, aromatherapy has been used as one of the nonpharmacological methods for reducing the symptoms of dysmenorrhea, releasing uterine cramps, and decreasing the pain and anxiety after childbirth[22-25].

However, a common complementary drug which has been used in aromatherapy is lavender that belongs to the Mediterranean region and it is found in Africa and India. Lavender is just a little herbaceous plant with narrow and long leaves, covered by white cottony fluff and purple flowers and is in the form of a spike. Its essence is made from distilling flowers from blossoming plants, and its muscle relaxant and antispasmodic effects have been confirmed by many studies.

Some studies suggest lavender and its main extract such as linalool acetate and linalool had topical analgesic effects on laboratory animals, which is made by increasing regional perfusion. Results of studies supporting aromatherapy suggest that essential oils stimulate the receptors in the olfactory bulb and transfer the message of olfaction to limbic system and cause releasing endorphin, encephalin, and serotonin, which results in the sense of relaxation and stress reduction[26,28]

About the prevalence of the primary dysmenorrhea and the side effects of the current treatments and because of lack of similar studies in this field, we decided to perform a study on the female

students in dormitories of Ardabil University of Medical Sciences with the aim of determining the effect of lavender aromatherapy on the pain severity in primary dysmenorrhea.

Dysmenorrhea is one of the most common gynecological problems among female students, which may be the leading cause of absenteeism from college. Dysmenorrhea is of two types. The primary is a menstrual pain without any organic pathology. When the pelvic pain is associated with identifiable pathological conditions, it is called secondary. Dysmenorrhea also may be due to anxiety, emotional instability, a faulty outlook on sex and menstruation, or imitation of the mother's feelings about menstruation. Researchers reported that excessive production of prostaglandins, which causes severe uterine contractions, which cause pain and decrease blood flow and oxygen to the uterus. Similar to labour pains, these contractions can cause significant pain and discomfort. Prostaglandins may also contribute to nausea and diarrhoea[49].

Most studies show that dysmenorrhea is a common problem affecting the majority of women in the community. Severe pain or pain limiting women's daily activities, however, occurred only in 2%–28% of adult women. Self-medication is a worldwide problem and it is common in developing countries like India every day, everywhere, consumers reach for self-care products to help them through their common health problems.[50]

The lowest rate of 2% reported in a longitudinal study was possibly due to potential underreporting as 74% of the included women were employed and thus less likely to stay in bed and miss work, which is used to define severe pain in the study [37]. The prevalence of severe pain reported in this review appears to be higher than the 12%–14% reported in community-based studies in the World Health Organization review [4] but comparable with 5%–20% reported in another review of the condition in developing countries [38].

Weissman et al. [37] found that dysmenorrhea persisted over the 6-year follow-up among the majority of women reporting it at baseline, and improvement or worsening of the symptom was equally likely. In their multivariable analysis, the presence of dysmenorrhea at baseline (excluding women with severe dysmenorrhea) was a strong predictor of reporting moderate or severe dysmenorrhea at follow-up (odds ratio = 7.48, 95% CI: 3.09, 18.15). More studies are

needed to explore the natural history of the symptom. Despite some disagreement, the majority of the previous literature generally demonstrates an inverse association between both age and parity and dysmenorrhea

This association was confirmed by the vast majority of studies included in this review, consistent across different types of study, although 3 studies failed to adjust for parity in their analysis on the association between age and dysmenorrhea [30]. Interestingly, Burnett et al. [26] found that the effect of age remained in the adjusted model including nulliparity, whereas the association between nulliparity and primary dysmenorrhea was no longer significant when controlled for age and smoking ($\beta = 0.93$; $P = 0.582$). It is unclear though what the proportion of nulliparous women was in the study.

Furthermore, the study did find that the women most debilitated by pain were significantly more likely to be nulliparous. Nevertheless, the longitudinal study by Weissman et al. [37] provides stronger support for the inverse association between both age (odds ratio = 0.92, 95% CI: 0.86, 0.98) and livebirth (odds ratio = 0.20, 95% CI: 0.08, 0.53) and the severity of dysmenorrhea after controlling for each other; however, parity clearly had a much stronger effect in their analysis. In addition, the study also found that gravidity was less influential than livebirth, consistent with other studies showing no effect from pregnancies ending in miscarriage or abortion [15, 41].

Different mechanisms have been proposed for the relation between livebirth and dysmenorrhea. One is related to the pathogenesis of primary dysmenorrhea of the close association with elevated prostaglandin levels in the secretory endometrium that triggers pain. After a term delivery, the endometrium may release a lower level of prostaglandins, resulting in decreased pain [41]. Another hypothesis is that neuronal degeneration in the uterus following term pregnancy, due to disappearance of uterine adrenergic nerves and a decrease in uterine noradrenaline in the third trimester of pregnancy, may explain the disappearance or reduction of menstrual pain after childbirth [15].

A strong effect of family history of dysmenorrhea and risk of dysmenorrhea was shown in 2 studies, which is in line with some previous studies reporting a similar association, suggesting genetic susceptibility to dysmenorrhea among women with variant genotypes in a number of

metabolic gene polymorphisms [20, 43]. However, other possible explanations are that the association could be related to conditioned behavior that is learned from mother or sisters for the possibility of societal reward or that control for pain exists [25-28]. Alternatively, it could be simply due to similar living patterns and lifestyles in the families [34]. Among the range of lifestyle and other demographic factors studied such as smoking, body mass index, and socioeconomic status, conflicting results were shown.

Previous studies on the association between smoking and dysmenorrhea are mixed. Although most cross-sectional studies show an increased risk among smokers [13, 14, 16], a negative effect was also seen [44]. Inconsistent results have also been observed for smoking and the incidence or the severity of dysmenorrhea in longitudinal studies [15, 18].Sundell et al. [15] found that the prevalence and severity of dysmenorrhea were increased in smokers and that the severity increased with the number of cigarettes smoked per day.

On the other hand, Harlow and Park [18] found that smoking was not associated with the probability of having pain or severe cramps but, among those with pain, smokers were more likely to have pain lasting longer than 2 days. Similarly, being overweight was found to be an important risk factor for the probability of experiencing pain and for increasing duration of pain in 1 longitudinal study [18], and severity of dysmenorrhea was not associated with either height or weight in another [15].

The systematic review also failed to detect a significant association between obesity and dysmenorrhea in the pooled analysis of 5 studies [40]. No association was shown among women's education, marital status, alcohol use, and the risk of dysmenorrhea in the current review, which is supported by the recent systematic review identified [40] and previous studies.

Dysmenorrhea or painful menses is unfortunately a very common condition and one that some women often come to accept as a normal occurrence in their lives. However anything beyond very mild discomfort at the onset of the cycle is a sign some aspect of the cycle needs to be addressed and Chinese Medicine has many tools to assist in this area.

There is an old saying in Chinese which translates as "Where there is free flow there is no pain and where there is pain there is no free flow". Basically stating that wherever we have pain there

is something restricting the optimal flow of blood and nutrients into an area. Common causes include; excessive tension causing sluggish flow, lack of warmth which allows the buildup of cold in the tissues and subsequent contraction or can be due to blood deficiency where it is not adequately nourishing the involved tissues and organs of the body and leading to complications.

When symptoms are worse in the days leading up the cycle and improve after the flow starts in earnest this is due to an excessive amount of tension in the system; often due to chronic mental stress and irritation or can be due to a blockage in the downward descent of Blood through the channels linked to the uterus .

Treatment is targeted towards re-establishing healthy movement of Blood and throughout the body mainly by working on the relationship between the Liver or GallBladder and the Spleen meridians.

Another common contributing factor is the accumulation of cold in the tissues of the lower abdomen. This can be due to environmental factors which lead to direct exposure such as; leaving the mid rift exposed during cooler weather, swimming in cold water during the menses, sitting on cold floors or excessive intake of iced products.

This accumulation can also arise from internal reasons. This can be due to lifestyle factors or convalescence from an illness that has led to the gradual exhaustion of the bodies. This exhaustion then allows the body temperature to drop and blood to stagnate and the tissues to contract manifesting with pain and inhibited menstrual flow.

When discomfort increases towards the end of menstruation this shows an underlying deficient condition in the body that has been exacerbated by the further loss of blood. Treatment then aims to build up and strengthen the body focusing on the quality and volume of the blood and the underlying organ deficiencies or lifestyle patterns that have contributed to the development of the condition. Excessive mental activity, worry or anxiety, heavy exercise or work schedule, chronic illness or dietary imbalances may all contribute to this condition.

However, caution should apply when interpreting the systematic review results, as heterogeneity was present in all analyses with multiple studies, and the review is based largely on case-control studies that are subject to recall bias. A protective effect of OCPs or other forms of hormonal contraceptive for dysmenorrhea is evident in the majority of previous studies [10, 13–15, 21], consistent across different study types.

This is largely confirmed by the current review, although 1 of the studies on a large sample of Indian women did not show any significant association between different methods of contraception, including OCPs, and moderate to severe dysmenorrhea [3].

The reason for this discrepancy may be due to the small number of women using OCPs (n = 43) in the study. There is evidence suggesting an association between early age at menarche and increased risk of dysmenorrhea [15, 18, 19], which is supported by the systematic review [40]. However, most of the studies reporting it in this review failed to show an association [26, 31, 35] with the exception of the Indian study [3].

The reason for the discrepancy is not readily apparent, and the association may be confounded or mediated by other factors. Furthermore, although there is suggestion of some associations of other reproductive factors such as age at first birth, cesarean section, and heavy and irregular menses [14, 18, 41], this review is unable to reach any firm conclusion because of the limited number of studies reporting these effects. Among the psychological factors studied, a positive association between perceived stress, related to work or general life events, and the risk of dysmenorrhea was shown in most included studies.

The biological mechanism for association between work stress and dysmenorrhea is not well understood, although potentially through a cascade of neuroendocrine responses [36]. Stress inhibits the release of follicle-stimulating hormone and luteinizing hormone, leading to impaired follicular development. This can alter progesterone synthesis and release, which may influence the activity of prostaglandin.

Besides progesterone, stress-related hormones, including adrenaline and cortisol, also appear to influence prostaglandin synthesis and/or binding in the myometrium [36].

Furthermore, mental health may act as a mediator in the relationship between stress and dysmenorrhea, with high job stress increasing the risk of mental health morbidity, which in turn is positively related to painful menses [28]. Of note is that these studies were generally conducted on groups of employed women, whereas another included crosssectional study of a random sample of Iranian women did not observe a significant association between stress level and the severity of pain in the adjusted analysis [34].

Conclusion

In conclusion, this review shows that dysmenorrhea is a significant symptom for a large proportion of women throughout the reproductive years. Severe dysmenorrhea limiting daily activities is much less common. Improvement of the symptom over time has been observed, although many women also experience unchanged or worse symptoms. From longitudinal or population data, this review has confirmed the following:

• Dysmenorrhea is inversely related to age, parity or number of livebirths, and oral contraception use;

• Dysmenorrhea is positively associated with stress related to both work and general life, as well as with family history of dysmenorrhea.

However, uncertainty still remains for a number of lifestyle factors, such as smoking, obesity and diet, psychological factors, and environmental factors. Furthermore, there is a lack of longitudinal datato study the natural history of dysmenorrhea and the effects of a range of modifiable risk factors over time. More research on these from population-based, prospective, longitudinal studies to generate robust evidence will help to support targeted preventive interventions.

Corresponding Author

M.Sankari,

Professor, Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai-600077, Tamil Nadu, India.

Phone Number: +91 9840285905

Email Id: msankari@gmail.com

References

1. Lentz G, Lobo R, Gershenson D, ComprehensiveGynecology. Philadelphia, PA: Mosby Elsevier; 2012; 53(11):837–846.
2. Impey L, Child T; Chicester, United Kingdom: Wiley-Blackwell; Obstetrics and Gynaecology; 2012; 20(3):234–241
3. Patel V, Tanksale V, Sahasrabhojanee M, The burden and determinants of dysmenorrhoea: a population-based survey of 2262 women in Goa, India.BJOG. 2006;113(4):453–463.
4. Latthe P, Latthe M, Say L, ;WHO systematic review of prevalence of chronic pelvic pain: a neglected reproductive health morbidity.BMC Public Health. 2006;6:177.
5. Ballagh SA, Heyl A Communicating with women about menstrual cycle symptoms; J Reprod Med. 2008;53(11):837–846.
6. Barnard K, Frayne SM, Skinner KM,;Health status among women with menstrual symptoms;J Womens Health (Larchmt). 2003;12(9):911–919.
7. Rodrigues AC, Gala S, Neves A, ;Dysmenorrhea in adolescents and young adults: prevalence, related factors and limitations in daily living; Acta Med Port. 2011;24(suppl 2): 383–392.
8. Sharma A, Taneja DK, Sharma P, et al;Problems related to menstruation and their effect on daily routine of students of a medical college in Delhi, India; Asia Pac J Public Health. 2008; 20(3):234–241.

9. TitilayoA, Agunbiade OM, Banjo O, et al;Menstrual discomfort and its influence on daily academic activities and psychosocial relationship among undergraduate female students in Nigeria;Tanzan J Health Res. 2009;11(4):181–188.
10. Harlow SD, Ephross SA;Epidemiology of menstruation and its relevance to women's health;Epidemiol Rev. 1995;17(2): 265–286.
11. Kennedy S;Primary dysmenorrhoea; Lancet. 1997;349(9059): 1116.
12. Parker MA, Sneddon AE, Arbon P;The menstrual disorder of teenagers (MDOT) study: determining typical menstrual patterns and menstrual disturbance in a large populationbased study of Australian teenagers; BJOG. 2010;117(2): 185–192.
13. Pullon S, Reinken J, Sparrow M;Prevalence of dysmenorrhoea in Wellington women; N Z Med J. 1988;101(839):52–54.
14. Messing K, Saurel-Cubizolles MJ, Bourgine M, ; Factors associated with dysmenorrhea among workers in French poultry slaughterhouses and canneries; J Occup Med. 1993; 35(5):493–500.
15. Sundell G, Milsom I, Andersch B;Factors influencing the prevalence and severity of dysmenorrhoea in young women;Br J Obstet Gynaecol. 1990;97(7):588–594.
16. Mishra GD, Dobson AJ, Schofield MJ;Cigarette smoking, menstrual symptoms and miscarriage among young women;Aust N Z J Public Health. 2000;24(4):413–420.
17. Parazzini F, Tozzi L, Mezzopane R,;Cigarette smoking, alcohol consumption, and risk of primary dysmenorrhea;Epidemiology. 1994;5(4):469–472.
18. Harlow SD, Park M;A longitudinal study of risk factors for the occurrence, duration and severity of menstrual cramps in a cohort of college women; Br J Obstet Gynaecol. 1996;103(11): 1134–1142.
19. 19.Ng TP, Tan NC, Wansaicheong GK;A prevalence study of dysmenorrhoea in female residents aged 15–54 years in Clementi Town, Singapore; Ann Acad Med Singapore. 1992; 21(3):323–327.
20. Parveen N, Majeed R, RajarUDM;Familial predisposition of dysmenorrhea among the medical students;Pak J Med Sci. 2009;25(5):857–860.
21. Juang CM, Yen MS, HorngHC,Natural progression of menstrual pain in nulliparous women at reproductive age: an observational study; J Chin Med Assoc. 2006;69(10):484–488.

22. GordleyLB,Lemasters G, Simpson SR,;Menstrual disorders and occupational, stress, and racial factors among military personnel;JOccup Environ Med. 2000;42(9):871–881.
23. Chen C, Cho SI, Damokosh AI, ;Prospective study of exposure to environmental tobacco smoke and dysmenorrhea;Environ Health Perspect. 2000;108(11):1019–1022.
24. Fowkes FG, Fulton PM;Critical appraisal of published research: introductory guidelines;BMJ. 1991;302(6785):1136–1140.
25. Abenhaim HA, Harlow BL;Live births, cesarean sections and the development of menstrual abnormalities;Int J Gynaecol Obstet. 2006;92(2):111–116.
26. Burnett MA, Antao V, Black A,;Prevalence of primary dysmenorrhea in Canada; J Obstet Gynaecol Can. 2005;27(8): 765–770.
27. Laszlo KD, Gyorffy Z, Adam S, Work-related stress factors and menstrual pain: a nation-wide representative survey;JPsychosom Obstet Gynaecol. 2008;29(2): 133–138.
28. Laszlo KD, Kopp MS;Effort-reward imbalance and overcommitment at work are associated with painful menstruation;results from the Hungarostudy Epidemiological Panel 2006. J Occup Environ Med. 2009;51(2):157–163.
29. Nohara M, Momoeda M, Kubota T,;Menstrual cycle and menstrual pain problems and related risk factors among Japanese female workers;Ind Health. 2011;49(2):228–234.
30. OhdeS, Tokuda Y, Takahashi O,Dysmenorrhea among Japanese women; Int J Gynaecol Obstet. 2008;100(1): 13–17.
31. PawlowskiB;Prevalence of menstrual pain in relation to the reproductive life history of women from the Mayan rural community; Ann Hum Biol. 2004;31(1):1–8.
32. Pitts MK, Ferris JA, Smith AM,Prevalence and correlates of three types of pelvic pain in a nationally representative sample of Australian women; Med J Aust. 2008;189(3): 138–143.
33. Santer M, Warner P, Wyke S;A Scottish postal survey suggested that the prevailing clinical preoccupation with heavy periods does not reflect the epidemiology of reported symptoms and problems; J Clin Epidemiol. 2005;58(11): 1206–1210.
34. TavallaeeM, Joffres MR, CorberSJ,;The prevalence of menstrual pain and associated risk factors among Iranian women;J Obstet Gynaecol Res. 2011;37(5):442–451.
35. Unsal A, Tozun M, Aslan G, Evaluation of dysmenorrhea among women and its impact on quality of life in a region of western Turkey; Pak J Med Sci. 2010;26(1):142–147.

36. Wang L, Wang X, Wang W,;Stress and dysmenorrhoea: a population based prospective study;Occup Environ Med. 2004; 61(12):1021–1026.
37. Weissman AM, Hartz AJ, Hansen MD,;The natural history of primary dysmenorrhoea: a longitudinal study; BJOG. 2004; 111(4):345–352.
38. Harlow SD, Campbell OM; Epidemiology of menstrual disorders in developing countries: a systematic review.BJOG. 2004;111(1):6–16.
39. Harlow BL, Missmer SA, Cramer DW, ;Does tubal sterilization influence the subsequent risk of menorrhagia or dysmenorrhea, Fertil Steril. 2002;77(4):754–760.
40. Latthe P, Mignini L, GrayR;Factors predisposing women to chronic pelvic pain: systematic review;BMJ. 2006; 332(7544):749–755.
41. Juang CM, Yen MS, TwuNF,;Impact of pregnancy on primary dysmenorrhea; Int J Gynaecol Obstet. 2006;92(3): 221–227.
42. Hong Ju, Mark Jones, and Gita Mishra; The Prevalence and Risk Factors of Dysmenorrhea;Epidemiol Rev 2014;36:104–113
43. Wu D, Wang X, Chen D,Metabolic gene polymorphisms and risk of dysmenorrhea;Epidemiology. 2000;11(6):648–653.
44. Andersch B, Milsom I;An epidemiologic study of young women with dysmenorrhea; Am J Obstet Gynecol. 1982; 144(6):655–660.
45. Christiani DC, Niu T, Xu X;Occupational stress and dysmenorrhea in women working in cotton textile mills;Int J Occup Environ Health. 1995;1(1):9–15.
46. Goldstein-Ferber S, Granot M; The association between somatization and perceived ability: roles in dysmenorrhea among Israeli Arab adolescents;Psychosom Med. 2006;68(1): 136–142.
47. Nidhi Sharma , M. Benjamin Sagayaraj, BalammaSujatha , Abdul Rahman A. M, Abirami M, Adhil Khan M. H, Aditya. K, Ajantha P; Prevalence of dysmenorrhoea in College Students;International Journal of Scientific and Research Publications;2014; 4(10):1-6.
48. G.V.S Nivashini; Prevalence of Primary Dysmenorrhea along With irregular Periods; Research Journal of Pharmacy and Technology;2015; 8(8):1099.

49. R Archana, Kumar Sai Sailesh , DeepthyPurushothaman , B Udaya Kumar Reddy, J K Mukkadan; Traditional Vestibular Stimulation: A Physiological Intervention for Dysmenorrhea;Journal of Medical Sciences and Health, 2015;1(3) :31-32.

50. Ms. I. Sigloria and Dr. P.Mangalagowri; Assess the prevalence of self-medication among people in a selected area; International Journal of Pharma and Bio Sciences; 2017 ; 8(1): 276 – 280.

51. R. Archana, V. Philominal, ShyamalaThirumeni; Effect of acupressure and changes in heart rate variability in dysmenorrhoea; Recent Research in Science and Technology 2011, 3(10): 01-06.

Effect of Bacopa Monnieri on Memory – A Review

Shasmitha.R [1], S.Saravanakumar [2], Madhusudhan Vasantharajan[3]

Graduate Student [1],Senior Lecturer [2], Department of Anatomy,

Senior Lecturer[3], Department of Pedodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

People believe and trust that plant based medicines are natural and are effective with no or fewer side effects. However it may or may not be true for each and every plant, unless they are clearly studied and identified its uses. Still there are numerous numbers of plants whose medicinal purposes are unknown to us, and there still under research of its uses. Bacopamonnieri is a perennial herb. Bacopamonnieri is an Indian system of medicine; it is most commonly called brahmi. Bacopamonnieri was the most important medicinal plants, for their medicinal importance, commercial value and its potential for future researches [1]. This herb has been taken to sharpen intellect and attenuate mental deficits [2]. In Ayurvedic, B. monnieri has been used to promote memory and to treat psycho neurological disorders, treating insanity, epilepsy, hoarseness and as a rejuvenator. In the last four decades the plant and the compounds isolated from it, especially the saponins, have been studied extensively regarding their memory-enhancing activities and their ability to improve cognitive function, including some clinical trials conducted to establish the activity[3][4]. There are adverse effects of Bacopamonnieri on humans, we can see about what it can do to rats.

Keywords: *Bacopamonnieri, Memory, Neurological disorders, Alzheimer's disease, Hypothermia,*

Introduction

Bacopamonnieri is an Indian folklore herbal medicine. In 1993, the Central Drug Research of India (CDRI) planned to start clinical trials using brahmi on human volunteers. They presented their research findings to numerous international brain research conferences. The research identified 2 active molecules: Bacoside A that assists in the release of nitric oxide allowing relaxation of the aorta and veins and blood to flow more smoothly through the body and aids circulation; and Bacoside B, a protein valued for nourishing nerve cells in the brain[5]. Subsequent studies showed that bacoside A and B were responsible for the cognition facilitating effect [6]. The aim of this study is to understand the features of Bacopamonnieri on memory.

Figure 1:Bacopamonnieri leaves

Chemical Components of Bacopamonnieri

Chemical constituents of Bacopamonnieri are mainly alkaloids Brahmin, herpestine, saponins, monnierins, hersaponin, bacoside–A and bacoside – B. Other constituents present in this plant are D- manitol,Betulic acid,B-sitosterol,Stigma sterol and its esters,Heptacosane,Octacosane,Nanocosane,Triacontane,Hentriacontane,Dotriacontane,Nicotin, 3-formyl – 4- hydroxyl – 2H pyran, Luteolin and its 7-glucoside, A-alanine, Aspartic Acid, Glutamic Acid and Serine[7].

Properties of Bacopamonnieri

This involves antispasmodic activity that the plant exhibited a tranquilizing effect in rat and dog. It caused smooth-muscle relaxation and showed antispasmodic activity [8, 9]. It also inhibits acetyl cholinesterase, activates choline acetyltransferase, and increases cerebral blood

flow [10]. Acetylcholinesterase, a major component of the central and peripheral nervous system, is ubiquitous among multi-cellular animals, where its main function is to terminate synaptic transmission by hydrolyzing the neurotransmitter, acetylcholine [11]. Prior studies on Bacopamonnieri on rats have shown that it improves the rate of learning in a brightness discrimination task and a conditioned avoidance task, that it improves retention, as demonstrated by savings in relearning, and that it attenuates amnesia induced by immobilization, electroconvulsive shock and scopolamine [12]. Anticancer, anti-ulcer, calcium antagonist, vasodilatory, smooth muscle relaxant, anti-addictive, and mast cell-stabilizing properties of Bacopamonnieri have also been demonstrated.There is a recent research that the anti-cholinesterase effect of Bacopamonnieri is a therapy for Alzheimer's disease in rats. Stress induced to rats is often studied to have inhibited hippocampal growth.

Hippocampus

The hippocampus, the brain's seat of memory, is located in the temporal (left and right) sides of the brain. It processes signals sent to the brain by the senses into the templates of memory, which are then stored in other parts of the brain, creating a long-term memory. Signals are converted into electrical impulses in the nerve cells due to a rapid change in protein composition. These impulses are conducted through the neurons and synapses. This process continues until the bonds between the nerve cells strengthen, and memory is created. Normal synaptic activity is a process mediated by neurotransmitters. Each neuron is a single nerve cell. It has one or more arms called axons that send signals (impulses) and one or more arms called dendrites that receive signals. When a signal is transmitted through an axon terminal,the vesicles fuse with its membrane. Neurotransmitters are released when the vesicles burst open into the synaptic space, the minute space between the sending and receiving cells used to discharge neurotransmitters. At the end of the signal, the axons reabsorb some neurotransmitters and the enzymes in the synapse neutralise the other neurotransmitters.

Improvement in the area of brain regions of a rat, affected by stress or severe disorders:

Alzheimer's disease (AD)

It is a slow progressive disease of the brain that is caused by impairment of memory and leading to disturbances in reasoning, planning, language, and perception. Alzheimer's disease (AD) is one of the most common forms of dementia affecting approximately 10% of the

population over the age of 65 years. Rats are similar to humans physiologically, genetically and morphologically. More importantly, the rat has a well-characterized behavioural display. Consequently, rat models of Alzheimer's diseaseshow accurate assessment of the pathology and novel therapeutics on cognitive outcomes. In a field of several theoretical options, the best approach has been the use of AChE inhibitors (AChEIs), which led to the introduction of tacrine as the first AChEI specifically approved for the treatment of Alzheimer's disease. Now, several kinds of AChEIs, such as donepezil,galantamine and rivastigmine are available for the symptomatic treatment of patients with mild to moderate Alzheimer's disease. However, these compounds have been reported to have the problems associated with the gastrointestinal disturbances and bioavailability. One of the most important approaches for treatment of this disease involves the enhancement of acetylcholine level in brain using AChE inhibitors [13]. Anticholinesterase activity of ethanol extracts of aerial parts of Bacopamonnieri are used in those studies. The ethanol extract of B. monnieri was found to contain saponins [14], mainly bacoside that has been used for memory and intellectual improvement. Subsequent studies showed that bacoside A and B were responsible for the cognition facilitating effect [6]. Other than facilitating learning and memory, these constituents in normal rats inhibited the amnesic effects of scopolamine, electroshock and immobilization stress [12]. Various experiments have identified potent antioxidant activity in Bacopamonnieri. Significant antidepressant activity has been observed in Bacopamonnieri extract using a rodent model of depression, comparable to imipramine after 5 days of oral administration. Additionally, anti-cholinesterase activity has been demonstrated [15][16]. Bacopamonnieri also demonstrates stress-decreasing activity in both acute and chronic stress situations. In a study the oral dose (100 mg/kg body weight) of ethanol extract of Bacopamonnieri inhibited acetyl cholinesterase (AChE) differentially in various brain regions viz. Cerebral cortex (51.6 %) > Cerebellum (51%) > Pons (44 %) > Thalamus (41.6 %) > Hippocampus (38.1 %) > Brain stem (34.3 %) > Striatum (24.9 %) [17]. Hippocampus is the major region of the brain belonging to the limbic system and plays an important role in epileptogenesis, memory and learning. The result demonstrated that the ethanol extract of Bacopamonnieri yields competitive inhibition in AChE kinetics in each Brain region of the rat. In a study, there is evidence of an antioxidant effect of Brahmi in the hippocampus[18]. The effect of the extract of Bacopamonnieri may be followed by antioxidant action within the hippocampus [19]. Central cholinergic system plays very important rolein regulation of cognitive functions. Loss of cholinergic neuronal in brain regions especially in hippocampal area is the major cause of Alzheimer's disease. The use of anti-cholinesterase in enhancement

of central cholinergic activity is presently the basis of treatment of dementia of Alzheimer's type [26].

Hypothermia on Rats

In a study, effect of BM was evaluated on acute stress (AS) and chronic unpredictable stress (CUS) induced changes in plasma corticosterone and monoamines-noradrenaline (NA), dopamine (DA) and serotonin (5-HT) in cortex and hippocampus regions of brain in rats. Treatment with BM (40 and 80 mg/kg) attenuated the stress induced changes in levels of 5-HT and DA in cortex and hippocampus regions [27]. The study involves the stress levels to cold exposure and oxygen levels on rats.When they had been pre-treated with Bacopa, these rats produced a protective protein, called a heat-shock protein, notably in hippocampus and cerebral cortex region. Bacopa helped these brain cell regions less susceptible to hypothermic and hypoxic stress by increasing the brain's protective enzymes storage. Bacopa somehow made those regions of the brain less susceptible to stress. In effect, it kept them in a relatively non-stressed state even in the presence of stress [20]. Stress goes with hand in hand on increased ascorbate demand because of its increased consumption and/or decreased synthesis and uptake [21]. Chronic stress-induced plasticity in the acute stress response may be important for stress adaptation, but may also contribute to pathophysiological conditions associated with stress. Thus, understanding the neural mechanisms underlying such adaptations may help us understand the aetiology of such disorders, and contribute to the future development of more effective treatment or prevention strategies. However, some data suggesting that differing plasma concentrations of ascorbic acid regulate in vivo steroid genesis by altering the activity of the membrane-bound enzyme adenylate cyclise. In spite of the fact that heat stress affected changes of AA in serum and adrenals, the same duration of cold exposure did not alter the concentration of AA in the liver, serum and adrenals. Here the results demonstrate that the exposure of rats to heat stress caused the ascorbic acid turnover e.g. the increase in the amount of ascorbate in blood due to the depletion of adrenals content, without de novo hepatic synthesis and support previous findings that heat is the stronger stressor in comparison to cold [22][23].

Conclusion

Hence Bacopamonnieri is used in indigenous systems of medicine for the treatment of various nervous system ailments such as insomnia, anxiety, epilepsy, and hysteria [24]. Preclinical and clinical studies have shown that BM improves memory and mental function.

It is believed that the components of BM extract aid in the repair of damaged neurons, neuronal synthesis, and the restoration of synaptic activity, and ultimately in nerve impulse transmission [25]. Bacopamonnieri has an adverse effect on rats on improvement in Alzheimer's disease and other clinical disorders. These experiments and researches make and provide a pathway in the treatment of complicated human disorders. We find that the active components of B. Monnieri i.e., bacoside A and B are the triterpenoid saponins and they may be responsible for the protective effect of this herb. Moreover, it seems that B. monniera extract possess certain additional antioxidative and neuroprotective properties due to which it may be used as herbal supplement for the treatment of neurodegenerative disorders and neuron degenerations [14].

Corresponding Author

Madhusudhan Vasantharajan

Senior Lecturer, Department of Pedodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University,

162, Poonamallee High Road,Chennai 600077,TamilNadu, India

Email: dr.madhusudhan24@gmail.com

Telephone Number: +91 9790166987

Reference

1. Export-Import Bank of India Indian Medicinal Plants: A Sector Study. Occasional paper No. 54, 2. Quest Publications, Bombay, India (1997).
2. J.C.Prakash, M. Sirsi, Effect of bacoside, alcoholic extract of Bacopamonnieri Linn. (Brahmi), on experimental amnesia in mice, J. Sci. Industry. Res.1962; 21C:93
3. H. K. Singh, B.N. Dhawan,Effect of Bacopa monniera Linn. (Brahmi) extract on avoidance responses in rat,J Ethnopharmacol. 1982 Mar; 5(2):205-14.
4. Singh HK, Dhawan BN, Drugs affecting learning and memory, In Lectures in Neurobiology 1, 1992:189-207.

5. Con Stough, Hemant Singh, and Andrea Zangara, Mechanisms, Efficacy, and Safety of Bacopamonnieri(Brahmi) for Cognitive and Brain Enhancement, Indian J. Pharm, 1994, 259-272.
6. H. N. Aithal, M. Sirsi,Ethanolic extract of Bacopa monniera (Brahmi) induces shortening of cell-cycle durations in naturally synchronous Physarumpolycephalum Indian J. Pharm 1961:23:2
7. D. K. Ganguly, C. L. Malhotra, A Review on major Bioactivities of Bacopamonnieri, Indian J. Med. Res.,1967; 55:473.
8. Aguiar S, Borowski T, Neuropharmacological Review of the Nootropic Herb Bacopamonnieri, Rejuvenation Research. 1995; 16(4):313-326.
9. Racchi M., Mazzuchelli M., Porrello E., Lanni C. And Govoni S. Acetyl cholinesterase inhibitions: novel activities of old molecules. Pharmacological Research. 2004; 50:441-451.
10. Singh HK, Dhawan BN, Neuropsycho pharmacological effects of the Ayurvedic NootropicBacopa Monniera Linn. (Brahmi). Indian J Pharmacology 1997; 29:359–365.
11. Schulz V., Ginkgo extract or cholinesterase inhibitors in patients with dementia; what clinical trial and guidelines fail to consider. Phytomedicine 10 2003;4: 74-79.
12. Garai S, Mahato SB, Ohtani K, Yamasaki K, Dammarane type triterpenoid saponin from Bacopamonnier, Phytochemistry 1996; 42:815-820.
13. Tripathi, Y.B., Chaurasia, S., Tripathi, E., Upadhyay, A., Dubey, G.P.,Bacopa monniera Linn. As an antioxidant: mechanism of action. Indian J. Exp. Biol. 1996; 34:523-526.
14. Sairam, K., Dorababu, M., Goel, R.K., Bhattacharya, S.K., Antidepressant activity of standardized extract of Bacopa monniera in experimental models of depression in rats. Phytomedicine 2002; 9:207-211.
15. Ahirwar S. , Tembhre M. , Gour S. , Namdeo A. Toxicology Laboratory, Sant Hirdaram Girls College, Bhopal - 462030, India.
16. OutiKontkanen, MerjaLakso, Garry Wong, and Eero Castrén, Chronic Antipsychotic Drug Treatment Induces Long-lasting Expression of fos and jun Family Genes and Activator Protein 1 Complex in the Rat Prefrontal Cortex, Neuropsychopharmacology 2002;27:152–162.

17. Bhattacharya SK, Bhattacharya A, Kumar A, Ghosal S, Antioxidant activity of Bacopamonnieri in rat frontal cortex, striatum and hippocampus. Phytother Res. 2000;14:174–179.
18. Chowdhuri DK, Parmar D, Kakkar P, Shukla R, Seth PK, Srimal RC.Anti-stress effects of bacoside of Bacopamonnieri: modulation of Hsp70 expression, superoxide dismutase and cytochrome P450 activity in rat brain. Phytother Res. 2002; 16:639-45.
19. Bánhegyi, G., Braun, L., Csala, M., Puskás, F., Somogyi, A., Kardon, T., and Mandl, J. Ascorbate and Environmental stress. Ann. N.Y. Acad. Sci. 1998;851:292-303.
20. Djordjevic, J., Cvijic, G., and Davidovic, V. Different activation of ACTH and corticosterone release in response to various stressorsin rats. Phys. Res. 2003; 52:67-72.
21. Doulas N.L., Constantopoulos, A. and Litsios, B., Effect of ascorbic acid on guinea pig adrenal adenylate cyclase activity and plasma cortisol. J. Nutr. 1987; 11(7):1108-1114.
22. Nadkarni, K.M., Indian MateriaMedica. Popular PrakashanPvt. Ltd., Mumbai, 1976:624-625.
23. Roodenrys, S., Booth, D., Bulzoni, S., Phipps, A., Micallef, C., Smoker, J., Chronic effects of Brahmi (Bacopamonnieri) on human memory. Neuropsychopharmacology 2002; 27:279-281.
24. Enz A., Amstutz R., Boddeke H., Gmelin G. And Malanowski J., Brain selective inhibition of acetylcholinesterase: a novel approach to therapy for Alzheimer's disease. Prog Brain Res. 1993;98:431-38.
25. Shiek N, Ahamd A, Siripurapu KB, Kuchibhotia VK, Singh S, Palit G, Effect of Bacopa monniera on stress induced changes in plasma corticosterone and brain monoamines in rats, J Ethnopharmacol. 2007; 111(3):671-6.

Effect of Pregnancy on Body Mass Index

Karthic.A [1], Gayathri Devi [2], Remmiya Mary Varghese [3]

Graduate Student [1],Senior Lecturer [2],Department of Physiology

Senior Lecturer [3],Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

The increase prevalence of obesity will lead to a critical challenge to healthcare service. So it is necessary to examine the BMI in the early stage of pregnancy. As a result increase in the BMI at the time of pregnancy may leads to many critical problems and even it may leads to miscarriage or stillbirth. Excessive leanness and obesity are associated with different adverse pregnancy outcomes with major maternal and fetal complications.A pregnancy woman are underweight due to genetics, metabolism, lack of food or illness .Being underweight is associated with certain medical conditions, including hyperthyroidism, cancer or tuberculosis. People with gastrointestinal or liver problems may be unable to absorb nutrients adequately. Pregnancy women who are malnutrative underweight raise special concerns, as not only gross caloric intake may be inadequate, but also intake and absorption of other vital nutrients, especially essential amino acids and micro-nutrients such as vitamins and minerals

Key Words :*Pregnancy, body weight, BMI, Mass index, obese*

Introduction

Our BMI is calculated based on the relationship between our height and weight. As a result BMI above is used to determine whether the person are underweight, just right , overweight or obese .The expanding predominance of weight in young ladies is a noteworthy general wellbeing concern. These patterns majorly affect pregnancy results in these ladies, which have been reported by a few scientists. The rising rate of corpulence is a noteworthy general wellbeing worry in the West, where 28% of pregnant ladies are overweight and 11% are stout [1]. In the United States, the occurrence of weight in pregnancy changes from 18.5% to 38.3% as indicated by the definition utilized [2-5]. In the UK, 56% of all ladies are over the suggested BMI, with 33% of them named overweight (BMI > 25) and 23% corpulent (BMI > 30). In spite of the fact that the correct rate of corpulence in pregnant ladies in the UK isn't known, the Confidential Enquiry into Maternal and Child Health 2004 [6] announced that 35% of every single maternal passing happening in the triennium 2000– 2002 were in fat ladies with Body Mass Index > 30 Kg/m2. Pregnancy entanglements in overweight ladies were considered as ahead of schedule as 1945 [7].

From that point forward, various investigations have revealed a reasonable relationship between maternal overweight and antagonistic obstetric and perinatal results. Information from North America have been bolstered by comes about because of Danish [8, 9] and Swedish investigations [10, 11]. In the UK, Sebire [12] examined the impacts of maternal corpulence on pregnancy results in a London companion of 287,213 ladies. From that point forward, comparable reports have been distributed from Wales [13] and Scotland [14]. There are prior examinations that have explored the effect of maternal weight pick up amid pregnancy on chose obstetric results, e.g., macrosomia, meconium recoloring, cesarean conveyance rate, fetal development and gestational length. The vast majority of them were confined to maybe a couple maternal BMI classes or the impact of gestational weight pick up was dissected after changes for maternal prepregnancy BMI.

The impact of maternal underweight on obstetric execution is less clear. While a few specialists [15] have discovered expanded frequencies of preterm conveyance, low birth weight

and expanded perinatal misfortune in these ladies, others [16] have detailed a defensive impact of maternal underweight on certain pregnancy complexities and intercessions. Past research has discovered a solid relationship between expanding BMI and pregnancy prompted hypertension. A meta-examination of the danger of pre-eclampsia related with maternal BMI [17] demonstrated that the danger of pre-eclampsia multiplied with every 5 to 7 Kg/m2 increment in pre pregnancyBMI.Late surveys [18] on weight and pregnancy have featured a few issues pertinent to research and administration approach. Initially, the absence of standard meanings of overweight and heftiness makes correlation of discoveries crosswise over examinations troublesome. While most reports characterize stoutness as an expanded weight file of more prominent than or equivalent to 30 Kg/m2 (IOM), others have characterized it as expanded abdomen perimeter, expanded midsection – hip proportion or body weight of in excess of 90 Kg. This makes correlation of concentrates troublesome and may have suggestions in the administration of typical pregnancy, as in the United States, prescribed gestational weight pick up is subject to ladies' pre pregnancy BMI classifications [19].

BMI Calculation Formula

BMI = (Weight in pound / Height in inches ^{2}) x 703.
BMI = (Weight in kilograms / Height in meters ^{2}).

Based on the calculation of BMI every individual is analysis as underweight, normal weight, overweight, obese.

BMI of

1) Under 18.5 – you are considered underweight and possibly malnourished.
2) 18.5 to 24.9 – you are within a healthy weight range for young and middle-aged adults.
3) 25.0 to 29.9 – you are considered overweight.
4) Over 30 – you are considered obese.

BMI Category and Its Risk During Pregnancy

Underweight

Underweight is a term describing a human whose body weight is considered too low to be healthy. The definition usually refers to people with a body mass index (BMI) of under 18.5 or a weight 15% to 20% below that normal for their age and height group.

The previous Studies have demonstrated that some results of pregnancy are related with low pre pregnancy weight. They found that underweight ladies demonstrated expanded hazard for SGA, and low birth weight babies in contrast with ordinary weight patients. The biologic systems basic the relationship between maternal undernutrition status and slower foetal development and improvement stay theoretical. Ross et al. discovered that ladies with underweight had a littler plasma volume, lower cardiac output, increments in peripheral vascular protection, and lower rennin-aldosterone reaction in pregnancy contrasted and typical weight ladies. It appears to be likely that such lacking maternal hemodynamic changes might be related with uteroplacental inadequacy and the expanded pervasiveness of little for gestational age babies watched. Then again late research shows that movement of placental transport instruments might be specifically balanced by maternal sustenance. Direct lack of healthy sustenance amid pregnancy caused an expansion in maternal-foetal limit with respect to glucose transport, which was in any event somewhat clarified by an expansion in all out glucose transporter (GLUT) wealth. These reactions permit to manage typical fetal development, regardless of interminable maternal hypoglycaemia and a decline in the maternal-fetal angle in blood vessel plasma glucose fixation. Amid endless supplement lack the advancement of significant foetal hypoglycaemia supports the maternal foetal inclination in glucose focus by confining the turnaround exchange of glucose to the placenta, and decreasing placental glucose utilisation. There is prompted fetal gluconeogenesis, what outcome is lessened foetal tissue protein combination and easing back of foetal development to a rate that can be supported by the decreased placental supplement supply.

Causes and Effect

A Pregnancy woman are underweight due to genetics, metabolism, lack of food (frequently due to poverty), or illness .Being underweight is associated with certain medical conditions, including hyperthyroidism, cancer or tuberculosis. People with gastrointestinal or liver problems may be unable to absorb nutrients adequately. Pregnancy women with anorexia nervosa become underweight due to self-starvation (often accompanied by excessive exercise)[20].

Underweight might be secondary to or symptomatic of an underlying disease. Unexplained weight loss may require professional medical diagnosis. Underweight can also be a primary causative condition. Severely underweight individuals may have poor physical stamina and a weak immune system, leaving them open to infection[21][22]."Underweight status and micronutrient deficiencies also cause decreases in immune and non-immune host defenses, and should be classified as underlying causes of death if followed by infectious diseases that are the terminal associated causes.

Pregnancy women who are malnutrative underweight raise special concerns, as not only gross caloric intake may be inadequate, but also intake and absorption of other vital nutrients, especially essential amino acids and micro-nutrients such as vitamins and minerals. Incase pregnancy women, being grossly underweight can result in amenorrhea (absence of menstruation), infertility and possible complications during pregnancy. It can also cause anaemia and hair loss [23].

Over Weight

Overweight is generally defined as having more body fat than is optimally healthy. Being overweight is a common condition, especially where food supplies are plentiful and lifestyles are sedentary. Excess weight has reached epidemic proportions globally, with more than 1 billion pregnancy woman being either overweight or obese. Increases have been observed across all age groups.

A healthy body requires a minimum amount of fat for the proper functioning of the hormonal, reproductive, and immune systems, as thermal insulation, as shock absorption for sensitive areas, and as energy for future use. But the accumulation of too much storage fat can impair movement and flexibility, and can alter the appearance of the body [24].

The past investigations demonstrated that the high chances and complications of DM and hypertension previously and amid pregnancy are expanded in corpulent and overweight ladies. A meta-investigation investigating the relationship between gestational DM and BMI evaluated that the danger of creating gestational DM is two and four times higher among overweight and stout ladies individually contrasted and typical weight pregnant ladies. Insulin protection assumes a vital part in these pathologies. In normal weight ladies, pregnancy is as of now connected with a dynamic diminishing in insulin affectability amid the pregnancy. This metabolic change gives off an impression of being amplified in stout ladies. The fringe as well as hepatic insulin protection apparently is expanded in glucose-tolerant pregnant ladies, when contrasted and thin or normal weight women; fringe insulin affectability is 40% less in stout women.

As for the gestational and pre-gestational pathology, no measurably critical connections between heftiness or overweight and rehashed urinary tract diseases were watched. However different examinations, have shower an expanded frequency of urinary tract contamination in hefty pregnant women.

Causes and Effect

Being overweight is generally caused by the intake of more calories (by eating) than are expended by the body (by exercise and everyday living). Factors that may contribute to this imbalance include:

1. Eating disorders (such as binge eating)
2. Genetic predisposition
3. Hormonal imbalances (e.g. hypothyroidism)
4. Insufficient or poor-quality sleep
5. Limited physical exercise and sedentary lifestyle
6. Poor nutrition
7. Metabolic disorders, which could be caused by repeated attempts to lose weight by weight cycling

8. Overeating
9. Psychotropic medication (e.g. olanzapine)
10. Stress

So Pregnant women who are overweight or obese should speak with their health care provider about limiting weight gain and being physically active during pregnancy.

Obese

If a pregnancy woman are suffering from Obesity is stated as medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems. Pregnancy woman are considered obese when their body mass index (BMI), a measurement obtained by dividing a woman's weight in kilograms by the square of the woman's height in metres, exceeds 30 kg/m2.

Indeed, even direct overweight is a hazard factor for gestational diabetes and hypertensive issue of pregnancy, and the hazard is higher in subjects with clear corpulence. Contrasted and ordinary weight, maternal overweight is identified with a higher danger of cesarean conveyances and a higher rate of sedative and postoperative intricacies in these conveyances. Low Apgar scores, macrosomia, and neural tube deserts are more successive in newborn children of fat moms than in babies of typical weight moms. The provincial dissemination of fat regulates the impacts of weight on starch resilience, hemodynamic adjustment, and fetal size. Maternal corpulence increments perinatal mortality .Long term intricacies incorporate compounding of maternal corpulence and advancement of weight in the newborn child. Previously established inclination directing, cautious pre-birth administration, tight checking of weight pick up, and long haul follow-up could limit the social and monetary results of pregnancies in overweight ladies.

Causes and Effect

Pregnancy women who is Obese increases the likelihood of various diseases, particularly heart disease, type 2 diabetes, obstructive sleep apnea, certain types of cancer, and osteoarthritis. Obesity is most commonly caused by a combination of excessive food energy intake, lack of physical activity, and genetic susceptibility, although a few cases are caused primarily by genes,

endocrine disorders, medications or psychiatric illness. Evidence to support the view that some obese pregnancy woman eat little yet gain weight due to a slow metabolism is limited; on average obese pregnancy woman have a greater energy expenditure than their thin counterparts due to the energy required to maintain an increased body mass[25].

Dieting and physical exercise are the mainstays of treatment for obesity. Diet quality can be improved by reducing the consumption of energy-dense foods such as those high in fat and sugars, and by increasing the intake of dietary fiber [21]. Anti-obesity drugs may be taken to reduce appetite or inhibit fat absorption together with a suitable diet. If diet, exercise and medication are not effective, a gastric balloon may assist with weight loss, or surgery may be performed to reduce stomach volume and/or bowel length, leading to earlier satiation and reduced ability to absorb nutrients from food.

Obesity is a leading preventable cause of death worldwide, with increasing prevalence in pregnancy woman and children, and authorities view it as one of the most serious public health problems of the 21st century. Obesity is stigmatized in much of the modern world (particularly in the Western world)[26], though it was widely perceived as a symbol of wealth and fertility at other times in history, and still is in some parts of the world. In 2013, the American Medical Association classified obesity as a disease

Effect of BMI during Pregnancy

If the BMI is high during the starting stage of pregnancy its lead to higher risk (ie If the body weight is over 100kg at the starting stage of the pregnancy and if the BMI is more than 35 than she should refer to an obstetrician[26].Being obese during pregnancy increase the risk of various pregnancy complication include

1. Gestational diabetes is refer to a woman who are obese during pregnancy will have diabetes.
2. Preeclampsia is refer to a woman who are obese during pregnancy will have high blood pressure

3. Infection is refer to a woman who are obese during pregnancy are at increased risk of urinary tract infection and it lead to high risk at the time of delivery

4. Thrombosis is refer to a woman who are obese during pregnancy will have a blood clot forms inside a blood vessel

5. Obstructive sleep apnoea is refer to a woman who are obese during pregnancy will have serious sleep disorder in which breathing repeatedly stops and starts

6. Pregnancy loss increase in BMI may even lead to miscarriage and stillbirth.

   So Pregnancy weight development was based on measurements generally carried out every fourth week during the first half of the pregnancy, every second week during weeks 20 to 32 and once weekly thereafter. Ninety-six percent of the infants were delivered between during Gestation week 36 to 42. Results presented demonstrate that data were not systematically biased by pregnancy duration.

Conclusion

Pregnancy outcome is worst in babies from mothers with low body mass index as compared to healthy weight mothers with respect to increased incidence of preterm birth, lower birth weight and increased neonate mortality on the neonatal ward.

To conclude, pregnancy complications related to maternal BMI is a growing problem. Both lean and obese mothers carry an increased risk of adverse perinatal outcome. Given the major economic and medical consequence of pregnancy in these women, all attempts should be made to maintain a normal BMI in women of childbearing age. Pre-pregnancy counseling, health programs and appropriate multidisciplinary management should be done. Last, we acknowledge the small sample size as being a weakness of the present study, but this was due to it being a prospective Study carried out in one unit of a tertiary care teaching hospital over a period of 1 year only, with many exclusion criteria.

Corresponding Author

Remmiya Mary Varghese

Senior Lecturer, Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id-remmiya@gmail.com

Phone Number: 9840488439

References

1. Bhattacharya S, Campbell DM, Liston WA, Bhattacharya S. Effect of body mass index on pregnancy outcomes in nulliparous women delivering singleton babies. BMC public Health. 2007 Dec;7(1):168.
2. Garbaciak Jr JA, Richter M, Miller S, Barton JJ. Maternal weight and pregnancy complications. American Journal of Obstetrics and Gynaecology. 1985 May 15;152(2):238-45.
3. Abrams BF, Laros RK. Prepregnancy weight, weight gain, and birth weight. American Journal of Obstetrics &Gynaecology. 1986 Mar 1;154(3):503-9.
4. Naeye RL. Maternal body weight and pregnancy outcome. The American journal of clinical nutrition. 1990 Aug 1;52(2):273-9.
5. Cogswell ME, Serdula MK, Hungerford DW, Yip R. Gestational weight gain among average-weight and overweight women—what is excessive?. American Journal of Obstetrics &Gynaecology. 1995 Feb 1;172(2):705-12.
6. Lewis G. Confidential enquiries into maternal and child health. Why mothers die. The Sixth Report of the United Kingdom Confidential Enquiries into Maternal Deaths in the United Kingdom. London. RCOG Press. 2004.
7. Odell Ld, MengertWf. The overweight obstetric patient. Journal of the American Medical Association. 1945 May 12;128(2):87-90.

8. Rode L, Nilas L, Wøjdemann K, Tabor A. Obesity-related complications in Danish single cephalic term pregnancies. Obstetrics &Gynaecology. 2005 Mar 1;105(3):537-42.
9. Kristensen J, Vestergaard M, Wisborg K, Kesmodel U, Secher NJ. Pre-pregnancy weight and the risk of stillbirth and neonatal death. BJOG: An International Journal of Obstetrics & Gynaecology. 2005 Apr 1;112(4):403-8.
10. Cedergren MI. Maternal morbid obesity and the risk of adverse pregnancy outcome. Obstetrics &Gynaecology. 2004 Feb 1;103(2):219-24.
11. Sebire NJ, Jolly M, Harris JP, Wadsworth J, Joffe M, Beard RW, Regan L, Robinson S. Maternal obesity and pregnancy outcome: a study of 287 213 pregnancies in London. International journal of obesity. 2001 Aug;25(8):1175
12. UshaKiran TS, Hemmadi S, Bethel J, Evans J. Outcome of pregnancy in a woman with an increased body mass index. BJOG: an international journal of obstetrics & gynaecology. 2005 Jun 1; 112(6):768-72.
13. Smith GC, Shah I, Pell JP, Crossley JA, Dobbie R. Maternal obesity in early pregnancy and risk of spontaneous and elective preterm deliveries: a retrospective cohort study. American journal of public health. 2007 Jan; 97(1):157-62.
14. Spinillo A, Capuzzo E, Piazzi G, Ferrari A, Morales V, Mario MD. Risk for spontaneous preterm delivery by combined body mass index and gestational weight gain patterns. ActaobstetriciaetgynecologicaScandinavica. 1998 Jan 1;77(1):32-6.
15. Sebire NJ, Jolly M, Harris J, Regan L, Robinson S. Is maternal underweight really a risk factor for adverse pregnancy outcome? A population-based study in London. BJOG: an international journal of obstetrics & gynaecology. 2001 Jan 1;108(1):61-6
16. O'brien TE, Ray JG, Chan WS. Maternal body mass index and the risk of preeclampsia: a systematic overview. Epidemiology. 2003 May 1;14(3):368-74.
17. Krishnamoorthy U, Schram CM, Hill SR. Maternal obesity in pregnancy: is it time for meaningful research to inform preventive and management strategies?. BJOG: An International Journal of Obstetrics & Gynaecology. 2006 Oct 1;113(10):1134-40.
18. Sahu MT, Agarwal A, Das V, Pandey A. Impact of maternal body mass index on obstetric outcome. Journal of Obstetrics and Gynaecology Research. 2007 Oct 1;33(5):655-9.

19. Ölin A, Rössner S. Factors related to body weight changes during and after pregnancy: the Stockholm Pregnancy and Weight Development Study. Obesity. 1996 May 1;4(3):271-6.
20. Ahmadu BU, Gofama MM, Ashir GM, Ayub-Eniola AA, Abdulrahman M, Ambe JP. The effect of maternal pregnancy body mass index as a measure of pregnancy weight gain on neonatal birth weight in Maiduguri metropolitan council of Borno state, Nigeria. Greener J Med Sci. 2012 Dec;2(6):168-72.
21. Yazdani S, Yosofniyapasha Y, Nasab BH, Mojaveri MH, Bouzari Z. Effect of maternal body mass index on pregnancy outcome and newborn weight. BMC research notes. 2012 Dec;5(1):34.
22. Nohr EA, Vaeth M, Baker JL, Sørensen TI, Olsen J, Rasmussen KM. Combined associations of prepregnancy body mass index and gestational weight gain with the outcome of pregnancy–. The American journal of clinical nutrition. 2008 Jun 1;87(6):1750-9.
23. Zaaba NA, Roy A, Lakshmi T. Perception of women on the adverse effect of drugs on the fetus during pregnancy. Journal of Advanced Pharmacy Education & Research| Apr-Jun. 2017;7(2).

Effects of vitamin D on Bones – A Review.

Sangavi R [1], Anitha Roy [2], Aravindh Kumar [3]

Graduate student[1] ,Senior Lecturer [2], Department of Biochemistry

Professor [3], Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract:

Vitamin D refers to a group of fat-soluble secosteroids responsible for enhancing intestinal absorption of calcium, iron, magnesium, phosphate and zinc. Cholecalciferol and ergocalciferol can be ingested from the diet and from supplements. The body can also synthesize vitamin D (specifically cholecalciferol in the skin, from cholesterol, when sun exposure is adequate hence its nickname, the "sunshine vitamin". This article summarizes the beneficial and importance of the Vitamin D and its action on bone.

Keywords:*Vitamin D, Hormone, Calcium, Osteoporosis, 24-25 dihydroxycholecalciferol.*

Introduction:

Vitamin D is a vitamin. Found in small amounts in foods like fatty fish such as herring, mackerel, sardines and tuna. In humans, the most important compounds in this group are vitamin D_3 (also known as cholecalciferol) and vitamin D_2 (ergocalciferol)[1].Vitamin D is made more available by adding it to dairy products, juices, and cereals known as “fortified with vitamin D.” But most vitamin D what the body gets is obtained through exposure to sunlight. Vitamin D can also be made in the laboratory as medicine. Vitamin D is used for preventing and treating rickets

which is a disease caused due to lack of vitamin D .Vitamin D is used in treatment of weak bones in cases like osteoporosis and also bone loss seen in conditions like hyperthyroidism. It is also used for preventing fractures in people who are at risk for osteoporosis and prevents low calcium and bone loss in people with kidney failure. Vitamin D is used for conditions of the heart and blood vessels, including high blood pressure and high cholesterol.

Dietary Sources:

Vitamin D is found in few dietary sources [2]. Sunlight exposure is the primary source of vitamin D for majority of people, other than supplements [3].

Vitamin D_2

Main article: Ergocalciferol

Fungus as obtained from USDA nutrient database (per 100 g) [4]. Low values in mushrooms for vitamin D below indicate incidental exposure to sunlight which activates synthesis of vitamin D2. Vitamin D levels can be controlled at much higher levels especially when fresh mushrooms or dried powders are purposely exposed to artificial sunlight with the use of an ultraviolet lamp [5]

- Mushrooms, portabella, exposed to ultraviolet light, raw: Vitamin D_2: 11.2 µg (446 IU)
- Mushrooms, portabella, exposed to ultraviolet light, grilled: Vitamin D_2: 13.1 µg (524 IU)
- Mushrooms, shiitake, dried: Vitamin D_2: 3.9 µg (154 IU)
- Mushrooms, shiitake, raw: Vitamin D_2: 0.4 µg (18 IU)

Vitamin D2 known as ergocalciferol which is found in fungi, is synthesized from viosterol when ultraviolet light stimulates and activates ergosterol.

Mushrooms enhanced with Vitamin D2 due to Ultraviolet irradiation have same bioavailability as compared to other Vitamin D2 supplement in humans. Vitamin D2 from UV-irradiated yeast baked into bread or mushrooms is bioavailable and increases blood levels of 25(OH) D.[6]

Plants

- Alfalfa (*Medicago sativa subsp. sativa*), shoot: 4.8 µg (192 IU) vitamin D_2, 0.1 µg (4 IU) vitamin D_3 (per 100 g).[7]

Vitamin D_3

Main article: Cholecalciferol

Staple foods are artificially fortified with vitamin D is practiced in some countries.

- Vegan sources

*lichen

*Vitamin D3 contents range from 0.67 to 2.04 µg g^{-1} dry matter in the thalli of C. arbuscula specimens grown under different natural conditions.[8]

- Animal sources[9]
  - Fish liver oils, such as cod liver oil, 1 Tablespoon (15 ml) provides 1,360 IU (90.6 IU/ml)
  - Fatty fish species, such as:
    - Catfish (wild), 85g (3 oz) provides 425 IU (5 IU/g)
    - Salmon, cooked, 100g (3.5 oz) provides 360 IU (3.6 IU/g)
    - Mackerel, cooked, 100g, 345 IU (3.45 IU/g)
    - Sardines, canned in oil, drained, 50g (1.75 oz), 250 IU (5 IU/g)
    - Tuna, canned in oil, 100g, 235 IU (2.35 IU/g)
    - Eel, cooked, 100g, 200 IU (2.00 IU/g)
  - A whole egg provides 20 IU if egg weighs 60g (0.333 IU/g)
  - Beef liver, cooked, 100g, provides 15 IU (0.15 IU/g)

Molecular Mechanism Of Vitamin D Action:

The hormonal metabolite of Vitamin D, Vitamin D_3 (1,25D) binds to the Vitamin D Receptor (VDR) and initiates biological responses. When occupied by 1,25D, VDR interacts with the Retinoid X Receptor (RXR) to form a heterodimer that binds to vitamin D responsive elements in the region of genes directly controlled by 1,25D. Ligand-activated VDR-RXR modulates the

transcription of genes encoding proteins which regulates the functions of vitamin D which is signaling intestinal calcium and phosphate absorption to affect skeletal and calcium homeostasis with the help of complexes like coactivators and corepressors. Action of Vitamin D depends on expression of adequate VDR and RXR co receptor proteins and cell-specific programming of transcriptional responses to regulate select genes that encode proteins that function in mediating the effects of vitamin D.

Vitamin D is widespread in nature, and one of its roles in vertebrate animals and humans is to promote the absorption of calcium and phosphate to enable normal mineralization of the skeleton [10]. Vitamin D deficiency in childhood makes children prone to rickets where defective mineralization of growth plate cartilage and bone leads to morphologically altered long bones, resulting in curvature and deformation [11]. Osteomalacia is a condition where the bone matrix fails to mineralize, resulting in bone pain, muscle weakness and increased risk of bone deformation and fracture which occurs as a result of vitamin D deficiency in adults [12]. The characteristic feature of a vitamin D deficiency is a low serum 25(OH) D [13] concentration, which is associated with decreases in both serum 1, 25-(OH) 2D [14]. In turn, low serum calcium results in increased secretion of parathyroid hormone (PTH)which promotes the production of 1,25-(OH) 2D [15]. As a result, serum concentrations return to normal but are accompanied by higher serum concentration of PTH, indicative of secondary hyperparathyroidism [16]. Increases in serum PTH concentration have been reported to stimulate the rate of bone turnover, causing a reorganization of the bone structure [17]. Hence, secondary hyperparathyroidism has been demonstrated as the predominant factor by which vitamin D deficiency results in increased susceptibility to bone fractures.

Vitamin D And Bone Formation:

Treatment of rachitic chicks with 1-hydroxyvitamin D3, the synthetic analogue of 1,25-dihydroxyvitamin D3, does not prevent most of the changes seen in bone affected by rickets, in spite of normal plasma levels of calcium and inorganic phosphate (unpublished). Similar findings have been reported for man [18], suggesting that vitamin D affects bone formation directly and that the effect is mediated by a metabolite(s) other than 1, 25-dihydroxyvitamin D3. In normocalcaemic animals, the predominant dihydroxylated metabolite of vitamin D in the

blood is 24, 25-dihydroxyvitamin D and, like 1,25-dihydroxyvitamin D, it is synthesised in the kidney[19][20]; but nothing is known of its function. Improved calcium retention has been reported in anephric patients treated with 24, 25-dihydroxyvitamin D3, which has been observed to accumulate preferentially in fetal bones of rat [21] and in callus tissue formed after experimental fracture (unpublished). It reports that when rachitic chicks are exposed to various therapeutic treatments with metabolites of vitamin D, the presence of 24, 25-dihydroxyvitamin D is essential for the healing process.

Vitamin D And The Actions Of Its Metabolites On Bone:

The vitamin D endocrine system has clear beneficial effects on bone as demonstrated by prevention of rickets in children and by reducing the risk of osteomalacia or osteoporosis in adults or elderly subjects. Depending on the design of the study of genetically modified animals, however, 1, 25(OH)2 D and the vitamin D receptor (VDR) may have no effect, beneficial or even deleterious direct effects on bone. In case of sufficient calcium supply, vitamin D and its metabolites can improve the calcium balance and facilitate mineral deposition in bone matrix largely without direct effects on bone cells, although some beneficial effects may occur via mature osteoblasts, as demonstrated in mice with osteoblast-specific overexpression of VDR or 1α-hydroxylase [22]. In case of calcium deficiency, however, 1, 25(OH) 2 D enhances bone resorption, whereas simultaneously inhibiting bone mineralization, so as to defend serum calcium homeostasis at the expense of bone mass. This dual role probably provides a survival benefit for land vertebrates living in a calcium-poor environment.

Vitamin D has a well-recognized role in bone biology, being required for normal bone formation and normal mineralization. The uncertainty that will be addressed in this review is how much of its effects on bone are secondary to its actions on gut calcium and phosphate absorption and how much relate to direct effects on bone. Moreover, if there are effects directly on bone, how much of any bone activity is on bone formation and how much on bone resorption. Conflicting data suggest that these actions may differ by timing, skeletal site and dietary calcium intake.

In the invivo studies, in vitamin D receptor knockout (Vdr−/−) models, there was the expected phenotype similar to various forms of vitamin D-deficient or -resistant rickets. There were

similar phenotypes in models of knockout of the 1α-hydroxylase (CYP27B1) enzyme. The findings in these studies underpin the critical role of vitamin D in normal calcium and bone/tooth/growth plate homeostasis. Vitamin D is generally associated not only with improved bone mineralization but also with increased bone resorption, and thus may seem to represent 'good' and 'bad' effects on bone. *In vitro* studies have readily demonstrated bone resorbing effects responses to 1, 25-dihydroxyvitamin D3 (1, 25(OH) 2D3) [23],

Interactions between cells of the osteoblast and osteoclast lineages, including osteocytes and possible role of chondrocytes. The regulatory pathways between osteoblasts and osteocytes and osteoclasts include multiple negative (red) and positive (green) feedback loops as indicated; all potentially modifiable by 1,25(OH)2D3. These include release of matrix components, such as transforming growth factor-β (TGFβ) and bone morphogenetic proteins (BMPs) that influence osteoblast activity, and humoral factors that influence mineral homeostasis at distant sites, such as fibroblast growth factor (FGF)-23 on renal phosphate handling. Given these loops, any regulatory effect on one cell type can influence the others. Chondrocyte effects on surrounding cells are also potentially involved. The complexity of these inter-relationships may explain the differential effects of 1, 25(OH) 2D3 in driving anabolic and catabolic outcomes. RANKL, receptor activator of nuclear factor-κB ligand [24].

Role Of Vitamin D In Bone Homeostasis:

One way to bring the divergent data on vitamin D and bone into a unifying hypothesis is to look at the evolution of vitamin D in vertebrates published in first issue (January 2014) of *BoneKEy Reports*) [25]. The vitamin D endocrine system has already been identified in early fish such as lamprey but still without any detectable effect on calcium and certainly not on the still nonexistent mineralized bone. In at least some late teleosts, vitamin D metabolites via VDR probably stimulate a positive calcium balance (possibly more in gills than in the intestine with TRPV channels as mediator). This seems logical as to allow the building up of a large bone structure (mainly acellular, thus without osteocytes and without multinuclear osteoclasts). This is apparent in all amphibians tested, and such an evolution would be logical as terrestrial animals

face a calcium-poor environment. So from this early stage in the evolution of vertebrates onwards, the role of vitamin D would be to enhance a positive external calcium balance. From amphibians onwards, PTH becomes a calcium-regulating hormone using bone as a calcium reservoir in times of shortage of nutritional calcium. Whether the vitamin D hormone is already cooperative with PTH to stimulate osteoclastogenesis and bone resorption in amphibians is plausible but needs further exploration. From reptiles onwards, a new dimension is added: a complex system is developing to regulate bone mineral deposition, with hormonal and local regulation of SIBLING proteins, several enzymes such as PHEX and a complex regulation of pyrophosphate PPi. Most of these factors are under the control of 1, 25(OH) 2D3 and have, as net result, an inhibition of mineralization. This is not illogical as high 1, 25(OH) 2D3 with PTH represents the first-line defense against a low nutritional calcium supply by increasing both fractional intestinal absorption and renal calcium reabsorption. Enhanced bone resorption and simultaneous inhibition of mineral deposition can thereby avoid a futile cycle of calcium resorption from bone and its immediate reuse for mineralization of bone.

Therefore, in the first place, the effect of vitamin D action would be to defend systemic calcium homeostasis by making calcium available for the extracellular fluid pool from the intestine if possible but from any internal source if required. In case of very low external calcium supply, high levels of 1, 25(OH) 2D3 would use the bone calcium reservoir for serum calcium homeostasis at the (temporary) expense of bone mass and strength. From a teleological standpoint, this seems a logical strategy as later access to nutritional calcium could then allow the rebuilding of the skeleton. Thus 1, 25(OH) 2D3 acting through the VDR may favor replication and maintenance of immature cells of the osteoblast lineage. These roles may be critical for bone resorption and ‘preventing’ futile bone mineralization under conditions of calcium stress. On the other hand, during a ‘recovery’ phase, 1, 25(OH) 2D3 acting through the VDR acting in more mature cells may encourage terminal differentiation of mineralizing cells. Consistent with studies of action of 1, 25(OH) 2D3 on osteoblastic cells at different stages of their maturation [26]. This may provide a drive to re-mineralization of the skeleton after surviving the calcium stress situation. This divergence between sacrifice of bone for calcium homoeostasis and rebuilding of the skeleton at other times is not dissimilar from the catabolic effect of chronically high PTH levels versus the anabolic effect of transiently high PTH levels.

Phosphate homeostasis is quite different from that of calcium as terrestrial animals live in a relatively phosphate-rich environment in comparison with marine animals. A phosphaturic hormone (or hormones) may therefore be more important than hormones stimulating phosphate uptake in the intestine or kidney. Although 1,25(OH)2D3 clearly stimulates phosphate uptake in the gut, the VDR endocrine system in the intestine seems to be redundant as selective Vdr−/− in the intestine does not modify serum or urinary phosphate concentration and therefore such animals do not show the typical growth plate abnormalities seen in vitamin D, calcium or phosphate deficiency.

That vitamin D has both beneficial and deleterious effects on bone are not an exception for ligand of nuclear receptors, such as estrogens, androgens or glucocorticoids. In fact, for each of these nuclear receptors it has allowed the development of structural analogs of the natural hormone that have more beneficial and less deleterious effects (for example, selective estrogen receptor modulators [DeLuca [27]]).

Vitamin D And Sclerostin:

Objective Serum sclerostin levels have been reported to be inversely associated with serum 25OHD levels, but the effect of vitamin D and calcium supplementation on serum sclerostin levels is unknown. Men and women appear to have different serum sclerostin responses to vitamin D and calcium supplementation [28].

Role Of Vitamin D In Healing Of Fracture:

Fracture healing is a process which involves response to injury, intramembranous bone formation, endochondral bone formation, and bone remodeling in order to restore the tissue to its original physical and mechanical properties. All this process is influenced by a variety of systemic and local factors. Vitamin D is said to play an intimate role in healing fracture, but very little data exists on how it does. Report suggests that the hypovitaminosis D is a possible cause of inadequate fracture healing and re fracture in children and it shows a clear effect of vitamin D supplementation on calcium.

Conclusion:

A greater understanding of how the local synthesis and activity of vitamin D within the bone micro-environment will contribute to the rationale for vitamin D and calcium supplementation. Vitamin D activities in bone provide evidence of actions regulating important aspects of bone mineral metabolism, integration of the evidence from the endocrine and autocrine activities of vitamin D are required to establish these strategies in the future.

Corresponding author:

Aravindh Kumar

Professor, Department of Orthodontics

Saveetha Dental College

Saveetha University, Saveetha Institute of Medical and Technical Science

Mobile Number: +919841299939

Email Id: dr.aravind.s@gmail.com.

REFERENCE

1) Holick MF. High prevalence of vitamin D inadequacy and implications for health. In Mayo Clinic Proceedings 2006 1; 81: 353-373.

2) Calvo MS, Whiting SJ, Barton CN. Vitamin D intake: a global perspective of current status. The Journal of nutrition. 2005 1; 135:310-6.

3) Harnly JM, Doherty RF, Beecher GR, Holden JM, Haytowitz DB, Bhagwat S, Gebhardt S. Flavonoid content of US fruits, vegetables, and nuts. Journal of agricultural and food chemistry. 2006 27; 54:9966-77.

4) Adams M. Vitamin D Posted 3 years ago under Health, Natural Health, Nutrition.

5) Eaton SB, Eaton III SB, Konner MJ. Review Paleolithic nutrition revisited: a twelve-year retrospective on its nature and implications. European journal of clinical nutrition. 1997; 51:207.

6) Hohman EE, Martin BR, Lachcik PJ, Gordon DT, Fleet JC, Weaver CM 2011 "Bioavailability and efficacy of vitamin D2 from UV-irradiated yeast in growing, vitamin D-deficient rats".*J. Agric. Food Chem.***59** :2341–6.

7) Wang T, Bengtsson G, Kärnefelt I, Björn LO "Provitamins and vitamins $D_2$and D_3 in Cladina Spp over a latitudinal gradient: possible correlation with UV levels". *J. Photochem. Photobiol. B, Biol.*62 2001: 118–22.

8) "Dietary Supplement Fact Sheet: Vitamin D".*Office of Dietary Supplements (ODS)*. National Institutes of Health (NIH). Retrieved 2010-04-11.

9) Matsumoto T, Takano T, Saito H, Takahashi F. Vitamin D analogs and bone: preclinical and clinical studies with eldecalcitol. BoneKEy Reports **3**, Article number: 513 (2014); doi:10.1038/bonekey.2014.8 (in press).

10) Lips P. Vitamin D deficiency and secondary hyperparathyroidism in the elderly: consequences for bone loss and fractures and therapeutic implications. Endocrine reviews. 2001 1; 22:477-501.

11) DeLuca HF. Overview of general physiologic features and functions of vitamin D. The American journal of clinical nutrition. 2004 1; 80:1689S-96S.

12) Rauch F, Schoenau E. Skeletal development in premature infants: a review of bone physiology beyond nutritional aspects. Archives of Disease in Childhood-Fetal and Neonatal Edition. 2002 1;86:F82-5

13) Whyte MP, Thakker RV. Rickets and osteomalacia. Medicine. 2009 1; 37:483-8.

14) Boonen S, Bischoff-Ferrari HA, Cooper C, Lips P, Ljunggren O, Meunier PJ, Reginster JY. Addressing the musculoskeletal components of fracture risk with calcium and vitamin D: a review of the evidence. Calcified tissue international. 2006 May 1; 78:257-70.

15) Hanley DA, Cranney A, Jones G, Whiting SJ, Leslie WD, Cole DE, Atkinson SA, Josse RG, Feldman S, Kline GA, Rosen C. Vitamin D in adult health and disease: a review and guideline statement from Osteoporosis Canada. Canadian Medical Association Journal. 2010 7; 182:E610-8.

16) Parfitt AM, Rao DS, Stanciu J, Villanueva AR, Kleerekoper M, Frame B. Irreversible bone loss in osteomalacia. Comparison of radial photon absorptiometry with iliac bone histomorphometry during treatment. The Journal of clinical investigation. 1985 Dec 1; 76(6):2403-12.

17) Bordier P, Zingraff J, Gueris J, Jungers P, Marie P, Pechet M, Rasmussen H. The effect of 1α (OH) D3 and 1α, 25 (OH) 2D3 on the bone in patients with renal osteodystrophy. The American journal of medicine. 1978 1; 64:101-7.

18) Holick MF, Schnoes HK, DeLuca HF, Gray RW, Boyle IT, Suda T. Isolation and identification of 24, 25-dihydroxycholecalciferol, a metabolite of vitamin D3 made in the kidney. Biochemistry. 1972 1; 11:4251-5.

19) Boyle IT, Omdahl JL, Gray RW, DeLuca HF. The biological activity and metabolism of 24, 25-dihydroxyvitamin D3. Journal of Biological Chemistry. 1973 Jun 25; 248:4174-80.

20) Suda T, Takahashi F, Takahashi N. Bone effects of vitamin D–Discrepancies between in vivo and in vitro studies. Archives of biochemistry and biophysics. 2012 1; 523:22-9.

21) Lieben L, Masuyama R, Torrekens S, Van Looveren R, Schrooten J, Baatsen P, Lafage-Proust MH, Dresselaers T, Feng JQ, Bonewald LF, Meyer MB. Normocalcemia is maintained in mice under conditions of calcium malabsorption by vitamin D–induced inhibition of bone mineralization. The Journal of clinical investigation. 2012 May 1; 122:1803-15.

22) Anderson PH, Atkins GJ. The skeleton as an intracrine organ for vitamin D metabolism. Molecular aspects of medicine. 2008 1; 29:397-406.

23) Ito N, Findlay DM, Anderson PH, Bonewald LF, Atkins GJ. Extracellular phosphate modulates the effect of 1α, 25-dihydroxy vitamin D3 (1, 25D) on osteocyte like cells. The Journal of steroid biochemistry and molecular biology. 2013 1; 136:183-6.

24) Bouillon R. Report on recent vitamin D research: ECTS 2012 and the 15th Vitamin D workshop 2012.

25) Shi YC, Worton L, Esteban L, Baldock P, Fong C, Eisman JA, Gardiner EM. Effects of continuous activation of vitamin D and Wnt response pathways on osteoblastic proliferation and differentiation. Bone. 2007 1;41:87-96.

26) DeLuca HF. The development of a bone-and parathyroid-specific analog of vitamin D: 2-methylene-19-Nor-(20S)-1 [alpha], 25-dihydroxyvitamin D3. BoneKEy reports. 2014 5; 3.

27) Dawson-Hughes B, Harris SS, Ceglia L, Palermo NJ. Serum sclerostin levels vary with season. The Journal of Clinical Endocrinology & Metabolism. 2014 1;99:E149-52.

Emerging Techniques In the Diagnosis and Treatment of Oral Candidiasis

Boopana .K [1], M.P.Brundha[2], Geo Mani[3],
Graduate Student[1], Senior Lecturer[2],Department of General Pathology,
Senior Lecturer[3],Department of Pedodontics and Preventive Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract:

Oral candidiasis is a fungal infection caused mostly by the species Candida albicans along with other causative agents like Candida tropicalis, Candida glabrata, Candida parapsilosis etc. The three main groups of candidiasis are acute, chronic and angular. The treatment involve the antifungal drugs like nystatin, miconazole and Amphotericin B. The recent advances in elucidating the interaction between the candidia organism and the environment are cellular and molecular mechanism. Polymerase chain reaction analysis, recombinant antigen technique and candida biofilm communication have found the existence of oral candidiasis. Traditional and innovative treatment modes are done to mange oral candidiasis.This review is done to learn about the advanced and emerging technique in the diagnosis and treatment of oral diagnosis.

Key Words : *fungal infection , oral lesion, Candida, advanced techniques , anti- fungal.*

Introduction:

Oral candidiasis is a significant and a familiar infection . Candida albicans is a most common fungal pathogen[1]. It is present in both pathogenic and non pathogenic states and has been able to develop a myriad of powerful mechanisms that allows for thriving colonisation and subsequent infection if the host under appropriate conditions . Candida albicans is an important pathogen that us capable if causing localised intra oral candida infection , but one that can permeate and initiate systemic effects as well [2,3]. It is considered the most important of the candida species and can be found in all areas of the mouth and on the surface of dentures. The fungi that cause oral candidiasis are considered as a part of the normal flora of the mouth [4]. Intra oral candidiasis can be based on the clinical recognition of a particular form of candida. Various clinical manifestations of oral candidiasis are present including pseudomembranous candidiasis , hyperplastic candidiasis (candidal leukoplakia), atrophic candidiasis , acute and chronic erythematous and angular cheilitis [8,9]. In addition to the clinical forms of candida for diagnosis , there are also various species of oral candida present . Some of the species commonly seen intra orally are c.albicans , c.glabarata, c.krusie, c.stellaroidea, c.tropicalis[7,8]. The main objective of this paper is to evaluate advances in the cellular and molecular mechanisms involved in the diagnosis and identification of oral candidiasis and assess the diverse novel treatment modalities for oral candidiasis in the clinical setup . Various cellular and molecular mechanisms have been employed that helps to elucidate the interactions between candida organisms and their environment [8,9]. Polymerase chain reaction analyses, candida biofilm communication , and recombinant antigen techniques have been found to facilitate explanation of the endurance and endurance and existence of oral candidiasis , specifically c.albicans. Various modes of treatment , both traditional and innovative are used utilised to manage oral candidiasis [9,10].

Cellular and Molecular Mechanism used in Oral Candidiasis for the Diagnosis of the Candida Species

The oral candida organism adhere to the oral mucosa and to the non-oral surface via the use of biofilms and it allows the species to grow and flourish[11]. Candida continues to grow in the mouth because they exist in mixed biofilms , where they can live together with bacteria and successfully live in those environments[12]. The cell wall structure if candida shows the properties that promote the formation of biofilms , thereby allowing it to infiltrate the tissue and ultimately inhabit various organs in the body, including the oral mucosa[14,15]. They have special mechanisms that allow them to switch back and forth from one form to another (from budding yeast from to pseudohyphal to hyphal forms. The formation of the candida biofilms creates serious clinical injurious effects because of their increased resistance to antifungal therapy . This property to adhere to the host cells of the epithelium is the virulence Factor of species of oral candidia. Additionally , it has been found that candida species and bacteria will communicate with each other by a mechanism called quorum-sensing[14,16]. The Candida albicans has two major quorumsensing molecules they are tyrosol and farnesol. Tyrosol stimulate the formation of hyphae in C.albicans and farnesol inhibits the shift from the yeast form to the hyphal form and regulates the structural morphology of the cell [16,17]. Quorum is a sensing phenomenon where the accrual of signalling molecules enables a single cell to sense the number of bacteria/fungi which are present.

Polymer Chain Reaction (PCR) Fingerprinting Method

Polymerase Chain Reaction (PCR) fingerprinting has been used to differentiate between various strains of C. Albicans and C. dubliniensis. This is a great problem because of the high degree of similarity in the phenotype between C. dubliniensis and C. Albicans [18]. Multiplex PCR has been used to identify various species, particularly C. Albicans, C. glabrata, and C. tropicalis[19].PCR has been successfully used to identify C. dubliniensis in gingival fluid of healthy individuals with periodontal disease [20]. Tests were

identified using conventional sampling methods as well as specific PCR assays. Results showed that C. dubliensis was isolated and identified in subgingival fluid of periodontal pockets of persons having healthy immune systems. Realtime PCR and lucigenin-based chemiluminescence have showed that Candida biofilms can exert resistance to many commonly employed antifungal in the clinical setup [21]. Real-time PCR chemistries allow for the detection of PCR amplification during the early phases; measuring the kinetics during the early phases provides an advantage over the traditional PCR where agarose gels are used for the detection of PCR amplification at the final endpoint phase of the reaction. C. Albicans genes have been identified that regulate the communication between oral epithelial cells in oropharyngeal candidiasis and vascular endothelial cells [22]. These genes increase their interaction as they come into contact with epithelial and endothelial cells and as they endure the stresses that they likely encounter as they grow in the oropharynx and in the blood. C. Albicans has a an assortment of pathogen-associated molecular patterns; recognizing this array of patterns on the cell wall of C. Albicans is mediated through many pattern-recognition receptors such as the Toll-like receptors [27]. These Toll-like receptors are a separate family of pattern-recognition receptors that help to recognize microbes and eventually activate phagocytosis and inflammatory responses. Various techniques have been used to identify Candida species. Multiplex PCR was used to quickly identify Candida species in samples of oral rinses without having to use an additional step of extraction in the process; ten species were identified in just a few hours [28]. Species that were identified included C. albicans, C. krusei, C. parapsilosis, C. famata, C. dubliniensis, C. kefyr, C. tropicalis, C. glabrata, C. guillermondii, and C. lusitaniae.

Immuno Flurosense Method

Use of recombinant antigens for the diagnosis and treatment of invasive candidiasis has been recently studied. The detection of antibodies directed against antigens which are expressed on the C. albicans germ tube surface by indirect immunofluorescence has been shown for diagnosis. Among the Candida enzymes detected in sera of patients who have systemic candidiasis, enolase has been found to be diagnostic. Advances in molecular

biological techniques have also allows completion of the Candida albicans genome sequence.These molecular biology techniques have allowed the production of recombinant antigens which are useful for the discovery and identification of antibodies against them. It has been shown that the detection of antibodies against purified and well-defined recombinant antigens allows for the diagnosis of Candidal organisms which are invasive in nature [23].

Gene Profiling Techniques

Host-fungus interactions were analyzed using a combination of microscopic, cellular and molecular techniques as well as an in vitro RHE (reconstituted human oral epithelium) model of oral candidiasis so to scrutinise the Candida infection process [24]. It was found from this research analysis that morphology and adaptive response of Candida organisms play important roles in the way the host and the fungus respond to one another if there is an infection. Candida has also been found by the study of structural mucosal and innate immune cells [25]. The activation of mechanisms which are protective in nature in both immune and non-immune effector cells depends on appropriate recognition of pathogens. Pattern-recognition receptors expressed on host cells allow for the rapid recognition of pathogens [26].

Immunological Methods

Denture stomatitis is a very real and common problem encountered within the dental setting; a significant emphasis has been placed in this review in both the immunology as well as in the treatment because of the practical implications that this topic carries for the general as well as specialty dental practitioner. In a denture-related stomatitis study, the main yeast strain isolated by far was C. Albicans. In the denture-related stomatitis group (versus the non-denture related stomatitis group), there were a greater variety of strains of Candida which were identified via microbiological sampling [29]. Candida mannan antigen detection was found to be a reliable as well as a sensitive method for the serologic diagnosis of systemic candidiasis [30]. The kit used in the study utilized an ELISA (Enzyme Linked Immunosorbent Assay) using polyclonal antibodies against Candida albicans mannan; the results revealed that the mannan assay had high positive

results for Candida in comparison to the standard of the culture medium. Denture stomatitis is a very real and common problem within the dental setting and a significant portion of this has been reviewed here with regards to immunologic aspects. It was noted in another investigation, that C. Albicans is the predominant fungal species isolated from those who wear dentures and that C. dubliniensis was isolated from the underlying mucosa but not from the prosthetic surface [31]. C. glabrata has been found to be an opportunistic pathogen in individuals who use long term immunosuppressants and broad-spectrum antibiotics-especially in the elderly [32]. This may be due to several factors, including decreased salivary gland hypofunction as well as pH changes noted in the elderly.

Microbial Culture

Identification of Candida species has been examined using CHROMagar[33]. CHROMagar can differentiate C. albicans as green colonies, C. krusei as pink colonies and C. tropicalis as blue colonies. Candida species were isolated and identified using this agar from HIV patients with oropharyngeal candidiasis. C. albicans and C. krusei were identified with a few strains of C. tropicalis. A few strains of C. tropicalis generated colonies with a trace of a greenish color making it complicated to differentiate it from C. albicans. In another identification and treatment analysis, fifty-five patients with complete upper dentures were examined before and after the use of a ten percent vinegar solution. Vinegar solution was used to disinfect their denture. Before the use of the solution, C. albicans, C. glabrata, C. krusei, C. lusitaniae, C. guillermondii, C. tropicalis, and C. parapsilosis were identified [34]. After the use of the vinegar solution, C. guillermondii, C. lusitaniae, and C. parapsilosis were not detected; all the other strains of Candida were still present. In a previous retrospective study of Candidal colonization and removable denture prostheses, forty-four patients exhibited various Candidal counts with respect to four groups of species: C. albicans, C. krusei, C. tropicalis, and unspecified Candidal species. The frequency of C. albicans was 87 percent with prostheses versus 90 percent without prosthesis; unspecified Candidal species was 58 percent versus 60 percent, C. krusei 14 percent with prosthesis versus 13 percent without prostheses, while the frequency of C. tropicalis was 6 percent for both.

Treatment of Oral Candidiasis

Various traditional modes of treatment have been used to manage oral candidiasis. Treatment can be accomplished with the use of appropriate antifungal medications and routine disinfection of removable intraoral prostheses [35]. Both topical and systemic medications have been used. Topical agents include Nystatin oral suspension and nystatin oral powder as well as clotrimazole troches. Systemic antifungals can be given by prescription and include itraconazole, ketoconazole, and fluconazole. Mycostatin® is a brand name for nystatin solution. Medications specifically for the treatment of angular cheilitis include nystatin ointment, miconazole cream (2%), and clotrimazole cream (1%). Disinfection of oral appliances can be accomplished via the use of Chlorhexidine and nystatin solution. (2% chlorhexidine gluconate, Peridex) [36]. Newer antifungals in the prophylaxis and maintenance of oral candidiasis therapy for invasive fungal infections include Posaconazole, Ravucanazole, and Echinocandins such as Caspofungin, Micafungin, and Anidulafungin[37]. They all have been found to be less toxic antifungal alternatives to Amphotericin B. Posaconazole was approved in 2006 for the treatment of invasive Aspergillosis and Candida infections. Ravucanazole is highly effective against a variety of fungi, including Candida pathogens and has been used to treat esophageal candidiasis in HIV (human immunodeficiency virus) patients. Caspofungin has inhibitory effects against various Aspergillosis and Candida species. Micafungin has been shown to be highly effective against esophageal candidiasis and has fewer side effects than Caspofungin. Anidulafungin is the newest echinocandin and appears to be very effective against many Candida species in vitro. Anidulafungin was found also to be more efficacious in one study than the traditional form of systemic fluconazole for the treatment of invasive candidiasis [38]. Recent developments in the treatment of oral candidiasis include the use of probiotic bacteria which are known to reduce the growth of pathogenic microbes [39]. A recent study used cheese containing a mixture of probiotics such as Lactobacillus rhamnosus GG, Lactobacillus rhamnosus LC705, Propionibacterium freudenreichii and shermanii JS. The use of probiotic cheese revealed

the reduction of the prevalence of oral Candida in the elderly and its use also allowed for the reduction of the prevalence of hyposalivation.

Another group of elderly individuals were evaluated for possible reduction of Candida overgrowth with the use of an amine fluoride-stannous fluoride mouth rinse and toothpaste combination. The number of elderly with high Candida albicans counts decreased after use of the combination. Other Candida counts (non-albicans) were also low in the participants of the study. In another novel application, fluconazolemucoadhesive buccal films were prepared and placed in the buccal mucosa where the drug was released over a five hour period and ultimately the disc eroded away. This topical method was used as another alternative to the traditional form of systemic fluconazole and seemed to reduce side effects of the medication that may have been encountered in the systemic form. There has been current acceptance by the FDA for the miconazole Lauriad in a mucobuccal tablet form to treat oropharyngeal candidiasis in a once-a-day dosing format [40]. In a different treatment of denture stomatitis, dentureliner containing nystatin was placed for a period of one week. This was compared to a denture-liner filled with nystatin and left on the denture surface for four days. Dentures left with nystatin containing denture liner for four days had higher yeast counts than those that were left in place for one week. Salivary yeast counts were much lower for individuals who had a denture liner that contained nystatin as the antifungal than individuals who did not use a denture liner containing an antifungal. These and other recent advances have allowed the practitioner to treat various forms of Candida species in an effective manner in the clinical setting.

Conclusion

Oral candidiasis is a fungal infection caused mostly by the species candida albicans along with the other causative agents like candida tropicalis, candida glabarata etc. The treatment involves the antifungal drugs like nystatin, miconazole and amphotericin B.

The recent advances that help in the diagnosis and treatment of oral candidiasis are cellular and molecular mechanisms, polymerase chain reaction, recombinant DNA, reconstituted human oral epithelium, immunological methods which allows the practitioner to treat various candida species in an effective manner in the clinical setting.

Corresponding Author

Geo Mani,

Senior Lecturer,Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha University,Saveetha Institute of Medical and Technical Sciences,

162 Poonamallee High Road, Chennai -600 077,

Tamil Nadu, India.

Email Id: geosdch@gmail.com

Telephone Number: 7358433246

Total Number of Words:3542

Reference

1. Lew MA. Diagnosis of systemic Candida infections. Annual review of medicine. 1989 Feb;40(1):87-97.

2. Coco BJ, Bagg J, Cross LJ, Jose A, Cross J, Ramage G. Mixed Candida albicans and Candida glabrata populations associated with the pathogenesis of denture stomatitis. Molecular Oral Microbiology. 2008 Oct 1;23(5):377-83.

3. Samaranayake YH, Cheung BP, Parahitiyawa N, Seneviratne CJ, Yau JY, Yeung KW, Samaranayake LP. Synergistic activity of lysozyme and antifungal agents against Candida albicans biofilms on denture acrylic surfaces. Archives of oral biology. 2009 Feb 1;54(2):115-26.

4. Lynch DP. Oral candidiasis: history, classification, and clinical presentation. Oral surgery, oral medicine, oral pathology. 1994 Aug 1;78(2):189-93.

5.Méan M, Marchetti O, Calandra T. Bench-to-bedside review: Candida infections in the intensive care unit. Critical Care. 2008 Feb;12(1):204.

6. Thaweboon S, Thaweboon B, Srithavaj T, Choonharuangdej S. Oral colonization of Candida species in patients receiving radiotherapy in the head and neck area. Quintessence International. 2008 Feb 1;39(2).

7.Soysa NS, Samaranayake LP, Ellepola AN. Antimicrobials as a contributory factor in oral candidosis–a brief overview. Oral diseases. 2008 Mar 1;14(2):138-43.

8. Davenport JC. The oral distribution of candida in denture stomatitis. Brit. dent. J.. 1970;129(4):151-6.

9.Samaranayake LP, Keung Leung W, Jin L. Oral mucosal fungal infections. Periodontology 2000. 2009 Jan 1;49(1):39-59.

10.Dahlen G, Blomquist S, Carlen A. A retrospective study on the microbiology in patients with oral complaints and oral mucosal lesions. Oral diseases. 2009 May 1;15(4):265-72.

11. Abraham CM, Haghighat N, Beach MM, Al-Hashimi I. Candidalcolonization in the presence and absence of removable dental prostheses. Texas dental journal. 2007 Jan;124(1):94-101.

12. Zunt SL. Oral candidiasis: Diagnosis and treatment. The Journal of Practical Hygiene. 2000 Sep;9(5):31-6.

13. DeLong L, Burkhart N. General and oral pathology for the dental hygienist. Lippincott Williams & Wilkins; 2013 Feb 25.

14. Ten Cate JM, Klis FM, Pereira-Cenci T, Crielaard W, De Groot PW. Molecular and cellular mechanisms that lead to Candida biofilm formation. Journal of dental research. 2009 Feb;88(2):105-15.

15. Mukherjee PK, Zhou G, Munyon R, Ghannoum MA. Candida biofilm: a well-designed protected environment. Medical mycology. 2005 May 1;43(3):191-208.

16. Hornby JM, Jensen EC, Lisec AD, Tasto JJ, Jahnke B, Shoemaker R, Dussault P, Nickerson KW. Quorum sensing in the dimorphic fungusCandida albicans is mediated by farnesol. Applied and environmental microbiology. 2001 Jul 1;67(7):2982-92.

17. Chen H, Fujita M, Feng Q, Clardy J, Fink GR. Tyrosol is a quorum-sensing molecule in Candida albicans. Proceedings of the National academy of Sciences of the United States of America. 2004 Apr 6;101(14):5048-52.

18.Neppelenbroek KH, Campanha NH, Spolidório DM, Spolidorio LC, Seó RS, Pavarina AC. Molecular fingerprinting methods for the discrimination between C. albicans and C. dubliniensis. Oral Diseases. 2006 May 1;12(3):242-53.

19. Liguori G, Di Onofrio V, Lucariello A, Gallé F, Signoriello G, Colella G, D'amora M, Rossano F. Oral candidiasis: a comparison between conventional methods and

multiplex polymerase chain reaction for species identification. Molecular Oral Microbiology. 2009 Feb 1;24(1):76-8.

20.Jewtuchowicz VM, Mujica MT, Brusca MI, Sordelli N, Malzone MC, Pola SJ, Iovannitti CA, Rosa AC. Phenotypic and genotypic identification of Candida dubliniensis from subgingival sites in immunocompetent subjects in Argentina. Molecular Oral Microbiology. 2008 Dec 1;23(6):505-9.

21.Seneviratne CJ, Wang Y, Jin L, Abiko Y, Samaranayake LP. Candida albicans biofilm formation is associated with increased anti-oxidative capacities. Proteomics. 2008 Jul 1;8(14):2936-47.

22. Park H, Liu Y, Solis N, Spotkov J, Hamaker J, Blankenship JR, Yeaman MR, Mitchell AP, Liu H, Filler SG. Transcriptional responses of Candida albicans to epithelial and endothelial cells. Eukaryotic cell. 2009 Oct 1;8(10):1498-510.

23. Abraham CM. Advances and emerging techniques in the identification, diagnosis and treatment of oral candidiasis. Open Path J. 2011;5:8-12.

24.Zakikhany K, Naglik JR, Schmidt-Westhausen A, Holland G, Schaller M, Hube B. In vivo transcript profiling of Candida albicans identifies a gene essential for interepithelial dissemination. Cellular microbiology. 2007 Dec 1;9(12):2938-54.

25. Geetha RV, Anitha RO. In vitro evaluation of anti mycotic activity of ethanolic extract of Glycyrrhizaglabra. Asian J Pharm Clin Res. 2013;6(3):205-6.
26. Mukhopadhyay S, Herre J, Brown GD, Gordon S. The potential for Toll-like receptors to collaborate with other innate immune receptors. Immunology. 2004 Aug 1;112(4):521-30.

27. Liguori G, Lucariello A, Colella G, De Luca A, Marinelli P. Rapid identification of Candida species in oral rinse solutions by PCR. Journal of clinical pathology. 2007 Sep 1;60(9):1035-9.

28. Figueiral MH, Azul A, Pinto E, Fonseca PA, Branco FM, Scully C. Denture-related stomatitis: identification of aetiological and predisposing factors–a large cohort. Journal of oral rehabilitation. 2007 Jun 1;34(6):448-55.

29.Kurita H, Kamata T, Zhao C, Narikawa JN, Koike T, Kurashina K. Usefulness of a commercial enzyme-linked immunosorbent assay kit for Candida mannan antigen for detecting Candida in oral rinse solutions. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics. 2009 Apr 1;107(4):531-4.

30. Marcos-Arias C, Vicente JL, Sahand IH, Eguia A, De-Juan A, Madariaga L, Aguirre JM, Eraso E, Quindós G. Isolation of Candida dubliniensis in denture stomatitis. Archives of oral biology. 2009 Feb 1;54(2):127-31.

31. Li L, Redding S, Dongari-Bagtzoglou A. Candida glabrata, an emerging oral opportunistic pathogen. Journal of dental research. 2007 Mar;86(3):204-15.

32.Sivakumar VG, Shankar P, Nalina K, Menon T. Use of CHROMagar in the differentiation of common species of Candida. Mycopathologia. 2009 Jan 1;167(1):47-9.

33. Pinto TM, Neves AC, Leão MV, Jorge AO. Vinegar as an antimicrobial agent for control of Candida spp. in complete denture wearers. Journal of Applied Oral Science. 2008 Dec;16(6):385-90.

34. Budtz-Jörgensen E, LöE HA. Chlorhexidine as a denture disinfectant in the treatment of denture stomatitis. European Journal of Oral Sciences. 1972 Dec 1;80(6):457-64.

35.Petrikkos G, Skiada A. Recent advances in antifungal chemotherapy. International journal of antimicrobial agents. 2007 Aug 1;30(2):108-17.

36.Hatakka K, Ahola AJ, Yli-Knuuttila H, Richardson M, Poussa T, Meurman JH, Korpela R. Probiotics reduce the prevalence of oral Candida in the elderly—a randomized controlled trial. Journal of dental research. 2007 Feb;86(2):125-30.

37.Meurman JH, Pärnänen P, Kari K, Samaranayake L. Effect of amine fluoride–stannous fluoride preparations on oral yeasts in the elderly: a randomised placebo-controlled trial. Gerodontology. 2009 Sep 1;26(3):202-9.

38.Yehia SA, El-Gazayerly ON, Basalious EB. Fluconazolemucoadhesive buccal films: in vitro/in vivo performance. Curr Drug Deliv 2009; 6(1): 17-27.

39.Dheepika B, Maheswari U. Aloe vera in oral diseases-A review. Int J PharmPharm Sci. 2014;6(2):64.

40. Geerts GA, Stuhlinger ME, Basson NJ. Effect of an antifungal denture liner on the saliva yeast count in patients with denture stomatitis: a pilot study. Journal of oral rehabilitation. 2008 Sep 1;35(9):664-9.

Endodontic Management of Various Developmental Anomalies of Tooth-A Review

Preethi Pavithra.R [1], Gheena Ranjith [2], Asha Ramesh [3]

Graduate student [1], Senior Lecturer [2],Department of Oral Pathology

Senior Lecturer [3],Department of Periodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Tooth development or odontogenesis is the complex process by which teeth form from embryoniccells, grow, and erupt into the mouth. Although many diverse species have teeth, non-human tooth development is largely the same as in humans. For human teeth to have a healthy oral environment, enamel, dentin, cementum, and the periodontium must all develop during appropriate stages of fetal development. If the teeth do not start to develop at or near these times, they will not develop at all, resulting in hypodontia or anodontia. These anomalies not only affect the esthetic appearance of teeth but also pose difficulties during dental treatment and sometimes are the cause of dental problems Dental anomalies are clinically evident abnormalities. They may be the cause of various dental problems.Careful observation and appropriate investigations are required to diagnose the condition and institute treatment.In this review, we describe the various developmental anomalies of tooth based on size,shape, number, and structure.

KeywordsOdontogenesis; Developmental anomaly; Esthetics; Dental management

Introduction

Developmental dental anomalies are prominent deviations in the form of normalcolor, contour, size, number, and degree of development of teeth. Local as well as systemic factors may be responsible for these developmental disturbances.The deciduous teeth begin their formation between the sixth and eighth week of prenatal development, and permanent teeth form around the twentieth week[1] Such influences may begin before or after birth, hence deciduous or permanent teeth may be affected. Aberrations in the normal number of teeth include supernumerary teeth (hyperdontia), i.e. excess teeth or hypodontia (teeth missing from the normal compliment) while oligodontia is a developmental absence of six or more teeth excluding the 3rd molars.[2]

Anomalies of shape of teeth include microdontia and macrodontia. Microdontia refers to teeth that are physically smaller in size than usual and macrodontia in turn refers to teeth that are physically larger in size than normal. Anomalies of shape include dens invaginatus (DI), talon cusp, dens evaginatus, gemination, fusion, root dilacerations, taurodontism, and concrescence.It is an anomaly resulting from invagination in the surface of a tooth crown or rarely the root and which is lined by enamel and dentin[3] Dens evaginatus is a focal area of the crown, projecting outward and giving rise to a horn-like protuberance on the affected surface that appears as an extra cusp [4] Talon cusp is an accessory cusp usually located on the lingual surface and rarely on the facial surface of permanent or deciduous incisors. It arises from the cingulum area, or cemento-enamel junction (CEJ) of maxillary or mandibular anterior teeth, in both the primary and permanent dentition [5].Fusion and gemination have been referred to as double teeth which appear as larger than normal sized teeth. Gemination is defined as a single enlarged tooth or joined (double) tooth in which the tooth count is normal when the anomalous tooth is counted as one [6].Fusion is defined as a single enlarged tooth or joined (double) tooth in which the tooth count reveals a missing tooth when the anomalous tooth is counted as one [7].

Taurodontism is a developmental anomaly that usually affects the molars in which the body of the affected teeth is very large and the associated roots are shortened, with bifurcation near the apex.Concrescence may be the Uni for two contiguous teeth Toward cementum. Dilaceration will be a abnormal twist in the root alternately crown of a tooth rot. The twist is that's only the tip

of the iceberg incessant in the root Be that might be exhibit anyplace along those length of the tooth root[8].

These anomalies not only affect the esthetic appearance of teeth but also pose difficulties during dental treatment and sometimes are the cause of dental problems. The present study was performed to evaluate the frequency of occurrence of dental anomalies of size, number, and shape in the adult population and their implications in the treatment of such conditons[9].Thongudomporn and Freer (1998) reported that 74.77% of their subjects had at least one anomaly. The most prevalent anomaly was invagination, while supernumerary teeth and root dilacerations were the least frequent anomalies.Early endodontic management of tooth can preserve the tooth.

Anomalies of Teeth

They are various types of anomalies based on the size and shape of teeth.

Hyperdontia and it's Management

Various reports of hyperdontia are mostly in children but it is increasingly common in patients with cleft lip and cleft palate. Salcido-garcía et al have reported a higher prevalence in males than in females.Among the various supernumerary teeth, mesiodens are the most commonly found followed by 4^{th} molars, premolars and maxillary lateral incisors [10]There was a higher occurrence of paramolars followed by mesiodens and supernumerary teeth in the anterior region. Impacted supernumerary teeth had equal occurrence in maxilla and mandible and occurred in the anterior rather than the posterior region.It can be managed orthodontically by extraction and root canal treatment of additional teeth.

Hypodontia and its Management

This occurs in permanent dentition in 0.3% of the population.Congenitally missing teeth most commonly occur with maxillary laterals, 2^{nd} premolars and mandibular central incisors and can be unilateral or bilateral[11]. Radiographs confirm the absence of missing teeth. The etiology is believed to be hereditary or developmental. In another study, mandibular central incisors

dominated the list compared to premolars and it was noted more in females[12]. Missing third molars were not considered in the study sample. Treatment generally requires a multidisciplinary approach including orthodontic correction, or prosthetic replacement with a removable or fixed appliance ,endodontic correction is not much needed Age of the patient, number of missing teeth, carious teeth, and condition of supporting tissues, occlusion and interocclusal space are the important factors determining treatment planning.[13]

Microdontia and its Management

Microdontia is a condition in which teeth appear smaller than normal. In the generalized form, all teeth are involved. In the localized form, only a few teeth are involved[14]The most common teeth affected are the upper lateral incisors and third molars. The affected teeth may be of normal or abnormal morphology.There are 3 types of microdontiaA. True generalized microdontia, B.Relative generalized microdontia, and C. Microdontia involving a single tooth. Teeth commonly affected are maxillary lateral incisors and third molars[15]. When lateral incisors are affected, there is a reduction in mesiodistal diameter and convergence towards the incisal edge and this is referred to as peg shaped incisors. The condition is largely of genetic origin. Treatment mainly requires root canal treatment followed by prosthodontic intervention as the teeth are smaller in size they are useful in preparing crowns and using the teeth as abutments.[16]

Macrodontia and its Management

Typically, only a few teeth are larger than normal. Macrodontia is a very rare anomaly mostly involving the second premolar and it is believed to be a variation of obscure genetic origin .Diffuse true macrodontia is observed in pituitary gigantism and pineal hyperplasia. It has also been reported that this condition can be associated with short stature, mental retardation, minor skeletal anomalies and craniofacial dysmorphism[17].Teeth with macrodontia can undergo conservative esthetic reconstruction using a minimally invasive technique. In the case presented, the correct diagnosis and detailed planning, followed by the use of composite resin, allowed a satisfactory result, with the recovery of the self-esteem of a young patient.[18]

Dens Invaginatus and its endodontic Management

This presents clinically as pit or fissure on the lingual surfaces of anterior teeth. The classical radiographic appearance of coronal DI is as a pear-shaped invagination of enamel and dentine with a narrow constriction at the opening on the surface of the tooth [19]. The infolding of the enamel lining is more radiopaque than the surrounding tooth structure aiding easy identification. The crown is almost always malformed if the coronal invagination is extensive

Oehlers et al grouped coronal DI into three types according to radiographic appearance:

1. Enamel lined invaginations confined to the crown of the tooth;
2. Extending towards the root but not crossing the cemento-enamel junction (CEJ);
3. Penetrating the surface of the root and 'bursting' apically or laterally to produce a second foramen in the root.[20].

The majority of cases are located in maxilla and in lateral incisors. Following the maxillary lateral incisors, in decreasing order, the central incisors, premolars, canines and molars are generally affected. DI involving mandibular teeth is rare[21]. There was a higher prevalence in male patients. The possibility of the pulp being affected without a clinically detectable lesion as a result of tortuous lingual anatomy makes DI clinically significant since the enamel is thin and close to the pulp, and so there can be easy involvement[22].Pulp sensitivity testing has to be performed if radiographically apical periodontium is unremarkable in clinically suspicious teeth. If the tooth is vital then it should be restored to prevent access of the DI to the oral environment. Steffen et al have stated that if no entrance to the invagination can be detected and there are no signs of pulp pathology, no treatment is required other than fissure sealing or a minimally invasive filling. Awareness of this anomaly when present is essential especially in a case of pulpitis in the absence of any history of trauma or clinical evidence of caries or restorations [23]. DI with pulpal involvement is usually managed by conventional endodontic treatment or special endodontic techniques capable of inducing an apexification if the condition necessitates [24]. Surgical treatment should be considered in cases of endodontic failure and in teeth which cannot be treated non-surgically due to anatomical problems or failure to gain access to all parts of the root canal[25].

Talon Cusp and its Endodontic Management

Talon cusp is composed of normal enamel and dentin containing varying extensions of pulp tissue. It may connect with the incisal edge to produce a T-form or, if more cervical, a Y-shaped crown contour. It is also known by other names such as interstitial cusp, tuberculated premolar, evaginatedodontoma, occlusal enamel pearl, occlusal anomalous tubercle or supernumerary cusp [26]

Hattabetalclassified anomalous cusps into three types based on the degree of cusp formation and extension:

1. Talon: A morphologically well-delineated additional cusp that prominently projects from the palatal surface of a primary or permanent anterior tooth and extends at least half the distance from the CEJ to the incisal edge.
2. Semi talon: An additional cusp of 1 mm or more, but extending less than half the distance from the CEJ to the incisal edge. It may blend with the palatal surface or stand away from the rest of the crown.
3. Trace talon: An enlarged or prominent cingula in any of its variants (i.e. conical, bifid or tubercle-like) originating from the cervical third of the root.[27]

Its prevalence ranges from less than 1% to 8% of the with a higher frequency in males than females. The anomaly has a greater predilection for the maxilla, and maxillary lateral incisors are commonly affected in the permanent dentition followed by central incisors and canines. In the present study, this anomaly accounted for 4.28% of dental anomalies and the semi-talon form was observed.[28]

Juan et al have emphasized that early diagnosis and management of talon cusp is important to prevent complications such as occlusal interference, compromised esthetics, caries and periapical pathologies, and periodontal problems. Prevention of accidental cusp fracture and attrition has also been stressed.[29]

The treatment of talon cusp involves careful clinical judgment and review of whether the cusp contains or is devoid of a pulp horn.[30] Earlier reports, based on radio graphic examination,

stated that removal of the cusp would inevitably lead to pulp exposure that would require endodontic treatment.However, radiographic tracing of the pulpal configuration inside the talon cusp has inherent difficulties because the cusp is superimposed over the affected tooth crown. Similarly, histological examination of extracted talon teeth failed to show the presence of a pulp horn in the talon cusp. [31]

Dens Evaginatus and it Endodontic Management

This is a relatively rare dental anomaly and primarily affects the premolars but can also occur on molars, canines, and incisors [32].Dens evaginatus is a condition affecting predominantly mandibular premolars and leading frequently to pulp necrosis because of occlusal trauma. When the tooth was nonvital at the time of diagnosis, apexification procedures using calcium hydroxide were successful some cases. Diagnosis of the condition before the tooth reached full occlusion (six teeth) allowed conservative management with preservation of pulp vitality, by prevention of traumatic occlusion. The dens evaginatus was ground down in small increments to avoid pulp exposure and promote deposition of secondary dentin[33].The early diagnosis of dens evaginatus before occlusal trauma can occur should permit protection of the pulp and preservation of tooth vitality.[34].

Taurodontism and its Treatment

The pulp chamber will be widened apico-occlusally and thus, the furcations are positioned added apically than normal with bull like tooth appereance. The prevalence of taurodontism is variable, depending on the altered alternation and groups studied. It is lower than 1 in modem man. Several research accept appear in which taurodontism appears as a allotment of assorted syndromes. Ackerman and assembly believed that tooth basis analysis is primarily bent genetically but that it may be environmentally modified. In both the patients discussed above, systemic disturbances or malformations could not be articular and appropriately advised to be of non syndromec taurodontism.[35]

The teeth most frequently affected are the molars, although it can be occasionally seen in premolar and incisors and are mostly diagnosed by radiographic study. Endodontic treatment in

taurodont teeth has been described as complex and difficult.Durr et al suggested that the morphology could hamper the location of the orifices, thus creating difficulty in instrumentation and obturation[36]. The number of root canals varied in each case. Since the pulpal chamber was huge in both the cases, mechanical debridement and shaping was achieved with circumferential filing using "K" files of standard ISO size and taper. Sodium hypochlorite irrigation was limited to the initial use as the apical foramina were wide open and as a precautionary measure to avoid a hypochlorite accident. In the first case custom made gutta-percha (roll-cone technique) was used, as the main pulp chamber was huge and large wide apical foramina were present. This technique allows good apical control and adapts well to the canal configuration. Thermo plasticized gutta-percha was used in the second case as distinct apical stops were present and not like the previous case where the use of it could have caused an apical extrusion.

The remaining dentin thickness of these roots is less, leading to chances of root fracture, which is very high in such cases. Hence it was decided to reinforce the root canal walls, by using light curing composite resin. Intra radicular rehabilitation with light cured composite resin using clear light transmitting post was therefore used.[37]Layered adhesion technique was used as it allows incremental composite build up inside the root canal. Aco responding size fibre reinforced resin post was used in the post space left by the light transmitting post and a monoblock effect was created which has the advantage of good stress distribution.

Fusion and Gemination and its Management

These anomalies are also referred to as double teeth, formed as result of total or partial union in dentin and possibly their pulps. They are known to occur in both deciduous and permanent dentitions.Fusion may be partial or complete and may present with two independent root canals or less often, a single root and one or two pulp chambers. As a result, the tooth may be of normal size or larger than normal. Fusion of central incisors and canines is more frequent than that of lateral incisors and canines. These anomalies are also referred to as double teeth, formed as result of total or partial union in dentin and possibly their pulps. They are known to occur in both deciduous and permanent dentitions.Fusion may be partial or complete and may present with two independent root canals or less often, a single root and one or two pulp chambers. As a result, the

tooth may be of normal size or larger than normal. Fusion of central incisors and canines is more frequent than that of lateral incisors and canines.[38]

Gemination is an incomplete division of one tooth germ, resulting in the formation of two partially or completely separated crowns formed on a single root. It is more frequent in the anterior teeth, but can also affect molars and bicuspids. It has a prevalence of 0.5% and 0.1% in deciduous and permanent dentitions, respectively. In this review, the evidence collated that fusion accounted for 4.85%, and gemination constituted 0.28% (only one patient) of all of the dental anomalies. Fusion was observed to occur unilaterally in accordance with other studies. Mandibular teeth were affected more than maxillary. Fusion can be suspected when the number of teeth in the arch is found to be reduced and/or two roots are seen radiographically.

Twinning will appear similar clinically and are larger than normal teeth, but by definition fusion must involve dentin. Gemination can differentiated from fusion by the presence of a full compliment of teeth and an incompletely divided tooth.Twinning of teeth may adversely affect esthetics, and may lead to dental crowding and difficulty in eruption of adjacent teeth. Treatment consists of managing asymmetry, either by extirpation of the unwanted dental portion in conjunction with root canal therapy, or restoration of the exposed area. Orthodontic intervention completes the treatment plan[38].

Root- canal treatment of fused or geminated teeth has been described by various authors (Tagger 1975, Libfeld 1986, Wong 1991). A literature review failed to reveal treatment of a trifid crown permanent maxillary first premolar. Although more than one separate root- canal lends support to the diagnosis of fusion, the diagnosis of gemination was based on the clinical examination and finding that three of the canals joined to form a common main canal. The formation of this tooth within the normal complement of teeth and the similar mirror- image effect of the three crowns supports this diagnosis. Gemination occurs because of a partial division of a single tooth bud through invagination, resulting in completely or incompletely separated crowns (Pindborg 1970). In this case, the aetiology of this abnormal tooth development is unknown. Consultation with the family members and the referring dentist revealed no hereditary or causal link.

The tooth exhibited percussion and thermal sensitivity in addition to pain on palpation, symptoms indicative of an irreversibly inflamed pulp with extension of inflammatory changes to the periapex. As the tooth was caries- free and no history of trauma was reported, it is likely that the unusual anatomy lead to the irreversible condition of the tooth and the patients symptoms. Though not detected clinically, enamel deficiencies may be present in the developmental grooves between the three crowns. This would allow bacteria to gain access to the pulp chamber in a similar manner to that which occurs in dens invaginatus.

Management of the case required careful consideration of the possible morphological aberrations. Clinical and radiographic evaluation indicated that access into each of the crowns was necessary. The complexities of the root- canal systems could then be addressed during treatment. This case illustrates that, despite the unusual morphology and the lack of a comparative case in the literature, the use of sound endodontic principles resulted in successful treatment.[38]

Accessory Roots and their Management

These are commonly known to occur in mandibular canines, premolars and molars (often in 3rd molars). There are no reported studies on the prevalence or occurrence of accessory roots in different populations except for individual case reports. Accessory roots were noted in mandibular premolars and 1st molars with higher numbers in males and comprising 2% of the total anomalies [39]. This abnormality is a definite hindrance for successful endodontic treatment leading to perforations and also poses difficulty in extractions. The radiographic signs of accessory roots include double periodontal spaces on one side of the root, periodontal space crossing roots and abrupt diminution of the root canal spaces. From a clinical standpoint, radiographs or other imaging resources provide clinicians with the most appropriate method to detect variations in both root and canal anatomy. However, only by correct clinical examination and interpretation of these images can the clinician detect variations and be aware of them before and during endodontic procedures.

Dilacerations and its Endodontic Management

These are thought to arise secondary to trauma during tooth formation, altering the angle between the tooth germ and the portion of the tooth already developed.Occasionally, the bend is created by pressure from adjacent cysts, tumors or odontogenic hamartoma. Frequently, the affected teeth are the maxillary incisors followed by the mandible anteriors. In the present study, dilacerations of the roots comprised 22.5% of the anomalie.A frequent error that may occur during endodontic procedure in dilacerated root is the failure to maintain root canal curvature, resulting in ledge formation, apical transportation, zipping, perforation.To avoid these mishaps, the basic principles of endodontic therapy must be followed, that is good preoperative radiograph, straight line access to apical foramen, pre-curving the endodontic hand instrument, recapitulation, thorough irrigation and use of flexible to reach the apical foramen; enough tooth structure was removed to get the straight line access to apical foramen, giving the access cavity a cloverleaf appearance. This modifed outline form of access cavity is known as shamrock preparation which allows the endodontic instrument unrestrained in the severely curved canals. In dilacerated teeth it is often diffcult to explore and negotiate the canal to its apical foramen; hence the use of scout fle is recommended in these kind of cases.[40]

Conclusion

The present reeview attempts to evaluate the frequency of occurrence of various developmental dental anomalies in a non-syndromic global population. It was noted that anomalies were more frequent in male patients than in females. Hyperdontia, root dilaceration, peg shaped laterals (microdontia), and hypodontia were more frequent compared to other anomalies of size and shape.Developmental anomalies of teeth are clinically evident abnormalities. They may be the cause of various dental problems. Careful observation and appropriate investigations are required to diagnose the condition and institute appropriate treatment.

Corresponding author

Asha Ramesh

Senior Lecturer,Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University

Chennai-600077

Mobile: +91 9841722113.

Email: doc.ash4587@gmail.com.

References

1. Guttal KS, Naikmasur VG, Bhargava P, Bathi RJ. Frequency of developmental dental anomalies in the Indian population. European journal of dentistry. 2010 Jul;4(3):263.
2. Neville BW, Damm DD, White DK, Waldron CA, Ferraro JW. Color Atlas of Clinical Oral Pathology. Plastic and Reconstructive Surgery. 1991 Aug 1;88(2):369.
3. Vasudev SK, Goel BR. Endodontic management of dens evaginatus of maxillary central incisors: a rare case report. Journal of endodontics. 2005 Jan 1;31(1):67-70.
4. Segura JJ, Jiménez-Rubio A. Talon cusp affecting permanent maxillary lateral incisors in 2 family members. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics. 1999 Jul 1;88(1):90-2.
5. Salcido-García JF, Ledesma-Montes C, Hernández-Flores F, Pérez D, Garcés-Ortíz M. Frecuencia de dientessupernumerarios en unapoblación Mexicana. Medicina Oral, Patología Oral y Cirugía Bucal (Ed. impresa). 2004 Dec;9(5):403-9.
6. Lisson JA, Scholtes S. Investigation of craniofacial morphology in patients with hypo-and oligodontia. Journal of Orofacial Orthopedics/Fortschritte der Kieferorthopädie. 2005 May 21;66(3):197-207.
7. Akkaya N, Kiremitçi A, Kansu O. Treatment of a patient with oligodontia: A case report. J Contemp Dent Pract. 2008 Mar 1;9(3):121-7.

8. Hersh JH, Ganzel TM, Fellows RA. Michel's anomaly, type I microtia and microdontia. Ear, nose, & throat journal. 1991 Mar;70(3):155-7.
9. Groper JN. Macrodontia of a single tooth: review of literature and report of case. The Journal of the American Dental Association. 1987 Jan 1;114(1):69.
10. Fryns JP, Haspeslagh M. Mental retardation, short stature, minor skeletal anomalies, craniofacial dysmorphism and macrodontia in two sisters and their mother. Clinical genetics. 1984 Jul 1;26(1):69-72.
11. Mupparapu M, Singer SR. A rare presentation of dens invaginatus in a mandibular lateral incisor occurring concurrently with bilateral maxillary dens invaginatus: case report and review of literature. Australian dental journal. 2004 Jun 1;49(2):90-3.
12. Steffen H, Splieth C. Conventional treatment of dens invaginatus in maxillary lateral incisor with sinus tract: one year follow-up. Journal of Endodontics. 2005 Feb 28;31(2):130-3.
13. Mellor JK, Ripa LW. Talon cusp: a clinically significant anomaly. Oral Surgery, Oral Medicine, Oral Pathology. 1970 Feb 1;29(2):225-8.
14. Hattab FN, Yassin OM, Al-Nimri KS. Talon cusp-clinical significance and management. Quintessence international-english edition. 1995 Feb 1;26:115.
15. Shifman A, Chanannel I. Prevalence of taurodontism found in radiographic dental examination of 1,200 young adult Israeli patients. Community dentistry and oral epidemiology. 1978 Aug 1;6(4):200-3.
16. Jafarzadeh H, Azarpazhooh A, Mayhall JT. Taurodontism: a review of the condition and endodontic treatment challenges. International endodontic journal. 2008 May 1;41(5):375-88.
17. Duncan WK, Helpin ML. Bilateral fusion and gemination: a literature analysis and case report. Oral Surgery, Oral Medicine, Oral Pathology. 1987 Jul 1;64(1):82-7.
18. Tannenbaum KA, Alling EE. Anomalous tooth development: case reports of gemination and twinning. Oral Surgery, Oral Medicine, Oral Pathology. 1963 Jul 1;16(7):883-7.
19. Ahmed HM, Abbott PV. Accessory roots in maxillary molar teeth: a review and endodontic considerations. Australian dental journal. 2012 Jun 1;57(2):123-31.

20. Chohayeb AA. Dilaceration of permanent upper lateral incisors: frequency, direction, and endodontic treatment implications. Oral Surgery, Oral Medicine, Oral Pathology. 1983 May 1;55(5):519-20.
21. Ando S, Oshima S, Nakamura Y, Sato A, Suzuki Y. Dilaceration in the root formation of the upper second premolar. The Journal of Nihon University School of Dentistry. 1968;10(3):91-4.
22. Arte S, Nieminen P, Apajalahti S, Haavikko K, Thesleff I, Pirinen S. (2001) Characteristics of incisor- premolar hypodontia in families. J Dent Res. 80(5): 1445-1450.
23. Backman B, Wahlin YB. Variations in number and morphology of permanent teeth in 7-year-old Swedish children. Int J Paediatr Dent. 2001; 11(1): 11-17.
24. Bruce C, Manning-Cox G, Stanback-Fryer C, Banks K, Gilliam MA. Radiographic survey of dental anomalies in Black pediatric patients. NDA J. 1994; 45(1): 6-13.
25. Cholitgul W, Drummond BK. Jaw and tooth abnormali- ties detected on panoramic radiographs in New Zealand children aged 10-15 years. N Z Dent J. 2000; 96(423): 10-13.
26. Constant DA, Grine FE. A review of taurodontism with new data on indigenous southern African populations. Arch Oral Biol. 2001; 46(11): 1021-1029.
27. Darwazeh AM, Hamasha AA, Pillai K. Prevalence of taurodontism in Jordanian dental patients. Dentomaxillofac Radiol. 1998; 27(3): 163-165.
28. Gabris K, Tarjan I, Fabian G, Kaa M, Szakaly T, Orosz M. Frequency of supernumerary teeth and possibilities of treatment. Fogorv SZ.2001; 94(2): 53-57.
29. Hamasha AA, AL-Khateeb T. Prevalence of fased and geminated teeth in Jordanian adults. Quintessence Int. 2004; 35(7): 556-559.
30. Hamasha AA, Al-Khateeb T, Darwazeh A. Prevalence of dilaceration in Jordanian adults. Int Endod J. 2002; 35(11): 910-912.
31. Hamasha AA, Alomari QD. Prevalence of dens invagi- natus in Jordanian adults. Int Endod J. 2004; 37(5): 307-310.
32. Laatikainen T, Ranta R. Taurodontism in twins with cleft lip and/or palate. Eur J Oral Sci. 1996; 104(2): 82-86.

33. Leifert S, Jonas IF. Dental Anomalies as a microsymptom of palatal canine displacement. J Orofac Orthop. 2003; 64(2): 108-120.
34. Liu PY, Sew WK. A controlled study of the association of various dental anomalies with hypodontia of permanent teeth. Pediatr Dent. 1995; 11(4): 291-296.
35. Mavrodisz K, Budai M, Tarjan I. Prevalence of talon cusp in patients aged 7-18. Forgorv SZ. 2003; 96(6): 257-259.
36. Ooshima T, Ishida R, Mishima K, Sobue S. The prevalence of developmental anomalies of teeth and their association with tooth size in the primary and permanent dentitions of 1650 Japanese children. Int J Paediatr Dent. 1996; 6(2): 87-94.
37. Sarr M, Toure B, Kane AW, Fall F, Wone MM. Taur- odontism and the pyramidal tooth at the level of the molar. Prevalence in the Senegalese population 15 to 19 years of age. Odontostomatol Trop. 2000; 23(89): 31-34.
38. Schalk-Vander Weide Y, Steen WH, Bosman F. Taur- odontism and length of teeth in patients with oligodontia. J Oral Rehabil. 1993; 20(4): 401-412.
39. Thesleff I. The genetic basis of normal and abnormal craniofacial development. Acta Odontol Scand. 1998; 56(6): 321-325.
40. Thongudomporn U, Freer TJ. Prevalence of dental anomalies in orthodontic patients. Aust Dent J. 1998; 43(6): 395-398.

Ethano-Medicine in Dentistry

Subhasri Raman [1] , Madhulaxmi M [2]

Graduate Student [1],Reader [2], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Herbal medicine has been used in dentistry for several years especially in countries like India and China. The photochemical properties of these herbs can provide an excellent alternative for synthetic antibiotics and have shown promising results as preventive and therapeutic medicine in oral care. Many people take herbal medicines nowadays for their health care in different national healthcare setups. Herbal extracts have been profoundly been used in dentistry for reducing inflammation, as antimicrobial plaque agents, for preventing release of histamine and as antiseptics, antioxidants, antimicrobials, antifungals, antibacterials, antivirals and analgesics. They aid in healing and are effective in controlling microbial plaque in gingivitis and periodontitis, thereby improving immunity.The main aim of this review is to study the biological activities of clove, neem and tulsi along with the importance of such traditional herbs in our daily life.

Key Words: Clove, Herbal, Remedies, Neem, Tulsi.

Introduction

Herbs are one of remedial agents which God has created for afflicted humans. Herbal extracts have been used in traditional medicine for several thousand and are staging a comeback and herbal 'renaissance' is happening all over the globe. The herbal products, today, symbolize safety, in contrast to the synthetics that are regarded as unsafe to humans and the environment. The knowledge on medicinal plants has been accumulated in the course of many centuries, based on different medicinal systems such as Ayurveda, Unani and Siddha. In India, it has been reported that traditional healers use 2,500 plant species and that 100 species of plants serve as regular sources of medicine. During the last few decades, there has been an increasing interest in the study of medicinal plants and their traditional use in different parts of the world [1, 2].

According to the World Health Organization (WHO), as many as 80% of the world's people depend on traditional medicine (herbal) for their primary healthcare needs. The development of indigenous medicines and the use of medicinal plants carry considerable economic benefits in the treatment of various diseases [3]. In the developed countries, 25% of the medical drugs are based on herbs and their derivatives [4].

Since tooth brushing is the most basic process in oral care, indigenous people all over the world use natural tooth brushes which are made from herbal healing plants. These brushes made from primitive twig actually work quite well and they provide natural-bristle which can be disposed with healing ingredients which have already been incorporated right in the plants. Herbalist, Lesley Tierra had mentioned in her, 'The Herbs of Life' (1992), volatile oils present in this twig can stimulate blood circulation and tannins can tighten and cleanse gum tissue and thereby maintain the gums healthy. Bay, eucalyptus, oak, fir and juniper, all work well for this'.

The Natural Dentist Healthy Gums Daily Oral Rinse (The Natural Dentist, Medford, Mass.) which was formerly named, Herbal Mouth and Gum Therapy, has been shown to have beneficial effects in the oral environment, such as reduction of gingival bleeding and gingivitis and inhibition of the growth of aerobic, micro-aerophilic and anaerobic bacteria [5]. This mouth rinse contains several naturally occurring, anti-inflammatory agents such as aloe vera and calendula and antimicrobial agents such as Golden Seal and grapefruit seeds. It was opined that when it was applied specifically against the broad spectrum of oral bacteria which are responsible for

gingivitis, plaque and periodontal disease, this herbal mouth rinse could prove beneficial for maintaining oral health [6].

The use of herbal remedies has assumed a global dimension, which has culminated in their being used in the treatment of various ailments in both developed as well as developing countries. Although today, only a few of these many herbs have been approved for their commendable medicinal properties, a large majority of naturally occurring herbs are only considered as food supplements, because of the lack of a randomized controlled clinical trials.. Herbal medicines are believed to be benign and to not cause severe toxicity. This, coupled with lower costs as compared to those of conventional medications, is the major attraction of these treatments. Despite the general belief, use of herbal medicines can cause severe toxicity and even death[7].

The use of herbal medicines continues to expand rapidly across the world. Many people take herbal medicines or herbal products now for their health care in different national healthcare settings. Herbal extracts have been used in dentistry for reducing inflammation, as antimicrobial plaque agents, for preventing release of histamine and as antiseptics, antioxidants, antimicrobials, antifungals, antibacterials, antivirals and analgesics. They also aid in healing and are effective in controlling microbial plaque in gingivitis and periodontitis and thereby improving immunity.

Ethnomedicine is a sub-field of ethnobotany or medical anthropology that deals with the study of traditional medicines: Not only those that have relevant written sources (traditional Chinese medicine, Ayurveda), but especially whose knowledge and practices have been orally transmitted over the centuries [8]. The current manuscript highlights various usage of clove, neem and tulsi related to oral health and suggests their derivatives in conventional dental practices.

Clove

Syzygiumaromaticum or clove an age old-spice has been used as traditional herbal medicine in Asia and Middle East Countries. Clove serves as a promisinganti-inflammatoryanti-fungal and anti-microbial, immune modulator, anti-carcinogenic and anti-mutagenic [9]. Cloves grow in hot tropical climates like the islands of Indonesia, having its native from Indonesia. The name

derives from French clou, a nailas the bud vaguely resemble small irregular nails in shape [10]. They are evergreen plants and reach a height of approximately thirty feet high. The leaves of the clove have a typical leathery textured and are usually covered with many tiny depressions along the entire length. The part of the clove that is used is the flower buds of the clove. The aromatic oils of the clove have a stimulant and irritant effect commonly encountered complaint by applicant. Cloves can bring about rise in blood pressure and body temperature [11].Cloves are the pink flowering bud of Eugenia aromatic an evergreen tree, which once dried until brown are used for medicinal and spicing purposes on a routine basis. Indigenous to the Moluccas spice islands of Indonesia, cloves also grow in India, the West Indies, Tanzania, Sri Lanka, Brazil and Madagascar [12]. Their strong aromatic flavor and powerful phenolic compounds have been the prime reason for their prominent usage for over hundreds of years as a nutritional spice for food and a remedy for health conditions. Arabic traders brought in clove buds to Europe in the fourth century A.D following which cloves became popular as a medicinal flower due to its brilliant ability to preserve food and mask poorly kept food in the seventh and eighth century A.D [13, 14].

Therapeutical Importance of Cloves

Germicidal property of clove has been exclusively used in dental care for relieving tooth ache, mouth ulcers and sore gums. Gargling with diluted clove oil can help in relieving sore throat. The aromatic efficiency of clove aids to control of halitosis [12]. Clove oil acts as an expectorant clears the respiratory tract treating many conditions like colds, bronchitis, sinus conditions and asthma [11]. Clove has a chemo- preventive role in lung and skin cancers [15, 16]. Antimutagenic property of clove was attributed to Phenylpropanoids which were isolated from the buds of cloves. These isolated products suppressed the expression of the gene. The suppressive compounds were localized in ethyl acetate extract portion of the processed clove [17].

Clove isolation has identified two anti-platelet components, eugenol and acetyl eugenol. They inhibited arachidonate, adrenaline- and collagen-induced platelet aggregation. Their inhibitory effect was reversible. These components behaved anti aggregatory based on two actions namely: (i) inhibition of formation platelet thromboxane and (ii) increased formation of 12-lipoxygenase products. It was observed eugenol and acetyl eugenol on being used in combination indicated

inhibition of platelet aggregation induced by arachidonate, adrenaline and collagen [18].Clove oil can be considered as an alternative to fishanesthetic[19].

A study was made on the effect of the cloves extract on the Cronobactersakazakii biofilm. C. sakazakii formed biofilm in the reconstituted milk after 24 hours incubation and the feeding tubes were used as the surfaces for the growth of C. sakazakii biofilm. The antimicrobial properties of clove extract were determined by comparing the thickness of biofilm on the feeding tubes before and after treatment with the clove extract. Increasing the concentration of the clove extract gave more effective result to inhibit the growth of C. sakazakii biofilm. Theresults proved that the clove extract had antimicrobial properties and can hence be used as disinfectant and solved biofilm problems in food industry [20].Specific components of cinnamon and cloves have been discovered to be efficient against mold growth and maturation till the stage of toxin production.The effects on Aspergillus parasiticus was studied using yeast extract sucrose broth being the substrate. It inhibited mold growth and toxin production. Levels of the eugenol above 200 ppm completely inhibited mold growth, or permitted only a small amount of growth [21]. Antioxidant property of spices is applied for preservation of lipids. Selected spices extracts (water and alcohol 1:1) were investigated on enzymatic lipid peroxidation. Water and alcoholic extract (1:1) of spices namely garlic, ginger, onion, mint, cloves, cinnamon and pepper on examination indicated dose-dependent inhibition of oxidation of fatty acid, linoleic acid in presence of soybean lipoxygenase. Clove exhibited highest inhibition and onion showed least. On a decreasing order the inhibitions were highest starting from cloves, cinnamon, pepper, ginger, garlic, mint and onion [22].

Home Remedies of Clove

Aromatherapy has been incorporated as a home remedy for respiratory conditions. A pomander can be hung at various places for a long standing continuous aroma therapy at home. The aroma from a hot tea with clove can also be inhaled to relieve cold [23]. Cloves can be used to remove offensive smells rather than using aerosols or other artificial air fresheners.An atomizer consisting of clove oil and water can be used. A clove pomander is another way. After the pomander has lost its effectiveness, the cloves still havemore aromas to share, which can then be crushed and used in potpourri. Cloves have excellent use in spicy potpourri. Sameatomizer can be used to freshen the air and if sprayed lightly on exposed skin it can be considered as an

effective mosquito repellent.The aromaalone from cloves will deter flies whetherit is in a potpourri an air freshener or apomander. Drops of clove oil will killants instantly. Clove oil along with cinnamon oil can kill ants [24].

Neem

Neem has been extensively used in Ayurveda, Unani and Homoeopathic medicine and has become a wonder tree of modern medicine [25]. It has been used traditionally for the treatment of inflammation, infections, fever, skin diseases and dental problems.

It is effective in several epidermal dysfunctions such as acne, psoriasis, eczema. Neem leaves have been reported to also possess antihyperglycemic, immunomodulatory, anti-inflammatory, antimalarial, antioxidant, antiviral, Antimutagenicandanti carcinogenic properties. Neem also exhibits antibacterial, antifungal, hepatoprotective, anti-ulcer, anti-fertility and anti-nociceptive activity [26].

Neem twigs are used as oral deodorant, toothache reliever and for cleaning of teeth. Neem bark possesses antibacterial and deodorant activity. The phytochemical constituents present in neem are nimbidin, nimbin, nimbolide, Azadirachtin, gallic acid, epicatechin, catechin, and margolone [27]. All these exhibit potent antibacterial activity. The chief active constituent of neem is azadirachtin, which is an effective antimicrobial agent. Neem has also been traditionally used as a skin moisturiser [28].

Azadirachtaindica is an evergreen tree having potential medicinal values. It has been found to be active against many dreadful disorders like hepatitis, viral infections, malaria and cancer. It is also effective against periodontal pathogens, and oral acidogenic bacteria responsible for dental caries and dental plaque. Nimbidin, a major active principle isolated from seed kernels of A. indicaexhibits several biological actions. From nimbidin other active constituents like nimbin, nimbinin, nimbidinin, nimbolide and nimbidicacid have been isolated which are responsible for its biological activities [27].

Antibacterial Activity

Neem is a natural antibacterial agent. Various scientific studies have revealed its antibacterial activity. The antimicrobial effects of Neem have been reported against S. mutans and S. faecalis [29]. Ethanolic extract of Neem leaves and sticks and bark exhibited significant antibacterial activity [30]. Dried chewing sticks of Neem showed maximum antibacterial activity against S. mutans compared to other dental caries-causing organisms, S. salivarius, S. mitis, and S. sanguis [31].

Anti-Candidial Activity

Ethanolic and aqueous extract of Neem leaf showed significant anti-candidial effect against C. albicans. A clinical study demonstrated the effects of the leaf aqueous extract from Azadirachtaindica(Neem) on adhesion, cell surface hydrophobicity and biofilm formation, which may affect the colonization by Candida albicans. The results suggest that Neem leaves have a potential anti-adhesive effect on the sample studied in vitro [32].

Anti-Cariogenic Activity

Mango and Neem extract showed antimicrobial activity against S. mutans, S. salivarius, S. sanguis and S. mitis. A combination of chewing sticks is found to be beneficial in eradicating the dental caries-causing organism [33]. Chloroform extract of Neem leaf inhibited Streptococcus mutans and Streptococcus salivarius also provides an aid for treating dental caries[34]. Acetone extract from the bark ofNeem is bactericidal against S. sobrinus hence indicates its anti-cariogenic activity. Antimicrobial activity of commercially available Himalaya herbal dental cream containing neem and fluoride-containing cheerio gel toothpaste has been assessed in school children. The study reported both the toothpastes showed a good antimicrobial effect on caries producing salivary streptococcus mutans. [35].

Anti-Plaque Activity

Neem oil shows significant antibacterial activity and has been suggested for use in treating dental plaque. Mucoadhesive dental gel containing Azadirachtaindica is found to be beneficial in

reducing the plaque index and salivary bacterial count comparatively better than chlorhexidine gluconate mouthwash [36].

Efficacy on Acidogenic Oral Bacteria on Fixed Orthodontic Appliance Wearing Patients

The primary acid-tolerant bacteria associated with dental plaque are Streptococcus mutans, Streptococcus oralis, Streptococcus sobrinus, Lactobacillus acidophilus, Streptococcus salivarius, Streptococcus mitis, Streptococcus sangui, Streptococcus intermedius, and Streptococcus anginosus that are seen around orthodontic appliances are one of the main complaints of the patients. It has been seen exclusively that orthodontic appliance often leads to compromised oral hygiene maintenance and plaque retention.

Ethanolic leaf extract of Azadirachtaindica has shown significant antibacterial activity against certain oral bacteria causing dental plaque in fixed orthodontic appliance patients. A study on orthodontic appliance wearers evaluated the anti-plaque activity of the extract against S. mutans, S. sanguis, and S. mitis. The extract did not inhibit L. acidophilus when tested [37].

Efficacy against Periodontal Pathogens

Brushing with Neem toothpaste after every meal and using a mouthwash with Neem extract is recommended treatment for preventing gingivitis. In a study, Neem-based mouth rinse was given to patients for assessing anti-plaque and anti-gingivitis activity. The findings conclude that Neem mouth rinse is as effective as chlorhexidine in reducing periodontal indices. Neem stick is found to be effective as a toothbrush in reducing dental plaque and gingival inflammation [38].

Studies indicate that leaf extract of A. indica-based mouth rinse is highly efficacious and that it may be used as an alternative therapy in the treatment of periodontal disease. Gingivitis has been prevented or even reversed with regular use of Neem toothpaste and mouthwash. Shefali sharma conducted a study on Soluneem (a water-soluble formulation from the Neem seed kernel from Shefali sharma conducted a study on Soluneem (a water-soluble formulation from the Neem seed

kernel from AzadirachtaIndica containing Azadirachtin) as an antimicrobial agent and the effective concentration of Soluneem required to inhibit periodontopathic bacteria and to compare it with a known antiplaque agent chlorhexidine (0.2%) in vitro. Study revealed that soluneem extract did not show activity against the organisms (Bacteroidsfragilis, B. distatonics, Prevotellacorporis, Prevotellamelagingogenica, Pepto streptococcus species) tested.

Also Botelho et al., and Behl et al., in their experiments and trials concluded that Azadirachtaindica is highly efficacious in the treatment of periodontal disease thus exhibiting its biocompatibility with human periodontal fibroblast [39].

Neem as Root Canal Irrigant

Sodium hypochlorite has been used as root canal irrigant for decades; it causes potential weakening of the tooth structure by decreasing the hardness and structural integrity of the dentin within the root canal. To overcome this

disadvantage herbal drugs are used effectively to inhibit E. faecalis that causes root canal failure in patients undergoing endodontic treatment.

Aqueous and ethanolic extract of Neem leaf inhibits S. mutans and E. faecaliswhich cause root canal failure in endodontic procedure. Its antioxidant and antimicrobial properties makes it a potential agent for root canal irrigation as an alternative to sodium hypochlorite [40]. Literature suggested that the Neem(Azadirachtaindica) leaf extract has significant antimicrobial effect against E. faecalis derived from infected root canal samples. The extract was found to be efficacious compared with 2% sodium hypochlorite.

Neem for Dental Care

Various parts of the Neem tree possess astringent and antiseptic activity. Leaf extracts have been widely used in both traditional and conventional times to manufacture toothpaste and mouthwash in the oral care dentistry. Its antibacterial properties due to the presence of nimbidin, Azadirachtin, and nimbinin help to remove many oral aerobic and anaerobic pathogens existing in the oral cavity.

Neem bark and leaf extract is most effectively used in preventing cavities and gum disease. Mouthwash containing Neem is a remedy for tooth decay, oral infections, prevents bleeding and sore gums. Twigs of Neem tree are used as chewing sticks by people all over India.

Tulsi

The plants are used for medicinal purpose since ancient times. The history of therapeutic use of plants is as old as 4000–5000 B.C. The Chinese were the first to use natural herbal preparations as medicines.The tulsi in sanskrit means 'incomparable or matchless'[41]. In ancient India the traditional medicine practitioners observed tulsi as greatest healing herb with high therapeutic potential. The botanical name of tulsi is 'Ocimum sanctum Linn'. In hindi it is known as 'Tulsi' and 'Holy Basil' in English. The tulsi plant is an erect softy hairy aromatic herb or under shrub of labiatae family belonging to genus 'Ocimum' and is found throughout India having high therapeutic potential.

Tulsi is known in different regions, languages and dialects of India by a variety of other names such as Tulasi, Surasah, Ajaka, Parnasa, Manjari, Haripriya and Bhutagni. Several species of tulsi has been seen worldwide. In India most commonly cultivated species is Ocimum sanctum Linn. The first type is green leaves type known as sri tulsi and second is purple leaves type known as krishna Tulsi. Other species of genus Ocimum which have high therapeutic potential and cultivated worldwide are Ocimum gratissium (Ram Tulsi), Ocimum canum (Dulal Tulsi), Ocimum basilicum (Ban Tulsi), Ocimum kilimandscharicum, Ocimum ammericanum, Ocimum camphora and Ocimum micranthum [42]. In Ayurveda tulsi has been well documented for its therapeutic potentials and described as DashemaniShwasaharni (antiasthmatic) and antikaphic drug. The leaves, stem, flower, root, seeds and even whole plant of ocimum sanctum Linn is used in traditional medicine. Tulsi is often enjoyed as a simple herbal tea and is frequently blended with other herbs and spices for various medicinal and culinary purposes. The Indian scientists and researchers have carried out several studies to find out the therapeutic potential of Ocimum sanctum Linn.

Chemical Constituents of Tulsi

Eugenol & Essential Oils - Ocimum sanctum L. (Tulsi) and Ocimum basilicum (Ban Tulsi) are cheaper sources for commercial extraction of eugenol. The aerial parts (leaves, flowers & stem)

of tulsi contain essential oils with good percentage of eugenol. The leaves of ocimum sanctum L. are chief source of essential oils followed by the inflorescence and stem however flowers contain more essential oils than leaves in ocimum basilicum. The roots and fruits of these plants are almost completely devoid of any essential oil. The essential oil extracted from the tulsi leaves by steam distillation largely contains eugenol.

The other important constituents of the essential oil are carvacrol, methyl eugenol, caryophyllene. To develop eugenol-rich ocimum variety scientists of Regional Research Laboratory (RRL), Jammu have developed a hybrid strain of ocimum gratissimum using recurrent selection (FCA) technique of breeding and named it as Clocimum. This Clocimum variety contains 60–65% eugenol. Another heterotic F1 strain of ocimum has been developed by Regional Research Laboratory (RRL) and named as Clocimum-3c.

This is an improved eugenol-rich ocimum variety containing 90–95% eugenol. 2. Carracrol and tetpene&Sesquiterpene b caryophylline 3. Linalool 4.Polyphenol rosmarinic acid 5.Vitamin A, C, Zinc & Iron [43].

Therapeutic Uses of Tulsi in Dentistry

Tulsi is generally considered as an Elixir of Life. Traditionally tulsi has been employed in hundreds of different formulations for the treatment of a wide range of disorders involving mouth and throat, lungs, heart, blood, liver, kidney, and the digestive, metabolic, reproductive and nervous systems [44]. The different part of tulsi like leaves, roots, flowers and stems have charismatic therapeutic potential. Tulsi has been used as expectorant, analgesic, anticancer, antiasthmatic, antiemetic, diaphoretic, antidiabetic, antifertility, hepatoprotective, hypotensive, hyolipidimic and antistress agent. As tulsi is a very effective in treatment of various medical disorders, It is also a very promising herb in management of oral diseases and dentistry. Tulsi leaves are quite effective in treating common oral infections. The tulsi leaves contains strong antibacterials like carracrol and tetpene&sesquiterpene b caryophylline.

The antibacterials present in tulsi leaves are approved by FDA as food additive. Ocimum sanctum leaves contain 0.7% volatile oil comprising about 71% eugenol and 20% methyl eugenol. Due to significant amount of eugenol (1-hydroxyl-2 methoxy-4 allyl benzene) tulsi is a strong COX-2 inhibitor. This antianalgesic property of tulsi is utilized in treatment of dental and mucosal pain[45].

The powdered tulsi leaves mixed with mustard oil can be used as toothpaste for tooth brushing. The powdered tulsi leaves used to encounter halitosis and maintaining good oral health. Massage with tulsi powder has reported to be highly effective in many gingival and periodontal diseases.The tulsi extract has high antimicrobial activity against streptococcus mutans.The streptococcus mutans has been reported to be key microorganism causing dental caries. In an in vitro study it was found that 4% concentration of tulsi extract has highest antimicrobial activity. The Tulsi also possesses a great antifungal activity. In a study conducted by Khan Ait was concluded that linalool and eugenol which are present in essential oil extracted from tulsi are effective against two strains of candida (C. albicans and Candida tropicalis) but linalool is more effective than eugenol against candidasis. [46].

The tulsi have property of immunomodulation. It also acts on skin and hemopoeitic tissues. So the tulsi can be used in treatment of oral lichen planus. However further studies are needed to evaluate efficacy of tulsi in treatment of oral lichen planus.Tulsi can also be used as antioxidant therapy in both leukoplakia and oral submucous fibrosis. The polyphenol rosmarnic acid is a strong antioxidant present in tulsi. So it can be used in treatment of all other oral precancerous lesions and condition. Due to immunomodulating property, the ocimum sanctum can be used in treatment of pemphigus. The tulsi causes healing of sores and blisters. More studies are needed to evaluate the potential use of immunomodulatory effect of tulsi in immunologically mediated mucosal disorders like pemphigus. The ocimum sanctum is a potent antiulcerogenic and have ulcer healing properties. Ocimum sanctum in dose of 100mg/kg was found to be effective against ulcers. [47]

The antiulcer effect of ocimum sanctum is reported to be due to cytoprotective effect rather than anti-secretory activity. Tulsi is effective in both oral ulcers and peptic ulcer. The tulsi is a rich source of vitamin A, Vitamin C, zinc and iron. It is also a rich source of chlorophyll and other polynutrients. So it can be used as dietary supplements in oral diseases arising due to deficiency of these nutrients. [48]

Conclusion

This review on the dental aspects of various herbal extract with their chemical constituents and biological activities helps in increasing their applications in dentistry. This information may give a bird's eye view for the dentist, and consequently this database might play a major role in future

research in the field of dentistry. The active principles of plants should be incorporated into modern oral health-care practices and dentists should be encouraged to use natural remedies in various oral health treatments.

Corresponding Author

Madhulaxmi M

Reader, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Email ID: madhulaxmi14@gmail.com

Telephone Number: 7373814000

REFERENCES

1. Hanazaki N, Tamashiro JY, Leitao-Filho H, Gegossi A. Diversity of plant uses in two Caicaras communities from the Atlantic forest coast, Brazil. Biodiversity and Conservation. 2000;9:597–615.
2. Rossato SC, Leitao-Filho H, Gegossi A. Ethnobotany of Caicaras of the Atlantic forest coast (Brazil) Economic Botany. 1999;53:387–395.
3. Azaizeh H, Fulder S, Khalil K, Said O. Ethnomedicinal knowledge of local Arab practitioners in the Middle East Region. Fitoterapia. 2000;74:98–108.
4. Principe P. Monetising the pharmacological benefits of plants. Washington, D.C: US Environmental protection Agency; 1991.
5. George D, Bhat SS, Antony B. Comparative evaluation of the antimicrobial efficacy of aloe vera tooth gel and two popular commercial toothpastes: An in vitro study. General Dentistry. 2008:238–240
6. Almas K. The antimicrobial effects of seven different types of Asian chewing sticks. Odonto-StomatologieTropicale. 2001;96:17–20.

7. Kumar, G., Jalaluddin, M., Rout, P., Mohanty, R., &Dileep, C. L.Emerging Trends of Herbal Care in Dentistry. Journal of Clinical and Diagnostic Research : JCDR, 2013;7(8), 1827–1829.
8. Acharya D, Shrivastava A. Indigenous Herbal Medicines: Tribal Formulations and Traditional Herbal Practices. India: Aavishkar Publishers Distributor; 2008;440.
9. FarzanehDehghani, AseyehHeshmatpour, Mohammad Reza Panjehshahin, TaherehTalaei-Khozani.Toxic effects of water/alcoholic extract of Syzygiumaromaticum on sperm quality, sex hormones and reproductive tissues in male mouse. IUFS Journal of Biology. IUFS J Biol 2012, 71(2):95-102
10. Alma, H., Ertas, M., Nitz, S., &Kollmannsberger, H.Research on essential oil content and chemical composition of Turkish clove (Syzygiumaromaticum L.). BioResources,2007;2(2): 265-269.
11. Debjit Bhowmik, K.P. Sampath Kumar , AkhileshYada , ShwetaSrivastava, ShravanPaswan, AmitSankarDutta.Recent Trends in Indian Traditional Herbs Syzygiumaromaticum and its Health Benefits. Journal of Pharmacognosy and Phytochemistry. 2012;1: 1-17.
12. Merril and Perry, syn. Eugenia caryophyllus (Sprengel) Bullock and S. Harrison International Organization for Standardization.Oil of clover leaf, Syzygiumaromaticum (Linnaeus).ISODirective 3141/1997, Geneva, Switzerland,2002.
13. Merril and Perry, syn. Eugenia caryophyllus (Sprengel) Bullock and S. HarrisonInternational Organization for Standardization Oil of clove buds Syzygiumaromaticum (Linnaeus).ISODirective 3142/1997, Geneva, Switzerland, 2002.
14. Merril and Perry, syn. Eugenia caryophyllus (Sprengel) Bullock and S.Harrison.International Organization for Standardization Oil of clove stems Syzygiumaromaticum (Linnaeus)ISODirective 3143/1997, Geneva, Switzerland, 2002.
15. The United States Pharmacopeia 31. Clove Oil, the National Formulary Edn 26, Vol. 1, Official Monography, 1113, the United States Pharmacopeial Convention, Rockville, MD, 2008
16. seidemann J. Syzygiumaromaticum (L.) Merr. LM Perry.World Spice Plants. Springer- Verlag, Berlin Heidelberg, Germany, 2005
17. MitsuoMiyazawa, MasayoshiHisama, Antimutagenic Activity of Phenylpropanoids from Clove (Syzygiumaromaticum). J. Agric. Food Chem., 2003; 51(22):6413–6422.

18. K.C. Srivastava, Antiplatelet principles from a food spice clove (Syzgiumaromaticum L) Prostaglandins, Leukotrienes and Essential Fatty Acids,1993;48:363-72
19. W. Gary Andersona, R.ScottMcKinleya and Maria Colavecchiaa, The Use of Clove Oil as an Anaesthetic for Rainbow Trout and Its Effects on Swimming Performance. North American Journal of Fisheries Management. 1997;17.
20. Hartmann I, Carranza P, Lehner A, Stephan R, Eberl L, Riedel K. Genes Involved in Cronobactersakazakii Biofilm Formation. Applied and Environmental Microbiology. 2010;76(7):2251-2261.
21. L. B. Bullerman, f. Y. Lieu, sally a. SeierInhibition of growth and aflatoxin production by cinnamon and clove oils. Cinnamic aldehyde and eugenol. Journal of food science.. Anaheim 1977;4.
22. S. Shobana, K. AkhilenderNaidu.Antioxidant activity of selected Indian spices. Prostaglandins, Leukotrienes and Essential Fatty Acids 2002; 62:107–110
23. Jirovetz L, Buchbauer G, Stoilova I, Stoyanova A, Krastanov A, Schmidt E. Chemical composition and antioxidant properties of Clove leaf essential oil. J Agric Food Chem. 2006;54:6303–6307
24. Heinrich M, Barnes J. Clove, Syzygiumaromaticum (L.) Merr. & L.M. Perry.Fundamentals of Pharmacognosy and Phytotherapy,Second edition. Churchill Livingstone, Edinburgh London, Great Britain, 2004
25. Athavale VB. New Delhi: Chaukhamba Sanskrit Pratishthan; 1999. Dentistry in Ayurveda [Danta-Shastra]
26. Khosla P, Bhanwra S, Singh J, Seth S, Srivastava RK. A study of hypoglycaemic effects of *Azadirachtaindica* (*Neem*) in normalandalloxan diabetic rabbits. Indian J Physiol Pharmacol. 2000;44:69–74.
27. van der Nat JM, Klerx JP, van Dijk H, de Silva KT, Labadie RP. Immunomodulatory activity of an aqueous extract of *Azadirachtaindica* stem bark. J Ethnopharmacol. 1987;19:125–131.
28. Tidjani MA, Dupont C, Wepierre J. Antiinflammatory activity of *Azadirachtaindica*. Planta Med Phytother. 1989;23:259–266.
29. Dhar R, Zhang K, Talwar GP, Garg S, Kumar NJ. Inhibition of the growth and development of asexual and sexual stages of drug-sensitive and resistant strains of the

human malaria parasite Plasmodium falciparum by *Neem* (Azadirachtaindica) fractions. J Ethnopharmacol. 1998;61:31-39.

30. Rao AD, Devi KN, Thyagaraju KJ. Ayurtox for body Detoxification. Enzyme Inhib. 1998;14:85–86.
31. Rao AR, Sukumar S, Paramasivam TV, Kamalakshi S, Parashuraman AR, Shantha M. Study of antiviral activity of tender leaves of Margosa tr*e*e (*Melia* azadericta) on vaccinia and variola virus: A preliminary report. Indian J Med Res. 1969;57:495–502
32. Balasenthil S, Arivazhagan S, Ramachandran CR, Ramachandran V, Nagini S. Chemopreventive potential of *neem* (*Azadirachtaindica*) on 7, 12-dimethylbenz (a) anthracene (DMBA) induced hamster buccal pouch carcinogenesis. J Ethnopharmacol. 1999;67:189–195.
33. Arivazhagan S, Balasenthil S, Nagini S. Modulatory effects of *Garlic* and *Neem*leaf extracts on N-methyl-N′-nitro-N-nitrosoguanidine (MNNG)-induced oxidative stress in Wistar rats. Cell Biochem Funct. 2000;18:17–21.
34. Baswa M, Rath CC, Dash SK, Mishra RK. Antibacterial activity of Karanj(Pongamiapinnata) and Neem (Azadirachtaindica) oil: A preliminary report. Microbios. 2001;105:183–189.
35. Jacobson M. Pharmacological and toxicological effects of Neem and China berryon Warm blooded animals. Neem Newslett. 1986;3:39–43.
36. Bhanwra S, Singh J, Khosla P. Effect of *AzadirachtaIndica (Neem)* leaf aqueous extract on paracetamol-induced liver damage in rats. Indian J Physiol Pharmacol. 2000;44:64–8.
37. Bandyopadhyay U, Chatterjee R, Bandyopadhyay R. US Patent 5,730,986. 1998corresponding to Indian Patent 1100/Del/95.
38. Saimbi CS. The efficacy of neem extract -Reported in Jeevaniya Health Care Magazine 1994.
39. Bhanwara S, Singh J, Khosla P. Antinociceptive activity of *AzadirachtaIndica (neem)* in rats. Indian J Physiol Pharmacol. 2000;18:17–21.
40. Sharma P, Tomar L, Bachwani M, Bansal V. Review on Neem (*Azadirechtaindica*): Thousand Problem One Solution. Int Res J Pharm. 2011;2:97–102.
41. Sirkar NN. Pharmacological basis of Ayurvedic therapeutics. In: Cultivation and utilization of medicinal plants. Editors: Atal CK and Kapoor BM (Published by PID CSIR) 1989.

42. Rai Y Holy Basil tulsi a herb, Navneet publications private limited,india.1984
43. Pandey BP, Anita. In: Economic Botany (Published by Chand and Company Ltd., Ramnagar, New Delhi),1990; 294.
44. Atal CK, Kapoor BM. Cultivation and utilization of medicinal plants (Eds. PID CSIR), 1989. 5. Sen P. Therapeutic potentials of Tulsi : from experience to facts. Drugs News & Views 1993; 1(2): 15–21.
45. Satyavati GV, Raina MK, Sharma M. Medicinal Plants of India (Published by ICMR, New Delhi), 1976
46. Gupta SK, Prakash J, Srivastava S. Validation of claim of Tulsi, Ocimum sanctum Linn as a medicinal plant. Indian J Experimental Biology 2002; 40(7): 765–773.
47. S. Rajeshwari. Ocimum sanctum. The Indian home remedy. In:Current Medical Scene, March-April 1992 (Edited and published by Cipla Ltd., Bombay Central, Bombay).
48. Mukherji S.P. Ocimum - a cheap source of Eugenol. Science Reporter 1987, p. 599. 31. Eugenol-rich Ocimum variety released. In: CSIR NEWS (Published by PID CSIR, New Delhi), 1995; 45:256.
49. Agarwal P, NageshL,Murlikrishnan. Evaluation of the antimicrobial activity of various concentrations of tulsi(ocimum sanctum) extract against streptococcus mutans: Ind J Dent Res.2010;21(3):357-359

Evolution of 3D Imaging in Dentistry

Divya T [1], Ashok V [2], Maragathavalli.G [3]
Graduate Student [1],Senior lecturer [2],Department of Prosthodontics,
Professor and Head [3],Department of Oral Medicine and Radiology,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

3D printing is atechnology in which many different materials are used to create a three dimensional model. It has the capacity to change the future of dental model production, due to the beneficial models it can create. The objective of this review is to explore the various advancementsin 3D printing that maypositively affect several aspects in dentistry. 3D printing is a revolutionary technology that has the potential to positively affect the work of dentalpractitioners, while improving the lives of others.

***Key words** : 3- dimensional , imaging, printing, technology, dentist*

Introduction

3Dprinting was previously most associated with science fictionThistechnology is now becoming an application within many industries such as manufacturing and medicine. Initially it was very expensive. As more research is being developed, 3D printing is rapidly becoming an affordabletechnology. It was originally based on ink jet principles which has the ability to print with many materials[1]. During its usage, a CAD file is processed through a specialisedsoftware and cut into a series of 2D layers. The printer then produces the object layer by layer with a supplemental support from a water soluble material[2].This reviewis focused on the applications of 3D printing in dentistry.Patients will no longer have to suffer through outdated techniques. A scan will capture the patient's mouth. This will be processed and a 3D printer will produce a replica of the mouth[3]. These major advancements are possible due to 3D printing.

History of 3-D Printing

Printing methods are constantly improved to keep up with the society. Themovable type in print technology was introduced by Johannes Gutenberg in 1400's. The rotary printing press was developed and in early 1900's lithography and letterpress machines were the dominant[4].In 1895, X-rays were discovered by W. C. Roentgen. This opened a new era in medicine and dentistry. Thirty-six years later, standardised methods for the producing cephalometric radiographs were introduced to the dental professionals by Broadbent and Hofrath simultaneously and independently, [5] and thus it remained comparatively unaltered until a few years back. Broadbent emphasised the importance of the position and the distance arrangements to produce distortionfree radiographs while taking the lateral and posteroanterior cephalometric radiographs [6]Therewere several disadvantages of 2-dimensional cephalometry as a scientific method. The fact that the conventional head filmreduces 3D objects to 2-dimensional view was the first and the most important reason. When 3D objects were displayed in a 2-dimension, the structures weredisplaceded as vertical and horizontal proportions to their distance from the film [7, 8].Secondly, cephalometric analyses were based on an excellent superimposition of the left and right sides at mid-sagittal plane. But such a superimposition was rarely observed because facial symmetry was infrequent. Third reason was that the manual data collection and processing in cephalometric analysis had been shown to have low correctness and precision [9].Finally, major errors in the cephalometric measurements were associated with uncertainties in locating anatomical

landmarks due to the deficiency of the well-defined outlines, hard edges, and shadows as well as the patient position [10].

In spite of these limitations, lots of cephalometric analyses had been developed to help in the diagnoses of skeletal and dental malocclusions and alsodent facial deformities [11,12].The quantitative errors associated with thetraditional 2D cephalometry had been substantial enough to make orthodontic diagnoses and treatment planning [13-17].

After the introduction of 3D imaging, dentists have had a great opportunity to evaluate anatomic structures 3-dimensionally in dental practice. Several investigators organised and conducted 3D imaging researches, and Singh and Savara [18]reported the first 3D analysis about the growth changes in maxilla. Computer softwares also helped to collect and analyse 3D coordinates directly from the digital cephalometric images, so that tracing manually and digitisingusingmouse on screen were abandoned [19,20].

3D imaging technique had been improved to use in different areas of medical sciences. Being improved from old photogrammetric techniques, stereophotogrammetry had been introduced so as to provide a more extensive and accurate assessmentof the captured objects. Using one or more convergingtypes of views, a 3D model could be constructed and monitored from any perspectiveand measured from any of the directions. In 1944, Thalmann-Degan recorded the facial differences after dental, more specifically, orthodontic treatment. This was the earliest clinical report about stereophotogrammetry [21].Computerisedstereo photometry has come into the market, parallel to the computer developments and has provided faster,and more comprehensive and correct taking of constructing sequences [22].

The first CT scanning device was developed around 40 years back. After a short duration of time, a stack of CT sectional images was used to obtain a3D information. At the beginning of 1980s, dentists used 3D imaging in craniofacial deformities [23]. For dento skeletal surgical needs, first simulation software was introduced in 1986. Then, the principles and the applications of 3D CT- and MRI-based imaging in dentistry were published. A specific discipline was established on 3D imaging, which dealt with different types of imaging, manipulation, and analysis of multi dimensional medical structures [24].

In 1960’s laser printing and 20 years later ink jet was brought. This technology sprays droplets of ink onto a substrate forming an image, known as 3D printing.The 3-D printing

was introduced by 3D systems in 1987. It was introduced as an additional print process which uses stereolithography for building models and prototypes. Later, 3D printing was developed and patented by the Massachusetts Institute of Technology in 1993 [25].There are several different types of 3D printing that includes Stereo lithography, Fused deposition modeling, Selective laser sintering, Traditional 3D printing, polyjet, and polyjet matrix[26]. Today,several methods of printing are used that follow traditional methods with improved capacities that produce high quality prints in a fast and efficient manner. 3D printing now with its advanced technology and versatility,can be applied to many fields[27]. This process has tremendous potential as it is valued by many industries.

Process of 3-D Printing

The 3-D print process consists of printing and post processing of the 3-D object being created. Printing with this new technique uses an additive rapid prototyping process, that is, it is based on inkjet technology. Similar to an inkjet printer that prints on a substrate with ink, 3-D printing uses this technology as a method to “jet” a binder material in the form of droplets. These droplets then conjoin with powder particles, which is deposited in layers, to create a substance[28]. In this process the powder material acts as the substrate and can be wet or dry. The liquid binder acts as an ink[29]. When printing, the goal is to distribute each layer as smooth and evenly as possible tocreate a consistent and accurate shape of the desired model. As mentioned, 3-D printing uses the principles of stereolithography to print a 3-D model layer by layer. These layers have been sliced by computer algorithms from a Computer Aided Design (CAD) file. The importantelementsinvolved in the three dimensional (3-D) printing of any object include the formulation of powder, binder material, printing of ink, and post processing which all contribute to the final formulated models[30].

Applications of 3-D Printing in Dentistry

3D printing was slow, inefficient andinconsistent process in the beginning. But today, the speed and accuracy of 3D printing are the main reasons that it is getting implemented into the fields of dentistry[31].Traditional practises were extremely uncomfortable and time consuming for patients. Time was lost waiting for moulding material to set and harden[32]. Although there wereimprovementsinthis process, it was still slow. One problemwhile using traditional casting was to create a teeth casting andto get an impression of the soft tissues of the mouth[33]. The usage of 3D printing solves some of these problems. In the olden days when a patient went to a dentalclinic - the dental practitioners had to pour goo into patients’

mouths to take impressions[34].Now, new technology has been developed that will do a digital scan of a patient's entire teeth[35]. Once it is captured by a scanner the operator delivers it to a lab where 3Dprinters are used to create the artificial dentition. With this process, it is possible to print not only teeth but also the jaw. By having a precise 3D printed mould, it is possible to create accurate prosthesis for jaw, facial reconstruction[36].Stereolithography is known to be a useful tool in maxillofacial surgery, reconstructive surgery, orthopaedics and in paediatric operations. Using this technology, templates or prosthesis can be tested and reconstructed before the operation. These three dimensional models are helpful for preoperative planning of surgical procedures[37].

Applications in Oral and Maxillofacial Surgery

Medical modelling, one of the earliest application of 3D printing in surgery, was used to produce an anatomical study model[38]. This was made even more accessible by another technology in dentistry, the CBCT[39,40].CBCT helps provide volumetric image data to a 3D printer before surgery [41] which is used in making detailed replicas of the patient's jaws. This helps review the anatomy so that a surgical approach can be planned or practised before surgery[42-43].

Applications in Prosthodontics

It is possible to develop a precise virtual model with the help of scanners[44,45]. In prosthodontics, treatment may be planned and restorations can be designed using CAD software. It may be used to mill or print crown or bridge copings, implant abutments and bridge structures.3D printing may be used for fabricating metal structures [46] either by printing in burn out resins or waxes, or in metals or metal alloys. The benefit of printing in resin/wax is that there is much less post processing involved than in the direct 3D printing of metals. Printing in metals requires the use of more costly technologies which have their own requirements and demands. When printing implant bridge structures, 3D printing may be used along with milling/machining technologies to produce an accurate mechanical connection to the implant, combining the best attributes of printing and with little waste.

Applications in Conservative Dentistry

Dentists maysometimes need a physical model of the scanned jaw for certain procedures which is now possiblewith the help of 3D printing. It may be used for fabricating a

restoration, like adding a veneering material. Patient model data may be digitally saved, and printed only when needed, easing storage requirements [47].

Applications in Orthodontics

Imaging is one of the most important tools for orthodontists as it is used to evaluate and record the size and form of craniofacial structures. Orthodontists regularlyuse 2-dimensional (2D) static imaging techniques, but the depth of structures cannot be obtained and localised with 2D imaging. So three-dimensional (3D) imaging has been developed in the early of 1990's and has gained a crucial place in dentistry, especially in orthodontics.

In orthodontics, treatment is planned, appliances are created, and wires are bent robotically using intra oral or CBCT to capture patient data. 3D printing digitally re-aligns the patients teeth to make a series of 3D printed models[48]. An example of printing is the manufacture of 3D printed, bracket bonding splints, printed in rigid and flexible materials for precise bracket placement [49]. In 3D diagnostic imaging, a series of anatomical data is gathered using certain technological equipment, processed by a computer and later showed on a 2D monitor to present the illusion of deepness [50].

Facial soft and hard tissues, and the dentition are the three main sections, also known as the triad, in orthodontics and orthognatic surgery. This triad has an important function in the planning of orthodontic treatment. Therefore, imaging of these structures is a useful diagnostic toolfor dentists to make a treatment modality [51]. 3D imaging for orthodontic uses contain preand posttreatment evaluation of dentoskeletal and craniofacial relationship, and eventually the facial appearance and beauty, evaluating the treatment results in terms of soft andhard tissues. 3D dental, facial, and skeletal images for making diagnostic decisions and treatment planning are the other benefits of three dimensional imaging in orthodontics [52].

Conclusion

3D printing technology is still in the developingstage. While it was in the marketplace since 1993, because of its limited usage, varying accuracy, high rate and slow production time, 3D printing was not an alternative to traditional modeling [53]. Presently it is used only in unique cases.Many dental practitioners are eager to implement 3D printing in their practice. This process will allow for the creation of common procedures and models to occur in a faster and

more precise manner allowing for quicker and better service to each patient[54].By reducing the time, a dental practitioner canprovide service to more patients and improvepatient satisfaction using the cutting edge technology[55]. Like other fields, using 3D technology requires a high cost. This is a major factor causing 3D printing of dental models from being a technology common in the field. In order for 3D printing to become a technologycommon withindental fields,the cost for 3D printers needs to reduce. But, due to the technology's initial stages of development, it may take a few decades before cost becomesaffordable[56]. If the cost reduces, 3D printing will become even more desirable by dentalpractitioners due to its exceptional accuracy.

Corresponding Author

G.Maragathavalli

Professor and Head,

Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai – 600077,Tamil Nadu, India

Mobile Number: +919445171146

Email ID:drgmvalli@yahoo.co.in

Reference

1. Schick.CDR System Requirements. Updated, March 2013. Patterson Dental, Patterson Technology Center, Effingham, Illinois.
2. Azari A, Nikzad S.The evolution of rapid prototyping in dentistry: A review. Rapid Prototyp Jr 2009; 15: 216-225.
3. Liu Q, Leu MC, Schmitt SM. Rapid prototyping in dentistry: Technology and application. Int J Adv Manuf Technol 2006; 29: 317-35.
4. Lightman AJ. Medical applications. Prinz, F.B. (Ed.), Rapid Prototyping in Europe and Japan, Loyola College of Maryland, Baltimore, MD, pg. 129-35.
5. Schubert c, Van Langeveld MC, Donoso LA. Innovations in 3D printing: a 3D overview from optics to organs, Br J ophthalmol. 2013 Nov 28.
6. Chua, C.K., Leong, K.F. (2000) Rapid Prototyping: Principles and applications in manufacturing, world scientific publishing.

7. Grimm T.User's Guide to Rapid Prototyping. Michigan: Society of Manufacturing Engineers; 2004.
8. da Rosa EL, Oleskovicz CF, Aragão BN. Rapid maxillofacial prototyping surgery in and traumatology. Braz Dent J. 2004; 15:243–247. Epub 2005 Mar 18.
9. Petzold R, Zeilhofer HF, Kalender WA. Rapid protyping technology in medicine--basics and applications. Comput Med Imaging Graph. 1999; 23:277–284.
10. "About 3D Systems." 3D Systems. 2009. Web. 1 May 2010.
11. Berman, A. "3-D Printing Making the Virtual Real." EDUCAUSE Evolving Technologies Committee. 2007. Web.1 May 2010.
12. Casey, Linda. "3D Printing is Reshaping Package Design and Prototyping." Packaging Digest. 2009. Web. 28 May 2010.
13. Cohen, A. "Print Future of Dentistry: 3-D Printing." Dental Health Magazine. Free Dentistry Information and Dental News for Patients. 26 Jan. 2010. Web. 3 May 2010.
14. Cush, J. "HP Partners with Stratasys for 3-D Printers." Laser, Wireless and Inkjet Printer Reviews. 20 Apr. 2010. Web. 2 May 2010.
15. Cygnus Business Media Site-Printing News. "3D Modeling: Using the Latest Innovation in Print." 27 May 2009. Web 6 June 2010.
16. Dynodan Print Solutions. "Printing History." 2010. Web. 1 May 2010.
17. Lozo, B., Stanic, M., Jamnicki, S., & Poljacek, SM. "Three-Dimensional Ink Jet Prints-Impact of Infiltrants." The Journal of Imaging Science and Technology. 2008. 52 5 (52), 1-8.
18. Kurenov S N, Ionita C, Sammons D, Demmy T L. Three-dimensional printing to facilitate anatomic study, device development, simulation, and planning in thoracic surgery. J Thorac Cardiovasc Surg 2015; 149: 973–979.
19. Adibi S, Zhang W, Servos T, O'Neill P. Cone beam computed tomography for general dentists. Open Access Scientific Reports 2012; 1: 519.
20. Scarfe W C, Farman A G, Sukovic P. Clinical applications of cone-beam computed tomography in dental practice. J Can Dent Assoc 2006; 72: 75–80.
21. Hatcher D C, Dial C, Mayorga C. Cone beam CT for pre-surgical assessment of implant sites. J Calif Dent Assoc 2003; 31: 825–833.
22. Sinn D P, Cillo J E Jr, Miles B A. Stereo lithography for craniofacial surgery. J Craniofac.Surg 2006; 17: 869–875.

23. Van Assche N, van Steenberghe D, Guerrero M E et al. Accuracy of implant placement based on pre-surgical planning of three-dimensional cone-beam images: a pilot study. Clin Periodontol 2007.
24. Logozzo S, Zanetti E M, Franceschini G, Makynen A. Recent advances in dental optics – Part I: 3D intraoral scanners for restorative dentistry. Opt Laser Eng 2014.
25. Akyalcin S, Cozad BE, English J D, Colville C D, Laman S. Diagnostic accuracy of impression-free digital models. Am J Orthod Dentofacial Orthop 2013; 144: 916–922.
26. Kruth J P, Vandenbroucke B, Van Vaerenbergh J, Naert I, Digital manufacturing of biocompatible metal frameworks for complex dental prostheses by means of SLS/SLM. pp 139–145. Proceedings of 2nd Int. Conf. on Advanced Research in Virtual and Rapid Prototyping, Leiria, 2005.
27. Birnbaum N S, Aaronson H B. Dental impressions using 3D digital scanners: virtual becomes reality. Compend Contin Educ Dent 2008; 29: 494, 496, 498–505.
28. Tuncay O. The Invisalign System. New Malden: Quintessence Publishing Co., Ltd, 2006.
29. Ciuffolo F, Epifania E, Duranti G et al. Rapid prototyping: a new method of preparing trays for indirect bonding. Am J Orthod Dentofacial Orthop 2006; 129: 75–77.
30. Utela, B., Storti, D., Anderson, R., & Ganter, M. “A Review of process development steps for new material systems in three dimensional printing.” Journal of Manufacturing Processes, 2008. Print. 1-9. 2010.
31. Teriman, D. “3-D Printing Changing Prosthetics Forever.” Cnet News. 2010. Web. 1 May 2010.
32. Laser Prototypes Europe Ltd. Stereolithography (SLA)- The Process. 2010. Web. 2 May 2010.
33. Webber RL, Horton RA, Tyndall DA, Ludlow JB. Tuned-aperture computed tomography (TACT). Theory and application for three-dimensional dento-alveolar imaging. Dentomaxillofac Radiol. 1997;26:53–62.
34. Ucar FI, Sekerci AE, Uysal T, Bengi AO. Standardization of records in orthodontics. Part 2: Craniofacial imaging techniques. Turkish Journal of Orthodontics. 2012;25:167–87.
35. Hajeer MY, Millett DT, Ayoub AF, Siebert JP. Applications of 3D imaging in orthodontics: Part 1. J Orthod. 2004;31:62–70.
36. Plooij JM, Maal TJ, Haers P, Borstlap WA, Kuijpers-Jagtman AM, Bergé SJ. Digital three-dimensional image fusion processes for planning and evaluating orthodontics

and orthognathic surgery. A systematic review. Int J Oral Maxillofac Surg. 2011;40:341–52.

37. Mavili ME, Canter HI, Saglam-Aydinatay B, Kamaci S, Kocadereli I. Use of three-dimensional medical modeling methods for precise planning of orthognathic surgery. J Craniofac Surg. 2007;18:740–7.
38. Karadeniz EI, Gonzales C, Elekdag-Turk S, Isci D, Sahin-Saglam AM, Alkis H, et al. The effect of fluoride on orthodontic tooth movement in humans. A two-and three-dimensional evaluation. Aust Orthod J. 2011;27:94–101.
39. Andresen V. Three contributions to orthodontological diagnosis. International Journal of Orthodontia, Oral Surgery and Radiography. 1926;12:235–51.
40. Van Loon J. A new method for indicating normal and abnormal relations of the teeth to the facial lines. Dental Cosmos. 1915;57:1093–101.
41. Schwarz R. New cephalometric method and apparatus and its application to orthodontia. Int J Orthod Oral Surg Radiol. 1925;11:989–1017.
42. Herman GT. 2nd ed. New York: Springer; 2009. Fundamentals of computerized tomography: Image reconstruction from projection; pp.
43. Scarfe WC, Farman AG, Sukovic P. Clinical applications of cone-beam computed tomography in dental practice. J Can Dent Assoc. 2006;72:75–80.
44. Ritman EL. Micro-computed tomography-current status and developments. Annu Rev Biomed Eng. 2004;6:185–208.
45. Paddock SW, Eliceiri KW. Laser scanning confocal microscopy: History, applications, and related optical sectioning techniques. Methods Mol Biol. 2014;1075:9–47.
46. Fechteler P, Eisert P, Rurainsky J. Fast and high resolution 3D face scanning. (III-4).ICIP. 2007;3:III–81.
47. Ras F, Habets LL, van Ginkel FC, Prahl-Andersen B. Quantification of facial morphology using stereophotogrammetry-demonstration of a new concept. J Dent. 1996;24:369–74.
48. Ferrario VF, Sforza C, Poggio CE, Serrao G. Facial three-dimensional morphometry. Am J Orthod Dentofacial Orthop. 1996;109:86–93.
49. Edelman RR, Hesselink J, Zlatkin M. Saunders; 2005. Clinical magnetic resonance imaging: 3-volume set.
50. Broadbent BH. A new x-ray technique and its application to orthodontia. Angle Orthod. 1931;1:45–66.

51. Athanasiou AE. London: Mosby-Wolfe; 1997. Orthodontic cephalometry; pp. 241–92.
52. Nalçaci R, Oztürk F, Sökücü O. A comparison of two-dimensional radiography and three-dimensional computed tomography in angular cephalometric measurements. Dentomaxillofac Radiol. 2010;39:100–6.
53. Burke PH, Beard FH. Stereophotogrammetry of the face: A preliminary investigation into the accuracy of a simplified system evolved for contour mapping by photography. Am J Orthod. 1967;53:769–82.
54. Baumrind S. Towards a general model for clinical craniofacial research. In: Hunter WS, Carlson DS, editors. Essay in Honor of Robert Moyers. Ann Arbor: The University of Michigan; 1991.
55. Han UK, Vig KW, Weintraub JA, Vig PS, Kowalski CJ. Consistency of orthodontic treatment decisions relative to diagnostic records. Am J Orthod Dentofacial Orthop. 1991;100:212–9.
56. Hixon EH. The norm concept and cephalometrics. Am J Orthod. 1956;42:898–906.

Evolution of Facial Muscles in Humans in Comparison to Other Organisms-A Review

Tamizh Paavai.Tha[1], Yuvraj Babu[2], Iffat Nasim[3]
Graduate student[1], Reader[2],Department of Anatomy
Reader[3],Department of Conservative Dentistry and Endodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Evolution is a slow, steady and a progressive process that brings about vital changes in various organisms with time to adapt to the environmental changes they encounter. The communication between organisms or individuals primarily involves facial expressions. Therefore this review article focuses on the evolution of facial muscles of humans with other organisms such as monkeys to study their adaptive pattern with time to various stresses.

Key Words: *Chimpanzee, Evolution, Facial muscles, Human, Mammals*

Introduction

The mammalian facial muscles are innervated by the facial nerve (seventh cranial nerve) and are a subgroup of hyoid muscles; they are inserted into the freely movable skin and produce facial expressions [1]. They are responsible for generating facial expressions during interactions and also play a role in various activities such as feeding, chemo sensation, hearing and speech. It is observed that the facial muscles attach via a subcutaneous musculoaponuerotic sheath unlike the vertebral muscles that attach via tendons.

The facial muscle of various organisms was studied using a new method used by Burrows and colleagues known as the 'face mask' technique wherein the superficial facial musculature, skin and fascia are removed from the skull. It is suggested that this method helps to preserve the facial musculature more effectively than the traditional method used earlier.

On comparing the facial muscles of humans with that of other organisms it is observed that modern humans possess varied facial and laryngeal muscles that are absent in majority of the other mammalian taxa [2]. Various dissections have proven distinct similarities between humans and chimpanzees in accordance to facial muscles[3].

The *Panthera tigris* facial muscles show certain similarities as well as differences when compared with human facial muscles [4] .It was also observed that many muscles that were absent in chimpanzees were present variably in humans [5][6]. Various studies prove the hypothesis that social and environmental variables have a great influence on the evolution of facial musculature in hylobatids, which is closely related to chimpanzees [7].

Humanvs*Panthera tigris*

Tigers occupy a wide range of habitats from mountain slopes to mangrove swamps and tropical rainforests [4], in particular, not much is known about the muscles of head and neck in tigers. Recent studies have helped us realize the various similarities and differences in facial muscles with comparison to that of humans. In tigers the masseter is divided into a ventral bundle, a superficial bundle and a deep bundle. However, a ventral bundle is not present as a distinct structure in humans [4][8].

According to a study conducted by Saban in the year 1968, the pars suprazygomatica of the temporalis is often present in the Carnivora also including domestic cats, and in

other mammals such as rodents and some primates but not in modern humans; this structure is effectively well developed in our serval and tiger, being similar to that reported in *Puma* by Schumacher (1961)[8][9][10].

The body of the temporalis into a deep, posterior bundle and an anterior, superficial bundle, this is a feature commonly found in most groups of carnivorans and other mammals, being often present in modern humans [8][9]. However, the broad attachment of the latter bundle onto a broad area of the orbital part of the frontal bone lying just posteriorly to the orbit, found in our serval and tiger, is a feature less commonly found within mammals and are not present in mammals [8][11].

The intermandibularis anterior is usually not present as a distinct muscle and the digastricus anterior is connected to the digastricus posterior by a tendinous intersection and not by a true tendon as seen in a few mammals that also modern humans [12].

A controversy about the presence or absence of a pterygoideus lateralis in carnivorans has been there for a long time wherein some authors defended that the muscle is usually completely missing in these mammals, others defended that it is present but displays a peculiar fusion with the pterygoideus medialis, and still others proposed that the two muscles are actually completely separable [8]. However, further studies by authors suggested that pterygoideus lateralis and the pterygoideus medialis are effectively deeply blended, and that the former muscle is undivided, as found in other felids such as domestic cats i.e. it is not differentiated into distinct superior and inferior heads as is often the case in various mammalian taxa, including modern humans [9].

When the hyoid muscles were studied, it was observed that the muscle originates from the skull, jugulohyoideus and/or proximal part of the hyoid apparatus and attaches onto a strong aponeurosis that indirectly connects it to the hyoid apparatus in case of tigers. But the modern humans the stylohyoideus originates from the styloid process and splits around the intermediate digastric tendon to insert onto the body of the hyoid bone [8][13].

Examples of other differences between modern humans and our serval and tiger include the following wherein the former the sphincter colli profundus,

interscutularis, levator anguli oculi medialis, retractor anguli oculi lateralis, platysma cervicale, and sphincter colli superficialis are usually not present, and the depressor supercilii, depressor septi nasi, depressor labii inferioris and depressor anguli oris are usually present, as distinct muscles [9].

It is also observed that similar to domestic cats, tigers have more facial muscle structures associated with the mobility of the auricular region than that of modern humans and most other primates, which usually only have three mainly undivided facial muscles connecting the ear to the skull, i.e., the auricularis anterior, auricularis superior and auricularis posterior [14].

Further when the branchial muscles are studied it is seen that the muscles stylopharyngeus and ceratohyoideus are seen in tigers whereas modern humans are observed to lack ceratohyoideus but possess stylopharyngeous[9].

Some other differences between tigers and humans are that the levator depressor septi nasi, depressor supercilli, depressor labii inferioris and depressor angulioris are usually present, as distinct muscles and also sphincter colli profundus, interscutularis, levator anguli oculi medialis, retractor anguli oculi lateralis, platysma cervicale, and sphincter colli superficialis are usually not present [9].

Humans vs other Therian mammals

It is observed that humans have a lesser number of mandibular muscles when compared to that of other mammals such as tree shrews, rats and fewer muscles than that of reptiles such as lizards [2]. Recent studies have rendered the cause for this phenomenon by establishing three reasons. Firstly, most of the evolutionary splitting that occurred during transitions has led to the origin of amniotes and mammals [2]. Further, some muscles that were present in our ancestors were lost during various stages of evolution [2]. Lastly, some of the muscles found in the sarcopteygian taxa are peculiar and features of that taxa[2]. It is observed that the number of laryngeal and facial muscles found in other mammals is much lesser than those seen in modern humans [2]. Excluding the extrinsic muscles in the ears, the modern humans have 24 facial muscles where as researchers have observed only 20 in case of rodents such as

rats and tree shrews such as *Tupaia* have 21. In case of laryngeal muscles, there are 4 muscles in *Tupaia* and rats whereas 6 muscles are present in human beings.
Many studies also suggest that the number of true branchial muscles found in modern humans are smaller than that found in most other mammals. Actually, in the case of these muscles if there is a 'trend' at the time of the evolutionary transitions that led to the origin of primates and subsequently to modern humans, then it is to reduce, and not increase, the total number of muscles. It is observed that the monotremes like platypus have 8 muscles; followed by rodents like rats with 7, Tree shrews like Tupaia have 6 muscles and the modern humans are observed to have 3 muscles [9].

In case of pharyngeal muscles, there is an increase in the number of muscles when evolutionary change that lead to therian mammals occurred but no increase was observed when an evolutionary transition lead to the existence of modern humans. It is observed that the platypus generally have 3 muscles, rodents like rats have 8 muscles, colugos have 8 muscles tree shrews such as Tupaia have 7 muscles while modern humans have 7 muscles [9].

On studying the hypobranchial muscles that does not include the intrinsic muscles of the tongue, it is observed that the modern humans have more muscles than the other taxa. For example, rodents like rats have 8 muscles, colugos have seven muscles, and tree shrews like Tupaia have 8 muscles while the modern humans have nine muscles [9].

Many studies were also conducted on the facial and laryngeal muscles, which shoed that humans possessed more muscles that any other taxa. Modern humans usually have 24 facial muscles without including the extrinsic muscles of the ear, monotremes like the platypus have 10 muscles, rodents such as the rats have 20, colugos have 19 muscles while the three shrew such as the Tupaia have 21 muscles [9].

Examples of facial muscles present in modern humans and which are lacking in most other mammals are the risorius, depressor supercilii, levator labii superioris alaeque nasi, depressor septi nasi, depressor labii inferioris and depressor anguli oris [15][16][17].

Human vs. Chimpanzee

Chimpanzees (*Pan troglodytes*) have an elaborate repertoire of facial signals, but little

is known about their facial expression (i.e. mimetic) musculature underlying these movements, especially when compared with some other catarrhines. It is observe that there are minimal anatomical differences between chimpanzees and humans.

One of the major differences found in a study conducted by Burrows on *P. troglodytes* and humans was the firm fusion and, often, intimate infiltration of the superficial fascia into some of the muscles, such as the deep head of the occipitalis muscle, the zygomaticus major muscle, and the depressors anguli oris and labii inferioris muscles. It was observed that in these muscles the superficial fascia was firmly blended with the muscle fascicles and slowed progression of the dissection. However, the relationship between the superficial fascia and musculature in human facesshowed the superficial fascia typically lies only loosely on top of the muscle [18].

Similar to modern humans, the facial expression musculature in *P. troglodytes* are observed to be thickest and most numerous in the area of the oral cavity. These organisms live inloose dispersed communities where the large group may frequently break out into numerous small groups to interact with other groups and then reunite with their old community [19]. Males are generally dominant with a clear dominance hierarchy and frequent territorial disputes [20]. In these intricate social settings, a variety of vocal, chemical and visual communication modes are employed to send information on social intentions, emotional states, and various aspects of an individual such as age, sex and reproductive status [21]. *P. troglodytes*are reported to use a number of facial expressions to communicate various intentions [20]. The silent bared-teeth and relaxed open-mouth displays are the most common facial expressions seen in social contexts which involve the lips, and very few displays are noted to include movements of the orbital region, scalp or pinna. The preponderance of musculature associated with the oral region may indeed reflect these behavioralobservations for *P. troglodytes* [22].

Human vs. Rhesus macaque

The rhesus macaque is one of the least-studied primates on the basis of facial musculature and this is considered as an essential exercise as these monkeys are used as models for understanding various human diseasesand abnormal emotional and social behaviors such as Parkinson’s disease, autism, AIDS and schizophrenia.

[24].

Macaques as a genus [Cercopithecoidea: Cercopithecidae] exhibit a number of social styles. They have the ‘tolerant’ species such as stump-tailed macaques (*M. arctoides*), Tonkean macaques (*M. tonkeana*) and lion-tailed macaques (*M. silenus*). These species are characterized by relatively relaxed and egalitarian dominance styles with tolerance toward subordinates, low levels of aggression, and high levels of reconciliation and affiliation. Social interactions between and among conspecifics tend to be bidirectional. They also have another extreme that are the ‘despotic’ species such as crab-eating macaques (*M. fascicularis*), pig-tailed macaques (*M. nemestrina*) and rhesus macaques (*M. mulatta*). A rigid dominance hierarchy characterizes these species with little tolerance toward subordinates, unidirectional and high levels of severe aggression and low levels of reconciliation in females. Any affiliations tend to be kin-based [24].

Muscle	**Rhesus Macaque**	**Chimpanzee**	**Humans**
Platysma	P	P	P

In both tolerant and despotic species individual must communicate with one another and this is done mainly via vocalizations and visual displays such as facial expressions. Both the vocal and facial display repertoires of some macaque species are relativelywellunderstood, especially in rhesusmacaques (*M. mulatta*). Facial displays and vocalizations in this species conveyinformationrelatedto the rank of the sender, individual identity, reproductivestatus and emotional state/intent of the signaler [17]. As in humans and chimpanzees, rhesusmacaquesintegrate vocalizations and facial displays and mayusethesemodes both individually and simultaneously.

Occipitalis	P	P	P
Frontalis	P	P	P
Superior auricularis	P	P	P
Posterior auricularis	P	P	P
Anterior auricularis	P/V	P	P
Inferior auricularis	P/V	A	A
Orbitoauricularis	P	A	A
Tragicus	P	P	P
Antitragicus	P	A	P/V
Orbicularisocculi	P	P	P
Orbicularis oris	P	P	P
Mentalis	P	P	P
Levator labii superioris	P	P	P
Depressor septi	P/V	P	P/V
Corrugatorsupercillii	P	P	P
Depressorsupercillii	P/V	P	P
Procerus	P	P	P
Levator labii superioris alaeque nasi	P/V	P	P
Caninus	P	P	P
Depressor anguli oris	P	P/V	P
Zygomaticus major	P	P	P
Zygomaticus minor	P/V	P	P/V
Risorius	A	P	P
Depressor labii inferiori	P	P	P

The most common facial display of *M. mulatta*that is well understood and documented extensively are the 'silent-bared teeth' and the 'relaxed open-mouth' . These displays have been cited as homologues to human laughter and smiling, respectively. Thus, an increased understanding of rhesus macaque facial display behaviors may inform our understanding of the evolution of human communication and social behavior [17]. The following table shows a comparison of muscles between Rhesus Macaque, Chimpanzee and modern humans [Table 1] [24][25].

Table 1: This table compares the facial musculature of Rhesus macaque with Chimpanzee and modern humans.

Key - P- Present; A- Absent; P/V- Muscle was variably present

Thus the results of various studies suggest that the rhesus macaque share a more similar facial musculature with chimpanzees and modern humans than it was previously considered.

Human vs. Hylobatids

Hylobatids, which include the gibbons and siamangs [Primates: Hominoidea: Hylobatidae), are small, arboreal, and territorial lesser apes found throughout the

evergreen forests of southeast Asia including Indonesia, Eastern India, Vietnam, Laos, Cambodia, and Southern China Together with humans and the great apes (chimpanzees, bonobos, gorillas, and orangutans) they comprise the superfamily Hominoidea [27]. During evolution of the hominoids, it is said that the hylobatids were the first to branch off, over 17 million years ago [28][29].

A total of 22 muscles were present when not counting the buccinatorius muscle, which is not typically involved in facial expressions. The depressor septi nasi muscle was found to be variable in two of the four specimens of hylobatids. The hylobatids, the phylogenetically distant rhesus macaque, and the more closely related chimpanzee have roughly the same number of mimetic muscles. One of the primary differences among these three taxa is that the risorius muscle is only consistently present in chimpanzees (as it is in humans and gorillas), and not in hylobatids and rhesus macaques [26].

Complexity of facial musculature in hylobatids in various studies conducted by Burrows suggested them to be mixed. In the musculature of the external ear, hylobatids had poor separation of these muscles from the occipitalis, frontalis, and auriculo-orbitilis muscles by way of intersecting fascial connections. This series of connections would, by definition, decrease complexity of the musculature and possibly the ability to move the external ear independently from the scalp. The procerus muscle was poorly separated from the frontalis muscle in hylobatids, which may decrease the ability to move the skin over the external nose separately from the skin of the superciliary region. In addition, the zygomaticus major and depressor labii inferioris muscles were exceptionally gracile relative to both the chimpanzee and rhesus macaque [26]. This study conducted by Burrows also mentioned a double-headed zygomaticus minor muscle in hylobatids, which may be viewed as having great complexity relative to the chimpanzee and rhesus macaque [26].

Micro anatomically, the hylobatids when examined the upper lip was found with approximately even distributions of connective tissue and muscle fibers, unlike the chimpanzee and rhesus macaque, which had relatively densely packed muscle fibers and very little connective tissue [30]. The present study also suggests that there was no indication of separate pars marginalis and pars peripheralis layers in the hylobatid orbicularis oris muscle, a contrast with the chimpanzee [30].

Thus the hypothesis derived from these studies suggests that environmental and social variables are influential in the evolution of morphology and complexity of facial musculature in hylobatids. Duetting (and loud vocalizations in general) is one of the key characteristics defining hylobatids and seems to be the primary mechanism of communicating with con- specifics outside of the small ‘‘family’’ group .It is suggested that the process and evolutionary mechanisms of hylobatid duetting would possibly have decreased selection pressure on development of facial displays and facial musculature complexity relative to both the distantly related rhesus macaque and the closely related chimpanzee [26].

Conclusion

Thus from the above review we can conclude the various evolutionary changes in the facial musculature of human beings when compared to various other mammalians and non-mammalians. This data will help in various experimental studies done to study the progression of a disease in humans with the other mammals or non-mammals as models. The researcher must be well aware of the similarities and variations present between humans and the model organisms to make a proper hypothesis and analyze the condition tested effectively[31][32]. It is also evident from this review that more evaluation of the facial musculature of various organisms is necessary as multiple investigations help in identifying many variants of the musculature that were not mentioned in the earlier studies. These studies also help us predict the future evolutionary pattern of the humans which the influence of social and environmental changes they are subjected to. This data helps various comparative anatomists in analyzing the musculature of the different evolutionary species of the same and various taxa.

Corresponding Author

Iffat Nasim

Reader,Department of Conservative Dentistry and Endodontics

Saveetha Dental College,

Saveetha University, Saveetha Institute of Medical and Technical Sciences

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

E-Mail: nasimiffat@gmail.com

Telephone number: +9940063567

References

1. RDiogo, B A Wood, M A Aziz, and A Burrows,On the origin, homologies and evolution of primate facial muscles, with a particular focus on hominoids and asuggested unifying nomenclature for the facial muscles of the Mammalia; J Anat. 2009 September; 215(3): 300–319
2. Diogo R, Abdala V, Lonergan N, Wood BA;From fish to modern humans--comparative anatomy, homologies and evolution of the head and neckmusculature.;J Anat. 2008 Oct;213(4):391-424.
3. Burrows, Waller BM, Parr LA, Bonar CJ, Muscles of facial expression in the chimpanzee (Pan troglodytes): Descriptive, comparative and phylogenetic contexts. Journal of Anatomy. 2006; 208:153–168
4. Rui Diogo,, Francisco Pastor, Felix De Paz, Josep M. Potau, Gaëlle Bello-Hellegouarch, Eva M. Ferrero, Rebecca E. Fisher; The Head and Neck Muscles of the Serval and Tiger: Homologies, Evolution, and Proposal of a Mammalian andaVeterinary Muscle Ontology Article first published online: 7 SEP 2012
5. Waller BM, Parr LA, Bonar CJ. Muscles of facial expression in the chimpanzee (Pan troglodytes): Descriptive, ecological, and phylogenetic contexts. J Anat. 2006; 208: 153–167.
6. Standring S.; Gray's Anatomy. 39th edn. London: Churchill Livingstone; 2004.
7. Anne M. Burrows, RuiDiogo, Bridget M. Waller, Christopher J. Bonar, Katja Liebal; Evolution of the Muscles of Facial Expression in a Monogamous Ape: Evaluating the Relative Influences of Ecological and Phylogenetic Factors in Hylobatids Article first published online: 2 MAR 2011
8. Saban R.. Musculature de la tête. In: Grassé PP, editor. Traité de Zoologie, XVI: 3 (Mammifères). Paris: Masson et Cie.1968 p 229–472.
9. Diogo R, Wood B..Comparative anatomy and phylogeny of primate muscles and human evolution. Oxford: Taylor and Francis.2012
10. Schumacher GH.. Funktionelle morphologie der kaumuskulatur. Jena: VEB

Gustav Fischer Verlag. 1961

11. Davis BW, Li G, Murphy WJ.. Supermatrix and species tree methods resolve phylogenetic relationships within the big cats, Panthera (Carnivora: Felidae). Mol Phylogenet Evol 201056:64–76.

12. Edgeworth FH. The Cranial Muscles of Vertebrates. Cambridge: University Press; 1935.

13. Weissengruber GE, Forstenpointner G, Peters G, Ku¨bber-Heiss A, Fitch WT. Hyoid apparatus and pharynx in the lion (Panthera leo), jaguar (Panthera onca), tiger (Panthera tigris), cheetah (Acinonyx jubatus) and domestic cat (Felis silvestris f. catus). J Anat 2002.201:195–209.
14. Gibbs S.. Comparative soft tissue morphology of the extant Hominoidea, including man. Unpublished PhD Thesis, The University of Liverpool.1999

15. Huber E, Evolution of Facial Musculature and Expression. Baltimore: The Johns Hopkins University Press; 1931.

16. Burrows AM The facial expression musculature in primates and its evolutionary significance. Bioessays30, 2008212–225.
17. Preuschoft S Primate faces and facial expressions. Soc Res 67, 2000, 245–271.
18. Stranding S Gray's Anatomy, 39th edn. London: Churchill Livingstone2004
19. Nishida T The social structure of chimpanzees of the Mahale Mountains. In: The Great Apes (eds Hamburg DA, McCown ER),. Menlo Park, CA: Benjamin- Cummings. 1979, pp. 73–121
20. Goodall J The Chimpanzees of Gombe: Patterns of Behavior. Cambridge: Harvard University Press 1986.

21. Burrows AM, Waller BM, Parr LA. 2009. Facial musculature in the rhesus macaque (Macaca mulatta): evolutionary and functional contexts with comparisons to chimpanzees and humans. J Anat 215: 320–334.
22. Parr LA, Hopkins WD, de Waal FBM The perception of facial expressions by chimpanzees, Pan troglodytes. Evol Comm 2,1998, 1–23.

23. Facial musculature and its innervation. In: Hartman CG, Straus WL Jr, editors. Anatomy of the Rhesus Monkey. New York: Hafner Publishing Co; 1933. pp. 176–188

24. Anne M Burrows. Bridget M Waller, and Lisa A Parr; Facial musculature in the rhesus macaque (Macaca mulatta): evolutionary and functional contexts withcomparisons to chimpanzees and humans; J Anat. 2009 September; 215(3): 320–334.

25. Fleagle JG.. Are there any fossil gibbons? In: PreuschoftH, ChiversDJ, BrockelmanWY, CreelN, editors. The lesser apes. Evolutionary and behavioural biology. Edinburgh: Edinburgh University Press.1984 P 431–447.
26. Burrows, A. M., Diogo, R., Waller, B. M., Bonar, C. J. and Liebal, K. (2011), Evolution of the Muscles of Facial Expression in a Monogamous Ape: Evaluating the Relative Influences of Ecological and Phylogenetic Factors in Hylobatids. Anat Rec, 294: 645-663
27. Groves C.. Order primates. In: Wilson DE, Reeder DM, editors. Mammal species of the World: a taxonomic and geographic reference. Vol.1. Baltimore: The Johns Hopkins University Press. 2005p 111–184

28. Pilbeam DR..Genetic and morphological records of the Hominoidea and hominid origins: a synthesis. Mol Phylogenet E1996vol 5: 155–168.

29. Cunningham C, Mootnick A. 2009. Gibbons. Curr Biol 19: R543–R544.
30. Rogers CR, Mooney MP, Smith TD, Weinberg SM, Waller BM, Parr LA, Docherty BA, Bonar CJ, Reinholt LE, Deleyiannis FW-B, Siegel MI, Marazita ML, Burrows AM. 2009. Comparative microanatomy of the orbicularis oris muscle between chimpanzees and humans: evolutionary divergence of lip function. J Anat 214:36– 44.
31. Selvaraj L, Rao SH, Lankupalli AS. Comparison of efficacy of methylprednisolone injection into masseter muscle versus gluteal muscle for surgical removal of impacted lower third molar. Journal of maxillofacial and oral surgery. 2014 Dec 1;13(4):495-8.

32. D'dharan SR, Kumar MS. Facial Palsy-A Case Report. Journal of Pharmaceutical Sciences and Research. 2016 Oct 1;8(10):1206

Failures In Composite Restoration – A Review

Mohammed Ahad [1], Remmiya Mary Varghese[2]

Graduate student [1] ,Senior lecturer [2],Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Aim: To review common causes of failure in composite restoration

Background: In recent years, the demand of tooth colored restorative material and its advantages has led to a rapid increase in the use of resins. The steady growth of interest in 'cosmetic' treatments led to increase in use of composite fillingswhich are naturally an attractive alternative to silver amalgam restorations, particularly in clinical situations where the restorations would otherwise be visible. This review is based on the failures of composite restoration and problems faced by dentist and patient. Some of the major problems are shrinkage of resins, post operative sensitive, fracture of restoration, wear, inadequate finishing and polishing etc

Reason: This review paper will be useful in understanding the problems related to the failures of composite restoration

Key words:*Composite, failures, shrinkage, wear, review*

Introduction

As per McLean, the main endeavour to create plastic restorative materials was uncovered in the Allied Field Information Technical Report No. 1185, distributed in 1947. A cold cured acrylic resin for use in restorative dentistry was produced in Germany [1]. The self-cured, unfilled acrylic materials could be put specifically into the prepared tooth. The polymer and monomer were combined and embedded into the pit where it polymerized. In any case, these amine-containing resins were not shading steady and turned dull on introduction to sunlight. They additionally had issues of over the excessive working time to set initially (1.5 minutes), poor compressive quality, low grating protection, low modulus of versatility, high water absorption and a polymerization shrinkage of 7% by volume. Their high coefficient of thermal expansion additionallypredis-postured they to miniaturized scale spillage and the issues related with microleakage [2]. In 1958, the main composite resin material, P-Cadwrit, was made accessible in Germany. In 1959, Bowen recorded his first patent in the U.S.A. on the renowned Bis-GMA resin. The upsides of composite resins in light of Bis-GMA resin over an acrylic resin include: bring down polymerization shrinkage, non-volatile, bring down exothermic properties, more prominent compressive quality and less lethal to the pulp [3]. Knight et al built up the urethane dimethacrylates in 1973. A resin was made for use in composite dental materials along these lines which have points of interest of higher molecular weight, lower viscosity and less in vivo stain-in with use than Bis-GMA (l) resins.

According to Lutz et al [4], filled remedial resin consists of three-dimensional mixes of at least two synthetically extraordinary materials with a surface interfacial stage. The 3 stages are: the matrix stage, the surface interfacial stage, and the dispersed stage. Every resin "should likewise incorporate a quickening agent initiator framework to start and finish polymerization. The artificially cured composites by and large utilize an amine-peroxide framework, though the light-cured resin utilizes a diketone-amine framework which is actuated by the extraordinary blue light. What's more, pigments and opaquers are added to control translucency and shade. The resin framework is a dimethacrylate oligomer such Bis-GMA or urethane-diacrylate. The surface interfacial stage comprises of either a bipolar coupling specialist to tie the natural resin grid to the inorganic fillers, or copolymeric of homopolymeric bond between the natural Fine-Particle measure Composite Resins network and fractional organic filler. The level of interfacial bond and compound solidness is basic for fruitful clinical utilization of any composite resin [5]. Lutz et al [4] grouped the dispersed stage in view of the three noteworthy classes of filler particles utilized. Conventional macrofillers comprise of quartz, glass, borosilicate, and ground or smashed ceramic. The measurement of macrofillers particles go from 0.1 to 100A.055/lm. Microfillers are normally pyrogenic silica which is indistinct, finely light cured. Contrasted with the large particle composite dispersed particles of around 0.04A.05.5/lm in estimate. The resins, they furnish a smoother restorative surface with micro filler-based complexes are generally one of three less surface degeneration, better shading solidness and higher writes: [1] fragmented prepolymerized particles of I to 200A.05 5/lm in estimate, [2] circular prepolymerized particles of 20 to 30A.055/lm in width, and [3] agglomerated microfillercomplexes of I to 25A.055/lm in diameter. Self-cured resins arefavourable where compos-degeneration at the

filler-matrix interface would in any case composite resin should be put in regions of the mouth where light can't reach adequately [2] Be that as it may, the visible light-cured resins have numerous preferences, including: control over the working time, quick completing of resins and cases of rebuilding and control over the profundity of cure. Since no mixing is required, it implies simpler dealing with and insignificant porosity, the real advantage is that the rebuilding will be significantly more shading stable contrasted with self cured resins. Subsequently, most of the composite resins now accessible are light-cured resins [2, 6].

Dental composites consist of a polymerizable resin matrix, particle fillers, reinforcing glass and silane coupling agents [7]. These glass particle/resin matrix composites have good aesthetic properties and strength, making them the most widely used materials for restorations of anterior teeth [8]. Development of modern dental composite restorative materials started in the late 1950s and early 1960s, when Bowen [9] began experiments to reinforce epoxy resins with filler particles.

Since 1990, numerous classifications of resins composites have been presented. Composite Resins have been utilized for almost 50 years and quite a long time upgrades have been made with respect to their composition and their properties. The majority of them contrasted chiefly in the specific filler matrix utilized, e.g. conventional/customary, little molecule composites, matrix (smaller scale hybrid, monohybrid, submicron cross breed and monohybrid) [10]. The consolidation of the filler in the composite material has improved the mechanical properties of the composite, for example, compressive and rigidity, surface hardness or resistance to surface space[11].On the other hand, posterior composite restorations have been shown to produce higher failure rates due to secondary caries [12,13].

Light Curing

Every composite requires a specific measure of energy (joules) for finish curing. This may differ by the manufacturer, by the sort of composite material, and the shade of the composite. It is a component of the energy of the curing light (mW/cm2) and the measure of time that the light is conveyed. Tragically, the measure of delivered energy can fluctuate because of the improper position of the light tip, movement of the light tip amid curing, separation of the light tip from the resin, shade and type of the resin material, state of the light curing unit, or thickness of the resin . Indeed, even the most capable curing light won't cure a composite on the off chance that it isn't legitimately set. Because the best layer of the composite is hard, that does not imply that the composite is cured at the base.[14]

All in all, insufficient consideration is given to legitimate position of the curing light or the state of the light. The best possible measure of irradiance is controlled by the manufacturer and the shade of the composite. Deficient or inadequate curing unfavorably influences the resin physical properties, lessens the bond quality to the tooth, diminishes the biocompatibility of the restoration, increments negligible wear and breakdown, and increments bacterial colonization of the restoration [15] A few reviews have demonstrated that numerous QTH (quartz-tungsten-halogen) curing lights in dental workplaces don't convey enough light energy to totally cure composites[16].

Size and Location of Restoration

The position of the tooth in the curve and the size of the cavity have been appeared to be a factor in the accomplishment of a restoration. One investigation found the risk of failure in the molar region to be twice as high with respect to premolars, [17] while another examination put the failure rate in bring down molars as three times that of the upper premolars. [18] Multi-surfaced restoration efforts are additionally more inclined to failure than single surface restoration. An investigation figured that for each surface added to the restoration, a 40% expansion in the failure rate resulted. [17]

Polymerisation shrinkage

Every single composite resin contract amid polymerization, such withdrawal is named polymerization shrinkage. Polymerization shrinkage of composite resin is critical as a result of its impact on cavosurface edges. It causes partition between a composite resin mass and the adjacent tooth structure. Negligible adjustment of a composite resin rebuilding is reliant on a few variables including: polymerization shrinkage, hygroscopic properties, holding between restorative material and the cavity walls. Coefficient of thermal expansion of the material, and the Finishing methods [19-22]

It has been exhibited that regardless of acid etching of enamel walls, hygroscopic expansion of composite resin, cautious completing strategies, and utilization of material with warm expanxivity like that of enamel, marginal gaps will in any case result from polymerization recoil age. Such shrinkage may cause minor gap development, or when the finish - resin bond stays in place, it might bring about damage inside the composite resins as micro cracks which may cause untimely disappointment of the restoration [23]. The shrinkage properties of composite resins are subject to both the physical components of the materials and how the materials are cured and dealt with clinically. Different composite materials have been appeared to display polymerization shrinkage from around 1.5 - 5.5% by volume [19]. Late investigations report shrinkage of around 1-2% volume for posterior compositeresins contrasted with around 4-5% volume for early ordinary composites [24]. Joining of a high division of filler particles alongside a fitting creation of the monomer matrix hypothetically would give a composite resin the most minimal conceivable polymerization shrinkage. The measure of volumetric change in back composite resins when cured has been expressed as one of the principle determinants of the life span of the composite resin reclamation.

When matrix monomer convert to polymer Composite shrinks immediately upon setting (2-3% by volume) stresses are invariably generated within the material at the margins on shrinking [25]. Larger the increment of compo- site, greater the total shrinkage, this will again increases the potent- tail for stress formation [26]

Consequence of polymerisation shrinkages

i. Weak bonds can occur due to polymerisation shrinkage this causes failure of composite in bonding with the tooth structure

ii. Post-operative sensitivity [26]

iii. Marginal gap formation

iv. Ingress of bacteria and secondary caries [26]

v. The contraction forces transmitted to enamel and dentin, causing cusp flexure, fracture or crazing of enamel and fracture in composite material [27].

Appropriate Placement Technique

Numerous specialists suggest incremental arrangement of the composite in light of the abatement in the "C" factor and the resultant reduction in the shrinkage stress. [28] This has been the prescribed strategy; in any case, with the presentation of the low shrinkage stretch composite materials, mass filling has turned out to be better known. Due to their low stresses and great profundity of cure, these composites might be what have to come. Regardless of which procedure that is utilized, legitimate matrix placement is critical for the restoration achievement. A restoration that has an open contact or a gingival overhanging isn't a worthy rebuilding. Nourishment impaction from an open contact does not prompt great gingival wellbeing and an open or rough edge may prompt bacterial development and possible intermittent decay. Sectional matrix frameworks and isolating rings may lead better shapes, contacts, and marginal seal.

Proximal Box Placement

Bonding failures have been usually credited to the gingival margins of Class II composite restorations. [29] In the proximal box of most Class II restoration, there is practically zero polish at the edge for bonding. Holes at the gingival edges have been ascribed to the potential for poor bonding at this margin.[30] The disparity between etching depth and adhesive invasion prompts a substantial region of exposed collagen at the gingival margin.[31] Another variable that may meddle with the bonding in the gingival region is water content. Expanded water content prompts lessened adhesive invasion and lower monomer/polymer transformation of the adhesive at the gingival edge when contrasted with the proximal wall. [32] Sometimes, the situation of a glass ionomer as the principal layer for these deep margins might be considered due to a portion of the great characteristics of this material [33].

Water Absorption

The specialized properties of composite resin are influenced by ingestion of water, which goes about as plasticizer and pressure consumption operator, debilitating the molecule network interfaces. Confined swelling happens at the filler-lattice between confront causing deboning, which may prompt hydrolytic breakdown. Separate on the surface of composite pitches may likewise be encouraged by temperature changes and dissolvable impacts. The

higher the temperature, the more fast the water absorption [34]. The measure of water ingestion in posterior composite resins utilized today is around 0.2 - 0.6% by weight. Water retention will prompt breakdown of the composite resin with utilize.

Wear

Wear might be characterized as the undesirable expulsion of strong material from surface because of mechanical action[35]. The customary vast molecule measure composite resin contains expansive filler particles which are impressively harder than the resin network. Amid rumination, stresses are transmitted onto the rebuilding surface and especially the particles anticipating from the occlusal surface. Since the particles are harder than the resin network in which they are implanted, a great part of the pressure is transmitted through the molecule into the resin itself. Stress will think and turn out to be too much high where the submerged portion of the molecule is angulated or irregular shape as a fiddle. Such a condition has a tendency to create little breaks around the molecule, along these lines debilitating the lattice locally [36]. Another age of composite resin has hence been produced which contain filler particles of decreased sizes yet expanded filler stacking. The measure of stress around every molecule is diminished which result in a huge lessening in loss of anatomical form. In some composite resin, softer filler particles have been incorporated keeping in mind the end goal to diminish the distinction in hardness between the filler and the network. At the point when softer filler particles are utilized, the masticator stresses are in part consumed by the molecule, instead of being completely transmitted into the encompassing matrix [23]the utilization of softer filler particles in this way diminish the likely hood of creating little splits around the filler and debilitating the framework locally.

Shade Matching

Correct shading coordinating of the restoration and the tooth is an imperative part of the aesthetic restoration. Numerous things can confound making the right shade choice, for example, the lighting framework in the operatory, the way that teeth lighten when got dried out, the shade of the operatory or the patient's dress, and the experience of the individual doing the shade determination. Shade choice requires learning of material science and the physiology of shading; subsequently, it is both an art and a science requiring top to bottom knowledge, precise clinical judgment, and discernment with respect to the dentist. [37] Unfortunately, the confinements of shade guides are critical variables that compromise shade matching methodology in dentistry and add to the dissatisfaction of clinicians, specialists, and patients. [38]

Improper manipulation:

i) Acid etching

Etching time is 15-20 sec on permanent tooth whereas its 60 sec on primary tooth, as it is more amorphous & does not form the deep resin tags. Enamel require more etching time as fluoride content is more & it is impervious to etch, The end results of etching that it appears as irregular surface and frosty white owing to light refraction [25]..

ii) AcidStrength

Buonocore utilized 85% phosphoric acid first. However, later examinations said higher concentration are less effective & are more likely to denude surface, so research suggest 37% phosphoric acid is the ideal concentration [39].Care ought be taken that acid should replenish before use as it evaporates during storage.

i) Under etching

Inability to accomplish a cold surface could come about because of under etching /hypo calcified enamel [39].

ii) Over etching

Can cause an insoluble reaction product monocalcium phosphate that gets dried out which avoids additionally etching and causes feeble bonding [39].

Average time for an adult permanent is 20 sec while for newly erupted Permanent teeth is 15 secs and for deciduous teeth it's 60 – 120 sec. Washing time is about 10 sec, Insufficient washing leaves debris that interferes with the flow of resin. 60 sec washing with heavy water spray actually weak resin- enamel bond as enamel rod crushed [25,39]

Drying

Electric hot air dryers are the most ideal approach to dry an etched enamel surface. They have appeared to enhance enamel bond quality by around 29%. The least desirable is three way syringe / liquid drying [25, 39]

Type of etching material

For pits & tissues, liquid is recommended. For smooth surface etching, liquids & gels results in similar etch patterns [39].

Oil sullying of hand pieces or/Air water syringes

The oil originates from air compressors, a large portion of which are not kept up well in dental workplaces. Any of the present dentin bonding agent combined with oil defilement gives an eccentric clinical outcome and potential clinical disappointment. Expelling all oil ought to be a prompt goal. Air filters should be placed on the air lines. [40]

Isolation and placement of rubber damn

Composite can be easily contaminated by moisture Isolation is necessary during adhesion and bonding of composite resin to tooth structure. Improper isolation causes decreased bond strength & ultimately physical & mechanical properties of compo- site restoration also decreases [26]. Isolation can be done with rubber dam, gingival retraction cords etc. But the placement of rubber dam is most important procedure. For success & longevity of composite restoration Appropriate contour and contacts are important. This can be achieved by proper placement of rubber dam [41, 42]. Although dentin is a wet substance, the constituents of saliva and blood create an environment that can destroy dentin bonding. Use of rubber dam or other dry field aids are necessary to avoid salivary or blood contamination during placement of tooth adhesion materials [40].

Selection criteria

Composites are indicated in class III, IV & V lesion in anterior teeth & small to moderate class I & II cavities [11]. Failure in case selection occurs, in case with poor oral hygiene as composite resin get easily attached to a higher level of pathogenic bacteria than other restoration, which may lead to recurrent caries due to microleakage [26]

Marginal failure and fracture of restoration

Poor marginal strength is the characteristic feature of composite [43]. So margins of restoration should be away from the occlusal contact points. If margins left open there may be chances of microleakage resulting in formation of secondary caries [44]. Marginal fracture is more common than bulk fracture [10].

Microleakage

Microleakage can cause postoperative sensitivity and bacterial invasion e.g. Streptococcus mutans. Microleakage results in subsequence inflammatory changes, secondary caries & discoloration of restoration [40]. Marginal gaps are primarily results from polymerization shrinkage on setting of resins [45]. After setting, dimensional changes occurs by masticatory forces, thermal changes & water sorption of composite restoration [44]

Inadequate finishing & polishing

A proper finishing& polishing is to be done, as all rough surfaces acts as an entry for microorganisms. Sharp projections irritate & inflame gingiva so interporximal areas should be given importance. Dentinal margins can be open at dentin-restoration when dry polishing & finishing is doneusing the burs having number of flutes, lesser will be the damage [42].

Post-operative sensitivity

Post-operative sensitivity can arise if we fail to avoid the cause of shrinkage and placement of restoration. It is caused when a Gap is created between restoration & tooth surfaces [44]. Pressure changes in dentinal fluids as flexural strength of composite restoration and tooth differs which transmitted to the pulp.

Conclusion

Composite material has a high rate success of longevity when used in posterior restoration. This review has highlighted the major causes for failures of composite restoration. To enhance the accomplishment of these restorations, factors related to the patient and operator is of great importance, which indicates the need for a conservative approach toward restoration replacement and its prevention.

Corresponding author:

Remmiya Mary Varghese

Senior Lecturer, Department Of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India Mobile: +91-9840488439

Email id: remmiya@gmail.com

References

1. McLean JW. Limitations of posterior composite resins and extending their use with glass ionomer cements. Quint Int 1987; 18(8): 517-528.

2. Valentine CWo composite resin restoration in esthetic dentistry. J Am Dent Assoc 1987; Special Issue 55E-6IE.ヸ.Branden M. Selection and properties of some new dental materials. Dent Update 1974; 1(10): 489-501.

4. Lutz F. Dental restorative resins: types and characteristics. Dent Clin North Am 1983; 27(4): 697-712. ヶ

5. Wei SHY & Jensen M. Composite Resin Restoration. In:Paediatric dentistry - total patient care, Wei ヸSHY ed. Lea&Febiger, Philadelphia, 1988; 12:99-223. ヶ

6. Farah JW & Powers JM. The Dental Advisor.1987; 4:4-7.

7.Ferracane JL. Current trends in dental composites.Crit Rev Oral Biol Med. 1995; 6:302–318.

8. Nicholson JW. Adhesive dental materials and their durability. Inter J AdhesAdhes. 2000; 201:11–16.

9. O'Brien WJ. Dental material and their selection.Quintessence publishing company, Inc; Polymeric Restorative Materials. 2002;113–131.

10. Joseph Sabhagh.Golden rules for Successful Composite Resin Restoration.2009;134

11. Albers HF. Tooth-coloured Restoratives, principles and techniques. Henry Ford: BC Decker Inc Hamilton.2002; 111

12. Bernardo M, Luis H, Martin MD, Leroux BG, Rue T, Leitao J. Survival and reasons for failure of amalgam versus composite posterior restorations placed in a randomized clinical trial. J Am Dent Assoc 2007;138:775–83.

13. Soncini JA, Maserejian NN, Trachtenberg F, Tavares M, Hayes C. The longevity of amalgam versus compomer/composite restorations in posterior primary and permanent teeth: findings From the New England Children's Amalgam Trial. J Am Dent Assoc 2007;138:763–72.

14. Author

15. Price RB, Strassler HE, Price HL, Seth S, Lee CJ. The effectiveness of using a patient simulator to teach light-curing skills. J Am Dent Assoc. 2014;145(1):32-43.

16. Martin FE. A survey of the efficiency of visible light curing units. J Dent. 1998;26(3):239-243.

17. Opdam NJ, Bronkhorst EM, Roeters JM, Loomans BA. Longevity and reasons for failure of sandwich and total-etch posterior composite resin restorations. J Adhes Dent. 2007;9(5):469-475.

18. Da Rosa Rodolpho PA, Donassollo TA, Cenci MS, et al. 22-Year clinical evaluation of the performance of two posterior composites with different filler characteristics. Dent Mater. 2011;27(10):955-963

19. Goldman M. Polymerization Shrinkage of resin base re- sportive materials. Aust Dent J 1983; 28(3):156-161. サ

20. Jorgensen KD, Asmussen E &Shimokobe H. Enamal Damages caused by contracting restorative resins. Scand J Dent Res 1975; 83 (2) : 120-122

21. Ehrnford L &Derand T. Cervical gap formation in Class II composite resin restorations. Swed Dent J 1984; 8: 15-19.

22. Braden M, Caauston BE & Clarke RL. Diffusion of water in composite filling materials. J Dent Res 1976; 55(5) :730-732.

23. Bausch lR, DeLange C & Davidson CL, et al. The Cinical significance of polymerization shrinkage of composite resin restorative materials. J Prosthet Dent 1982; 48(1): 59-67.

24. Rees IS &lacobson PH. The polymerization shrinkage of composite resins. Dent Mater 1989; 5(1): 41-44.

25. Henry Wadsworth Longfellow, Albers. Resin Polymerization BC Decker Inc Hamilton London.2002;6(9):81-110

26. Bohaty BS, Ye Q, Misra A, Sene F, Spencer P.Review article Clinical , Cosmetic and Investigational Dentistry Posterior Composite Restoration Update : Focus on factors influencing form and function2013;5:33-42

27. Kinomoto Y, Torii MTakeshgi F, Ebisu S. Polymerization Contraction stress of Composite Resin restorations in a model Class I cavity configuration using photoelastic analysis. J of Esthetic and Restorative Dentistry.200; 012(6):309-319.

28. Alomari QD, Reinhardt JW, Boyer DB. Effect of liners on cusp deflection and gap formation in composite restorations. *Oper Dent*. 2001;26(4):406-411.

29. Bouillaguet S, Ciucchi B, Jacoby T, et al. bonding characteristics to dentin walls of class II cavities, in vitro. Dent Mater. 2001;17(4):316-321.

30. Roulet JF. Benefits and disadvantages of tooth-coloured alternatives to amalgam. J Dent. 1997;25(6):459-473.

31. Bohaty BS, Ye Q, Misra A, et al. Posterior composite restoration update: focus on factors influencing form and function. Clin Cosmet Investig Dent. 2013;5:33-42.

32. Wang Y, Spencer P. Interfacial chemistry of class II composite restoration: structure analysis. J Biomed Mater Res A. 2005;75(3):580-587.

33. Hagge MS, Lindemuth JS, Mason JF, Simon JF. Effect of four intermediate layer treatments on microleakage of Class II composite restorations. Gen Dent. 2001;49(5):489-95.

34. Lundin S-A. Studies on Posterior composite resins with special reference to Class II restorations.Swed Dent. 1990.

35. Roulet IF. A material scientist's view: assessment of wear and marginal intergrity. Quint Int 1987;18(8):543-552.

36. Leinfelder KF. Current development in posterior composite resins. Adv Res 1988;2(1): 115-121

37. Judd DB, Wyszecki G. Color in Business, Science, and Industry. 3rd ed. New York: Wiley-Interscience.1975:26-40.

38. Rodrigues S, Shetty SR, Prithviraj DR. An evaluation of shade differences between natural anterior teeth in different age groups and gender using commercially available shade guides. J Indian Prosthodont Soc. 2012;12(4):222-30.

39. Harry F. Albers. Resin Bonding Marconi, BC Decker Inc Hamilton • London 2002;8(9):126-156.

40. Sarvesh Kumar j and Jayalakshmi. /J. Pharm. Sci. & Res. Vol. 8(7), 2016, 627-63.

41. Hickel R.,Manhart J. Longevity of restorations in posterior teeth and reasons for failure. J Adhes Dent. 3(1):45-64.

42. Sturdevants- Art and Science Of Operative Dentistry Compo- site restoration. TheodoreM.RobinsonDDS,Elsevier publication, 2006;5: 497-597

43. Ryan Francisco Alberto, Ida de Noronha de Ataide. Effect of cyclical lateral forces on microleakage of cervical resin composite Restoration, journal of conservative dentistry. 2007;10 (1):5-13.

44. V. Ritter DDS ,Ms. Masters of Esthetic Dentistry, Posterior Composite Revisited Andre journal compilation , blackwell2008; 20(1)

45. S.Kubo ,H Yokota ,Y Hayashi.Effect of light curing modes on the microleakage of cervical resin composite restorations. . J of Dentistry. 2002;32(3):247-254.

Flexible Dentures

S. Mohamed Jaqfer Sadiq[1],Kirubha Robin[2], Aarthi Vignesh[3],
Graduate Student[1],Senior Lecturer[2], Department of Prosthodontics**,**Senior Lecturer[3],
Department of Oral and Maxillofacial Surgery,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University,

.

Abstract

Doctors experience play a major role in fabricating and designing the dentures,but the selection of dental resin is also important. Dentists are advising flexible partial dentures because they make better and stronger appliances that are comfortable and long lasting. The strong and flexible nature of the material perfectly suits the variety of natural conditions in the mouth, simplifying design and enabling the flexible nylon resin to act as a built-in stress-breaker in order to provide superior function and stress distribution in a removable partial denture. Unilateral or bilateral undercuts are frequently encountered and may complicate successful fabrication of denture prosthesis. This review is to ensure the better purpose of flexible dentures compared to conventional acrylic denture its advantages and its disadvantages.

Key Words*: Denture base, Denture resin, Valplast, Retention, Stability.*

Introduction

Loss of teeth, which may be due to trauma, dental diseases, pathology, or otherwise not only alters the psychological thought of the patients but also disturbs the esthetics, phonetics, and functional occlusion[1]. Replacement of missing teeth is highly essential in order to restore the defect and regain function as best as possible.Modern dentistry offers many options for the restoration of partially edentulous mouth, like removable partial dentures (RPD), fixed bridges and dental implants. Removable partial dentures became very popular with the introduction of acrylic polymers and chrome cobalt alloys in dentistry. Many patients choose removable partial dentures due to factors ranging from cost to physiology[2]. Dentists are advising flexible partial dentures because they make better and stronger appliances that are comfortable and long lasting. The strong and flexible nature of the material perfectly suits the variety of natural conditions in the mouth, simplifying design and enabling the flexible nylon resin to act as a built-in stress-breaker in order to provide superior function and stress distribution in a removable partial denture. Unilateral or bilateral undercuts are frequently encountered and may complicate successful fabrication of denture prosthesis[3,4]. The advancement of the nylon-inferred denture base material in the 1950s prepared for another kind of dentures. Adaptable dentures are a magnificent contrasting option to customarily utilized methyl methacrylate dentures, which give great style and solace as well as adjust to the consistent development and adaptability in incompletely edentulous patients.Adaptable fractional dentures are dental prosthesis made of thermoplastic nylon sap called polyamides utilized as a part of expert dentistry since 1954[5,6]. Dental practitioners utilize adaptable dentures to supplant at least one of your missing teeth either in the upper or lower jaw.Adaptable halfway dentures are agreeable, delightful, and a reasonable decision in this cutting edge time. It was for some time imagined that removable fractional dentures must be unbending to be powerful[7]. The development of the adaptable incomplete dentures enables the reclamation to adjust to the steady development and adaptability in the mouth. This is the fundamental reasoning behind the inventive adaptable, removable halfway denture. Flexible temporary dentures are suggested by dental specialists amid restorative scenes. After careful reproduction of the upper jaw, there is a need to oblige the patient amid the period amongst medical procedure and the creation of a last dental machine[8,9]. For this reason, an adaptable denture offers an awesome arrangement that enables the patient to recommence day by day exercises. Midline break of finish dentures has been accounted for to be the second most basic sort of crack in denture prosthesis. Adaptable denture material has been accounted

for to have restorative preferred standpoint in beating midline denture cracks. Adaptable dentures frame an astounding other option to customary hard fitting denture. [10,11]. Material being delicate and solid can be made more slender and are light in weight contrasted with customary dentures that advances better adjustment of the tongue and cheek to the denture base. These dentures retain little measures of water to make the denture all the more delicate tissue good[12]. Because of their capacity of superb mouldability, light weight to thickness proportion and high warm quality, adaptable dentures have been demonstrated as a fantastic treatment alternative for finish and halfway edentulism[13]. Be that as it may, watchful case choice and clinical judgment is required to utilize adaptable dentures in fitting circumstances keeping in mind the end goal to acquire a fruitful treatment result. The adaptability, joined with quality and lightweight, gives add up to solace and awesome looks [14].Management of these situations conventionally includes alteration of the denture prosthesis bearing area, adaptation of the denture base, careful planning of the path of insertion and the use of resilient lining material. An alternative denture prosthesis design in which optimal flange height and thickness can be achieved is by using flexible denture base material. Soft dentures are an excellent alternative to traditional hard-fitted dentures [15]. Traditionally relining dentures with a soft base increases comfort at the cost of chewing efficiency. To make up for the loss of chewing efficiency, denture wearers would use dentures adhesive which causes its own problems. A flexible material is now an option that does not trade off the ability to eat[16].

Functional Benefits of Flexible Materials

Acrylic partial dentures offer a relative ease of fabrication as compared to the metal frame. The cast partials require accurate tooth preparations for guide planes and placement of occlusal rest. Very accurate surveying is required on the diagnostic cast to help inform about the tooth preparation [17]. However, the main limitations from these materials come from a steady loss of function as the edentulous ridge undergoes a natural process of resorption and the obvious non-aesthetic visible metal clasps. The patient needs to maintain the partial dentures routinely in terms of clasp adjustment and relines, and if any of the requirements are slightly compromised, the design will fail to work as intended. Irrespective of the accuracy with which the metal partial denture is designed for its fit, this perfection is gradually lost after the partial denture is placed.Soft dentures are generally only used when traditional dentures cause discomfort to the patient that cannot be solved through relining.

Soft dentures are not the same as a soft reline for traditional dentures [18] .Soft relines use a soft putty-like substance to separate gums from the hard acrylic in dentures. Flexible dentures use a special flexible resin that prevents them from chafing the gums, allows the wearer to chew properly. It also provides a soft base that prevents the gums from being rubbed raw.Flexible dentures have got various advantages over the traditional rigid denture bases. Translucency of the material picks up underlying tissue tones, making it almost impossible to detect in the mouth [19].

Valplast

Valplast is a flexible denture base resin that is ideal for partial dentures and unilateral restorations. The resin is a biocompatible nylon thermoplastic with unique physical and aesthetic properties that provides unlimited design versatility and eliminates the concern about acrylic allergies. The Valplast Flexible Partial allows the restoration to adapt to the constant movement and flexibility in your mouth. The flexibility, combined with strength and light weight, provides total comfort and great looks[20,21].

Advantages

1. Flexible dentures have got various advantages over the traditional rigid denture bases. Translucency of the material picks up underlying tissue tones, making it almost impossible to detect in the mouth.
2. No clasping is visible on tooth surfaces (when used in manufacturing of clear clasps), improving aesthetics. The material is strong and flexible. Free movement is allowed by the overall flexibility [22].
3. Complete biocompatibility is achieved because the material is free of monomer and metal, these being the principle causes of allergic reactions in conventional denture materials. Clinicians are able to use Valplast, Duraflex, Flexite, Proflex, Lucitone, Impak where as valplast and lucitone are monomer free. Areas of the ridge that would not be possible with conventional denture and partial techniques can be accessed by these flexible dentures.
4. Patient can wear appliances that would normally not be comfortable. Flexible dentures will not cause sore spots due to negative reaction to acrylic resins and will absorb small amounts of water to make the denture more soft tissue compatible. Flexible dentures may be used as an alternate treatment plan in rehabilitating the anomalies such as ectodermal dysplasia [22].

5. Metal-free restorations and prosthesis are future of dentistry. Flexible partial is the optimal choice whenever partial is the choice of treatment or the patient prefers not to use a fixed restoration. Patients, who have used both conventional RPD and a Flexible partial, report that the later feels more natural and is more comfortable to wear [23].

6. It also provides a higher standard of function by using the flexibility of the material to balance masticatory forces over the entire supporting ridge instead of individual support points (Phoenix et al, 2004). The balanced distribution of forces can often lead to longer lasting appliances that may not require frequent relines. Following are some of the advantages of a flexible partial denture: More acceptable esthetics, since there are no metal clasps. The material has good flexibility like Titanium. Therefore, even if there is a little bit of bending, it comes back to the original shape and position [24].

7. Ease of insertion in the mouth with alveolar undercuts because of the flexibility. Even if there is slight shifting of the remaining teeth over time, the flexibility of the denture material, allows the use of prosthesis with little adjustment. There is no need of modification of the remaining teeth to receive occlusal rests as for the metal clasps. In cases of undercut due to tilted teeth, flexibility of the material makes it possible to insert the prosthesis over the angulated teeth[22].

8. The denture can be heated up in hot water for about a minute and can easily be adjusted and inserted in the undercut area. A real boon for patients with compromised oral conditions. Opens up scope to address the needs of such patients with ease[24]

Ultra-light and thin.

The material thickness may shift from 0,6 mm to 1,8 mm so every nylon denture is five times lighter than standard dental prosthesis and much more if contrasted with metal cast partials. Adaptable dentures influence you to disregard the cumbersome inclination that made wearing fractional apparatuses so awkward previously.

Other Advantages

- Conceivably Unbreakable

The nylon base makes the prosthesis adaptable in this way, it might ingest well an inadvertent tumble to the ground amid the home oral cleanliness.

On account of its unbreakable element, a few brands, for example, Valplast offer lifetime guarantee that apply to the adaptable material as it were. At the end of the day, prosthetic

teeth (made of sap or porcelain) are not secured by the guarantee. If you don't mind make sure to deal with the dental machine with mind[23].

- Anallergic and Profoundly Biocompatible

Polyamides (otherwise called thermoplastic nylon gum) doesn't contain bisphenol (BPA) consequently it is the most biocompatible material for building dental pitches. Polyamides dentures is a substantial option for patients that experience the ill effects of sensitivities to acrylic or certain metals, for example, chromium and cobalt utilized as a part of the system of conventional fractional dentures. Nylon dentures are viewed as sans metal dental apparatuses.

- Agreeable and Fantastic feel

Adaptable partials benefits incorporate agreeable. Contaminate, they adjust to the steady development and adaptability in the mouth. Because of delicate material, they don't cause gingival bothering.

Since there are no metal catches around your common teeth, they are essentially imperceptible.

The shade of the base material mixes with the characteristic shade of the gums in your mouth. Nobody will find you're wearing an adaptable fractional denture[25].

- Stain and Smell Safe

Regardless of whether adaptable incomplete denture are more impervious to catch scent and to recolor (we should consider the pigmentation because of espresso, tea, cigarettes smoke and wine) than conventional acrylic dentures despite everything they should be legitimately cleaned and kept up as portrayed underneath

- Non-Deformable

Depicting standard fractional denture, we said that while you are not wearing it, It is smarter to place it in a glass of water or in a sodden little towel. The reason is basic, If a denture gets dry it tends to change its shape. With adaptable dentures this hazard doesn't exist since they generally have a tendency to return to their unique shape. Anyway, dental practitioners dependably counsel their patients to keep their adaptable partials hydrated utilizing legitimate fluid[23].

Disadvantages

1. Being a plastic material, it cannot be made into thin sections like metal.

2. It is likely to break if cut thin sections. Since they need to be made bulkier than cast partials, it may take longer to get used to a flexible partial denture. It does not conduct heat and cold like metal.

3. The patient may not enjoy certain food like hot soup or ice cream. Since flexible dentures utilize the gaps (because of some missing teeth) for the 'Retento-Grip Tissue-bearing Technique' (Iselin et al, 1990) for retention, the remaining teeth have to be in fairly good periodontal health[25].

4. The patients that have periodontal problem may have several teeth that are mobile due to bone loss. Therefore, the whole area keeps on flexing causing unfavorable forces that in turn result in more bone loss. The laboratory fee is a little higher.Requires more chair-side time for adjustment.

5. A Flexible denture is very hard to repair if fractured. No additions can be made onto it. In such cases, rebasing is recommended.Flexible dentures generally not used for long-term restorations and is intended only for provisional or temporary applications. Flexible dentures tend to absorb the water content and will discolor often. Metal frame partial dentures remain the" standard" for long-term restorations. When grinding this prosthesis, proper ventilation, masks, and vacuum systems should be used and the procedure is technique sensitive[9].

Conclusion

Flexible dentures were previously selected by few patients and the clinician but now a days it has become an elective treatment option.Flexible partial dentures can be a good option for the replacement of missing teeth when patient is concerned about aesthetics.The rehabilitation of orofacial structures demands the restoration of esthetic and function irrespective of the individual's dietary / parafunctional habits and structure left. They have given a option of thinking beyond complex designing of cast partial dentures. The fabrication of the optimum restoration is depending on the clinicians skills in selection of the type of the restorations which is required for the patientFlexible dentures will stand in a superior are allergic to monomer. Demand for more retentive and aesthetic treatment needs. Flexible dentures were previously selected by few patients and the clinician but now a day it has become an elective treatment option. Position in fulfilling the various patients. VALPLAST plaster is the

flexible partial denture allowing the restoration to adapt to constant movement and comfort along with great looks. VALPLAST is the most commonly used.

Corresponding Author

Aarthi Vignesh,

Senior Lecturer, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077, Tamil Nadu, India.

Email Id: draarthiomfs@gmail.com

Phone Number: +91 8056985855

References

1. Zarb GA, Bolender CL, Carlsson GE. St Louis: Mosby. Boucher's Prosthodontic Treatment for Edentulous Patients; .1997:11; 337–42.
2. NAYLOR WP, Manor RC. Fabrication of a flexible prosthesis for the edentulous scleroderma patient with microstomia. J Prosthet Dent. 1983;50(4):536-8.
3. Strohaver RA. Comparison of Changes in Vertical Dimension between Compression and Injection Molded Complete Dentures. J Prosthet Dent. 1989;62:716–8.
4. Jagger DC, Harrison A, Jandt KD. The Reinforcement of Dentures. J Oral Rehabil. 1999;26:185–94.
5. Yunus N, Rashid AA, Azmi LL, Abu Hassan MI. Some Flexural Properties of a Nylon Denture Base Polymer. J Oral Rehabil. 2005;32:65–71.
6. Dhiman RK Col, Roy Chowdhury SK. Midline Fracture in Single Complete Acrylic vs Flexible Dentures. MJAFI. 2009;65:141–5.
7. Antonelli JR, Hottel TL. The "flexible augmented flange technique" for fabricating complete denture record bases. Quintessence Int. 2001;32(5):361-4.
8. Lowe LG. Flexible denture flanges for patients exhibiting undercut tuberosities and reduced width of the buccal vestibule: a clinical report. J Prosthet Dent. 2004;92(2):128- 31.
9. Naylor WP, Manor RC. Fabrication of a flexible prosthesis for the edentulous scleroderma patient with microstomia. J Prosthet Dent. 1983;50(4):536-8.

10. Antonelli JR, Hottel TL. The "flexible augmented flange technique" for fabricating complete denture record bases. Quintessence Int. 2001;32(5):361-4.
11. Dr. Sunitha N Shamnur1, Dr. Jagadeesh KN1, Dr. Kalavathi SD1, Dr. Kashinath KR. 2002;34:323-333.
12. Iselin W, Meier C, Lufi A, Lutz F: The flexible gingival epithesis. The practical procedure, laboratory technics and clinical experience. 1995;18:12-14.
13. SchweizerMonatsschrift for Zahnmedizin. Kaplan P. Flexible removable partial dentures- design and clasp concept. Dentistry Today, 2008;27(12): 120,122-123.
14. Lowe LG: Flexible denture flanges for patients exhibiting undercut tuberosities and reduced width of the buccal vestibule: a clinical report. The Journal of Prosthetic Dentistry, 2004;92(2):128-131.
15. Shamnur SN, Jagadeesh KN, Kalavathi SD, Kashinath KR: “Flexible dentures”- an alternate for rigid dentures? Journal of Dental Sciences & Research, 2005; 1(1):7479. 10.
16. Zhao X, Cao J, Zhang Y: Clinical application of flexible gingival epithesis material. West China Journal of Stomatology, 2003; 21(4):324-326.
17. G.K. Thakral, Himanshu Aeran, Bhupinder Yadav, *Rashmi Thakral.2005;5(2):66-68.
18. Maurice N, Stern. Valplast flexible partial dentures. New York State Journal.1964;30:123 -36.
19. Kutsch VK, Whitehouse J, Schermerhorn K, Bowers R. The evolution and advancement of dental thermoplastic. Dental Town Magazine. Negrutiu M, Sinescu C, Romanu M, Pop D, Lakatos S. Thermoplastic resin for removable partial dentures. TMJ.2003;5(3):456-458.
20. Keenan PL, Radford DR, Clark RK. Dimensional change in complete dentures fabricated by injection molding and microwave processing. J Prosthet Dent. 2003;89(1):37-4.
21. Lowe LG. Flexible denture flanges for patients exhibiting undercut tuberosities and reduced width of the buccal vestibule: A clinical report. J Prosthet Dent, 2004;92:128-31.
22. MedaNegrutiu,CosminSinescu,MihaiRomanu, aniela Pop, Sorin Lakatos. Thermoplastic Resins for Flexible Framework Removable Partial Dentures. TMJ 2005;55:295-9.

23. WöstmannB, Budtz-JørgensenE,JepsonN,MushimotoE,PalmqvistS,Sofou A. Indications for removable partial dentures: A literature review. Int J Prosthodont 2005;18:139-45.
24. Beaumont AJ Jr. An overview of esthetics with removable partial dentures. Quintessence Int, 2002;33:747-55.
25. Lowe, L.G. Flexible denture flanges for patients exhibiting undercut tuberosities and reduced width of the buccal vestibule: a clinical report. J. Prosthet Dent., 2004; 92(2):12831.

Foramen Magnum Dimention Variations Shown Among Male And Female Population

Aslin Sanofer A[1], Saravana Kumar[2], Jayanth Kumar V[3]
Graduate Student [1],Senior Lecturer [2] , Dept of Anatomy ,
Reader [3], Dept of Oral Medicine & Radiology,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract:

The aim of the study is to show the various dimensions of foramen magnum shown by the male and female population. The objective is to study the various anteroposterior , longitudinal and transverse dimensions seen among the population in the foramen magnum. Sex determination between people is most commonly identified based upon morphological shape, size and structure changes seen with them. Many craniofacial skeletal structures damaged after air accidents, mass disasters, fire, explosion makes difficult to determine the gender and the consequent identification. This identification can be done and studied based upon morphological changes in the individuals. This study makes the forensic people to estimate the gender and sex identification in simple manner. In additional to this, the article gives the review on different dimensions of foramen magnum shown by the population.

Key words : *Foramen magnum, sex, identification, males , females*

Introduction:

Personal identification is the act of establishing the identity of a person—by linking him/her to the stream of data in the information systems. It beholds a major portion in forensics and usually relies on the comparison of the known features to the unknown specimen. Identification of an individual is vital for the family not only from an emotional standpoint but is also a medicolegal requirement [1]. In living individuals, identification plays a pivotal role in cases such as property disputes, insurance claims, issuance of passports, and various other licenses, whereas after death, it becomes important to identify the deceased as in case of a murdered victim, or file closure of a person missing for a prolonged period to facilitate the rituals of body disposition following death and permission of remarriage. So, the society's duty to preserve human rights and dignity beyond life begins with identity. Hence, positive identification is required both for legal and humanitarian reasons.

Sex determination reduces the search of individuals by 50%[2] and is 98% accurate when the whole skeleton is available [3]. But finding an intact skeleton is not always likely.In such circumstances, pelvic bone possesses highest sex discriminant accuracy of 95% [4]. The skull is the second best alternative [5] and is 90% accurateto determine the sex of the decedent [4]. However, in explosions, warfare and mass disasters, human remains are often obtained in fragmented states. In such circumstances, only strong bones that greatly resist fracture are likely to be recovered intact. Forensic investigators have thus attempted to utilize them for personal identification. The base of the skull is one such bone and establishing sex discriminant value of the skull base has attracted attention. The aim of this article is to highlight the anatomical and clinical features of foramen magnum followed by its forensic role in human identification.

Foramen magnum [FM] is an important structure of the skull base. It is located inferior to the sagittal suture and surrounded by the basilar, squamous, and lateral parts of the occipital bone. Situated in the deepest part of the posterior cranial fossa and covered by a large volume of soft tissue, it is an ideal structure for sex determination. Sexual dimorphism in FM dimensions is population-specific and highly influenced by environmental, socioeconomic, and genetic factors. The foramen magnum [Latin: great hole] is a large opening in the occipital bone of the human skull. It is one of the several oval or circular openings [foramina]

in the base of the skull. The foramen magnum is found in the most inferior part of the posterior cranial fossa [6].The spinal cord, an extension of the medulla, passes through the foramen magnum as it exits the cranial cavity. Apart from the transmission of the medulla oblongata and its membranes, the foramen magnum transmits the vertebral arteries, the anterior and posterior spinal arteries, the tectorial membranes and alar ligaments. It also transmits the spinal component of the accessory nerve into the skull.

The diameter and area of the foramen magnum are greater in males than in females, hence its dimensions can be used to determine sex in the medico legal conditions, especially in the following circumstances, such as explosions, aircraft accidents and war fare injuries [7]. Foramen magnum is about 3cm wide by 3.5cm anteroposteriorly [8]. It is located midway between and on a level with mastoid processes. The foramen magnum is surrounded by different parts of the occipital bone, squamous part lies behind and above, basilar part in front and a condylar part on either sides [9]. On each side its antero-lateral margin is encroached by occipital condyles, hence the foramen magnum is narrow anteriorly. The anterior edge of the foramen magnum is slightly thickened and lies between the anterior ends of the condyles. The posterior half of the foramen magnum is thin and semicircular. Upper ends of anterior and posterior atlanto-occipital membranes are attached to the anterior and posterior margins of the foramen magnum respectively, and their lower ends are attached to the superior surface of anterior and posterior arches of the atlas respectively. The foramen magnum is a wide communication between posterior cranial fossa and the vertebral canal. The narrow anterior part of the foramen magnum has apical ligament of dens, upper fasciculus of the cruciate ligament and membrana tectoria, both are attached to the upper surface of basioccipital bone infront of the foramen magnum. Its wide posterior part contains the medulla oblongata and its meninges. In subarachnoid space spinal rami of the accessory nerve and vertebral arteries, with their sympathetic plexus, ascend into the cranium; the posterior spinal arteries descend posterolateral to the brain stem, where as anterior spinal artery descends anteromedian to the brain stem. The cerebellar tonsils may project into the foramen magnum.

Interest in the anatomy of the foramen magnum [FM] is largely interdisciplinary-made evident by numerous anatomical studies from the fields of physical anthropology, forensic anthropology, comparative anatomy, evolutionary biology, and surgery.

Development Of The Foremen Magnum :

During fetal development, the anterior and lateral aspects of the FM are formed from the basioccipital, the exoccipitals, and their respective interoccipital synchondroses anterior while the posterior boundary of the foramen is formed by the supraoccipital, the exoccipitals, and their corresponding interoccipital synchondroses posterior. The fetal growth centers do not fully ossify until approximately 10 years of age until which time both the size and shape of the FM are undergoing change. Between the 7th month in utero and birth, the rate of FM growth in its sagittal dimension is 5.4% greater than that of its transverse dimension.Between birth and 0.5 years, the opposite trend is observed – the rate of transverse growth is 7.6% faster than that of sagittal growth.Mean adult sagittal lengths are attained by 5 years of age, whereas mean transverse lengths are not attained until 10 years of age.

Landmark Of Foramen Magnum:

The opisthion is the centre on the posterior margin of the hiatus and could be a cephalometric landmark. Another landmark is that they basion situated at the centre on the anterior margin of the hiatus.

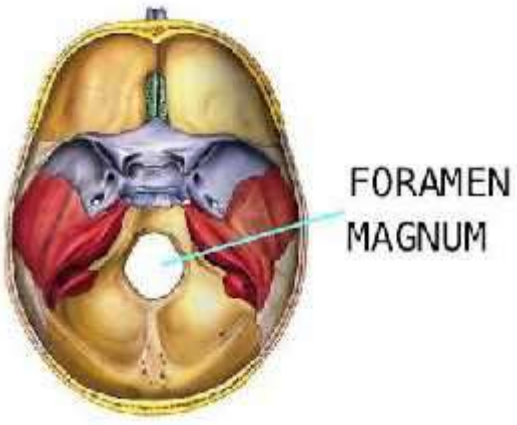


Image 1 : Showing Foramen Magnum

Relation Of Foramen Magnum:

anteriorly - basilar part of occipital bone

anterolaterally - occipital condyles, hypoglossal canal, jugular foramen
posteriorly - squamous part of occipital bone with the internal occipital crest

Contents :

medulla oblongata
meninges
spinal root of the accessory nerve
vertebral arteries
anterior and posterior spinal arteries
tectorial membrane and alar ligaments

Radiographic Features :

CT :

at the amount of the hiatus CSF is seen to surround the medulla that is reduced or absent in tonsillar hernia.

Measurements Of Foramen Magnum:

-Longitudinal diameter [LD] of the foramen magnum - It is distance between basion and opisthion.
-Transverse diameter [TD] of the foramen magnum - It is maximum distance between two lateral margins.
Measurements of the foramen magnum were taken by vernier callipers to the nearest of 0.1mm. Measurements were taken twice and average of two values was taken as final measurement. Area of the foramen magnum It is surface area of the foramen magnum calculated by the following formula [10].

AREA [A] = ¼ X p X w X h w = Width, transverse diameter h = Height, longitudinal diameter = 22/7, mathematical constant.

Morphometry And Morphologyoftheforemenmagnum :

Adult females have a reported mean sagittal FM diameter ranging from 27.1 to 36.66 mm whereas adult males have a reported mean sagittal diameter ranging from 32 to 40.0 mm [11].Adult females have a reported mean transverse diameter ranging from 25.45 to 31.34 mm,whereas adult males have a reported mean transverse diameter ranging from 26.92 to 38.0 mm [12] . Studies reporting both female and male mean sagittal and transverse diameters agree that male FM diameters are longer than those of females [13] . Aside from documenting the ratio between sagittal and transverse diameters [i.e., FM-index], the study of FM shape has been largely limited to assigning shape categories to foramina. The shape of the FM has been classified with ambiguous terminology such as “asymmetrical,” “biconvex,” “bi-pointed oval,” “bi-rounded oval,” “circular,” “dorsally convergent oval,” “egg,” “heart-like,” “heptagonal,” “hexagonal,” “irregular,” “oval,” “pentagonal,” “pear,” “polygonal,” “rhomboid,” “round,” “symmetrical,” “tetragonal,” “two semicircles,” “ventrally wide oval,” and “wide oval ” [14].

Sahoo et al. [15] reported the FM to be mostly in the shape of “two semicircles,” and furthermore, both pear- and egg-shaped more often than oval-shaped; however, similar to the Henríquez-Pino et al.'s [16] study, Sahoo et al. [15] did not identify a tetragonal category. Indeed, Aragão et al. [17] classified FM into eight categories and still noted that “in the remaining 6.36% of the FM, it was not possible to define a regular and specific form.

Knowledge of morphometric and morphologic variation of the FM in different populations has implications for a variety of scientific fields; however, the structure of the FM has not been studied among several geographically and craniofacially distinct populations.

Discussion :

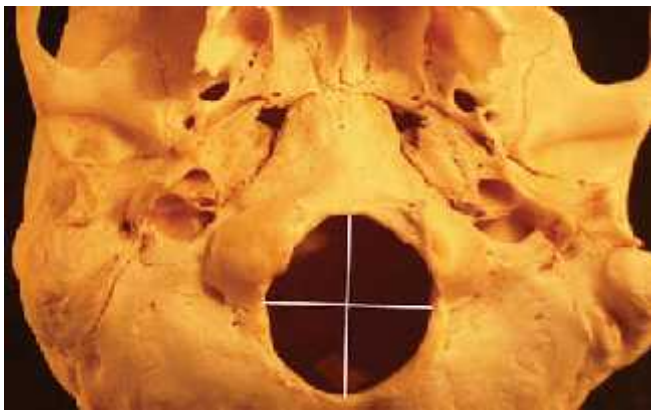
Sex determination helps to channelize the investigation by deducing the search to half the population, thus conserving both resources and the time required for identification. When the entire skeleton is present, sex determination is possible with 100% accuracy. However, in many instances human remains are likely to be obtained in fragmented states. In such situations, the pelvis has demonstrated maximum accuracy in sex determination followed by the pelvis if available with cranium, pelvis with long bones, and long bones or skull in isolation. The base of the skull, precisely the occipital bone is often recovered intact, even in cases of severe trauma due to its well-protected anatomical position and large amount of overlying soft tissue [18].

FM is a vital and prominent structure of the skull base. It has been found to exhibit differences in its dimensions between males and females. But this sexual dimorphism is population-specific, as demonstrated by studies on the populations of Iraq, Turkey, Brazil, Poland, and Nigeria. This is also seen in India in diverse geographical locations such as Uttar Pradesh, Gujarat, Chandigarh, and Madhya Pradesh. The dimensions of FM are also influenced by genetic, environmental, and social factors. Many authors have demonstrated sexual dimorphism in the South Indian population as well [19]. FM completes its growth by early childhood. It is unresponsive to secondary sexual changes, with no influence of musculature on its size and shape, making it considerably stable beyond adolescence [20].

Babu [21] found the mean LD values to be 35.68 mm in males and 32.57 mm in females; Kanchan et al. [22] reported values of 34.51 mm in males and 33.6 mm in females. The mean values of TD as reported in these studies were 28.91 mm in males and 28.19 mm in females [Babu R]; 27.36 mm in males and 26.74 mm in females [Kanchan et al.].

In CT scan images, the longitudinal diameter and area of the foramen magnum of male subjects was higher than females, whereas transverse diameter of the foramen magnum of male was not significantly higher than females. In a morphometric evaluation of the foramen magnum of normal adults by CT scan showed. In males sagittal diameter varied from 31-45mm with an average of 37.2±3.43 mm, transverse diameter varied from 27-40mm with an average of 31.6±2.99 mm and area of foramen magnum was varied from 710-1266mm2 with an average of 931.7±144.29mm2. In females the sagittal diameter was varied from 28-42mm with an average of 34.6±3.16mm transverse diameter was

varied from 28-42mm with an average of29.3±2.19mm and area of the foramen magnum was varied from 671-1006mm2 with an average of 795.0±99.32mm2.



The correlation coefficient between stature and breadth of foramen magnum shows extreme significance [p = 0.006] in the southern Chinese population, but is not statistically significant [p = 0.932] in the northern Chinese population [24].

The morphological differences between skulls of different genders are mainly determined by genetic factors rather than nutrition, hormones, or muscles [GÜNAY, 2000]. Thus, the determination of gender in human skulls is based on morphological differences, mainly on the size and strength of certain structures [ROSING, 2007], which may be characteristics of each population that are influenced by genetic, environmental, and socioeconomic factors [25] .

Conclusion:

From this review study , we noticed that males show significant value than females in the sexual dimorphism of foramen magnum dimensions. The skull base and the occipital bone are protected by a large amount of soft tissue, and the anatomical position is useful for many craniofacial skeletal structures are damaged after air accidents, mass disasters, fire, explosion, or injuries resulting from violence.

Corresponding Author

Dr. V.Jayanth Kumar,

Saveetha Dental College, Saveetha institute of medical and technical sciences.
Saveetha University,
162, Poonamallee High Road,Velapanchavadi, Chennai 600077.
Email : doctorjayanth@gmail.com
Mob: 80561 35807

REFERENCE:

1. Rai B, Kaur J. Evidence Based Forensic Dentistry. 1st ed. Berlin Heidelberg: Springer-Verlag; 2013.

2. Babu R, Manjunath S, Rastogi P, Kumar Mohan TS, Bhat VJ, Yoganarasimha K, et al. Determination of sex by foramen magnum morphometry in south Indian population. Indian Journal of Forensic Medicine and Pathology. 2011;4:29–33.

3. Uysal S, Gokharman D, Kacar M, Tuncbilek I, Kosar U. Estimation of sex by 3D CT measurements of the foramen magnum. Journal of Forensic Science . 2005;50:1310–4.

4. Uthman AT, Al-Rawi NH, Al-Timimi JF. Evaluation of foramen magnum in gender determination using helical CT scanning. Journal of Dentomaxillofacial and Radiology. 2012;41:197–202.

5. Santhosh CS, Vishwanathan KG, Ashok Gupta, Siddesh RC, Tejas J. Morphometry of the Foramen Magnum: An Important Tool in Sex Determination. Journal of Medical and Health Sciences . 2013;2:88–91.

6. Russo, Gabrielle A.; Kirk, Christopher E. "Foramen magnum position in bipedal mammals". Journal of Human Evolution. 2013; 65 [5]: 656–670.

7. Sgouros S, Goldin HJ, Hockely AD, Wake MJ, et al. Intracranial volume change in childhood. Journal of Neurosurgery . 1999;91:610-616.

8. Gunay Y, Altinkok M. The value of the size of foramen magnum in sex determination. Journal of Clinical forensic andMedicine.2000;7[3]:147-149.

9. Oliveira Ed, Rhoton AL, Peace D. Microsurgical anatomy of the region of the foramen magnum. Journal of Surgical Neurology. 1985;24:293-35.

10. Routal RR, Pal GP, Bhagwat Ss, Tamankar BP. Material studies with sexual dimorphism in foramen magnum of human crania. Journal of Anatomy . 1984;33[2]:85-89.

11. Edwards K, Viner MD, Schweitzer W, Thali MJ. Sex determination from the foramen magnum. Journal of Forensic Radiology and Imaging. 2013;1:186–92.

12. Loyal P, Ongeti K, Pulei A, Mandela P, Ogeng'o J. Gender related patterns in the shape and dimensions of the foramen magnum in an adult Kenyan population. Anatomy Journal of Africa. 2013;2:138–41.

13. Kamath VG, Muhammed A, Radhakrishna S, Radhakrishna A. Binary logistic regression analysis of foramen magnum dimensions for sex determination. Anatomy Research International. 2015;2015:1–9.

14. Loyal P, Ongeti K, Pulei A, Mandela P, Ogeng'o J. Gender related patterns in the shape and dimensions of the foramen magnum in an adult Kenyan population. Anatomy Journal of Africa. 2013;2:138–41.

15. Sahoo S, Giri SK, Panda SK, Panda P, Chandra Sahu M, Mohapatra C. Morphometric analysis of the foramen magnum and the occipital condyles. International Journal of Pharmaceutical Sciences Review and Research. 2015;33:198–204.

16. Henríquez-Pino J, Cricenti SV, Didio LJ. Morphometry of the foramen magnum and its relantioship to skull types on Brazillian individuals. International Journal of Morphology. 1995;13:159–64.

17. Aragão JA, de Oliveira Pereira R, de Moraes RZ, Reis FP. Morphological types of foramen magnum. Annual Research & Review in Biology. 2014;4:1372.

18. Uthman AT, Al-Rawi NH, Al-Timimi JF. Evaluation of foramen magnum in gender determination using helical CT scanning. Journal of Dentomaxillofacial Radiology . 2012;41:197–202.

19. Kanchan T, Gupta A, Krishan K. Craniometric analysis of foramen magnum for estimation of sex. International Journal of Medical and Health Pharmaceuticals Biomedical Engineering . 2013;7:166–8.

20. Krogman WM. The human skeleton in forensic medicine. International Journal of Medical and Health Pharmaceuticals Biomedical Engineering . 1955;77:48–72.

21. Raghavendra Babu YP, Kanchan T, Attiku Y, Dixit PN, Kotian MS. Sex estimation from foramen magnum dimensions in an Indian population. Journal of Forensic and Legal Medicine. 2012;19:162–7.

22. Kanchan T, Gupta A, Krishan K. Craniometric analysis of foramen magnum for estimation of sex. International Journal of Medical and Health Pharmaceuticals Biomedical Engineering . 2013;7:166–8.

23. Suazo , G. I. C.; Russo, P. zavando , M. D. A.; & SMITH, R. L. Dimorphism sexual dimensions. International Journal of Morphology. 2009. 27[1]:21-23.

24. Murshed K A, Cicekeibasi A E, Tuncer I. Morphometric evaluation of the foramen magnum and variations in its shape. A study of computerized tomographic images of normal adults. Turkish Journal of Medical Sciences. 2003;33:301-306.

25. Adams , DC., Rohlf , FJ. and slice DJ. Geometric morphometrics: ten years of progress following the ‘‘revolution.’’ Italian Journal of Zoology. 2004; 71: 5-16.

G6PD Deficiency

Benazir Hussain M[1], Sivakumar M[2]

Graduate Student[1],Senior Lecturer[2], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

Abstract

Glucose-6-phosphate dehydrogenase deficiency (G6PDD) is an inborn error of metabolism that predisposes to red blood cell breakdown.Most of the time, those who are affected have no symptoms.Following a specific trigger, symptoms such as yellowish skin, dark urine, shortness of breath, and feeling tired may develop.Complications can include anemia and newbornjaundice.Some people never have symptoms.It is an X-linked recessive disorder that results in defective glucose-6-phosphate dehydrogenase enzyme.Red blood cell breakdown may be triggered by infections, certain medication, stress, or foods such as fava beans.Depending on the specific mutation the severity of the condition may vary. Diagnosis is based on symptoms and supported by blood tests and genetic testing.A high degree of variation exists between human populations and individuals with respect to malaria incidence, parasite density and disease severity and reactions to anti-malarials. Although in some studies glucose-6-phosphate dehydrogenase deficiency (G6PDd) is associated with protection from severe malaria deficient individuals are reported to suffer from haemolytic anaemia when treated with primaquine, which is the only available drug against *Plasmodium falciparum* gametocytes and a radical cure of *Plasmodium vivax*. Further individuals with G6DPd are reported to suffer from haemolysis when treated with tafenoquine, another anti-malarial with proposed indication of treating hypnozoites of *P. vivax* and *Plasmodium ovale* .G6PDd is one of the most common human enzymopathies which is in a very high frequency in tropical and subtropical malaria endemic regions. In sub-Saharan Africa (SSA), the prevalence of G6PDd can reach up to 35% although organized reports are lacking and different allelic variants are unevenly distributed in the region. However, with progressive improvements in detection methods past G6PDd prevalence figures may need continuous revision. Glucose-6-phosphate dehydrogenase (G6PD) deficiency is the most common enzyme deficiency in humans, affecting 400 million people worldwide. It has a high

prevalence in persons of African, Asian, and Mediterranean descent. It is inherited as an X-linked recessive disorder. G6PD deficiency is polymorphic, with more than 300 variants. G6PD deficiency confers partial protection against malaria , which probably accounts for the persistence and high frequency of the responsible genes. This review gives a detailed description on diagnosis, clinical manifestation and treatment of g6pd deficicency.

Key Words*: g6pd deficiency, favism, jaundice,cancer, haemolytic anemia*

Introduction

The structure of enzyme G6PD monomer consists of 515 amino acid subunits with a calculated molecular weight of 59,256 daltons. The active enzyme exists as a dimer and contains tightly bound NADP. Aggregation of the inactive monomers into catalytically active dimers and higher forms requires the presence of NADP.[1] Thus, NADP appears to be bound to the enzyme both as a structural component and as one of the substrates of the reaction. The binding sites for this coenzyme have not been identified at the structural level, but examination of mutants has suggested that amino acids 386 and 387, the basic amino acids lysine and arginine, respectively, seem to bind one of the phosphates of NADP.[2] The evidence that this site is involved in the binding of NADP is as follows:

(1) all mutants that rapidly lose activity at a 10 pmol/L NADP concentration, but are reactivated at high concentrations of NADP have been shown to have mutations in this region;

(2) mutations in this region result in paradoxical electrophoretic migration of the enzyme as if it had become more positively charged, even when the amino acid change adds a negative charge, suggesting failure of binding of negatively charged NADP.

It has also been suggested, on the basis of the deduced conformation of the peptide chain of the yeast enzyme, that the NADP binding site may be elsewhere, but the data on the human enzyme seems much more compelling to me.[3] The glucose-6-phosphate binding site has been identified at amino acid by locating a lysine at this position that is reactive with pyridoxal phosphate in competition with glucose-6-phosphate.

Enzymology

G6PD catalyzes the first step in the hexose monophosphate pathway (HMP). It oxidizes glucose-6-phosphate to 6- phosphogluconolactone, reducing NADP to NADPH. The HMP is the only source of NADPH in the erythrocytes and it also serves to produce the ribose needed for synthesis of nucleotides in the salvage pathways.[4] The main function of the pathway seems to be to protect the RBC against oxidative damage. Glutathione peroxidase (GSHPx) removes peroxide from the erythrocyte. Reduced glutathione (GSH) serves as a substrate for this enzyme, and because NADPH is required for the reduction of oxidized glutathione and protein sulfhydryl groups, it is an essential factor in the chain of reactions that defends the RBC against peroxide. RBCs are a particularly rich source of catalase, but this enzyme is relatively inefficient at removing low levels of peroxide levels. Moreover, catalase has the ability to bind NADPH tightly and the inactive form, compound 11, is reactivated by NADPH. Thus, the activity of the HMP serves to remove peroxide not only through the action of GSHPx, but also by activating catalase.[5]

The long-standing controversy about which is the more important, catalase or futile. Clearly, both enzymes may play a role and serve as backup mechanisms for each other. Which is more active at any one time may depend on the particular conditions under which the measurements are being made and very likely the particular peroxide substrate that is being catabolized. The Km of G6PD for NADP is very low, roughly 2 to 4 pmol/L, and the enzyme is strongly inhibited competitively by NADPH. Thus, the NADPWNADP ratio within the RBC controls the rate of the reaction in an autoregulatory manner. In the quiescent state, the NADPWNADP ratio is very high, and G6PD is nearly completely inhibited. When NADPH is oxidized, as when oxidized glutathione is reduced in the glutathione reductase reaction, NADPH is converted to NADP and G6PD becomes active, reducing NADP to NADPH. G6PD-deficient cells are unable to respond adequately to such an oxidative stress.[6] When the susceptibility of a mutant enzyme to inhibition by NADPH is greater than normal, this compounds the metabolic difficulty of the cell.

Clinical Manifestation

A.HemolyticAnemia

Drug-Induced Hemolysis

The first and best-known morbid effect of G6PD deficiency was drug-induced hemolysis. Primaquine is but one of many drugs that shortens RBC life span in G6PD-deficient

persons.[1] The administration of such drugs is followed, after a 1- or 2-day delay, by a fall in the hemoglobin (Hb) concentration. Particles of denatured protein adherent to the RBC membrane which is Heinz bodies, appear in the early stages of drug administration and disappear as hemolysis progresses. Appearance of RBCs that have variously been designated in such “irregularly contracted RBCs,” “eccentrocytes,” “hemighosts,” “double-colored RBC,” and “cross-bonded cells” are another morphologic feature observed on the blood film.[2] The Hb of these cells is confined to one side of the erythrocyte, leaving the other part as a flat, Hb-free ghost. In this portion of the cell, the inside surface of the membrane is tightly bonded.” Often Heinz bodies are included in the flattened region, where they may bulge visibly out of the leaflet. The urine turns dark and the patient may complain of back pain when the hemolysis is severe. When G6PD deficiency is relatively mild, as in the class 3 G6PD A-, the hemolyticanemia is self-limited because only the older RBCs are destroyed and young RBCs have normal or nearnormal enzyme activity. In patients with more severe forms of enzyme deficiency such as G6PD Mediterranean, young cells are severely deficient in G6PD,I5 and as a consequence, hemolysis continues until well after the administration of drug is stopped.[3]

As a matter of fact, it is difficult to be certain in some cases, whether a cause-and-effect relationship exists between ingestion of a drug and hemolysis. The most robust data regarding the potential hemolytic effect of drugs and chemicals comes from clinical investigations with Cr-labeled erythrocytes. However, even results obtained using RBC survival studies can be misleading. Individual inherited differences in drug metabolism such as acetylator status play a significant role in determining whether a drug will be hemolytic.[4] Thus, if a recipient who efficiently catabolizes the active hemolytic metabolite of a drug is challenged, hemolysis will not be apparent, but the drug may be hemolytic in a subset of individuals who metabolize the drug less efficiently. Moreover, even when a drug does shorten RBC life span, as shown by performing sensitive studies with Crlabeled erythrocytes, the degree of hemolysis may be so modest as to be of no clinical significance. Sulfamethoxazole, a component of the commonly used combination Septraa and Bactrima, has been shown to produce shortening of the RBC life span in Asian subjects with G6PD deficiency, but no significant hemolysis could be shown when this combination was used clinically in patients with G6PD A-.[5] RBCs from subjects with severe class 2 variants such as G6PD Mediterranean may be sensitive to drugs when those with milder defects such as G6PD A- are not. The data obtained from Cr survival must be supplemented with less reliable information gained from clinical observations.

Clinical studies are confounded by the effect of intercurrent infections which may be responsible for hemolysis rather than the drug that has been administered.[6]

Favism

A clinical manifestation of G6PD deficiency closely related to drug-induced hemolysis is the hemolyticanemia induced by ingestion of the fava bean, Viciafaba. Favism, this hemolyticanemia, has been known since antiquity.[7] Indeed, the demise of Pythagoras has been attributed to unwillingness to enter a bean field, possibly because of favism, although the evidence supporting this interpretation is feeble. Patients with favism are always G6PD deficient, but not all G6PD-deficient individuals develop hemolysis when they ingest fava beans.[8] Thus, G6PD deficiency is a necessary but not sufficient cause of favism. Presumably some other factor, probably also genetic and very likely related to metabolism of the active ingredients in the beans, is involved. The vast majority of cases of favism occurs in individuals with severely deficient (class 2) variants of G6PD, but occasionally favism has been observed in a patient with G6PD. Although at times the onset of hemolysis in favism may be more explosive than occurs as a result of drug administration, in general the course of hemolysis in favism is very similar to that occurring after drug ingestion.[9]Hemolysis does not usually begin for 24 hours after ingestion of the beans and hemoglobinuria may continue for several days.

Mechanism of Hemolysis

The mechanism by which drugs and fava beans produce hemolyticanemia is not well understood. Such drugs do not lyse RBCs in vitro.[10] Instead, they appear to inflict oxidative injury on the erythrocytes and, therefore, are often designated as oxidative drugs. Because of its relatively high frequency in some areas in the Mediterranean region, the mechanism by which fava beans produce hemolysis has received special attention, with the suggestion that the pathogenesis of favism and drug-induced hemolyticanemia may be essentially the same.[11]Vicine, convice, ascorbate, and L-DOPA are abundant in fava beans and have been considered candidate toxins. The most likely offenders are vicine and convicine, &glucosides of pyrimidine compounds that are converted by glucosidases to their aglycones, vicine and isouramil, respectively. These compounds form reactive semiquinoid-free radicals and can generate active oxygen species. This results in the formation of ferrylhemoglobin, methemoglobin, and inactivation of various enzymes. The reactions that occur are complex and varied and therefore, largely unpredictable.[12]

Infection-Induced Hemolysis

Although, for historical reasons, drug-induced hemolysis has attracted the most attention, it is likely that hemolysis induced by infection may be a more common cause of clinically significant hemolysis.[13] Numerous reports attest to the importance of infection in causing hemolyticanemia. It is clear that many different types of infections may trigger hemolysis in the G6PDdeficient patient. The mechanism by which this occurs is not clear, but an imaginative suggestion has been that during phagocytosis, leukocytes damage erythrocytes in their environment by discharging active oxygen species during phagocytosis. Perhaps nitric oxide might also play such a role.[14] It is unlikely that such a mechanism is operative in the case of viral infections such as hepatitis, but it may play a role in some infections.

Diabetes Mellitus-Induced Hemolysis

It has been suggested that episodes of diabetic acidosis may precipitate hemolytic episodes in persons with G6PD deficiency, but in one study no evidence was found that such an effect existed.[15] It has also been reported that hypoglycemia may precipitate hemolysis in G6PD deficiency.

Hereditary Non-SpherocyticHemolyticAnemia

It was in 1958, not long after G6PD deficiency was identified as the cause of primaquine sensitivity, that it was recognized that the enzyme deficiency could cause chronic hemolysis as well.[16] The syndrome of hereditary nonspherocytichemolyticanemia did not occur in persons who inherited the common, polymorphic variants of G6PD such as G6PD A- or G6PD Mediterranean, but rather in patients who had inherited rare mutations, designated class 1 because of their association with chronic hemolysis. The severity of hemolysis varies greatly. Although it is usually mild, the patient with G6PD Campina has transfusion-dependent hemolysis resembling thalassemia major. Presumably class 1 variants produce chronic hemolysis because the functional severity of the defect is so great that the erythrocyte cannot even withstand the normal stresses that it encounters in the circulation.[17] The functional severity in these patients is not usually reflected by the level of the enzyme as it is measured in the laboratory. The RBCs of patients with class 1 variants may have residual G6PD activity as high as 35% of normal when measured under standard conditions. The functional impairment that leads to the shortening of the RBC life span in these patients may

include such factors as susceptibility to inhibition by NADPH and in vivo lability.[18] Possibly the most consistent common feature of class l variants is the location of the mutation. In the great majority of cases, it is in the region of the putative NADP-binding or glucose-6-phosphate binding site of the molecule.

Neonatal Jaundice

Neonatal jaundice is one of the most life- and health threatening consequences of G6PD deficiency, and kemicterus may occur in these It is often erroneously assumed that the jaundice is the result of hemolysis.[19] However, this is apparently not usually the case. Anemia is not present in G6PD-deficient infants that develop neonatal icterus. Instead of the icterus being a manifestation of accelerated RBC destruction, it now seems likely that it is largely the result of the impairment of liver function, presumably because of a deficiency of the enzyme in the liver.[20] It is entirely possible that some shortening of RBC life span also plays a role. Neonatal jaundice has occurred primarily in Asian and Mediterranean infants. In one study, G6PD Aures has been associated particularly high incidence of jaundice. Early reports from the United States suggested that African-American infants did not have a significantly increased incidence of neonatal jaundice. However, anecdotal observations from the United States and surveys in Jamaica and in Africa all suggest that an increased incidence of neonatal icterus may occur also in infants with G6PD A-.[21]

Transfusion with G6PD-Deficient Blood

There is evidence that G6PD-deficient RBCs maintain viability less well than do normal cells even without being subjected to oxidative stress. However, the consequences of transfusing a single unit of G6PD-deficient RBCs into an adult are probably minor.[22] It has been pointed out that in the case of G6PD A-, the number of cells that would be destroyed if a hemolytic stress occurred would be no greater than the number of nonviable cells in a unit of blood nearing its expiration date. Transfusion of G6PD-deficient blood may be an issue of greater potential importance in parts of the world in which the incidence of the defect is very high and where more severely deficient class 2 variants such as G6PD Mediterranean are prevalent.[23] In such areas, it is possible for a patient to receive, by chance, several units of deficient blood. In one instance, it has been suggested that fatal hemolysis occurred in a young woman as a result of receiving G6PD-deficient blood, but the reports of such severe consequences are not themselves convincing of a cause-andeffect relationship. In a controlled study, only minor increases in bilirubin levels were found in individuals receiving a unit of

severely G6PD-deficient blood, but the changes might be greater if a hemolytic stress were present.[24] In general, it has not been the practice to screen blood bank blood for G6PD deficiency, even in areas in which the gene frequency is very high. However, caution is justified in the exchange transfusion of newborn infants. Here, in contrast to adults, the proportion of deficient cells could be very high, and the products of Hb catabolism disposed of inefficiently by the immature liver.[25]

Other Manifestations of G6PD Deficiency

It is reasonable to assume that a genetic trait that has reached such high frequencies in many populations that it is carried by some 200,000,000 persons would not have a readily apparent effect on fitness. For this reason, if no other, it has been generally assumed that those who carry polymorphic genes for G6PD deficiency would not suffer from any morbidity.[26] Nonetheless, a number of studies have suggested that G6PD-deficient individuals might, even in the absence of any stress, have some clinical abnormalities.

Tissue Distribution of The Deficiency

Early studies indicated that the deficiency of G6PD activity was limited to the RBCs; liver and leukocyte activity was reported to be normal, and it was even suggested that the defect might not be in the G6PD gene itself, but rather some other gene that influenced the stability of the enzyme in the erythrocyte.[27] Platelet activity was found to average about 40% of normal. These studies were probably performed on patients with G6PD A-, and subsequent investigations in patients with more severely deficient variants indicated that other tissues were indeed involved in G6PD deficiency.

Life Expectancy

Large-scale studies have assessed the effect of G6PD A- on the overall health of Afro-American veterans. In an investigation of 1,413 black males Petrakis et al found that the incidence of G6PD deficiency was 12.1% in the 5- to 20-year age group, 5.6% in the 21 to 49 year age group, and only 3.8% of those above the age of 49.[28] While acknowledging that there might be a number of explanations for this, they concluded that G6PD-deficient subjects had a reduced life span.[29] However, this seems unlikely in view of the fact that it would require a very high excess mortality rate among persons with G6PD deficiency. Indeed, a study of 65,154 black male patients admitted to US Veteran's Administration

hospitals showed no increased mortality among patients who were G6PD deficient and no significant difference in the mean ages of G6PD-deficient and nondeficient patients.[30]

Cancer

Epidemiologic studies suggested to some that the incidence of cancer may be lower in G6PD-deficient person. However, these investigations were generally based on screening methods that do not efficiently ascertain G6PD deficiency in heterozygotes, and even in hemizygotes who have a disorder that might decrease lU3C life span.[31] Indeed, in one study it was shown that the RBC G6PD activity of cancer patients is higher than that of controls. More recent studies tend not to show any differences between the incidence of cancer in G6PD-deficient and normal subjects.[32]

Diagnosis

Detection of G6PD deficiency

Before the underlying defect, G6PD deficiency, had been uncovered, two methods for detecting individuals sensitive to the hemolytic effect of primaquine had been developed, the Heinz body test and the GSH stability test.[33] Although still occasionally used, these surrogate tests are obsolete and no longer have a role in the diagnosis of G6PD deficiency.[34]Instead, quantitative assays or screening tests that detect severe deficiency should be used to diagnose the disorder.

Quantitation of G6PD Activity in Erythrocytes

The simplest type of quantitative assay measures the reduction of NADP to NADPH in the presence of glucose-6-P and hemolysate.[35] In reality, this type of assay measures both G6PD and 6-phosphogluconate dehydrogenase (6-PGD) activity.[36] In the reaction mixture, as in the cell, the immediate product of the G6PD reaction, 6-phosphogluconolactone is converted to 6-phosphogluconate which serves as substrate for the 6- PGD reaction. Thus, 2 moles of NADP are reduced for each mole of glucose-6-P consumed in the mixture.[37]Although methods that measure G6PD activity independently of 6- PGD deficiency have been available for many years, such methods have little additional utility in diagnosing the deficiency state, because 6-PGD does not usually limit the rate of the reaction, particularly in G6PD-deficient individuals.[38]

Screening for G6PD Deficiency

In hemizygous males who are not undergoing hemolysis, as will be found in population surveys, semi-quantitative or nonquantitative screening methods are entirely adequate.[39] Dye reduction tests, tirst introduced by Motulsky and Campbell-Kraut as the brilliant cresyl blue decolorization test, have been widely used.[40] Other receptors for the electrons from NADPH generated in the G6PD and 6-PGD reactions include methylene blue, MTT tetrazolium, and methemoglobin.[41] A test in which protection against denaturation of Hb under oxidative stress serves as an endpoint has also been developed.[42] Although all of these tests are still sometimes used, particularly in population surveys, they have largely been replaced by the fluorescent spot test, in which the generation of NADPH is detected directly visually under ultraviolet light.

Detection of G6PD Deficiency in Patients Undergoing Hemolysis.

While the diagnosis of deficient males ordinarily poses no special difficulties, the same cannot be written about the detection ofG6PD deficiency in patients with some of the milder G6PD-deficient variants (class 3) undergoing a hemolytic episode.[43] Because the older members of the RBC population are selectively removed in patients with variants such as G6PD A-, leaving the younger cells with nearnormal activity in the circulation, a screening test may give quite normal results, at least for a week or two after the hemolytic episode. The same problem in diagnosis does not exist in the case of severe (class 2) variants because in these variants, even the very young cells are severely enzyme deficient.[44]

Several different approaches may be used to diagnose patients who have just undergone hemolysis. The simplest is merely to wait for a week or two or to perform family studies. Alternatively, one may deplete the sample being studied of reticulocytes by centrifugation. The denser cells, although not truly old as has sometimes been believed, are depleted of very young RBCS.[45] Accordingly, it has been found that even during hemolysis, the dense fraction of cells is G6PD deficient. Another approach is to compare the activity of G6PD with that of another age-dependent RBC enzyme such as hexokinase or glutamic oxaloacetic transaminase. This approach has been used also to detect the G6PD A- genotype in patients with sickle cell disease, in which the mean RBC age is greatly decreased. The most powerful approach for establishing the diagnosis in the context of hemolysis is analysis of genomic

DNA obtained from circulating leukocytes . Neither the presence of young erythrocytes nor, for that matter, of transfused cells confounds the results obtained from such an analysis.[46]

Treatment

When hemolytic episodes occur in G6PD-deficient individuals, the inciting agent, drug or infection, should be removed whenever possible. However, in patients who have class 3 variants such as G6PD A-, it may be possible to continue essential drug therapy with careful monitoring of the blood count.[47] Blood transfusion is only occasionally required to support patients who have undergone severe hemolytic episodes, usually in patients with favism.

It has been suggested that attacks of favism may be ameliorated by the administration of desfemoxamine. In one study patients with favism who received a single 500-mg dose of desfemoxamine and packed RBC transfusions had a shorter duration of hemoglobinuria, greater rise in Hb level and more rapid drop in reticulocyte count than control patients who received packed cells alone.[48] However, it was not clear that both groups received the same volume of transfusion.

To permit NADPH to be produced by a different route, xylitol administration has also been proposed as a way to prevent or treat hemolysis of G6PD deficiency. Clinical studies in which two severely G6PD-deficient volunteers were pretreated with 10 g xylitol per day and then given primaquine and 20 g xylitol per day showed no protection against hemolysis.[49]

It has been suggested that vitamin E, by virtue of its antioxidant effect, might protect against chronic hemolysis in G6PD deficiency causing chronic hemolyticanemia. Some studies have shown a favorable response to this vitamin; others have not.

The most dangerous consequence of G6PD deficiency is neonatal icterus. Kernicterus has been documented repeatedly in populations in which class 2 variants are common and it has been pointed out this is an important preventable form of mental retardation. Phototherapy has been used to reduce bilirubin levels, and phenobarbital has been used prophylactically with some success.[50] Agar, given to reduce bilirubin reabsorption, was found to be ineffective. When the bilirubin level exceeds 20 mg/dL exchange of transfusion is required but G6PD-deficient blood should not be used for this purpose.

Corresponding Author

Sivakumar M

Senior Lecturer, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences,

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India

Telephone number: +919443602627

Email id: drsivaomfs@gmail.com

References

1. Beutler E.The hemolytic effect of primaquineand related compounds. J Pharm. 1997;14:103

2. Carson PE, Flanagan CL, Ickes CE, Alving AS.Enzymatic deficiency in primaquine-sensitive erythrocytes. Science 1956;124:484.

3. Zanella A, Colombo MB. Rossi F, Merati G, SirchiaG.Congenital non-spherocytic haemolytic anaemias. Haematologica.1989;53:23-101

4. BeutlerE. Glucose-6-phosphate dehydrogenase deficiency and other enzyme abnormalities, in Beutler E. Lichtman MA, Coller BS, KippsTJ : Williams Hematology. New York, NY, McGrawHill. 1995;12;30-564

5. Valentine WN.Erythroenzymopathies and haemolytic anemia: The many faces of inherited variant enzymes. J Lab Clin Med 1990;115:12

6. Valentine WN, Tanaka KR, Paglia.Hemolyticanemias and erythrocyte enzymopathies. Ann Intern Med 1998;103:245

7. Persico MC, Viglietto G, Martino G, Toniolo D, Paonessa G, Moscatelli C, Dono R, Vulliamy T, Luzzatto L, D'Urso M: Isolation of human glucose-6-phosphate dehydrogenase (G6PD) cDNA clones: Primary structure of the protein and unusual 5' non-coding region. Nucleic Acids Res 1991;14:25.

8. Takizawa T, Huang IY, lkuta T, Yoshida. A Human glucose6-phosphate dehydrogenase: Primary structure and cDNA cloning. Proc Natl Acad Sci. 1996;83:4157.

9. Beutler E. The red cell A tiny dynamo, in Wintrobe MM. Blood Pure and Eloquent, New York, NY, McGraw-Hill.1980;141:10-27

I0. Dern RJ, Beutler E, Alving.The hemolytic effect of primaquine. J Pharm, 1982:23;12-45

11. The natural course of the hemolyticanemia and the mechanism of its self-limited character. J Lab Clin Med 1956;44:171

12. Fischer TM, Meloni T, Pescarmona GP, Arese P. Membrane cross bonding in red cells in favic crisis: A missing link in the mechanism of extravascular haemolysis. Br J Haematol 1985:9:132-201

13. Betke K, Beutler E, Brewer GJ, Kirkman HN, Luzzatto L, Motulsky AG, Ramot B, Siniscalco.Standardization of procedures for the study of glucose-6-phosphate dehydrogenase.Report of a WHO scientific group.1967;12:871-1001

14. Beutler E, Dern RJ, Alving.The hemolytic effect of primaquine. IV. The relationship of cell age to hemolysis. J Lab Clin Med 1954;44:439

15. Piomelli S, Corash LM, Davenport DD, Miraglia J, Amorosi .In vivo lability of glucose-6-phosphate dehydrogenase in GdA and Gd Mediterranean deficiency. J Clin Invest 1968;47:940

16. Pannacciulli I, Tizianello A, Ajmar F. SalvidioE.The course of experimentally-induced hemolyticanemia in a primaquine- sensitive Caucasian.A case study. Blood 1965;25:92.

17. George JN, Sears DA, McCurdy P, Conrad ME.Primaquine sensitivity in Caucasians: Hemolytic reactions induced by primaquine in G-6-PD deficient subjects. J Lab Clin Med 1967;70:80.

18. Dern RJ, Beutler E, Alving**.**The hemolytic effect of primaquine. V. Primaquine sensitivity as a manifestation of a multiple drug sensitivity. J Lab Clin Med 1955;45:30.

19. Beutler E.HemolyticAnemia in Disorders of Red Cell Metabolism. New York, NY, Plenum, 1978;23:16-78

20. Woolhouse NM, Atu-Taylor**.** Influence of double genetic polymorphism on response to sulfamethazine. Clin Pharmacol Ther 1982;3:377.

21. Magon AM, Leipzig RM, Zannoni VG, Brewer**.**Interactions of glucose-6-phosphate dehydrogenase deficiency with drug acetylation and hydroxylation reactions. J Lab Clin Med 1981;97:764

22. Kellermeyer RW, Tarlov AR. Brewer GJ, Carson PE, Alving**.**Hemolytic effect of therapeutic drugs.Clinical considerations of the Primaquine-type hemolysis.1962;180:388.

23. Chan TK, McFadzean AJS. Haemolytic effect of trimethoprim: sulphamethoxazole in G-6-PD deficiency. Trans R Soc Trop Med Hyg 1974:68:67.

24. Markowitz N. Saravolatz**.**Use of trimethoprim-sulfamethoxazole in a glucose-6-phosphate dehydrogenase-deficient population. Rev Infect Dis 1987;12:54:218

25. Lakshmi. Haemolytic crisis after acetaminophen in glucose-6-phosphate dehydrogenase deficiency. KlinWochenschr 2012;67:1068

26. Sarbeen JI, Sethu G. Glucose-6-Phosphate Dehydrogenase Deficiency. Research Journal of Pharmacy and Technology. 2015 Jun 1;8(6):792.

27. Beutler E: Acetaminophen and G-6-PD deficiency. Acta Haematol 1984;72:211

28. Casassus P, Vannetzel JM, Lortholary P:Hemolysedeficitent glucose 6 phosphate dehydrogenase treatment d'un myelome role du melphalan. Nouv Press Med 1982;11:2296

29. Janakiraman N, Seeler RA, Royal JE, Chen MF.Hemolysis during BAL chelation therapy for high blood lead levels in two G6 PD deficient children. Clin Pediatr. 1978;17:485.

30. Doll DC: Oxidative haemolysis after administration of doxorubicin. BMJ. 1983;287:180.

31. Michot F, Rastetter J, Gronauer H: Durch Neo-salvarsanausgelosteHamolysebei Glukose-6-phosphat-dehydrogenase-mangel, kombiniertmithepatischem Icterus. Schweiz Med Wochenschr. 1996;96:985

32. Arie THD: Pythagoras and beans. Oxf Med School Gaz 1959:75:11-30

33.Stamatoyannopoulos G, Fraser GR, Motulsky AG, Fessas P, AkrivakisA,amyloidosis. Am J Hum Genet. 1966; 18:253

34. Calabro V, Cascone A, Malaspina P, Battistuzzi G: Glucose6-phosphate dehydrogenase (G6PD) deficiency in southern Italy: A case of G6PD A(-) associated with favism. Haematologica. 1999; 74:71

35. Galiano S, Gaetani GF, Barabino A, Cottafava F, Zeitlin H, Town M, Luzzatto L: Favism in the African type of glucose-6-phosphate dehydrogenase deficiency (A-). BMJ 1990; 300:236,

36. Sansone G, PigaAM, Segni G.Favismo. Torino, Minerva Medica, 1958;50:45-124

37. Beutler E, Dern RJ, Alving AS: The hemolytic effect of primaquine. 111. A study of primaquine-sensitive erythrocytes. J Lab Clin Med. 1954; 44:177.

38. Repine JE, Eaton JW, Anders MW, Hoidal JR, Fox RB: Generation of hydroxyl radical by enzymes, chemicals, and human phagocytes in vitro. J Clin Invest 1979;64:1642.

39. Rakitzis ET, Papandreou FT: Ascorbate-induced generation of free radical species in normal and glucose-&phosphate dehydrogenase-deficient erythrocytes. Biochem SOC Trans 1989;17:371

40. Niki E, Komuro E, Takahashi M, Urano S, Ito E, Terao K. Oxidative hemolysis of erythrocytes and its inhibition by free radical scavengers. J Biol Chem 1988;63:19-304

41. Saltman P: Oxidative stress: A radical view. Semin Hematol 1989;26:249.

42. Mehlhom RJ: Ascorbate- and dehydroascorbic acid-mediated reduction of free radicals in the human erythrocyte. J Biol Chem. 1981;66:27;24.

43. Davies KJA, Goldberg AL: Oxygen radicals stimulate intracellular proteolysis and lipid peroxidation by independent mechanisms in erythrocytes. J Biol Chem 1987;262:8220.

44. Hochstein P: Perspectives on hydrogen peroxide and drug induced hemolyticanemia in glucose-6-phosphate dehydrogenase deficiency. J Free Radical Biol Med 1988;5:387

45. Gaetani GD, Mareni C, Ravazzolo R, Salvidio E: Haemolytic effect of two sulphonamides evaluated by a new method. Br J Haematol 1976;32: 183.

46. Bashan N, Peleg N, Moses SW: Attempts to predict the haemolytic potential of drugs in glucose-6-phosphate dehydrogenase deficiency of the Mediterranean type by an in vitro test. Isr J Med Sci 1998;24:61.

47. Bloom KE, Brewer GJ, Magon AM, Wetterstroem N: Microsomal incubation test of potentially hemolytic drugs for glucose-6-phosphate dehydrogenase deficiency. Clin Pharmacol Ther 1983;33:403

48. Mela Q, Perpignano G, Ruggiero V, Longatti S: Tolerability of tiaprofenic acid in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency. Drugs 1998;35:107.

49. Smith JE: Low erythrocyte glucose-6-phosphate dehydrogenase activity and primaquine insensitivity in sheep. J Lab Clin Med 1968;71:826.

50. Horton HM, Calabrese EJ: Predictive models for human glucose-6-phosphate dehydrogenase deficiency. Drug Metab Rev 1989;17:261.

General Dental Practitioners Knowledge Of Dental Radiography Among Dentist In Chennai ,India.

Vijayaalaksmi.L.G[1].,Shantha Rajan [2], Jayanth Kumar [3]

Graduate Student [1], Senior Lecturer [2] ,Department Of Pedodontics,

Senior Lecturer [3] ,Department Of Oral Medicine,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Aim:
The aim of this research is to evaluate the general dental practitioners knowledge of dental radiography .

Objective:
Dental radiographs is one of the principal diagnostic methods used in dentistry.The safe and effective use of x-ray equipment is important to the protection of the patient and members of the dental team.

Materials and methods :
The data was collected from 50 general dental practitioners at self convenience using a structured questionnaire consisting of 9 item questionnaire.The sample was selected based on inclusion criteria and exclusion criteria.Inclusion criteria includes undergraduate,postgraduate and chennai dental practitioners. The exclusion criteria includes the students and dental radiograph assistants.
In the questionnaire, details were asked about the age of the intraoral radiographic unit, kVp and mA of the equipment, the type of timer on the machine and about the geometry of the machine and the geometric technique used for intraoral radiography.Furthermore, the questionnaire contained questions about the type of intraoral detectors that were used and whether the dentists were also using panoramic machines and, if so, which type of detector they had. Among the possible answers, one option was ‘‘no idea’’.The data thus obtained was subjected to statistical evaluation.

Conclusion:
The results of this study showed that the knowledge of dentist in South Chennai regarding dentomaxillofacial radiology is excellent.The level of awareness of practitioners regarding radiation hazards and safety was found to be acceptable.

Keywords:*general practitioners,knowledge, radiology, radiation protection,exposure.*

Introduction

As a dental professional, the utilization of radiograph for finding and further treatment is significantly more regular than different fields of medicine.It is accounted for that around

45% of dental patients require radiographs for determination. [1]Dental radiographs is one of the essential demonstrative techniques utilized as a part of dentistry.Intra oral and extra oral radiographs are two sorts of radiographs available.The sheltered and powerful utilization of x-beam hardware is imperative to the assurance of the patient and individuals from the dental team.Its assumes a critical part in the identification of oral disease.[2]Exposure to such radiation is related with an expanded hazard in the long haul of threatening sickness in those persons.Furthermore, it is accepted that the likelihood of event of these antagonistic impacts is straightforwardly corresponding to the level of presentation, with no measurements threshold.The dangers related with the vital introduction to ionizing radiation might be generous, and must be limited through careful adherence to great practice.[3]

Specialized advances in radiological equipment significantly affect lessening of radiation dosages to patients during intra oral and extra oral radiographs. During extra oral radiography, the utilization of uncommon earth escalating screens has diminished the radiation presentation significantly.[1,4,5]Although the quantity of radiographs taken every day in essential dental care is over the top, the most serious hazard emerges when there is proof of poor picture quality or non-diagnostic images inferable from poor specialized information and the insufficient handling of films [6-10]. It has been evaluated that the disposal of non-beneficial examinations could prompt a 30% lessening in the aggregate populace measurements, got from medical radiology.[11] Dental hygienists assume an imperative part in handling and they should accept the accountability for poor preparing rehearses, the majority of which concern not supplanting preparing arrangements as oftentimes as required, overdeveloping and overfixing. Accordingly, numerous dental practitioners tend to build the presentation time to make up for the disgraceful preparing, therefore expanding the radiation measurements to patients. [12]

For dispensing with unnecessary X-beam examinations, each expert who works X-beams ought to be appropriately prepared as per the Ionizing Radiation Medical Exposure Regulations 2000.[13] We live in an ocean of radiations. We are constantly exposed to naturally occurring ionizing radiation i.e.,background radiation and furthermore presented to ionizing radiation from manmade sources, for the most part through medical procedures.On an average, doses from a diagnostic X-ray are much lesser, in dose effective terms, than the natural background radiation.Radiation has turned into a part of present day living, achieving each section of our general public. The essential hazard from dental radiography is radiation actuated malignancy. The writing on conceivable destructive impacts of expert analytic presentation for dental specialists isn't steady. The hazard required with dental radiography is surely little in correlation with numerous different dangers that are a typical piece of regular daily existence. In any case, no premise exists to accept that it is zero. Likewise, the biologic impacts of ionizing radiation ingested during dental radiography are questionable. The radiographic examination utilized as a part of all fields of medicinal administrations and adds to the advancement of the well being, both separately and broadly. Radiographic examination has a basic impact of dental practice. Certain measure of radiation is unavoidably conveyed to patients, it ought to be as low as reasonably achievable [ALARA]. International Commission for Radiation Protection [ICRP] is the administrative body which sets down

standards for radiation security at the universal level. In India, it is the Atomic Energy Regulatory Board [AERB] which gives the standards to radiation assurance.

AERB prescribes standards for reasonable dosages of radiation from X-beam tubes, the protecting required for the dividers of a X-beam tube room, the lead equal protecting attire to be worn by radiation specialists and sets down safe measurements limits for radiation laborers and for the overall population.Before undertaking any radiological examination, it is important that the provider comprehends the potential dangers and the advantages of radiation. The dangers can be stochastic [of which likelihood increments with dosage] and deterministic [of which severe increments with measurement].Cancer induction and hereditary effects are stochastic impacts and blood dyscrasias,cataracts and imparired fertility are cases of deterministic effects.Biological effects of ionizing radiation are gathered into stochastic and deterministic effects. Over a specific decided dosage of radiation organic harm starts to show up, this is called as deterministic impact. Stochastic impact is that in which there is no specific dosage level above which natural change happens in the body.

Ionizing radiation causes both the impacts relying upon radiation doses and body's reaction to these radiations. Dental practitioners and in addition patients are more inclined to the danger of stochastic effects because of its lack of a dose threshold limit. The positive parts of diagnosis of disease and detection of disease should be considered while assessing the risk of impacts of radiation. It is essential for dental practitioner to refresh their insight about new patterns in analytic strategies, protective measures, etc.This can be accomplished by methods for proceeding with training, continuing education, journals,workshops, and other media.In India, diagnostic radiation facilities are administered by Atomic Energy Regulatory Board [AERB].The role of the AERB is to guarantee that utilization of ionizing radiation and atomic vitality in India does not make undue hazard the well being of individuals and the environment.It is required to enlist all diagnostic radiation facilities in e-Licensing of Radiation Application [eLORA] system of AERB. From December first, 2013, it is mandatory for dental specialists and dental institutions to enroll in eLORA and get a license to operate dental X-ray units, panaromic machines, and cone bean computed tomography. It is additionally essential for manufacturers of X-ray machines to get a license available to be purchased in India by AERB.

The current radiation protection standards are based on 3 principles : Justification of a practice, optimization and dose limitation.In spite of the fact that the radiation dosage levels in dental practice are generally low, one ought to think about the combined impact of repeated exposures. There ought to be a making progress toward radiation assurance measures in the private dental offices.[14]There is an association between knowledge and the use of low-dose techniques and attitudes towards risks.[15]A dental professional should always be prepared to improve his or her knowledge through continuous education, it is a well-known fact that this is more likely to occur if the continued education is mandatory for all dental professionals, for whatever local or federal reasons.[16]

The reason for performing this study was to alert the dental professional societies that more attention should be paid to radiation protection in dentistry in our country. The aim of this research is to evaluate the general dental practitioners knowledge of dental radiography .

Materials and method

Data was collected from 50 general dental practitioners using a structured questionnaire consisting of 9 item questionnaire.The sample was selected based on certain inclusion and exclusion criteria.

Inclusion criteria :

1. Undergraduate
2. Postgraduate
3. Chennai dental practitioners

Exclusion criteria:

1.Students

2.Dental radiograph assistants

In the questionnaire, details were asked about the age of the intraoral radiographic unit, kVp and mAofthe equipment, the type of timer on the machine and, about the geometry of the machine and the geometric technique used for intraoral radiography.Furthermore, the questionnaire contained questions about the type of intraoral detectors that were used and whether the dentists were also using panoramic machines and, if so, which type of detector they had. Among the possible answers, one option was ‘‘no idea’’.The data thus obtained was subjected to statistical evaluation.

Result

Table-1 responses from the study subjects to the questionnaire in percentage[%]

Intra - Oral Radiographic Machine	Condition	Percentage
Practice dates from	After 2000	100%
Works at (Kv)	70Kv	20%
Works at (Kv)	60Kv to 70Kv	80%
Works at (mA)	4 or 8mA	92%
Works at (mA)	No Idea	8%
Timer	Digital Timer	100%
Shape	Long Cone	6%
Shape	Short Cone	94%
Rectangular Collimator	Yes	20%
Rectangular Collimator	No	80%
Parallel Technique	Yes	18%
Parallel Technique	No	82%
Works with	Digital Censor - CCD	24%
Works with	Digital Censor - PSPP	76%
Panaromic Machine Disposal	No	10%
Panaromic Machine Disposal	Yes - Analogue Film	80%
Panaromic Machine Disposal	Yes - CCD Technology	10%

Discussion

As mentioned above the data was collected from 50 general dental practitioners using a structured questionnaire consisting of 9 item questionnaire.The sample was selected based on certain inclusion criteria and exclusion criteria.Inclusion criteria which includes the Chennai dental practitioners,undergraduates,postgraduates and Exclusion criteria includes Students,dental radiograph assistants.Regarding the age of the intraoral radiographic equipment used by the questioned population of chennai dentists, it was found that almost 100%[50 dentist] were using radiographic machines for intraoral radiography equipment that was after 2000.

As the answers about the tube voltage of the intraoral equipment was mild difference the answers were condensed into two groups. Therefore, tube voltage ranging from 60 to 70 kV was considered as one category. 80%[40 dentist]of the equipment used in South chennai dentist was reported to operate between 60 and 70 kVp. ,whereas 20%[10 dentist] claimed to be working at a tube voltage of 50 kVp.

The majority of the South Chennai dentists were using 4 or 8mA[easy to change] equipment for intraoral radiographs ; 8%[4 dentist] claimed they not knew what they were working .

100% [50 dentist]claimed to be working with a digital exposure timer, whereas none said they were still working with a manual [clock-like] exposure timer.

It was found that 94% [47 dentist]said that they were working with a short cone geometry, whereas 6% [3 dentist]claimed to be working with a long cone geometry.

20% [10 dentist]said they were using a rectangular collimator.80%[40 dentist] responded that they were using circular collimatior.

18%[9 dentist]claimed to be working with the parallel technique, whereas 82%[41dentist] said they were working with the bisecting angle technique.

76%[38 dentist]of the dentists in South Chennai were still using PSPP sensor films for intraoral radiography. 24%[12 dentist] had digitized to intraoral sensors as charge coupled device [CCD].

About 10% [5 dentist]of the dentists has no intention idea what sensor they using.10%[5 dentist] of South Chennai dentist don't not posses a dental Panaromic machine. Others 80% use analogue films and 10% [5 dentist]use CCD technology.

A dental professional should know his or her equipment and it is expected to know the main guidelines on radiation protection.The overall conclusion of this study,is that a great deal of work done to ameliorate the quality of radiographs and the knowledge and attitude of general dental practitioners regarding dentomaxillofacial radiology.[2] This is a task for dentomaxillofacial radiology specialists, who will have to concentrate in the first place on undergraduate teaching and then on postgraduate teaching. The latter is not less important than the first as the working dentist population needs to change their attitude regarding radiation protection and dentomaxillofacial radiology general knowledge. [3]This has also been stressed in other surveys. From the results ,it is evident that continued education in dentomaxillofacial radiology is essential, especially when considering a change from analogue to digital radiology.[4]That 92%[46 dentist]of the dentists in the present survey claimed to be working with a short cone radiographic machine is probably biased by the fact that they are not aware of the difference between a short cone and a short spacer cone, which

is also called a beam indicating device or position indicating device.[17]It can be assumed that dentists owning a machine with a short spacer cone, because the manufacturer positioned the focus near the rear end of the machine, gave the wrong answer. Therefore this result should be interpreted with care. The opposite for long cone could also be the case, of course.
[18]The results are, therefore , excellent.This could mean, for instance,increased in the number of hours of under and /or postgraduate education. If this can be decided by the local authorities and enforced at university level, it had a chance of succeeding. However, at present, every university has its own curriculum.And as known from other studies, it is not always easy to change curricula and convince faculty staff colleagues of this issue.[19]
Regarding the knowledge of Chennai's general dental practitioners, there are various reasons for the results of this questionnaire being excellent . Perhaps the quality and quantity of the undergraduate education is good.However, it is the personal impression that, for many years, dentomaxillofacial radiology has been taught at every university in the country by medical radiologists or general dentists with specific dentomaxillofacial radiology training.The major problem with the undergraduate education is the number of hours that should be dedicated to dental radiology.The age of the dentists was not assessed and also the university where they had studied. Age was not considered an important question in this study, as the age of a dentist does not always reflect that person's knowledge of dental radiology.Furthermore, it is known that not every young dentist starts with new equipment and, vice versa, that not every "older" dentist is working with old equipment. Therefore, the age of the intraoral radiographic equipment was considered more important and more relevant as a question.[20]The level of knowledge and the attitude of dentists regarding radiation safety will have a direct impact on patient exposure to radiation [Shahab et al. 2012]. Many studies have been done to determine the knowledge and attitude of dentists regarding radiation safety.
Shahab et al reported a study pertaining to radiation safety to assess the knowledge of dentists with regard to basic information in relation to radiation protection and methods of reducing the radiation dose to the patient. The majority of dentists did not employ appropriate procedures to decrease exposure to unwanted radiation.[21]
Mutyabule and Whaites conducted a study to assess radiation protection measures in dental practices in Uganda. It was found that operators lacked sufficient knowledge regarding radiation safety protection measures.[22]
Apsassessed dentists' knowledge regarding radiation safety. The dentists were asked about various methods of dose reduction to the patients. The results of the study highlighted the need to increase knowledge of dental practitioners regarding radiation safety and methods of dose reduction.[23]
Lee and Ludlow assessed the attitude of dentists regarding radiation safety. The dentists were asked about primary knowledge of radiation safety and the method of reducing the dose to the patient such as speed of the film, collimation of the X-ray tube and the regular use of shielding. Results of the study confirmed that there is a demand to reinforce the dentist's working knowledge about the issue of radiation safety.
Math et al assessed the understanding of dentists regarding radiation safety standards with regards to the X-ray machine, collimation of the tube, regular use of a film holder, shielding

and the position of the operator during radiation exposure. The result of this study also highlights the need to increase the practitioner's awareness and attitude regarding radiation hazard and use of appropriate methods to reduce the radiation dose.[24]

Jacobs et alconducted a study regarding the perceptions of the dentists of radiation protection. The results of this study concluded that there was a need to apply strict guidelines toward radiation safety.[25]

Majority of the practitioners still followed the position and distance rule, few were found to be using lead barriers while mere used lead aprons and some using a combination of various safety techniques. Distance in radiation protection refers to distance from the source and the individual. As the distance increases, radiation exposure reduces. Moreover, shieldingincludes both protective barriers such as lead shield and personnel protective measures such as lead apron. Ninety-eight percent reduction in scattered radiation and attenuate dose to 0.04 µR can be achieved using lead aprons.Patient should wear thyroid collar during radiation exposure as it reduces attenuation of scattered radiation to 92%[46 dentist].Proper shielding from radiation and by increasing the distance from source protect radiographer as well as patient for unnecessary exposure to radiation. According to position distance rule, radiographer position should be at least 6 feet from the source at an angle of 90 to 135° to the central ray of X-ray beam.

Conclusion

The results of this study showed that the knowledge of dentist in South Chennai regarding dentomaxillofacial radiology is excellent.This emphasizes that there is better under and postgraduate education in dentomaxillofacial radiology in Chennai. The present report is therefore an important statement for the dental professional society.The level of awareness of practitioners regarding radiation hazards and safety was found to be acceptable. However, implementation of their knowledge with respect to patient and personnel safety was found wanting. Insisting that they follow the protocols and take necessary safety measures by means of continuing medical education programs, pamphlets, articles, and workshops is strongly recommended.From the time of its discovery, X-rays have played a vital role in the field of medical and dental science. Ranging from diagnostic to therapeutic applications, the use of X-rays is manifold. Probably, the most widespread application is in the field of dentistry from the simple diagnosis of incipient caries, multiple fractures to aiding in more complex procedures such as precision implant planning. The modalities at the disposal of dentists range from intraoral radiography to cone beam computed tomography. During the course of their training, all health-care personnel are trained regarding radiation hazards and requisite safety measures. However, the sincerity with which the matter is considered needs to be assessed from time to time. In the present study, we attempt to evaluate the awarenessand validate the knowledge of radiograpy among general dental practitioners in Chennai, India.Protection of one's self and patient from all kinds of health hazards is the hallmark of concerned doctors. The AERB recommendations should reach out through the dentists'

platform [e.g., IDA] to the dental practitioner. Better “safe than sorry” remains no more a virtue but a fundamental necessity.

Corresponding Author

Jayanth Kumar

Senior Lecturer ,Department of Oral Medicine

Saveetha Dental College,Saveetha Institute of Medical And Technical Sciences,

Saveetha University, Chennai, India

162, Poonamalle high Road,Chennai -600077, Tamil Nadu, India.

REFERENCES :

1.TamizhPaavi.Tha,Jayanth Kumar.V[Saveetha Dental College and Hospitals].To study awareness about radiation protection among dental students of Chennai-A questionnaire based study.Int J Pharm Bio Sci 2017 Jan ; 8[1]: B 542-551.
2.Gijbels F, Debaveye D, Vanderstappen M, Jacobs R. Digital radiographic equipment in the Belgian dental office. RadiatProt Dosimetry 2005; 117: 309–312.
3. Ilguy D,Ilguy M, Dincer S, Bayirli G. Survey ofdental radiological practice in Turkey. DentomaxillofacRadiol 2005; 34: 222–227.
4.Tugnait A, Clerehugh V, HirschmannPN.Radiographic equipment and techniques used in general dental practice. A survey of general dental practitioners in England and Wales. JDent2003; 31: 197–203.
5. Salti L, Whaites EJ. Survey of dental radiographic services in private dental clinics in Damascus, Syria. DentomaxillofacRadiol 2002; 31: 100–105.
6.Rushton VE,Horner K. A laboratory evaluation of four quality control devices for radiographicprocessing.JDent1994;22:213–222.
7.Platin E, Ludlow JB. Knowledge and adoption of radiographic quality assurance guidelines by general dentists in North Carolina. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 1995; 79: 122–126.
8.Rushton VE, Horner K, Worthington HM. Aspects of the use of panoramic radiography in general dental practice. Br Dent J 1999; 186: 342–344.
9.Tugnait A, Clerehugh V, Hirschmann PN. Radiographic equipment and techniques used in general dental practice: a survey of general dental practitioners in England and Wales. J Dent 2003; 31: 197–203.
10.Alcaraz M, Martinez Y, Perez L, Jodar S, Velasco E, Canteras M. The estatus of Spain′s dental practices following the European Union directive concerning radiological installations. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 2004; 98: 476–482
11.Brown FR, Shaver JW, Lamel DA. The Selection of Patients for X-ray Examination. U.S. Department of Health, Education and Welfare. HEW Publication [FDA]. Rockville, MD, Bureau of Radiological Health1980; 80–8104

12.Yakoumakis EN, Tierris CE, Stefanou EP, Phanourakis IG, Proukakis CC. Image quality assessment and radiation doses in intraoral radiography. Oral Surg Oral Med Oral Pathol Oral RadiolEndod 2001; 91: 362-368
13.Tan RT, Burke FJT. Response rates to questionnaires mailed to dentists. A review of 77 publications. Int Dent J 1997;47:349–354
14.Tugnait A, Clerehugh V, Hirschmann PN. Radiographic equipment and techniques used in general dental practice: a survey of general dental practitioners in England and Wales. J Dent 2003; 31: 197–203.
15.Svenson B, Soderfeldt B, Grondahl HG. Knowledge or oral radiology among Swedish dentists. DentomaxillofacRadiol 1997; 26: 219–224.
16.Swan ES, Lewis DW. Ontario dentists: Radiologic practices and opinions. J Can Dent Assoc 1993;59:62–67
17. Mutyabule TK, Whaites EJ. Survey of radiography and radiation protection in general dental practice in Uganda. DentomaxillofacRadiol 2002; 31: 164–169.
18.Svenson B, Soderfeldt B, Grondahl HG. Attitudes of Swedish dentists to the choice of dental X-ray film and collimator for oral radiology.Dentomaxillofac Radiol 1996; 25: 157–161.
19.Davies C, Grange S, Trevor MM. Radiation protection practices and related continuing professional education in dental radiography: a survey of practitioners in the North-east of England. Radiography 2005; 11: 255–261.
20. Smith NJ. Continuing education in radiation protection: assessment of a one-day course. Br Dent J 1991; 170: 186-188.
21.Shahab.S,Kavosi.A,Nazarinia.H,Mehralizadeh.SMohammadpour.M,Emami.M.
Compliance of Iranian dentists with safety standards of oral radiology. Dentomaxillofacial Radiology 2012;41:159-164.
22.Mutyabule.T,Whaites.E. Survey of the radiography and radiation protection in general dental practice in Uganda. Dentomaxillofacial Radiology 2002;31:164-169.
23.Aps,JKM.Flemish general dental practitioner knowledge of dental radiology. Dentomaxillofacial Radiology2010;39: 113-118.
24.Math S,MurugeshappaD,Annigeri R,Kalra,DCompliance of Indian dentists with oral radiology safety measures. Journal of Oral and Maxillofacial Radiology 2014;1[3]: 104-110.
25.Jacobs.R,Vanderstappen.M,Bogaerts.R,Gijbels.F.Attitudeof the Belgian dentist population towards radiation protection. Dentomaxillofacial Radiology2004 ;33: 334-339.

1. The intra-oral radiographic machine in my practice dates from
   - ☐ before 1980
   - ☐ 1980 – 1990
   - ☐ 1991 – 2000
   - ☐ after 2000

2. The intra-oral radiographic machine in my practice works at
   - ☐ 50 kV
   - ☐ 60 kV
   - ☐ 65 kV
   - ☐ 70 kV
   - ☐ 60 to 70 kV (easy to change)
   - ☐ no idea

3. The intra-oral radiographic machine in my practice works at
   - ☐ 4 mA
   - ☐ 8 mA
   - ☐ 10 mA or more
   - ☐ 4 or 8 mA (easy to change)
   - ☐ no idea

4. The intra-oral radiographic machine in my practice has a
   - ☐ manual (clock) timer
   - ☐ digital timer
   - ☐ no idea

5. The intra-oral radiographic machine in my practice is a
   - ☐ long cone
   - ☐ short cone
   - ☐ no idea

6. The intra-oral radiographic machine in my practice has a rectangular collimator
   - ☐ yes
   - ☐ no
   - ☐ no idea

7. For intra-oral radiography I usually use the paralle! technique
   - ☐ yes
   - ☐ no
   - ☐ no idea

8. For intra-oral radiography I work with
   - ☐ analogue film
   - ☐ a digital sensor
     - ☐ CCD
     - ☐ CMOS
   - ☐ no idea
   - ☐ PSPP (phosphor plates)

9. I have a panoramic machine at my disposal
   - ☐ no
   - ☐ yes
     - ☐ analogue film
     - ☐ CCD technology
     - ☐ photostimulable phosphor system
     - ☐ no idea

Genetic Biology in Age Determination

Anitha.D.R [1], Nithya Jaganathan [2], Sri Sakthi .D [3]
Graduate Student [1],Senior Lecturer [2],Department of Oral Pathology,
Reader [3],Department of Public Health Dentistry,
Saveetha Dental College, Saveetha University, Saveetha Institute of Medical And Technical Science

Abstract

To review the use of genetic biology in age determination.Chronological age fails to provide an accurate indicator of the ageing process. Biological age estimates the functional status of an individual in reference to his or her chronological peers on the basis of how well he or she functions in comparison with others of the same chronological age. This concept leads to the notion that biological age can be calculated for any given age group, thus being applicable to unselected pedigree members from the general population.The development of a biological age estimate using a combination of reliable quantitative biomarkers will also provide an instrument for assessing the effectiveness of any experimental manipulation or intervention in human aging. The search for genes which contribute to aging will benefit from the development of such a measure of biological aging of an organism as a whole.

Key Words:*Genetic biology, Ageing, Biomarkers, Skeletal age, Biological age.*

Introduction

Biological (synonymous with "functional" and "physiological") age has been proposed as an elective phenotype of intrigue. Since tissues age at various rates and in light of the fact that sicknesses change massively among people, people turn out to be progressively unique in relation to each other with age[1]. Ordered age neglects to give a precise pointer of the maturing procedure. Organic age gauges the utilitarian status of a person in reference to his or her sequential companions based on how well he or she works in correlation with others of the same ordered age. This idea prompts the thought that organic age can be computed for any given age gathering, accordingly being pertinent to unselected family individuals from the overall public. Diverse individual rates of the maturing procedure prompt contrasts amongst ordered and natural age, in this way singular estimations of organic age can shift broadly at any given sequential age, and at last are relied upon to relate to the bury singular varieties in life span and timing and additionally extent of spin-offs of the maturing process[2].

It has been suggested that natural age may fill in as a marker of a person's general wellbeing status, staying sound life expectancy, and dynamic future. Organic age may help in distinguishing people in danger for age-related disarranges, filling in as a measure of relative wellness, and foreseeing handicap in later life and mortality free of ordered age Other attributes, for example, slightness and allostatic stack, have been proposed as elements to consider while surveying heterogeneity of wellbeing status in elderly people, however these builds are not appropriate for an investigation of moderately aged people.

Individuals who work inadequately are believed to be "naturally more established" than their sequential companions; on the other hand, individuals who work well may be regarded "organically more youthful." The idea of organic age might be best spoken to by the development of a file got from a few organic parameters of a life form (supposed biomarkers of maturing), which are firmly identified with the support of life and associated to some degree with ordered age[3]. Investigation and approval of biomarkers remains a need in gerontology. Since tissues and organs age at various rates, there is a need to get biomarkers from numerous frameworks and to join them in the most proficient approach to reflect general maturing of a living being.

The majority of the proposed strategies for organic age assurance utilize far reaching multifactorial estimating frameworks or batteries of tests, incorporating an expansive number of biochemical, physical, mental, and utilitarian parameters that change with age[4]. The more far reaching the decision and evaluation of parameters of dysregulation in all the critical administrative frameworks, the better the capacity of their mix to foresee results. Some physiological frameworks might be more significant than others to the rate of maturing then again, some biomarkers can reflect dysregulation of numerous frameworks. The look for worldwide and framework particular markers will proceed until there is a superior comprehension of the connection between the imperative real frameworks.

The most clear biomarkers incorporate silver hair, presbyopia, and skin inelasticity. An expanded survey and dialog of biomarkers and their connection with age and prescient capacity of wellbeing results was given by Anstey and associates 10 years ago[5]. Most as of late, new biomarkers of maturing and new batteries of tests have been proposed, incorporating changes in telomere length, cross-connecting of collagen, glycosylated hemoglobin, beat wave speed, hearing misfortune, number of solid teeth and additionally sarcopenia. Seeman and partners additionally propose the utilization of fiery markers, coagulating variables, and safe capacity tests as biomarkers[6].

Numerous mainstream biomarkers decay generally directly with age with a slant of <1% per annum. Albeit certain biomarkers may veer off from linearity in a few bits of the age dispersion, a straight relationship is typically proposed for most biomarkers as a best fit. Psychosocial factors demonstrate minimal relationship with age, though measures of respiratory capacity demonstrate the most grounded affiliations. By and large, educational biomarkers should: a) foresee, superior to anything sequential age, decreases in one or a few organ frameworks as well as the probability of a malady occasion; b) be exceptionally connected with age-related loss of capacity, as opposed to just being associated with ordered age or survival; and c) be insignificantly obtrusive and promptly noticeable, dependably estimated, ideally on a quantitative scale[7].

We understand that the biomarkers by and large chose at introduce are simply previews of vital administrative frameworks; there is no data on framework elements if each biomarker is estimated just once. Despite the fact that the predominance of longitudinal over cross-sectional information is entrenched, its leeway in investigations of maturing might be

especially huge in light of the fact that longitudinal examinations allow the immediate examination of individual change and recognizable proof of elements related with that change[8]. A direct association with age that is expected for most biomarkers as a best fit may not generally be right because of deviation from linearity in a few bits of the age dispersion. An extra constraint of a cross-sectional estimation of natural age is an inability to represent bury singular contrasts in factors that are not identified with maturing. Each biomarker's change comprises of both honest to goodness maturing contrasts amongst people and contrasts not identified with maturing. A critical case of this is found in an investigation by Nakamura and partners, which demonstrated that hereditary variables may impact biomarkers at one time point as well as their rates of progress with time (age). There is an undeniable need to comprehend the status of the biomarker factors at various time focuses, and in addition to survey singular rates of natural age changes[9].

Choice of Biomarkers- Statistical Modelling

A fruitful future area of investigation in gerontology should focus on incorporating genetic profiles (variations in gene sequence and expression) along with other biomarkers of aging into prediction of health outcomes. Comprehensive studies, combining molecular genetic approaches with genetic epidemiology, gene expression profiling, proteomics, and functional genomic technology, will greatly accelerate the pace of discovery of genetic causes of aging. From a medical point of view, the endpoints of anti-aging therapy are clear: extending a healthy life span and maintaining and improving individual well-being, both physical and psychological, with the ultimate aim of exploring new ways of halting or delaying the onslaught of functional decline in elderly persons. Genotype information can be particularly useful in case–control studies ultimately, results of these studies will lead to developing novel intervention strategies, such as personalised geriatric medicine seeking to slow aging. It should become possible to assess a genetic risk profile for individuals that will enable them to rationally alter their lifestyle. Continuous efforts should be made to develop batteries of candidate genes and biomarkers of aging. As pointed out by Le Bourg: “Aging can be delayed if the right tool is discovered”.

The essential factors that have been utilized as an intermediary for maturing related attributes incorporate life span, absence of utilitarian decay, "successfull ageing," dynamic future (the

term of survival without disability), and wellbeing anticipation (survival with no one of a predefined set of disease, for example, cardiovascular infection, cancer, fracture, or dementia). In spite of the fact that there seem, by all accounts, to be built up methods to decide maturing related attributes, our comprehension of important hazard factor(s) for these results is still exceptionally simple. A basic situation is to utilize individual natural markers and covariates as indicator factors with one of the results as the ward variable[10]. In any case, the assessment of individual biomarkers (e.g., SBP, lipid measures, or hand hold) as essential indicators has been embroiled as a less proficient approach when contrasted with the utilization of rundown files. One of the key highlights of maturing considers has been the endeavor to choose an arrangement of factors that catches the "aggregate natural weight," which can be communicated as an element of watched singular hazard factors (biomarkers). In this way, Seeman and associates proposed a solitary rundown score (allostatic stack) as a measure of aggregate natural weight autonomous of result measures[6]. Further, Karlamangla and associates got a weighted synopsis score of hazard factors in view of standard connection investigation (hence characterizing allostatic stack restrictive on result measures)[11].

The kinds of strategies used to characterize composite factors incorporate those in view of basic midpoints or wholes of individual biomarkers, accepted examination, factor (central parts) investigation, and different straight relapse procedures. The propriety of these strategies relies upon the objectives of the investigation and the sort of information being examined. Reasonable lucidity and the basic rationale of picking these or different methodologies are key for the exactness of the examination.

One generally utilized strategy for natural age estimation is a different relapse of numerous biomarkers with age as the reliant variable. There are three normally utilized determination methods: forward choice, in reverse disposal, and stepwise relapse. Regardless of whether one uses forward determination, in reverse end, or stepwise relapse, these procedures are not guided by any substantive hypothesis—i.e., they utilize measurable calculations with little respect for the examination questions and additionally targets; and they don't really create the best model or an ideal choice of the subset of biomarkers. A typical issue with these strategies emerges when there are excess predictors[12]. Since in complex living beings, hypothetically, forms are for the most part related, changes in a single organ framework may influence numerous others. Notwithstanding, there are redundancies in compensatory frameworks in complex creatures that save work. There is accordingly a need to adjust the

want to incorporate however many qualities as could be allowed with the need to limit the quantity of attributes in the model.

Factor investigation has been proposed as a method for choosing factors instead of varieties of biomarkers of maturing. Primary part investigation is helpful to develop a straight blend among exceptionally perplexing, corresponded (colinear) biomarkers, and along these lines lessen the quantity of factors catching the maturing rate. The strategy gives rules to decide the quantity of parts to hold and the factor scores to characterize outline factors (primary components)[13]. In fact, a key part can be characterized as a direct mix of ideally weighted watched factors. When all is said in done, the bigger the span of the factor stacking for a variable, the more essential the variable is in translating that factor.

The capacity of both relapse conditions and factor answers for sum up crosswise over examinations isn't high. In any case, the two strategies are important as an estimation of an unpredictable system of natural maturing, and may distinguish various biomarkers that have reliably contributed a noteworthy piece of organic age changeability in numerous studies[11,12,13] A contrasting option to relapse incorporates the recursive parceling to recognize nonlinear examples of relationship among biomarkers. Development of weighting frameworks in light of accepted connection examinations has likewise been proposed, which shapes a straight blend of the factors from each set (called a sanctioned variable), with the end goal that the connection between the two standard factors is expanded. Karlamangla and associates utilized this way to deal with enable unequal weight to mirror a more practical situation in which the distinctive segments of the joined score contribute differentially to outcomes[11].

There is by all accounts a developing agreement with respect to the procedure for assessing organic age. Indeed, late examinations (counting our own) have acknowledged the accompanying factual definition: Biological age can be computed as a leftover between real age and anticipated age (from a measurably demonstrated blend of numerous biomarkers).

Biomarkers and Biological Age

There has been a hidden desire that a measure, for example, natural age ought to have the capacity to anticipate demise and incapacity freely of sequential age. Endeavors to assess the

prescient capacity of death by batteries of biomarkers started numerous years back. For instance, Uttley and Crawford built up a battery of biomarkers from pneumonic capacity (constrained expiratory volume), neuromotor execution, pulse, blood science, quality/adaptability, and anthropometric factors in their Mennonite tests. Organic age demonstrated along these lines did not definitively anticipate death[14]; be that as it may, a few individual biomarkers were related with higher relative hazard for mortality. Thus, Brant and partners found that circulatory strain, heart analysis, constrained expiratory volume, and visual sharpness were related with mortality in the Baltimore Longitudinal Study of Aging[15].

A contention could likewise be made that significant biomarkers ought not be required to anticipate mortality. Actually, the capacity of natural maturing records to foresee life traverse is a flawed rule beforehand used to approve these markers. There are numerous hazard factors that anticipate future in moderately aged individuals, yet are not firmly identified with a basic procedure of maturing, frequently in light of the fact that they are surrogates of weakness practices or particular infection chance. More critical is the capacity of a portion of the biomarkers and organic age measures to have the capacity to separate between antagonistic maturing related occasions, for example, stability, organization, or penchant to fall. An outline record of a few biomarkers ("allostatic stack") has been shown to be a noteworthy indicator of real wellbeing results, to be specific, mortality and decreases in psychological and physical working, both over a 2.5-year and 7.5-year development. Quite, none of the individual biomarkers, segments of this list, had solid prescient capacity for the above results, recommending that the outline list for sure measures a property past minor brokenness in one organ system[16].

Common and New Biomarkers

SBP, hold quality, constrained expiratory volume, cholesterol and glucose blood levels, and psychological and additionally neuropsychological information are the most regularly utilized biomarkers in investigations of natural age. SBP has been one of the beforehand examined biomarkers for a few reasons. There are changes in vascular solidness with maturing that influence SBP; these progressions might be because of the lessening in versatile consistence of maturing vascular tissues. In a longitudinal investigation of Japanese men, SBP essentially expanded over a 7-year interim ($r = 0.58$), and in a few cross-sectional examinations, SBP

likewise has been shown to connect well with age in both genders (r = 0.45 in guys and 0.50 in females)[17]. Heartbeat weight, a distinction amongst systolic and diastolic circulatory strain, logically increments with age because of dynamic hardening of corridors, and consequently may likewise fill in as a biomarker of blood vessel maturing.

Sarcopenia is showed as muscle quality and bulk diminish with age, basically because of the adjustments in muscle morphology. This for the most part is because of diminished extent and cross sectional territory of sort II filaments, in any case, single fiber investigation recommends that the contractile proteins may turn out to be less powerful with age. Hand grasp quality is known to be related with solid working in other muscle gatherings and with exercises of day by day living. In a few cross-sectional investigations, grasp quality corresponded with age (r = 0.61 in the two guys and females). Hand grasp quality predicts cause-particular and aggregate mortality and inability. Symptomatic hand osteoarthritis additionally has been appeared to be conversely connected with hold strength[19].

The histological changes in lung parenchyma, a decline in ventilatory muscle quality, and an expansion in lung consistence with maturing add to bring down crucial limit and expiratory stream rates in elderly people. Constrained expiratory volume in 1 second (FEV1) and constrained imperative limit (FVC) are the best measures of aspiratory work. Both essentially diminished with age (r = −0.60 to −0.75 in cross-sectional and −0.48 to −0.60 in longitudinal examination, for FVC and FEV1, respectively)[18,19].

Cross-sectional and longitudinal changes in cholesterol levels with maturing have been accounted for in both genders. Eminently, serum cholesterol levels expanded by 18% more than 15 years in ladies and by just 6% in men. LDL cholesterol levels likewise increment with age in most human populaces, and relate to higher cardiovascular pathology and mortality. HDL cholesterol levels have been appeared to be significantly higher in the posterity of Ashkenazi centenarians than in controls, proposing that HDL might be engaged with remarkable life span. A few examinations have discovered a diminished grouping of aggregate cholesterol in an elderly populace, maybe clarified by their deficient nourishment or lower physical movement. Age subordinate decrease of aggregate cholesterol levels has been recommended to be a marker of poor health[15,16,17,18].

Hindered glucose resilience and non-insulin-subordinate diabetes turn out to be dynamically more typical with propelling age, because of diminished hepatic affectability to insulin or insufficient emission of insulin. Coming about hoisted glucose focuses (hyperglycemia), specifically, potentiate damage to mitochondrial DNA. Blood glucose levels have been appeared to relate with age in Japanese men ($r = 0.17$ in cross-sectional and $r = 0.13$ in longitudinal examination).

Decrease in cerebral blood stream, white issue injuries, and a critical decrease in the quantities of mind cells add to intellectual decrease in elderly people. With expanding age, there is an expanded hazard for memory hindrance and additionally signs of age-related decrease of other psychological capacities in generally typical more seasoned grown-ups. Along these lines, in the Framingham Study, the most established members had the least execution levels estimated by a neuropsychological test battery (be that as it may, they likewise have had the least years of formal instruction). When all is said in done, the mean execution of less instructed elderly people was fundamentally poorer than that of their better taught peers[20].

Skeletal Biomarkers

The process of bone involution seems to be a general phenomenon and a normal manifestation of tissue atrophy with age. After the skeleton has reached maturity, bone remodelling is responsible for the complete replacement of old bone tissue with new tissue. With advanced age, the placement and quality of new bone are altered[21]. This alteration is caused by the change in areas of remodelling due to disease, by variations in muscle strength, and by trauma. Also, at different phases of life during which there are various kinds of work and leisure activity, bone activity may be altered. The amount of bone reformed and replaced during the remodelling cycle progressively decreases with advanced age due to decreased proliferative capacity of osteoblasts as well as to cumulative damage of uncorrected molecular errors. The longer an individual lives, the more remodelling cycles he or she undergoes, and the more such errors accumulate[22]. Finally, the contribution of catabolic illness, such as surgery, injury, palsy, and immobilisation, may contribute to rapid bone degeneration. The net result of the age-related bone changes in older persons is chronic degenerative disease, which is characterized by the loss of bone mass and bone strength, development of fragility, and the alteration of bone morphology. These processes involve

both cortical and trabecular bone compartments, juxtaarticular regions, and bones of axial and appendicular skeleton, and are common in humans of different ethnic groups, both modern and prehistoric.

There is a strong correlation of age-related skeletal features with the state of many vital functions ,age-related conditions of different bodily systems, and ultimately survival rates. For example, lower bone mineral density and higher osteoarthritis are associated with a higher risk of mortality independent of age and comorbidities. In particular, low bone ultrasound indices have been associated with a higher risk of all-cause and cancer mortality.

Adaptive mechanisms, which compensate for skeleton's deficient remodelling, are also evident in degenerative bone disease. Thus, endosteal expansion causes subperiosteal outgrowth; excessive bone resorption (such as that occurring when the skeleton is unloaded) brings about heterotopic calcification in neighbouring soft tissues[24]. Similarly, new bone formation is demonstrated by endochondral ossification of enthesis fibrocartilage and development of osteophytes. Longitudinal studies have shown that incident bone degenerative traits never regress. These features of the skeleton make it an attractive organ system for assessing biological age. Bone changes are relatively stable, unlike most non-skeletal biomarkers that may vary seasonally or diurnally or may be dependent on external conditions.

Age-related bone and joint changes manifest as visible and measurable traits, which has facilitated paleoanthropological and forensic applications of skeletal ageing methods and has ultimately paved the way for the development and usage of a radiographic (osteographic) scoring system as an integrative measure of adult bone ageing[25].

Conclusion

Human aging is a complex and irreversible process that is genetically determined and influenced by the environment. The study of aging genetics in humans is an enormous challenge. The complexity of the aging phenotype and the challenges inherent in the study of human aging oblige researchers to resort to models and extrapolations. Because different individual rates of the aging process lead to differences between chronological and biological age, the deviation between the actual age and age predicted using validated measurements may be one approach to estimating the biological age of an individual. An interdisciplinary

approach is needed to further develop reliable methods of biological age evaluation and to validate this phenotype as a predictor of important health outcomes of aging. The development of a biological age estimate using a combination of reliable quantitative biomarkers will also provide an instrument for assessing the effectiveness of any experimental manipulation or intervention in human aging. The search for genes which contribute to aging will benefit from the development of such a measure of biological aging of an organism as a whole. Empirically, not all valid biomarkers proposed in one study may be replicated in other samples, due to missing measurement or differences in technique. Until a consensus on a minimal set of biomarkers is achieved, it would be premature to recommend the use of biological age for changes in policy such as life insurance, elderly driving, and retirement benefits. Differences between ethnic and geographical groups, both in terms of their biological age (at a given age) and its rate of change, as well as genetic effects of aging-related genes in different races, are almost totally unexplored. Similarly, the extent to which biological age interacts with sex is not clearly established.

Corresponding Author

D.Sri Sakthi

Reader,Department of Public Health Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai – 600077, Chennai, India

Mobile Number- +91 – 9962787531

Email ID: drsrisakthiphd@gmail.com

Reference

1. Shephard R. Constitution or Environment? In: Shephard R, ed. Gender, Physical Activity, and Aging Boca Raton (FL): CRC Press; 2002:151–174.

2. Tominaga K, Olgun A, Smith JR, Pereira-Smith OM. Genetics of cellular senescence. Mech Ageing Dev 2002;123:927–936.

3. Finkel D, Whitfield K, McGue M. Genetic and environmental influences on functional age: a twin study. J Gerontol B Psychol Sci Soc Sci 1995;50:P104–P113.

4. McGue M, Vaupel JW, Holm N, Harvald B. Longevity is moderately heritable in a sample of Danish twins born 1870–1880. J Gerontol 1993;48:B237–B244.

5. Anstey, Herskind AM, McGue M, Holm NV, Sorensen TI, Harvald B, Vaupel JW. The heritability of human longevity: a population-based study of 2872 Danish twin pairs born 1870–1900. Hum Genet 1996;97:319–323.

6. Seeman, Mitchell BD, Hsueh WC, King TM, Pollin TI, Sorkin J, Agarwala R. Heritability of life span in the Old Order Amish. Am J Med Genet 2001;102:346–352.

7. Yashin AI, Iachine IA, Harris JR. Half of the variation in susceptibility to mortality is genetic: findings from Swedish twin survival data. Behav Genet 1999;29:11–19.

8. Yashin AI, Iachine IA. Genetic analysis of durations: correlated frailty model applied to survival of Danish twins. Genet Epidemiol 1995;12:529–538.

9. Nakamura, Scott W, Gaskell P, Jackson C, Haines JL, Pericak-Vance MA. Combinatorial mismatch scan for successful aging loci in the Amish. Am J Hum Genet 2003;73:124.

10. Reed T, Dick DM, Uniacke SK, Foroud T, Nichols WC. Genome-wide scan for a healthy aging phenotype provides support for a locus near D4S1564 promoting healthy aging. J Gerontol A Biol Sci Med Sci 2004;59:B227–B232.

11. Karlamangla, Brand FN, Kiely DK, Kannel WB, Myers RH. Family patterns of coronary heart disease mortality: the Framingham Longevity Study. J Clin Epidemiol 1992;45:169–174.

12. Terry DF, Wilcox M, McCormick MA, Lawler E, Perls TT. Cardiovascular advantages among the offspring of centenarians. J Gerontol A Biol Sci Med Sci 2003;58:M425–M431.

13. Uttley, Barzilai N, Gabriely I, Gabriely M, Iankowitz N, Sorkin JD. Offspring of centenarians have a favorable lipid profile. J Am Geriatr Soc 2001;49:76–79.

14. Brant, Evert J, Lawler E, Bogan H, Perls T. Morbidity profiles of centenarians: survivors, delayers, and escapers. J Gerontol A Biol Sci Med Sci 2003;58:232–237.

15. Barzilai N, Atzmon G, Schechter C. Unique lipoprotein phenotype and genotype associated with exceptional longevity. JAMA 2003;290:2030–2040.

16. Geesaman BJ, Benson E, Brewster SJ. Haplotype-based identification of a microsomal transfer protein marker associated with the human lifespan. Proc Natl Acad Sci U S A 2003;100:1411514120.

17. Hadley E, Rossi W, Albert S. Genetic epidemiologic studies on age-specified traits. Am J Epidemiol 2000;152:1003–1008.

18. Duggirala R, Uttley M, Willams K, Arya R, Blangero J, Crawford M. Genetic determination of biological age in the Mennonites of the Midwestern United States. Genet Epidemiol 2002;23:97109.

19. Puca A, Daly M, Brewster S. A genome-wide scan for linkage to human exceptional longevity identifies a locus on chromosome 4. Proc Natl Acad Sci U S A 2001;98:10505–10508.

20. Framingham, Jackson AU, Galecki AT, Burke DT, Miller RA. Mouse loci associated with life span exhibit sexspecific and epistatic effects. J Gerontol Biol Sci 2002;57A:B9–B15.

21. Finkel D, Pedersen NL, Berg S, Johansson B. Quantitative genetic analysis of biobehavioral markers of aging in Swedish studies of adult twins. J Aging Health 2000;12:47–68.

22. Terry DF, Wilcox MA, McCormick MA, Perls TT. Cardiovascular disease delay in centenarian offspring. J Gerontol A Biol Sci Med Sci 2004;59:385–389.

23. Helfand SL, Inouye SK. Rejuvenating views of the ageing process. Nat Rev Genet 2002;3:149–153.

24. Vane swetha, Nitya jaganathan. Age estimation using pulp volume of mandibular first Pre molar obtained from CBCT in Indian population. Int. J. Pharm. Sci. Rev. Res.2017;119-122.

25. Sisira padavela, Gheena. Estimation of age using cementum annulations.J. Pharm. Sci. & Res.2015:7(7);461-463.

Green Tea in Oral Health

S.Thaminee[1],Nithya Jaganathan[2],Ashok V[3],
Graduate Student[1],Professor[2], Department of Oral Pathology, Reader[3], Department of Prosthodontics[3],
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Green tea is biologically important in oral protection.It is an antioxidant, antimutagen, antimicrobial, anticollagnese contains fluoride that prevents the dental carries. It is one of the most consumed beverages, which also provides a very good health. Green tea induces the biological activity due to the presence of Polyphenols namely catclines. Cateclines are considered to be the most predominant among polyphenols egigallo catechin 3 gallatecatechinswhich are proved to be helpful in the treatment of chronic diseases. It protects against bacterial induced dental carries.

Key Words: *Polyphenol, Anti Oxidant, Anti Viral, Dental Carries, Bleeding Gums, Oral Cancer, Periodontal Pathogens*

Introduction

Green tea is important source of polyphenol antioxidants.Green tea protects against bacteria that inducing dental caries. Generally, green tea defends healthy cells from malignant transformation and locally has the ability to induce apoptosis in oral cancer cells (10). There is a need for more clinical and biological studies to support guidelines for green tea intake as part of prevention and treatment of specific oral pathology(1). The effect has been attributed to the poly phenol content which is known to induce apoptosis of tumor cells and has little or nil effect in normal cells (9).Green tea is particularly rich in health-promoting flavonoids (which account for 30% of the dry weight of a leaf), including catechins and their derivatives. The most abundant catechin in green tea is epigallocatechin-3-gallate, which is thought to play a pivotal role in the green tea's anticancer and antioxidant effects. Catechins should be considered right alongside of the better-known antioxidants like vitamins E and C as potent free radical scavengers and health-supportive for this reason. It has been suggested that green tea also promotes periodontal health by reducing inflammation, preventing bone resorption and limiting the growth of certain bacteria associated with periodontal diseases.Green tea is a leading beverage in the Far East for thousands of years; it is regarded for a long time as a health product. Green tea is important source of polyphenol antioxidants. Polyphenols including epigallocatechin 3 gallate (EGCG) constitute the most interesting components in green tea leaves. Green tea has the potential to protect against various malignant, cardiovascular and metabolic diseases. There is a growing body of evidence pointing a beneficial role of green tea and its polyphenols in oral health. Green tea protects against bacterial induced dental caries. Tea polyphenols possess antiviral properties, believed to help in protection from influenza virus. Additionally, green tea polyphenols can abolish halitosis through modification of odorant sulphur components. Oral cavity oxidative stress and inflammation, consequent to cigarette smoking and cigarettes' deleterious compounds nicotine and acrolein, may be reduced in the presence of green tea polyphenols. Generally, green tea defends healthy cells from malignant transformation and locally has the ability to induce apoptosis in oral cancer cells. All together, there is an increasing interest in the health benefits of green tea in the field of oral health. Nonetheless, there is still a need for more clinical and biological studies to support guidelines for green tea intake as part of prevention and treatment of specific oral pathologies.

Cavity Prevention

Green tea controls bacteria and lowers the acidity of saliva and dental plaque, it may be a useful tool in preventing cavities. A recent study tested people before and after they gave their mouths a five-minute rinse with green tea.The test subjects had less bacteria and acid in their mouths, as well as reduced gum bleeding(2). Few researches had found that drinking green tea shows promise when it comes to preventing tooth decay.The effects of green tea extract on caries inhibition of hamsters and on acid resistance of human tooth enamel have been suggested by both *in vivo* and *in vitro* studies. The dialyzed tea solution in which the fluoride was removed almost completely also showed remarkable effects, similar to the original tea extract. The results obtained from this study suggested that fluoride in green tea may play a role in increasing the cariostatic action along with other components in tea. However, the action of fluoride does not seem to be so important because its concentration is very low. The effect of green tea on caries inhibition as well as on the increment of acid resistance appears to be more correlative with the nondialysable substances in tea.

Gum Health

Green tea's anti-inflammatory powers seem to help control periodontal (gum) diseases. German study found similar positive results in people who were asked to chew candies containing green-tea extracts.

Less Tooth Loss

It has made sense that a substance that helps prevent cavities and gum disease will help you keep yourteeth. Japanese research published in 2010 reported that men and women who drink green tea a day were more likely to hold on to their natural teeth(3).

Cancer Control

The antioxidants properties of green tea appear to protect against cellular damage and cancerous tumour growth. In University of Texas, green-tea extract was given to patients with precancerous lesions in their mouths, and it slowed the progression to oral cancer.Animal studies have also

found that tea compounds can inhibit cancer growth(4).The intake of green tea and its components such as catechin,had a preventive effect against cancer development.

Bad Breath

The University of British Columbia's Faculty of Dentistry measured the level of smelly compounds in people's mouths after they were given green-tea powder or another substance that supposedly helps with bad breath. Green tea outperformed mints, chewing gum and even parsley-seed oil in this study.

Role of Green Tea in Periodontitis

Gum disease, periodontal disease and periodontitis are different terms for the same condition.It occurs when the gums begin to separate from the teeth, leaving open spaces or pockets. A recently published study in the Journal of Periodontology, the official publication of the American Academy of Periodontology, has revealed benefit of drinking Green Tea(5). Author-dr. Yoshihiro Shimazaki of Kyushu University in Fukuok, Japan and his colleagues wanted to explore which is the impact of Green Tea consumption on periodontal health, with special emphasis on the link between periodontal and general health. Finally, they came to some interesting conclusions.Each cup of Green Tea daily resulted in a great decrease of the mentioned problems,as it dramatically reduced the incidence and stage of periodontal disease in males who followed it constantly(6).

Green Tea and its Polyphenols

Green tea catechins and polyphenols have a positive influence on the inflammatory reaction of periodontal structures. Streptococcus mutans plays an important role in causing dental caries. Leaves are rich in fluoride which is known to prevent dental caries. Besides fluoride, several green tea polyphenolshave preventative effects on dental caries(7).

Green Tea's Polyphenol Antioxidant

The green tea also possesses antiviral properties, which could help protect against the influenza virus that often enters through the mouth or nasal cavities (11). Additionally, green tea

polyphenols can be used as a natural remedy to treat bad breath. The sulfur compounds in green tea are thought to be responsible for its breath freshening effect (12). Green tea could help induce death in oral cancer cells. Polyphenols, antioxidants found in green tea, is believed to neutralize the risk of cancer caused by free radicals and can actually kill mouth cancer cells (15). Consuming this tea can prevent tooth decay. A new study shows drinking green tea or gargle use may inhibit the formation of dental plaque and prevent dental caries. Green tea has an antimicrobial effect on S. mutans, the bacteria responsible for tooth decay (13).

Oral Herbal Green Tea Brushing Salt

Olgani Green Tea Brushing Salts are a mild antibacterial and antifungal brushing salt formulation that can be used as an alternative to our powerful Original Salts while still providing an effective gum disease treatment (14). The main antioxidant in green tea, polyphenol, is a bioactive substance vital for maintaining, restoring and improving health in the human body (8).

Benefits of Green Tea in Oral Health

1. Protection against bacteria-induced dental caries

2. Prevent halitosis (bad breath) due to “modification of odorant sulfur components”

3. Prevent malignant formation of cells found in the mouth which could lead to oral cancer

Conclusion

Green tea extract may have numerous effects on periodontal pathogens and periodontal tissues. Greater the concentration of catechins better the health benefits. The consumption of green tea in comparison to other beverages may be widely recommended.

Corresponding Address

Ashok V,

Reader,Department of Prosthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Science,

Saveetha University, Chennai, India
162, Poonamallee High road, Chennai 600077,TamilNadu,India.
Email Id:ashokprostho@gmail.com

Mobile Number:+91 9962422299

References

1. Babu Venkateswara, K. Sirisha, andVijay K. Chava. Green tea extract forperiodontal health. J Indian SocPeriodontol, 2011; 15(1): 18–22.
2. Socransky SS, Haffajee AD, Cugini MA, Smith C, Kent RL Jr. Microbial complexes in subgingival plaque. J ClinPeriodontol,1998; 25:134-144.
3. Haffajee AD, Socransky SS, Taubman MA, Sioson J,Smith DJ. Patterns of antibody response in subjectswith periodontitis. Oral Microbiol Immunol, 1995; 10:129-137.
4. Hamidreza Arab, Ahmad Maroofian,ShayanGolestani, Hoomanshafaee, KeyvanSohrabi and Ali Forouzanfar.Review of the therapeutic effects ofCamellia sinensis (green tea) on oral andperiodontal health. Journal of MedicinalPlants Research,2011; 5(23): 5465-5469.
5. Sakanaka S, Aizawa M, Kim M, Yamamoto T. Inhibitory effects of green tea polyphenols on growth and cellular adherence of an oral bacterium,Porphyromonasgingivalis. BiosciBiotechnol Biochem,1996; 60: 745-749.
6. Masatomo Hirasawa, Kazuko Takada,Masaharu Makimura, Shigeo Otake.Improvement of periodontal status bygreen tea catechin using a local delivery system: A clinical pilot study. JPeriodont Res 2002;37:433-438.
7. Diplock AT, Aggett PJ, Ashwell M, Bornet F, Fern EB, Roberfroid MB. Scientific concepts of functional foods in Europe consensus document. Br J Nutr. 1999; 81:1–27.
8. Roberfroid MB. Global view on functional foods: European perspectives. Br J Nutr. 2002; 88:133–8.
9. M. Priya, JaiganeshRamamurthy. Effects of Green Tea in the Treatment of Periodontal Disease Department of Periodontics, Saveetha Dental College.JPSR 2016; 33(4):4-5.
10. Graham, H. N. Green tea composition, consumption, and polyphenol chemistry. Prev. Med.1992;21:334-350.

11. S.Jayakeerthana.Benefits of Green Tea: A Review;Saveetha Dental College, Chennai-600077; J. Pharm. Sci,2016; 8(10): 1184-1187.
12. V.Gopinath.Oral practices and hygiene among dental professionals in Chennai;Saveetha Dental College;Chennai-600077; RJPT 2010;21(2):55-57.
13. Dr.Vipin Kumar.Green Tea – The Periodontal Health Booster;Department of periodontic;Saveetha Dental College.IJPSR 2014; 1(4):73-77
14. Sarah Sathiyawathie, Jaiganesh Ramamurthy.Nature's Receipe of oral health;Saveetha Dental College and Hospital;chennai;RJPBCS 2015;.6(5):244.
15. S. AafrinThasleema.Green Tea as an Antioxidant- A Short Review;Saveetha Dental College. J. Pharm. Sci. & Res 2013;5 (9): 171 - 173.
16. Haffajee AD, Socransky SS, Taubman MA, SiosonJ,Smith DJ. Patterns of antibody response in subjectswith periodontitis. Oral Microbiol Immunol, 1995; 10:129-137.
17. Hamidreza Arab, Ahmad Maroofian,ShayanGolestani, Hoomanshafaee, KeyvanSohrabi and Ali Forouzanfar.Review of the therapeutic effects ofCamelliasinensis (green tea) on oral andperiodontal health. Journal of MedicinalPlants Research,2011; 5(23): 5465-5469.
18. Sakanaka S, Aizawa M, Kim M, Yamamoto T. Inhibitory effects of green tea polyphenols on growth and cellular adherence of an oral bacterium,Porphyromonasgingivalis. BiosciBiotechnol Biochem,1996; 60: 745-749.
19. MasatomoHirasawa, Kazuko Takada,MasaharuMakimura, Shigeo Otake.Improvement of periodontal status bygreen tea catechin using a local delivery system: A clinical pilot study. JPeriodont Res 2002;37:433-438.
20. Diplock AT, Aggett PJ, Ashwell M, Bornet F, Fern EB, Roberfroid MB. Scientific concepts of functional foods in Europe consensus document. Br J Nutr. 1999; 81:1–27.

Halitosis – A Review

Boopana .K [1], Geo Mani[2]

Graduate Student [1], Senior Lecturer [2],Department of Pedodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Halitosis is very common among any population, some believe that this distressing condition to be within a normal breath odour. Halitosis is common among the adult population group. Halitosis interferes with the social relationship and brought more awareness among the public. It becomes more necessary for the dentists for the proper understanding of oral as well as systemic factors that cause halitosis. Patient with halitosis may seek treatment from dental clinicians for their perceived oral malodour. Halitosis is not a disease but it is rather a symptom of underlying oral, systemic or psychological condition . psychosomatic halitosis is more difficult to diagnoise and manage , and patients with this condition are often mismanaged in that they get only treatments for genuine halitosis , even though they do not have a oral malodour. Management of halitosis include periodontal or restorative treatment or both, as well as simple treatment such as the introduction in oral hygiene, tongue cleaning and mouth rinsing. This article aims to address some misconceptions, and reviews on the cause, types, diagnosis, treatment, with the halitosis originating in the oral cavity

Key Words*: malodor, volatile sulphur compounds, oral cavity, oral hygiene , bacteria.*

Introduction:

Halitosis is the term which is used to describe any disagreeable odour in the air exhaled by the individual ,regardless of whether the odour originates in the oral cavity or elsewhere within the body[1]. Bad breath or halitosis is experienced by most of the people some time. It can simply be a morning breath but can also be persistent chronic condition which affects a person's everyday life and confidence. Halitosis emit a variety of volatile and non volatile molecules that are influenced by genetics , diet ,stress and disease. Halitosis causes embarrassment , may affect interpersonal social communication and it also has become an important market for the pharmacological and cosmetic industries. The prevalence of halitosis is reported to be as high as 50 percent[2]. Halitosis is not a disease but rather a symptom of underlying oral, systemic or psychological conditions [3]. Although the prevalence of halitosis and the incidence between male and female have been reported to be the same , women seem to seek more treatment more often than the men, in which some of the individuals who have halitosis but are totally unaware of it [4].

Causes of Halitosis :

The primary cause for halitosis is due to the release of odoriferous volatile sulphur compounds in the exhaled air. There are several things that may contribute to the bad breath , which include certain food , or food that are trapped between your teeth, gum disease, and tooth decay, dry mouth, tobacco products or a medical disorder. The basic that is present behind the origin of bad breath is the bacteria that live in the mouth if the individual which produce the volatile sulphur products . if there is enough amount of sulphur compounds there will b clinical bad breath5. Other reasons for bad breath are poor cleaning of teeth and tongue allowing bacteria to accumulate. It results from the breakdown or the decomposition of the bacteria left on the teeth and the tongue. This produces the VSC compounds which are exhaled while we are talking and breathing. The most common volatile sulphur compounds are the hydrogen sulphide which smells like a rotten egg, methyl mercaptan which smells like the animal faeces and the dimethyl sulphide smells like a cabbage6. Dry mouth called xerostomia can lead to the increase in the bacteria, as saliva usually has a protective function in the mouth. Morning breath and saliva flow

reduces , post nasal drips or sinus, smoking especially stale tobacco, intake of garlic and onion, dehydration[7].

Types of Halitosis :

One of the recent and frequently used classification is the halitosis id classified into three types the exogenous, endogenous and psychogenic halitosis. Halitosis is also classified as genuine halitosis, physiological halitosis, pathological halitosis, pseudo halitosis, halitophobia8

Genuine halitosis: presence of malodour that is beyond socially acceptable level .

Physiological halitosis: neither a specific disease nor a pathological condition that could cause halitosis is found, e.g . malodour from putrefactive processes within the oral cavity, temprorary halitosis due to the dietary factors9

Pathological halitosis

Oral: halitosis caused by disease, pathological condition or malfunction of oral tissues. Halitosis derived from the tongue coating, modified by pathological condition, e.g. periodontal disease, xerostomia.

Extra oral: malodour originates from nasal, paranasal sinuses, laryngeal regions, respiratory or upper digestive tract. Malodour originates from anywhere in the body whereby the odour is blood borne and emitted through the lungs. E.g diabetes mellitus, hepatic cirrhosis, uraemia and internal bleeding[10]

Pseudo halitosis: obvious maldour is nit perceived by others, although the patient complains of its existence. Condition is improved by counseling and simple oral hygiene measures[10].

Halitophobia: no evidence of any halitosis being present. After treatment for genuine halitosis or pseudo-halitosis, the patient fears that he/she has halitosis.

Diagnosis of Halitosis :

A complete dental examination is needed along with the medical and halitosis history, is essential to determine the cause of the halitosis. In subjects with the genuine halitosis, halitosis of oral origin has to be distinguished from halitosis of extraoral origin by comparing mouth breath, nasal breath and the lung air [11]. The first step in assessment is to determine if halitosis is present. This is more important as many of them are poor judges of their own breath odor. The assessment is usually based upon organoleptic assessment of exhaled air, namely the clinician sniffs the air exhaled from the mouth and nose and subjectively defines the presence or absence of malodor. Gas chromatography is considered by many to be the method of choice for the differentiation and quantifying the volatile sulfur compound and can also other classes of compounds like indole[12]. Examination of oral flora , for example using the BANA test or dark field microscopy, can be helpful, atleast for the patient education. Assay for the major glysocidic enzyme in the saliva, which deglycosylates the oral mucins, leading to their subsequent proteolysis and putrefaction , may also be very useful[13].

Treatment of Halitosis :

Treatment for physiological halitosis: explanation of halitosis and instructions for oral hygiene(support and reinforcement of a patient’s own self-care for further improvement of their oral hygiene[14]

Treatment pathological halitosis: oral and extra oral : explanation of halitosis and instructions for oral hygiene and the support and reinforcement of a patient’s own self care for further improvement of their oral hygiene and oral prophylaxis , professional cleaning and treatment for oral diseases, especially periodontal diseases[15] . Treatment for pseudo halitosis: explanation of halitosis and the instructions for the oral hygiene for the support and the reinforcement of a patient’s own self-care for further improvement of their oral hygiene. Explanation of examination data, further professional instruction, education and reassurance [16].Treatment for halitophobia: explanation of halitosis and the instructions for the oral hygiene for the support and

the reinforcement of a patient's own self – care for further improvement of their oral hygiene. Referral to a clinical psychologist, psychiatrist or other psychological specialist[17]

Control and Prevention of Halitosis :

Control and prevention include active periodontal therapy either at our office or reffered to a specialist. Treat any existing decayed tooth and the drymouth issues. Eliminate any tobacco use habits. Maintain optimal oral care at home. Use a carbamide peroxide gel in custom made halitosis trays[18,19] .All patient need to do the following : brush with oxyfresh tooth paste regularly as well as your fluoride containing toothpaste and brush twice a day. Floss the teeth atleast one daily. Brush the teeth twice a day for atleast 1-2 minutes with a small drop of the oxyfresh tooth paste. Use of oxyfresh mouthrinse at least twice a day. Avoiding the alcohol containing mouth wash and use of ACT fluoride mouth wash that are alcohol free and helps in the prevention of tooth decay and halitosis20. For the person who have halitosis all day custom made halitosis trays with carbamide peroxide gel half an hour in the evening. For the persons who bad breath in the morning they can wear custom made halitosis trays with carmbamide peroxide at night when you sleep. For the patients who have evening breath they can wear custom made halitosis with carbamide peroxide about 3 hours before the onset of when you feel the bad breath occurs. Wear it for half an hour[21]. The other general measures that are to adopted is having a scale and polish to remove the hard and soft deposits and also tongue brushing. Regular checkups ,smoking cessation , reduce alcohol consumption , reduce the intake of food such as garlic and onion and a balanced healthy diet[22].

Conclusion:

The patients seek a lot of therapy from the dentist than from the physicians. It is therefore very crucial for the dentists to have an understanding of all types of halitosis , especially those that arise from the oral cavity. Several misconception about halitosis need to put a step side . the

available evidences indicates that the volatile sulpur compounds are nit just odoiferous , but that some of them are deleterious to the periodontal health. Therefore treatment of halitosis can no longer be considered as just a cosmetic therapy.

Corresponding Author

Geo Mani

Senior Lecturer,Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College and Hospitals, Saveetha Institute of Medical and Technical Sciences, Saveetha University,

162 Poonamallee High Road,Chennai -600 077,Tamil Nadu, India.

Email Id: geosdch@gmail.com

Telephone Number: 7358433246

Reference

1. Teixeira AC. Estudo da halitose em pacientes com tonsilitecronicacaseosa, tratadosporcriptolise com laser de'CO IND. 2'.

2. Lee SS, Zhang W, Li Y. Halitosis update: a review of causes, diagnoses, and treatments. Journal of the California Dental Association. 2007 Apr;35(4):258-60.

3. Tonzetich J. Production and origin of oral malodor: a review of mechanisms and methods of analysis. Journal of periodontology. 1977 Jan;48(1):13-20.

4. Iwakura M, Yasuno Y, Shimura M, Sakamoto S. Clinical characteristics of halitosis: differences in two patient groups with primary and secondary complaints of halitosis. Journal of dental research. 1994 Sep;73(9):1568-74.

5. Miyazaki H, Sakao S, Katoh Y, Takehara T. Correlation between volatile sulphur compounds and certain oral health measurements in the general population. Journal of periodontology. 1995 Aug;66(8):679-84.

6. Rosenberg M. First international workshop on oral malodor. Journal of dental research. 1994 Mar;73(3):586-9.

7. Lavezy LA. The relationships between malocclusion, gingival inflammation, plaque and calculus. Journal of periodontology. 1981 Jan 1;52(1):35-40.

8.Yaegaki K, Coil JM. Examination, classification, and treatment of halitosis; clinical perspectives. JOURNAL-CANADIAN DENTAL ASSOCIATION. 2000 May;66(5):257-61.

9. Murata T, Yamaga T, Iida T, Miyazaki H, Yaegaki K. Classification and examination of halitosis. International dental journal. 2002 Jun 1;52(S5P1):181-6.

10. Yaegaki K, Coil JM. Genuine halitosis, pseudo-halitosis, and halitophobia: classification, diagnosis, and treatment. Compendium of continuing education in dentistry (Jamesburg, NJ: 1995). 2000 Oct;21(10A):880-6.

11. McDowell JD, Kassebaum DK. Diagnosing and treating halitosis. The Journal of the American Dental Association. 1993 Jul 1;124(7):55-64.

12. Donaldson AC, Riggio MP, Rolph HJ, Bagg J, Hodge PJ. Clinical examination of subjects with halitosis. Oral diseases. 2007 Jan 1;13(1):63-70.

13.Sterer N, Bar-Ness Greenstein R, Rosenberg M. β-galactosidase activity in saliva is associated with oral malodor. Journal of dental research. 2002 Mar;81(3):182-5.

14. Ozeki H, Amemiya H, Tanaka N, Kanamori K, Tojo H, Ushiyama T, Ishii Y, Narita Y, Uchida Y. Therapy of psychogenic halitosis and its problems. Japanese Journal of Psychosomatic Dentistry. 1991 Jun 25;6(1):76-80.

15.Sugiura M. Approach to psychogenic halitosis. Shikaitenbo= Dental outlook. 1982 Nov;60(5):859.

16. Yaegaki K, Coil JM. Questionnaire for detecting the halitosis patients with psychological condition. injournal of dental research 2000 jan 1 (vol. 79, pp. 582-582). 1619 duke st, alexandria, va 22314 usa: amer assoc dental research.

17. iwata K, horikawa T , Namikawa I , editors. Medical and dental microbiology , Tokyo : ishiyaku publishing ; 1985. P. 267.

18. pedrazzi V, sato S, de mattos MG, lara EH, panzer H. tongue cleaning methods: a comparative clinical trial employing a toothbrush and a tongue scraper. J periodontal 2004;75:1009-1012

19. Rosenberg M, geleniter l, barki M, bar –ness R. daylong reduction of malodor by a two phase oil: water mouth rinse, as compared to chlorhexidine. J peridontol 1992;63:39-43.

20. Gnanasekhar JD. Aetiology, diagnosis and management of halitosis: a review. Periodontal Practice Today. 2007 Jun 1;4(3).

21. Harini G, Kaarthikeyan G. Advanced Drug Delivery Systems in Treating Periodontal Diseases-A Review. Journal of Dental and Medical Sciences (IOSR-JDMS). 2014;13:27-32.

22. Lakshmi T, Rajendran R, Krishnan V. Perspectives of oil pulling therapy in dental practice. Dental Hypotheses. 2013 Oct 1;4(4):131.

Health Eating Index for Children –A Review

NirishaSriram[1],R.Navaneethan[2],G.Deepa[3],
Graduate Student[1],Reader[2], Department of Orthodontics, Professor[3], Department of Pedodontics[3],
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Children are more prone to various diseases as their immune system is still in development. A good immune system can be builtonly when the child is at the best of health and good health in turn is dependent on a healthy diet and good exercise. A healthy diet means a good intake of food which has all the essential nutrients in the right amounts. The diet would vary slightly with individuals depending on their day to day activity. It also varies with the age and gender of the individual. Thus the quantity and the quality of food that is consumed are of most importance. When we see the health of the individuals in a community, it becomes important to quantify as to what constitutes a healthy diet both qualitatively as well as quantitatively. This quantification can be done with the help of Indices that measure the type and quantity of food consumed. Various committees have proposed different diet eating indices to standardize the type of food to be consumed. The indices serve not only to know the diet status of the individuals in a community but also serve as a guide to the individuals regarding their daily dietary requirements.

***Key words*:** *Diet indices, Health eating Index, Food Pyramid, Children,Healthy diet.*

Introduction

A good healthy smile boosts the self-confidence of an individual weather it is an adult or child. A good smile is dependent on strong, healthy teeth surrounded by a healthy periodontium which in turn depends on a healthy diet. The nutrition deficiency manifestations in the oral cavity serve as a testament to the importance of a healthy diet. This is where the role of indices comes in as they assess and quantify the nutritional status of various food products. These dietary indices calculate the food habits of an individual and the amount of food consumed which offers a brief knowledge of the person's health in relation to their diet.

Different diet quality assessment indices assign scores which indicate the health status of an individual based on the consumption of these food items[1]. At present two methods are used to assess the nutritional quality of the food items namely priori which gives scores to the food items based on food guides and a posteriori analysis which uses statistics to find the diet of a particular population[2]. The four most commonly used indices used are a) Health eating index, b) Diet quality index, c) Mediterranean diet score and d) Overall nutritional quality index[3]. Of the four Indices, the Health Eating Index is the most widely used and it has been modified in different countries to estimate the diet quality in the respective populations.

Health Eating Index (Hei)

It is a measure of diet quality that is in line with the Dietary Guidelines for Americans (DGA), which is the basis of nutrition policy for the US government. The health eating index

is revised every 5 years. The first HEI was introduced in 1995(Table 1). The HEI 1995 included 10 components with each component being given a score of 0-10. Of the 10 components, 5 components were for the type of foods that are present in the original food pyramid, 4 components for the five major food types that should be consumed in moderation or in limited amounts like fats, sodium etc. and 1 component measures the variety in the diet. The criteria for scoring were also clearly defined based on the number of servings, energy obtained from fats etc. An overall score of 80 and above was suggestive of a healthy diet, while a score between 80-50 was considered to require improvement while a score of less than 50 was considered poor. Further the scoring was categorised based on age and gender so as to take into considerations the different requirements at each age group and for each gender[4]. The HEI of American children estimated during from 1994 to 1996 showed that the children were in the category that needed improvement as the scores varied between 66.4 (lowest score over the years) to 74.4 (highest score over the years)[5]. The scores for children in 1999-2000 period ranged from 66 to 75.7 which was still in the need improvements group.[6]

Table 1: HEI-1995

Component	Score Ranges	Criteria for Maximum Score of 10	Criteria for Minimum Score of 0
Grain consumption	0 to 10	6 - 11 servings	0 servings
Vegetable consumption	0 to 10	3 - 5 servings	0 servings
Fruit consumption	0 to 10	2 - 4 servings	0 servings
Milk consumption	0 to 10	2 - 3 servings	0 servings
Meat consumption	0 to 10	2 - 3 servings	0 servings
Total fat intake	0 to 10	30% or less energy from fat	45% or more energy from fat
Saturated fat intake	0 to 10	Less than 10% energy from saturated fat	15% or more energy from saturated fat

Cholesterol intake	0 to 10	300 mg or less	450 mg or more
Sodium intake	0 to 10	2400 mg or less	4800 mg or more
Food variety	0 to 10	8 or more different items in a day	3 or fewer different items in a day

The HEI was updated over the years and in 2005 and very an updated version, HEI-2005 (Table 2) was created. The HEI 2005 was based on food patterns on the MyPyramid[7] and had 12 components. The various components are classified into two groups. The first was the "adequacy components" which contained all the essential food groups namely Total fruit, Whole Fruit, Total Vegetables, Dark green & Orange vegetables & Legumes, Total grains, Whole Grains, Milk, Meat and Beans and Oils. The second group was the moderation group which contained all the foods that have to be consumed in moderation namely the saturated fat, Sodium and Calories from Solid Fat, Alcohol and Added Sugars (SoFAAS). The first six components of the adequacy group had a scoring of 0-5 while milk, Meat & Beans and Oil had a scoring of 0-10 each. Among the moderation group, saturated fat and sodium had a scoring of 0-10 each while SoFAAS had a scoring of 0-20. The total score was again 100. The amount of food intake was estimated on a density basis that is the amount per 1000 calories intake[8]. The average HEI-2005 score for children aged between 2 to 17 years of age was 55.9 during 2003-2004[9]. This indicated that there required a large improvement in the diet patterns in the American children.

Table 2:HEI-2005

Component	Maximum points	Standard for maximum score	Standard for minimum score of zero
Total Fruit (includes 100% juice)	5	≥0.8 cup equiv. per 1,000 kcal	No Fruit

Whole Fruit (not juice)	5	≥0.4 cup equiv. per 1,000 kcal	No Whole Fruit
Total Vegetables	5	≥1.1 cup equiv. per 1,000 kcal	No Vegetables
Dark Green and Orange Vegetables and Legumes	5	≥0.4 cup equiv. per 1,000 kcal	No Dark Green or Orange Vegetables or Legumes
Total Grains	5	≥3.0 oz equiv. per 1,000 kcal	No Grains
Whole Grains	5	≥1.5 oz equiv. per 1,000 kcal	No Whole Grains
Milk	10	≥1.3 cup equiv. per 1,000 kcal	No Milk
Meat and Beans	10	≥2.5 oz equiv. per 1,000 kcal	No Meat or Beans
Oils	10	≥12 grams equiv. per 1,000 kcal	No Oil
Saturated Fat	10	≤7% of energy[5]	≥15% of energy
Sodium	10	≤0.7 gram per 1,000 kcal[5]	≥2.0 grams per 1,000 kcal
Calories from Solid Fats, Alcoholic Beverages, and Added Sugars (SoFAAS)	20	≤20% of energy	≥50% of energy

The HEI-2005 was later updated to HEI-2010 (Table 3) and the HEI 2010 had nearly the same components of the HEI 2005 with slight modifications.Like the HEI 2005, the new version has an “Adequacy group” and a “Moderation group”. The Adequacy group in HEI 2010 consists of the following components a) Total Fruit, b)Whole Fruit, c)Total Vegetables, d)greens and beans, e)Whole Grains, f)Dairy g)Total Protein foods, h) Seafood and Plant proteins and i) fatty acids. The scoring was between 0-5 for total fruit, whole fruit, total vegetables, greens and beans, total protein foods and seafood and plant proteins while dairy, whole grains and fatty acids have a scoring of 0-10 each. The moderation group consisted of Refined Grains, Sodium and Empty Calories. Refined grains and sodium had a scoring of 0-10 each while ‘empty calories’ was scored 0-20[10].

Table 3: HEI 2010

Component	Maximum points	Standard for maximum score	Standard for minimum score of zero
Adequacy:			
Total Fruit	5	≥0.8 cup equiv. per 1,000 kcal	No Fruit
Whole Fruit	5	≥0.4 cup equiv. per 1,000 kcal	No Whole Fruit
Total Vegetables	5	≥1.1 cup equiv. per 1,000 kcal	No Vegetables
Greens and Beans	5	≥0.2 cup equiv. per 1,000 kcal	No Dark Green Vegetables or Beans and Peas
Whole Grains	10	≥1.5 oz equiv. per 1,000 kcal	No Whole Grains
Dairy	10	≥1.3 cup equiv. per 1,000 kcal	No Dairy
Total Protein Foods	5	≥2.5 oz equiv. per 1,000 kcal	No Protein Foods
Seafood and Plant Proteins	5	≥0.8 oz equiv. per 1,000 kcal	No Seafood or Plant Proteins
Fatty Acids	10	(PUFAs + MUFAs)/SFAs ≥2.5	(PUFAs + MUFAs)/SFAs ≤1.2
Moderation:			
Refined Grains	10	≤1.8 oz equiv. per 1,000 kcal	≥4.3 oz equiv. per 1,000 kcal
Sodium	10	≤1.1 gram per 1,000 kcal	≥2.0 grams per 1,000 kcal
Empty Calories	20	≤19% of energy	≥50% of energy

Table 4:HEI 2015

Component	Maximum points	Standard for maximum score	Standard for minimum score of zero

Adequacy:			
Total Fruits	5	≥0.8 cup equiv. per 1,000 kcal	No Fruit
Whole Fruits	5	≥0.4 cup equiv. per 1,000 kcal	No Whole Fruit
Total Vegetables	5	≥1.1 cup equiv. per 1,000 kcal	No Vegetables
Greens and Beans	5	≥0.2 cup equiv. per 1,000 kcal	No Dark Green Vegetables or Legumes
Whole Grains	10	≥1.5 oz equiv. per 1,000 kcal	No Whole Grains
Dairy[5]	10	≥1.3 cup equiv. per 1,000 kcal	No Dairy
Total Protein Foods	5	≥2.5 oz equiv. per 1,000 kcal	No Protein Foods
Seafood and Plant Proteins	5	≥0.8 oz equiv. per 1,000 kcal	No Seafood or Plant Proteins
Fatty Acids	10	(PUFAs + MUFAs)/SFAs ≥2.5	(PUFAs + MUFAs)/SFAs ≤1.2
Moderation:			
Refined Grains	10	≤1.8 oz equiv. per 1,000 kcal	≥4.3 oz equiv. per 1,000 kcal
Sodium	10	≤1.1 gram per 1,000 kcal	≥2.0 grams per 1,000 kcal
Added Sugars	10	≤6.5% of energy	≥26% of energy
Saturated Fats	10	≤8% of energy	≥16% of energy

The next updated version HEI 2015 (Table 4), is yet to be published but based on the dietary guidelines 2015 by the US government, it is expected to retain all the components of the HEI 2010. The only change expected is the extra calories component being split into two namely a) added sugars and b) saturated fats with each component having a scoring of 0-10[11]. The HEI is the most commonly used dietary index and it has been used to estimate the quality of various diets[12-15], in different population samples and had also been used to associate diet with nutritional and other diseases[16-18].

The HEI-2010 scores for children ranged from 43.59 to 52.11, which was lower than the minimum score of 80[19]. The scores were closer to the “poor” category (51 and below). This showed the dire need to improve on the diet requirement in children. Similar findings have been found among pre-schoolers in Greece[20]with most children (80%) scoring below 50 while only 0.4% children were in the good category. Fernanda Rauber et al[21] studied thediet

quality of children of the low economic status using an adapted version of the HEI and got mean scores of 65.7 for children of 3to 4 years of age and a score of 65 for children aged 7 to 8 years. These values, though higher than those of the American and Greece children, cannot be compared to them as the HEI index used was modified for the Brazilian diet. Another Brazilian studyused HEI to standardise the study and found that rice fortified with micronutrients improved the folic acid, thiamine, haemoglobin concentrations in the blood. This study suggested that using fortified rice in school programs could improve the intake of micronutrients[22]. Another American study showed that the diet of children with autism did not vary much from the diet of normal children as measured by the HEI[23].Angelopoulos et al[24] found that majority (84.5%) of the participants fell under the HEI score of 50-80 which was the 'needs Improvement' category while 12 % had a 'poor diet' and only 3.5% children had a 'good diet'.

All the above studies show that there is a dire need for improvement in the diet of children, irrespective of which country they belong to. There is an overall deficiency in children who have of a well-balanced diet. And when such deficiency is noted in children of developed countries, the situation of the children in the developing and the underdeveloped world would paint a scary picture.

Conclusion

To conclude, indices help in correlating diet and the risk of nutritional and systemic diseases which indirectly affect the overall status of the oral cavity. There is a more need of calculation of these indices in children as they are more prone to have an irregular diet which might lead to nutritional deficiency. In such conditions the teeth of the children are equally affected as that of the general health. Moreover thechances of deformation are more common in primary teeth which will interrupt the growth of permanent teeth and its function. Having a hale and healthy diet will indirectly result in a good smile. For a healthy life appropriate proportion of all types of food is essential which is calculated with the help of dietary indices.

Corresponding Author

R.Navaneethan,

Reader, Department of Orthodontics

Saveetha Dental College,Saveetha Institute Of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai-600077, Tamil Nadu, India

Mobile Number: +91-9962787531

Email Id: ethan.nava@gmail.com

References

1. Waijers PMCM, Feskens EJM, Ocke MC. A critical review of predefined diet quality scores. Br J Nutr. 2007; 97(2):219-31.
2. Kastorini CM, Papadakis G, Milionis HJ, Kalantzi K, Puddu PE, Nikolaou V, et al. Comparative analysis of a-priori and a-posteriori dietary patterns using state-of-the-art classification algorithms: A case/case-control study. ArtifIntell Med. 2013; 59(3):175-83.
3. Carvalho KMB, Dutra ES, Pizato N, Gruezo ND and Ito MK. Diet quality assessment indexes. Rev. Nutr., Campinas. 2014; 27(5): 605-617.
4. Kennedy ET, Ohls J, Carlson S, Fleming K. The Healthy Eating Index: design and applications. J Am Diet Assoc. 1995; 95: 1103–1108.
5. Bowman SA, Lino M, Gerrior SA, Peter BP. The Healthy Eating Index, 1994-96. Family Economics and Nutrition Review;Washington. 1998; 11(3): 2-14.
6. Basiotis PP, Carlson A, Gerrior SA, Juan WY and Lino M.The Healthy Eating Index, 1999-2000: Charting Dietary Patterns of Americans. Family Economics and Nutrition Review; Washington. 2004; 16(1): 39-48.
7. Britten P, Marcoe K, Yamini S and Davis C. Development of food intake patterns for the MyPyramid food guidance system. Journal of Nutrition Education and Behavior. (2006); 38(6S): S78-S92.
8. Guenther PM, Reedy J, Krebs-Smith SM, Reeve BB and Basiotis PP. Development of the Healthy Eating Index-2005, Journal of the American Dietetic Association. 2008; 108(11): 1896-1901.
9. Fungwe T, Guenther PM, Juan WY, Hiza H, Lino M, (2009). The quality of children's diets in 2003-04 as measured by the Healthy Eating Index-2005.

USDA. Nutrition Insight 43. Online link https://www.cnpp.usda.gov/sites/default/files/ nutrition insights uploads/ Insight43.pdf.

10. Guenther PM, Casavale KO, Kirkpatrick SI, etal.Update of the Healthy Eating Index: HEI-2010, Journal of the Academy of Nutrition and Dietetics. 2013; 113(4): 569-580.
11. U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015–2020,(2015). Dietary Guidelines for Americans. 8th Edition. December 2015. Available at http://health.gov/dietaryguidelines/2015/guidelines/
12. Krebs-Smith SM, Reedy J, Bosire C. Healthfulness of the U.S. food supply: Little improvement despite decades of dietary guidance. Am J Prev Med. 2010; 38(5):472-477.
13. Hornick BA, Krester AJ, Nicklas TA. Menu modeling with MyPyramid food patterns: Incremental dietary changes lead to dramatic improvements in diet quality of menus. J Am Diet Assoc. 2008; 108(12): 2077-2083.
14. Kirkpatrick SI, Reedy J, Kahle LL, Harris JL, Ohri-Vachaspati P, Krebs-Smith SM. Fast food menu offerings vary in dietary quality, but are consistently poor. Public Health Nutr. 2014;17(4):924-31.
15. Reedy J, Krebs-Smith SM, Bosire C. Evaluating the food environment: Application of the Healthy Eating Index-2005. Am J Prev Med. 2010; 38(5):465-471.
16. Guo X, Warden BA, Paeratakul S, Bray GA.Healthy Eating Index and obesity. Eur J Clin Nutr. 2004; 58(12):1580-1586.
17. Schwingshackl L and Hoffmann G. Diet quality as assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension score, and health outcomes: a systematic review and meta-analysis of cohort studies. J AcadNutr Diet. 2015 ;115(5):780-800.e5.
18. McCullough ML, Feskanich D, Stampfer MJ, Giovannucci EL, Rimm EB, Hu FB, Spiegelman D, Hunter DJ, Colditz GA, and Willett WC. Diet quality and major chronic disease risk in men and women: moving toward improved dietary guidance. Am J Clin Nutr. 2002; 76(6): 1261-1271.
19. Banfield EC, Liu Y, Davis JS, Chang S, Alexis C. Frazier-Wood. Poor Adherence to US Dietary Guidelines for Children and Adolescents in the National Health and

Nutrition Examination Survey Population. Journal of the Academy of Nutrition and Dietetics. 2016; 116(1): 21-27.

20. Manios Y, Kourlaba G, Kondaki K, Grammatikaki E, Birbilis M, Oikonomou E and Roma-Giannikou E. Diet Quality of Preschoolers in Greece Based on the Healthy Eating Index: The Genesis Study, J American Diet Assoc. 2009; 109(4): 616-623.
21. Rauber F, LouzadaMLdC, Vitolo MR. Healthy Eating Index Measures Diet Quality of Brazilian Children of Low Socioeconomic Status. Journal of the American College of Nutrition. 2014; 33(1): 26-31.
22. Lucia CMD, Rodrigues KCC, Rodrigues VCC, Santos LLM, Cardoso LM, Martino HSD, Franceschini SCC and Pinheiro-Santana HM. Diet Quality and Adequacy of Nutrients in Preschool Children: Should Rice Fortified with Micronutrients Be Included in School Meals?Nutrients. 2016; 8(5): 296.
23. Graf-Myles J, Farmer C, Thurm A, et al. Dietary Adequacy of Children with Autism Compared to Controls and the Impact of Restricted Diet. Journal of developmental and behavioral paediatrics. 2013;34(7): 449–459.
24. Angelopoulos P, Kourlaba G, Kondaki K, Fragiadakis GA and ManiosY.Assessing children's diet quality in Crete based on Healthy Eating Index: The Children Study.European Journal of Clinical Nutrition. 2009; 63: 964–969.

Heart Rate Variability in Cardiovascular Syndromes

S.Rohini[1], Gayathiri[2], Rakshagan. V[3]

Graduate Student[1], Senior Lecturer[2], Department of Physiology , Senior Lecturer[3], Department of Prosthodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Heart rate variability (HRV) is a reliable reflection of the many physiological factors modulating the normal rhythm of the heart. In fact, they provide a powerful means of interplay between the sympathetic and parasympathetic nervous systems. It shows that the structure generating the signal is not only simply linear, but also involves nonlinear contributions. Heart rate (HR) is a nonstationary signal, and the variations in that may contain indicators of current disease, or warning about any impending cardiac diseases. This indicator may be always present or may occur at certain intervals of the day. HR variation analysis has become a popular non-invasive tool for assessing the activities of the autonomic nervous system. Heart rate variability (HRV) is widely used for quantifying neural cardiac control, and low variability is particularly predictive of death in patients after myocardial infarction.

Key Words:*Cardiac Death, Epinephrine andnor Epinephrine,Heart Rate Variability,Myocardial Infarction (MI), Vagus Nerve.*

Introduction

Heart rate, measured as beat to beat intervals, which is not constant and varies in time. Heart rate variability (HRV), the variation over time of the period between consecutive heartbeats, is predominantly dependent on the extrinsic regulation of the heart rate (HR). HRV is the ability of heart to adapt to changing circumstances by detecting and quickly responding to unpredictable stimuli. (1) Heart rate variability provides a non-invasive and indirect measurement of the autonomic control of the heart. A high variability in heart rate implies a healthy individual with a well-functioning autonomic nervous system. A low HRV indicates an abnormal autonomic system. Abnormalities in HRV have been reported in a variety of medical conditions including myocardial infarction, diabetes, the metabolic syndrome, end stage renal disease, chronic liver disease, hypoxic lung disease, and chronic heart failure and HIV infection. (2) HRVcan beanalysed with the variations in normal consecutive heart beats or R-R intervals.Analysis of Heart rate variability is very important and gives the overall cardiac health and state of the autonomic nervous system (ANS) which is responsible for regulating cardiac functions. The normal variability in HR is due to autonomic neural regulation of the heart and the circulatory system. (3) The balancing actions of sympathetic nervous system and parasympathetic nervous system controls the HR. The degree of variability in the heart rate provides information about the functioning of the nervous system on the heart rate and the heart's ability to respond.(4)

The amount of HR fluctuations around the mean HR can be used as cardio respiratory control system. It is an important tool to investigate the sympathetic and parasympathetic function of ANS. It also gives information regarding the sympathetic-parasympathetic autonomic balance and about the risk for sudden cardiac death (SCS).(5) It is influenced by various physiological factor including age, postural changes, and time of day. Pathological conditions such as congestive heart, diabetic neuropathy and coronary heart disease also are associated with alterations in heart rate variability. Heart rate and rhythm are under the control of the autonomic nervous system. (6) The parasympathetic influence on heart rate is regulated via release of acetylcholine by the Vagus nerve. Muscarinic acetylcholine receptors respond to this release mostly by an increasing the K^{2+} conductance in cell membrane. The sympathetic influence on heart rate is mediated by epinephrine and norepinephrine. Sympathetic stimulation, occurring due to stress, exercise and heart disease, increases the heart rate by increasing the firing rate of pacemaker cells in the heart's sino atrial node. (7)

Parasympathetic activity, results from the functions of internal organs, trauma, and the allergic reactions, decreases the firing rate of pacemaker cells and the HR, providing a regulatory balance in physiological autonomic function. Rhythms from sympathetic and parasympathetic activity contribute separately for the modulation of heart rate (RR) intervals of the QRS complex in the electrocardiogram (ECG)at distinct frequencies.[8] The RR interval variations present during resting conditions represent beatbybeat variations in cardiac autonomic inputs.

Heart Rate Variability in Myocardial Infarction

A reduction in parasympathetic cardiac control and predominance of sympathetic activity are seen in patients with acute myocardial infarction.HR variability in AMI was related to age, as older patients tended to have lower HR variability values than younger AMI patients. (9) It was found that low HR variability at an early stage of AMI is significantly associated with early mortality and major complications. It was shown that, the HRV decreases withthe recent MI. Depressed HRV after Myocardial infarction indicates decrease in vagal activity directed to the heart. (10) HRV in patients surviving an acute MI reveal a reduction in total and in the individual power of spectral components.Clinical depression is associated with an increased risk for mortality in patients with a recent myocardial infarction (MI). (11)

Decreased HRV in Chronic Obstructive Pulmonary Disease

Patients with COPD are less able to respond to sympathetic and parasympathetic stimuli, in other words their control system are working at saturation level, so they could not have the normal range of variation and even opposite stimuli cannot modify their autonomic pattern. In patients with heart failure, the autonomic system is driven by an exaggerated sympathetic tone.(12) Similarly, patients with COPD, have altered sympatho-adrenal response to chronic moderate bronchoconstriction. One possible reason for the lack of RR variability in patients withCOPD might be a light constant compensatory sympathetic activity in response to an increase in airwayresistance. Larsson et al. suggested that this impaired sympatho-adrenal response is not from an alteration of plasma noradrenalin concentrations. Another possibility is that sympathetic outflow response may be normal, but that there could be a block to the

action circulating catecholamine, due to receptor down regulation. The down regulation could be due in part to long-term therapy with theophylline or d-agonists and in part to increased circulating endogenous catecholamine.(14) Usually in patients with COPD an imbalance is seen in autonomic nervous system activity. This is apparently driven at rest by an increase in vagal activity and through the lack of responses to sympathetic stimulation. This altered balance could contribute to the airways obstruction in COPD.(15)

Heart Rate Variability in Chronic Heart Failure

Despite recent advancement in management of chronic heart failure, mortality remains high and death often occurs suddenly. Identification of patients with chronic heart failure at risk for sudden death remains difficult. Low heart rate variability (HRV) is an indicator of increased risk of sudden cardiac death after myocardial infarction and in patients with chronic heart failure.(16) Regular physical activity could modify the risk of sudden cardiac death. Chronic heart failure is characterised by significant autonomic dysfunction consisting of sympathetic activation, parasympathetic withdrawal, and peripheral organ unresponsiveness.

Chronic heart failure (CHF) is also associated with major abnormalities of autonomic cardiovascular control. Among several prognostic factors, the extent of neurohormonal activation (17),particularly plasma norepinephrine levels, are found to be independently related to survival ofpatients with heart failure. Profound abnormalities in autonomic control, characterized by generalized sympathetic over activity and parasympathetic withdrawal, are a typical feature of neuroendocrine activation in heart failure.(18) It is also expected that increased sympathetic activity would be accompanied by a relative predominance of Low frequency(LF)oscillations –domain analysis of HRV.However, both increased and reducedLF power were found to be associated with an increased risk of cardiac death. In the present studies it has been stated that reduced LF power (particularly duringcontrolled breathing) independently predicted sudden, presumably arrhythmic death in a multivariate model testing the majority of the recognized pathological determinants of CHF in both the derivation and validation series. (19) In CHF patients, abnormal breathing patternsplays a confounding role thatis abolished by controlled respirationor O2administration. Moreover the absence of a sustained abnormal breathing pattern alters the regularity of tidal volume (i.e. irregular) in CHF patients, thus affecting the HRV assessment. (20) The main finding isthat in patients

with chronic heart failure, heart rate variability, as assessed by time domain and spectralanalysis, has prognostic value not only for the identification of patients at increased risk for mortality but, most importantly, also for sudden death. Among time domain indexes, a lower SDNN is a significant and independent predictor of all-cause mortality and of progressive heart failure death. Among spectral measures, reduced power within the low frequency band during daytime is a significant and independent predictor of sudden death risk. Hence reduced heart rate variability has been observed consistently in patients with cardiac failure.(21)

Effect of Exercise Training on Heart Rate Variability

There are two types of exercise training: Resistance & dynamic exercise training. The adaptation of cardiovascular system to resistance exercise training is different from adaptation to dynamic exercise training. Heart muscle shows signs of concentric hypertrophy in resistance training, whereas in dynamic training the adaptation is characterised by increase on heart cavities & limited heart wall thickening.(22) In such situation, hypertrophy is considered as eccentric. Hence dynamic training is considered to be more efficient than resistance training on the decrease of heart rate & systolic blood pressure at rest and also on the increase of stroke volume at rest and during exercise. Thus it is possible that various types of exercises influence the HRV in a different manner. Hence Regular physical exercises can reduce the risk of sudden cardiac death. (23)

Effect of HRV in Coronary Artery Disease

Autonomic nervous controls cardiovascular system and has a distinct circadian rhythm,and this may be animportant mechanism underlying the diurnal distribution of cardiac events such as myocardial ischemia, myocardial infarction, and cardiac death.(24) Analysis of heart rate variability (HRV) in the frequency domain is a novel method of studying cardiovascular neural regulation. Decrease of HRV is frequently associated with coronary artery disease (CAD). Theamplitude of circadian rhythm of the HF component of HRV was blunt in the patients with CAD compared with healthy ones suggesting that the vagal responses to endogenous or exogenous stimuli are reduced in CAD.(25)The circadian rhythm of

normalized power of the LF component of HRV was also blunted in the patients with CAD, suggesting that the diurnal fluctuation of both vagal and sympathetic activity is altered in CAD. There was less variation in heart rate during deep breathing in patients with coronary artery disease. Heart rate variation during deep breathing was not sufficiently related to age in patients with CAD. (26)

Autonomic nervous system imbalance in the form of reduced vagal and enhanced sympathetic activity is supposed to be the basis of diminished heart rate variability in coronary artery disease patients. Previous literature suggests that distorted vagal nerve endings and enhanced release of norepinephrine from sympathetic nerve fibers is the underlying mechanism of altered activity of autonomic nervous system. Preceding myocardial ischemiacauses alteration in distribution ofautonomic nerve fibers. Heterogeneity of autonomic innervation leads to augmented sympathetic influence and reduce vagal intonation in coronary artery disease patients leading to reduced heart rate variability.(27)

Chronic myocardial infarction (MI) causes destruction of the ventricular neural receptors, which results in altered autonomic regulation and ageing has significant effects on vagal heart rate control. One of the recent study done in patients with CAD, but without a prior MI, indicated that the 24-h average frequency domain measures of HRV were not significantly reduced in CAD patients compared with age-matched healthy subjects.(28)Healthy subjects have a distinct circadian rhythm of HRV, while this rhythm seems to be blunted in CAD patients, and their autonomic responses to the sleep-awake rhythm are abnormal, suggesting that the central modulation of the autonomic control of the heart rate is altered in CAD. Similar abnormalities in the circadian rhythm of HRV have been observed in hypertensive patients. (29)

Low HRV is always associated with an increased risk of overall mortality in post infarction patients. According to the results of study done by Farrell et al. and Bigger et al. (30) suggested that impaired HRV was particularly related to an increased risk of arrhythmic sudden death. Reductions in the VLF and LF spectral components of HRV seem to be more closely related to an increased risk of sudden arrhythmic death than the HF component. On the other hand, Bigger et al. also reported that reduced HRV also predicts cardiac mortality in patients with remote MI and Rich et al.(31) showed in a small population with CAD, but

without a prior MI, that reduced HRV increased significantly the risk of cardiac mortality. The incidence of arrhythmic cardiac death is relatively low in these patient populations, suggesting that low HRV may also be associated with the risk of non-arrhythmic cardiac death, such as acute coronary events or progressive heart failure.(32)

Depressed Mood and Herat Rate Variability

Depression is an independent risk factor for mortality after MI Parasympathetic tone is an important factor in the development of cardiac arrhythmia.(33) Among healthy individuals, parasympathetic tone helps to prevent ventricular arrhythmias by maintaining the electrical stability of the heart. When parasympathetic tone is decreased, cardiac arrhythmias are more likely to occur (34). Although stress decreases parasympathetic tone, under normal conditions enough parasympathetic tone remains during stress to prevent ventricular arrhythmias. Neural fibres are damaged due to MI, such as when the autonomic influences on heart on compromised, extreme stress may provoke lethal arrhythmias and sudden death.

Power Spectral Analysis of HRV Identifies Congestive Heart Failure

Autonomic dysfunction seems to be involved in the progression and prognosis of severe congestive heart failure. Power spectrum analysis of heart rate variability in congestive heart failure may identify patients with a more pronounced sympathovagal imbalance and, as a consequence, could be at major risk of the disease worsening and have a poorer prognosis. Heart rate variability was reduced in patients with congestive heart failure. Two spectral patterns were identified .The first was characterised bya predominant LF component and a reduced HF component, suggesting sympathetic predominance and parasympathetic withdrawal. In the second a major reduction intotal power, an undetectable LF componentand a low amplitude HF peak. Patients with undetectable LF were more severely affected and with depressed left ventricular function and a higher degree of sympathoexcitation, reflected by the higher plasma noradrenalinconcentrations. It was also suggested that parasympathetic activity is inversely related and that sympathetic activity and noradrenaline concentrations are directly related to the severity of heart failure and prognosis.

Therapeutic Interventions and Heart Rate Variability

It is always important to find therapeutic methods which would improve heart rate variability. Beta-blocking therapy has been shown to increase HRV both in healthy subjects and in patients with Coronary artery disease.(36)This improvement in HRV has been suggested to contribute to the protective effects of beta-blockade in ischaemic heart disease. The exact reason is unknown for increase in the measures of HRV after beta-blockade but itis merely due to the decrease of heart rate or increases the vagal modulation of heart rate. Scopolamine, which is a vagotonic agent, has also been shown to increase the respiratory component of HRV. Angiotensin-converting enzyme inhibitors may also improve the HRVand physical training has been reported to increase it. Despite these potential methods for improving HRV, there are other pharmacological or other methods to improve HRV and prevent cardiovascular morbidity or mortality.

Effect of Beta Blockers and Other Cardioactive Drugs on Heart Rate Variability

Previous studies showed that beta-blocker therapy augmented heart rate variability in patients with coronary artery disease. Thusbetablockers significantly increased high frequency power spectra and the rootmean square of the differences of successive RR intervals. Power in “the low frequency band tend to be higher with beta blockers, but the difference relative to placebo was not significant. The standard deviation of normal RR intervals and very low and ultra- low frequency power, which are independent predictors of mortality in patients after acute myocardial infarctionwere also significantly augmented by betablockers.

Mechanisms of Modifying Heart Rate Variability

Use of beta blockers resulted in significant argumentation of high frequency power and the root mean square successive difference of normal RR intervals, which is considered to reflect parasympathetic activity. However, reduction of the average heart rate itself (i.e., increase in the mean RR intervals) might result in increase in both the time and frequency domain measures of RR interval variability. Thus beta blocker induced changes of variability indexes may be due to increased vagal tone, reduced sympathetic beta receptors stimulation or a

combination of the two. The equal effects of metoprolol and atenolol on heart rate and high frequency power suggests that these two beta blockers with different lipophilicity elicited similar changes of cardiac vagal sympathetic balance.

Conclusion

Heart rate variability has proved to be a valuable tool to investigate the sympathetic and parasympathetic function of the ANS.HRV analysis has become important in cardiology , because its measurements are easy to perform,non-invasiveand provide prognostic information on patients with heart disease.

Corresponding Author

Rakshagan. V

Senior Lecturer, Department of Prosthodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email Id:rakshga@yahoo.com

References

1. Saul JP. Beat-to-beat variations of heart rate reflect modulation of cardiac autonomic outflow.News Physiol Sci 1990 5:32–3.
2. Frasure-Smith N, Lesperance F, Talajic M. Depression following myocardial infarction: impact on 6-month survival. JAMA 1993; 270:1819 –25.
3. Rector TS, Olivari MT, Levine TB, Francis GS, Cohn JN. Predicting survival for an individual with congestive heart failure using the plasma norepinephrine concentration. Am Heart J 1987; 114:148-52.

4. Airaksinen KEJ, Ikaheimo MJ, Linnaluoto MK, Niemela M, Takkunen JT. Impaired vagal heart rate control in coronary heart disease. Br Heart J. 1987; 58:592-597.
5. Bigger JT, Albrecht P, Steinman RC, Rolnitzky LM, Fleiss JL, Cohen RJ. Comparison of time and frequency domain-based measures of cardiac parasympathetic activity in Holter recordings after myocardial infarction. Am J Cardiol. 1989; 64:536-566.
6. Carney RM, Blumenthal JA, Stein PK, Watkins L, Catellier D, Berkman LF, Czajkowski SM, O'Connor C, Stone PH, Freedland KE. Depression, heart rate variability, and acute myocardial infarction. Circulation 2001, 104:2024.
7. Kienzle MG, Ferguson DW, Birkett CL, Myers GA, Berg WJ, Mariano DJ. Clinical, Hemodynamic and Sympathetic Neural correlates of heart rate variability in congestive heart failure. Am J Cardiol 1992; 69:761-7
8. Hayano J, Yamada A, Mukai S, Sakakibara Y, Yamada M, Ohte N, Hashimoto T, Fujinami T, Takata K. Severity of coronary atherosclerosis correlates with the respiratory component of heart rate variability. Am Heart J. 1991; 4:1070-1079.
9. Swedberg K, Eneroth P, Kjekshus J, Wilhelmsen L, for the CONSENSUS Trial Study Group. Hormones regulating cardiovascular function in patients with severe congestive heart failure and their relation to mortality. Circulation 1990; 82: 1730–6.
10. Ahern DK, Gorkin L, Anderson JL, Tierny C, Hallstrom A, Ewart C, Capone RJ, Schron E, Kornfeld D, Herd A, Richardson DW, Follick MJ. Biobehavioral variables and mortality or cardiac arrest in the Cardiac Arrhythmia Pilot Study (CAPS). Am J Cardiol 1990; 66:59-62
11. Frasure-Smith N, Lesperance F, Talajic M. Depression and 18-month prognosis after myocardial infarction. Circulation 1995; 91:999 –1005.
12. Ponikowski P, Anker SD, Chua TP, Szelemej R, Piepoli M, Adamopoulos S, Webb-Peploe K, Harrington D, Banasiak W, Wrabec K, Coats AJ. Depressed heart rate variability as an independent predictor of death in chronic congestive heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. Am J Cardiol. 1997; 79:1645–1650.
13. Huikuri HV, Kessler KM, Terracall E, Castellanos A, Linnaluoto MK, Myerburg RJ. Reproducibility and circadian rhythm of heart rate variability in healthy subjects. Am J Cardiol. 1990; 65:391-393.

14. Szabó BM, van Veldhuisen DJ, van der Veer N, Brouwer J, De Graeff PA, Crijns HJ. Prognostic value of heart rate variability in chronic congestive heart failure secondary to idiopathic or ischemic dilated cardiomyopathy. Am J Cardiol. 1997; 79: 978–980.
15. Malliani A, Pagani M, Lombardi F, Cerutti S. Cardiovascular neural regulation explored in the frequency domain. Circulation. 1991;84:482-49
16. Ho KKL, Pinsky JL, Kannel WB, Levy D. The epidemiology of heart failure: The Framingham Study. J Am Coll Cardiol 1993; 22 (Suppl): 6A–13A.
17. Larsson K, Hjemadahl P, Theodorsson E. Acute bronchoconstriction is not a stimulus for sympatho-adrenal activation in asthmatic or healthy subjects. Eur Respir J 1990; 3:273-81.
18. Binkley PF, Nunziata E, Haas GJ, Nelson SD, Cody RJ. Parasympathetic withdrawal is an integral component of autonomic imbalance in congestive heart failure: demonstration in human subjects and verification in a paced canine model. JAm Coll Cardiol 1991; 18:464-72.
19. Carney RM, Blumenthal JA, Freedland KE, Stein PK, Howells WB, Berkman LF, Watkins LL, Czajkowski SM, Hayano J, Domitrovich PP, Jaffe AS. Low heart rate variability and the effect of depression on post—myocardial infarction mortality. Arch Intern Med 2005, 165:1486–1491.
20. Hayano J, Sakakibara Y, Yamada M, Ohte N, Fujinami T, Yokoyama K, Watanabe Y, Takata K. Decreased magnitude of heart rate spectral components in coronary artery disease: its relation to angiographic severity. Circulation. 1990; 81:1217-1224.
21. Turton MB, Deegan T. Circadian variations of plasma catecholamine, cortisol, and immunoreactive insulin concentrations in supine subjects. Clin Chim Acta. 1974; 55:389-397.
22. Casolo G, Balli E, Taddei T, Amuhasi J, Gori C. Decreased spontaneous heart rate variability in congestive heart failure. Am J Cardiol 1989; 64:1162-7
23. Eckberg DL, Drabinky M, Braunwald E. Defective cardiac parasympathetic control in patients with heart disease. N Engl J Med 1971; 285:877-83.
24. Ramaswamy C. Effect of mobile phone radiation on heart rate variability. J. Evolution Med. Dent. Sci 2016; 5(90): 6717-6721.
25. Frasure-Smith N, Lesperance F, Juneau M, Talajic M, Bourassa MG. Gender, depression, and one-year prognosis after myocardial infarction. Psychosom Med 1999; 61:26 –37.

26. Hinkle LE, Carver ST, Plakum A. Slow heart rates and increased risk of cardiac death in middle-aged men. Arch Intern Med 1972; 129:732-50.
27. Hirsch AT, Dzau VJ, Creager MA. Baroreceptor function in congestive heart failure: effect on neurohumoral activation and regional vascular resistance. Circulation 1987; 75 (Suppl IV): IV-36–48.
28. Ladwig KH, Kieser M, Konig M, Breithardt G, Borggrefe M. Affective disorders and survival after acute myocardial infarction: results from the post-infarction late potential study. Eur Heart J 1991; 12:959 – 64.
29. Lombardi F, Sandrone G, Pernpruner S, Sala R, Garimoldi M, Cerutti S, Baselli G, Pagani M, Malliani A. Heart rate variability as an index of sympathovagal interaction after acute myocardial infarction. Am J Cardiol. 1987; 60:1239-1245.
30. Samuel Sundar Doss D, Anandhalakshmi S, Rekha K, Akhil Antony K. Effect of smoking on heart rate variability in normal healthy volunteers. Asian J Pharm Clin Res vol 9, Issue 4, 2016, 230-234.
31. Mortara A, Sleight P, Pinna GD, Maestri R, Prpa A, La Rovere MT, Cobelli F, Tavazzi L. Abnormal awake respiratory patterns are common in chronic heart failure and may prevent evaluation of autonomic tone by measures of heart rate variability. Circulation. 1997; 96:246–252.
32. Muller JE, Tofier GH, Stone PH. Circadian variation and triggers of onset of acute cardiovascular disease. Circulation. 1989; 79: 733-743.
33. R.Archana, R.Mukilan. Beneficial effect of preferential music on exercise induced changes in heart rate variability. Journal of clinical & Diagnostic research. 2016, vol - 10(5): CC09-CC11.
34. Rothschild M, Rothschild A, Pfeifer M (1988) Temporary decrease in cardiac parasympathetic tone after acute myocardial infarction. Am J Cardiol 18:637–639
35. Saul JP, Aray Y, Berger RD, Lilly LS, Colucci WS, Cohen RJ. Assessment of autonomic regulation in chronic congestive heart failure by heart rate spectral analysis. Am J Cardiol 1988; 61:1292-9
36. Tristani FE, Kamper DG, McDermott DJ, Peters BJ, Smith JJ. Alterations of postural and Valsalva responses in coronary heart disease. Am J Physiol. 1977; 233:H694-H699.

Hibiscus *rosasinensis* – AReview of Literature

Sameer Mohideen Gani[1],Anitha Roy[2], Aravind Sivakumar[3],
Graduate Student[1],Head[2], Department of Pharmacology,Senior Lecturer[3], Department of Orthodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Hibiscus rosasinensis Linn. is certain to emerge in the near future as a major player in the growing field of herbal health supplements and medicines both in daily self-care and in professionally managed health care system.The principal constituents of Hibiscus rosasinensis Linn. areflvones. Flvones contain quercetin-3-diglucoside, quercetin-3,7-diglucoside, cyaniding-3,5-diglucoside, quercetin-3-sophorotrioside, kaempferol-3xylosylglucoside, cyaniding-3-sophoroside-5-glucoside and other constituent are cyclopeptide alkaloid, cyanidin chloride, hentriacontane, riboflavin, ascorbic acid, thiamine, taraxeryl acetate, ß-sitosterol , cyclicacidssterculic and malvalic acids. All the parts of Hibiscus rosasinensis Linn and chemical constituents are used as anti-tumor, antifertility, antiovultory, antiimplantation, anti-inflammatory, analgesic, antiestrogenic, antipyretic, antispasmodic, antiviral, antifungal, antibacterial, hypoglycaemic, spasmolytic, CNS depressant, hypotensive and juvenoid activity. This article compile all the information related to Hibiscus rosasinensis Linn.

Key Words:*Hibiscus rosasinensis, drugs, anti-inflammatory, juvenoid activity, antioxidants*

Introduction

Plants have been a source of medicine from time immemorialand a number of modern drugs have been isolated from natural sources, many based on their use in traditional medicines.over the years, world health organization has advocated traditional medicines as safe remedies for aliments of both microbial and non microbial origins[1].Hibiscus rosa-sinensis, known colloquially as the chinese hibiscus, china rose and shoe flower, is an evergreen flowering shrub native to east asia.it is widely grown as an ornamental plant throughout the tropics and subtropics. The flowers are large, generally red in the original varieties, and firm, but generally lack any scent. Also many colors are available in a single, double or multi-shades including white, yellow, orange, red, pink, salmon, purple, etc. Hibiscus rosa-sinensis, is the national flower of malaysia. The leaves are alternate, simple, ovate to lanceolate, often with a toothed or lobed margin. The flowers are large, conspicuous, trumpet-shaped, with five or more petals. The fruit is a dry five-lobed

capsule, containing several seeds in each lobe, which are released when the capsule dehisces (splits open) at maturity. Uses: to induce abortion, ease menstrual cramps and to help in childbirth. To treat headaches. A preparation from the leaves is used to treat postpartum relapse sickness, to treat boils, sores and inflammations. Good for hairs. Hibiscus flowers are reported to possess anti-fertility property by ancient ayurvedic texts. It is also used for the worship of devi and especially the red variety takes an important part in tantra. Hibiscus is a popular herbal plant which belongs to the family malvaceae and has high medicinal value.its 250 species are widely distributed in tropical and subtropical regions and are reported to posses various medicinal properties including antitumor, antihypertensive, antioxidant and anti ammonemic.[1–4] the extracts from different parts of the plant are used as emmenagogues,abortifacients, contraceptives and labour inducers. It has various pharmacological actions like ameliorative potential,anti fertility activity, anti inflammatory activity, antidiabetic activity, anti fungal activity and cardio protective functions. A literature review is done to study these properties of hibiscus plant.

Common Names

China rose, jaba, jasum ,sadaphool , sambathoochedi , jasunt , mandasa (5).

Chemical Constituents

Composition: various parts of the plant contain a number of chemical constituents, that are of major pharmacological importance. The stem and leaves contain stigmasterol, β-sitosterol, taraxeryl acetate and three other cyclopropane compounds along with their derivatives. Flowers are composed of flavonoids, diglucoside, and vitamins like thiamine, riboflavin, ascorbic acid and niacin (ghani, 2003). Quercetin-3-diglucoside, 3, 7- diglucoside, cyanidin-3-sophoroside-5-glucoside and cyaniding-3,5-glucoside have been isolated from the bright yellow flowers of this plant. Along with these compounds, kaemferol-3-xylosyl glucoside has been isolated from white ovary flowers (kumar, 2012). Five new phyto constituents have been isolated from the flowers of hibiscus rosasinensis. These were found to be n-docosane, n-entacos-4-en-3-one-18, 23-diol and henicos-11-ene-8-one, stigmast-5-ene-3 β, 4a- diol, stigmast-5-ene-3 β-benzyloxy-12 β-ol (kasture, 2000; siddiqui, 2006). The total anthocyanin composition was reported to be 165 mg/kg with about 6% reduction due to fermentation. Tannin content is reported as 11.8g/kg. Ascorbic acid and total polyphenol composition is established as 478 mg /kg and 14.4 mg/g, respectively. The phytochemical screening reveals the presence of alkaloids and saponins. The antioxidant activity of the extract is significantly lower (ec50 = 43.9 µg/ml) compared to ascorbic acid (e 50 = 3.3 µg/ml) (mak, 2013). Total phenolic content in hibiscus rosasinensis was reported to be 48.4±1.03mg catechol equivalent/g and the total flavonoid content was established as 24.26 ± 1.1mg catechol equivalent/g (shirwaikar, 2005).leaves and stems contain β-sitosterol, stigmasterol, taraxeryl acetate and three cyclopropane compounds and their derivatives. Flowers contain cyanidindiglucoside, flavonoids and vitamins, thiamine, riboflavin, niacin

and ascorbic acid (ghani, 2003). Quercetin-3-diglucoside, 3,7-diglucoside, cyanidin3,5-diglucoside and cyanidin-3-sophoroside-5glucoside have been isolated from deep yellow flowers.kaempferol-3xylosylglucoside has been isolated from ovary white flowers (rastogi&mehrotra, 1993)(5).

Ecology of Hibiscus

The genus 'hibiscus' includes both annual and perennial herbaceous plants, woody shrubs and small trees. The leaves are generally simple, ovate to lanceolate, having a lobed or toothed margin. The flowers are generally big, conspicuous and trumpet shaped having five or more petals. They are coloured flowers ranging from white to purple, red, pink and sometimes yellow. The petals are very broad ranging from 4 to 15cm. Flowers have nectarines, that are composed of several tightly packed glandular hairs, and are generally positioned at the sepals. The fruit is a capsule made up of dry five-lobes. It contains numerous seeds in each of its lobes, which then are released at the time of maturation, when the capsule splits open (alarcon, 2007; ames, 2013). Hibiscus plants are a source of beneficial ecological, aesthetic, culinary and medicinal values. Ecologically, the large flowers give nectar to large pollinators such as hummingbirds. Many insects use these flowers as a source of food. It is also well known for its aesthetic values. There are a wide range of varieties of this plant. They can be of many colours such as orange, red, white and pink having a single or double set of petals. Being large and trumpet shaped, the hibiscus is often used as a garden plant as well as a potted plant. They issn: 0974-2115 www.jchps.com journal of chemical and pharmaceutical sciences october-december 2015 971 jcps volume 8 issue 4 are more popular among hawaiian women who traditionally wear a single flower. This plant is also used in the preparation of commercial teas. Hence, this plant, while advancing its own individual functions such as reproduction and survival, also advances a larger function for the ecosystem as well as for humans (vitullo, 2009; ames, 2013).

Bioactivity of Hibiscus Components

Hibiscus is one of the medicinal plants that generally find applications in treating diseases such as oxidative stress like hypertension and cancer. This is because they are made up of strong antioxidants like alkaloids and flavonoids / anthocyanins. Anthocyanins are useful in therapy against cardiovascular diseases and cancer (trevisanto, 2000) and age related conditions such as dementia or alzheimer's disease (commenges, 2000; milugo, 2013). Flavonoids have the ability to induce human protective enzyme systems. They have also been found to have protective effects against many infectious bacterial and viral diseases including degenerative diseases such as cardiovascular diseases, cancers, and other age-related disorders (cook, 1996; kumar, 2013; filippo, 2014). Flavonoids also have antibacterial, antiviral (critchfield, 1996; cushnie, 2005), anticancer and anti-inflammatory properties. Flowers of the plant contain ascorbic acid whose biological role is to act as a reducing agent by donating electrons in several enzymatic reactions and a few non-

enzymatic ones. The one-and two-electron oxidized forms of vitamin c that is, semi hydro ascorbic acid and dehydro ascorbic acid, respectively, can be reduced in the body by glutathione and nadh- dependent enzymatic mechanisms (meister, 1994; ewadh 2014). The leaves and stem contain cyclopropane compounds that are inactive at the gaba and glycine receptors. They instead act as an nmda- receptor antagonist (hemmings, 2006; hudson, 2011). It inhibits the ampa receptor and nicotinic acetylcholine receptors, and activates certain k channels (hudson, 2011; borghese, 2012)

Traditional Uses

Hibiscus rosasinensis has been used in siddha medicine, a traditional tamil system from south india, for many centuries. Hibiscus extracts have been used for ages in ayurveda to cure many ailments. The plants have the natural health benefit that can be used to cure diseases naturally. They are used to cure ailments such as cough cold, hair loss and hair greying also. The flowers and leaves of this plant play a major role in hair treatment. These are ground into a fine paste with water and this is generally used as a shampoo plus conditioner. The plant also helps to improve the overall texture and health of hair. Hibiscus is a sweet sour herb and is used in the preparation of herbal teas. It acts as an antioxidant and also helps in the reduction of cholesterol levels (esa, 2010). It has also been used in the traditional medicine for treating colds, loss of appetite, disorders of the respiratory tract. The plant is beneficial as a mild laxative, expectorant and diuretic. The hibiscus rosasinensis has been found to have emmenagogue effects which can stimulate menstruation and in some women, cause an abortion (ernst, 2002; ali, 2005; de boer, 2014).

CardioprotectiveActivity

The medicinal values of the flowers of hibiscus rosasinensishas been mentioned inliterature as useful in disorders of the heart(6).in recent times, both experimental and clinical studies have shown that the dried flower powder of hrs has significant protective effects in ischemic heart disease [ihd] [7,8]. Oral administration of flowers of hibiscus rosasinensisprevents the oxidative stress and the structural changes associated with iri. The mechanism of such protection of chronic oral administration of hibiscus rosasinensismay be due to myocardial adaptation, oxidative stress is mediated through augmentation of cellular antioxidants such as glutathione, sod, catalase [9].Flowers of hibiscus rosasinensisare particularly useful agents, as they could enhance myocardial endogenous antioxidants without producing any cytotoxic effects.A study conducted to investigate the cardioprotectiveeffect of hrs flowers(dried pulverized) showed that hrs flowers augmentedendogenous antioxidant compounds of rat heart and also prevented the isoprotenelol induced myocardial injury.

Antioxidant ActivityAnd Cognitive Enhancing

Hibiscus rosasinensisflowers million contain anthocyanins, which may be responsible for its antioxidant effects.(10)alzheimer's disease (ad) is a neurodegenerative disease causing memory loss and dementia, which mostly affects the elderly population.(11)the pathophysiology of ad is complex including defective beta-amyloid (aβ) protein metabolism, abnormalities of glutaminergic, adrenergic, serotonergic and dopaminergic neurotransmission, and the potential involvement of inflammatory and oxidative pathways.[12] the drugs with antioxidant effects might be beneficial for preserving brain function. Antioxidant enzymes are involved in the reduction of oxidative stress.[13] antioxidant enzymes display the reduced activities in the affected brain region of alzheimer's disease patients. Antioxidant enzymes display
The reduced activities in the affected brain region of patients of alzheimer's disease. Moreover, the reduction in the level of intracellular oxidized protein under these conditions has been associated with the improvement of cognitive and/or psychomotor functions (14). Hibiscus rosasinensis may be useful in the treatment or prevention of various cognitive disorders.

Anti – Bacterial Activity

Most of the flower extracts have an antibacterial activity against the human pathogens such as e. Coli, b. Subtillis, p. Aeruginosa, s. Aureus, streptococcus sp. Salmonella sp. The crude extracts containing multiple organic components including flavonoids, tannins, alkaloids, triterpenoids, all of which are known to have antibacterial affects. Flower extract contain phenolics compounds like tannins that are very good antimicrobial agent[15]. Flavonoids especially are known to be effective antimicrobial agent against a wide arrayof microorganisms. The activity is attributed to their ability to complex with extra cellular and soluble proteins and with bacterial cell wall[16].Most of the extracts showantibacterial activity against e. Coliwhich are common member of the normal flora of large intestine. It is predominant facultative organism in the gastrointestinal tract and colonizes the tract within hours or few days and is responsible for causing diarrhea.Pseudomonas sp. Causes urinary tract infection, respiratory system infection, dermatitis soft tissue infection, gastrointestinal infection and a variety of systemic infection. Thus the extractsof hibiscus rosasinensiscan be used as an important antibiotic to cure above mentioned disorders caused by the different strains of bacteria.

Wound Healing Activity

Wound healing is the process of repair that follows injury to the skin and other soft tissues. Following injury, an inflammatory response takes place and the cells below the dermis begin to increase collagen production. Later, the epithelial tissue (the outer skin layer) is regenerated.(17) topical application of h. Rosa sinensis improved wound contraction and closure and the effects were distinctly visible starting from 4th post-wounding day. Flavonoids promote the wound-healing process mainly due to their astringent and

antimicrobial properties which appear to be responsible for the wound healing.[18] the wound-healing property of h. Rosa sinensis flower extract may be attributed to its high flavonoid content.

Collagen is a major protein of the extracellular matrix and is the component that ultimately contributes to wound strength.[19] the healing process depends, to a large extent, on the regulated biosynthesis and deposition of new collagens and their subsequent maturation.[20] assessment of collagen content in granulation tissues of control and experimental wounds clearly suggests that h. Rosa sinensis enhances collagen synthesis and deposition. Wound contraction, increased tensile strength activity support further evaluation of *h. Rosa sinensis* in the topical treatment and management of wounds.

Anti-Inflammatory Activity

Inflammation is a normal protective response to tissue injury caused by physical trauma, noxious chemical or microbial agents. It is the body's response to inactivate or destroy the invading organisms, to remove irritant and set the stage for tissue repair. It is triggered by the release of chemical mediators from specimen injured tissue and migrating cells[21]. Ethanol extracts of flower(22] and leaf(23] of hibiscus rosasinensis l. (red) has been reported to exhibit anti-inflammatory activity.

Corresponding Author

Aravind Sivakumar,

Senior Lecturer,Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India.

Email: aravind.sivakumar@gmail.com

Telephone Number: 8220552400

References

1. Hou dx, tong x, terahara n, luo d, fujii m. Delphinidin 3-sambubioside, a *hibiscus* anthocyanin, induces apoptosis in human leukemia cells through reactive oxygen species-mediated mitochondrial pathway. Arch biochembiophys. 2005;440:101–9.
2. Hirunpanich v, utaipat a, moralesnp, bunyapraphatsara n, sato h, herunsale a,Hypocholesterolemic and antioxidant effects of aqueous extracts from the dried calyx of *hibiscus sabdariffa* l. In hypercholesterolemic rats. J ethnopharmacol. 2006;103:252–60.
3. Chang yc, huangkx, huang ac, ho yc, wangcj. Hibiscus anthocyanins-rich extract inhibited ldl oxidation and oxldl-mediated macrophages apoptosis. Food chemtoxicol. 2006;44:1015–23.

4. Herrera-arellano a, flores-romero s, chávez-soto ma, tortoriello j. Effectiveness and tolerability of a standardized extract from *hibiscus sabdariffa* in patients with mild to moderate hypertension: a controlled and randomized clinical trial. Phytomedicine. 2004;11:375–82..
5. Puckhaberls, stipanovicrd, bostga. Analyses for flavonoid aglycones in fresh and preserved *hibiscus flowers*. In: janick j, whipkey a, editors. Trends in new crops and new uses. Alexandria, va: ashs press; 2002;1:556–63.
6. Nadkarni ak. In indian*materia medica* popular prakashan (pvt) ltd, bombay, india. 1976;1:1199.
7. Jonadet m, bastide j, bastide p, boyer b, carnatap. In vitro enzyme inhibitory and in vivo cardioprotective activities of hibiscus (hibiscus sabdariffa l) j pharm belg. 1990;45:120–4.
8. Yamasaki h, uefuji h, sakihama y. Stress proteins and myocardial protection . Arch biochembiophys. 1996;332:183–186. Doi: 10.1006/abbi.1996.0331.
9. Das dk, maulik n, moraru ii. Cell biology of trauma. Journal of molecular cell cardiology. 1995;27:181–193.
10. Yamasaki h, uefuji h, sakihama y. Stress proteins and myocardial protection . Arch biochembiophys. 1996;332:183–186. Doi: 10.1006/abbi.1996.0331.
11. Francis pt, palmer am, snape m, wilcockgk. The cholinergic hypothesis of alzheimer's disease: a review of progress. J neurolneurosurg. 1999;66:137–47.
12. Kang SY, Lee KY, Koo KA, Yoon JS, Lima SW, Kima YC. ESP-102, a standardized combined extract of Angelica gigas, Saururuschinensis and Schizandrachinensis, significantly improved scopolamine-induced memory impairment in mice. Life Sci. 2005;76:1691–05.
13. Wilson jx. Antioxidant defense of the brain: a role for astrocyte. Can j physiolpharmacol. 1997;75:1149–63.
14. El-sherbiny da, khalifaae, attia as, eldenshary eel-d. *Hypericumperforatum* extract demonstrates antioxidant properties against elevated rat brain oxidative status induced by amnesic dose of scopolamine. Pharmacol biochembehav. 2003;76:525–33.
15. Scalbert ac. Antimicrobial properties in tannins. Phytochem. 1991;30:3875–3883.
16. Ionela dc, ion ib. Plant products as antimicrobial agents. Secţiuneageneticăbiologiemolecula. 2007;8:104–111.
17. Clark ra. Cutaneous tissue repair. I basic biologic consideration. J amacaddermatol. 1985;13:701–25. [Tsuchiya h, sato m, miyazaki t, fujiwara s, tanigaki s, ohyama m, et al. Comparative study on the antibacterial activity of phytochemical flavanones against methicillin-resistant staphylococcus aureus. J ethnopharmacol. 1996;50:27–34.
18. Hassan sw, abubakar mg, umarra, yakubu as, maishanuhm, ayeni g. Pharmacological and toxicological properties of leaf extracts of kingeliaafricana (bignoniaceae) j pharmacoltoxicol. 2011;6:124–32.

19. Puratchildy a, nithya c, nagalakshmi g. Wound healing activity of cyperusrotunduslinn. Ind j pharmaceutical sci. 2006;68:97–101.
20. Bhitremj, fulmali s, kataria m, anwikar s, kadri h. Antiinflammatory activity of the fruits of piper longumlinn. Asian jchem 2008; 20(6): 4357- 4360.
21. Birarirb, jalapuress, changranisr, shidsl, tote mv, habade bm. Anti-inflammatory, anti-analgesic and anti-pyretic effect of hibiscus rosasinensislinn flower. Pharmacologyonline 2009; 3: 737-747.
22. Shimizu n, tomoda m, suzuki t, takada k. Plant mucilages. Xliii. A representative mucilage with biological activity from the leaves of hibiscus rosasinensis. Bio pharmceu bull 1993; 16: 735-739.

Huntington's Disease

Anitha.D.R [1], Ezhilarasan.D [2], Sri Sakthi.D [3]
Graduate Student [1], Senior Lecturer [2], Department of Pharmacology,
Reader [3],Department of Public Health Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Huntington's disease (HD) is a neurodegenerative genetic disorder that affects muscle coordination and leads to cognitive decline and behavioral symptoms. HD is the most common genetic cause of abnormal involuntary writhing movements called chorea,i.e the name of the disease is called Huntington's chorea. Tetrabenazine (Xenazine) is specifically approved by the Food and Drug Administration to suppress the involuntary jerking and writhing movements associated with Huntington’s disease.Currently available drugs like Citalopram, Fluoxetine, Sertraline, Tetrabenazine, Amantadine, levetiracetam (Keppra)are only symptomatic.In this review we are discussing about Amantadine ,Levetiracetam ,clonazepam and Tetrabenazine for the treatment of Huntington’sdisease.It is one of the common disease in old age people,so to study the drugs like Amantadine,Levetiracetam,Clonazepam,Tetrabenazine are effective in the treatment of Huntington's disease

Key Words: Huntington’s disease, neurodegenerative diseases, chorea, Parkinsonism

Introduction

Globally, millions of people each year are getting affected by various neurodegenerative diseases and the incidences are increasing further significantly with the extended life expectancy. World Health Organization (2006) reported that the number of older people of more than 65 years age is expected to increase to approximately 1 billion in 2030 worldwide [1]. Therefore, the percentage of aged population in developing countries would increase from 59% to 71% worldwide[2]. Thus, aging is implicated in onset and progression of several neurodegenerative diseases including Huntington's disease(HD),Alzheimer's disease(AD),Parkinson's disease(PD) are characterized by selective loss of anatomically or physiologically related neuronal systems[3].Neuropathologically,these are characterized by abnormalities of relatively specific regions of the brain and specific population of neurons [4].Among the above said neurodegerativediseases, HD is movement disorder is characterized by choreatic movements, dystonic symptoms and rigidity. Hence, this review focuses the Huntington's disease and its treatment.

Huntington's Disease

Huntington's disease (HD) is an autosomal dominant, neurodegenerative disorder characterized by by a triad of chorea, cognitive symptoms and behavioral changes, as well as various hyper-and a hypokinetic movement disorder, of which chorea is more prominent[5]. In patients with HD, chorea has a deleterious effect on coordinated limb movements [6,7]. Although there is no established treatment to delay the onset or forestall the progression of HD, symptomatic treatment of chorea may be beneficial in some individuals as it may have a

favourable impact on motor function, quality of life and safety[8].

Molecular Pathogenesis of HD

Gene mutation play pivotal role in the mechanism of the pathogenesis of HD. The dynamic gene mutation of CAG (cytosine-adenine-guanine) repeat in the Huntington (HTT) gene, which is transcribed into an abnormal protein with an elongated polyglutamine tract. Polyglutamine HTT accumulates and is changed in its function in multifaceted ways related to the numerous roles of the normal protein. This Polyglutamine HTT protein is reported to expressed in variousregions of the brain and in other organs[8]. The striatum of the brain is the major region involved in the disease process, it is also reported that other systems are involved as well. Thus, the overwhelming knowledge in the pathogenesis of HD led to the development of new therapeutic strategies based on specific molecular pathways for symptomatic and disease course-modifying treatment. It has been said that the most proximal way to handle the disturbed protein is to hinder the gene transcription, translation and proteins synthesis. Other mechanisms include modulation of energy and intracellular signaling, induction of factors potentially leading to neuroprotection, as well as modulation of glial function. Several clinical trial studies have shown based on these approaches andtherapy will be a based on the combination of these approaches harmonized with symptomatic treatments[8].

The transcription of DNA into mRNA is highly regulated process in the cell. The expression of protein coding genes is regulated in an orchestrated and elaborate manner, involving several transcription factors which interact with each other and with the regulatory DNA elements that specify the activity of the gene. Currently, it came to light that HTT has an important role in transcription; a range of methods have shown that both wild-type and mutant HTT interact with numerous transcription factors. Further, microarray studies haveconfirmed that various transcriptional pathways are impaired in HD.Other important transcription factors seem to interact with HTT in the nucleus. Among them, the Gln-Ala repeat transcriptional coactivator CA150 has been found to associate with both normal and mutant HTT[9]. Moreover, it has been demonstrated that CA150 protein levels increase concurrentlyas disease increases, which implies that CA150 accumulates in response to HTT aggregation and that this interferes with gene transcription in HD brain samples. Furthermore, an expansion in the polyQ region of TATA box-binding protein (TBP) is known

to induce spinocerebellar ataxia type 17 (SCA-17)diseases, which shows overlapping features with HD[10]. Interestingly, HTT might also function as a transcriptional repressor,it has been shown to interact directly with repressor complexes containing nuclear corepressor protein and Sin3A in a polyQ-dependent manner as demonstrated by yeast two-hybrid analysis[11]. Nuclear corepressor protein and Sin3A act with other transcriptional DNAbinding proteins to repress the transcriptional activation of nuclear receptors such as the thyroid and retinoic acid receptors. Accordingly, mutant HTT might interfere not only with nuclear co repressor protein and Sin3A function, but also with signaling pathways involving nuclear receptors.

Chaperones and the Proteasome

One feature of HD, along with other neurodegenerative diseases, is that inclusions have been found to be ubiquitinated and associated with several proteasome subunits, which strongly suggests a failure in the degradative machinery of the cell. If chaperones cannot refold abnormal proteins correctly, they then promote their subsequent ubiquitination, which ultimately directs them to the proteasome for degradation[12].Several lines of evidence have indicated that impairment of the ubiquitin-proteasome system (UPS) is central to the pathogenesis of polyQ disease*i.e.,* in HD. The eukaryotic proteasome is incapable of digesting peptides containing stretches of 9–29Q residues, and these peptides must be released for further hydrolysis by unidentifiedpeptidases[13]. Therefore, the attempted digestion of pathogenic polyQ tracts might result in aggregation-prone polyQ-containing fragments being released from the proteasome, or the failure of these tracts to exit might impair proteasome function. Consistent with this, transient transfection of HTT fragments containing a pathogenic polyQ repeat caused almost complete inhibition of the UPS in a cell-based UPS reporter system, which implies that protein aggregation directly impairs the function of the UPS [14].

Etiology

Huntington's disease is an autosomal dominantly inherited disease caused by an elongated CAG repeat on the short arm of chromosome in the Huntingtongene. This gene codes for the Huntingtonprotein on exon 1, it contains the CAG tract. The wild-type contains a CAG repeat, coding for a polyglutamine stretch in the protein at that site in the range from 6 to 26. Huntington's disease is associated with 36 repeats or more. Definite clinical manifestation

will occur if the number of repeats exceeds more than 40. The range 36-39 leads to an incomplete penetrance of the disease or to a very late onset. The range between 29 and 35, they called as intermediate alleles and it is unstable, which means that these alleles are prone to changes during reproduction. Copying the gene may lead to mistakes and very often leads to elongation and seldom to shortening. This phenomenon is mainly seen in the male line of reproduction[15].

An inverse correlation has been described between the length of the repeat and the age at onset, determined by the first motor manifestation. The longer the CAG repeat, the earlier the onset. When the disease starts before the age of 20 years, so-called juvenile Huntington's disease (JHD), the repeat often exceeds 55[16]. The length of the repeat determines about 70% of the variance in age at onset and gives no indication at all about the initial symptom, the course, or the duration of illness. The only correlation now described is the faster weight loss associated with a longer CAG repeat[17]. Anticipation phenomenon is seen in Huntington families in the paternal line of inheritance.

The normal wild-type Huntington protein plays a role in synaptic function, is necessary in the post-embryonic period, possibly has an anti-apoptotic function and is possibly protective against the toxic mutant, Huntington[4]. There is evidence that the mutant form leads to a gain of function as well as to a loss of function.

Clinical Description

The nuclear symptoms and signs of Huntington’s disease (HD) consist of motor, cognitive and psychiatric disturbances. Other less well-known, but prevalent and often debilitating features of HD include unintended weight loss, sleep and circadian rhythm disturbances and autonomic nervous system dysfunction[5]. The mean age at onset is between 30 and 50 years, with a range of 2 to 85 years. The mean duration of the disease is 17-20 years. The progression of the disease leads to more dependency in daily life and finally death. The most common cause of death is pneumonia, followed by suicide.

Signs and Symptoms

The characteristic motor changes are involuntary, unwanted movements. Initially, the movements often occur in the distal extremities such as fingers and toes, but also in small

facial muscles. For bystanders these muscle twitches are often invisible or can be explained as nervousness. In daily life, walking becomes unstable and the person can look as slightly drunk. Gradually the unwanted movements spread to all other muscles from distal to more proximal and axial. Choreatic movements are present all the time the patient be awake. No single pattern exists, but facial choreatic movements can lead to a continuous movement of facial muscles where for instance an eyebrow is lifted, an eye closed, the head is bent or turned while the tongue is protruded with the lips pouting. The most prominent are the extension movements of the long back muscles. Talking and swallowing gradually become more problematic leading to choking at any time in some patients. In later stages the patient even becomes mute. Dysarthria and dysphagia become very prominent during the course of the disease. All patients develop hypokinesia, akinesia, and rigidity leading to a slower pace of all activities (bradykinesia: slowness of movement) and a severe hesitation in embarking on a movement (akinesia: difficulty in starting movements)[11]. The balance between chorea and hypokinesia is determined individually. The extremes are on the one hand the younger patient with an overwhelming rigidity (Westphal variant) and on the other hand the very old patient severely affected in the last stage of the disease with a long duration of illness, bed-bound with rigidity and flexion contracture in the extremities. Dystonia is characterised by slower movements with an increased muscle tone leading to abnormal posture, for instance torticollis, but also rotation of the trunk or limbs. Dystonia (for instance torticollis) can be the first motor sign in Huntington's disease. Other unwanted movements include tics, comparable to the ones seen in Tourette syndrome, but these are fairly rare. Cerebellar signs can appear sporadically, similar to the presence of hypo- and hypermetria[7]. Walking is often described as 'drunk' or 'cerebellar ataxia'-like. Distinguishing between choreatic and ataxic walking is very difficult. Pyramidal signs (Babinski sign) are present incidentally.

Cognitive decline is the other main sign of HD and can be present long before the first motor symptoms appear, but can also be very mild in far advanced stages of the disease. The cognitive changes are particularly in relation to executive functions. In normal conditions, cognitive and motor behaviour is goal-directed and planned. Normally individuals are able to distinguish what is relevant and what can be ignored, but patients with HD lose this capability. The patients are no longer able to organise their life or to plan things which in the past were simple [8]. They lose flexibility of mind, and can no longer make mental adjustments. Misjudgements lead to complicated situations, with patients no longer reacting

as they did in the past or in a way that the environment expects. Language is relatively spared. Memory certainly becomes impaired, although the semantic memory can be spared to a certain extent [9]. All psychomotor processes become severely retarded.

If the first symptoms and signs start before the age of 20 years, the disease is called Juvenile Huntington's disease (JHD). Behaviour disturbances and learning difficulties at school are often the first signs. Motor behaviour is often hypokinetic and bradykinetic with dystonic components. Chorea is seldom seen in the first decade and only appears in the second decade. Epileptic fits are frequently seen. The CAG repeat length is over 55 in most cases. In 75% of the juveniles the father is the affected parent[8].

Secondary Signs and Symptoms

From early on, an unintended weight loss has been reported in all patients. As more attention is now paid to this phenomenon, the loss seems to be a little less severe, the cause being diverse. Although it seems logical to think that chorea should play the main role in weight loss, it has been shown that there is no relation between weight loss and chorea or other movement disorders[10]. More practical issues, such as slower functioning, decreased appetite, difficulty handling food and swallowing certainly play a role. But hypothalamic neuronal loss is also a causative factor.

Attention has only recently been focused on sleep- and circadian rhythm disturbances of patients with HD .Autonomic disturbances can result in attacks of profuse sweating.

Psychiatric symptoms are very frequently present in the early stage of the disease, often prior to the onset of motor symptoms. The percentage of patients with psychiatric signs varies between 33% and 76% depending on the methodology of the study[4]. Because of their impact on daily life, these symptoms and signs usually have a highly negative impact on functioning and on the family [5]. The most frequently occurring sign is depression. The diagnosis is difficult because weight loss, apathy and also occur in HD. Usually there is low self-esteem, feelings of guilt and anxiety. Apathy is related to disease stage, whereas anxiety and depression are not. Suicide occurs more commonly in early symptomatic individuals and also in pre manifest gene carriers. Around the time of the gene test and the stage when

independence diminishes are the most risky periods for suicide. Anxiety also occurs frequently (34-61%), sometimes in relation to uncertainty about the start and or the course of the disease. Obsessions and compulsions can disturb the patient's life and also lead to irritability and aggression. Irritability is often the very first sign, in retrospect, but in fact occurs during all stages of the disease [7]. The way irritability is expressed varies from serious disputes to physical aggression. A loss of interest and increasing passive behaviour are seen as part of the apathy syndrome. It can be difficult to discriminate apathy from depression. Psychosis may appear, mainly in the later stages of the disease. In most cases this goes together with cognitive decline. The complete clinical picture is comparable to schizophrenia with paranoid and acoustic hallucinations. In the early stages, hyper-sexuality can cause considerable problems in a relationship. In the later stages hypo-sexuality is the rule.

Diagnosis

The clinical assessment of the symptoms and signs of HD is important for patient, family and care-givers. To follow the patient systematically, mainly for research purposes, several scales have been developed. The best known are the Shoulson and Fahn capability scale and the Unified Huntington Disease Rating Scale (UHDRS). The UHDRS consists of a motor, behaviour, cognitive and functional part, preceded by a history and medication scheme. For the behaviour signs a new scale was developed by Craufurd, the Problem Behaviour Scale (PBS).The diagnosis is based on the clinical symptoms and signs in a person with a parent with proven HD. First, it is obligatory to take a precise history from the person with symptoms followed by a detailed family history[5,6,7]. When all information has been obtained the diagnosis is not very difficult, although non-specific clinical pictures can be misleading. Also when the parent is not known or has died due to another cause at a young age, the clinical picture can be difficult to recognise. It is often necessary to request old information in the form of medical records and autopsy reports. The current gold standard is DNA determination, showing a CAG-repeat of at least 36 on the *Huntington gene* on chromosome 4. Before 1993, a family history with clinical and morphological verification in at least one of the parents or grandparents was obligatory[14].The clinical criteria currently necessary are still motor changes with or without psychiatric or cognitive changes. However,

in most cases a combination of the three main signs is present. The combination with the family history is sufficient for diagnosis. No imaging, general blood tests or other diagnostic tools are helpful. Several studies are now focussing on changes in function and changes in brain imaging (MRI) before clinical overt manifestation is present.

Treatment of Huntington's Disease

Many agents and surgical procedures have been evaluated in HD for their anti-choreic efficacy including dopamine depleting agents, dopamine antagonists, benzodiazepines, glutamate antagonists, acetylcholinesterase inhibitors, dopamine agonists, anti-seizure medications, cannabinoids, lithium, deep brain stimulation, and fatal cell transplantation.Therapeutic drug for the HD is usually minimize its chorea and other movement disorders.The monoamine depletortetrabenazine (TBZ) has been shown to effectively reduce hypokinetic movement disorders especially chorea in HD[18]. TBZ carriesa less severe risk of developing tardive dyskinesia[19]. TBZ is well tolerated and serves as a good drug candidate to start medical treatment of choreatic movements even in early stages[20].Possible side effects of TBZ include depression, Parkinsonism, insomnia, akathisia, and sedation. Coming to the drug regimen, the dosage initially starts with 12.5 mg/day an6+d it can be increased as 12.5 mg three times per day. There is no marked maximum dose for this is reported in literature however it is usually limited to 100 mg/day. Its pharmacokinetics effectincludes strong cytochrome P450 2D6 inhibitors for instance, selective serotonin reuptake inhibitors such as paroxetine, fluoxetine, or other antidepressants such asbupropion or the individual turns out to be a CYP2D6 slow metabolizer by genotyping, the maximum dose should be restricted to half, although no distinguishing features have been found in patients exhibiting various CYP2D6 activities[21].Undoubtedly, in spite of itsbeneficial effects on chorea certain cognitive functions can worsen under medication with TBZ[22].

Neuroleptic drugs are also preferentially used for the HD If both chorea and psychiatric symptoms like agitation and psychosis are predominant features, these drugs are act by blocking the dopamine transmission[22].Neuroleptic drugs such as haloperidole, fluphenazineor chlorpromazine are commonly used in the HD patients if chorea is very severe or psychosis is accompanied by aggressiveness.Neuroleotics treatment is contraindicated in HD patients show Parkinsonian-like symptoms because medication with

neuroleptics can worsenParkinsonism. Several atypical neuroleptics have been chosen to treat chorea in HD patients. Clinically, Olanzapine, risperidoneand aripiprazole have been reported to improve chorea in HD patients.Sedation and obesityis a common side effectassociated with neuroleptics. Weight gain can even be of advantage for HD patients since unintended weight loss is in many cases a progressing burden in HD[23].

Benzodiazepines Chorea is known to worsen in psychologically demanding situations or under stress. Hence, low dosages of antidepressant *i.e.*,benzodiazepines can be added to the medication transitionally to cushion these emotional effects. The risk of drug abuse and dependency is the most drawbacks[22].

Amantadine, an antiviral and an antiparkinsonian drug also employedfor treatment of chorea is discussed controversially, since results of the present trials are not concordant[24,25]. Accordingly, this N-methyl-D-aspartate excitatory receptor antagonist could be an option in individual patients to improve chorea.

Riluzole is used for thetreatment of chorea in HD have diminished. Notable side effects were reported, but restricted to dose of 200 mg/day. However, Riluzole does not serve as first-line medication in the treatment of chorea in early symptomatic HD patients but it is recommended in third position for treating in HD[22].

During the course of the disease, the patient requires more care, which can also help his/her partner, for example by having a nurse at home to help with showering. The burden for the caregiver can become too heavy and so help must be found in day-care institutions, usually connected to nursing home facilities. In the period that follows the patient moves into a transition phase and eventually a 24-hour care situation. Throughout the whole process of increasing dependency, psychological help is often needed for the caregiver, who has to deal with increasing responsibilities while losing contact with his or her former partner. Partner groups can be very useful. In general, lay organisations play an enormous role in educating caregivers, patients and families.Medical and non-medical treatment must be individually tailored, as the symptoms and signs differ by person and over time tremendously. Ideally

treatment of patients and their families should be organised by a multidisciplinary team. Treatment is intended to improve quality of life. To date, no cure is available unfortunately.

Conclusion

In light of this review, it has been confirmed that the therapeutic treatment of HD include only to improve the symptoms and afford neuroprotection. Therapeutic options are still limited to symptomatic medication and supportive approaches. Current drug therapy has no effect on the progression of disability, and the need for any pharmacological treatment should be carefully considered. Hyperkinesias and psychiatric symptoms may respond well to pharmacotherapy, but neuropsychological deficits and dementia remain untreatable. Pharmacological intervention in the treatment of the movement disorder of HD is aimed at restoring the balance of neurotransmitters in the basal ganglia.A low level of evidence exists to support the use of physiotherapy for addressing impairments of balance, muscle strength, and flexibility. There was a small amount of evidence to support the use of speech pathology for the management of eating and swallowing disorders. The current evidence is insufficient to make strong recommendations regarding the usefulness of physiotherapy, occupational therapy, or speech pathology for people with Huntington's disease.

As discussed in this review, the molecular physiopathology of HD is very complex. Hence,the future therapy should combine number of drugs targeting different molecular targets in order to improve the chorea like symptoms and thereby afford neuroprotection. Since the single cause of HD is precisely known, and this disorder may be considered a paradigm for disease modifying treatment of other neurodegenerative disorders. Further studies are warranted in the arena of clinical, genetic and neuropathological factors that causes HD.

Corresponding author

Sri Sakthi. D

Reader,Department of Public Health Surgery

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University,Chennai,India

162, Poonamallee High Road,Chennai – 600077,Tamil Nadu, India.

Mobile Number: +91 – 9962787531

Email ID: drsrisakthiphd@gmail.com

Reference

1. National Institute of Health. Why Populations Aging Matters. A Global Perspective. 2013; 45(6): 4-5.
2. Guo CY, Sun L, Chen XP, Zhang DS. Oxidative stress, mitochondrial damage and neurodegenerative diseases. Neural Regen Res. 2013; 8(21): 206-210.
3. Martin LJ. Mitochondrial and Cell Death Mechanisms in Neurodegenerative Diseases. Pharmaceuticals (Basel). 2010; 3(4): 839-915.
4. Singh RP, Sharad S, Kapur S. Free Radicals and Oxidative Stress in Neurodegenerative Diseases: Relevance of Dietary Antioxidants. JIACM. 2004; 5(3): 218-25.
5. Ferrara JM, Mostile G, Hunter C, Adam OR, Jankovic J. Effect of Tetrabenazine on Motor Function in Patients with Huntington Disease. Neurol Ther. 2012, 1: 5. 4-5.
6. Fenny A, Jog MS, Duval C. Bradykinesia is not a "systematic" feature of adult-onset Huntington's disease; implications for basal ganglia pathophysiology. Brain Res. 2008; 1193: 67-75.
7. Reilmann R, Kristen F, Quinn L, Henningsen H, Marder K, Gordon AM. Objective assessment of progression in Huntington's disease: a 3- year follow up study. Neurology. 2001; 57: 920-924.
8. Burgunder JM. Translational research in Huntington's disease: opening up for disease modifying treatment. TranslNeurodegener. 2013; 2(1): 2.
9. Halbert S, Denghien I, Kiechle T, Rosenblatt A, Wellington C, Hayden MR, Margolis RL, Ross CA, Dausset J, Ferrante RJ, Néri C. The Gln-Ala repeat transcriptional activator CA150 interacts with Huntington: neuropathologic and genetic evidence for a role in Huntington's disease pathogenesis. Proc Natl Acad Sci U S A. 2001; 98(4): 1811-1816.
10. Stevanin G, Fujigasaki H, Lebre AS, Camuzat A, Jeannequin C, Dode C, Takahashi J, San C, Bellance R, Brice A, Durr A. Huntington's disease-like phenotype due to trinucleotide repeat expansions in the TBP and JPH3 genes. Brain. 2003; 126(7):1599-1603.
11. Boutell JM, Thomas P, Neal JW, Weston VJ, Duce J, Harper PS, Jones AL. Aberrant interactions of transcriptional repressor proteins with the Huntington's disease gene

product, Huntington. Hum Mol Genet. 1999; 8(9): 1647-1655.

12. Ciechanover A, Brundin P. The ubiquitin proteasome system in neurodegenerative diseases: sometimes the chicken, sometimes the egg. Neuron. 2003; 40(2): 427-446.
13. Venkatraman P, Wetzel R, Tanaka M, Nukina N, Goldberg AL. Eukaryotic proteasome cannot digest polyglutamine sequences and release them during degradation of polyglutamine-containing proteins. Mol Cell. 2004; 14(1): 95-104.
14. Bence NF, Sampat RM, Kopito RR. Impairment of the ubiquitin-proteasome system by protein aggregation. Science. 2001; 292(5521): 1552-1555.
15. Trottier Y, Biancalana V, Mandel JL: Instability of CAG repeats in Huntington's disease: relation to parental transmission and age of onset. J Med Genet. 1994, 31: 377-82.
16. Wheelock VL, Tempkin T, Marder K, Nance M, Myers RH, Zhao H, Kayson E, Orme C, Shoulson I, Huntington Study Group: Predictors of nursing home placement in Huntington disease. Neurology. 2003, 60: 998-1001.
17. Bonelli RM, Hofmann P: A systematic review of the treatment studies in Huntington's disease since 1990. Expert Opin Pharmacother. 2007, 8: 141-53.
18. Armstrong MJ, Miyasaki JM; American Academy of Neurology. Evidence-based guideline: pharmacologic treatment of chorea in Huntington disease: report of the guideline development subcommittee of the American Academy of Neurology. Neurology. 2012; 79(6): 597-603.
19. Jankovic J. Treatment of hyperkinetic movement disorders. Lancet Neurol. 2009 Sep;8(9):844-56.
20. Chen JJ, Ondo WG, Dashtipour K, Swope DM. Tetrabenazine for the treatment of hyperkinetic movement disorders: a review of the literature. Clin Ther. 2012; 34(7): 1487-1504.
21. Mehanna R, Hunter C, Davidson A, Jimenez-Shahed J, Jankovic J. Analysis of CYP2D6 genotype and response to tetrabenazine. Mov Disord. 2013; 28(2): 210-215.
22. Schiefer J, Werner CJ, Reetz K. Clinical diagnosis and management in early Huntington's disease. DegenerNeurolNeuromuscular Dis. 2015; 5:37-50.
23. Brusa L, Orlacchio A, Moschella V, Iani C, Bernardi G, Mercuri NB. Treatment of the symptoms of Huntington’s disease: preliminary results comparing aripiprazole and tetrabenazine. Movdisord. 2009; 24:126–129.
24. O'Suilleabhain P, Dewey RB Jr. A randomized trial of amantadine in Huntington

disease. Arch Neurol. 2003; 60(7):996-998.

25. Heckmann JM, Legg P, Sklar D, Fine J, Brier A, Kies B. IV amantadine improves chorea in Huntington’s disease: an acute randomized, controlled study. Neurology. 2004; 63:597–598.

Hyperacusis-Review Article

Rebecca Rachel George[1], Saravana Kumar[2], Ravindra Kumar Jain[3]

Graduate student[1,] Asst Professor[2], Department of Anatomy,

Reader[3], Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Disorders related to perceiving loud sounds can represent a serious challenge to patients in a dental setting. In this paper a review of what is known of hyperacusis—the prevalence, causes, symptoms and approaches to treatment. Beginning with definitions, because even basic terminology still varies in this under-researched area. Hyperacusis (also spelled hyperacousis) is a health condition characterized by over-sensitivity to certain frequency and volume ranges of sound (a collapsed tolerance to usual environmental sound). Difficulties in tolerance towards listening to everyday sounds is observed in severe hyperacusis, some of which may seem unpleasantly or painfully loud to that person but not to others [1][2].There is no objective test for hyperacusis. Most people who develop hyperacusis will be referred to an ear doctor and receive a thorough evaluation including a full audiological assessment to determine the state of the auditory-vestibular system. For patients, hypersensitivity of hearing may evoke anxiety and even fear. This can be true for specific sounds or for sound in general. The links between the central auditory system and areas of the brain implicated in anxiety and fear are now under close scrutiny.Hyperacusis can come on suddenly or gradually. It can in the early stages affect the ear on one side but generally speaking, within a short time, the condition is almost always bilateral. It can be mild or severe. Often, people who have hyperacusis also have tinnitus or phantom noises in their auditory system (ringing, buzzing, chirping, humming, or beating). Research has shown that about half of all people who have tinnitus, also have reduced tolerance for moderate or loud sounds, known as hyperacusis.

Key Words :*hyperacusis, over-sensitivity, auditory, tinnitus*

Introduction

Hyperacusis is described as an‘unusual tolerance to ordinary environmental sounds’ [3] and, more pejoratively, as ‘consistently exaggerated or inappropriate responses to sounds that are neither threatening nor uncomfortably loud to a typical person’ [4]. Common to both is the implication that the experience can be evoked by sounds of low intensity and that sounds in general, rather than specific sounds, are problematic. This is less true of phonophobia (fear of sound) and the recently proposed misophonia (dislike of sound) [5], both of which carry a suggestion that the intolerance may be specific to certain sounds with emotional associations. In terms of neurology, phonophobia tends to be utilized for the loudness intolerance reported by some patients with migraine. For the wider types of hearing hypersensitivity, therefore, the term hyperacusis is preferable. Loudness recruitment[6][7] describes an experience commonly associated with cochlear hearing loss and specifically with dysfunction of the outer hair cells of the organ of Corti: with a rising sound level, the perceived loudness becomes increased faster than normal [8].This phenomenon may be distinguished from hyperacusis if the individual perceives sound of moderate intensity as uncommonly loud (recruitment) or sound of reduced intensity as loud in an uncomfortable manner (hyperacusis) but both the experiences are not mutually exclusive.

Decreased amount of significant epidemiological data is a major shortcoming of the published work on hyperacusis. Fabijanska et al. [9] undertook a postal questionnaire of tinnitus in Poland which included an unspecified question on hyperacusis. Of the 10349 respondents, 15.2% reported hyperacusis (12.5% of males, 17.6% of females). Regional differences were also reported. A weakness of this report is the lack of specificity. More recently Andersson and co-workers [10]investigated the prevalence of hyperacusis in the adult Swedish population. Two methods were used—an internet study, wherein visitors to the website of a Swedish broadsheet newspaper were invited to complete a web-based questionnaire; and a postal population study [11]. Ofa total of 1167 individuals, 595 responded upon the web banner, response rate of 52%. The point prevalence of hyperacusis in this group was 9%. The postal group comprised 987 individuals of whom 589 responded (response rate 60%) and the point prevalence was 8% [12]. Participants were not asked if they had ever sought a medical opinion regarding their hyperacusis. Incidence data for hyperacusis do not seem to have been reported anywhere [13].

A coincidence of tinnitus complaint and of experiences of hyperacusis has been widely note. In patients going to tinnitus clinics with a primary complaint of tinnitus, the prevalence of hyperacusis is about 40%; 9–11 and in patients with a primary complaint of hyperacusis the prevalence of tinnitus has been reported as 86% [14]. The apparent link has led to speculation about common mechanisms [15]. Until recently it has not been possible to quantify the handicap associated with hyperacusis, but two instruments have now been published for this purpose. Khalfa et al. [16] describe data from a self-report hyperacusis questionnaire with 14 items 'normalized' on 201 individuals who had answered a recruitment advertisement. Principal component analysis indicated that three factors accounted for 48% of the variance—attentional, social and emotional. With a 27-item questionnaire examined in 226 patients with hyperacusis Nelting et al. [17] reached similar conclusions: 51% of the variance was accounted for by cognitive reactions, actional/somatic behaviour and emotional factors.

Causes

In the great majority of cases, no underlying medical condition can be found. The conditions in which hyperacusis has been reported as a symptom have been reviewed by Katzenell and Segal[18].Since the facial nerve innervates the stapedial reflex, which is a mechanism for reducing the perceived intensity of impulse sound, these conditions may reduce the efficacy of that reflex and hence increase the perceived intensity of sound.

Williams syndrome is a disorder characterized by deficits in conceptual reasoning, problem solving, motor control, arithmetic ability and spatial cognition, with an incidence of 1 in 20 000 live births. As many as 90% of individuals with this syndrome report hyperacusis [19]. The common causes of hyperacusis are found to be hearing loss, noise injury, head injury or whiplash type injury,acoustic trauma, i.e. airbag explosion or gunshot, adverse reaction to medication or surgeries, particularly central nervous system drugs, chronic ear infections,auto immune disorders. The most common causes of hyperacusis have been found to be noise injuries and head injuries by far. Neck injuries can also contribute, i.e., whiplash. There remain huge areas of medical scientific research and data accumulation that must be explored before we can really begin to define and understand the different types of hyperacusis, and the origins of this troubling disorder.The frequently encountered cause of hyperacusis is overexposure to excessively high decibel levels (or sound pressure levels) [20].

Mechanisms

Sahley and Nodar considered the observation that hyperacusis (and tinnitus) appear to increase in extent at times of tiredness, anxiety or stress. They hypothesize that, during stress, endogenous dynorphins are released into the synaptic region beneath inner hair cells. This might potentiate the neurotransmitter glutamate, causing sound to be perceived with excessive loudness. The model applies both to externally generated and to internally generated (tinnitus) sound, but empirical evidence in support has not yet been forthcoming [21].

The high prevalence of hyperacusis in Williams syndrome led Marriage and Barnes to consider the mechanism in that condition and the extent to which it might be generalized to other individuals. Their suggestion that 5-HT might be implicated was based partly on the clinical observation that hyperacusis tends to occur in other conditions where 5-HT unction is thought to be disturbed—namely, migraine, depression and post-traumatic stress disorder. 5-HT does appear to have a role in modulating auditory gain and the determination of significance of sound. However, there is no evidence that 5-HT disturbance contributes to hyperacusis of non-syndromic types. Moreover, even in Williams syndrome the excessive auditory gain may be explained partly by the high incidence of otitis media with effusion and the associated conductive hearing loss [22].

Symptoms

In cochlear hyperacusis (the most common form of hyperacusis), the symptoms are ear pain, annoyance, and general intolerance to any sounds that most people don't notice or consider unpleasant. Cochlear hyperacusis may cause crying spells or panic attacks. As many as 86% of hyperacusis sufferers also have tinnitus [2][23] .In vestibular hyperacusis, the sufferer may experience feelings of dizziness, nausea, or a loss of balance when sounds of certain pitches are present. The extent to which a hyperacusis patient is affected relies not only on the overall severity of that person's symptoms but also on whether the person can detect sounds in that frequency range at the volume in question, as well as on the person's preexisting tone and severity of startle response. Anxiety, stress, and/or phonophobia can be found in both types of hyperacusis. Individuals suffering from either form of hyperacusis may develop avoidant behavior in order to try to avoid a stressful sound situation or to avoid embarrassing themselves

in a social situation that might involve noise. Regular everyday sounds such as closing doors, ringing phones, running water, ticking clocks, chewing gum, cooking, normal conversation, eating, dishes, and other sounds will hurt his/her ears.

Hyperacusis in children

Reduced tolerance to sounds seems particularly common in children who have had a temporary hearing loss due to wax or otitis media with effusion that has then been corrected. It is also noted in children with learning and/or social and communication difficulties including those on the autistic spectrum or with Williams Syndrome. Common troublesome sounds include sounds of vacuum cleaners, washing machines or emergency vehicles. The child's reaction ranges from covering ears to crying and screaming. For children with mild distress, management may involve an explanation of the symptom and advice on coping strategies. For those children who are markedly distressed, the management may involve auditory desensitization, behavioral desensitization or both. A multi-agency or multi-disciplinary approach is recommended [24].

Assessment

Tyler et al. (University of Iowa, USA) suggested the following classification of hyperacusis:

- "Loudness hyperacusis" - some moderately loud sounds are very loud;
- "Annoyance hyperacusis" - some sounds are annoying (not always loud);
- "Fear hyperacusis" - patients are afraid of some sounds (not always loud).

Questionnaires to assess the severity and disability associated with each of the above categorieswere used in a study of over 200 people with hyperacusis. This showed that louder sounds are generally more annoying and softer sounds are less likely to evoke fear hyperacusis. The most common etiological factor is noise exposure. In 10% the symptom is unilateral. About 90% report concurrent tinnitus. Music, screaming and sirens are the most common sounds associated with fear hyperacusis. Loudness and annoyance hyperacusis are more closely linked to one another than they are to fear hyperacusis. Some patients with severe hyperacusis are also bothered by strong smells, tastes and bright lights and report headaches and balance problems. It

was suggested that use of this classification facilitates the treatment of hyperacusis patients using a protocol called tinnitus activities treatment [25].

Jastreboff and Jastreboff (Emory University, USA) proposed a different taxonomy for decreased sound tolerance (DST); defining DST as the presence of negative reactions experienced by a subject as a result of exposure to sounds that would not evoke such aversive reactions in the average listener. They suggested that DST can be subdivided into hyperacusis, misophonia, or a combination of these conditions.In this classification, hyperacusis is defined as a negative reaction to a sound dependent only on its physical characteristics, namely its spectrum and intensity. The sound's meaning and the context in which it occurs are irrelevant. Misophonia is characterized as negative reactions to a sound with a specific pattern and meaning to an individual patient. In misophonia the physical characteristics of the sound are secondary and the reactions to the sound are thought to depend on non-auditory factors such as the patient's previous evaluation of that sound and the context in which the sound is presented. Misophonia can include a variety of negative emotions such as dislike, annoyance, hate, discomfort, fear. In this classification a further type of DST, phonophobia, is defined as a subset of misophonia in which fear is the dominant factor. A hypothesis for the mechanisms of hyperacusis and misophonia was proposed together with a treatment paradigm based on tinnitus retraining therapy (TRT) [26].

How Hyperacusis Affects People

People with hyperacusis can have very different levels of distress, and our clinical experience is that they often are more handicapped than those with tinnitus. Hyperacusis can influence emotional well-being, hearing, sleep, and concentration. This is the basis for Tinnitus Activities Treatment [27]which is also applied to hyperacusis patients.Jüris, Andersson, Larsen, and Elselius (2013) studied a group of 62 patients with hyperacusis and reported that about 47% fitted the diagnosis of having an anxiety disorder. Some patients complain that their hyperacusis interferes with speech perception, particularly in noise. How hyperacusis affects the coding of speech and separating speech from noise at these higher levels is unknown. Because many people with hyperacusis also have hearing loss, it is difficult to isolate peripheral and central effects. Furthermore, we should not forget that many with hyperacusis appear to have normal hearing thresholds. Some people with hyperacusis report that they are awakened from sleep by

sounds or that they do not sleep well because of the anticipation of a loud or annoying sound. Some report that they have difficulty concentrating in anticipation of a loud or annoying sound. A particular problem for those with hyperacusis, and one that is not always appreciated, is that they often have to move around in different areas with varying noise levels throughout the day. Some areas might be quiet, some might have moderate noise levels, and some might have high enough levels that noise protection is warranted. This might naturally lead to fear hyperacusis. It would be of interest to apply the World Health Organization categorization of Functional Impairments as has been done for tinnitus (Tyler, 1993; Tyler et al., 2009). For example, the primary functions impaired could be emotions, hearing, sleep, and concentration. This would limit activities of socialization, work, and education and would have an economic impact. Many with severe hyperacusis, notably fear hyperacusis, experience dire emotional problems (notably anxiety and depression). Juris et al. (2013) reported that 56% of patients (35 of 62) with hyperacusis met criteria for a psychiatric disorder. Most had a social phobia and/or a generalized anxiety disorder. A common perspective among clinical otologists and audiologists is that hyperacusis is primarily a psychological disorder. One can imagine at least two scenarios. Auditory system abnormalities could lead to hyperacusis. This could lead to anxiety and depression. Another scenario is that brain abnormalities could lead to mental illness and dysfunction (and, therefore, a psychiatric disorder) and that hyperacusis is one symptom resulting from this. This distinction will likely be important in understanding mechanisms. Counseling and sound therapy management addressing both the hyperacusis and the psychological issues resulting from hyperacusis could (at present) be applied regardless of the underlying mechanisms.

Therapy

For many patients, the first reaction to hyperacusis is to protect themselves with ear plugs, muffs or other devices. There is, however, reason to believe that such strategies to decrease the intensity of sound entering the auditory system may further increase the central gain, exacerbating rather than improving the hyperacusis. In the past, patients had little choice but to resort to hearing protection devices since hyperacusis was not widely regarded as a genuine symptom. For tinnitus, tinnitus retraining therapy (TRT) was introduced in 1993,and with minor modifications this has been advocated also for hyperacusis. After evaluation in audiological and

medical aspects, the protocol11 requires classification of the patient according to the tinnitus and hyperacusis state, and then ‘directive counseling’ about the auditory system, about mechanisms of tinnitus and hyperacusis and about the distress associated with them. Binaural sound therapy, from ear-level wide-band generators, is under- taken even when the symptoms are unilateral. Treatment is based on the notion of desensitization, and the sound intensity is increased from a low level gradually over time. No randomized controlled trials have been done on retraining therapy for hyperacusis; they would be hard to design in view of the twin elements of counseling and sound therapy.

Several observational studies [28]have pointed to improvements in loudness tolerance, but the nature of training to do TRT (attendance at an examined course run by the originators) raises concerns about objectivity. For the psychological distress associated with tinnitus, cognitive–behavioral therapy (CBT) has been identified as the treatment of choice [29], and this seems a reasonable strategy to counter the anxiety and stress associated with hyperacusis, together with information counseling, relaxation therapy and sound therapy. No evidence as to the efficacy of such an approach is yet available, and at present CBT therapists in the UK shows little interest in tinnitus or hyperacusis. There is at present some tension between advocates of retraining therapy and advocates of psychological therapy, but the differences between the two are not great. Patients would probably benefit if the insights from both could be brought to bear[30][31].

Conclusion

Hearing is an ability that can be affected or distracted by various means [32][33]. Most jobs involve some level of noise. In some cases, the patient may need to seek other employment or attempt to secure disability with the help of an understanding doctor. Loud noise exposure generally makes the condition worse and exacerbates the accompanying tinnitus. Patients report they perceive sound - even their own voice - as uncomfortably loud and this not only causes tinnitus to increase but may also cause inner ear discomfort or a popping reflex in the ear. In fact some patients actually try to change the pitch of their own voice to accomodate their ears. This may help their ears but a patient can become hoarse in the process.

Corresponding Author

Ravindra Kumar Jain

Reader,Department of Orthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Telephone Number: +91 9884729660

E-mail id: orthoravi2@gmail.com

References

1. Baguley DM (December 2003)."Hyperacusis". J R Soc Med 96 (12): 582–5.
2. Andersson G, Jüris L, Kaldo V, Baguley DM, Larsen HC, Ekselius L (2005). "[Hyperacusis--an unexplored field. Cognitive behavior therapy can relieve problems in auditory intolerance, a condition with many questions]".Lakartidningen (in Swedish) 102 (44): 3210.PMID .
3. Vernon JA. Pathophysiology of tinnitus: a special case—hyperacusis and a proposed treatment. Am J Otol 1987;8:201
4. Klein AJ, Armstrong BL, Greer MK, Brown FR. Hyperacusis and otitis media in individuals with Williams syndrome. J Speech Hear Disord 1990;55:339–4
5. Jastreboff PJ. Tinnitus retraining treatment for patients with tinnitus and decreased sound tolerance. Otolaryngol Clin N Am 2003;36:2
6. Silberstein SD, Saper JR, Freitag FG. Migraine: diagnosis and treatment. In: Silberstein SD, Lipton RB, Dalessio DJ, eds. Wolff's Headache and other Head Pain, 7th edn. Oxford: Oxford University Press, 2001:121–237
7. Fowler EP. A method for the early detection of otosclerosis. Arch Otolaryngol 1936;24:731–41
8. Moore ECJ. Cochlear Hearing Loss. London: Whurr, 1998
9. Fabijanska A, Rogowski M, Bartnik G, Skarzynski H. Epidemiology of tinnitus and hyperacusis in Poland. In: Hazell J W P, ed. Proceedings of the Sixth International Tinnitus Seminar. London: The Tinnitus and Hyperacusis Centre, 1999:569–71

10. Andersson G, Lindvall N, Hursti T, Carlbring P. Hypersensitivity to sound (hyperacusis): a prevalence study conducted via the Internet and post. Int J Audiol 2002; 41:545–54
11. Sood SK, Coles RRA. Hyperacusis and phonophobia in tinnitus patients. Br J Audiol 1998;22:228
12. Bartnik G, Fabijanska A, Rogowski M. Our experience in treatment of patients with tinnitus and/or hyperacusis using the habituation method. In: Hazell J W P, ed. Proceedings of the Sixth International Tinnitus Seminar. London: The Tinnitus and Hyperacusis Centre, 1999:416–17
13. Jastreboff PJ, Jastreboff MM. Tinnitus retraining therapy (TRT) as a method for treatment of tinnitus and hyperacusis patients. J Am Acad Audiol 2000;11:162–77
14. Anari M, Axelsson A, Eliasson A, Magnusson L. Hypersensitivity to sound. Questionnaire data, audiometry and classification. Scand Audiol 1999;28:219–30
15. Andersson G, Vretblad P, Larsen HC, Lyttkens L. Longitudinal follow-up of tinnitus complaints. Arch Otolaryngol Head Neck Surg 2001;127:175–9
16. Khalfa S, Dubal S, Veuillet E, Perez-Seliaz F, Jouvent R, Collet L. Psychometric normalisation of a hyperacusis questionnaire. Otorhinolaryngology 2002;64:438–42
17. Nelting M, Rienhoff NK, Hesse G, Lamparter U. The assessment of subjective distress related to hyperacusis with a self-rating questionnaire on hypersensitivity to sound. Laryngorhinootologie 2002;81:32–4
18. Katzenell U, Segal S. Hyperacusis: review and clinical guidelines. OtolNeurotol 2001;22:321–6
19. Levitin DJ, Menon V, Schmitt JE, et al. Neural correlates of auditory perception in Williams syndrome: an FMRI study. Neuroimage 2003;18:74–82
20. Størmer CC, Stenklev NC (March 2007). "[Rock music and hearing disorders]". Tidsskr. Nor. Laegeforen. (in Norwegian) 127 (7): 874–7. PMID 17435808
21. Sahley TL, Nodar RH. A biochemical model of peripheral tinnitus. Hearing Res 2001;182:43–54
22. Marriage J, Barnes NM. Is central hyperacusis a symptom of 5 hydroxytryptamine (5-HT) dysfunction? J LaryngolOtol 1995;109: 584 915–21)
23. Nelson JJ, Chen K (July 2004). "The relationship of tinnitus, hyperacusis, and hearing loss". Ear Nose Throat J83 (7): 472–6. PMID 15372918
24. Coelho CB, Sanchez TG, Tyler RS. Hyperacusis, sound annoyance, and loudness hypersensitivity in children. Prog Brain Res 2007;166:169-78.

25. Tyler RS, Noble W, Coelho C, Haskell G, Bardia A. Tinnitus and hyperacusis. In: Katz J, Burkard R, Medwetsky L, Hood L, editors. Handbook of Clinical Audiology. 6 thed. Baltimore: Lippincott Williams and Wilkins; 2009
26. Jastreboff MM. Using TRT to treat hyperacusis, misophonia and phonophobia. ENT Audiol News 2013;21:88-90.
27. Tyler, R. S., & Baker, L. J. (1983). Difficulties experienced by tinnitus sufferers. Journal of Speech and Hearing Research, 48, 150–154
28. Kandel ER,KupfermannI,Iversen S. Learning and memory. In: Kandel ER,Schwartz JH, Jessell TM eds. Principles of Neural Science. New York: McGraw-Hill,2000:1227–46.
29. Jastreboff PJ, Hazell JWP. A neurophysiological approach to tinnitus: clinical implications. Br J Audiol 1993;27:7–17
30. Gold S, Formby C, Frederick EA, Suter C. Shifts in loudness discomfort level in tinnitus patients with and without hyperacusis. In: Patuzzi R, ed. Proceedings of the Seventh International Tinnitus Seminar 2002. Perth: University of Western Australia, 2002: 170–2
31. Hazell JWP, Sheldrake JB, Graham RL. Decreased sound tolerance: predisposing factors, triggers and outcomes after TRT. In: Patuzzi R ed. Proceedings of the Seventh International Tinnitus Seminar 2002. Perth University of Western Australia, 2002:255–61.
32. Arshiya S., Dhanraj S. The effects of brain games and music on short term memory. Research Journal of Pharmaceutical, Biological and Chemical Sciences. March-April 2017:200-04.
33. Hemashree J., Preetha S. Audiometry in type 2 diabetic and non-diabetic patients of the age group 45 to 65 years: a comparative study.Asian J Pharm Clin Res, Vol 9, Suppl. 1, 2016, 177-178.

Implant Surface Texturing- A Literature Review

S. Mohamed Jaqfer Sadiq[1], Dhanraj M[2], Aarthi Vignesh[3],

Graduate Student[1],Professor[2], Department of Prosthodontics,Senior Lecturer[3], Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Aim of the dental treatment is to replace the damaged or lost part of tooth structure with materials that restores functions and aesthetics of that tooth. Over the past few years various materials have been introduced for these purposes, amongst those categories implants are more popular in the past few decades. An implant is defined as a "biomaterial which is inserted either partially or completely, into the body for therapeutic, diagnostic, or prosthetic purposes". The attachment of cells to titanium surfaces is an important phenomenon in the area of clinical implant dentistry. The key factor in implant designs is to create surfaces that promote desirable responses in the cells and tissues. Biological fixation between the dental implant surfaces and jaw bones determine the longevity of dental implants. Implant morphology influence bone metabolism, rougher surface stimulates differentiation, attachment and growth of bone cells, and increases bone mineralization,furthermore, the degree of roughness is important. Textured surface also allows ingrowth of the tissues. Various techniques of surface modifications have been studied and applied to improve biological surface properties, which favours osseointegration. The major methods that are studied in the literature review to create implant roughness are acid etching, grit-blasting, titanium plasma spraying, Calcium phosphate coating, electrochemical anadizing and laser treatment.

***Key Words**: Titanium Plasma Spraying, Sand Blasting, Acid Etching, Laser Treatment, Grit Blasting.*

Introduction

Aim of the dental treatment is to replace the damaged or lost part of tooth structure with materials that restores function and esthetics of that tooth. Over the past few years various materials have been introduced for these purposes, amongst those categories implants are more popular in the past few decades. An implant is defined as a “biomaterial which is inserted either partially or completely, into the body for therapeutic, diagnostic, or prosthetic purposes”.[1,2] The attachment of cells to titanium surfaces is an important phenomenon in the area of clinical implant dentistry. The key factor in implant designs is to create surfaces that promote desirable responses in the cells and tissues. Biological fixation between the dental implant surfaces and jaw bones determine the longivity of dental implants.[3] Implant morphology influences bone metabolism: rougher surfaces stimulates differentiation, growth and attachment of bone cells, and increases mineralization; furthermore, the degree of roughness is important. Textured surface also allows ingrowth of the tissues [4,5]. Various techniques of surface modifications have been studied and applied to improve biological surface properties, which favours osseointegration [6,7]. The major methods that are studied in the literature review to create implant roughness are acid etching, grit-blasting, titanium plasma spraying,Calcium phosphate coating, electrochemical anadizing and laser treatment. Dental implant surface technologies have evolved rapidly in the recent times to enhance the bone formation on their surface. Following the placement of the implant, there is a predictable sequence of bone turnover and replacement at the interface that allows the newly formed bone to adapt to microscopic roughness on the implant surface, and even nanotopography has been shown to preferably influence the formation of bone. Chemical composition of the implant surface can differ markedly from bulk composition due to manufacturing, finishing, thermal treatment, blasting, etching, coatings, and even sterilization procedures. Based on these considerations, a careful control of implant surface composition becomes a relevant procedure to produce high-quality devices. Dental implants have become a standard procedure for single tooth replacement in the esthetic zone, providing many advantages but also challenges in sophisticated patients.

Titanium Plasma Spraying

This method consists in injecting titanium powders into a plasma torch at high temperature. These titanium particles projected onto the surface of the implants condense and fuse together

resulting in titanium plasma sprayed (TPS) coating with an average roughness of around 7 μm. [8]This procedure helps in increasing the surface area of the implants. TPS are recommended in regions with low bone density as it increases the surface roughness profile and consequently the surface area. [9] It is considered that the increase in surface area represents an effective increase in osseointegration area providing spaces greater than 50 μm that facilitates the migration of pathogens when the implant surface is exposed to the oral fluids.[10] Advantages of TPS are Reported to increase the surface area of bone implant interface and act similarly to 3D surface, which may stimulate adhesion osteogenesis, Surface area to increase by 600%, Increases tensile strength of bone implant interface and Improves primary stability. [11] modified surfaces can also present osteoconductivity which allows for cell migration to the implant surface promoting the formation of extracellular matrix and bone apposition.[12] It proposed to modify the implant surface without leaving the residues found after the sandblasting procedures. It helps to avoid the nonuniform treatment of the surface and to control the loss of metallic substance from the body of the implant. Baths of hydrochloric acid (HCl), sulfuric acid (H2 SO4), hydrofluoric acid (HF), and HNO3 are used in different combinations. Roughness before etching, the acid mixture, the bath temperature, and the etching time all affect the acid-etching process. The OsseoSpeedimplant was introduced to the market in 2004. The specific surface texture is a result of two subtractive, sequential manufacturing steps. Titanium oxide blasting produces the microscale surface roughness. The subsequent etching with hydrofluoric acid shapes the nanostructure of the implant. A pleiotropic manufacturing effect is the accumulation of fluoride on the surface. Fluoride containing surfaces have been hypothesized to propagate the host-to-implant reaction in early osseointegration. Cell studies have demonstrated that the OsseoSpeed surface promotes a branched cell morphology of osteoblasts and an osteogenic gene expression profile as well as osteoinduction and osteogenesis in mesenchymal stem cells compared to TiOblast implants (DENTSPLY Implants, Mannheim, Germany), the titanium oxide blasted precursor.

Grit Blasting or Abrasive Blasting

Grit blasting, also known as abrasive blasting, is another technique which is used to create surface topographies on the implant surfaces. Blasting is a diffuse mechanical treatments done to obtain rough implant surfaces.[13] grit-blasting procedure is performed by propulsion of particles of different sizes at high velocity such as of silica (sand), alumina, titanium oxide or CaP. [14] This technique is generally employed for descaling and surface roughening of commercial implants there by increasing the surface area of the implant for better osseointegration. [15] Shot peening is a modified method of grit blasting and is used primarily for introducing compressive stresses in the material's surface. It is most commonly used for producing specific surface topographies on various biomaterials surfaces. Surface topography depends on size of particles used blasting.[16] Alumina particles in the size range of 25-75 µm result in mean surface roughness in the range 0.5-1.5 µm, [17,18] where as roughness in the range of 2-6 µm are reported for surfaces blasted with particles of size between 200-600 µm. Use of fine particle size glass particles of 150-230 ìm results in relatively smooth surface.[19] Advantages of blasting includes a secure 3-D interlocking interface with bone, minimal crestal bone remodeling, Short healing timeProvide space, volume for cell migration and attachment and thus support contact osteogenesis.[20] Abrasive blasting can be used to remove - or clean - excess or unwanted materials from a surface. The advantage of the procedure is that it keeps the surface intact, while everything else is removed. There are two main types of blasting: dry and wet. The type of abrasive used in a particular application is usually specific to the blasting method. Dry blasting is usually done with the following: Sand Metallic grit Shot Aluminum oxide (alumina) Silicon carbide Wet blasters are operated with: Sand Glass beads Other materials that remain suspended in water. The exact equipment used depends, to a large extent, on the specific application and type of abrasive. Industries that use abrasive blasting include: Shipbuilding, Automotive, Metal finishing, Foundry, Surface coating. Other industries that involve surface preparation and painting. Common applications are engravings for monuments and buildings and cleaning boat hulls, etc.

Acid Etching

Metallic implant is immersed into an acidic solution, which erodes its surface, creating pits of specific diameter and shape. [21]Most commonly used acid-etching agents are hydrofluoric, nitric, sulfuric acids and combinations. Etching of the implant surface produces irregular

micropores with approximately 3-5 μm in diameter and 2-3 μm in depth. [14] Such a etched surface is considered to be in topographical aspects, while surface treatment methods alter both the topography and chemistry of an implant surface [6]. An acid etched surface was reported to show four times greater resistance to removal torque, the measurement of which has been one of the tools evaluating the quality of binding at the interface between the bone and the implant surface[22]. It includes Higher adhesion and expression of platelet and extracellular genes even which helps in colonization of osteoblasts at the site and promote osseointegration. Acid-etching can lead to hydrogen embrittlement of the titanium, creating micro cracks on its surface that could reduce the fatigue resistance of the implants.[23] This hydrogen embrittlement of titanium is also associated with the formation of a brittle hybrid phase, leading to a reduction in the ductility of the titanium wich is related to the occurrence of fracture in dental implants [24]. Acid etching – to create a new titanium surface combining all the aforementioned surface texture features. The present study showed that precise acid selection and the sequence of processing played the main role in preparation of the rough titanium surface. The surface was poorer if it was etched with hydrochloric acid and then with sulphuric one. The very similar results were demonstrated processing implants only with hydrochloric acid or with sulphuric/hydrochloric acids and phosphoric one. Sulphuric and hydrochloric acids in sequence and time showed the best results. The newly created titanium surface according its topography was very much alike to a SLA surface. It combines the main properties of roughened titanium surface: glossily micro rough and large waviness. In general, the experimental surface was rougher when compared tocommercially available implants. Although the implant surface created using specific acid etching methods resembles an SLA created surface, further research is necessary to study the biological response to it. Characteristics of titanium implantsurfaces have been modified by additive methods(e.g. titanium plasma spray) to increase the surface area and provide a more complex surface macro-topography. Subtractive methods(e.g. blasting, acid etching) have also been used to increase the surface area and to alter its micro-topography or texture

Electrochemical Anodization

Another method that has been shown to increase surface microtexture and change surface chemistry is electrochemical anodization. The combination of potentiostatic or galvanostatic anodization of titanium in strong acids at high current density or potential, results in thickening

of the titanium oxide layer. Anodized surfaces interfere positively in bone response with higher values for biomechanical and histomorphometric tests when compared to machined surfaces [26,27]. In electro-polishing technique, a controlled dissolution of the surface takes place under the influence of electrochemical reaction. Choice of the electrolyte is generally a mixture of an acid and alcohols. Anodization of titanium surfaces at high voltages causes crystallization of surface oxide and there by produces desired roughness and porosity. In anodic oxidation electrode reactions in combination with electrical-field driven by metal and oxygen ion diffusion lead to the formation of an oxide film at the anode surface. When anodizing process is carried out below 100 V in sulfuric acid, phosphoric acid, or acetic acid, it produces microporous surfaces [28,29]. This process is used to produce Nanoporous. This electrochemical process results in an increased thickness and modified crystalline structure of the titanium oxide layer. However, it is a complex procedure and depends on various parameters such as current density, concentration of acids, composition and electrolyte temperature [30].

Calcium Phosphate Coatings

Calcium phosphate (CaP) coatings, mainly composed by hydroxyapatite, has been used as a biocompatible, osteoconductive and resorbable blasting materials [31] The idea behind the clinical use of hydroxyapatite is to use a compound with a similar chemical composition as the mineral phase of the bone in order to avoid connective tissue encapsulation and promote peri-implant bone apposition [32]. For this matter, the CaP coatings disclose osteoconductive properties allowing for the formation of bone on its surface by attachment, migration, differentiation and proliferation of bone-forming cells. the release of calcium phosphate into the peri-implant region increases the saturation of body fluids and precipitates a biological apatite onto the surface of the implant [33].This layer of biological apatite might contain endogenous proteins and serve as a matrix for osteogenic cell attachment and growth [34] and therefore, improve osteointegration. Plasma Sprayed Hydroxyapatite (PSHA) coatings are the most commonly found among the commercially available calcium phosphate coatings. The HA ceramic particles are heated to extremely high temperatures and deposited at a high velocity onto the metal surface where they condense and fuse together forming a 20–50 µm thick film. [35]

Laser Treatment

Implant surface roughening using the previously discussed methods would cause surface contamination. Laser techniques have recently been developed as an alternative to these techniques. Laser enables implant surface treatment without direct contact and provides better control Surface Roughness of Implants.Laser treatments are clean and easy method to perform. The average surface roughness of the laser treated acid-etched implant was 2.28 ìm [36].

Conclusion

Various Methods discussed in this review are well established and are widely used by the manufacturers of current day dental implants. Although these methods have been successfully developed and employed to produce varying surface topographies, the effect of the surface topographies on the long term biological compatibility and osseointegration has not been established very well. However, research in these area are very much active and several new technologies and methods will be introduced in future to produce various surface topographies on the implants surfaces.

Corresponding Author

Aarthi Vignesh,

Senior Lecturer, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077, Tamil Nadu, India.

Email Id: draarthiomfs@gmail.com

Phone Number: +91 8056985855

References

1. KamachiMudali U., T M Sridhar, BaldevRaj, Corrosion of bio implants, Sadhana, 2003; 28(3&4), 601–637.

2. Brunette DM, Mechanical, Thermal, Chemical and Electrochemical surface treatment of Titanium, in Titanium in Medicine (Ed) Brunette DM, Tengvall P, Textor M, Thomson (eds) Springer-Verlang, Berlin Heidelberg, 2001;24: 231–266.
3. Wong M, Eulenberger J, Schenk R, Hunziker E. Effect of surface topology on the osseointegration of implant materials in trabecular bone. J Biomed Mater Res 1995;29:1567-1575.
4. William R. Lacefield, Materials Characteristics of Uncoated/Ceramic-coated Implant materials, Adv Dent Res, 2007; 13: 21-26.
5. Albrektsson T, Berglundh T, Lindhe J, –Osseointegration: Historic background and current concepts. In:–(eds) –Clinical Periodontology and Implant Dentistry., Blackwell Munksgaard, Oxford, 2003;4: 809 – 820.
6. Ellingsen J E. "Surface configurations of dental implants". Periodontol. June. 1998;2000 17:36–46.
7. Wennerberg A, Albrektsson T. Effects of titanium surface topography on bone integration: a systematic review. Clin Oral Implants Res 2009;20:172-184.
8. Ask M, Rolander U, Lausmaa J, Kasemo B, –Microstructure and morphology of surface oxide films on Ti-6Al-4V–, J Mater Res,1990; 5: 1662 – 1667.
9. Coelho PG, Granjeiro JM, Romanos GE, Suzuki M, Silva NR, Cardaropoli G,. Basic research methods and current trends Braz Dent J 21(6) 2010 480 A.B. Novaes Jr. of dental implant surfaces. J Biomed Mater Res B Appl Biomater 2009;88:579-596.
10. Deligianni DD, Katsala N, Ladas S, Sotiropoulou D, Amedee J, Missirlis YF. Effect of surface roughness of the titanium alloy Ti- 6Al-4V on human broadcast marrow cell response and on protein adsorption. Biomaterials 2001;22:1241-1251.
11. Schroeder A, Van der Zypen E, Stich H et al. the reactions of bone, connective tissue and epithelium to endosteal implants with titanium sprayed surface. J Maxillofac Surg 1981;9:15-25.
12. Wennerberg A, Albrektsson T, Johansson C, Andersson B, –Experimental study of turned and grit-blasted screw-shaped implants with special emphasis on effects of blasting material and surface topography– Biomaterials,1996;17(1):15 – 22.

13. Wieland M, Chehroudi B, Textor M, Brunette DM, –Use of Ti-coated replicas to investigate the effects on fibroblast shape of surfaces with varying roughness and constant chemical composition– J Biomed Mater Res,2002; 60, 434 – 444.
14. Sammons RL, Lumbikanonda N, Gross M, Cantzler P. Comparison of osteoblast spreading on microstructured dental implant surfaces and cell behavior in an explants model of osseointegration. A scanning electron microscopy study. Clin Oral Implants Res 2005;16:657-666.
15. Bowers KT, Keller JC, Randolph BA, Wick DG, Michaels CM, –Optimization of surface micromorphology for enhanced osteoblast responses in vitro– Int J Oral Maxillofac Implants,1992;7:302 – 310.
16. Wennerberg A, Albrektsson T, Johansson C, Andersson B, –Experimental study of turned and grit-blasted screw-shaped implants with special emphasis on effects of blasting material and surface topography– Biomaterials, 1996; 17(1):15 – 22.
17. Wennerberg A, Albrektsson T, Lausmaa J, –Torque and Histomorphometric evaluation of c.p. titanium screws blasted with 25- and 75- microns- sized particles of Al2 O3 –, J Biomed Mater Res, 1996; 30(2):251 – 260.
18. Pypen CMJM, Plenk H, Jr, Ebel MF, Svagera R, Wernisch J, –Characterization of microblasted and reactive ion etched surfaces on the commercially pure metals niobium, tantalum and titanium– J Mater Sci: Mater Med, 1997; 30(2):251 – 260.
19. Cochran DL, Schenk RK, LussiA et al. bone response to unloaded and loaded titanium implant with a sandblasted and acid etched surface: a histometric study in the canine mandible. J Biomed Mater Res 1998; 40:1-11.
20. BuserD ,Nydegger T et al . removal torque values of titanium implants in the maxillae of miniature pigs. Int J Oral Maxillofac Implants 1998; 13:611-619.
21. Lim YJ, Oshida Y, Andres CJ, Barco MT. Surface characterizations of variously treated titanium materials. Int J Oral Maxillof Imp 2001;16:333-342.
22. Klokkevold P R, Nishimura R D, Adachi M, Caputo A. "Osseointegration enhanced by chemical etching of the titanium surface.A torque removal study in the rabbit . Clin. Oral Implants Res. Dec. 1997;8:442–447.

23. Klokkevold PR, Johnson P, Dadgostari S, Caputo A, Davies JE, Nishimura RD. Early endosseous integration enhanced by dual acid etching of titanium: a torque removal study in the rabbit. Clin Oral Implants Res 2001;12:350-357.
24. Isabel De Monserrat Osorio Bernal, Ito Risa, Katagi Hiroki, Tsuboi ken-Ichiro, Yamada Naoko, Tanabe Toshi-Ichiro, NagaharaKuniteru, Mori Masahiko, –Dental implant surface roughness and topography: A review of the literature–, J. Gifu. Dent. Soc, 2009; 35(3): 89 – 95.
25. Rocci A, Martignoni M, Gottlow J. Immediate loading of Branemark System TiUnite and machined-surface implants in the posterior mandible: a randomized open-ended clinical trial. Clin Implant Dent Relat Res 2003;5:557-563.
26. Burgos PM, Rasmusson L, Meirelles L, Senerby L. Early bone tissue responses to turned and oxidized implants in a rabbit tibia. Clin Impl Dent Relat Res 2008;10:181-190.
27. Hahn H, Palich W. Preliminary evaluation of porusmeta surfaced titanium for orthopedic implants. J Biomed Mater Res,1970; 45:71-77.
28. Larsson C, Thomsen P, Lausmaa J, Rodahl M, Kasemo B, Ericson LE, –Bone response to surface modified titanium implants: studies on electropolished implants with different oxide thickness and morphology– Biomaterials,1994; 15(13): 1062 – 1074.
29. Larsson C, Thomsen P, Aronsson BO, Rodhal M, Lausmaa J, Kasemo B, Ericson LE, –Bone response to surface modified titanium implants: studies on the early tissue response to machined and electropolished implants with different oxide thickness and morphology–, Biomaterials, 1996; 17(6):605-616.
30. Pan J, Thierry D, Leygraf C, –Electrochemical and XPS studies of titanium for biomedical applications with respect to the effect of hydrogen peroxide–, J Biomed Mater Res, 1994; 28(1): 113-122.
31. Brunette DM, –Mechanical, Thermal, Chemical and Electrochemical surface treatment of Titanium–, in Titanium in Medicine (Ed) Brunette DM, Tengvall P, Textor M, Thomson (eds) Springer-Verlang, Berlin Heidelberg, 2001;32:231-266.
32. Urban, R.M. Dissemination of wear particles to the liver, spleen, and abdominal lymph nodes of patients with hip or knee replacement. J Bone Joint Surg Am, 2000; 82(4): 457-76.

33. Mombelli, A., N. Muller, and N. Cionca, The epidemiology of peri-implantitis. Clin Oral Implants Res, 2012; 23(6): 67-76.
34. John E. Davies, B., Understanding Peri-Implant Endosseous Healing. Journal of Dental Education, 2003; 67: 8.
35. Wennerberg, A. and T. Albrektsson, Suggested guidelines for the topographic evaluation of implant surfaces. Int J Oral Maxillofac Implants, 2000; 15(3):331-44.
36. Tomisa A, L.M., Lee J, Mankani M, Wegst U, Saiz E., Nanotechnology approaches to improve dental implants. Int J Oral Maxillofac Implants, 2011; 26: 25-44.

Importance of Dental Procedure on Infective Endocarditis

Anitha.D.R [1], Brigit Varghese [2], Sri Sakthi.D [3]
Graduate Student [1], Senior Lecturer [2],Department of Oral and Maxillofacial Surgery,
Reader [3],Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Science,
Saveetha University, Chennai, India

Abstract

In some cases, bacteria in the mouth may trigger endocarditis in people at higher risk. Bacteria found in tooth plaque may multiply and cause gingivitis (gum disease).When gums bleed, the bacteria can enter the bloodstream and can infect other parts of the body. In the case of endocarditis, this affects the inner lining of the heart and the surfaces of its valves. During dental procedures, bacteria may involve manipulation of gingival tissue (around bone and teeth) or the periapical region of teeth (tip of the tooth root).As few studies have already been reported in this topic, we have further more elucidated the importance of dental procedure on infective endocarditis patients .

Key Words: Infective endocarditis, Bacteraemia, Blood stream, Staphylococci, FimA.

Introduction

Infective endocarditis (IE) is commonly a disorder analysis that is resolved based on the nearness of various discoveries as opposed to a solitary conclusive test outcome. Despite the fact that the nearness of IE might be clear when a patient with inclining heart injuries has bacteraemia without an undeniable source, it is frequently a troublesome finding to set up in different patients who are found in routine clinical practice[1]. At the point when the highlights of IE are atypical or conceal by existing together infections, misdiagnosis may prompt clinical disaster. Then again, finished finding of IE may prompt various iatrogenic issues emerging from antimicrobial treatment or its simultaneous vascular access.

Infective endocarditis is a genuine contamination happening on the endothelial surfaces of the heart, particularly at the valves.It is a perilous disease[2].It is constantly lethal if untreated, and it keeps on causing considerable horribleness and mortality regardless of present day antimicrobial and surgical treatments.oral microscopic organisms are most imperative etiological factor in this disease.Common dental procedures,even non-surgical dental methods, can frequently cause bacteraemia of oral commensals.scaling,gingivectomy, dental water system, brushing the teeth likewise instigates bacteraemia.Periodontally sick patients are in danger from bacteraemia even in the wake of brushing the teeth.Infective endocarditis happens in 3 conditions,1) inclining weaknesses in the heart, 2) presentation of microorganisms into the circulation system ,3) harmfulness of bacteria.Antibiotics must be sufficiently used to keep this infection.Antibiotic prophylaxis ought to be given before dental procedure[3].

Infective Endocarditis

Infective endocarditis (IE) is characterized as a disease of the endocardial surface of the heart , which may incorporate at least one heart valves, the painting endocardium, or a septal deformity. Its intracardiac impacts incorporate extreme valvular deficiency, which may prompt obstinate congestive heart disappointment and myocardial abscesses[4].Fungi,chlamydia,rickettsia can cause contamination, however most regular reason for this sickness are microscopic organisms, so they are additionally called bacterial endocarditis.According to common history of ailment, it has been grouped in to intense and subacutetypes.It has been arranged in to prosthetic valve endocarditis and local valve

endocarditis related with valve replacement.It can likewise be named intravenous medication mishandle endocarditis and nosocomial endocarditis related with intravenous catheters[5].

The rate of IE runs starting with one nation then onto the next inside 3– 10 scenes/100 000 man years. This may reflect methodological contrasts between studies instead of genuine variety. Of note, in one studies, the rate of IE was low in youthful patients however expanded drastically with age—the pinnacle rate was 14.5 scenes/100 000 man a long time in patients in the vicinity of 70 and 80 years of age[6]. In every epidemiological investigation of IE, the male:female proportion is ≥2:1, in spite of the fact that this higher extent of men is inadequately comprehended. Moreover, female patients may have a more terrible anticipation and experience valve surgery less as often as possible than their male counterparts[7].

Pathophysiology

Infective endocarditis grows most usually on the mitral valve, once in a while on the aortic valve, the consolidated mitral and aortic valve, the tricuspid valve, and, infrequently, the pulmonic valve.IE creates from basic process,1) Bacteraemia that conveys the living beings to the surface of the valve,2)Adherence of the organisms,3)Eventual attack of the valvular pamphlets. IE comes about because of adherence of microorganisms to injured cardiovascular surfaces and their multiplication at the adjacentsite.Damaged heart valves as a sequela of rheumatic fever or past endocarditis, obtained valve injuries, roughened heart surfaces because of-fly stream impact from blood crossing innate cardiovascular sores, for example, septal imperfection, and prosthetic heart valve are the typical inclining condition for infective endocarditis sterile vegetations made out of platelet-fibrin cluster and thrombus are at first framed on harmed endothelial surfaces, at that point microscopic organisms are acquainted in with blood stream[8].Subsequent development of the infective vegetation causes nearby myocardial sore, that restrains valvular capacity and it brings about congestive heart disappointment.

The advancement of IE is the net consequence of the unpredictable collaboration between the circulation system pathogen with framework atoms and platelets at locales of endocardial cell harm. What's more, a significant number of the clinical indications of IE exude from the host's resistant reaction to the tainting microorganism[9]. The accompanying succession of occasions is thought to bring about IE: development of nonbacterial thrombotic endocarditis

(NBTE) on the surface of a heart valve or somewhere else that endothelial harm happens, bacteraemia, adherence of the microscopic organisms in the circulatory system to NBTE, and multiplication of microorganisms inside a vegetation.

Formation of NBTE

Turbulent blood stream delivered by specific kinds of innate or obtained coronary illness, for example, spill out of a high-to a low-weight chamber or over a limited opening, damages the endothelium. This makes an inclination for statement of platelets and fibrin on the surface of the endothelium, which brings about NBTE. Attack of the circulation system with a microbial animal categories that has the pathogenic potential to colonise this site would then be able to bring about IE.

Transient Bacteraemia

Mucosal surfaces are populated by a thick endogenous microflora. Injury to a mucosal surface, especially the gingival cleft around teeth, oropharynx, GI tract, urethra, and vagina, discharges a wide range of microbial species temporarily into the circulation system. Transient bacteraemia caused by viridian assemble streptococci and other oral microflora happens usually in relationship with dental extractions or other dental methods or with routine every day activities[10]. Albeit dubious, the recurrence and power of the subsequent bacteraemia are accepted to be identified with the nature and size of the tissue injury, the thickness of the microbial vegetation, and the level of irritation or disease at the site of injury. The microbial species entering the course relies upon the extraordinary endogenous microflora that settlements the specific damaged site.

Bacterial Adherence

The capacity of different microbial species to hold fast to particular destinations decides the anatomic localisation of contamination caused by these microorganisms. Middle people of bacterial adherence fill in as harmfulness factors in the pathogenesis of IE. Various bacterial surface parts introduce in streptococci, staphylococci, and enterococci have been appeared in creature models of test endocarditis to work as basic attachments. Some viridian aggregate streptococci contain a FimA protein that is a lipoprotein receptor antigen I (LraI) that fills in as a noteworthy bond to the fibrin platelet lattice of NBTE. Staphylococcal bonds work in no less than 2 ways. In one, microbial surface parts perceiving cement lattice atoms encourage

the connection of staphylococci to human extracellular grid proteins and to restorative gadgets that wind up covered with network proteins after implantation[11]. In the other, bacterial extracellular structures add to the arrangement of biofilm that structures on the surface of embedded restorative gadgets. In the two cases, staphylococcal bonds are imperative destructiveness factors.

Both FimA and staphylococcal grips are immunogenic in exploratory diseases. Antibodies arranged against FimA and staphylococcal bonds give some defensive impact in trial endocarditis caused by viridian assemble streptococci and staphylococci[12].The consequences of these exploratory examinations are very captivating, in light of the fact that the advancement of a successful immunisation for use in people to anticipate viridian gather streptococcal or staphylococcal IE would be of real significance.

Multiplication of Bacteria Within a Vegetation

Microorganisms follower to the vegetation invigorate promote statement of fibrin and platelets on their surface. Inside this segregated concentration, the covered microorganisms duplicate as quickly as microscopic organisms in stock societies to achieve maximal microbial densities of 108 to 1011 province shaping units for each gram of vegetation inside a brief timeframe on the left half of the heart, evidently uninhibited by have guards in left-sided lesions[13]. Right-sided vegetations have bring down bacterial densities, which might be the result of host barrier components dynamic at this site, for example, polymorphonuclear action or platelet-determined antibacterial proteins. Over 90% of the microorganisms in develop left-or right-sided valvularvegetations are metabolically dormant as opposed to in a dynamic development stage and are in this manner less receptive to the bactericidal impacts of anti-infection agents.

Dental Techniques and Infective Endocarditis

The circulatory system is clean under typical solid condition.The presentation of microscopic organisms into the circulation system is vital for an intra heart contamination to occur.Such bacteraemia happens after tooth extractions and other oral surgery procedures.Non surgical dental techniques, for example, organization of nearby anaesthesia, orthodontic band position, periodontal testing, dental prophylaxis, scaling, root arranging and even after tooth brushing and flossing. Most mash and periapical ailment is caused by microbial infection[14].

Agent techniques for its administration run from straightforward mash topping, through traditionalist root channel treatment to crisis seepage and surgical endodontics. Practically speaking, any of these mediations and their aides including neighbourhood analgesic organization and elastic dam arrangement including contaminated tissues are equipped for inciting bacteraemia and setting patients in danger of infective endocarditis. Bacteraemia does not happen or is a phenomenal and fleeting occasion following pulpotomy or root channel preparation short of root-apex[15]. Contemporary examinations utilizing thorough microbiological methods have yielded more positive blood societies, and even the utilization of rubber dam was as of late appeared in kids to deliver bacteraemia of practically identical extent to a dental extraction[16]. Profoundly delicate, sub-atomic strategies presently can't seem to be connected in the distinguishing proof and sourcing of cultivable and non-cultivable organisms in the dissemination after cautious endodontic treatment. It is likely that a wide assortment of creatures would be identified, however their importance as for infective endocarditis isn't known. It might create the impression that endodontics as a hazard factor for IE relies on the idea of instrumentation, including the think or unintentional expansion of instruments past the root end. More obtrusive, surgical endodontic strategies with fold withdrawal and periapical curettage are related typically with a high predominance of bacteraemia[17].

Periodontitis and Infective Endocarditis

Bacteraemia is all the more habitually inducible in patients with serious periodontal maladies than the individuals who have more beneficial periodontal tissue after tooth brushing and periodontal pocket probing.Gum irritation extricates the gingival epithelial tissues and it regularly end up ulcerative at inward piece of periodontal pockets. It can give oral microscopic organisms the course to getting in to the circulation.Periodontopathic microorganisms, for example, An actinomycetemcomitans and P gingivalis have the ability to attack the host epithelial and connective tissues, brings about augmentation of incendiary sore [18].Periodontopathic microbes which assume a critical part in acceptance of IE by causing gum aggravation and opening a course toward the blood dissemination for endocardopathic viridian streptococci.

Counteractive Action of Infective Endocarditis

It has for some time been exhorted that Antibiotic prophylaxis is required before dental techniques prone to create a bacteraemia in view of the high bleakness and mortality identified with IE. Two systems are believed to be included. Initial, a diminishment in the quantity of life forms in the blood and second, a decrease in the bond of living beings to the nonbacterial thrombotic vegetation.cephalaxine and cefadoxil can be utilized for patients with history of penicillin hypersensitivity, yet the utilization of beta lactate operators for patients with penicillin sensitivity might be addressed, different specialists, for example, clindamycin,azithromycin,clarithromycin ought to be prescribed. Notwithstanding anti-toxin prophylaxis of IE, different techniques for lessening bacteraemia from an oral root have been looked for. The utilization of pre-surgical 1% povidone-iodine has been shown to cause noteworthy lessening in bacteraemia from oral sources ,albeit routine utilise may incite the choice of safe microorganisms[19].

The rule of prophylaxis for IE was produced based on observational examinations in the mid twentieth century[20]. The essential speculation depends on the presumption that bacteraemia consequent to therapeutic systems can cause IE, especially in patients with inclining factors, and that prophylactic anti-infection agents can anticipate IE in these patients by limiting or forestalling bacteraemia, or by changing bacterial properties prompting decreased bacterial adherence on the endothelial surface. The suggestions for prophylaxis are situated to some degree on the consequences of creature thinks about demonstrating that anti-microbial could keep the advancement of test IE after vaccination of microscopic organisms.

Standards of the New ESC Guidelines

Recent guidelines proposed impediment of prophylaxis to patients at expanded danger of unfriendly result of IE or even entire discontinuance of anti-infection prophylaxis in any patient gatherings, the Task Force decided[21]:

– to maintain the principle of antibiotic prophylaxis when performing procedures at risk of IE in patients with predisposing cardiac conditions, *but*

– to limit its indication to patients with the highest risk of IE (*Table 1*) undergoing the highest risk procedures (*Table 2*).

Table 1: Cardiac conditions at highest risk of infective endocarditis for which prophylaxis is recommended when a high risk procedure is performed

RECOMMENDATIONS: PROPHYLAXIS	CLASS [a]	LEVEL[b]
Antibiotic prophylaxis should only be considered for patients at higher risk of IE 1.Patients with prosthetic valve or prosthetic materials used for cardiac valve repair. 2. Patients with previous IE 3. Patient with congenital heart disease	II_a	C
Antibiotic prophylaxis is no longer recommended in other forms of valvular or congenital heart disease	III	C

[a]Class of recommendation.

[b]Level of evidence.

Table 2: Recommendations for prophylaxis of infective endocarditis in highest risk patients according to the type of procedure at risk

Recommendations : prophylaxis	Class[a]	Level[b]
A - Dental procedure Antibiotic prophylaxis should only be considered for dental procedures requiring manipulation of the gingival or periapical region of tooth or perforation of oral mucosa	II_a	C
Antibiotic prophylaxis is not recommended for LA injections in non infected tissue, removal of sutures, dental X-rays, placement or	III	C

adjustment of removable prosthesis or orthodontic appliances or braces.		
B – Respiratory tract procedures* Antibiotic prophylaxis is not recommended for respiratory tract procedures, including bronchoscopy or laryngoscopy, transnasal or endotracheal intubation.	III	C
C – Gastrointestinal or urogenital procedures* Antibiotic prophylaxis is not recommended for gastroscopy, colonoscopy, cystoscopy or transoesophageal echocardiography.	III	C
D – Skin and soft tissues* Antibiotic prophylaxis is not recommended for any procedures	III	C

[a]Class of recommendation.

[b]Level of evidence.

*For management when infections are present.

1**. Patients with the Most Elevated Danger of Infective Endocarditis** (Table 1)

They incorporate three classes of patients:Although AHA rules suggest prophylaxis in cardiovascular transplant beneficiaries who create heart valvulopathy, this isn't upheld by solid confirmation. What's more, in spite of the fact that the danger of unfriendly result is high when IE happens in transplant patients, the likelihood of IE from dental starting point is

to a great degree low in these patients.The ESC Task Force does not suggest prophylaxis in such circumstances.

a. Patients with a prosthetic valve or a prosthetic material utilized for heart valve repair: these patients have a higher danger of IE, a higher mortality from IE and more regularly create intricacies of the sickness than patients with local valves and an indistinguishable pathogen.

b. Patients with past IE: they likewise have a more serious danger of new IE, higher mortality and rate of confusions than patients with a first scene of IE.

c. Patients with intrinsic coronary illness (CHD), specifically those with complex cyanotic coronary illness and the individuals who have post-agent palliative shunts, courses, or different prostheses. After surgical repair with no leftover imperfections, the Task Force suggests prophylaxis for the initial a half year after the method until the point when endothelialization of the prosthetic material happens.

Prophylaxis isn't suggested for some other type of local valve infection (counting the most generally distinguished conditions, bicuspid aortic valve, mitral valve prolapse, and calcific aortic stenosis).

2. **Most Astounding Danger Strategies**(Table 2)

a. **Dental Strategies**

Strategies in danger include the control of the gingival or periapical district of teeth or aperture of the oral mucosa (counting scaling and root channel methodology). Prophylaxis should just be considered for patients depicted in Table 1 experiencing any of these methods, and isn't prescribed in different circumstances. The fundamental focuses for anti-toxin prophylaxis in these patients are oral streptococci. Table 2 abridges the primary regimens of anti-microbial prophylaxis prescribed before dental methods. The effect of expanding protection of these pathogens for the adequacy of anti-microbial prophylaxis is indistinct.

Fluoroquinolones and glycopeptides are not prescribed because of their vague viability and the potential enlistment of protection.

b. **Other In Danger Methods**

There is no convincing proof that bacteraemia coming about because of either respiratory tract techniques, gastrointestinal or genitorurinary systems, dermatological or musculoskeletal methodology cause IE[22]. Subsequently, prophylaxis isn't prescribed in patients experiencing these systems.

Respiratory Tract Methodology

Patients recorded in Table 1 who experience an obtrusive respiratory tract methodology to treat a built up contamination, e.g. seepage of a canker, ought to get an anti-toxin regimen which contains an against staphylococcal penicillin or cephalosporin. Vancomycin ought to be given to patients unfit to endure a β-lactam. Vancomycin or another appropriate specialist ought to be managed if the disease is known or suspected to be caused by a methicillin-safe strain of S. aureus (MRSA).

Gastrointestinal or Genitourinary Methods

On account of a set up disease or if anti-infection treatment is demonstrated to forestall wound contamination or sepsis related with a gastrointestinal or genitourinary tract strategy in patients portrayed in Table 1, it is sensible that the anti-microbial regimen incorporates a specialist dynamic against enterococci, e.g. ampicillin, amoxicillin, or vancomycin. Vancomycin should just be regulated to patients unfit to endure β-lactams. On the off chance that contamination is caused by a known or associated strain with safe enterococcus, conference with an irresistible infections expert is recommended[23].

Dermatological or Musculoskeletal Methods

For patients depicted in Table 1 experiencing surgical methods including tainted skin (counting oral abscesses), skin structure, or musculoskeletal tissue, it is sensible that the restorative regimen contains an operator dynamic against staphylococci and β-haemolytic streptococci, e.g. an against staphylococcal penicillin or cephalosporin. Vancomycin or clindamycin might be utilized as a part of patients unfit to endure a β-lactam. On the off chance that the disease is known or suspected to be caused by MRSA, vancomycin or another reasonable specialist ought to be directed.

Body Puncturing and Inking

These developing social patterns are a reason for concern, especially for those people with CHD who are at expanded defencelessness for the procurement of IE. Case reports of IE in the wake of penetrating and inking are expanding, especially while puncturing includes the tongue, in spite of the fact that production inclination may overestimate the issue since a large number of individuals are inked and penetrated far and wide and CHD concerns just 1% of the overall public. Right now no information are accessible on (a) the frequency of IE after such strategies and (b) the adequacy of anti-microbials for counteractive action. Training of patients in danger of IE is fundamental, and penetrating and inking techniques ought to be demoralized. On the off chance that attempted, strategies ought to be performed under entirely sterile conditions however anti-infection prophylaxis isn't recommended[24].

Heart or Vascular Surgery

In patients experiencing implantation of a prosthetic valve or intravascular prosthetic or other remote material, peri-agent anti-microbial prophylaxis ought to be considered because of the expanded hazard and unfavourable result of a contamination. The most regular microorganisms hidden early (<1 year after surgery) prosthetic valve diseases are CNS and S. aureus. Prophylaxis ought to be begun instantly before the methodology, rehashed if the strategy is drawn out, and ended 48 h subsequently. It is firmly prescribed that potential wellsprings of dental sepsis are wiped out no less than 2 weeks before implantation of a prosthetic valve or other intracardiac or intravascular remote material, unless the last technique is critical.

Methods Causing Medicinal Services Related IE

They speak to up to 30% of all instances of IE and are portrayed by an expanding frequency and a serious guess, subsequently speaking to a critical medical issue. Albeit routine antimicrobial prophylaxis directed before most intrusive strategies isn't prescribed, aseptic measures amid the inclusion and control of venous catheters and amid any obtrusive techniques are obligatory to diminish the rate of this disease.

1. Blood Culture

Positive blood societies remain the foundations of analysis and give live microscopic organisms to powerlessness testing. Three sets (counting no less than one high-impact and

one anaerobic), each containing 10 mL of blood acquired from a fringe vein utilizing fastidious sterile strategy, is for all intents and purposes constantly adequate to recognise the standard microorganisms—the analytic yield of rehashed testing from there on is low. Inspecting from focal venous catheters ought to be evaded in perspective of the high danger of contaminants (false positives, ordinarily staphylococcal) and deluding discoveries. The requirement for culture before anti-infection organization is plainly obvious, despite the fact that studies of contemporary practice uncover visit infringement of this run the show. In IE, bacteraemia is relatively consistent, which has two ramifications: (1) there is no justification for postponing blood testing to harmonise with pinnacles of fever; and (2) for all intents and purposes all blood societies (or a greater part of them) are certain. Accordingly, a solitary positive blood culture ought to be respected carefully to establish the conclusion of IE, particularly for conceivably 'contaminants, for example, CNS or corynebacteria.

Albeit IE caused by anaerobes is phenomenal, societies ought to be hatched in both oxygen consuming and anaerobic environments to distinguish living beings, for example, Bacteroides or Clostridium species. At the point when societies stay negative at 5 days, subculture onto chocolate agar plates may permit ID of an exacting life form. Drawn out culture is related with rising probability of pollution, and elective procedures (or an elective determination) ought to be considered at this stage.

2.Histological/Immunological Strategies

Obsessive examination of resected valvular tissue or embolic pieces remains the best quality level for the determination of IE and may likewise control antimicrobial treatment if the causative operator can be recognised by methods for extraordinary stains or immunohistological systems. Electron microscopy has high affectability and may describe new microorganisms, yet is tedious and costly. Coxiellaburnetii and Bartonella species might be effortlessly identified by serological testing utilizing backhanded immunofluorescence or chemical connected immunosorbent examine (ELISA), and late information exhibit comparable utility for staphylococci. Immunological examination of pee may permit location of microorganism corruption items, and ELISA discovery of Legionella species has been portrayed utilizing this technique[25]. Joining of these strategies into acknowledged symptomatic criteria anticipates planned approval.

Treatment for Infective Endocarditis

Staphylococcal Endocarditis

Intravenous treatment for a month is suggested for staphylococcal NVE, which ought to be stretched out to no less than a month and a half in patients with intracardiacprostheses[26,27].

NVE, methicillin-safe: vancomycin AND rifampicin.

NVE, methicillin-safe, vancomycin-safe: daptomycin AND rifampicin OR gentamicin.

PVE, methicillin, rifampicin-powerless staphylococci: flucloxacillin AND rifampicin AND gentamicin.

PVE, methicillin-safe, vancomycin-powerless or penicillin sensitivity: vancomycin AND rifampicin AND gentamicin.

PVE, methicillin-safe, vancomycin-safe: daptomycin AND rifampicin AND gentamicin.

Streptococcal Endocarditis

Benzylpenicillinmonotherapy; ceftriaxone monotherapy; benzylpenicillin AND gentamicin; ceftriaxone AND gentamicin.Treatment of streptococci in patients with hugepenicillin allergy: vancomycin AND gentamicin; teicoplanin AND gentamicin.

Enterococcal Endocarditis

Amoxicillin OR penicillin AND gentamicin; vancomycin AND gentamicin; teicoplanin AND gentamicin; amoxicillin (for amoxicillin-helpless AND abnormal state gentamicin safe disconnects).

Parasitic Endocarditis

Against parasitic operators utilized incorporate fluconazole, voriconazole, amphotericin, micafungin, caspofungin, anidulafungin, posaconazole and flucytosine. Itraconazole ought not be utilized.

CandidalEndocarditis

Starting treatment ought to be with an echinocandin or amphotericin. Surgical valve substitution is suggested.

Conclusion

Regular dental methods, even non-surgical dental techniques, frequently cause bacteraemia that can bring about infective endocarditis in individuals who have an inclining hazard for this illness, for example, valvular heart ailments including prosthetic valve, inborn heart diseases,cardiomyopathy,coronary course malady, pacemaker implantation etc.Common dental methodology frequently cause bacteraemia and periodontally unhealthy patients may significantly experience the ill effects of bacteraemia after tooth brushing.Antibiotics must be utilized sufficiently so as to avert such contaminations amid dental systems.

Corresponding author
Sri Sakthi. D
Reader,Department of Public Health Surgery
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,
Saveetha University,Chennai,India
162, Poonamallee High Road,Chennai – 600077,Tamil Nadu, India.
Mobile Number: +91 – 9962787531
Email ID: drsrisakthiphd@gmail.com

Reference

1. Al-Karaawi ZM, Lucas VS, Gelbier M, Roberts GJ. Dental procedures in children with severe congenital heart disease: a theoretical analysis of prophylaxis and non-prophylaxis procedures. Heart 2001;85:66–8.
2. Bahn SL, Goveia G, Bitterman P, Bahn AN. Experimental endocarditis induced by dental manipulation and oral streptococci. Oral Surgery, Oral Medicine, Oral Pathology. 1978; 45:549–59.
3. Tomas Carmona I, Diz Dios P, Scully C. An update on the controversies in bacterial endocarditis of oral origin. Oral Surgery Oral Medicine Oral Pathology 2002;93:660–70.
4. Gendron R, Grenier D, Maheu-Robert LF. The oral cavity as a reservoir of bacterial pathogens for focal infections. Microbes and Infection. 2000; 2:897–906.
5. Tsitasika A, Pefanis A, Perdikaris GS, Donta I, Karayiannakos P, GiamarellouH .Single oral dose azithromycin prophylaxis against experimental streptococcal or staphylo- coccal aortic valve endocarditis. Antimicrobial Agents and Chemotherapy 2000;44:1754–56
6. Habib G, Hoen B, Tornos P. Guidelines on the prevention, diagnosis, and treatment of infective endocarditis : the Task Force on the Prevention, Diagnosis, and Treatment of Infective Endocarditis of the European Society of Cardiology (ESC). Eur Heart J. 2009;30(19):2369–2413.
7. Murdoch DR, Corey GR, Hoen B. Clinical presentation, etiology, and outcome of infective endocarditis in the 21st century: the International Collaboration on Endocarditis-Prospective Cohort Study. Arch Intern Med. 2009;169(5):463–473.
8. Karchmer AW. Infectious endocarditis.Harrison's Principles of Internal Medicine. 2008; 45: 789–797.
9. Durack DT, Lukes AS, Bright DK. Duke Endocarditis Service. New criteria for diagnosis of infective endocarditis: utilization of specific echocardiographic findings. Am J Med. 1994;96(3):200–209.
10. McKinsey DS, Ratts TE, Bisno AL. Underlying cardiac lesions in adults with infective endocarditis. The changing spectrum. Am J Med. 1987;82(4):681–688.

11. Cherubin CE, Neu HC. Infective endocarditis at the Presbyterian Hospital in New York City from 1938–1967. Am J Med. 1971;51(1):83–96.
12. MorcillonP. Endocarditis and endarteriftis,In:Infectiousdiseases.ArmstrongD,CohenJ,eds.Harcourt Publishers,London,1999;2.50.1-2,50.9
13. HorderT .Infective endocarditis.Q J Med 2011:319-323.
14. Durack DT, Lukes AS, Bright DK. Duke Endocarditis ServiceNew criteria for diagnosis of infective endocarditis: utilization of specific echocardiographic findings, Am J Med , 1994; 96:200-9.
15. Bayer AS, Ward JI, Ginzton LE, Shapiro. Evaluation of new clinical criteria for the diagnosis of infective endocarditis, Am J Med .1994;96 : 211-9.
16. Hoen B, Selton-Suty C, Danchin N. Evaluation of the Duke criteria versus the Beth Israel criteria for the diagnosis of infective endocarditis, Clin Infect Dis.1995;21: 905-9.
17. Arguello EA, Varini S, Romorini A. Infective endocarditis in the Argentine Republic, 3d International Symposium on modern concepts in endocarditis (Boston). 1995;54:78.
18. Kanavos K, Antoniadou A, Venetis C. Retrospective analysis of Duke's criteria in 60 cases of infective endocarditis, 3d International Symposium on modern concepts in endocarditis (Boston) .1995;4:56.
19. Gubler JGH, Kuster M, Dutly F. Whipple endocarditis without overt gastrointestinal disease: report of four cases, Ann Intern Med . 1999;131:112-6.
20. Roe MT, Abramson M, Sexton DJ. Positive and negative effects of transesophageal echocardiography on the Duke criteria for infective endocarditis, J Am Coll Cardiol .1996;27:2-22
21. Krivokapich J, Child JS. Role of transthoracic and transesophageal echocardiography in diagnosis and management of infective endocarditis, Cardiol Clin .1996;14:363-82.
22. Raoult D. AfebrilebloodculturenegativeendocarditisAnnIntern Med .1999;131:144-5.
23. Fowler VG, Sanders LL, Kong LK. Infective endocarditis due to Staphylococcus aureus:prospectively identified cases with follow-up, Clin Infect Dis.1999;28: 106-14.

24. Lamas CC, Eyken SJ. Suggested modifications to the Duke criteria for the clinical diagnosis of native valve and prosthetic valve endocarditis: analysis of 118 pathologically proven cases, Clin Infect Dis.199725:713-9.
25. Fowler VG, Li J, Corey GR. Role of echocardiography in evaluation of patients with Staphylococcus aureus bacteraemia: experience in 103 patients, J Am Coll Cardiol .1997;30:1072-8.
26. Shathariya A. Antibiotic prophylaxis for Infective endocarditis.J. Pharm. Sci. & Res. 2015; 7(8):571-572
27. Santhosh kumar MP, Sneha S. Knowledge and awareness regarding antibiotic prophylaxis for infective endocarditis among undergraduate students. Asian J Pharm Clin Res. 2016;9(2):154-159.

Importance of Drug Enantiomers in Clinical Pharmacology- An Overview

KausalyahKrisna Malay[1], DuraisamyRevathi[2], Ezhilarasan[3]

Graduate Student[1],Senior Lecturer[2], Department of Prosthodontics,Assistant Professor[3], Department of Pharmacology,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

The single enantiomers are considered to be safer and better alternatives to racemates, has resulted in a need for developing single isomers as drug products. Most of the drugs in use today are chiral. It is well established that the pharmacological activity is mostly restricted to one of the enantiomers and the individual enantiomers of racemic drugs frequently differ in their biological effects. In many cases, the inactive enantiomer shows unwanted side effects or even toxic effects. A pharmacological study reveals that there is a need for separating and quantifying enantiomers in biological samples to assess the toxic effects of such drugs. Single enantiomer products are being re-marketed by so called the chiral switch process. (Racemate to single enantiomer).Therefore, it is important to promote the chiral separation and analysis of racemic drugs in pharmaceutical industry as well as in clinic in order to eliminate the unwanted isomer from the preparation and to find an optimal treatment and a right therapeutic control for the patient. In this article, we review the nomenclature, pharmacology, toxicology, pharmacokinetics, metabolism etc of some usual chiral drugs as well as their mechanisms. Different techniques used for the chiral separation in pharmaceutical industry as well as in clinical analyses are also examined.About more than half of the drugs currently in use are chiral compounds and near 90% of the last ones are marketed as racemates consisting of an equimolar mixture of two enantiomers. Although they have the same chemical structure, most isomers of chiral drugs exhibit marked differences in biological activities such as pharmacology, toxicology, pharmacokinetics, metabolism etc. Some mechanisms of these properties are also explained.

KeyWords: *enantiomers, chiral drug, thalidomide, stereoisomers, pharmacology, enantioselective*

Introduction

Enantiomers chemistry was discovered by Louis Pasteur, a French chemist and biologist, when he separated by hand for the first time, in 1848, the two isomers of sodium ammonium tartrate. However, it needed about a century later to find that the phenomenon of chirality plays a key role not only in the life of plants and animals but also in pharmaceutical, agricultural and other chemical industries. All proteins, enzymes, amino acids, carbohydrates, nucleosides and a number of alkaloids and hormones are chiral compounds. In pharmaceutical industries, 56% of the drugs currently in use are chiral products and 88% of the last ones are marketed as racemates consisting of an equimolar mixture of two enantiomers (1). In contrast to chiral artificial products, all natural compounds are under single enantiomeric form, for example, all natural amino acids are l-isomer (levorotatory) as well as all natural sugars (carbohydrates) are d-isomer (dextrorotatory). Although they have the same chemical structure, most enantiomers of racemic drugs exhibit marked differences in biological activities such as pharmacology, toxicology, pharmacokinetics, metabolism etc. The mechanisms of chiral drugs with biological environment are now explained. Therefore, it is important to promote the chiral separation and analysis of racemic drugs in pharmaceutical industry as well as in clinic in order to eliminate the unwanted isomer from the preparation and to find an optimal treatment and a right therapeutic control for the patient (2).

An enantiomer is one of two stereoisomers that are mirror image of each other that are non-identical. Their isomers have the same chemical formula but with different orientations of the side groups around the central atom. They share the same chemical and physical properties, the same chemical formula, the same bond angles, the same boiling point, and the same melting point. In fact, their only difference lies in the direction in which they rotate polarized light, which are dextrorotary (clockwise rotation) and levorotary (counterclockwise rotation). (3) Due to these changes, sometimes only one of the enantiomers have a therapeutic effect while the other may have an entirely different effect, no effect at all, or even have undesired side effects. Our body

can only recognize one enantiomer which in result producing therapeutic effect. This will bind along with its chiral receptor whereas another enantiomer disturbs the metabolic and regulation process and causes side effect.This problem has been overcome with the production of single enantiomer drug.

Enantiomer was a top-class subject for academic research as well as for pharmaceutical development. Accounting for the important role of enantiomers separation, the 2001 Nobel Prize in Chemistry has been awarded to three scientists, Dr. William S. Knowles and Pr. K. Barry Sharpless in USA and Pr. RyoriNyori in Japan, for their development of asymmetric synthesis using chiral catalysts in the production of single enantiomer drugs or chemicals. Thanks to a wide range of new technologies for enantiomers separation, US Food and Drug Administration (FDA) recently recommends the assessments of each enantiomer activity for racemic drugs in body and promotes the development of new chiral drugs as single enantiomers(3).

Few separation techniques are introduced and single enantiomer is formulated. Examples of single enantiomer drug used clinically nowadays are omeprazole, propranolol, albuterol etc.In many cases, one enantiomer is the active pharmaceutical ingredient while the other can be benign or even toxic. Thalidomide racemate was one of the first drugs recognized to cause birth defects in humans, which is due to (-)(S)thalidomide. It causes severe side-effects and thousands of babies were born with missing or abnormal arms, hands, legs, or feet. One side of the drug is known to efficacy whereas the other is the cause of side effect which causes teratogenic effect. As the next step in newer generation the scientists introduces the separation technique to prevent the side effects. Here is where the single enantiomers is introduced commercially. Only single-enantiomers are provided in market nowadays. (5)

In this article, we review the nomenclature of chiral compounds, the biological activities such as pharmacology, toxicology, pharmacokinetics, metabolism etc of some usual racemic drugs in therapeutics as well as their mechanisms. Different techniques used for the chiral separation in pharmaceutical industry as well as in clinical analyses are also examined

History of Enantiomers

Previously, all drugs were made from racemic mixtures or mixtures with equal amounts of both enantiomers. Separation of the two enantiomers thought to be unnecessary due to its high cost. Therefore, enantiomer drugs were given to patients without purification. In 1960s, to inhibit the effects of morning sickness in pregnant woman the drug named Thalidomide is introduced. It is a racemic mixture composed of both R-thalidomide and S-thalidomide. As a result after its release, over 10,000 children of women using this drug were born with severe birth defects such as phocomelia, the development of flipper-like limbs.(6)

Consequently, Thalidomide was sealed as one of the most severe pharmaceutical failures of the 20th century. Research is quickly done by scientists but it was fiasco. However within a few years, scientists discovered the cause of the fatal flaw, it was the nature of enantiomers which was far more complex than ever imagined. The R-form of the enantiomer showed the reputed characteristic of inhibiting morning sickness. It seemed effective in preventing morning sickness and producing desired results. However, the S-form of the organic compound thalidomide produced teratogenic effects which caused the fatal defects in embryonic formation. (7) These effects were believed to result from the increased affinity of S-thalidomide with embryonic cerebrum proteins, which have a direct effect on limb formation. The Thalidomide incident had invested greater interest in a previously recondite and unknown region of the enantiomers.

Pharmaceutical expects spent much of their resources developing better techniques to separate enantiomers in the process of forming aenantiopure drug. Various techniqueswere experimented to separate enantiomers. However the first successful method which is reproducible in separation process was the gas chromatograph, was done in 1966 using an isoleucine lauryl ester mixture as the stationary phase for separating two amino acid enantiomers. Once the racemic mixture was separated into enantiopure mixtures, the optical purity, or the ratio of the two enantiomers, was measured to determine the efficacy of the separating process. (8)

Pharmacology of Enantiomers

Enantiomers (also sometimes called stereoisomerism or chirality or dissymmetry) are a property of an object which is non-superimposable with its mirror image. When a molecule cannot be superimposed on its mirror image, this molecule and its image are called chiral. It is like left and

right hands. The two non-superimposable mirror-image forms of chiral molecules are called enantiomers. Enantiomers are most commonly formed when a carbon atom contains four different substituents (asymmetric carbon atom or stereogenic carbon or also called chiral center). A chiral molecule is a molecule having at least one asymmetric carbon. Carbon is not the only atom that can act as an asymmetric center. Sulfur, phosphorus and nitrogen can sometimes form chiral molecules such as omeprazole, cyclophosphamide and methaqualone, respectively. Chiral molecules exhibit optical activity, so enantiomers are also sometimes called optical isomers. The two enantiomers of such compounds may be classified as levorotary (l-isomer) or dextrorotary (d-isomer) depending on whether they rotate plane-polarized light in a left (-) or right (+) -handed manner, respectively. An equimolar mixture (50/50) of the two enantiomers of a chiral compound is called a racemic mixture (racemate) with sign (±) or (d, l) that does not exhibit optical activity. Optical isomers or enantiomers are molecules having the same chemical formula, the same physical and chemical properties, but differing in their optical activity and their spatial arrangement.

Enantiomers are now determined by their spatial arrangement (3 dimensions) of substituents (groups) around a chiral center (asymmetric carbon) in the molecule. This configuration follows the Cahn-Ingold-Prelog (CIP) convention which is used to assign priorities to substituent groups. This system is based on a set of rules for ordering the substituents attached to the asymmetric atom by using sequence rules to assign priorities. The spatial arrangement of a chiral compound is determined by nuclear magnetic resonance or/and X-ray crystallography diffraction. The R/S tri-dimensional configuration allows explaining the interaction of enantiomers with their biologic receptors.

Diastereomers are any molecules which have two or more chiral centres. A diastereomer with two chiral carbon has four isomers. Unlike enantiomers, the physical and chemical properties of diastereomers can differ and consequently, their chemical characterization is easy and their biological activities are often different. This is the basis for derivatization of enantiomers to form diastereomers in chiral separation and also for the explanation of enantiomer activities with their chiral receptors in the body. Diastereomers, enantiomers and geometric isomers form a family called stereoisomers that are molecules having the same chemical formulas but differing only

with respect to the spatial arrangement.Eutomer refers to bioactive enantiomer or enantiomer having higher pharmacological activity. Its opposite is called distomer.Epimers are two diastereoisomers having a different configuration at only one chiral centre.

Enantioselectivity is a property of a process whereby one enantiomer is expressed exclusively or predominantly over the other. In pharmacological terms, that means a biological structure (enzyme, antibody or receptor) which exhibits affinity towards one enantiomer over the other. Enantioselective assay is an analytical method capable of separating and quantifying enantiomers.

In a stereoselective synthesis, one of a set of isomers is predominantly or exclusively formed whereas in a stereospecific synthesis, one isomer leads to one product while another isomer leads to the opposite product.Homochirality is the biological chirality in which all biologic compounds have the same chirality such as all amino acids are levorotary isomers. Chiral switch is a procedure used to transform an old racemic drug into its single active enantiomer. This new enantiomeric drug developed by a pharmaceutical manufacturer will receive additional patent protection and a new generic name. (9)

Technique Used for Separation of Enantiomers

The main techniques of enantiomer separations are capillary electrophoresis, asymmetric biotransformation, liquid-liquid extraction, membranes, sensors, chromatography and crystallization. Enantiomer separation methods, is emphasized on separation by chiral inclusion complexes and crystallization, biological methods, preparative liquid and gas chromatographic methods have been reported. Conventionally optical isomers of racemic compounds can be separated but this has always been difficult and expensive. Hence, chiral separationis usednowadays to producedual isomer recovery, as well as to generate only single-isomer recovery.

In industry, there are two main categories of techniques that are often applied for enantiomer separation, the classical methods and the modern technology.First of all the classic method, it’s the most widely used technique, is the resolution by diastereomeric salt formation. In this

method, an acid-base reaction is involved between a racemic drug and a pure single enantiomer called resolving agent. This reaction is responsible for the formation of two diastereomeric salts which have different physical and chemical properties and can be easily separated either by filtration or by crystallization if one is insoluble and the other is soluble. Finally, the pure enantiomer is obtained by salt decomposition by treatment with either acid or base. It can also be separated by classical achiral liquid chromatography. This method has been used in the resolution of -methyl-L-dopa, asparagine and glutamic acid which is widely been using clinically.[10]

Other classical method that is being used is enzymatic or kinetic resolution. In this methodology, formation of resolution is achieved with the biochemical process which destroys one enantiomeric form that causes side effect. Certain microorganisms such as yeasts, molds, bacteria can only degrade one of two isomers of a racemate by enzymatic assimilation. Whereas, the other side which is not digested remains in the solution will be isolated. Enzymatic resolution has been used in the preparation of lorafiban (benzodiazepine), levofloxacin (antibacterial drug), and S-naproxen (anti-inflammatory drug). (11)

The modern technologies, ensures the preparative high-performance liquid chromatography (HPLC) as the method of choice for the enantiomer separation. This method has proven to be one of the best methods for the direct separation and analysis of enantiomers. In chromatographic methods, indirect and direct methodologies are used. The indirect HPLC involves derivatization of samples with a chiral derivatization reagent, for an example a pure single enantiomer, resulting in the formation of two diastereomers which can be separated by a classical reversed-phase column. This indirect HPLC method is rarely used in industry, but due to its high sensitivity, it is frequently performed in biological analysis. Whereas, the direct HPLC utilizes in chiral stationary phases (CSPs) or in the mobile phase called chiral mobile phase additives (CMPA). (12)

Direct chiral separations using CSPs are more widely used and are more predictable, in mechanistic terms, than those using chiral additives in the mobile phase. Among a hundred HPLC CSPs commercially available, only some types of chiral sorbents following arepresently

the most widely used for preparative HPLC in industry. Choosing the right column for the Enantio separation of a racemic compound is difficult. The decision relies mostly on empirical data and experience. However, the understanding of the recognition mechanisms of chiral selectors with enantiomers can help the chromate graphists to resolve some problems of resolution and to economize time-consuming. (13)

According to Aboun-Enein and Ali all chiral selectors provide a chiral surface to enantiomers, which form with the selectors temporary complexes, having different bonding energies. The enantiomers differ in their binding energies because they fit differently into the chiral selector structures. Consequently, the two enantiomers can be eluted at different times by the mobile phase and then separately collected. Briefly, in general, the recognition mechanism on a chiral selector is based on a key-and-lock arrangement. However, many other factors such as mobile phase composition (pH, electrolytes, solvent nature), size and length of column, temperature, etc also play a key role for chiral resolution. (14)

There is also a new technique called simulated moving bed (SMB) chromatography is recently developed for industry. Basic concept of SMB technology is the continuous countercurrent movement of stationary and mobile phases in which the movement of a stationary phase is simulated. The small particles in this component are packed into single columns and connected to form a circle. Four external valves allow the addition and subtraction of feed and effluent. The mobile phase is pumped through the circle and when it passes the stationary phase a slight separation occurs, the less absorbable compound running in front and the more absorbable compound staying behind. When steady state is reached, the system can be operated continuously. If all flow rates and the shift time are determined correctly, raffinate and extract fractions can be withdrawn in high purity. An example of a pharmaceutical compound separated by SMB chromatography is tramadol. (16)

To avoid the racemization during chiral drug preparation, an asymmetry synthesis using chiral catalysts has been developed by W.S. Knowles, R. Noyori and K.B. Sharpless, the Nobel Prize in chemistry 2001 (17). Most of the available asymmetric chemical catalysts are organometal types including transition metals such as titanium, and noble metals such as osmium, palladium, and rhodium. Chiral catalysts are like enzymes in that both have a high degree of specificity. They allow stereospecific reactions to take place and therefore avoid the formation of racemates. L-Dopa (anti-Parkinson agent), naproxen (anti-inflammatory drug) are some examples of single enantiomer drugs produced by this catalytic asymmetric synthesis. (18)

Biological Activities of Enantiomers

The enantiomers may vary in their interactions with chiral environments such as enzymes, proteins, receptors, etc of the body. These variations may lead to differences in biological activities such as pharmacology, pharmacokinetics, metabolism, toxicity, immune response etc. Biological systems can recognize the two enantiomers as two different substances, and their interaction each other will therefore elicit different responses.

Easson and Stedman has proposed the reason for enantiomers recognition by drug receptors is a three-point interaction of the drug with the receptor site (19). In this case, one enantiomer is biologically active while the other enantiomer is not. In this case, this fitting interaction can produce an active biological effect. In addition, the inactive enantiomer cannot bind in the same way with its receptor when it rotates in space, consequently, there is no active response.

As a conclusion, the attachment of an enantiomer to the chiral receptor is similar to a hand fitting into a glove or to a key into a lock. Indeed, a right hand can only fit into a right hand glove, therefore a particular enantiomer can only fit into a receptor site having the complimentary shape. The other enantiomer will not fit, like a right hand in a left glove, but may fit into a receptor site elsewhere in the body and cause an eventual unwanted or toxic effect. On the other hand, enantiomers can show different chemical behaviour due to different chiral discrimination by diastereomeric formations with a chiral environment. (20)

Uses of Enantiomers

The enantiomers of chiral drugs are known to be exhibiting different pharmacological and pharmacokinetic activities because they interact with enzymes and receptors consisting of aminoacids andother chiral biomolecules.There are many enantiomers that are formulated as single enantiomers which having only the side of efficacy without a side effect phase.

For example,DOPA the precursor of dopamine that is effective in the treatment of Parkinson disease, was used under racemic form (d, l- dopa), but knowing to its toxicity (agranulocytosis) of d-isomer, only levoratory form called L-Dopa is being used nowadays in therapeutics. It has been renewed the interest in uses of thalidomide due to its immunomodulatory, anti-angiogenic, and anti-inflammatory effects. In addition, it has a strong inhibition in the tumor necrosis factor alpha (21).

Thalidomide has given spectacular results in the treatment of erythema nodosumleprosum, aptosis and Behcet's syndrome. Moreover, it has been assayed for organ transplantation, some autoimmune diseases such as chronic lupus erythematosus, rheumatoid arthritis, some forms of cancer, etc. Single thalidomide enantiomers and its derivative which is N-hydroxythalidomide, were also synthetized by asymmetric technique in order to study their individual biological and chemical activities (22).

(S)-albuterol is one of the single formulated enantiomer which is a selective alpha-2 adrenergic receptor agonist used in the treatments of asthma. The other example is (S)-omeperazole, the proton pump inhibitor for the treatment of gastrointestinal tract reflux[9]. Other than that, (S)-citalopram which is primarily responsible for re-uptake of serotonin and act as the antagonism. The drug that responsible for the local anesthetic effect which is (L)-propranolol is also an single formulated enantiomer. Other examples are Ethambutol for treatments of tuberculosis and Naproxen for the treatment of arthritis pain[23].

Conclusion

Drug enantiomers are widely being used clinically nowadays. Thus, the enantiomers need to undergo separation methods as this stereoisomers causes side effect which caused by the inefficacy side of the drug. The chiral separation of racemic drugs is a necessary in

pharmaceutical industry as well as in clinical therapeutics. However, the usage of a single isomer must be taken after long clinical assessments between the racemate and single enantiomer actions because in some cases, racemates have more therapeutic advantages than single isomers. It is also important to give more information about chiral drugs especially racemic form to healthcare professionals in order to help them for finding an optimal treatment and a right therapeutic control.

Corresponding Author

RevathiDuraisamy

Senior Lecturer, Department of Prosthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Contact Number: +91 7358448777

Email Id: revathygounder@yahoo.com

References

1. Davies NM, Teng XV. Importance of chirality in drug therapy and pharmacy practice. Implication for psychiatry. Advances in Pharmacy. 2003; 1(3):242–252.Davankov, Vadim A. Analytical Chiral Separation Methods. International Union of Pure and Applied Chemistry, 1997. PDF
2. Chen J, Korfmacher WA, Hsieh Y. Chiral liquid chromatography-tandem mass spectrometric methods for stereoisomeric pharmaceutical determinations. Journal of Chromatography B. 2005; 820:1–8.
3. Lorenz H, Capla F, Polenske D et al, Crystallization based separation of enantiomers, Journal of the University of Chemical Technology and Metallury 2007: 42: 5-16.
4. Bojarski J, Aboun-Enein HY, Ghanem A. What's new in chromatographic enantioseparations. Current Analytical Chemistry. 2005;1:59–97.

5. Boralli VB, Coelho EB, Cerqueira PM, Lanchote VL. Steroselective analysis of metoprolol and its metabolites in rat plasma with application to oxidative metabolism. Journal of Chromatography B.2005; 823:195–202.
6. Zaaba NAAB, Roy A. Perception of women on the adverse effect of drugs on the fetus during pregnancy. J Adv Pharm Edu Res 2017; 7(2):72-75.
7. Raikumar SV. Thalidomide:Tragedy past and promising future. Commentary. Mayo Clinic Proceedings,PubMed.2004
8. Scott, Raymond PW. "Chiral Gas Chromatography." Chromatography Books Online (free Access Pdfs). Mar. 2011.
9. Wisor, Jonathan P., William C. Dement, Lisa Aimone, Michael Williams, and Donna Bozyczko-Coyne. "Armodafinil, the R-enantiomer of Modafinil: Wake-promoting Effects and Pharmacokinetic Profile in the Rat." Pharmacology Biochemistry and Behavior 85.3 (2006): 492-99. ScienceDirect - Home. Nov. 2006.
10. Drayer DE. The early history of stereochemistry. In: Wainer IW, editor. Drug stereochemistry. Analytical methods and pharmacology. 2nd. New York: Marcel Dekker Publisher; 1993. pp. 1–24.
11. Waldeck B. Three-dimensional pharmacology, a subject ranging from ignorance to overstatements.Pharmacology and Toxicology. 2003;93(5):203–210.
12. Davies NM, Teng XV. Importance of chirality in drug therapy and pharmacy practice. Implication for psychiatry. Advances in Pharmacy. 2003;1(3):242–252.
13. Stoschitzky K, Lindner W, Zernig G. Racemic beta-blockers-fixed combinations of different drugs.Journal of Clinical and Basic Cardiology. 1998;1(1):15–19.
14. Darrow, Jonathan J. "The Patentability of Enantiomers: Implications for the Pharmaceutical Industry." Stanford Technology Law Review, 2007. Web. 7 Mar. 2011.
15. Matthijs N, Maftouh M, Heyden YV. Screening approach for chiral separation of pharmaceuticals. IV. Polar organic solvent chromatography. Journal of Chromatography B. 2006;830:114–119.
16. Murphy-Poulton SF, Boyle F, Gu XQA, Mather LE. Thalidomide enantiomers: Determination in biological samples by HPLC and vancomycin-CSP. Journal of Chromatography B. 2006;831:48–56.

17. Staroverov SM, Kuznetsov MA, Nesterenko PN, Vasiarov GG, et al. New chiral stationary phase with macrocyclic glycopeptide antibiotic eremomycin chemically bonded to silica. Journal of Chromatography A.2006;1108:263–267.
18. Bhupinder Singh Sekhon, Enantioseparation of chiral drugs-an overview, International Journal of PharmTech Research 2010 2(2).
19. Mann A, Wermuth CG. Prix Nobel de Chimie 2001: William S. Knowles, RyoriNoyori, K. Barry Sharpless. La catalyseasymetrique. Medicine/Sciences. 2001; 17:1230–1232. (French)
20. Soh NHBC, Roy A. Stress, anxiety, and depression in clinical practice of undergraduates and awareness of its effective management -A survey. JAdv Pharm Edu Res 2017; 7(2):82-85.
21. Harrower ADB, Fyffe JA, Horn DB, Strong JA. Thyroxine and triiodothyronine levels in hyperthyroid patients during treatment with propranolol. Clinical Endocrinology. 1977; 7:41–44.
22. Echizen H, Manz M, Eichelbaum M. Electrophysiologic effects of dextro- and levo-verapamil on sinus node and AV node function in humans. Journal of Cardiovascular Pharmacology. 1988; 12:543–546.
23. Barrett A, Cullum C. The biological properties of the optical isomers of propranolol and their own cardiac arrhytmias. British Journal of Pharmacology. 1968; 34:43–55.

Influence of Aerosol in Dental Office

Benazir Hussain M[1], Muralidharan NP[2], Sivakumar M[3]

Graduate Student[1], Reader, Department of Microbiology, Senior Lecturer[3], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Aerosol is a suspension of solid or liquid particles in air or other gaseous environment. Sources of bacterial aerosols exist within and outside the dental clinic. The generation of bacterial aerosols and splatters appears to be highest during dental procedures. The use of rotary dental and surgical instruments and air-water syringes generates visible infectious spray that encloses large-particle splatter of water, saliva, microorganisms, blood, and other debris. Air-water aerosol produced during dental treatment procedures emerges from a patient's mouth and mixes with the surrounding air, thus influencing its composition. The air contained in this space is the air breathed by both dentist and patient, its composition is extremely important as a potential threat to the dentist's health as well as to the patient and the dental assistant. From an infectious point of view, dentistry has never been safer than it is today for both patients and dental team. The aerosols and splatter generated during dental procedures have the potential to spread infection to dental personnel and other people in the dental office. While, as with all infection control procedures, it is impossible to completely eliminate the risk posed by dental aerosols, it is possible to minimize the risk with relatively simple and inexpensive precautions with immunization protocols requires attention. CDC and ADA protocol should be followed with at most consciousness to minimize the risk among the patients and the dentists.This review is done to recapitulate the formation of dental bio aerosols, its hazardous nature to the clinician, patient and the assistant and also the protective measure need to be taken against it thereby preventing it.

Key Words: Dental aerosol, splatter, dental office, infectious disease, ultra sonic scaling, pre-procedural rinses

Introduction

The spread of infection through aerosol and splatter has long been considered one of the main concerns in the dental community. Even before the discovery of specific infectious agents such as bacteria and viruses, the potential infection by the airborne route was recognized. A number of sources of bacterial aerosols exist within and outside the dental clinic. Dentists use high-energy equipment, such as drills and scalars, in the presence of bodily fluidssuch as blood, saliva and dental plaque. Thiscombination has been shown to generate aerosols of oral micro-organisms, and blood. Combined effect, referred as Bio-aerosols present a considerable microbial challenge to the patients, the dentist and nursing staff. Several infectious diseases could be transmitted to staff and patients by airborne bacterial and other contaminants in the dental clinic.

Aerosols are defined as liquid or solid particles suspended in the air by humans, animals, instruments, or machines. Bio-aerosols are aerosols consisting of particles of any kind of organism. [1,2] Aerosols, which are responsible for the transmission of airborne micro-organisms by air, consist of small particles named droplet nuclei (1–5μm) or droplets (>5μm). Splatter is usually described as a mixture of air, water and/or solid substances. Water droplets in splatter are from 50 micron to several millimeters in diameter and are visible to the naked eye. Droplets nuclei can stay airborne for hours, transport over long distances and contaminate surfaces by falling down [1] Humans generate bio-aerosols by talking, breathing, sneezing or coughing [1]. Based on the infectious status of a person, the bio-aerosols are proven to contain influenza or rhinoviruses[3,4] Mycobacterium tuberculosis [5], Staphylococcus aureus, Varicella Zoster Virus, Streptococcus species or Aspergillus species. [6] It is inferred from one of the study that the spread of aerosols was maximum at 2 feet distance in all directions irrespective of the environmental conditions. Whereas it was a minimal to negligible spread at 6 feet distance in all directions under all environmental conditions simulated. There was no color change in litmus paper after 6 feet, which indicates that the minimum safe distance that has to be followed around a dental chair is 6 feet.[7]

In this article, we review relevant literature that has addressed the presence and makeup of dental aerosols and splatter. We also assess the threats that may be inherent in this airborne material, including risk potential to patients and the dental team. We make recommendations for the control of dental aerosols and splatter.

Contents of Aerosol

The terms “aerosol” and “splatter” in the dental environment were used by Micik and colleagues [8,9] aerosols were defined as particles less than 50 micrometers in diameter. Particles of this size are small enough to stay airborne for an extended period before they settle on environmental surfaces or enter the respiratory tract. The smaller particles of an aerosol (0.5 to 10 µm in diameter) have the potential to penetrate and lodge in the smaller passages of the lungs and are thought to carry the greatest potential for transmitting infections. Splatter was defined by Micik and colleagues as airborne particles larger than 50 µm in diameter. Micik and colleagues stated that these particles behaved in a ballistic manner. This means that these particles or droplets are ejected forcibly from the operating site and arc in a trajectory similar to that of a bullet until they contact a surface or fall to the floor.

Composition of aerosols probably varies with each patient and operative site. However, it is reasonable to suppose that components of saliva, nasopharyngeal secretions, plaque, blood, tooth components and any material used in the dental procedure, such as abrasives for air polishing and air abrasion, all are present in dental aerosols. Several recent studies have analyzed the presence of blood components in dental aerosols.[10,11]

Aerosols may accommodate micro organisms like multidrug resistant Staphylococcus aureus, influenza, legionnaire’s disease, pneumonia, mumps, chicken pox, cytomegalovirus infection, hepatitis B and C virus infection, herpes simplex virus Types 1 and 2 infection, human immunodeficiency virus etc [12].They may also harbor mycotoxins, fungal cells such as molds, spores, yeasts, saliva, blood, protozoa and dead cell debris.

Dental Procedure That Generates Aerosol

The source of dental aerosol during dental procedures includes:

1. Patient

Dental aerosol can be produced from the patients. The amount of contamination of dental aerosol depends on the quality of saliva, nasal and throat secretion, blood, dental plaque, periodontal infection, blood and presence of any dental infection.[13] Therefore aerosol composition differs from patient to patient depending on the site and type of the procedure like tooth preparation and oral prophylaxis. [13, 14]

2. Dental Unit Waterline

The tubing in DUWL is constructed in such a way that, the centre of the lumen has the maximum flow of water and the periphery has the minimal flow. Reasons of contamination of DUWL may be due to narrow bore water lines, water stagnation, heating of dental chair unit, anti retraction valve failure and contamination of reservoir bottles. [15] Many environmental organisms identified in dental treatment water have been associated with opportunistic infections in hospitalized or immune compromised patients. For example, Pseudomonas species, non-tuberculous mycobacteria, and Legionella species all have been isolated from dental unit water. Legionella, the causative agent of Legionnaires' disease, may pose a particular concern, as it appears to be transmitted by inhaling aerosols or aspirating water contaminated with the bacteria. Biofilm is a community of bacterial cells and other microbes that adhere to surfaces and form a self protective slime layer. Found in virtually all places where moisture meets a suitable solid surface, biofilm can contain many types of bacteria as well as fungi, algae, protozoa, and nematodes. Although biofilm can form in all non-sterile fluid environments, dental waterlines provide particularly well-suited conditions. One article reports the death of an 82-year-old Italian woman died in February due to complications related to Legionnaires’ disease, a life-threatening type of pneumonia caused by Legionella bacteria, which she acquired while receiving dental care. In the Dimensions of Dental Hygiene Journal, [16] the elderly woman was hospitalized after becoming ill with a fever and respiratory distress.

3. Instruments

Instruments used for dental procedures includes: ultrasonic scalars, high speed hand pieces, air turbines, three in one syringes, and air water syringes. Most dental procedures that use mechanical instrumentation will produce airborne particles from the site where the instrument is used. Dental hand pieces, ultrasonic scalars, air polishers and air abrasion units produce the most visible aerosols. Each of these instruments removes material from the operative site that becomes aerosolized by the action of the rotary instrument, ultrasonic vibrations or the combined action of water sprays and compressed air. The water spray usually is the portion of the aerosol that is most visible to the naked eye and is noticed by the patient and dental personnel. One study, however, showed that when an ultrasonic scalar was used in vitro without any coolant water, there still was a large amount of aerosol and splatter formed from small amounts of liquid placed at the operative site to simulate blood and saliva.[18]

Investigations have evaluated the presence or absence of blood contamination in the aerosols produced during root planning when an ultrasonic scalar is used. [10, 11] These studies have shown that blood is present universally in ultrasonic scalar aerosols during root planning. While the presence of blood has not been directly studied, it would seem logical that blood also would be present in any dental aerosol that is produced by an instrument in a blood-contaminated field. This would include restorative procedures that extend subgingivally, as well as periodontal and oral surgery procedures.

4. Ventilation

There is little evidence that ventilation directly reduces the risk of disease transmission, but many studies suggest that insufficient ventilation increases disease transmission(19-21). Detection of pathogens in room air and buildings may suggest a possible, indirect association between ventilation and disease transmission [22] Lack of ventilation or low ventilation rateis associated with increased infection rates or outbreaks of airborne diseases. No information exists on the impact of ventilation rate on transmission of droplet-transmitted diseases. This agrees with the physics of droplet transmission, which shows that general ventilation should not affect large droplet transmission.A higher ventilation rate can provide a higher dilution capability and consequently potentially reduce the risk of airborne infections. [23]

Table1: Dental devices and procedures known to produce airborne contamination:

[13,24,25]

Ultrasonic and Sonic Scalars	Considered the greatest source of aerosol contamination; use of a high-volume evacuator will reduce the airborne contamination by more than 95 percent
Air Polishing	Bacterial counts indicate that airborne contamination is nearly equal to that of ultrasonic scalars; available suction devices will reduce airborne contamination by more than 95 percent
Air-Water Syringe	Bacterial counts indicate that airborne contamination is nearly equal to that of ultrasonic scalars; high-volume evacuator will reduce airborne bacteria by nearly 99 percent
Tooth Preparation With Air Turbine Hand piece	Minimal airborne contamination if a rubber dam is used
Tooth Preparation With Air Abrasion	Bacterial contamination is unknown; extensive contamination with abrasive particles has been shown

Susceptible Areas of Contamination

1) The dental clinician

2) The assistant

3) Patient

4) Dental instruments, dental handpieces along with burs, Ultrasonic scaler tips, Polishing cups

Risks of Aerosol Contamination

The role of the air as a carrier of infection is seen in Tuberculosis, measles, severe acute respiratory syndrome, multi-resistant Staphylococcus aureus, influenza, legionnaires disease, pneumonia, mumps, chicken pox, cytomegalovirus infection, hepatitis B and C virus infection, herpes simplex virus Types 1 and 2 infection, human immunodeficiency virus, etc. The dispersion of bacteria and viruses (reservoir being mouth with saliva, blood, sub gingival fluids, and moisture from the nasopharynx) into the air due to coughing, sneezing, general exhalation, and exacerbated by the use of the high-speed instruments such as ultrasonic scalars and hand pieces starts the chain of airborne infection. During dental treatment, several thousand droplets are aerosolized. The larger droplets fall quickly to the floor and onto other surfaces, the smaller droplets evaporate quickly, leaving dry microscopic droplet nuclei which remain suspended in the air for extended periods of time and require prolonged hours to settle down. The bacteria and viruses become highly mobile in the air and circulate from one room to another by convection currents. Microorganisms in excess of 5 times that of outdoor air are present in dental surgery units. [12]

Periodontitis is an inflammatory disease of the supporting tissue of the teeth caused by specific microorganisms or groups of specific microorganisms resulting in progressive destruction of the periodontal ligament and the alveolar bone with pocket formation, recession or both [26]. Dental plaque being the prime etiological agent comprises complexes of microorganisms, both bacterial and viral origin in the gelatinous matrix. It gets dispersed when mechanically acted upon pressure of food, friction of soft tissues or dislodgement by the ultrasonic devices [27]. Therefore the aerosols produced by ultrasonic scalar units are heavily contaminated by microorganisms which have the capability of disease transmission leading to various diseases like mild flu, pneumonia [28, 29 & 30] streptococcal and staphylococcal infections, viral infection, conjunctivitis [31], amoebic keratitis [32], tuberculosis and severe acute respiratory syndrome (SARS). Blood borne pathogens like HIV, HBV, and HCV can be transmitted through the inhalation of blood containing aerosol via the microlesion in the mucosa of the airways which acts as the potential access for such viruses. [33, 34] It can also contaminate the nearby instruments on the instrument trays which can further act as a source of infection to the patient

Contamination from dental instrumentation is the result of organisms on instruments and in DUWLs. Routine cleaning and sterilization procedures should eliminate contamination of all dental instruments except those being used with the current patient. The use of ADA-recommended methods to treat the DUWL also should minimize or eliminate airborne contamination from the DUWL, because contamination from these sources is controlled relatively easily by standard procedures.Snophia S et al, in 2011 reported two patients contaminated with Pseudomonas Aerugiosa when treated in a dental clinic, where DUWL was the source of infection. The microorganism which was isolated from the oral abscess developed in these patients was the same strain isolated from the DUWL [35]. Other respiratory infections reported were mild flu and pneumonia, which was caused by legionella pneumophilia, non pneumophillaspecies and mycobacterium species including mycobacterium avium, staphylococcal and streptococcal infection. [31, 29, 30]

The most serious potential threat present in aerosols is M. tuberculosis, the organism that causes TB. In the past, TB was viewed as an occupational hazard of dentistry.[36, 37] Patients known to have active TB should be treated using special respiratory precautions so that the aerosols produced during treatment can be controlled. Patients with undiagnosed, active, infectious TB, however, remain a risk for the dental team and other patients.

The saliva and nasopharyngeal secretions also may contain other pathogenic organisms. These may include common cold and influenza viruses, herpes viruses, pathogenic streptococci and staphylococci, and the SARS virus. The use of universal precautions with all patients initially was based on the assumption that all patients may have an infectious blood borne infection, such as with hepatitis B virus, hepatitis C virus and HIV. It also should be assumed that all patients may have an infectious disease that has the potential to be spread by dental aerosols; thus, universal precautions to limit aerosols also should be in place.Recent studies have highlighted the spread of infection through the air resulting from the most intensive aerosol and splatter emission that occur from an ultrasonic scalar tip and bur on a high-speed hand piece. [38, 39]Basu et al. [40] did a survey of aerosol-related symptoms in dental hygienists using ultrasonic scalars. They found that symptoms such as nasal irritation, persistent cough, runny eyes, and itchy and dry skin were more common in dental hygienists than in nurses and hospital staff.

Dental prostheses should be disinfected before they are sent to the laboratory and upon return to the dental clinic [41] but, despite rigorous control of sterilization and disinfection of instruments in dental clinics; prosthetic appliances do not receive adequate infection control. Potentially pathogenic microorganisms, such as Gram-negative bacilli of the genus Acinetobacter, as well as Micrococcus, Pseudomonas, Moraxella and Alcaligeneshave been detected contaminating pumice in commercial laboratories [42, 43]. These bacteria, which are not part of normal oral flora, can cause serious diseases if passed to patients whose dentures are polished with contaminated material and to the technician by exposure to contaminated aerosol. Williams et al. [44] reported, in 1985, the increase of cases of pneumonia in individuals exposed to lathe aerosol. Sande et al. [45] reported 10 cases of infection by Mycoplasma pneumoniae involving persons working in dental prosthetic laboratories, suspecting that these infections derived from manipulation of prostheses contaminated by these microorganisms. Therefore, the use of aprons, gloves and protective glasses by the professionals should be a routine. [46]

The dentist's face is at high-risk of infection transmission. During dental practice, central areas of the face such as inner part of the eyes and around the nose were most contaminated areas. These parts are the important areas for transmission of infection. Among occupational threats for this group, transmission of infections especially viral diseases such as Hepatitis B, Hepatitis C and acquired immune deficiency syndrome is more critical. [47] World health organization has reported 2.5% of human immunodeficiency virus (HIV) and 40% of Hepatitis B and Hepatitis C infections in healthcare staff by occupational contacts. [44]Comparing prosthetic and periodontal procedures, the latter caused more contamination in the present study. This could be because of more bleeding and soft-tissue irritation during scaling. [49] Water droplets in splash are from 50 μm to several millimeters in diameter and move 15-120 cm from a patient's oral cavity.[38, 50, 39] Splatter shows limited penetration into the respiratory system,[50] but can come into contact with the mucosa of nostrils, open mouth, eyes, and skin. They are deposited on hair, clothes and in the immediate surroundings of the splatter source.

Allergens and Irritants in Dental Clinic Through the Use of Volatile Chemical and Dust Particles Generated

The work at a dental clinic implies getting in contact with different chemical substances. The chemical substances may originate from dental materials, such as mercury from amalgam and monomers from resin based materials. The substances may be released during preparation, polishing and removal of restorations. Visual field constriction related to mercury exposure is reported.[51] Color vision examination has been shown as a sensitive indicator of subtle neurotoxic effects from exposure to solvents and heavy metals.[51]The dusts generated during routine procedures by dental technicians such as mixing powders, using silica sand for abrasive blasting,trimming and polishing of acrylic dentures and orthodontics appliances can't be avoided. Research has shown us that the dust from the types of materials that are used in dental technology contain between 54-70 per cent of respirable particles (that is, particles of less than 5 microns in size). A particular concern is the silica content of this dust, which can reach 30 per cent and may exceed maximum recommended levels during the sandblasting and grinding processes. Another is exposure to dusts from heavy metals like the cobalt-chromium-molybdenum alloys. Both exposures may lead to a lung condition known as pneumoconiosis, of which several cases have been reported as being linked to dental technology. [52] The damage can be prevented by measures such as the use of high-volume evacuation reducing spatter and aerosol formation, "no touch-technique" and proper use of personal protective equipment in the form of protective gloves and eyewear and surgical masks.

Among the risks which are fatal includes tuberculosis (TB) and severe acute respiratory syndrome (SARS). Splash of blood to the eyes, nose or mouth occurs frequently during dental procedure. The risks of transmission of HBV, HCV and HIV as a result of injury with a contaminated needle are approximately 30% (HBV when the source is e-antigen positive), 3% (HCV) and 0.3% (HIV). (Table 2) Cases reported so far, hypothesized that blood borne pathogens like HIV, HBV, and HCV can be transmitted through the inhalation of blood containing aerosol via the microlesion in the mucosa of the airways which acts as the potential access for such viruses [54].

Table 2: Risk to dental surgeon and patients through aerosol

Condition	Habitat	Routes of transmission
1. Common cold	Upper respiratory tract	Aerosol, contact
2.Sinusitis	Upper respiratory tract	Aerosol, droplet
3.Pharangitis	Upper respiratory tract	Aerosol, droplet
4.Pneumonia	Respiratory tract	Aerosol, droplet
5.Tuberculosis	Respiratory tract	Aerosol, droplet
6.SARS	Respiratory tract	Aerosol, droplet, intimate contact
7.Avian influenza(H5N1 flu)	Respiratory tract	Aerosol, droplet, intimate contact
8. Avian influenza(swine flu)	Respiratory tract	Aerosol, droplet, intimate contact

Barrier Techniques Used To Reduce the Risk of Contamination

Universal Barrier Precaution

When the patient enters the dental clinic, every attempt should be made from the dentist's end to avoid cross contamination. A thorough case history, clinical examination and all the necessary investigations should be carried out. The dental team should be vaccinated, protective equipment which include gloves, gowns; face shields, hair protection, mouth masks, and goggles/eye wear are the other preventive steps to be taken.

Immunization of Dental Personal

Immunization of dental personnel against Hepatitis A [39], Hepatitis B [39,55], Influenza, Mumps, Measles, Tetanus, Rubella, Tuberculosis, whooping cough [55], Varicella, MMR , DPT, Rubeola, Meningitis, Polio and Diptheria for infection control should be done at proper periodic intervals.

Pre Procedural Mouth Rinse

One of the study demonstrated that the microbial load in the saliva was reduced in both chlorhexidine and herbal group (p value = <0.05). It was found that chlorhexidine mouthwash could reduce the bacterial load to 99.91% in saliva while herbal mouthwash could reduce the bacterial load to only 58.27%. Hence chlorhexidine mouthwash has better antibacterial activity than herbal mouthwash. However the percentage reduction of the bacterial load in the aerosol in chlorhexidine group was much higher in comparison with the herbal group. Therefore chlorhexidine is proved to be better than herbal mouthwash. The reason to this could be due to better penetration of chlorhexidine to dental plaque. [56] Hence Pre-procedural rinses with water and 0.12 to 2% chlorhexinegluconate or essential oil containing mouthwashes for duration of 60 seconds can cause substantial reduction in bacterial counts.

Use of Rubber Dam

During many dental procedures, the use of a rubber dam will eliminate virtually all contamination arising from saliva or blood. If a rubber dam can be used, the only remaining source for airborne contamination is from the tooth that is undergoing treatment. This will be limited to airborne tooth material and any organisms contained within the tooth itself.

Use of High Volume Evacuator

The use of a high-volume evacuator, or HVE, has been shown to reduce the contamination arising from the operative site by more than 90 percent. During restorative dentistry, the HVE often will be used by an assistant who is able to guide and aim the vacuum in a manner that eliminates or greatly reduces the visible water spray produced during dental procedures. It has been shown that the number of CFUs produced during dental procedures is reduced greatly when an assistant uses an HVE.[57]

Instrument Sterlization

Patient-care items are categorized according to Spaulding classification system as critical, semi-critical, or noncritical, depending on the potential risk for infection associated with their intended use.[58, 59] The biological indicators (spore strips of Bacillus stearotheromophillus) must be checked for every sterilization cycle and if not then at least once in a week with physical and

chemicals methods of monitoring of sterilization cycles. Maintain the record of all these monitoring systems.

Disinfection

Prostheses, inter treatment materials and non-sterilizable equipment must be cleaned with soap and water and disinfected with a hospital-level disinfectant if they become contaminated. If ultrasonic cleaners are used for cleaning or the disinfecting step, care must be taken not to overheat the material or disinfectant while in the ultrasonic cleaner. Spraying or soaking these items in the disinfectant in a separate container or bag is the method of choice[60].

Laboratory Asepsis

On the polishing lathe the pumice solution should be made by suspending the pumice in tincture of green soap or other surfactant and possibly adding an effective disinfectant solution to the mix. [61]This will prevent colonization from airborne and other organisms that may find themselves in the warm wet pumice environment. The pumice should be changed daily and the machine should be cleaned and disinfected daily. Bench tops and work areas should be cleaned daily or. Surface disinfection protocols are the same in a Dental laboratory as that in a dental clinic. [61]

Conclusion

From an infectious point of view, dentistry has never been safer than it is today for both patients and dental team.The aerosols and splatter generated during dental procedures have the potential to spread infection to dental personnel and other people in the dental office. While, as with all infection control procedures, it is impossible to completely eliminate the risk posed by dental aerosols, it is possible to minimize the risk with relatively simple and inexpensive precautions with immunization protocols requires attention. CDC and ADA protocol should be followed with at most consciousness to minimize the risk among the patients and the dentists.

Corresponding Author

Sivakumar M

Senior Lecturer, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India.

Telephone Number- +919443602627

Email id: drsivaomfs@gmail.com

References

1.Ashrae.position document on airbone infectious diseases. 2014;100:102-405

2. Barker J, Jones MV. The potential spread of infection caused by aerosol contamination of surfaces after flushing a domestic toilet. Journal Applied Microbiology. 2005;99(2):339–47.

3. Brankston G, Gitterman L, Hirji Z, Lemieux C, Gardam M. Transmission of influenza A in human beings. The Lancet infectious diseases. 2007;7(4):257–65.

4. Tellier R. Aerosol transmission of influenza A virus: a review of new studies. Journal of the Royal Society Interface. 2009;6 Suppl 6:S783–90.

5. WHO. Infection prevention and control measures for acute respiratory infections in healthcare settings: An update. 2007:39–47.

6. Gralton J, Tovey E, McLaws ML, Rawlinson WD. The role of particle size in aerosolised pathogen transmission: a review. Journalof Infection. 2011;62(1):1–13.

7. Sandeepchiramana, et al. Evaluation of minimum required safe distance between two consecutive dental chairs for optimal asepsis.10.5005/jp-journals-10026-1056

8.Micik RE, Miller RL, Leong AC. Studies on dental aerobiology, 3: efficacy of surgical masks in protecting dental personnel from airborne bacterial particles. Journal of Dental Research 1971;50:626-30.

9. RE, Abel LC, Miller RL, MicikRyge G. Studies on dental aerobiology, IV: bacterial contamination of water delivered by dental units. Journal ofDental Research 1971;50:1567-9

10. King TB, Muzzin KB, Berry CW, Anders LM. The effectiveness of an aerosol reduction device for ultrasonic scalars. Journal Periodontology 1997;68(1):45-9.

11. Logothetis DD, Gross KB, Eberhart A, Drisko C. Bacterial airborne contamination with an air-polishing device. Academy of General Dentistry 1988;36: 496-9.

12.Yadal. HEPA filters and control of air borne infections, SRM Journal of Research in Dental Sciences | Vol. 6 | Issue 4 | October-December 2015.

13. Zymańska J, Dental bioaerosol as an occupational hazard in a dentist's workplace.Annals Agricultural Environmental Medicine. 2007, 14(2), 203-207

14. YogeshwariSwaminathan, Julie Toby Thomas. "Aerosol"-A Prospective Contaminant of Dental Environment.Saveetha dental college and hospital.IOSR Journal of Dental and Medical Sciences. 2013:11;2;45-50.

15. Mary J O, Maria A B, Ronnie J R, David CC, Management of dental unit waterline biofilms in the 21st century, Future microbiology, 6(10), 2011, 1209-1226

16. Raymond britto.P, N.P. Muralidharan. Contamination of Dental waterline and its control measures. Asian journal of pharmaceutical and clinical research.2013:4;6(12)

17. Oshafor dentistry, Jeannie cordova,2011:3;13.

18. Harrel SK, Barnes JB, Rivera-Hidalgo F. Aerosol and splatter contamination from the operative site during ultrasonic scaling. JADA 1998;129:1241-9.

19. Miller RL, Micik RE. Air pollution and its control in the dental office. Dental Clinics of North America. 1978;22:453-476.

20. Kahn RC, Lancaster MV, Kate Jr W. The microbiologic cross-contamination of dental prostheses. Journal of Prosthetic Dentistry. 1982;47:556-559.

21. Fisher WT, Chandler HT, Brudvik JS. Reducing laboratory contamination. Journal of Prosthetic Dentistry. 1972;27:221-225.

22. Artenstein MS, et al. Large-volume air sampling of human respiratory disease pathogens. American Journal of Epidemiology. 1967;85(3):479–485.

23. Atkinson J, Chartier Y, Pessoa-Silva CL, et al. Natural Ventilation for Infection Control in Health-Care Settings. Geneva: World health organisation; 2009.

24. Bentley CD, Burkhart NW, Crawford JJ. Evaluating spatter and aerosol contamination during dental procedures.Journal of American Dental Association. 1994;125(5):579-584.

25. Al Maghlouth A, Al Yousef Y, Al Begieh N. Qualitative and quantitative analysis of bacterial aerosols. Journal of Contemporary Dental Practices. 2004;5(4):91-100.

26. Newman MG, Takei HH, Klokkevold PR. Caranza'sClincal Periodontology. 10th ed. Los Angeles , California: Elsevier Inc Inc; 2006.

27. Shanti priya Reddy, M.G.S Prasad, Sanjay Kaul, K.Satish, SabanaKakaral, Nirjhar Bhowmik: Efficacy of 0.2%tempered chlorhexidine as a pre-procedural mouth rinse: a clinical study. J Indian Soc Periodontol 2012; 16(2): 213 – 217.

28. CarolineL, Pankhurst, NW Johnson: Microbial contamination of dental unit waterlines: the scientific arguments. Int Dent J 1998; 48(4): 359-368.

29. Patricia MM, Alexandrine C, Cristina P, Helder O, Maria CM: Air quality assesmnet during dental practice: Aerosol bacterial counts in an university clinic. Rev Port Estomatol Med Dent Cir Maxilofac 2013; 54(1): 2-7.

30. Rautemaa R, Nordberg A, Wuolijoki S K, Meurman JH: Bacterial aerosol in dental practice-a potential hospital infection problem. J of hospital infection 2006; 64(1): 76-81

31.Seetharam KD, Sudheep N: Aerosols: a concern for dentist. Ind J of Dental Advancement 2011; 2(1): 100-102.

32.Williams J F, Johnston A M, Johnson B: Microbial contamination of dental unit waterlines: prevalence, intensity and microbial characteristics. JADA 1993; 124(10): 59–65.

33. Maria LC, Anna MS, Marina S, Maurizio D, Gianluca O, Roberto L, Fernanda P: Evaluation of risk of infection through exposure to aerosols splatters in dentistry. Am J Infect Control 2008; 36(4): 304-307.

34. Lucia B, Ioan D, Infection Control in Dentistry –present requirements: OHDMBSC 2003;2(4).

35.Snophia S, M.Manimegalai, Uma S, Sopia, Comparison of efficacy of preprocedural rinsing with chlorhexidine and essential oil mouth in reducing viable bacteria in dental aerosols- a microbiological study, Int J of Contemporary Dentistry, 2(6), 2011 Dec, 1- 6

36. Smith WH, Davies D, Mason KD, Onions JP. Intraoral and pulmonary tuberculosis following dental treatment. Lancet 1982;1:842-4

37. Belting CM, Haberfelde GC, Juhl LK. Spread of organisms from dental air rotor. JADA 1964;68:648-51.

38.Leggat PA, Kedjarune U. Bacterial aerosols in the dental clinic: A review. Int Dent J 2001;51:39-44.

39.Szymanska J. Dental bioaerosol as an occupational hazard in a dentist's workplace. Ann Agric Environ Med 2007;14:203-7

40.Basu MK, Browne RM, Potts AJ, Harrington JM. A survey of aerosol-related symptoms in dental hygienists. J Soc Occup Med 1988;38:23-5

41.The Council on Dental Therapeutics, The Council on Prosthetic Services and Dental Laboratory Relations. Guidelines for infection control in the dental office and the commercial dental laboratory. J Am Dent Assoc 1985;110:969-972.

42.Katberg Jr JW. Cross-contamination via the prosthodontic laboratory. J Prosthet Dent 1974;32:412-418.

43. Williams HN, Falkler Jr WA, Hasler JF. Acinetobacter contamination of laboratory dental pumice. J Dent Res 1983;62:1073-1075.

44. Williams HN, Falkler Jr WA, Hasler JF, Libonati JP. The recovery and significance of nonoral opportunistic pathogenic bacteria in dental laboratory pumice. J Prosthet Dent 1985;54:725-730.

45. Wenzel RP. Point source epidemic of Mycoplasma pneumoniae infection in a prosthodontics laboratory. Am Rev Respir Dis 1975;112:213-217.

46. Henderson CW, Schwartz RS, Herbold ET, Mayhew RB. Evaluation of the barrier system, an infection control system for the dental laboratory. J Prosthet Dent 1987;58:517-521.

47.Ayatollahi J, Ayatollahi F, Ardekani AM, Bahrololoomi R, Ayatollahi J, Ayatollahi A, et al. Occupational hazards to dental staff. Dent Res J (Isfahan) 2012;9:2–7.

48. Geneva: WHO; 2002. World Health R. Reducing Risks, Promoting Healthy Life; pp. 1–167.

49.The functions of saliva.Mandel ID.J Dent Res. 1987 Feb; 66 Spec No():623-7.

50.Bennett AM, Fulford MR, Walker JT, Bradshaw DJ, Martin MV, Marsh PD. Microbial aerosols in general dental practice.Br Dent J. 2000 Dec 23; 189(12):664-7.

51. JamshidAyatollahi, et al. Occupational hazards to dental staff.Dent Res J (Isfahan). 2012 Jan-Mar; 9(1): 2–7.

52. The Dangers of Dust in the Dental Laboratory.Allergy ,Jpsr. 2013;13:100-203

53. McCarthy GM, Koval JJ, MacDonald JK. Occupational injuries and exposures among Canadian dentists: the results of a national survey.1999; 20:3316.

54. Puttaiah R, Youngblood D, Verma M, Reddy A. The rationale for dental safety.World Journal of Dentistry. 2010;1: 129-134.

55. Dutch Working Party on Infection Prevention, Infection Prevention in dental practice (2012)

56. Yogeshwari swaminathan, Dr julie Toby Thomas, N.P Muralidharan. The efficacy of pre-procedural mouth rinse of 0.2% chlorhexidine and commercially available herbal mouth containing salvadora persica in reducing the bacterial load in saliva and aerosol produced during scaling.Asian journal of pharmaceutical and clinical research. 2014:21;31-89

57.Micik RE, Miller RL, Mazzarella MA, Ryge G. Studies on dental aerobiology, I: bacterial aerosols generated during dental procedures. J Dent Res 1969;48(1):49-56

58. Spaulding EH. Chemical disinfection of medical and surgical materials [Chapter 32]. In: Lawrence CA, Block SS, eds. Disinfection, sterilization and preservation. Philadelphia, PA: Lea &Febiger, 1968:517--31.

59. DM Kohn WG, Collins AS, Cleveland JL, Harte JA, Eklund KJ, Malvitz. Guidelines for infection control in dental health-care settings.MMWR. 2003;52:1-61

60. Dr Julie George Alapatt1 , Dr Neenu Mary Varghese2 , Dr Joy P T3 , Dr Mohamed Saheer K4 , Dr Bennett AtlinCorreya. Infection Control In Dental Office: A Review. IOSR Journal of Dental and Medical Sciences. 2008:23;100-302

61. Infection control recommendations for the dental office and the dental laboratory. ADA Council on Scientific Affairs and ADA Council on Dental Practice. J Am Dent Assoc 1996;127:672-80.

Intravenous Immunoglobulin Therapy in Myasthenia Gravis – A Review

Rebecca Rachel George[1], Naufal Rizwan[2], Ravindra Kumar Jain[3]
Graduate Student[1],Senior Lecturer[2],Department of General Medicine
Reader[3],Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Myasthenia gravis is an autoimmune disorder characterized by antibody meditated destruction of acetylcholine receptors at the neuromuscular junction. Treatment with intravenous immunoglobulin (IVIg) has been shown to have fewer side effects and should therefore be considered as a promising therapy in the treatment of myasthenia gravis. This review aims to recognize the potential of intravenous immunoglobulins in treating myasthenia gravis.

Key Words*: intravenous immunoglobulins, myasthenia gravis, autoimmune.*

Introduction

Myasthenia gravis is an autoimmune disorder of neuromuscular transmission associated with a deficiency of acetylcholine receptors. Current therapies that influence the immune response include: thymectomy, corticosteroids, non steroidal immunosuppression and plasmapheresis.Treatment of neurological disorders by means of intravenous immunoglobulin (IVIg) is an increasing feature of our practice for an expanding range of indications. For some there is evidence of benefit from randomized controlled trials, whereas for others evidence is anecdotal.The management of patients with myasthenia gravis usually involves a combination of these therapies to produce an adequate response[1-3]. Such a combination exposes the patient to asignificant level of toxicity. However, treatment with intravenous immunoglobulin (IVIg) hasshown fewer side effects and shouldtherefore be considered a promising therapy in the treatment ofmyasthenia gravis.

Myasthenia Gravis

Myasthenia gravis is adisorder of neuromuscular transmission that is antibody mediated and that leads to loss of functional acetylcholine receptors at the endplate and consequentfatiguable muscle weakness. This disease is characterized by weakness and fatigability of voluntary muscle changing over shorter or longer periods. Acute exacerbations are life-threatening because of swallowing difficulties or respiratory failure.The prevalence of myasthenia gravis among middle-aged and older patientshas increased. The disease occurs, for the most part, in the third decade, and is rare before the age of 15, or after 70. In approximately 60 percent of the patients the disease develops between the ages of 20 and 40[4]. Recent studies have shown an increased prevalence of the disease among middle-aged and older patients[5-8]. This may be a result of both an improved prognosis among elderly patients.The need for universally accepted classifications, grading systems, and methods of analysis for patients undergoing therapy for myasthenia gravis is widely recognized and is particularly needed for therapeutic research trials.

Natural History of Myasthenia Gravis

Myastheniagravisistwiceascommoninwomenasinmen.Theratioishigherinthefirstthreedecadesandreversesinthesixthdecade,beingmorecommoninmalesinthelaterpartoflife,sothatthemeanageatonsetofthediseaseisslightlylessinwomen(26yr.)thaninmen(31yr.),butthemodalageisabout20yearsforbothsexes.Thereisptosis(oftenasymmetrical),diplopia,facialweaknesswithatypicalvertical'snarl'onshowingtheteeth.Weaknessofthejawandneckmusclescausethecharacteristicpostureofthemyasthenic,sittingsupportingheadandjawwithherhand.Thelimbandtrunkmusclesmaybeweak.Weaknessoftheton gueandlaryngealmusclescausesdysarthria.Involvementofskeletalmuscleoftheupperpharynxandoesophaguscausesdysphagia.Vocalcordandrespiratoryparesiscausethevoicetofadeandbreathlessnesstointerferewithspeechandotheractivities.Themostfrequentlyinvolvedaretheextra-ocularmuscles(includingorbicularisoculi)andthenthemusclesoftheneck,shouldergirdleandhipflexors.Theproximallimbmusclesaremoreseverelyaffectedthanthedistal.Extensormusclesaremoreinvolvedthanflexorsintheupperlimbs,butflexorsmorethanextensorsinthelowerlimbswhichareusuallylessseverelyaffectedthantheupper.Trunkmuscles,otherthanerectorspinae,areleastaffected.Fortunatelyfewpatientsshowthegeneralizeddistribution.

Itismuchmorecommonforthedisordertoinvolveagroupofmuscles,asinglemuscle,oreven partofacompoundmuscle(e.g.extensordigitorumlongus)andtheinitialweaknessmayaffectanymuscle.Sevenpatientshadunilateralfacialpalsywhichwasconsideredtobe Bell'spalsyforseveralmonthsuntilfurtherweaknessdeveloped.Fourpatientshadsuddensevereinspiratorydyspnoeaasthefirstsymptom. Commonlythecomplaintisrelatedtoamusclefatiguedbyaparticularmovementrequiredbythepatient'swork.Systematicexaminationmayrevealunsuspectedweaknessorfatiguabilityofothermusclesbutexaminationmustbethoroughandincludecontractionmaintainedagainstresistanceforanadequateperiod.Ifthisruleisstrictlyfollowed,myastheniaislessfrequentlyconfinedentirelytotheextraocularmusclesthanotherwritershaveclaimed.Certainlythepresentseriesgivesbutscantysupporttothestatementthatmyastheniaiscommonlyconfinedtothesemuscles.Grob(1953)statesthat ifweaknesshasnotspreadfromtheextraocularmusclestoothersin2yearsitisunlikelytodoso.Weaknessincreaseswithrepeateduseofamuscleorwithlongmaintainedcontraction. If thepatientisaskedtogazeupwardstheeyelidsgraduallydroop.Eventuallysheblinksandimmediatelytheptosislessensandmaydisappear.Butitagainreappearsonrefixingthegaze,andeachtimethishappensthefatigueappearsmorerapidlyuntilfinallynoimprovementoccurs.Exactlythesamehappenstotheoutstretchedarms.Theslowdroopingfollowedbythecyclesofsuddendropandrenewedeffortuntilfatigueiscomplete,lookslikeahystericalmanifestationtotheinexperienced.

Electromyographyshowsthat thepatientis takingadvantageofpost-tetanicfacilitationaccumulatingduringabriefresttorestorefullactivitytemporarily.Asimilarphenomenonintheextraocularmusclescausescoarsenystagmuswhichmaybemonocular.Thepatient,ifobservant,will state thattheobjectsheislookingat'suddenlyslips'.Thereflexesareoftenunusuallybrisk (indeedankleclonusissometimesobserved)butmaybefatiguedifelicitedrepeatedly.Thisshort-termfatiguabilityishighlycharacteristicofmyasthenia.Itisnotsurprisingthatfacilitativecompensation shouldbecome lesseffectiveaftertheday'sactivities, andmostpatientsdocomplain ofincreasedweakness towardsevening,butthisisnotsoinvariableas isoftenbelieved.Indeedone ofthemajordifficulties intreatment isdue tothefactthatthepatient isoftenextremelyweakonfirstwakingin themorning.Thisisnotconfined tothoserequiringfrequentdosage whomightbedeprivedoftheirdrugduring thenight,asitoccursbefore treatmentisstarted.Indeed there have beentenpatients(eightofwhomweremales)whohavenoticedthattheyimprovedasthedayadvanced,andtwoactuallyusedexerciseasameansofincreasingtheirstrength.Thisisprobablyassociatedwiththe'de-curarization'phenomenon.Permanentweakness,notrespondingtoneostigmine,andatrophyofmusclearemuchcommonerthangenerallybelieved(10%inthis series).Thisistheso-called ‘myasthenicmyopathy’. Itmayaffectanymuscle.

Thetriple-furrowedtongue,named afterKinnierWilsonthoughpreviouslydescribedbyErb(1879)andBuzzard(1905),wasonlyfoundinsixcases. Theextra-ocularmusclesquitefrequentlybecomeunresponsivetoneostigmine,andthetricepsbrachiiand ilio-psoasnextmostcommonly.Electromyographicandhistologicalchangesinthese'myopathic'musclesareindistinguishablefrompolymyositis.Theonsetisofteninsidiousandprogresssloworrapid,but insomeithasaverysuddenbeginning.Itcommonlyfollowsafebrile disease,usuallyanupperrespiratoryinfection.Only a little less common is precipitation by an emotional upset and this can be most dramatic. One patient, thrown to the ground by blast in the blitz, was unable to rise again, Another developed ptosis and diplopia the day after an accident at work, in which he was unharmed but was certain he was about to be killed. Two women had ptosis for the first time at their wedding, and one when she found evidence of her husband's infidelity, Very many attributed the onset to anger, quarrels, worry etc. with details too circumstantial to discount. All had myasthenia from then on, (as one woman ruefully remarked 'girls with myasthenia shouldn't fall in love'). A few women first had symptoms during

pregnancy, and four cases were detected by abnormal response to relaxant drugs used by anaesthetists.

Later relapses were attributed to the same factors, but other causes were the pre-menstrual period, cold, warmth (especially a hot bath) and allergy to shell fish (two cases) and one case followed soon after inoculation. Sunlight often caused ptosis and blurred vision, and a few patients asserted that it also caused generalized weakness.There can be quite surprising variation from day to day and even hour to hour. Physical exertion is certainly a factor, but I believe that the emotional state is' more important. This raises the question of remissions. Remissions are generally considered so characteristic of myasthenia as to cause difficulty in evaluating therapy. It is quite true that sudden improvement can occur, but complete remission lasting for more than a month is seen in less than half of the patients, and more than one long remission is very rare. Furthermore, most of the 'worthwhile' re- missions occur during the first 3 years, though there are notable exceptions to this rule. This is the period which sees most of the deaths from myasthenia (especially the first year of the disease) and thymectomy, to be beneficial, is best done during the first 5 years.

The impressionthat the 'active' stage of the disease is limited to this period and the subsequent course depends on the extent of the damage occurring then. This is not to say that the patient is out of all danger if he survives for 5 years. On the contrary, the 'brittleness' of the remissions and the delicate balancetakes place is significant. Sudden death may occur 'from apparently trivial respiratory obstruction, and many examples could be quoted. Nevertheless these catastrophes are due to the pre-cariousness of an adaptation which is sufficient for normal existence without permitting mobilization of reserve muscular power in emergency. They are notusually due to sudden relapses or to a progressive disease process, though second waves of renewed activity do occur sometimes, particularly after upper respiratory infection or renewed emotional stress.

Pain is quite common in weak muscles, usually an ache which is presumably due to the extra effort required to maintain posture. This often causes headache, pain round the eyes, backache etc. Sometimes the muscles are actually tender, and pain has been noted to persist even while resting in bed. One isirresistibly reminded of polymyositis. A few patients have complained of mid-sternal pain, often on stooping, which is sometimes associated with palpitations. Other sensory symptoms are rare but cannot be ignored as they have been present in other series. Most often there is tingling of the hands, thighs, or face, sometimes unilaterally. It can be considered that there was a mechanical explanation, as from shoulder- girdle drop, or a misinterpretation by

the patient of muscle 'stiffness'. Deafness, often variable, sometimes occurs, from Eustachian block in pharyngeal paresis or an unusual deafness for low frequencies which may be caused by paralysis of the tensor tympani. If the stapedius muscle is weak the patient has hyperacusis. Some authors describe objective sensory changes including a few cases of loss of taste or altered olfactory sensation which are difficult to explain. Epilepsy was present in two of my cases and unexplained blackouts in another three, These were thought to be coincidental, but Hoefer et al. (1958) found an unusually high incidence of epilepsy associated with myasthenia [9]. Raised protein in the cerebrospinal fluid was present in nine cases, but the fluid was only rarely examined. There is a substantial collection of similar cases in the literature.

Intravenous Immunoglobulins

Intravenous immunoglobulins (IVIg) were developed 20 years ago for the prophylaxis support of immunodeficient patients. However, IVIg have been increasingly used since 10 years, in the treatment of many autoimmune and inflammatory diseases raising the possibility of product shortages and ever increasing costs in the near future. Surprisingly, the immunomodulatory mechanisms of action of IVIg are unclear because of the diversity and often contradictory Fc, F(ab′)2, and non-IgG-related mechanisms that have been proposed from clinical observations and from results obtained in various in vitro and in vivo experimental model. For a increased and rationalized clinical use of intravenous immunoglobulins and for developing substitutes for some of the immunomodulatory indications in order to ensure immunodeficiency patients of long-term availability of plasma-derived IVIg, the briefing of mechanisms of action of IVIg is crucial.

Intravenous immunoglobulin (IVIG) is a therapeutic compound prepared from pools of plasma obtained from several thousand healthy blood donors. Intravenous immunoglobulin has been used in the treatment of a wide range of primary and secondary immunodeficiencies for more than 20 years. Experimental evidence retrieved in the previous years are suggestive of the fact that the therapeutic beneficial effect of IVIG in immunodeficiencies reflects an active role for IVIG, rather than a mere passive transfer of antibodies.

The large donor pool ensures the diversity of antibody repertoires that include antibody specificities to a wide spectrum of antigens. IVIG contains a sampling from the complete array of variable regions of antibodies similar to those present in normal human serum [10].

Antibodies that occur in the absence of pathological conditions or deliberate immunizations are referred to as natural antibodies (NAb), and IVIG represents a privileged source of natural antibodies. NAb have been attributed with a variety of functions, including first- line defence against pathogens [11-13].. The presence of antibodies to recurrently occurring pathogens may thus be critical for the replacement therapy of patients with humoral immune deficiencies.

IVIG has been used in agammaglobulinaemia therapy for the past 20 years [10,14]. IVIG is significantly seen as a recommended treatment for most primary immunodeficiencies (PID) [15]. IVIG is used in patients with X- linked agammaglobulinaemia (XLA), common variable immunodeficiency, X- linked hyper- immunoglobulin (Ig)M, severe combined immunodeficiency, Wiskott–Aldrich syndrome and selective IgG class deficiency. In addition, IVIG is used extensively in the treatment of an increasing number of autoimmune and inflammatory disorders.

Intravenous Immunoglobulins in Myasthenia Gravis

The extensive range of effects associated with IVIg treatment reflects the functions of circulating immunoglobulins in the maintenance of tolerance to self and immune homeostasis in healthy individuals. The mode of action of IVIg is complex, but efficacy depends on two general types of mechanism: antigen binding and modification of various effector functions. Antigen binding is mediated by the Fab part. The effector functions include binding to the various Fc receptors resulting in modulation of expression and function of Fc receptors, complement activation, complement binding, anti-inflammatory effects resulting from interference with the cytokine network, provision of anti-idiotypic antibodies, and modulation of T and B-cell activation. [16-25] It should be emphasized that these mechanisms overlap, and the outcome of IVIg treatment manifests as a combination of many different effects.

IVIg has been studied in a number of trials over the past ten years. Intravenous immunoglobulin treatment for myasthenia gravis was first done inthe 1980s [14,15]. Several such short earlyseries were reviewed in 1994 [26]. 78% of patientsimproved overall, but most received additional immunological therapy, which may account for theexceptionally long duration of improvement (30 days to 2 years). An open retrospective study ofIVIg (2.0 g/kg body weight) in 14 patients with generalized MG, many of whom also receivedadditional immunological therapy, reported significant improvement that began within a few daysof treatment initiation and peaked at 2

weeks[27]. Whereas severe cases usually responded,thosewith mild disease did not (level IV evidence).

In 10 patients with severe generalized MG reportedimprovement in all in an open prospective study of 5 days IVIg followed by single treatments every 6 weeks [28]: each patient was receiving additional immunological therapies, but itproved possible to reduce these over the year long study (level IV evidence. A retrospectivestudy of the reports of IVIg therapy in 10 patients with juvenile MG (age range 2–18) reported improvementin eight [29] and treatment was well tolerated. A consensus meeting on IVIg [30]came to a conclusion that IVIg therapy was very helpful in acute deterioratingdisease, minimizing the risk of bulbar or respiratoryweakness requiring intensive care support. A role inchronic state of the disease was not established however, and its use as a primarytreatment in MG was not recommended (level IV evidence).Aseptic meningitis has recently been reported as a complication of IVIg therapy in the treatment of myasthenia gravis [31].

Conclusion

A review of recent studies enables us to put forwardsome positive and negative aspects of IVIgtreatment in MG. The negative aspectsinclude: the temporary nature of theresponse, the high cost of the product, andthe injection of human blood and the accompanyingrisk of infection. However, the positiveimpact of IVIg therapy on MG areevident in the high rate of response, rapidonset of improvement, the lack of recognized short- and long-term toxicity, the reproducibilityof the response (the temporarynature of response may be overcome by usingrepetitive courses) resulting in the reductionof corticosteroid therapy in the majority ofpatients. Myasthenia gravis can cause facial paralysis [32][33].

Corresponding Author

Ravindra Kumar Jain

Reader,Department of Orthodontics

Saveetha Dental College,

Saveetha University, Saveetha Institute of Medical and Technical Sciences

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Telephone Number: +91 9884729660

E-mail id.: orthoravi2@gmail.com

References

1. Pascuzzi RM, Coslett HB, Johns TR. Long-term cortisteroid treatment of myasthenia gravis: report of 116 patients. Ann Neurol 1984;15:291-8.
2. Verma P, Oger J. Treatment of acquired autoimmune myasthenia gravis: a topic review. Can J Neurol Sci 1992;19:360-75.
3. Drachman DB, McIntosh KR, Reim J, Balcer L. Strategies for treatment of myasthenia gravis. Ann NY Acad Sci 1993;681:515-25.
4. Aird RB. Muscular atrophies and dystrophies. In: Baker AB, ed. Clinical Neurology. Vol 3. New York, NY: Hoeber & Harper; 1955:1498-1562.
5. Christensen PB, Jensen TS, Tsiropoulos I, et al. Incidence and prevalence of myasthenia gravis in western Denmark: 1975 to 1989. Neurology. 1993;43:1779-1783.
6. Phillips LH, Torner JC, Anderson MS, Cox CG. The epidemiology of myasthenia gravis in central and western Virginia. Neurology. 1992;42:1888-1893.
7. Phillips LH, Torner JC. Epidemiologic evidence for a changing natural history of myasthenia gravis. Neurology. 1996;25:56-58.
8. Schon F, Drayson M, Thompson RA. Myasthenia gravis and elderly people. Age Ageing. 1996;25:56-58.
9. Hoefer Pf, Aranow H, Rowland Lp. Myasthenia gravis and epilepsy. AMA Archives of Neurology & Psychiatry. 1958 Jul 1;80(1):10-7.
10. Simpson JA. Myasthenia gravis: a new hypothesis. Scottish Medical Journal. 1960 Oct;5(10):419-36.
11. Kazatchkine MD, Kaveri SV. Immunomodulation of autoimmune and inflammatory diseases with intravenous immune globulin. New England Journal of Medicine. 2001 Sep 6;345(10):747-55.
12. Bayry J, Lacroix-Desmazes S, Kazatchkine MD, Kaveri SV. Intravenous immunoglobulin for infectious diseases: back to the pre-antibiotic and passive prophylaxis era?. Trends in pharmacological sciences. 2004 Jun 1;25(6):306-10.
13. Ochsenbein AF, Fehr T, Lutz C, Suter M, Brombacher F, Hengartner H, Zinkernagel RM. Control of early viral and bacterial distribution and disease by natural antibodies. Science. 1999 Dec 10;286(5447):2156-9.

14. Zhou T, Georgiev I, Wu X, Yang ZY, Dai K, Finzi A, Do Kwon Y, Scheid JF, Shi W, Xu L, Yang Y. Structural basis for broad and potent neutralization of HIV-1 by antibody VRC01. Science. 2010 Aug 13;329(5993):811-7.
15. Orange JS, Hossny EM, Weiler CR, Ballow M, Berger M, Bonilla FA, Buckley R, Chinen J, El-Gamal Y, Mazer BD, Nelson RP. Use of intravenous immunoglobulin in human disease: a review of evidence by members of the Primary Immunodeficiency Committee of the American Academy of Allergy, Asthma and Immunology. Journal of Allergy and Clinical Immunology. 2006 Apr 1;117(4):S525-53.
16. Notarangelo LD. Primary immunodeficiencies. Journal of Allergy and Clinical Immunology. 2010 Feb 1;125(2):S182-94.
17. Ippoliti G, Cosi V, Piccolo G. High-dose intravenous immunoglobulin for myasthenia gravis. Lancet 1984; ii:809.
18. Arsura EL, Bick A, Brubber NG. High dose immunoglobulin in the management of myasthenia gravis. Arch Intern Med 1987; 146:1365–8.
19. Kazatchkine MD, Kaveri SV. Immunomodulation of autoimmune and inflammatory diseases with intravenous immune globulin. N Engl J Med 2001;345:747-755.
20. Dwyer JM. Manipulating the immune system with immune globulin. N Engl J Med 1992;326:107-116.
21. Dalakas MC. Intravenous immune globulin therapy for neurologic diseases. Ann Intern Med 1997;126:721-730.
22. Yu Z, Lennon V. Mechanism of intravenous immune globulin 1476 Boros. therapy in antibody-mediated autoimmune diseases. N Engl J Med 1999;340:227-228.
23. Gelfand EW, Landwehr LP, Esterl B, Mazer B. Intravenous immuneglobulin: an alternative therapy in steroid-dependent allergic diseases. Clin Exp Immunol 1996;104:61-66.
24. Dalakas MC. Mechanism of action of intravenous immunoglobulin and therapeutic considerations in the treatment of autoimmune neurologic diseases. Neurology 1998;51:2-8.
25. Van Schaik IN, Lundkvist I, Vermeulen M, Brand A. Polyvalent immunoglobulin for intravenous use interferes with cell proliferation in vitro. J Clin Immunol 1992;12:325.

26. Modiano JF, Amran D, Lack G, Bradley K, Ball C, Domenico J. Posttranscriptional regulation of T cell IL-2 production by human pooled immunoglobin. Clin Immunol Immunopathol 1997:83:77-85.
27. Andersson U, Bjork L, Skansen SU, Andersson J. Pooled human IgG modulates cytokine production in lymphocytes and monocytes. Immunol Rev 1994:139:21-42.
28. Vuist W, Van Schaik I, Van Lint M, Brand A. The growth arresting effect of human immunoglobulin for intravenous use is mediated by antibodies recognizing membrane glycolipids. J Clin Immunol 1997;17:301-310.
29. Edan G, Landgraf F. Experience with intravenous immunoglobulin in myasthenia gravis: a review. J Neurol Neurosurg Psychiatry 1994;57(suppl):55–6.
30. Jongen JLM, Van Doorn PA, Van der Meche FGA. High dose intravenous immunoglobulin therapy in myasthenia gravis. J Neurol 1998;245:26–31.
31. Achiron A, Barak Y, Miron S. Immunoglobulin treatment in refractory Myasthenia gravis. Muscle Nerve 2000;23:551–5.
32. Nivetha D, Kumar S. Bell Palsy's and its Clinical Significance–A Review.
33. D'dharan SR, Kumar MS. Facial Palsy-A Case Report. Journal of Pharmaceutical Sciences and Research. 2016 Oct 1;8(10):1206.

Invisible Braces in Orthodontic Treatment

Bharadwaj.B [1], Saravana Pandiyan [2], Balakrishna R.N [3]
Graduate Student [1],Senior Lecturer [2], Department of Orthodontics
Senior Lecturer [3], Department of Oral and Maxillofacial Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

The frequency of malocclusion in adults is equal to or greater than observed in children and adolescents. Align Technology Inc addressed the demand for an esthetic alternative to braces by developing an ‘‘invisible’’ method of orthodontic treatment (Invisalign) that uses a series of computer-generated, clear, removable aligners to move the dentition. Esthetic concerns during orthodontic treatment may be a significant factor, with many patients not wanting to show metal or partially clear fixed appliances with arch wires when they smile.The aim of this review is to provide comprehensive information about invisible braces in dental practice.

Key Words: Ceramic brackets, Invisalign, Essix appliance, Lingual braces, Clear aligners

Introduction

The face is said to be the most important physical characteristic in the development of self-image and self-esteem, as positive social interactions have been shown to result in better interpersonal relationships and more self-confidence. Any significant deviations from the norm may result in feelings of insecurity related to appearance, inhibition in social contacts, and comparison of self with others considered to be 'superior', all of which may negatively affect the quality of life of the individual [1].

Orthodontic treatment mainly involves the correction of facial asymmetry, malocclusion, thus improving the appearance and function of the patient. There are basically two different types of appliances, removable and fixed. Both these appliances help in the movement of teeth, retraining of muscles as well altering the growth of the jaws. These appliances apply pressure over the muscle for producing the desired effects. The treatment likely to be given for each and every patient is purely based on the severity of the malocclusion. The treatment mostly involves the metal brackets and wires, which may make some patient to neglect the treatment. Thetooth coloured brackets and wires were gradually upgraded. But this did not account for the amount of force that was given by the metal brackets and wires. The cost and the bulk of tooth coloured brackets combined with the wires were expensive when compared to the metal. The clear plastic aligners, also known as the clear aligners then came into picture. They are tough to straighten the teeth. They are made from clear plastic, so they are nearly invisible. The lingual orthodontics has also gained importance in the last few years. But the technical complexity in the placement of brackets in the lingual and palatal region and its cost remain to be the same and has restricted its use.Although Orthodontic treatment has many recognized benefits, including improvement in dental health, function, appearance, and self-esteem, nevertheless orthodontic appliances can cause unwanted complications if adequate care is not taken during the treatment. It is important that the patients are aware of these potential risks, so that they can know their responsibilities and the expectations placed on them during the treatment [2].

In orthodontics the long term stability of the achieved result remains to be a fundamental issue of concern and debate. Orthodontic retainers are exclusively made appliances, which are usually made of wires or clear acrylic, that hold the tooth in its stable position after orthodontics treatment, orthognathic surgery or any method of realigning teeth. They are most often used after orthodontic correction to keep the teeth in position while allowing remodelling of the surrounding tissues and other structures and to hold the teeth in an ideal

aesthetic and functional relation and to prevent the inherent tendency of the teeth to return to their former position (relapse) [3].

One of the major challenges that are usually faced in orthodontics is to achieve an excellent result with appliances that are both aesthetic as well as comfortable. There are currently enormous interests in so-called 'invisible orthodontics'. This has been contributed to by the intensive marketing campaigns run by the manufacturers themselves for various removable clear aligner systems. However, the availableresearch tells us that the mean accuracy of tooth movement with Invisalign is only41%. Aligner systems, because of their inherent biomechanical limitations, can only accomplish certain types of tooth movement. Their role is therefore limited to the correction of specific malocclusions [4]. In this review, invisible braces in orthodontic treatment have been elaborated.

Invisible Orthodontics

History

The possibility of using clear overlay orthodontic appliances was introduced in 1946, when Keslingdevised the concept of using a series of thermoplastic tooth positioners to progressively move misaligned teeth to improved positions. In 1997, Align TechnologyG (Santa Clara, Calif) adapted and incorporated modern technologies to introduce the clear aligner treatment (CAT). Although CAT has been cited as a safe, esthetic, and comfortable orthodontic procedure for adult patients, only a few investigations[5, 6] have focused on the predictability of orthodontic tooth movement. In 2005 Lagrave`re and Flores-Mir6 published a review in which only two studies met their inclusion criteria related to Invisalign therapy efficacy. In 1971, Ponitz [7] introduced a similar appliance called the "invisible retainer" made on a master model that prepositioned teeth with base-plate wax. He claimed that this appliance could produce limited tooth movement. Sheridan and others [8] later developed atechnique involving interproximal tooth reduction and progressive alignment using clear Essix appliances.

Types of Invisible Braces

Ceramic Brackets

After their introduction in 1986, various types of ceramic brackets are currently available by all major orthodontic manufacturers, thus gaining widespread popularity and becoming an integral part of the clinician. All currently available ceramic brackets are composed of aluminum oxide. However, because of their distinct differences during fabrication, there are

two types of ceramic brackets, namely, the polycrystalline alumina and the single crystal alumina. The manufacturing process plays a very important role in the clinical performance of the ceramic brackets. Because production of polycrystalline brackets is less complicated, these brackets are more readily available at present. The most apparent difference between polycrystalline and single crystal brackets is in their optical clarity. Single crystal brackets are noticeably clearer than polycrystalline brackets, which tend to be translucent. Fortunately, both single crystal and polycrystalline brackets resist staining and discoloration. Ceramic brackets come in a variety of edgewise structures including true Siamese, semi-Siamese, solid, and Lewis/Lang designs. Many brackets are made by specialized ceramic manufacturers and are sold under proprietary names by manufacturers or distributors of orthodontic products [9].

Ceramic brackets appeared in the mid-1980s and were better able meet patients' functional and esthetic demands; plastic brackets had left much to be desired, particularly regarding color stability, deformation, torque, friction, tie-wing fracture, and debonding.With improvement in the physical and mechanical properties of esthetic brackets by the incorporation of ceramic or glass particles and a metal channel as reinforcement in plastic brackets,in addition to surface preparation of the channel in the ceramic brackets, the demand for esthetics became the main target. Less visual perception, to which the term esthetic is related, was desired. Various ceramic brackets are monocrystalline brackets, Polycrystalline brackets and zichronia brackets. The major advantages of ceramic brackets are esthetics, tooth colored material and acceptance by patient.But there are certain disadvantages of ceramic brackets. Ceramic brackets have a higher incidence of fracture during debonding, particularly with the conventional debonding techniques. Ceramic brackets are unable to withstand strong torsional forces, especially after the bracket surface has been nicked during treatment. Enamel wear occurs if ceramic brackets contact opposing tooth surfaces. Therefore, placement of ceramic brackets is contraindicated on the lower anterior teeth in cases with deep overbite and minimal overjet. In such cases, sufficient overjet has to be created before bonding the lower incisors. Similarly, during maxillary incisor retraction, the overbite should be reduced first so that the maxillary incisors do not contact the mandibular ceramic brackets. Ceramic brackets can cause nicks in the arch wires, resulting in more friction between the bracket and the arch wire. This can decrease the efficiency of tooth movement. The use of ceramic brackets in patients who will undergo orthognathic surgery should be discouraged. The fracture of the brackets before, during, or after surgery creates the potential for undesirable and avoidable cornplications [10].

Self-ligating ceramic brackets have become very popular over the course of the last decade. Effective ligation is important, as it ensures that forces generated between the archwire and the brackets are transmitted to the tooth. In later stages of treatment, when single teeth or groups of teeth are slid along the archwire by traction from elastics or coil springs, the ligation maintains the existing alignment. As a consequence, the control of the archwire in the slot is always coupled with resistance to sliding (RS) arising in the mechanical system [11].

Invisalign

Joffe [12] suggested that the Invisalign appliance is most successful for treating mildlymalaligned malocclusions (1 to 5 mm of crowding or spacing), deep overbite problems (e.g.,Class II division 2 malocclusions) when theoverbite can be reduced by intrusion oradvancement of incisors, nonskeletally constricted arches that can be expanded with limitedtipping of the teeth and mild relapse after fixed-appliance therapy. Use of the Invisalignappliance is relatively new fororthodontists and is still being developed. Currently, fewclinical studies and case reports have assessed the effectiveness of this technique. AlthoughAlign Technology has suggested guidelines for its appropriate use,clinicians haveencountered numerous limitations when using the appliance.

Method of Fabrication

The Invisalign appliance involves a series of aligners made from a transparent, thin (typically less than 1 mm) plastic material formed with CAD-CAM laboratory techniques. Thesealigners are similar to the splints that cover the clinical crowns and the marginal gingiva.Each aligner is designed to move the teeth a maximum of about 0.25 to 0.3 mm over a 2-week period, and is worn in a specific sequence. The Invisalign appliance is currently recommended for adults and for adolescents with fullyerupted permanent teeth who meet an acceptable standard of compliance.

Advantages of Invisalign orthodontics are:1) It is removable when compared to metal braces, 2)Convenient as there is no worry of breakage of wires or brackets, 3)Highly aesthetic and 4)As they are made of plastic, they are highly durable.

It has few disadvantages too. Temporary sensitivity of teeth and gingival and the Invisalign appliances, because of their removability, have very limited control over precise tooth movements.

Lingual Braces

With invent of lingual appliances an entirely new treatment biomechanics emerged. Orthodontists are also determined towards this goal and have given a new dimension by changing from the labial to the lingual so as to give rise to a new concept the Invisible Braces concept or Lingual Orthodontics[13-14]. Due to increasing social awareness and esthetic demands of young patients, the application of lingual orthodontics today is not only restricted to adults, but has been extended to adolescents as well.

This kind of braces is used in cases where stability is questionable and long term retention is planned. Has four main indications such as, maintaining lower incisor position, diastema maintainence, implant or Pontic space maintainence, retaining closed extraction spaces.

Types of lingual braces: Banded canine to canine retainers,bonded lingual retainers,Band and spur retainers.

Self ligation in lingual orthodontics was first described by Macchiet in 2002 the Philippe Self Ligating Lingual Brackets that can be bonded directly to the lingual tooth surfaces. Self ligation in lingual orthodontics has come with interactive lingual brackets. It has a non-locking rotating clip resulting in unique flexibility. It comes with a self-ligating clip flexes like an elastomeric ligature and therefore responds to the actual malocclusion without losing force, which reduces binding and prevents notching especially with highly rotated teeth. The Evolution SLT Smart cap is a unique indirect bonding system based on the principles of the HIRO system. It offers the practitioner the benefits of bonding each tooth individually, offering at the same time the speed of a full bonding tray.

Another precise Lingual Self ligating system comes as Inovation L by GAC which is an interactive, twin, self-ligating lingual bracket system. Only an interactive bracket provides full functionality throughout the course of treatment. Passive function is achieved with a round Sentalloy NiTi wire that slides freely for efficient leveling and alignment. This is created with the light seating of a BioForce wire into the base of the slot so programming may be expressed and rotations are corrected. Active function with full control is provided by the introduction of a full-size Resolve Beta-Ti wire, enabling full expression of the bracket’s torque and achievement of the desired result. Dentaurum has come up with the World‘s first Nickel free Lingual Brackets called Magic. Due to the magic bracket's unique geometrical form, arch wires can be inserted occlusally. The innovative combination of occlusal and horizontal slot directions enables the arch wire to be automatically pressed into the slot.

The Incognito Lingual Appliance

Lingual orthodontics has advanced to a highly sophisticated level where CAD/CAM (computer-aided design/computer-aidedmanufacture) technology is employed to manufacture both thebrackets and arch wires for each patient individually in the incognitoappliance system. The incognito lingual appliance system is used to treat both teenagers and adults. It can be used in combination with functional appliances like the Herbst appliance, and 'bite jumpers' like the Forsus appliance. It can be used without difficulty in the management of orthodontic patients who require orthognathicsurgery.

Technological developments mean that lingual orthodontics is now far more acceptable to the patient, as discomfort and interference with speech and mastication have been minimised. Although lingual orthodontics is more difficult than labial orthodontics to perform, it can achieve a high standard of orthodontic result comparable to labial orthodontics when properly applied. It is, like labial orthodontics, dependent on patient compliance.

2D Lingual Brackets

2D Lingual Brackets are bonded to the inside of the dental arch. They are invisible from the front and give you that carefree smile from the beginning. 2D Lingual Brackets offer the most important advantage of braces: they work around the clock and you do not need to motivate yourself to wear them regularly. 2D Lingual also acts close to the so-called resistance centre of the teeth and moves them effectively and quickly. 2D Lingual Brackets are the attest brackets on the market at a height of only 1.3 to 1.65 mm, depending on the design. Due to the integrated clips, no further retaining elastics are required, which would only add volume and impair wearing comfort. The optimally rounded edges ensure that the brackets are not uncomfortable or irritating. Very soon after placement you will be able to speak with- out a noticeable difference and eat as usual.

A three-dimensional (3D) surface scanning system was recently introduced in dental fields and has been used most extensively for example, for assessing morphological changes in maxillofacial surgery or in orthopedic treatment with a functional appliance. Another use for the 3D data acquisition in orthodontics is bending art system (BAS) or Invisalignsystem introduced as a new treatment modality [15].However, research on the various clinical

applications of 3D digital model is still in its early stage, as it has been used as a simple model analysis, a digitized data storage. It can be divided into 7 parts:

1. The measuring accuracy and process of the 3D model scanning technique was evaluated in terms of linear, surface and volumetric parameters. The diverse clinical applications of model analysis, including measuring basal arch width, or sectional areas concerned will be presented.
2. Giving the evidences that the superimposition of the 3D digital maxillary model is clinically as reliable as cephalometric superimposition for assessing orthodontic tooth movements.
3. Presenting the clinical cases, using the superimposition technique for the 3D measuring of orthodontic tooth movement in maxilla.
4. Describing the clinical procedure for digital diagnostic setup.
5. Introducing a novel method concerning the volumetric assessment of tooth wear using 3D reverse engineering technology.
6. Presenting a quantitative 3D soft tissue facial analysis using a color coding system.
7. Presenting feasible methods of the integrating 3D digital model into a 3D facial image to visualize the anatomic position of the dentition [16].

The main advantage is that, the facial surfaces of the teeth are not damaged, facial gingival tissues are not adversely affected, he position of the teeth can be more precisely seen, facial contours are truly visualized since the contour and drape of the lips are not distorted by protruding labial appliances, most adult and many young patients would prefer "invisible" lingual appliances if costs, treatment times, and results were comparable to those of labial appliance treatment.

But there are certain cons for the same. They are, increased chair time, banded variation may interfere with oral hygiene maintainence, more cumbersome to insert, more prone to breakages and finally loss of healthy tooth material.

Clear Aligners

Orthodontic treatment is mainly surrounded with metals like wires and brackets. This esthetic compromise repels adult orthodontic patient from accepting treatment. Tooth colored brackets and wires tried to overcome these limitations but could not proved to be good option to metals because of its bulk, and cost. In last few decades lingual orthodontics has gained

worldwide popularity in terms of esthetics but its widespread use is restricted because of complexity in bracket positioning, treatment mechanics, limited access to the lingual / palatal surface and cost. In addition, bracket bonding procedure endangers the precious enamel to chemical insult, making it unaesthetic and susceptible to microbial attack. So, clear aligners have emerged as a promising option for adult orthodontic patients.

Clear Aligner Therapy is an orthodontic treatment modality in which the patient wears a series of customized clear, removable aligners that gradually moves the teeth to a desired position. The total number of aligners varies depending on severity of malocclusion. Clear aligners do have some limitations and inconveniences. The benefits of Clear Aligner Therapy generally outweigh the drawbacks. Clear aligner treatment falls into two basic categories. The first category consists of those thermoformed appliances, sometimes known as Essix Retainers that are fabricated by making adjustments to the tooth positions on plaster or stone models and fabricating one or more aligners to correct a minor malocclusion. These types of appliances are sometimes fabricated in the orthodontist's office or sometimes sent out to commercial laboratories. These types of in-office prepared aligner systems have been in use since around 1994. Invisalign is a proprietary orthodontic technique that uses a series of computer generated custom plastic aligners to gradually guide the teeth into proper alignment [17-19].

In one study, metallic brackets in use for one month were found to be colonized by cariogenic microorganisms and periodontal pathogens [20]. In another study, a negative effect on microbial flora was observed with long-term utilization of orthodontic appliances. The study recommended patients be put on short recare intervals during therapy [21]. During treatment, there are strategies that can be used to minimize caries and its precursor, demineralization. When used to bond brackets, resin-modified glass ionomer cement and fluoride-releasing resin composite have been successfully usedto inhibit demineralization [22].After retreatment of anterior crowding and open-bite relapse with the Invisalign appliance, Womack and others [23] found that the position of the maxillary central incisors was superior to that of the canines and posterior teeth. Djeu and others found that fixed appliances were superior to the Invisalign appliance for treating buccolingual crown inclinations, occlusal contacts, occlusal relationships, and overjet. In their 1998 study comparing Essix and Hawley retainers, Lindaurer and Shoff found that one sixth of their patients lost their appliances; the majority of these losses were ascribable to the appliances being clear and removable. Aligners from the Invisalign appliance have very similar properties to those of Essix appliances [24].

Conclusion

Recently the success of a complete orthodontic treatment is based on achieving facial balance, and a balance between functionality, aesthetically treatment and patient's aspirations. The aesthetic aspirations are now found in both male and female, most commonly in the younger population of patients. In the aesthetic aspect, the invisible orthodontics is playing a crucial rolein also the functionality aspect. Without compromising the efficiency of the appliance and also having a better aesthetic harmony for the patient invisible orthodontics is found to be one of the best treatment options. Thorough efforts, methodological approach, and vast experience gained over the years has proved that this specialty of orthodontics has the answer to the ever increasing cosmetic demands of the patients who insist on appliances which are not seen to the outside world and at the same time want to get their malocclusion treated. Today, the main goal is to achieve facial balance, and the development of orthodontic treatment is the balance between esthetical treatment, functionality and a patient's aspirations. Esthetic aspirations are now universal and involve younger patients including both male and female adolescents. In this regard, invisible orthodontics, which is highly esthetic play a fundamental part in achieving dental alignment. A smile revealing esthetic harmony is very important at any age as difficult as that of adolescence. Invisible orthodontics thus represents the best solution for meeting the needs of the patients without the risk of damaging functional efficiency.

Corresponding Author

Balakrishna R.N

Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamalle High Road,Chennai 600077,Tamil Nadu, India

E-mail Id – balakrishdr8394@gmail.com

Mobile number: 8939896729

References

1. Shilpa.R. Self Perceived Orthodontic Treatment Need in Dental Students. Int. J. Pharm. Sci. Rev. Res 2017; 46(2): 39-44.
2. Suhashini Ramanathan, Navaneethan Ramasamy. Avoidable Complication and Patient Care during orthodontic Treatment. J. Pharm. Sci. & Res 2015; 7(12): 1096-98.
3. Vignesh,A. SumathiFelicita. Long Term Effectiveness of various orthodontic Retention - A Review. IOSR Journal of Dental and Medical Sciences2015; 14(2): 56-59.
4. Kravitz ND, Kusnoto B, Agran B, Viana G. How well does Invisalign work? A prospective clinical study evaluating the efficacy of tooth movement with Invisalign. Am J Orthod Dentofacial Orthop 2009;35:27–35.
5. Krieger E, Seiferth J, Marinello I. InvisalignH treatment in the anterior region: were the predicted tooth movements achieved? J OrofacOrthop 2012;73:365–376.
6. Ponitz RJ. Invisible retainers. Am J Orthod 1971; 59(3):266–72.
7. Sheridan JJ, LeDoux W, McMinn R. Essix retainers: fabrication and supervision forpermanent retention. J Clin Orthod 1993; 27(1): 37–45.
8. Ehsani S, Mandich MA, El-Bialy TH, Flores-Mir C. Frictional resistance in self-ligating orthodontic brackets and conventionally ligated brackets. Angle Orthod 2009;79:592–601.
9. John Buckley. Lingual orthodontics: an illustrated review with the incognito fully customised appliance. Journal of the Irish Dental Association 2012; 58(3): 149-155.
10. Kaphoor AA, Sundareswaran S. Aesthetic nickel titanium wires-how much do they deliver? Eur J Orthod 2012; 34: 603-9.
11. Sagar Hirani, Unnati Patel, Nirav Patel. Invisible Orthodontics -A Review. IOSR Journal of Dental and Medical Sciences 2015; 15(6): 56-62.
12. Alexander CM, Alexander RG, Gorman JG, Lingual orthodontics: A status report, Part I, J Clin Orthod 1982; 16: 255.
13. Wiechmann D, Customised Brackets and archwires for lingual orthodontic treatment. Am J Orthod Dentofacial Orthop 2003; 124: 593-9.
14. Joffe L. Invisalign. Early experiences. J Orthod 2003; 30(4):348–52.
15. Choi, D.S.; Jeong, Y.M.; Jang, I.; Jost-Brinkmann, P.G. & Cha, B.K. Accuracy and reliability of palatal superimposition of three-dimensional digital models. Angle Orthod 2010; 80(4): 497-503.

16. Andrucioli MC, Nelson-Filho P, Matsumoto MA, Saraiva MC, Feres. Molecular detection of in-vivo microbial contamination of metallic orthodontic brackets by checkerboard DNA-DNA hybridization. Am J Orthod Dentofacial Orthop. 2012; 141(1):24-9.
17. Aricis, Regan D. Alternatives to Ceramic Brackets: the Tensile Bond Strengths of Two Aesthetic Brackets Compared Ex Vivo with Stainless Steel Foil-mesh Bracket Bases, British Journal of Orthodontics 1997;24: 133–137.
18. Wong B, Invisalign A to Z, Am J Orthod Dentofacial Orthop 2002; 121: 540-1.
19. Paquette D, Clear Aligner Treatment Orthodontics: Current Principals And Techniques 2005; 5:639-90.
20. Topaloglu-Ak A, Ertugrul F, Eden E, Ates M, Bulut H. Effect of orthodontic appliances on oral microbiota—6 month follow-up. Clin Pediatr Dent 2011; 35(4):433-6.
21. Wilson RM, Donly KJ. Demineralization around orthodontic brackets bonded withresin-modified glass ionomer cement and fluoride-releasing resin composite. Pediatr Dent 2001; 23(3):255-9.
22. Womack WR, Ahn JH, Ammari Z, Castillo A. A new approach to correction of crowding. Am J Orthod Dentofacial Orthop 2002; 122(3):310–6.
23. Djeu G, Shelton S, Maganzini A. Outcome assessment of Invisalign and traditional orthodontic treatment compared with the American Board of Orthodontics objective grading system. Am J Orthod Dentofacial Orthop 2005; 128(3):292–8.
24. Lindauer SJ, Shoff RC. Comparison of Essix and Hawley retainers. J Clin Orthod 1998; 32(2):95–7.

Liposomes

B.Valli[1], Vishnupriya[2], Jayanth Kumar V[3]

Graduate Student[1], Professor[2], Department of Biochemistry,Reader[3], Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Liposomesaresphereshaped vesicles, consists of oneor morephospholipid bilayers. Theyarenon-toxic, flexible, biocompatible, biodegradable and non-immunogenic. Theyfunction in drugtargetingcarryboth hydrophobic and hydrophilic molecules. Liposomes deliver drugs bydiffusion rather than bydirect cellfusion.Liposome has the natural abilityto target cancercells. Theyare also used in textile industries.

Key Words:*Drug deliverysystemusingliposomes, surfactant,encapsulation, lipid spherules.*

Introduction

Liposomes was initially explained by the British hematologistDrAlec D Bangham FRSin the year 1961 and was published in the year1964,at theBabrahamInstitute,which is situated in Cambridge.Liposomesare colloidal and vesicularstructures that are madeof oneor morelipid bilayers with acentral coreof equal numbers of aqueous solution. Theword liposomeis obtained from twogreekwords namely the “Lipos” which means fat and “Soma” which means body. These liposomes are concentricbleeder vesicles inside which an aqueous volumeis totallyenclosed bya membraneous lipid bilayer. Membranes areusuallymadeof phospholipids, which are molecules that haveahydrophilic headgroupandahydrophobic tailgroup. Thehead of the liposome is attracted to water, and thetail of the same , which is madeofalonghydrocarbonchain,is repelled by water[1]. While the phospholipids aredispersed in water, they instantlyform closed structurewith internalaqueous environment surrounded by thephospholipidbilayermembranes, this kind of vesicular system is calledas liposome[2]. The phospholipid bilayer is formed when the hydrophobic tail of both layer faces each other. Liposomes arebiocompatible, biodegradable and target drugto organsystems.

Liposomes technologyis used in various fields, like the pharmaceuticals,cosmetics, foods, detergents, textiles, and many other applications, because of their abilitytoliberate and

encapsulate slowly[3,4].Liposomes are basicallymadeofnaturallyderivedphospholipids that aremixed with lipid chainslike the eggphosphatidylethanolamineor they are made ofpuresurfactant components namely the DOPE which is the dioleoylphosphatidylethanolamine. Liposomes generallythough notby definition, it is found to be made up of a coreof aqueous solution ; and lipid spheres which contain noaqueous materials called micelles, anyways , reverse micelles could be made to encompass an aqueous environment also[5].

Based upon the composition and the molecular structure , liposomes can separate hydrophobic or hydrophilic molecules from the solution[6,7]. These vesicles are not considered to be strong formations but rather they are fluid entities which are complex supramolecular assemblies. Because of the dynamic properties they possessand therelatively easy manipulating ability, these liposomes have been used in a wide range in the analytical sciences as well as for drug and gene delivery.

Mechanism of Liposome Formation

Since the liposomes are made up of phospholipids, they are usually amphipathic in nature and they tend to have the ability to bind both with aqueous and polar moiety. They also have a polar head and a non polar tail.

The polar end is mainly made up of phosphoric acid and it will get bound to water soluble molecules.In aqueous medium the molecules are found in self- assembled structure and it is oriented in such a way that the pole side of the molecule tend to remain in contact with in polar environment and also at same time shieldsthe non polar part. Liposomes are being formed when these thin films are getting hydrated and stacks of liquid crystalline players tend to become fluid and gets swollen.

Once these vesicles are formed, there is a change in vesicle shape and also morphology required energy input in the form of sonic energy to obtain SUVs and mechanical energy to obtain LUVs.

Yet, in aqueous mixtures these molecules are able to get converted to various phases, and some of them are constantly stable and other tend to remain in metastable form.

Types ofLiposomes

There are various types of liposomes identified.

Liposomes areclassifiedon the basisof

-Structural parameters

-Method ofpreparation

-Composition and applications

Structural Classification

There are three types of liposomes based on structural classification . They are:

a. Unilamellar vesicles –Unilamellar is further classified into three based on the size of e vesicles as Large unilamellar vesicles orLUVs, whose size varies between 100nm-1000nm , small unilamellar vesicles or SUVs whose size varies between 20nm-40nm, and the medium unilamellar vesicles whose size varies from 40nm-80nm
b. Oligolamellar vesicles- These kinds of liposomes are made by 2-10 bilayers of lipids which surrounds an internal volume[8].
c. Multilamellar vesicles- These kind of vesicles have multiple bilayers within which they can organize their aqueous volume . This kind of organization of aqueous volume can be done in several ways. These vesicles differ from each other in the method of preparation. They are arranged in concentric circles resembling an onion where in there is a layer of large unilamellar vesicle alternating medium unilamellar vesicles and both surrounding the core of small unilamellar vesicles. With the exceptionofmultilamellar vesicles,theseothermorphologies aredifficultto obtain withouthighlycontrolled processesforformation. Giant vesicles also deservespecial attention because their sizes arelarge, rangingfrom 1 µm to morethan 100 µm[9]. These largevesicles arestudied and wellcharacterized, partiallyduethe easeofobservation via optical microscopy.

Methods ofLiposome FormationandDrug Loading

There aremanyways ofpreparinglipososmes which arelisted below. Some oftheimportant methods havebeenexplained with their steps.

Hydration oflipids in presenceof solvent , Ultrasonication, French Pressure cell, Solvent injection method , Detergentremoval , Reversephase evaporation technique, High pressureextrusion , Miscellaneous methods , Drylipid film , Emulsions , Micelle-formingdetergents, Alcohol injection technology[10].

Solvent Injection Method

In this method lipids are initially dissolved in an organic solution and then they are mixed with aqueous phase containing materials to be entrapped with liposome.

- Ethanol injection method- This was first described in the year 1972. Ethanol injection method relevance remains in the observation that a smaller distribution of small unilamellar liposomes (under 100 nm) can be attained by easily injecting an ethanolic lipid solution in water, in single step, without extrusion or sonication
- Ether injection method - The ether injection method varies from the ethanol injection method as the ether is immiscible along with the aqueous phase, which is also heated so that the solvent getsseparated from the liposomal product. The method involves the

injection of ether-lipid solutions into the hot aqueous phases above the boiling point of the ether. The ether vaporizes when it comes in contact withthe aqueous phase, and the dispersed lipid forms primarily unilamellar liposomes[11].

Detergent Removal Method

Detergent can be removed by either

- Dialysis- Liposomes, that are in the size range of 40–180 nm, arecreated when lipids are solubilised with detergent, yielding defined mixed micelles[12]. When the detergent is consequently removed by controlled dialysis, phospholipids tends to form homogeneous unilamellar vesicles with usefully large encapsulated volume.
- Column chromatography
- Bio-beads

Miscellaneous Methods

- Slow-swelling in non-electrolyte solution
- Removal of chaotropic ion
- Freeze-thawing- Freeze thawing method is totally based upon freezing of small unilamellar vesicles dispersion (SUV). And then thawing by keeping at room temperature for about 15min. Atlast subjecting it to a brief Sonication cycle that considerably causes reduction in the permeability of the Liposomes membrane. In order to make giant vesicles of diameter ranging between 10 to 50um, the freeze thaw method is being modified to involve a dialysis step against hypoosmolar buffer in the place of sonication. The method is simple, rapid and mild for entrapped solutes, and results in a high proportion of large unilamellar vesicle formation which are useful for study of membrane transport phenomenon.

Other than these there are few other methods used for the preparation of particular types of liposomes namely

- Reverse phase evaporation method- used in the preparation of single or oligolamellar vesicles
- Reverse phase evaporation method- used in the preparation of Multilamellar vesicle
- Stable plurilamellar vesicles
- Frozen and thawed medium lamellar vesicles
- Vesicles prepared by extrusion technique
- Dehydration - rehydration method.

Reverse phase evaporation method- A lipidic film is being prepared by evaporating the organic solvent with the help of reduced pressure. The system is mixed with nitrogen and the lipids are finelyre-dissolved in a second organic phase that is generallymade by diethyl ether and/or isopropyl ether. Large unilamellar and oligolamellar vesicles are prepared when an aqueous buffer

is being introduced into this already prepared mixture. The organic solvent is consequently removed and the system is constantly maintained under continuous flow of nitrogen. These vesicles are found to have aqueous volume to lipid ratios which are 30 times greater than the sonicated preparations and 4 times greater than the multilamellar vesicles. An important thing ,is that a substantial fraction of that aqueous phase which is up to 62% at low salt concentrations is being entrapped inside the vesicles, encapsulating even when large macromolecular assemblies with high efficiency[13].

Super critical reverse evaporation method:The super critical reverse evaporation method is a single-step newly developed method which has been used for liposomes preparation with the help ofsupercritical carbon dioxide. This super critical reverse evaporation method allows the aqueous dispersions of liposomes that areto be obtained through emulsion homogeneous mixture of supercritical carbon dioxide/L-R-dipalmitoylphosphatidylcholine/ethanol under adequate stirring and subsequent pressure reduction . Transmission electron microscopy results revealed that vesicles are found to be large unilamellar vesicleswith thediameters of 0.1– 1.2 microm. The trapping efficiency of these liposomes were indicated to be more than 5 times higher values for the water-soluble solute when compared to multilamellar vesicles which are prepared by the Bangham method. The trapping efficiency for an oil-soluble substance,like the cholesterol, was found to be about 63%. And the final results showed that the super critical reverse evaporation method is an excellent method that allows one-step preparation of large unilamellar liposomes which exhibits a high trapping efficiency for both the water-soluble and oil-soluble compounds.

Composition and Applications

Liposomes can alsobeclassified in terms of composition and mechanisms of intracellular deliveryinto sixtypes:

1. Conventional liposomes- Neutral or negatively charged phospholipids and cholesterol.
2. pH-sensitive liposomes- Phospholipids namely PE or DOPE with either OA or CHEMS
3. cationic liposomes- Along with DOPE
4. Immuno-liposomes- LCL or CL with attached monoclonal antibody or recognition sequence.
5. long-circulatingliposomes- Polyethylene glycol is attached to the surface membrane to prevent them from being identified by the phagocyte system. These attachments to liposomes increases the liposome circulating time in the body by decreasing the clearance from the blood stream.
6. Fusogenic liposomes- reconstituted Sendai virus envelope.

Advantages

1. Provides selectivepassive targetingto tumor tissues (Liposomal doxorubicin).
2.Increased efficacyandtherapeuticindex[9]
3. Biodegradable, biocompatible, flexible
4. Non ionic
5. Can carryboth watersoluble and lipid solubledrugs
6. Drugcan bestabilized from oxidation.
7. Altered pharmacokinetics and pharmacodynamics
8. Can be administered through various route
9. Act as reservoirof drug.
10. Flexibilityto couplewith sitespecific ligandsto achieveactivetargeting[11].
11. Efficient control of release of liposomes.
12. Resembles the natural membrane structures.
13. It has shown increased targeting prospects.
14. Liposomes gives both the lipophilic environment and aqueous “milieu internee” in a single system. It Canalso protect the encapsulated drug.
15. Easy to construct.
16. Does not accumulate in the heart and thus there is no possibility for cardiotoxicity.
17. Prevention of oxidation of the drug.

Disadvantages

1. Production cost is high.
2. Leakage and fusion ofencapsulated drug/molecules.
3. Short half-life.
4. At times phospholipid tend to undergo oxidation and hydrolysis like reaction.
5. Lower solubility.
6. Fewer stables.
7. The development of Liposomes at industrial level is very difficult becauseof its physiological and physicochemical instability.
8. They are highly prone to degradation by oxidation and hydrolysis.

Characteristics

Liposomesare generallyformed bythe selfassemblyof dissolved lipid molecules, each ofwhich contains ahydrophilic headgroup and hydrophobic tails. Theselipids takeon associations whichyield entropicallyfavorable statesoflow free energy, in some cases formingbimolecularlipid leaflets .Such leaflets are characterized by hydrophobichydrocarbon tails facingeach otherand hydrophilicheadgroups facingoutward to associatewith

aqueoussolution[14]. At this point thebilayer formation is still energeticallyunfavorablebecause thehydrophobicparts ofthe moleculesarestillin contact with water, aproblem that is overcome throughcurvatureof the formingbilayer membrane upon itself to form a vesicle with closededges[15]. This free-energy-driven self assemblyis stable and has been exploited asapowerful mechanismfor engineeringliposomesspecificallytothe needsofagiven system[16].Thevesiclesizewas foundto be varied from146±0.6 nm to 198±0.6 nm.

Applications

The following are the list of areas where liposomes are being used

1. Cancer chemotherapy
2. Gene therapy
3. Liposomes as carriers for vaccines
4. Liposomes as carriers of drug in oral treatment
5. Liposomes for topical applications
6. Liposomes for pulmonary delivery
7. Against leishmaniasis
8. Lysosomal storage disease
9. Cell biological application
10. Metal storage disease
11. Role of liposomes in Ophthalmic delivery of drugs

Liposomes used inParasitic Diseases andInfections

Since conventional liposomes areingestedbyphagocyticcells in thebodyafter intravenous administration, theyareconsidered to be ideal vehiclesforthetargettingof drugmoleculesinto these macrophages. Theyincludeleishmaniasisand several fungal infections.Leishmaniasis is a parasitic infection ofmacrophages whichaffects over 100million peoplein tropical regions and is often fatal.All the Liposomesgetsaccumulated in the verysame cellpopulation which is infected and thereforeoffer an ideal drugdeliveryvehicle.

Liposomes used inAnticancer Therapy

Manydifferent liposome formulations of multipleanticancer agents weredescribed to beless toxic than the freedrug[17]. Anthracyclines aredrugs which stop thegrowth of dividingcells byintercalatinginto theDNA and thereforekill predominantlyquicklydividingcells. Thecells are presentin tumours, butalso in gastrointestinal mucosa, hair,and blood cells and thus this class of drugs ishighlytoxic.Manydifferent formulations weretried.Inmostcases the toxicitywas reduced

about 50%. This includes both, short term and chronictoxicitiesbecause liposome encapsulation reduces thedistribution ofthedrugmolecules towards those tissues. Forthe samereason, on theotherhand, theefficacywas inmanycases compromised due to the reduced bioavailabilityofthe drug, especiallyifthe tumour was not phagocytic, or located in the organs ofmononuclear phagocytic system.In fewcases, namely thesystemic lymphoma, the effect ofliposome encapsulation had shownenhancedefficacy becauseto the sustained release effect, i.e. longer presenceof therapeutic concentrations in the circulation[18]whilein several other cases thesequestration ofthe druginto tissues of mononuclear phagocyticsystem actuallyreduced its efficacy. The most used and studied is Adriamycin which is the commercial name for Doxorubicin HCl. Liposomes can be used to deliver many such drugs and thereby minimize their toxic effects on healthy cells. Targeted delivery of drugs to cancer cells can be achieved by coating the monoclonal antibodies (MAbs) which are raised against tumor-cell specific antigens. Both In vitro and in vivo studies done by Ahmad et al. of squamous-cell carcinoma in mouse models gave the evidence that antibody-coated polyethylene glycol liposomes that contains doxorubicin were found to be more effective and less toxic than the free drugs, drugs that are incorporated into antibody-free liposomes, and also antibody- coated conventional liposomes[19,20].

Liposomes in Ophthalmic Drug Delivery

Liposome were found to be examined for ophthalmic drug delivery as it provides advantages as a carrier system. It is a biodegradable and also a biocompatible nano carrier. It can help in enhancing the permeation of poorly absorbed drug molecules by getting bound to the corneal surface and also improving residence time. It encapsulates both the hydrophilic and the hydrophobic drug molecules. In addition to this, liposomes can also improve pharmacokinetic profile, and enhance therapeutic effect, and help to reduce toxicity associated with higher dose. Owing to the versatile nature of liposomes ,they have been widely examined for the treatment of both anterior and posterior segment eye disorders. Recent approaches for the anterior segment drug delivery are being focused on improving corneal adhesion and permeation by administeringdifferent bio-adhesive and penetration enhancing polymers. Yet, in the case of posterior segment disorders, improvising the intravitreal half-life and targeted drug delivery to the retina is very much necessary. At present verteporfin is being used in the clinical aspect in photodynamic therapy for the management of subfoveal choroidal neovascularization (CNV), ocular histoplasmosis, or pathological myopia very effectively. Verteporfin is a light-activated drug that is beinginjected by intravenous infusion. In photodynamic therapy, once the

drug is being injected, a low-energy laser is applied to the retina through the contact lens so that it can activate verteporfin that results in closure of the abnormal blood vessels. But, photodynamic therapy generally does not permanently shut the abnormal vessels and choroidal neovessels tend to reappear after several months.

Liposomes in MacrophageActivationandVaccination

The automatictargeting ofliposomes bymacrophages can bestopped in various other ways, which includesthe macrophage activation and in vaccination. Few natural toxins causesstrong macrophage responsewhich leads tomacrophage activation. This process can berepeated in a similar way and also improvisedwiththe useof liposomes due tosmall molecules with immunogenic properties like the haptensand cannot causeimmune response without being attached toalarger particle.

Other Applications

Liposomes areusedfordrugdeliverydueto theirunique properties. Aliposome encapsulates aparticular region on the aqueous solution inside ahydrophobicmembrane; dissolved hydrophilic solutes cannot readilypass through the lipids. Hydrophobic chemicals are identified to bedissolved into the membrane, and in a through wayliposomesare facilitated tocarryboth thehydrophobic molecules andhydrophilic molecules.

Liposomes in Drug Targetting

The approach for drugtargettingvia liposomes involves theuse of ligands(e.g., antibodies, sugar residues,apoproteinsorhormones), whicharetagged on the lipid vesicles. Theligand identifies specific receptor sites and, therefore, causesthe lipid vesicles to concentrate at such target sites. Bythis approach the otherwisepreferential distribution of liposomes into the reticulo-endeothelial system RES (liver, spleen andbonemarrow) is avertedor minimized.Liposomes have been proved that restricting the distribution of the drug to the particular target site should cause efficacy increase even at low dosage with attendant decrease of toxicity. In fact , pumping a drug through the entire body surface is not only wasteful but also, more fundamentally, it increases the undesirable side effects. Hence, the advantages of drug targeting includes reducing of drug waste, and it is also possible to deliver a drug to a tissue or cell region which is not usually accessible to the free or untargeted drug[21]. Liposomes have been widely applied in drug targeting especially in

cancer treatment. Effective chemotherapy is highly limited by the toxic side effects of the drugs.

Liposomes inDrugDelivery

Liposomes are majorly used in drug delivery becauseof their unusual but unique structural properties. A liposome is capableof encapsulating a region of aqueous solution present inside a water repelling membrane[22]. This membrane is otherwise called as hydrophobic membrane , that will not permit dissolved hydrophilic solute to easily pass through the lipids. Whereas hydrophobic chemicals on the other hand willget dissolved into the membrane thatpermits liposome to carry both the hydrophobic and the hydrophilic molecules[23]. The molecules are transferred to the sites of action while the lipid bilayer fuses with the other bilayer such as the cell membrane.There is another way of delivering drugs which is by targeting the endocytosis events. Liposomes would be made as targets for macrophages that are present inside the body. The drugs are released and meanwhile the liposomes are being phagocytosed by the macrophage present in the body. For Endocytosis to take place in other cells , it can be initiated by administrating liposomes decorated with opsonins and ligands[24].The application ofliposomes on theskin surfaceis found to beeffectivein drug deliveryinto the skin.Liposomes tends to increasethe permeabilityof skin formultiple differententrapped drugsand at thesametime diminish theside effect of thesedrugs due to this reasonslower doses are now required.

Treatment ofHumanImmunodeficiency Virus (HIV) Infections

Several antiretroviral nucleotide analogues havebeen developed forthetreatment of patients sufferingfrom the acquired immunodeficiencysyndromes (AIDS). Theseinclude antisense oligonucleotide, which is a new antiviralagent that has shown potential therapeutic application againstHIV-virus.

Applications of Liposome inBleaching

The effect of liposomes was studied in peroxide bleachingon cellulosic and its blendswith polyester and wool fabrics[25].Thetreatment of wool fabric with chlorine-liposomes suspension was alsoreported[26].Awettingagent is required in theconventional bleachingbath ofcotton fabrics, but this step can be eliminated byusingliposomes. Thepresenceofliposomes in theperoxide bleachingbath can improvethe mechanicalproperties offabricsandtheirbrightness

Liposomes in Agro-Food Industry

The capability of Liposomes to solubilize compounds by demanding solubility properties, protect compounds from potentially harmful milieu, and helps in releasing incorporated molecules in a sustained and predictable fashion that can be used also in the food processing industry. For example, lecithin and few other polar lipids are continuously extracted from nutrients, such as egg yolks or soya beans.

Conclusion

As a conclusion, liposomes have been found to have a wide range of uses ever since it was initially noted that it was capable to self-assemble into vesicles. Their capability to form a bilayer liposome made it to be an efficient carrier. Newly formed liposomes, with highly efficient lipid molecules, and fine new formulations has managed to give various possibilities for safely and effectively treating many diseases which includes cancers.Liposomes areusedfordrug targeting,cancer therapies , in textile industryand in manyother fields.Further research is required to explorethe vast functions of liposomesand to get adeeper knowledgeabout thesignificanceof this wonder molecule.

Corresponding Author

Jayanth Kumar V,

Reader, Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai -600077, Tamil Nadu, India

Mobile Number: +91 8056135807,

Email: doctorjayanth@gmail.com

References

1.Kimball'sBiologyPages,"CellMembranes." Stryer S.Biochemistry,1981,213.

2. Grubber SM.Liposome BiophysicsTherapeutics,MarcelDekker:NewYork,1987

3.Nelson, G.(2002).Applicationof microencapsulationintextile.IntJ Pharm242:55–62.

4. Gomes,J.,Genoves,M.C.(1997).Controllingof reactive dyeson woolbymicroencapsulationwithliposome. Textile ResJ67(7):537–41.

5.VyasS.P.&Khar R.K.“Targeted&controlleddrugdelivery:Novelcarriersystem”.CBS publishersanddistributors,2007.

6. Anna Jone. Liposomes: A short Review. J. Pharm. Sci. & Res. 2013; 5(9):181 – 183

7. Amarnath Sharma, Uma S. Sharma, “Liposomes in drug delivery: progress and limitations,” International Journal of Pharmaceutics 1997;154:123 140.

8.RajanK. Verma andSanjayGarg,“CurrentStatusof DrugDelivery TechnologiesandFuture Directions,”PharmaceuticalTechnologyOn-Line. 2001;25 (2):1–14

9.Jesorka, Aldo,andOwe Orwar. Liposomes:Technologiesandanalytical applications. AnnualReviewof AnalyticalChemistry2008; 1:801-32

10. Svetina,S.,andB.Zeks.Shape behavior of lipidvesiclesasthe basisofsome cellularprocesses. The AnatomicalRecord2002; 268:215-25.

11. P. Stano, S. Bufali, C. Pisano, F. Bucci, M. Barbarino,M. Santaniello, P. Carminati, and P. L. Luisi, J. Liposome Res.2004; 14: 87

12. Chunlei, L.,Yingjie, D.Anovel methodfor the preparationof liposomes:Freezedryingofmonophase solutions.JPharmSci 2004; 93(6):1403–14.

13. F. J. Szoka and D. Papahadjopoulos, Proc. Natl. Acad. Sci. USA 1978; 75:4194.

14. Margineanu, D. G. Equilibriumandnon-equilibriumapproachesin biomembrane thermodynamics. ArchivesInternationalesDe Physiologie EtDe Biochimie1987; 95:381-422.

15. Shimomura, Masatsugu, and Tetsuro Sawadaishi . Bottom -upstrategyof materials fabrication: Anewtren dinnanotechnology of soft materials. Current OpinioninColloid&Interface Science (2001)6:11-6.Thevesicle size was foundtobe variedfrom146±0.6nmto198±0.6nm

16. GabizonA. Liposomesas a drugdeliverysystemincancertherapy,in:Drug Carrier Systems,edsF.H.D.RoerdinkandA.M.Kron(Wiley,Chichester).1989;185–211

17. StormG,RoerdinkFH,SteerenbergPA,de JongWHandCrommelinDJA. Influence oflipidcompositionontheantitumor activityexertedbydoxorubicin containing liposomesinaratsolidtumormodel,Cancer Res.1987;47:3366–3372.

18. Sheveleva,I. A.,Belokurova, O. A.Polyfunctionalproperties of liposomesinpreparationoftextile materials.Fibre Chemistry2003; 35(1):48–52.

19. I. Ahmad. “Antibody-Targeted Delivery of Doxorubicin Entrapped in Sterically Stabilized Liposomes Can Eradicate Lung Cancer in Mice,” Cancer Res. 1993; 53: 1484–1488.

20. G. Lopez-Berestein et al., “Liposomal Amphotericin B for the treatment of Systemic Fungal Infections in Patients with Cancer: A Preliminary Study,” J. Infect. Dis. 1985; 151:704–710.

21. R. M. Fielding, Clin. Pharmacokinet 1991; 21:155.

22. J.S. Dua, Prof. A. C. Rana, Dr. A. K. Bhandari, “LIPOSOME: METHODS OF PREPARATION AND,” International Journal of Pharmaceutical Studies and Research. E-ISSN 2229-4619.
23. Renuka Devi SK. Immunotherapy Monitoring Through Liposomes-An Altered Form of Bio-Sensing. J Aller Ther 2012; 3:122. doi:10.4172/2155- 6121.1000122
24. Abhijit Ray “Liposome in Drug delivery system,” Asian J. Res. Pharm. Sci. 2012; 2(2):41-44
25. dela Maza, A.,Parra J. L. Usingliposomesinwool chlorination:Stabilityofchlorineliposomesandtheir applicationonwoolfibers. Textile ResJ1991; 61(6):357–62
26. Kling, A.,Hofstetter, V.(1975).Bleachingof cellulose containing textile fiber withasilicate-free stabilizedperoxidebleachingbath. UnitedStates Patent,3810391.

Liver Fibrosis

Sangeetha Shankar[1], Gowri Sethu[2], E. Roja[3],

Graduate Student[1], Professor[2], Department of Physiology, Tutor[3], Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Liver fibrosis is defined as the excessive accumulation of extracellular matrix proteins including collagen. It represents a major worldwide health care burden (1). It represents a significant health problem worldwide of which no acceptable therapy exists (2) and not just that liver fibrosis is a major cause of morbidity and mortality worldwide due to chronic viral hepatitis and more recently from fatty liver disease associated with obesity (3). Progressive liver fibrosis is the main cause of organ failure in chronic liver diseases of any etiology. The primary source of the extracellular matrix that is responsible for fibrosis is the myofibroblast. Hepatic myofibroblasts are not present in the normal liver but transdifferentiate from heterogeneous cell populations in response to a variety of fibrogenic stimuli. It is usually considered that hepatic stellate cells and portal fibroblasts have fibrogenic potential and are the major origin of hepatic myofibroblasts. But there are at least three potential sources of myofibroblasts in the liver:

1. Resident mesenchymal cells (consisting of the quiescent hepatic stellate cell and portal fibroblasts).
2. Hepatocytes, cholangiocytes and endothelial cells.
3. The bone-marrow derived cells.

Advanced liver fibrosis usually results in liver cirrhosis, liver failure and portal hypertension and often requires liver transplantation. It is considered that the hepatic cells and portal fibroblasts have fibrogenic potential which serves as a thmajor origin of hepatic myofibroblasts. Although many therapeutic interventions are effective in experimental models of liver fibrosis, their efficacy and safety in humans is unknown.

Key Words: *Myofibroblast, Liver, Fibrosis, Hepatic, Fibroblasts*

Introduction to Liver Fibrosis

Liver fibrosis represents a major worldwide health care burden (1). It represents a significant health problem worldwide of which no acceptable therapy exists (2) and not just that, liver fibrosis is a major cause of morbidity and mortality worldwide due to chronic viral hepatitis and more recently from fatty liver disease associated with obesity (3).

Liver fibrosis is the excessive accumulation of extracellular matrix (ECM) proteins including collagen (scar tissue) that occurs in most types of chronic liver diseases (4-6).Advanced fibrosis results in cirrhosis and is characterized by an accumulation of extracellular matrix (ECM) rich in fibrillar collagens. Although various types of collagen are involved in liver fibrosis, type IV is said have a major role (7). It results in liver failure and portal hypertension and is associated with an increased risk of liver cancer (2). Progressive liver fibrosis is the main cause of organ failure in chronic liver diseases of any etiology. The accumulation of ECM proteins distorts the hepatic architecture by forming a fibrous scar and the subsequent development of nodules of regenerating hepatocytes defines cirrhosis (5,8). Cirrhosis will in turn result in hepatocellular carcinoma (9). Myofibroblasts are the cells which form the extracellular matrix (mainly Type-1 collagen fibres), which is responsible for the fibrous scar in liver fibrosis (10, 11). The main causes of liver fibrosis and cirrhosis in industrialized countries include chronic HCV infection, alcohol abuse and nonalcoholic steatohepatitis (NASH) (5, 12). Advanced liver fibrosis results in cirrhosis, liver failure and portal hypertension and often requires liver transplantation. It is considered that hepatic stellate cells (HSC's) and myofibroblasts play a major role in liver fibrosis.

Stages of Liver Damage

The following are the stages of liver damage or failure (13):-

Stage 1. Fatty Liver

Stage 2. Liver Fibrosis

Stage 3. Liver Cirrhosis

Stage 1. Fatty Liver

In this stage, there is excessive fat accumulation. This may usually be due to chronic alcoholism. This prevents the breakdown of fat which ultimately gets accumulated in large amount and casue liver enlargement.

Stage 2. Liver Fibrosis

As already stated above there is excessive accumulation of extracellular matrix proteins including collagen. In detail, this topic will be discussed below.

Stage 3. Liver Cirrhosis

Long standing liver fibrosis will result in liver cirrhosis. This will result in reduced or retarded liver function along with reduced blood supply causing death of liver tissues and ultimately liver failure.

Liver Fibrosis- Mechanism

As already disccused above, liver fibrosis is due to abnormal proliferation and accumulation of fibrous connective tissue (scar tissue) in the liver (14). It is quite normal for our body to undergo healing process in reponse to an injury and result in scar tissue formation. But in fibrosis, there is chronic or increased activation of this scarring tissue formation i.e collagen deposition. This is stimulated by a wide variety of factors such as chronic alcohilsm, the most common factor, others include chronic infection by hepatitis B, C viruses and parasite Schistosoma, certain drugs and toxins, infections, non-alcoholic steatohepatitis (NASH), inherited disorders like Wilson's disease, α-1 antitrypsin deficiency, autoimmune diseases, etc (14,15). It begins with the stimulation of chemical messengers such as cytokines, etc which will activate hepatic stellate cells (HSCs) which in turn will cause synthesis of collagen, glycoproteins, etc. All this along with the impairment of collagenolysis will result in increased extracellular matrix deposition resulting in liver fibrosis.

Stages Of Liver Fibrosis

The following are the stages in liver fibrosis (13) :-

Stage 0: which indicates no fibrosis.

Stage 1: which indicates enlargement of the portal areas by fibrosis.

Stage 2: which indicates fibrosis extending out from the portal areas.

Stage 3: which indicates a stage of fibrosis that link up portal and central areas of the liver.

Stage 4: which indicates liver cirrhosis.

Hepatic Stellate Cells (Hsc's)

These cells play a major role or infact are responsible for liver fibrosis. They are also known as fat-storing cells and generally represent 5-8% of all healthy liver cells (14). These cells are mainly located near the hepatocyte in the perisinusoidal space of Disse by means of their dendritic cytoplasmic processes extending along and around endothelial cells. Basically, they exhibit two phenotypes- quiescent and activated hepatic stellate cells. Quiescent HSC's are star shaped and their cytoplasm contains various components like lipid droplets, which contain retinoids, triglycerides, cholesterol, and free fatty acids . In fact, storage and limited release of retinoids is a major function of HSC in the healthy liver. A number of stimuli such as chronic alcohilsm, viral infections, etc, as already discussed above, will stimulate the conversion of quiescent to active HSC's. These cells later develop into myofibroblast-like cell types, which is carried out mainly by the loss of lipid droplets and other features such as lack of glial fibrillary proteins and increased proliferation of cells (14, 17).

Molecular Basis Of Liver Fibrosis

Liver fibrosis is basically a resultant of increased collagen deposition and decreases collagen destruction which in turn results in increased extracellular matrix.

It has two main phases- inflammation and fibrogenesis (14). Hepatotoxic factors like alcoholism, etc induces the synthesis of chemical mediators like cytokines, etc which will cause inflammatory reactions within hepatic cells converting them into active HSC's from quiescent HSC's (14). These activated HSC's, during their transformation process, becomes proliferative and morphologically begins to appear like myofibroblasts, which are devoid of lipid droplets, lack glial fibrillary-acidic protein and cause excessive production of ECM components (14).

Activated HSCs migrate towards regions of injury, in reponse to the chemical messengers

released from the site of injury and start accumulating around damaged tissue. Activated HSCs express the following : cytoskeleton protein, α-smooth muscle actin (α-SMA) and various connective tissue proteins including collagen types I, III, and IV (14,18-20). In addition to all of the above, liver fibrosis exhibits increased levels of tissue inhibitors of matrix metalloproteinases (TIMPs). These in turn are capable of inhibiting the action of MMP-1 (interstitial collagenase), thereby causing the accumulation of collagen fibers.

Myofibroblasts

Myofibroblasts are another group of cells that also play a major role in liver fibrosis. They are a unique group of smooth-muscle-like fibroblasts that have a similar appearance and function. In other words they are alpha smooth muscle actin positive cells. Through the secretion of extracellular matrix proteins (including fibrillar collagen) and proteases, they play a very important role in liver fibrosis and organogenesis, oncogenesis, inflammation repair in most organs and tissues. That produce extracellular matrix proteins. Myofibroblasts are the cells that are prominent in liver fibrosis (21-23).

They are generally characterized immuno-phenotypically by spindle or stellate shape, pale eosinophilic cytoplasm, expression of abundant pericellular matrix and fibrotic genes (vimentin, a-smooth muscle actin (a-SMA), non-muscle myosin, fibronectin and collagen Type I). Ultrastructurally, myofibroblasts are defined by prominent rough endoplasmic reticulum, a golgi apparatus producing collagen, peripheral myofilaments, fibronexus (no lamina) and gap junctions . In liver fibrosis, the myofibroblasts are imbedded in the fibrous scar. (21-23)

In both experimental and clinical liver fibrosis, there is a close correlation between the regression of liver fibrosis and the disappearance of these myofibroblasts. There is a general fact that these myofibroblasts are ultimately responsible or act as the source of excessive extracellular matrix proteins in liver fibrosis. Therefore, identifying the origin of these myofibroblasts will provide information about the pathology of liver fibrosis and perhaps even about new therapeutic targets.

Origin of Myofibroblasts

The origin of myofibroblasts in liver fibrosis is stillquestionable and debatable, although morphologic evidence has suggested that they are derived from fat storing cells-lipocytes (24).

There are at least three potential sources of myofibroblasts in the liver:

1. Resident mesenchymal cells (consisting of the quiescent hepatic stellate cell and portal fibroblasts).
2. Hepatocytes, cholangiocytes and endothelial cells.
3. The bone-marrow derived cells.

1. Resident Mesenchymal Cells (The Quiescent Hepatic Stellate Cells – Hsc And The Tissue Fibroblasts)

The resident mesenchymal cells, which mainly consists of the quiescent hepatic stellate cells (HSC) and the tissue fibroblasts, can become myofibroblasts. These cells are characterized by cell markers like CD45-, CD34-, desmin+, glial fibrillar associated protein (GFAP)+ and thy-1+ (25). Fibroblasts are primarily located in the portal tract in the normal liver (26,27). Recent studies (28) had demonstrated that thy-1 is a potential marker of activated myofibroblasts in the injured liver. Many studies have demonstrated an overlap in experimental fibrosis between thy-1 and alpha smooth muscle actin, indicating that some myofibroblasts are derived from fibroblasts in liver fibrosis. Studies from other researchers have proposed that TE-7 (Human Thymic Fibroblasts Antibody), an antibody against elastin, specifically identifies fibroblasts in the liver (29,30).

As already stated above, generally in a normal liver, HSCs reside in the space of Disse and they are the major storage sites of vitamin A. Following a chronic injury, hepatic stellate cells activate or gets transdifferentiated into myofibroblast-like cells, acquiring the contractile, pro-inflammatory and fibrogenic properties. These activated HSCs will migrate and accumulate at the sites where tissue repair is required, start secreting large amounts of ECM and regulate ECM degradation (31,32).

Those myofibroblasts which are derived from small portal vessels will proliferate around biliary tracts in cholestasis-induced liver fibrosis in order to initiate collagen deposition (23,

33) HSCs and portal myofibroblasts differ in specific cell markers and response to apoptotic stimuli (34). Several markers have been proposed to be specific for hepatic stellate cells, whether in the quiescent or activated state. These markers include florescence of Vitamin A in lipid droplets, GFAP, p75 NGF receptor, and synaptophysin (35-37). Using these markers, it is possible for one to distinguish between myofibroblasts that originate from fibroblasts or from hepatic stellate cells in experimental liver fibrosis.

2**. Bone-Marrow Derived Cells (Fibrocytes And Circulating Mesenchymal Cells)**

Bone-marrow derived cells, consisting of fibrocytes and circulating mesenchymal cells, can also be recruited to the injured liver to become myofibroblasts. These cells are CD45+ (fibrocytes), CD45+/- (circulating mesenchymal cells), collagen type I +, CD11d+ and MHC class II+.

Culture of CD34+CD38– hematopoietic stem cells with various growth factors has been shown to generate HSCs and myofibroblasts of bone marrow origin that infiltrate human livers undergoing tissue remodelling (38,39). These data suggest that cells originating in bone marrow can be a source of fibrogenic cells in the injured liver.

3. **Hepatocytes, Cholangiocytes, And Endothelial Cells**

Few reccent studies have proposed the fact that hepatocytes, cholangiocytes, and endothelial cells can actually become myofibroblast through epithelial or endothelial mesenchymal transition (EMT). These cells include CD45-, albumin+ (i.e. hepatocytes), CD45-, CK19+ (i.e. cholangiocytes) or Tie2+ (endothelial cells) (25).

Anti-Fibrotic Drugs And Liver Fibrosis

The high prevalence of insults with the potential to cause liver fibrosis, including chronic viral hepatitis, non-alcoholic steatohepatitis, toxic damage through alcohol consumption, etc indicates that fibrosis and cirrhosis of the liver remain major causes of morbidity and mortality worldwide (40). Hepatic Fibrosis and cirrhosis represent the consequences of a sustained wound healing response to chronic liver injury from a variety of causes including viral, autoimmune, drug induced, cholestatic and metabolic diseases (41).

Extensive research is being conducted and performed primarly to understand the mechanisms responsible for the development of liver fibrosis. Few drugs of liver fibrosis are effective in experimental models but their safety and efficacy in case of humans is questionable.

Conclusion

Liver fibrosis represents a significant health problem worldwide and is a major cause of morbidity and mortality across the globe. From the above, it can be concluded that liver fibrosis is a very complex disease. Not just medical professionals but even the general population should be made aware of this medical condition. Hence, in that aspect this article will be useful in understanding about liver fibrosis.

Corresponding Author

E. Roja,

Tutor**,** Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India.

Email Id: itsrojaa@gmail.com

Phone Number: +91 9994174480

References

1. Virginia Hernandez-Gea and Scott L. Friedman. Pathogenesis of Liver Fibrosis. Annual Review of Pathology. 2011; 6: 425-456.
2. Shigeki Tsukada , of liver fibrosis. Invited critical review . 2006, 2005;364(1):33–60.
3. Hernandez-Gea V1 and Friedman SL. Pathogenesis of liver fibrosis. Annu Rev Pathol . 2011; 6: 425-56.

4. Bataller R1 and Brenner DA. Liverfibrosis. JClin Invest . 2005;115(2):209-18.
5. Ramón Bataller and David A. Brenner. Liverfibrosis. JClin Invest . 2005; 115(4): 1100.
6. Friedman SL. Liver fibrosis - from bench to bedside. *J. Hepatol.* 2003; 38(Suppl. 1):S38–S53.
7. Emer Fitzpatrick and Anil Dhawan. Type IV Collagen: A Non Invasive Bio Marker To Detect Non - Alcoholic Steato Hepatitis (NASH), Among Non - Alcoholic Fatty Liver Disease (NAFLD) Patients. World J Gastroenterol. 2014 ; 20(31): 10851–10863.
8. Surapaneni K M, Saraswathi P, Shyama S, Subramaniam S. Type IV Collagen: A Non Invasive Bio Marker to Detect Non - Alcoholic Steato Hepatitis (NASH), Among Non - Alcoholic Fatty Liver Disease (NAFLD) Patients. Journal of Clinical and Diagnostic Research, 2010; 1(4): 2483-2488.
9. Savitha.G, Vishnupriya.V, Surapaneni Krishnamohan. Hepatocellular Carcinoma- A Review. J. Pharm. Sci. & Res. 2017; 9 (8): 1276-1280
10. Albanis E, Friedman SL. Hepatic fibrosis. Pathogenesis and principles of therapy. *Clin. Liver Dis.* 2001;5:315–334, v–vi.
11. Alcolado R, Arthur MJ, Iredale JP. Pathogenesis of liver fibrosis. Clin Sci (Lond). 1997; 92: 103 – 12.
12. Friedman S.L. Molecular regulation of hepatic fibrosis, an integrated cellular response to tissue injury. J.BiolChem . 2000; 275: 2247–2250.
13. Stages of Liver Disease. Available from URL: http://mysickliver.weebly.com/mdash-stages.html
14. Areeba Ahmad and Riaz Ahmad. Understanding the Mechanism of Hepatic Fibrosis and Potential Therapeutic Approaches. Saudi J Gastroenterol. 2012; 18(3): 155–167.
15. Friedman SL. Mechanism of hepatic fibrogenesis. Gastroenterology 2008;134:1655-69.
16. Geerts A. On the origin of stellate cells: mesodermal, endodermal or neuroectodermal. J Hepatol 2004;40:331-4.

17. Vogel S, Gamble MV, Blaner WS. Biosynthesis, absorption, metabolism and transport of retinoids. In: Nau H, Blaner WS, editors. Retinoids: The biochemical and molecular basis of vitamin A and retinoid action. Berlin: Springer; 1999. p. 31-95.

18. Pinzani M, Marra F. Cytokine receptors and signaling in hepatic stellate cells. Semin Liver Dis 2001;21:397-416.

19. Maher JJ, Lozier JS, Scott MK. Rat hepatic stellate cells produce cytokine-induced neutrophil chemoattractant in culture and *in vivo*. Am J Physiol 1998;275:G847-53.

20. Lotersztajin S, Julien B, Teixeira-Clerc F, Grenard P, Mallat A. Hepatic fibrosis: molecular mechanisms and drug targets. Annu Rev Pharmacol Toxicol 2005;45:605-28.

21. John P. Iredale. Models of liver fibrosis: exploring the dynamic nature of inflammation and repair in a solid organ. JClin Invest . 2007 Mar 1; 117(3): 539–548.

22. Watsky MA.,New insights into the mechanism of fibroblast to myofibroblast transformation and associated pathologies. Int Rev Cell MolBiol . 2010, 282: 165-192.

23. Eyden B. The myofibroblast:phenotypic characterization as a prerequisite to understanding its functions in translational medicine. J Cell Mol Med . 2008, 12: 22-37.

24. David A Brenner, Origin of myofibroblasts in liver fibrosis. Fibrogenesis & Tissue Repair . 2012, 5(Suppl 1): S17 doi:10.1186/1755-1536-5-S1-S17

25. Magness ST, A dual reporter gene transgenic mouse demonstrates heterogeneity in hepatic fibrogenic cell populations. Hepatology . 2004, 40: 1151-1159.

26. Bataller R, Brenner DA. Hepatic stellate cells as a target for the treatment of liver fibrosis. *Semin. Liver Dis.* 2001;21:437–451.

27. Marra F. Hepatic stellate cells and the regulation of liver inflammation. *J. Hepatol.* 1999;31:1120–1130.

28. Dudas J, Thy-1 is an in vivo and in vitro marker of liver myofibroblasts. Cell Tissue Res . 2007, 329: 503-514.

29. Wells RG, Autocrine release of TGF-beta by portal fibroblasts regulates cell growth. FEBS Lett . 2004, 559: 107-110.

30. Dranoff JA, Wells RG. Portal fibroblasts:Underappreciated mediators of biliary fibrosis. Hepatology . 2010, 51: 1438-1444.

31. Milani S, Procollagen expression by non-parenchymal rat liver cells in experimental biliary fibrosis. Gastroenterology . 1990. 98: 175-184.

32. Marra F. Hepatic stellate cells and the regulation of liver inflammation. J. Hepatol . 1999. 31:1120-1130.

33. Kinnman N, Housset C. Peribiliary myofibroblasts in biliary type liver fibrosis. Front Biosci . 2002; 7: d496–d503.

34. Knittel T, Rat liver myofibroblasts and hepatic stellate cells: different cell populations of the fibroblast lineage with fibrogenic potential. Gastroenterology . 1999; 117: 1205–1221.

35. Geerts A:History, heterogeneity, developmental biology and functions of quiescent hepatic stellate cells. Semin Liver Dis . 2001, 21: 311-335.

36. Senoo H , Vitamin A-storing cells (stellate cells). VitamHorm . 2007, 75: 131-159.

37. Forbes SJ., A significant proportion of myofibroblasts are of bone marrow origin in human liver fibrosis. Gastroenterology . 2004; 126: 955–963.

38. Iredale J.P. Cirrhosis: new research provides a basis for rational and targeted treatments. BMJ . 2003. 327:143–147.

39. Suskind DL, Muench MO. Searching for common stem cells of the hepatic and hematopoietic systems in the human fetal liver: CD34+ cytokeratin 7/8+ cells express markers for stellate cells. J. Hepatol . 2004; 40: 261–268.

40. Iredale J.P. Cirrhosis: new research provides a basis for rational and targeted treatments. BMJ . 2003. 327:143–147.

41. Fattovich G., Morbidity and mortality in compensated cirrhosis type C: a retrospective follow-up study of 384 patients. Gastroenterology 112 (1997) 463-472.

Local Anaesthesia Failure in Endodontics

Anandadeeban K B S [1], Balakrishna [2]
Graduate Student [1], Senior Lecturer [2],Department of Oral and Maxillofacial Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Science,
Saveetha University, Chennai,India

Abstract

Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage Local anaesthesia is essential for successful completion of endodontic treatment. Although pain treatment is well managed in many endodontic patients, there exists a group of patients who do not receive adequate local anaesthesia inspite of proper administration of local anaesthesia.This article reviews about the reasons for failure of local anaesthesia during endodontic treatment.

Key Words:Local anaesthesia,pain,mandibular molars, inflammation

Introduction

During dental procedure localanaesthesia plays a major role in controlling the pain.They are the safest and most effective drugs in all of medicine for the prevention and management of pain during dental procedure.Extraction of teeth, root canal treatment, minor surgical procedures and periodontal procedures,tooth preparation mandatorily need administration of a local anaesthetic in order to minimize patient discomfort and make patient co-operative during dental treatment[1]. Fear of pain is the main issue which causes patients to refuse dental treatment. Pain is defined as an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. This discomfort signals actual or potential injury to the body[2].The success of local anaesthetic administration depends on various factors like such as site of administration if local anaesthesia, injection techniques, dosage and allergic complications. Many studies saysthat local anaesthetic failures are more likely to occur because of choosing improper.site for injection and inappropriate techniques to administer it.. [3].

It has been estimated that about 20% of patients experience moderate- to- severe pain after treatment [4].Very few patientswill experience a sudden ‘flare- up’ of severe pain or swelling after endodontic treatment has been over. Thus clinical management of endodontic patients is often problematic due to inadequate local anaesthesia.

Various Reason for Failure of Local Anaesthesia

1) Anatomical Factors

Few suggest that the inability of the operator to load the localanaesthesia in close proximity to the corresponding nerve would lead to inadequate blockade in both normal and uninflamed states, it may be possible that a partial blockade would be adequate in neurons that were not sensitized by inflammatory mediators. Thus, it is important to know the nerve supply to the tissue to be anesthetized, as well as the anatomy of the injected site and its variations present.Anatomic variation would have a lesser impact on infiltration anaesthesiawhich is commonly used in maxilla.

Usually, the pulps of mandibular teeth will anesthetized by blocking of the inferior alveolar nerve via an intraoral approach to deliver the local anaesthetic to the pterygomandibular

space. In the classic technique, the needle is advanced to a point where a pool of anaestheticis deposited near the mandibular foramen, which lies below the lingula, and in the sulcus colli mandibulae[5]. As the bony prominence of the lingula projects medially, it is often difficult to place the tip of the needle in the sulcus colli, and it has been suggested that the bevel should be orientated towards the midline in order to take advantage of the lateral deflection that would be provide via tissue resistance . However, even when needle placement is given with proper technique there occur the failures of local anaesthesia.This may be due to the erratic post- injection distribution of anaesthetic solution in the pterygomandibular space over which the operator has no control [6]

Accessory innervation to the mandibular teeth from several sources has also been known as the cause for inadequate localanaesthesia. In particular, the nerve tothe mylohyoid muscle has been implicated in carrying afferent fibers from the mandibular teeth. In a study of 37 cadavers, it has been found the point at which the mylohyoid nerve branched from the inferior alveolar nerve to be an average of 14.7mm above the foramen of mandible a distance which is sufficient to prevent a blockade of the mylohyoid nerve when the classic technique is used. To overcome accessory innervation from the mylohyoid nerve, the clinician has several options, including the use of a block technique that deposits anaesthetic solution higher in the pterygomandibular space (i.e. Gow- Gates or Akinosi), infiltration on the lingual surface of the mandible adjacent to the tooth operated, or techniques that deposit anaesthetic solution in the medullary space surrounding the operated tooth, such as the intraligamentary or intraosseous routes of injection.Lingual nerve, buccal nerve are the other nerves which gives stimulus to the mandibular teeth.Regardless of the origin, the technique that would predictably block allsources of accessory innervation to the mandibular teeth would be one in which the anaesthetic solution is deposited at the apices of the teeth in question (i.e. intraligamentary or intraosseous routes). Even though both of these techniques appear to increase the efficacy of inferior alveolar block anaesthesia, research show a greater duration of lower molar pulpal anaesthesia (as determined by a reading of 80 on an electric pulp tester) to be provided by the intraosseous technique [7].

2. Effect of Local Tissue Inflammation.

Usually local anaesthetics diffuse through the cell membrane and then block the sodium channel by accessing the protein from the cell's cytoplasm. This action requires the shift of

drug between its acid form (an ionized or charged molecule) and its base form (an uncharged molecule). The pH of most local anaesthetics in cartridge form is purposefully low (pH = 3–4), because the charged, acid form of the molecule is more stable (as is the vasoconstrictor) at a low pH, and thus gives a longer shelf life [8].Once injected, the local tissue pH and the drug's strength as an acid regulate the distribution of the local anaesthetics between the acid and base forms according to the well- known Henderson- Hasselbalch equation.The proportion of the drug that exists in the uncharged base form is available to diffuse across the cell membrane. As it enters the cell, the drug repartitions into the acid and base forms, and it is the acid form of the drug that blocks the sodium channel.

This becomes as an potentially important issue because inflammation- induced tissue acidosis may cause ‘ion trapping’ of local anaesthetics. According to this hypothesis, the low tissue pH will result in a greater proportion of the local anaesthetics being trapped in the charged acid form of the molecule and, therefore, unable to cross cell membranes. This has been advanced as a major mechanism for failures of local anaesthesia in conditions such as endodontic pain .The reduction in tissue pH results in a substantial proportion of the drug being trapped in the charged acid form. Thus, over the pH range of 7.4–6.6, in comparison to lidocaine or bupivacaine, mepivacaine is relatively resistant to ion trapping. To the extent that this hypothesis explains local anaesthetics failure, mepivacaine represents a logical local anaesthetics for use in patients with irreversible pulpitis.

The relationship between the proportions of local anaesthetics in the cationic acid form of the drug as a function of tissue pH. Note that the cationic acid form cannot diffuse across cell membranes and is referred to as the ‘ion trapped’ proportion of the molecule. This proportion is derived from the Henderson- Hasselbach equation and the pKa value for each drug.

However, there are considerations that may limit the local pH hypothesis. First, the acidosis may be minor in magnitude. Although severe forms of liquefaction necrosis may have pH levels as low as 4 to 5, the affected area is restricted to the actual abscess. Studies on cutaneous inflammation indicate that tissue pH may be only marginally reduced to pH values of about 5.8–7.2 [9]. In addition, inflamed tissue possesses greater buffering capacity than normal tissue (possibly due to extravasation of protein or erythrocytes into the inflamed tissue). Thus, the actual pH change may not be large enough to produce substantial ion trapping of local anaesthetics. In addition, a reduction in tissue pH is likely to be a localized event and, with the exception of mandibular second and third molars, most probably does not

involve distinct facial space compartments that isolate the site for an IAN nerve block from the mandibular teeth. Thus, even in severe forms of inflammation, local tissue pH may explain problems with infiltration anaesthesia in maxillary teeth, but is unlikely to explain local anaesthetics failures in nerve block anaesthesia.

To the extent of its validity, the local pH hypothesis has at least two clinical implications. Firstly, it suggests that local anaesthetics with lower pKa values are likely to be more effective in endodontic pain patients. This recommendation is based on the physical properties and available formulations of these drugs, and it should be evaluated in a prospective clinical trial. Secondly, the temporary adjustment of tissue pH may be used to augment clinical anaesthetics. This strategy has been employed by anaesthesiologists with sodium bicarbonate to alkalinize the local anaesthetics and tissue pH and thereby enhance local anesthesia. Addition of sodium bicarbonate also raises the pCO2 of the anesthetic solution bathing the nerve. When CO2 crosses the nerve membrane and decreases the intracellular pH, the ionized form of the drug is favoured, and as mentioned previously, it is this form that binds to the sodium channel to effect blockade.

Although alkalinisation may have theoretical utility, there is a paucity of clinical trials in dental pain patients to support its use. In one study, compared with a standard lidocaine formulation, a buffered lidocaine formulation demonstrated no significant difference when given by infiltration injection into inflamed maxillary incisors [10]. Although other formulations may warrant testing in additional studies, there does not appear to be a preponderance of clinical evidence to support the use of alkalinisation of local anesthetic solutions

3.Effect of Inflammation on Central Sensitization

Inflammation also induces changes in the central nervous system's pain processing system. Activation and sensitization of nociceptors in pulpal and periradicular tissue results in a barrage of impulses sent to the trigeminal nucleus and brain. This barrage, in turn, produces central sensitization. Central sensitization is the increased excitability of central neurons and is thought to be a major central mechanism of hyperalgesia [11]. Under conditions of central sensitization, there is an exaggerated CNS response to even gentle peripheral stimuli. A common example to this is sunburn, where even the innocuous stimulation of wearing a t- shirt is considered painful. Similarly, percussing a tooth with an inflamed periodontal

ligament (e.g. acute apical periodontitis) may produce an exaggerated pain response which is due, in part, to central sensitization.

Although we often consider central sensitization when discussing endodontic pain mechanisms (65), this same process may contribute to local anesthetic failures. Under normal conditions, many patients tolerate dental procedures, even though a slight or occasional sensation may still be felt. In other words, under normal conditions, a local anesthetic injection that blocks most of the fibers (say, 90%) may still be clinically successful. This has been reported in other clinical models (for example, IV cannulation of the arm), where patients treated with a topical anesthetic reported that they did not experience pain, even though their visual analog pain scores were greater than zero [12]. However, under conditions of central sensitization, there is an exaggerated response to peripheral stimuli and, under these conditions, the same 90% block may permit sufficient signaling to occur to lead to the perception of pain. Thus, central sensitization may contribute to local anesthetic failures.

Unfortunately, there are no selective drugs for blocking central sensitization. The only clinical implication would be to reduce the afferent barrage and thereby reduce central sensitization. This is done routinely by clinicians via cleaning and shaping techniques, but this is a conundrum, as the endodontic treatment is performed after local anesthesia. One interesting study has demonstrated that intraosseous injection of steroid (methylprednisolone acetate 40 mg) reduces endodontic pain in 24 h . If confirmed, then this approach may reduce peripheral and central mechanisms sufficiently to obtain predictable local anesthesia.

4.Psychological Factors

Patient anxiety may also contribute to local anesthetic failure. Experienced clinicians understand that apprehensive patients have a reduced pain threshold and are more likely to report an unpleasant dental experience [13] Fear of seeing and/or feeling the needle and the sound of the dental handpiece are routinely cited as causative agents in the creation of anxiety in the dental patient. Moreover, patients may be particularly anxious about impending root canal therapy. Investigators have also demonstrated that patient anxiety predicts a poor outcome for clinical procedures involving local anaesthetics applied to the arm before IV cannulation .Thus, patient anxiety should be considered when managing the endodontic pain patient.

Several methods have been advocated for managing anxious emergency pain patients. First, the clinician should establish a positive and confident relationship and avoid exposing the

patient to obvious fear- producing stimuli. Many clinicians report that a sense of humor often helps to relax apprehensive patients. For extremely fearful patients, cognitive behavior- based programs have shown significant long- term reduction in predental treatment anxiety. Other studies have demonstrated that instructing patients to focus on sensory stimuli significantly reduces intraoperative endodontic pain. This effect was most evident in patients who were characterized as having a high desire for control and a low perceived control over their clinical care.

Second, pharmacologic agents can be administered to control patient anxiety. While these agents can be delivered via oral, inhalation (N2O) or intravenous routes, a decreased likelihood of serious morbidity, reduced monitoring and demonstrated efficacy have made oral or a combination of oral and inhalation routes attractive [14]. Kaufman et al showed that oral triazolam 0.25 mg was equally effective in comparison to intravenous diazepam in reducing anxiety in patients undergoing oral surgery.

One could certainly consider an integrated approach that involves both non- pharmacologic and pharmacologic techniques. Regardless of the technique utilized, providing some means of anxiety control should enhance the clinician's ability to provide adequate local anesthesia for endodontic pain patients.

5. Acute Tachyphylaxis of Local Anaesthetics

It is well known in pharmacology that administration of receptor agonist drugs often leads to reduced responsiveness to a subsequent administration of the drug,an effect called tachyphylaxis. Since local anaesthetics are often administered together with vasoconstrictors, there is the possibility that the drug persists in the tissue for a sufficient amount of time to produce tachyphylaxis at the sodium channel. It has been proposed that this contributes to reduced anesthetic effectiveness, especially after repeated injections.

However, it is not clear that local anaesthetics produce substantial, or in fact any, tachyphylaxis under clinical conditions. Several clinical trials have evaluated repeated or continuous local anesthetic administration to treat chronic pain patients. Despite continuous infusion or daily administration for periods of up to several years, these studies have not reported tachyphylaxis to local anaesthetics[15].Thus, this hypothesis may have comparatively little merit for explaining local anesthetic failures.

6. Effect of Nociceptors Inflammation

Substances released from inflamed tissue have two major effects on nociceptive (‘pain detecting’) neurons [16]. Firstly, they change the functional activity of these neurons. As might be expected, nociceptors are thought to be quiescent throughout much of our lives and only discharge in the presence of stimuli strong enough to damage the tissue or chemicals that stimulate receptors on these neurons. Inflammatory mediators activate or sensitize these neurons by interacting with specific receptors. An example of a mediator that activates nociceptors is bradykinin: its administration causes a brisk firing of unmyelinated C nociceptors via activation of cell surface bradykinin receptors (BK1 or BK2). Prostaglandin E2 is an example of a mediator that sensitizes nociceptors: administration of PGE2 reduces the threshold for firing to the point where gentle stimuli can now activate these neurons. For example, the throbbing nature of pulpal pain is thought to be due to pulpal nociceptors sensitized to the point where they discharge in response to the patient's heartbeat. Thus, activation and sensitization are two major mechanisms by which inflammatory mediators alter the activity of these normally quiescent neurons. Although local anaesthetics display use- dependent blockading properties, peripheral sensitization and activation have been reported to cause an increase in the resistance of nerves to anaesthetics.

In addition, inflammatory mediators, including certain growth factors, have profound effects on these neurons by altering their structural properties. In particular, the elegant studies by Byers and her colleagues have led to the realization that the terminals of peripheral nerves literally grow (‘sprout’) into areas of inflammation in dental pulp and periradicular tissue ([17]. Clinical studies have confirmed that a similar sprouting occurs in inflamed human dental pulp. This increase in nerve terminals in inflamed tissue increases the size of their receptive field, indicating that pain neurons may now be more easily activated by a spatial summation of stimuli.

Inflammation also changes the synthesis of several proteins in nociceptors, leading to an increase in neuropeptides, such as substance P and calcitonin gene- related peptide. These neuropeptides play important roles in regulating pulpal inflammation. In addition, tissue injury may alter the composition, distribution or activity of sodium channels expressed on nociceptors. The effect of inflammation on these sodium channels may have profound implications in local anesthetic failures.

Several types of sodium channels have been discovered over the last decade. One particular group of channels is characterized as being resistant to the puffer fish toxin, tetrodotoxin (TTX). At least two channels are members of the TTX- resistant class, including the PN3

(also known as SNS, or NaV 1.8) and NaN (also known as the SNS2 or NaV 1.9) sodium channels. The TTX- resistant class of sodium channels is of interest since they are less sensitive to lidocaine [18]. The TTX- resistant class of sodium channels is expressed on nociceptors under normal conditions. In addition, their activity more than doubles after being exposed to prostaglandin E2.Thus, we hypothesize that the TTX- resistant class of sodium channels represents a logical mechanism for local anesthetic failures: the channels are relatively resistant to lidocaine, they are expressed on nociceptors, and their activity is increased with PGE2.

7.Effect of Inflammation on Blood Flow

Inflammation has several other effects on local tissue physiology. For example, it has been proposed that peripheral vasodilation induced by inflammatory mediators would reduce the concentration of local anaesthetics by increasing the rate of systemic absorption [19].This is a potentially important mechanism, because local anaesthetics are well- recognized vasodilators that, in most cases, require formulation with vasoconstrictor agents. Although inflamed dental pulp experiences regional changes in blood flow, less is known about inflammation- induced vascular changes in periradicular tissue. Moreover, it is likely that this vasodilation may be localized and not evident at distant sites of injection (i.e. nerve block injection sites). Thus, this hypothesis may have greater utility in explaining difficulties with infiltration anesthesia when compared with nerve block anesthesia.

To the extent that this hypothesis predicts local anesthetic failure, there are clinical implications that may improve the success of local anesthesia. If vasodilation leads to increased drug absorption, then the use of higher concentrations of vasoconstrictors may produce more profound or longer duration anesthesia. Thus, in patients who can tolerate it, the use of 1:50000 epinephrine may improve clinical success in anesthetizing patients with endodontic pain. However, to date, the results from clinical trials have been equivocal. The use of 1:50000 epinephrine produces a greater degree of vasoconstriction in patients than 1: 100000 epinephrine [20]and yet, there is no difference in the magnitude or duration of clinical anesthesia in normal subjects .In this latter study, the clinical anesthesia for 2% lidocaine was the same, regardless of whether the epinephrine was present at 1:50000, 1:80000 or 1:100000 . Knoll- Kohler and Fortsch, however, showed a dose- dependent relationship between the onset and duration of anesthesia and the concentration of epinephrine(1:200000, 1:100000, 1:50000) when used with 2% lidocaine for infiltration

anesthesia [21].It should be noted that these studies were conducted in normal subjects and, to date, no clinical trial has tested whether these higher concentrations of epinephrine alter anesthesia in endodontic pain patients in whom tissue vasodilation may be increased.

Conclusion

Analgesia is essential for successful completion of modern dental procedures. Difficulty experienced in obtaining satisfactory analgesia after proper administration especially of an acutely inflamed mandibular molar, remains a common clinical problem. This article reviews about the various reason for failure of local anaesthesia during endodontic procedure.

Corresponding Author

Balakrishna
Senior Lecturer,Department of Oral and Maxillofacial Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162 Poonamallee High Road,Chennai 600077,Tamil Nadu, India
Phone: +91 8939896729
Email id:balakrishdr8394@gmail.com

Reference

1. B.Vijayalakshmi, Dr.M.P.Santhosh kumar, Knowledge of students about Local anaesthetics
   used during oral surgical procedures. J.Pharm. Sci. & Res. 2015; 7(11):1011-1014
2. Yen Lai Kee,Dr.Prasanna Neelakantan, Local Anaesthetics in Dentistry – Newer Methods of Delivery, International Journal of Pharmaceutical and Clinical Research 2014; 6(1): 4-6
3. Dr.M.P.Santhosh kumar, Newer Delivery Systems for Local Anesthesia in Dentistry, J. Pharm. Sci. & Res. 2015; 7(5):252-255
4. Harrison JW, Baumgartner JC, Zielke DR. Analysis of interappointment pain associated with the combined use of endodontic irrigants and medicaments. J Endodont 1981; 7:272–276.

5. Hannan L, Reader A, Nist R, Beck M, Meyers WJ. The use of ultrasound for guiding needle placement for inferior alveolar nerve blocks. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1999: 87:658–659
6. Berns JM, Sadove MS. Mandibular block injection: a method of study using an injected radiopaque material. J Am Dent Assoc 1962; 65:735–745.
7. Childers M, Reader A, Nist R, Beck M, Meyers WJ. Anesthetic efficacy of the periodontal ligament injection after an inferior alveolar nerve block. J Endod 1996: 22: 317–319
8. Punnia- Moorthy A. Buffering capacity of normal and inflamed tissues following the injection of local anaesthetic solutions. Br J Anaesth 1988: 6:154–159.
9. DiFazio C, Carron H, Grosslight K. Comparison of pH- adjusted lidocaine solutions for epidural anesthesia. Anesth Analg 1986: 64:760–764.
10. Buckley J, Ciancio S, McMullen J. Efficacy of epinephrine concentration in local anesthesia during periodontal surgery. J Periodontol 1984: 55:653–657.
11. Knoll- Kohler E, Fortsch G. Pulpal anesthesia dependent on epinephrine dose in 2% lidocaine. Oral Surg Oral Med Oral Pathol 1992: 73: 537–547
12. Lander J, Hodgins M, Nazarali S, McTavish J, Ouellette J, Friesen E. Determinants of success and failure of EMLA. Pain 1996: 64:89–97.DOI: 10.1016/0304-3959(95)00100-x
13. Gallatin E, Reader A, Nist R, Beck M. Pain reduction in untreated irreversible pulpitis using an intraosseous injection of Depo- Medrol. J Endod 2000: 26:633–638.
14. Fiset L, Getz T, Milgrom P, Weinstein P. Local anesthetic failure: diagnosis and management strategies. General Dent 1989: 37:414–417.
15. Wong M, Jacobsen P. Reasons for local anesthesia failures. J Am Dent Assoc 1992: 12: 69–73.
16. Gale EN. Fears of the dental situation. J Dent Res 1972: 51: 964–966.
17. Thom A, Sartory G, Johren P. Comparison between one- session psychological treatment and benzodiazepine in dental phobia. J Consult Clin Psychol 2000: 68:378–387.
18. Dionne R. Oral sedation. Compend Contin Educ Dent. 1998: 868– 870, 872, 874 passim.
19. Haas DA. Oral and inhalation conscious sedation. Dent Clin North Am 1999: 43:341–359.

20. Kaufman E, Hargreaves KM, Dionne RA. Comparison of oral triazolam and nitrous oxide with lacebo and intravenous diazepam for outpatient sedation. Oral Surg Oral Med Oral Oathol 1993: 433-435
21. Smith GN, Walton RE. Periodontal ligament injection: Distribution of injected solutions. Oral Surg Oral Med Oral Pathol 1983: 55:232–238.

Macrophages – A Review

Maria Kurian[1] , R. Gayatri Devi [2], Sreedevi Dharman[3]

Graduate Student [1],Senior Lecturer[2], Department of Physiology

Senior Lecturer[3], Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

The involvement of macrophages in chronic inflammatory conditions hasbeen the subject of considerable experimental investigation in recentyears. Macrophages are able to secrete a wide range of biologically activecompounds. They are important in the control of infection and, throughtheir immunological and non-immunological receptors; macrophagesinteract with other cell types and a wide variety of proteins.Monocyte proliferation is apparent in a number of disorders, includingcollagen diseases, many infectious diseases, neo-plastic conditions andchronic inflammatory gastrointestinal diseases. Macrophage dysfunction may also result from adrenocorticosteroidadministration or exposure to ionising radiation.Macrophage cell function has been extensively investigated in disorderssuch as lepromatous leprosy, miliary tuberculosis, disseminated fungalinfections and in Hodgkin's disease. Intrinsic or acquired functionaldefects, however, have not been convincingly proved in these conditions.Recent information on the tissue damaging potential of compoundsreleased from activated macrophages has focused interest on a wide varietyof chronic inflammatory conditions where macrophages may potentiatetissue damage in a non-specific manner.The purpose of this review is to highlight recent advances concerningmacrophage origin, function and toconsider how far macrophages may be involved in the promotion ofinflammation in a variety of diseases.

Key Words: *Macrophage, Monocyte, Immunity, Blood, Defense.*

Introduction

The blood consists of monocytes from which the tissue macrophages develop. The capacity of both monocytes and tissuemacrophages to proliferate is limited, and therefore the provision ofadequate numbers of macrophages, especially at an inflammatory focus isdependent upon delivery of blood monocytes. Circulating bloodmonocytes are derived from a rapidly proliferating precursor pool of cellsin the bone marrow, termed promonocytes. The same basic stem cell maygive rise to either monocytes or granulocytes and differentiation iscontrolled by specific colony stimulating factors derived from monocytes,lymphocytes or endothelial cells.

Macrophages are myeloid immune cells. They are present throughout thebody tissues and have the function of ingesting and degrading dead cells, debris and foreign material. They also facilitate the inflammatory processes. Most of the tissue-resident macrophages are formed prenatally and reach until adulthood by their longevity and self renewal properties.The development and maintenance of macrophages maybe independent from the process of haematopoiesis, but the cells may develop alongside the adult monocyte-derived macrophages. Aside from being immune sentinels,tissue macrophages form integral components of their host tissue. Thus these cells are able to respond to the local environmental factors. By this method, it helps in the development and specialized functioning of the host tissue. Factors that govern tissue macrophagespecialization are emerging. Tissue specialization is due to gene expression of the macrophages.

Macrophages are myeloid immune cells that are characterized by avid phagocytosis.Very befittingly, Ilya (Eli) Metchnikoff, the father of cellular immunology, hence referredto these cells as ‘the big eaters’ in Greek [1]. With extraordinary intuition Metchnikoff, atrained comparative zoologist, foresaw the fascinating biology of macrophages and theircentral role in health and disease states and was rightfully awarded a Nobel Prize.Using intra-vital microscopy to visualize macrophage swarms in pricked starfish larvaeand infected water fleas, Metchnikoff noted the striking similarities of invertebratephagocytosis and vertebrate blood cells. He proposed that macrophages had originallyevolved to regulate development through phagocytosis of unwanted or obsolete cells,such as in the pruning of digits and frog morphogenesis. Activities of these professionalphagocytes might then have been recruited for innate immune effector

functions,including pathogen clearance. Importantly, beyond regulating development andcontrolling pathogens, Metchnikoff also recognized that macrophages play a major roleduring injury repair and appreciated their contribution to sterile ‘physiological’inflammation [1]. He thereby anticipated the role of inflammation and macrophages inhomeostatic maintenance processes of the healthy organism, very much in line withcurrently held views [2, 3].

Development of Macrophages

Macrophages have been classified as part of the mononuclear phagocyte systemestablished by Van Furth and Cohn [4, 5], alongside with monocytes and dendriticcells (DC), that were included upon their discovery by Ralph Steinman in the mid 1970s[6-8]. The mononuclear phagocyte concept [5] proposed that tissue-residentmacrophages rely in adulthood on replenishment by bone-marrow (BM)-derived bloodmonocytes. However, the notion of an inherent developmental link between tissuemacrophages and blood monocytes has remained disputed throughout the years [9].Long-term persistence of adult tissue macrophages relying on self-renewal is well established. Moreover, certain embryonic macrophage populations are establishedbefore the emergence of circulating monocytes. Finally, monocytopenic animals displayseemingly normal tissue macrophage compartments and tissue-resident macrophagenumbers are also largely unaffected in human patients suffering from monocytopenia.Collectively, these findings motivated fate mapping experiments to addressmacrophage origins and that revealed the major alternative, non-monocytic origin ofthese cells and thereby corrected the notion of an obligatory steady state monocytemacrophageaxis.The revised concept of the mononuclear phagocyte system, though still mainly basedon data from the mouse, now accommodates the existence of tissue macrophages oftwo distinct origins. (1) The majority of macrophages that reside in healthy tissue areestablished prenatally. These cellular compartments self-maintain locally independentfrom each other within their tissue of residence and are self-sufficient and independentfrom further hematopoietic input. (2) In addition, macrophages can develop in adulthoodfrom tissue-infiltrating monocytes. This pathway is associated with pathological, but alsohomeostatic inflammatory reactions. Monocyte-derived macrophages generally displaylimited life spans, although exceptions have been reported. Embryonic- and adultderivedmacrophages can co-exist

in given tissues and their respective abundancelikely reflects the nature and history of their tissue of residence. Whether embryonicandadult-derived macrophages differ with respect to functional contributions inhomeostasis or following challenge, remains however to be shown and a key questionwith major clinical relevance.

The different types of Macrophages are,

- **Prenatally established tissue macrophages**

Embryonic macrophages

Adult tissue macrophages of embryonic origin

- **Postnatally-generated tissue macrophages**

Monocytes - the precursors

Monocyte-derived macrophages in pathology-associated Inflammation

Monocyte-derived macrophages in homeostatic inflammation

Functions of Macrophages

The mononuclear phagocyte system is involved in five major areas: (a)defense against microorganisms, (b) removal of dead or damaged cells, celldebris and inorganic material, (c) regulation of haematopoiesis, (d)cooperative and effector functions in the immune response, (e) synthesis ofbiologically active compounds such as complement components, prostaglandins,interferon and neutral proteases.

These other functionsof the macrophage may, however, be highly relevant to infections, allergicand autoimmune mechanisms in inflammatory bowel and liver disease, andto the development of immunological abnormalities such as the hyperglobulinaemiaof liver disease.

Defense against Microorganisms

Macrophages are the principal cells involved in killing intracellular parasites such as mycobacteria, toxoplasma and cryptococci [10-12].Macrophage activation was a term used to describe the enhancedbactericidal properties of macrophages previously exposed to intracellularbacteria [13]. This term has now adopted a wider meaning.Macrophages must be able to show effective ability of chemotaxis, phagocytosis and also intracellular killing so that they can be efficient tools in killing the microorganisms. Monocyte chemotaxis is slow in

comparison withneutrophil movement. Chemotaxis is mediated by various enzymes such as the complementcomponents (C5b), the lymphokines and the various derivatives of phospholipids [14].Inhibitors of chemotaxis and inactivators are present in serum in differentdisease states [15, 16]. Once macrophages arrive at an inflammatory site, theyremain there under the influence of a lymphokine, migration inhibitionfactor which activates the cells and results in increased adherence. Thismolecule is probably identical to macrophage activating factor.Cells that have been exposed to a chemoattractant show enhancedexpression of surface complement receptors and are thus better able toadhere to opsonised particles [17]. Subsequent particle ingestion is an energydependent process involving the activation of actin binding protein, whichleads to actin polymerisation to microfilaments. These microfilamentsprovide the forces for pseudopod formation and particle engulfment [18].Particle ingestion depends on the surface receptors involved and the stateof activation of the cell.Macrophages possess multiple mechanisms for killing or degradingingested organisms. The various mechanisms by which macrophages kill the microorganisms includeproduction of hydrogen peroxide [19] and by the production of various oxygen derived products [20]. Myeloperoxidase and otherperoxidase activity [21] have been shown in these cells. Lysosomal fusion withthe phagaocytic vacuole suggests that cationic proteins, hydrolases andcatalases are also important in bacterial degradation [22, 23].

Removal of Damaged Cells, Debris and Inorganic Material

The mononuclear phagocyte system kills the erythrocytes which have been damaged by physical or chemical injury, surface carbohydrate alterations oraging [24]. Alveolar macrophages clear particulate matter from inspiredair [25]. Various inorganic materials such as beryllium, barium salts, zirconium and silica are localised within tissues, often within macrophages.

Regulation of Haematopoiesis

The substances with specific colony stimulating activity determine the formation of granulocyte and monocyte colonies in culture.Circulating monocytes and macrophages are the main source of colonystimulating activity in man [26]. Granulocytes are able to produce compoundssuch as lactoferrin, with colony-inhibiting activity. These compounds blockthe resting but not the activated production of colony stimulating activityby monocytes [27].Macrophages therefore control theproliferation of their own and other progenitor cells through both positiveand negative

feedback systems. Bone marrow macrophages have beenshown recently to influence the growth of early and late committederythoid precursors through the synthesis of soluble factors [28].

Macrophage Activation

The functional efficiency of the macrophage depends on its state of activation. The term 'activated macrophage' was introduced byMackaness [13] to describe the enhanced bactericidal properties ofmacrophages previously exposed to intracellular bacteria. In recent yearsthis term has been applied to describe a wide range of functional changes inthe macrophage, not necessarily resulting in increased bactericidal activity.Plasminogen activator which is a neutralproteinase, when it is secreted, it is seen to go hand in hand with the macrophage activation [29].

The induction of the synthesis and secretion ofplasminorn activator may depend on a number of sequential metabolicchanges [30]. The latter may occur earlier than those changes required for fullmicrobicidal activity. The macrophage activation can be best described by its function under study such as bactericidal, phagocytic or its metabolic activity [31]. Manyagents have been used to increase the yield of macrophages from theperitoneal cavity. Cells thus elicited show some of the features of activatedmacrophages, but not all the biochemical changes associated with fullyactivated cells are found [32].

Functional Specialization of Tissue Macrophages

Generic Macrophage Functions

Macrophages are as immune cells specialized in the phagocytosis and neutralization ofcellular debris and potentially hazardous agents, including pathogens. They can assuch be considered an adaptation of the multicellular organisms to cope with thechallenge arising from progressive cell specialization and complexity by outsourcingimmuno-surveillance to specialized phagocytes. Tissue macrophages are non-migratorycells that monitor their immediate, local environment, as strikingly visualized for microglia [33, 34]. Moreover, also many macrophage effector functions might beexpected to be concentrated on their closer surrounding. In this respect, macrophagescritically differ from highly mobile DC, which are also immune sentinels, but specializedin the triggering of remote T cell responses upon their trans-location to tissue-

drainginglymph nodes [8].To fulfill their role as guardians, tissue macrophages are equipped with a vastarray of sensing molecules, including scavenger receptors, pattern recognitionreceptors (TLRs, CLRs, RLRs, NLRs), nuclear hormone and cytokine receptors, as wellas adhesion molecules. The repertoire of these receptors though varies among tissuemacrophages and likely reflects local adaptation.Generic effector functions of macrophages include activities associated with their highlydeveloped lysosomal compartment that bears critical protease and bactericidal activity[35]. Moreover, macrophages play a critical role in the orchestration of theinflammatory reaction by provision of chemokines and cytokines that recruit and activate neutrophils, monocytes and lymphocytes. Macrophage activation andpolarization has been under intense study, mainly using in vitro system. Inresponse to challenge, macrophages also produce a plethora of effector molecules,including a battery of growth factors, such as PDGF (platelet-derived growth factors),IGFs (insulin-like growth factors), HGFs (hepatocyte growth factors), and FGFs (fibroblastgrowth factors).Expression of sensors renders macrophages responsive to changes in theirenvironment, but bears the inherent risk of macrophage hyper-activation and resultingcollateral damage. Counterbalancing their stimulation, macrophages are hence subjectto silencing programs that most likely set tissue-specific thresholds for their activationand allow the cells to gradually respond and gauge the quality and intensity of thestimulus. The exact nature of these silencing circuits differs between tissues andincludes innate inherent suppression, as well as acquired de-activation triggered inresponse to activating stimuli [36, 37].Of note, the robust silencing programs oftissue resident macrophages might necessitate the need of more plastic cells to copewith acute challenges. The latter seems to be accomplished through on demandrecruitment of monocytes, as flexible emergency squad [38].The immune sentinel task of tissue macrophages could arguably be performedby a stereotypic macrophage prototype, with minor adjustments to the particular tissuecontext. Surprisingly though, depending on their tissue of residence macrophagesdisplay highly distinct and characteristic gene expression signatures [39]. Collectively, these findings suggest that tissue macrophages exertbeyond their unifying features, functions associated with homeostasis of their particulartissue of residence. Of note, this is well compatible with the newly discovered fact thatthese cells develop alongside with their tissue of residence, rather than being eventuallyseeded.

Heterogeneity of tissue macrophages likely results from their adaptation tospecific host tissue environment and reflects a functional polarization governed by localtissue-derived signals; with

few exceptions, however, the identity of these signalsremains unknown. It is also unclear, whether functional heterogeneity is a resultof irreversible lineage-specific differentiation or a consequence of continuous, butreversible induction of diverse functional programs.

Tissue and Organ-Specific Contributions of Macrophages

Lung

The lung is the essential respiration organ and constantly exposed to microbes,pollutants and dusts. A prime task of the lung is to maintain a balance betweentolerances to inhaled innocuous stimuli, as well as the recently appreciated lung microbiome [40], while preserving the capability to mount an immune responseagainst opportunistic pathogens. Dysregulation of the response can lead to infectionsand sustained susceptibility to allergic airway inflammation. This challenge resemblesthat of the intestinal tract and indeed, both organs host equivalent innate immune cellsin the lamina propria, such as monocyte-derived macrophages[41]. In the lung, these cells are directly involved in acute immuneresponses, as well as tolerance induction [42].

Importantly, these macrophages are not located in the interstitial space,but in the alveoli of the healthy airways. Whether the reliance on embryonic originapplies to all lung macrophages remains to be determined.AM also excel in bacterial phagocytosis and produce significant amounts of typeI interferon upon pulmonary virus infection [43]. They thus contribute to thedevelopment of inflammation, but may also be involved in its resolution, since AMdepletion before bacterial or viral infections results in decreased pathogen clearance,but also in exacerbated inflammation [44, 45]. This restorative activity of lung AM canpotentially be explained by their robust anti-inflammatory phenotype, which needs to beovercome by the pathological stimuli. The unique microenvironment of the lung, as a classical environmentalbarrier tissue, significantly contributes to the phenotype of AM by providing factors thatcontrol hyper-activation and thereby govern the characteristic dampened tolerogenicimmunological response in this tissue.

Liver – Kupffer cells (KC)

The liver is a vital organ in charge of efficient uptake of nutrients, amino acids,carbohydrates, lipids and vitamins and their subsequent storage. It further promotesmetabolic conversion and

release of metabolites into the blood and bile, as well as thedetoxification of ingested hazardous chemicals. Liver lobules receive continuous bloodsupply from the arterial circulation and the portal vein, which drains the gastro-intestinaltract. The latter results in prominent liver exposure to food antigens, microbial productsand xenobiotics. The liver hence stars prominently in host defense and theestablishment of a local and systemic tolerance. Blood entering the hepatic parenchymapasses through a network of thin-walled ‘sinusoids’, which contain specialized nonparenchymalcells, also including a fenestrated layer of liver sinusoidal endothelial cells.These features confer effective antigen capture and presentation in thismicroenvironment [46]. KC, the resident macrophage population of the liver, is most abundant in theupstream periportal regions of the liver sinusoids. KC display high phagocytic andlysosomal activity, which highlights their specialization in the surveillance and filtering ofblood entering the sinusoids. They thereby form a protective barrier to prevent systemicpathogen circulation. In addition, KC remove potentially harmful endogenouscompounds, such as complement and fibronectin-coated particles, material releasedfrom dying cells and extracellular matrix components, as well as immune complexes toavoid intravascular coagulation. Despite their continuous exposure to gut-derivedantigens and bacterial endotoxins, and maybe because of the latter, KC exhibit in thehealthy liver a tolerogenic phenotype. However, under disease conditions, KC can shiftto a pathologically activated state and then cause hepatocellular injury and damage[47, 48].KC also performs liver-specific metabolic functions. KC furthermoreregulates plasma cholesterol levels through the removal of native and modifiedlipoproteins from the circulation. Specifically, they express receptors for both high-densitylipoproteins (HDLs) and low-density lipoproteins (LDLs), but are primarilyinvolved with the catabolism of LDLs and modified LDLs [48]. Kupffer cells areembryo-derived macrophages and the compartment seems to resists replacement byadult monocyte-derived cells during inflammation [49].

Bone – Osteoclasts

Bone is a unique, highly vascularized, rigid, yet dynamic organ designed to providemaximal strength with minimal mass. Bone shape is dictated by constant remodelinginvolving bone synthesis by mesenchymal osteoblasts and bone resorption byspecialized bone resident macrophages, the osteoclasts. Abnormalities in the balanceof osteoclast development or function,

relative to osteoblast activity, result in skeletalpathologies, such as osteopetrosis or osteoporosis [50]. Osteoclasts are moreoveressential for normal long bone formation during embryogenesis [51]. They aremultinucleated cells, or 'polykaryons', that form by cell fusion near the bone surface.RANKL is expressed by osteoblasts and induces polarization of mature osteoclasts, which is manifested in structural changes essentialfor their bone-resorption. This includes cytoskeletal rearrangements that enable theformation of a ruffled membrane structure and a “matrix attachment” sealing actin ringtowards the bone surface [52]. Pre- and post-natal osteoclast ontogeny remains poorly defined. Specifically, it isunclear whether osteoclasts originate from circulating blood monocytes or from bonetissue-resident precursors. The stimulus that recruits the monocytes tothe bones remains however to be defined.

Brain – Microglia

Microglia represents the tissue resident macrophages of the CNS and the sole residenthematopoietic, mesoderm-derived cell type in the CNS parenchyma. Microglia fulfillstwo main functions in the adult CNS: homeostatic maintenance and immune defense.Beside the neuronal circuitry, the CNS comprise of additional microglia populations,such as the myelin sheath-forming oligodendrocytes and astrocytes, which are involvedin physiological and biochemical tasks to functionally support the BBB. The lattercritically contributes to the immune-privileged state of the CNS and secures therestricted passage of substances from the bloodstream into the CNS.Microglia was, aside from epidermal LC [53], the first cells of the innateimmune system that were experimentally proven to be long-lived and independent ofBM-derived myeloid cell replenishment during adulthood [54, 55]. However,compared to LC and other peripheral tissue resident macrophages, murine microgliaexclusively derives from the embryonic yolk sac, without a contribution from fetal liver[56, 57]. This unique feature might have its cause in the BBB that could limit CNS entryof myeloid cells to embryonic yolk sac macrophages. Alternatively, embryonic yolk sacmacrophages might be uniquely able to infiltrate the neuro-ectoderm, due to theirexpression repertoire of matrix metalloproteinases or chemokine receptors [58].In the developing embryonic

CNS, microglia undergoes rapid proliferation and increase incell numbers, but steady state proliferation largely ceases in adulthood [57].Existence of these mechanisms suggests that amicroglia-free brain parenchyma is incompatible with brain function and viability, andthus support the emerging concept that microglia are critical for CNS homeostasis.Microglia is indeed tightly imbedded into the cellular communication with neuroglialcells. Early infiltrating embryonic microglia is highly phagocytic and probably involvedin clearance of cellular debris [58]. Microglia actively engulfs synaptic material andperform synaptic pruning during postnatal development [59-61]. In summary, recent advance in our understanding of microglial biology indicates thatthese cells represent an important component of the CNS, which is imprinted by itsneuroglial surrounding and linked to its unique secluded location behind the BBB.

Splenic Macrophage Populations

The spleen is the largest lymphoid organ in the body and the main site where T and Bcell responses to blood-borne antigens are initiated. Since this organ lacks afferentlymphatics, functions of the spleen are centered on the systemic circulation. The spleenserves as filter of the blood for senescent cells, including erythrocytes, and potentialnoxious materials. Many of the above functions are essentially attributed to specificmacrophage subpopulations that are compartmentalized into different splenic domains,and well adapted to perform site-specific homeostatic roles [62].Red Pulp Macrophages (RPM)are specialized in iron recovery and prominentlyexpress proteins involved in all recycling phases starting from the scavenging ofsenescent or damaged erythrocytes, the uptake of hemoglobin and finally hemebreakdown and iron export [63]. Similarly to KC, RPM also use CD163 for endocytosisof hemoglobin-haptoglobin complexes released from dying erythrocytes [64].

RPM are generated prenatally and maintained throughsteady state adulthood without substantial monocytic input [65, 66]. Of note, the splenic red-pulp is also a site for storageand rapid deployment of monocytes, a potential resource that the body can exploit toregulate inflammation [67].The interface of the splenic white- and red-pulp, called 'marginal zone', is a reticularnetwork and hosts two distinct resident macrophage populations that are strategicallypositioned to screen

entering arterial blood: the marginal zone macrophages (MZM)and the marginal metallophilic macrophages (MMM). As a result of the splenicanatomy, blood flow slows down in the marginal zone and pathogens present in thesystemic circulation are efficiently removed by the macrophage subsets [68, 69].Elimination of MZM and MMM leads toaccumulation of apoptotic cells in the white pulp and their uptake by RPM, resulting inincreased inflammatory cytokine production, enhanced adaptive immunity andaccelerated autoimmunity in disease prone mice [70]. This suggests that MMM andMZM are, as opposed to RPM, specialized in the quiescent removal of their cargo.As for distinct roles of the two cell populations, Marginal Zone Macrophages(MZM) are positioned within the outer layer of the marginal zone and express a unique set of patter recognition receptors (PRR). MZM intimatelyinteract with a specialized non-circulating type of mature B cells that segregateanatomically into the marginal zone.

Marginal Metallophilic Macrophages (MMM) are located adjacent to the white pulpand marginal sinus and form an inner ring of macrophages.Macrophages are also located in the white pulp, which is the site where immuneresponses to blood-borne antigens are initiated and which structurally resembles lymphnodes [68]. **White pulp macrophages** can be identified by their common expressionof the pan macrophage marker CD68. They include 'tingible body macrophages', whichare located in the germinal center (GC) of the B cell follicles. Collectively and in line with the complex architecture and multiple tasks of their organ ofresidence, splenic macrophage subsets display considerable phenotypic and functionalheterogeneity. It remains though unresolved whether these macrophage subsets aredifferent cellular entities with independent ontogeny or alternatively, share precursorsthat are differentially educated in the distinct splenic micro-anatomical niches. Thus, thespleen might represent an excellent model to study site-specific regulatory networks ofdevelopment and / or function.

Conclusion

There are clear examples in which tissue-specific factors drivehighly specialized macrophage functions irrespective of theirontological origin, suggesting tremendous plasticity and redundancyin the mononuclear phagocyte system. Whether embryonicand adult macrophages possess specialized roles has yetto be formally tested. However, our conceptual understandingand genetic tools are now sufficiently developed to preciselyfollow both embryonic and adult macrophage subsets in healthand disease, which should allow important and

unansweredquestions in the field to be addressed. In order todevelop novel therapies, a critical future goal is to harness thisnewfound understanding that different macrophage lineagesexist within tissues and clarify whether these distinct lineagesdifferentially contribute to tissue damage and repair.

Corresponding Author

Sreedevi Dharman

Senior Lecturer, Department of Oral Medicine and Radiology

Saveetha Dental College,

Saveetha University, Saveetha Institute of Medical and Technical Sciences

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

E-mail: sanjamrut@gmail.com

Telephone number: +919841009003

References

1. Cavaillon JM. The historical milestones in the understanding of leukocyte biology initiated by Elie Metchnikoff. Journal of leukocyte biology. 2011 Sep 1; 90(3):413-24.
2. Medzhitov R. Inflammation 2010: new adventures of an old flame. Cell. 2010 Mar 19; 140(6):771-6.
3. Medzhitov R. Origin and physiological roles of inflammation. Nature. 2008 Jul 23; 454(7203):428.
4. van Furth R, Cohn ZA. The origin and kinetics of mononuclear phagocytes. Journal of Experimental Medicine. 1968 Sep 1; 128(3):415-35.
5. Van Furth R, Cohn ZA, Hirsch JG, Humphrey JH, Spector WG, Langevoort HL. The mononuclear phagocyte system: a new classification of macrophages, monocytes, and their precursor cells. Bulletin of the World Health Organization. 1972; 46(6):845.

6. Steinman RM, Kaplan G, Witmer MD, Cohn ZA. Identification of a novel cell type in peripheral lymphoid organs of mice. V. Purification of spleen dendritic cells, new surface markers, and maintenance in vitro. Journal of Experimental Medicine. 1979 Jan 1; 149(1):1-6.
7. Merad M, Sathe P, Helft J, Miller J, Mortha A. The dendritic cell lineage: ontogeny and function of dendritic cells and their subsets in the steady state and the inflamed setting. Annual review of immunology. 2013 Mar 21; 31:563-604.
8. Guilliams M, Ginhoux F, Jakubzick C, Naik SH, Onai N, Schraml BU, Segura E, Tussiwand R, Yona S. Dendritic cells, monocytes and macrophages: a unified nomenclature based on ontogeny. Nature Reviews Immunology. 2014 Aug; 14(8):571.
9. Ginhoux F, Jung S. Monocytes and macrophages: developmental pathways and tissue homeostasis. Nature Reviews Immunology. 2014 Jun 1; 14(6):392-404.
10. Ando M, Dannenberg Jr AM. Macrophage accumulation, division, maturation, and digestive and microbicidal capacities in tuberculous lesions. IV. Macrophage turnover, lysosomal enzymes, and division in healing lesions. Laboratory investigation; a journal of technical methods and pathology. 1972 Nov; 27(5):466.
11. Anderson SE, Remington JS. Effect of normal and activated human macrophages on Toxoplasma gondii. Journal of Experimental Medicine. 1974 May 1; 139(5):1154-74.
12. Shahar A, Kletter Y, Aronson M. Granuloma formation in cryptococcosis. Israel J Med Sci 1969; 5: 1164-72.
13. Mackaness GB. Cellular resistance to infection. Journal of Experimental Medicine. 1962 Sep 1; 116(3):381-406.
14. Cline MJ, Lehrer RI, Territo MC, Golde DW. Monocytes and macrophages: functions and diseases. Annals of internal medicine. 1978 Jan 1;88(1):78-88.
15. Snyderman R, Pike MC. An inhibitor of macrophage chemotaxis produced by neoplasms. Science. 1976 Apr 23; 192(4237):370-2.
16. Maderazo EG, Ward PA, Quintiliani R. Defective regulation of chemotaxis in cirrhosis. Translational Research. 1975 Apr 1; 85(4):621-30.
17. Glass EJ, Kay AB. Enhanced expression of human monocyte complement (C3b) receptors by chemoattractants. Clinical and experimental immunology. 1980 Mar; 39(3):768.

18. Robinson BV. The pharmacology of phagocytosis. Rheumatology. 1978 Jan 1; 17(suppl):37-46.
19. Cline MJ. Bactericidal activity of human macrophages: analysis of factors influencing the killing of Listeria monocytogenes. Infection and immunity. 1970 Aug 1; 2(2):156-61.
20. Johnston JR, Godzik CA, Cohn ZA. Increased superoxide anion production by immunologically activated and chemically elicited macrophages. Journal of Experimental Medicine. 1978 Jul 1; 148(1):115-29.
21. Davis GS, Mortara M, Pfeiffer LM, Green GM. Bactericidal and biochemical peroxidase activity in human alveolar macrophages. Am Rev Respir Dis. 1977; 115:210A.
22. Hunninghake GW, Gadek JE, Young Jr RC, Kawanami O, Ferrans VJ, Crystal RG. Maintenance of granuloma formation in pulmonary sarcoidosis by T lymphocytes within the lung. New England Journal of Medicine. 1980 Mar 13; 302(11):594-8.
23. Springmeyer SC, Altman LC, Kopecky KJ, Deeg HJ, Storb R. Alveolar macrophage kinetics and function after interruption of canine marrow function. American Review of Respiratory Disease. 1982 Mar; 125(3):347-51.
24. Maruta H, Mizuno D. Selective recognition of various erythrocytes in endocytosis by mouse peritoneal macrophages. Nature New Biology. 1971 Dec 22; 234(51):246.
25. Sherman MP, Ganz T. Host defense in pulmonary alveoli. Annual review of physiology. 1992 Mar; 54(1):331-50.
26. Golde DW, Cline MJ. Identification of the colony-stimulating cell in human peripheral blood. The Journal of clinical investigation. 1972 Nov 1; 51(11):2981-3.
27. Broxmeyer HE, Smithyman AN, Eger RR, Meyers PA, De Sousa M. Identification of lactoferrin as the granulocyte-derived inhibitor of colony-stimulating activity production. Journal of Experimental Medicine. 1978 Oct 1; 148(4):1052-67.
28. Gordon LI, Miller WJ, Branda RF, Zanjani ED, Jacob HS. Regulation of erythroid colony formation by bone marrow macrophages. Blood. 1980 Jun 1; 55(6):1047-50.
29. Nogueira NA, Gordon SI, Cohn ZA. Trypanosoma cruzi: the immunological induction of macrophage plasminogen activator requires thymus-derived lymphocytes. Journal of Experimental Medicine. 1977 Jul 1; 146(1):172-83.

30. Gordon S, Unkeless AC, Cohn ZA. Induction of macrophage plasminogen activator by endotoxin stimulation and phagocytosis: evidence for a two-stage process. Journal of Experimental Medicine. 1974 Oct 1; 140(4):995-1010.
31. North RJ. Opinions: the concept of the activated macrophage. The Journal of Immunology. 1978 Sep 1; 121(3):806-9.
32. Karnovsky ML, Lazdins JK. Biochemical criteria for activated macrophages. The Journal of Immunology. 1978 Sep 1; 121(3):809-13.
33. Davalos D, Grutzendler J, Yang G, Kim JV, Zuo Y, Jung S, Littman DR, Dustin ML, Gan WB. ATP mediates rapid microglial response to local brain injury in vivo. Nature neuroscience. 2005 Jun; 8(6):752.
34. Nimmerjahn A, Kirchhoff F, Helmchen F. Resting microglial cells are highly dynamic surveillants of brain parenchyma in vivo. Science. 2005 May 27; 308(5726):1314-8.
35. Stuart LM, Ezekowitz RA. Phagocytosis: elegant complexity. Immunity. 2005 May 1; 22(5):539-50.
36. Bogdan C, Vodovotz Y, Nathan C. Macrophage deactivation by interleukin 10. Journal of Experimental Medicine. 1991 Dec 1; 174(6):1549-55.
37. Gordon S. Alternative activation of macrophages. Nature reviews immunology. 2003 Jan; 3(1):23.
38. Mildner A, Yona S, Jung S. A close encounter of the third kind: monocyte-derived cells. In Advances in immunology 2013 Jan 1 (Vol. 120, pp. 69-103). Academic Press.
39. Gautier EL, Shay T, Miller J, Greter M, Jakubzick C, Ivanov S, Helft J, Chow A, Elpek KG, Gordonov S, Mazloom AR. Gene-expression profiles and transcriptional regulatory pathways that underlie the identity and diversity of mouse tissue macrophages. Nature immunology. 2012 Nov; 13(11):1118.
40. Beck JM, Young VB, Huffnagle GB. The microbiome of the lung. Translational Research. 2012 Oct 1; 160(4):258-66.
41. Guilliams M, Lambrecht BN, Hammad H. Division of labor between lung dendritic cells and macrophages in the defense against pulmonary infections. Mucosal immunology. 2013 May; 6(3):464.
42. Nishanthi R, Arjunkumar R. Interleukin Level 1 in Smokers GCF. Journal of Clinical Periodontology.; 22:885-90.

43. Kumagai Y, Takeuchi O, Kato H, Kumar H, Matsui K, Morii E, Aozasa K, Kawai T, Akira S. Alveolar macrophages are the primary interferon-α producer in pulmonary infection with RNA viruses. Immunity. 2007 Aug 24; 27(2):240-52.
44. Archambaud C, Salcedo SP, Lelouard H, Devilard E, De Bovis B, Van Rooijen N, Gorvel JP, Malissen B. Contrasting roles of macrophages and dendritic cells in controlling initial pulmonary Brucella infection. European journal of immunology. 2010 Dec 1; 40(12):3458-71.
45. Tate MD, Pickett DL, van Rooijen N, Brooks AG, Reading PC. Critical role of airway macrophages in modulating disease severity during influenza virus infection of mice. Journal of virology. 2010 Aug 1; 84(15):7569-80.
46. Ishibashi H, Nakamura M, Komori A, Migita K, Shimoda S. Liver architecture, cell function, and disease. InSeminars in immunopathology 2009 Sep 1 (Vol. 31, No. 3, p. 399). Springer-Verlag.
47. Dixon LJ, Barnes M, Tang H, Pritchard MT, Nagy LE. Kupffer cells in the liver. Comprehensive Physiology. 2013 Apr 1.
48. Toth CA, Thomas D. Liver endocytosis and Kupffer cells. Hepatology. 1992 Jul 1; 16(1):255-66.
49. Zigmond E, Samia-Grinberg S, Pasmanik-Chor M, Brazowski E, Shibolet O, Halpern Z, Varol C. Infiltrating monocyte-derived macrophages and resident kupffer cells display different ontogeny and functions in acute liver injury. The Journal of Immunology. 2014 Jul 1; 193(1):344-53.
50. Teitelbaum SL, Ross FP. Genetic regulation of osteoclast development and function. Nature Reviews Genetics. 2003 Aug; 4(8):638.
51. Ortega N, Wang K, Ferrara N, Werb Z, Vu TH. Complementary interplay between matrix metalloproteinase-9, vascular endothelial growth factor and osteoclast function drives endochondral bone formation. Disease models & mechanisms. 2010 Mar 1; 3(3-4):224-35.
52. Burgess TL, Qian YX, Kaufman S, Ring BD, Van G, Capparelli C, Kelley M, Hsu H, Boyle WJ, Dunstan CR, Hu S. The ligand for osteoprotegerin (OPGL) directly activates mature osteoclasts. The Journal of cell biology. 1999 May 3; 145(3):527-38.

53. Merad M, Manz MG, Karsunky H, Wagers A, Peters W, Charo I, Weissman IL, Cyster JG, Engleman EG. Langerhans cells renew in the skin throughout life under steady-state conditions. Nature immunology. 2002 Dec; 3(12):1135.
54. Mildner A, Schmidt H, Nitsche M, Merkler D, Hanisch UK, Mack M, Heikenwalder M, Brück W, Priller J, Prinz M. Microglia in the adult brain arise from Ly-6C hi CCR2+ monocytes only under defined host conditions. Nature neuroscience. 2007 Dec; 10(12):1544.
55. Ramanathan S, Jagannathan N. Tumor associated macrophage: a review on the phenotypes, traits and functions. Iranian journal of cancer prevention. 2014; 7(1):1.
56. Hoeffel G, Wang Y, Greter M, See P, Teo P, Malleret B, Leboeuf M, Low D, Oller G, Almeida F, Choy SH. Adult Langerhans cells derive predominantly from embryonic fetal liver monocytes with a minor contribution of yolk sac–derived macrophages. Journal of Experimental Medicine. 2012 Jun 4; 209(6):1167-81.
57. Ginhoux F, Greter M, Leboeuf M, Nandi S, See P, Gokhan S, Mehler MF, Conway SJ, Ng LG, Stanley ER, Samokhvalov IM. Fate mapping analysis reveals that adult microglia derive from primitive macrophages. Science. 2010 Nov 5; 330(6005):841-5.
58. Kierdorf K, Erny D, Goldmann T, Sander V, Schulz C, Perdiguero EG, Wieghofer P, Heinrich A, Riemke P, Hölscher C, Müller DN. Microglia emerge from erythromyeloid precursors via Pu. 1-and Irf8-dependent pathways. Nature neuroscience. 2013 Mar; 16(3):273.
59. Stevens B, Allen NJ, Vazquez LE, Howell GR, Christopherson KS, Nouri N, Micheva KD, Mehalow AK, Huberman AD, Stafford B, Sher A. The classical complement cascade mediates CNS synapse elimination. Cell. 2007 Dec 14; 131(6):1164-78.
60. Tremblay MÈ, Stevens B, Sierra A, Wake H, Bessis A, Nimmerjahn A. The role of microglia in the healthy brain. Journal of Neuroscience. 2011 Nov 9; 31(45):16064-9.
61. Schafer DP, Lehrman EK, Kautzman AG, Koyama R, Mardinly AR, Yamasaki R, Ransohoff RM, Greenberg ME, Barres BA, Stevens B. Microglia sculpt postnatal neural circuits in an activity and complement-dependent manner. Neuron. 2012 May 24; 74(4):691-705.
62. Den Haan JM, Kraal G. Innate immune functions of macrophage subpopulations in the spleen. Journal of innate immunity. 2012; 4(5-6):437-45.

63. Ganz T. Macrophages and systemic iron homeostasis. Journal of innate immunity. 2012; 4(5-6):446-53.
64. Kristiansen M, Graversen JH, Jacobsen C, Sonne O, Hoffman HJ, Law SA, Moestrup SK. Identification of the haemoglobin scavenger receptor. Nature. 2001 Jan; 409(6817):198.
65. Yona S, Kim KW, Wolf Y, Mildner A, Varol D, Breker M, Strauss-Ayali D, Viukov S, Guilliams M, Misharin A, Hume DA. Fate mapping reveals origins and dynamics of monocytes and tissue macrophages under homeostasis. Immunity. 2013 Jan 24; 38(1):79-91.
66. Hashimoto D, Chow A, Noizat C, Teo P, Beasley MB, Leboeuf M, Becker CD, See P, Price J, Lucas D, Greter M. Tissue-resident macrophages self-maintain locally throughout adult life with minimal contribution from circulating monocytes. Immunity. 2013 Apr 18; 38(4):792-804.
67. Swirski FK, Nahrendorf M, Etzrodt M, Wildgruber M, Cortez-Retamozo V, Panizzi P, Figueiredo JL, Kohler RH, Chudnovskiy A, Waterman P, Aikawa E. Identification of splenic reservoir monocytes and their deployment to inflammatory sites. Science. 2009 Jul 31; 325(5940):612-6.
68. Mebius RE, Kraal G. Structure and function of the spleen. Nature Reviews Immunology. 2005 Aug; 5(8):606.
69. Kraal G, Mebius R. New insights into the cell biology of the marginal zone of the spleen. International review of cytology. 2006 Jan 1; 250:175-215.
70. McGaha TL, Chen Y, Ravishankar B, Van Rooijen N, Karlsson MC. Marginal zone macrophages suppress innate and adaptive immunity to apoptotic cells in the spleen. Blood. 2011 May 19; 117(20):5403-12.

Management of Anaphylaxis- A Short Review

Sisira Padavala[1], V. Ashok[2],
Graduate Student[1],Reader[2],Department of Prosthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Anaphylaxis is a serious allergic reaction that is rapid in onset and may lead to death.During anesthesia,neuromuscular blocking agents,antibiotics and latex are the most common causes.The rapidness determines its severity.Prompt recognition and management of this condition is imperative.Diagnosis and management are challenging since reactions are often immediate and unexpected.To understand the severity of anaphylaxis and to manage anaphylaxis to determine the risk of death caused by anaphylaxis.

Key Words:*Anaphylaxis,Management,Epinephrine,Allergy, Emergency, Dental office*.

Introduction

Anaphylaxis is an acute, potentially fatal systemic reaction with varied mechanisms and clinical presentations[1,21]. Although prompt recognition and treatment of anaphylaxis are imperative, both patients and healthcare professionals often fail to recognize and diagnose early signs and symptoms of the condition. It is a life-threatening syndrome that requires prompt recognition and treatment.Immediate management includes oxygen administration, epinephrine and i.v fluids[2].Anaphylaxis has a rapid onset with multiple organsystem involvement and is mostly caused by specific antigens in sensitized individuals[3]. It results from the immunologically induced release of mast cell or basophil mediators after exposure to a specific antigen in previously sensitized individuals. Anaphylaxis is a medical emergency that requires immediate attention. Several working definitions of anaphylaxis have been formulated to aid clinical diagnosis and management. Due to differences in recognition, diagnosis and reporting of anaphylactic reactions estimates of lifetime prevalence range between 0.05% and 2%. As with other allergic diseases, several surveys suggest a rising incidence of anaphylaxis[4,5].The most common causes of IgEmediated anaphylaxis are insect, stings,medications,latex,peanuts,tree nuts,fish,milk,eggs,wheat.Idiopathic and exercise induced anaphylaxis also occur[6].In this review, we discuss the current recommendations for its management.All physicians will benefit from knowledge of its recognition and appropriate treatment.

Causes of Anaphylaxis

Most episodes of anaphylaxis are triggered through an immunologic mechanism involving immunoglobulin E (IgE) which leads to mast cell and basophil activation and the subsequent release of inflammatory mediators such as histamine, leukotrienes, tryptase and prostaglandins[7,22].The most common causes of IgE-mediated anaphylaxis are: foods, particularly, peanuts, tree nuts, shellfish and fish, milk, eggs and wheat;medications (most commonly penicillin), and natural rubber latex. Exercise, aspirin, non-steroidal anti-inflammatory drugs (NSAIDs), opiates, and radio contrast agents can also cause anaphylaxis, but anaphylactic reactions to these agents often result from non-IgE-mediated mechanisms.Sometimes it is idiopathic[1,2,27].In children, anaphylaxis is most often caused by foods, while venom-and drug-induced anaphylaxis is more common in adults.

Signs and Symptoms of Anaphylaxis

A wide variety of clinical signs and symptoms involving the skin, gastrointestinal and respiratory tracts and cardiovascular system can be observed.Symptoms have their own onset within minutes but occasionally occur as late as 1 hour after exposure to the offending antigen.The signs and

symptoms may follow a uniphasic course, with resolution of symptoms within hours of treatment, but about 20% of anaphylactic reactions will follow a biphasic course[3].

Table 1: Lists the Symptoms of Anaphylaxis

Table 1	Signs and symptoms
Skin	Urticaria Angioedema Pruritus Erythema Maculopapular rash
Cardiovascular	Tachycardia Hypotension Arrhythmia Syncope Cardiac arrest Dizziness Myocardial infarction
Respiratory ->Upper airway ->Lower airway	Stridor Cough Sneezing Hoarseness Laryngeal edema Nasal congestion Cyanosis Respiratory arrest Tachypnea Accessory muscle use

Table 1	Signs and symptoms
Ocular	Lacrimation Conjunctival injection
Gastrointestinal	Diarrhoea Nausea Vomiting Abdominal pain

Diagnosis

It is not necessary to differentiate between anaphylactic and anaphylactoid reactions at the time of patient presentation since both respond to the same treatment but anaphylactoid shock must be differentiated from other circulatory collapse causes.Anaphylaxis is an unusual, exaggerated allergic reaction. It is mediated by the IgE antibodies and occurs due to re-exposure to an antigen. Anaphylactoid reaction does not involve mediation by the IgE antibodies and occurs without previous exposure to the antigens. The most common conditions that mimic anaphylaxis are vasodepressor reactions characterized by weakness, nausea, bradycardia, pallor, hypotension, vomiting, diaphoresis[1,8,24].Acute respiratory decompensation from severe asthma attacks, pulmonary embolism, foreign body aspiration, myocardial infarction, seizures, acute poisoning can be featured as symptoms suggestive of anaphylaxis but other characteristics like pruritus, urticaria, angioedema are lacking[9].

Management

Anaphylaxis is a medical emergency that may require resuscitation measures such as airway management, supplemental oxygen, large volumes of intravenous fluids, and close monitoring.Administration of epinephrine is the treatment of choice with antihistamines and steroids used as adjuncts[10,25].

Epinephrine

Epinephrine (adrenaline) is the primary treatment for anaphylaxis with no absolute contraindication to its use.The world health organization classifies it as an essential drug.The recommended dose of epinephrine is 0.01mg/kg intramuscular to as much as 0.3mg and it may be repeated within 5 minutes if symptoms worsen or severe symptoms persist[11].The more advanced the anaphylactic reaction, the less likely epinephrine is to reverse the reaction. Epinephrine is life-saving because of its alpha-1 adrenergic vasoconstrictor effects in most body organ systems (skeletal muscle is an important exception) and its ability to prevent and relieve airway obstruction caused by mucosal edema, and to prevent and relieve hypotension and shock[12,13].

Adjuncts

Corticosteroids are unlikely to make a difference in the current episode of anaphylaxis, but may be used in the hope of decreasing the risk of biphasic anaphylaxis[14]. Nebulized salbutamol may be effective for bronchospasm that does not resolve with epinephrine.Methylene blue has been used in those not responsive to other measures due to its presumed effect of relaxing smooth muscle[15].

Treatment

Acute Management

The acute treatment of anaphylaxis begins with rapid assessment of the airway, breathing and circulation. Epinephrine is the drug of choice for anaphylaxis and should be given immediately to any patient with a suspected anaphylactic episode[1].Oxygen therapy should also be considered in any patient with symptoms of anaphylaxis, particularly for those with prolonged reactions. Intravenous fluids should also be provided since massive fluid shifts can occur rapidly in anaphylaxis due to increased vascular permeability[16,17].Vasopressors, such as dopamine, can also be considered if epinephrine injections and volume expansion with intravenous fluids fail to alleviate hypotension[18].If intramuscular epinephrine and intravenous fluids fail to improve anaphylactic symptoms, intravenous infusions of epinephrine may be required.Following acute treatment, patients should be observed for a period of time due to the risk of a biphasicresponseorpossiblerecurrenceofthereactionas epinephrine wears off.

Long Term Management

Assessment

Specialist assessment is a part of longterm management.It helps in assessment of future risk of anaphylaxis for an acute anaphylaxis patient.It helps in analysing causes of anaphylaxis and educating them on proper avoidance strategies, drafting an action plan and advising on immunotherapy[12].

Epinephrine Auto-Injector

Epinephrine auto-injector should be provided to all the patients who have experienced anaphylaxis even those with rapid onset reaction or diffuse hives to insect stings or food.Thereare two kinds of auto-injectors EpiPen and Twinject.They are prescribed according to the weight.0.15 mg for those who weigh between 15-30 kg[1].They should be stored properly and replaced before the expiration date.

Education on Avoidance Measures

Avoidance measures should be considered based on the individual and on age, occupation, activities, personal anxietyand medical care access.

Conclusion

Most of the cases of anaphylaxis are not given timely recognition and remain untreated. This may lead to life-threatening consequences so intramuscular administration of epinephrine is lifesaving.There has to be frequent monitoring of the recovered patients.Recommending management of anaphylaxis in schools and other community settings will help in managing anaphylaxis to a large extent.

Corresponding Author

V. Ashok,

Reader,Department of Prosthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai- 600077, Tamil Nadu, India.

Email Id: ashokprostho@gmail.com

Telephone Number: 9962422299

References

1. Kim H,Fischer D.Anaphylaxis.Allergy,Asthma& Clinical Immunology 2011;7:1-6.

2. Ryder SA,Waldmann C.Anaphylaxis.Continuing Education in Anaesthesia,Critical Care &Pain 2004;4(4):111-113.
3. Ellis.AK,DayJH.Diagnosis and Management of anaphylaxis.CMAJ 2003;169(4):307-312.
4. Dhami S,A sheik, A Muraro The acute and long-term management of anaphylaxis:protocol for a systemic review.Clinicaland Translational Allergy 2013;3(14):1-5.
5. Grabenhenrich L, Hompes S,Implementation of Anaphylaxis Management Guidelines:A Register-Based Study.PLoS ONE 2012;7(5):1-10.
6. Lee J, Kim M, Chung YB,Anaphylaxis to husband's seminal plasma and treatment by local desensitization.Clinical and Molecular Allergy 2008;6(13):1-3.
7. Maus VM, Haas A R,T Cells Expressing Chimeric Antigen Receptors Can Cause Anaphylaxis in Humans.Cancer Immunol Res, 2013;1:26-31.
8. Walker S, Sheikh A. Managing anaphylaxis: effective emergency and longterm care are necessary. Clin Exp Allergy, 2003; 33(8):1015–1018.
9. Waserman S, Watson W. Food allergy. Allergy, Asthma & Clinical Immunology, 2011;7(1):1-7.
10. Simon FE. Anaphylaxis, killer allergy: long-term management in the community. J Allergy Clin Immunol, 2006;117:367-77.
11. LiebermanP, Nickolas RA, Kemp SF, The diagnosis and management of anaphylaxis practice parameter: 2010 update. J Allergy Clin Immunol, 2010;126:477-80.
12. Sampson HA Symposium on the Definition and Management of Anaphylaxis: Summary report.The Journal of Allergy and Clinical Immunology, 2005;115(3):584-91.
13. Simons FER, Ardusso LRF, Bilo MB, EL-Gamal YM, World Allergy Organization Guidelines for the Assessment and Management of Anaphylaxis.WAO Journal, 2011;4:13-37.
14. Muraro A, Roberts G, Clark A,.EAACI Task Force on Anaphylaxis in Children. The management of anaphylaxis in childhood: position paper of the European academy of allergology and clinical immunology. Allergy 2007;62(8):857–871.
15. Smith CE, Eagar TN, Strominger JL, Miller SD. Differential induction of IgE-mediated anaphylaxis after soluble vs. cell-bound tolerogenic peptide therapy of autoimmune encephalomyelitis. PNAS 2005;102(27):9595-9600.
16. Simons FER, Gu X, Simons KJ. Epinephrine absorption in adults: intramuscular versus subcutaneous injection. J Allergy Clin Immunol 2001;108(5):871-3.
17. Saxon A. Immediate hypersensitivity reactions to beta-lactam antibiotics. Ann Intern Med 1987;107(2):204-15.

18. Sussman GL, Beezhold DH. Allergy to latex rubber.Ann Intern Med 1995; 122(1):43-6.
19. Sheffer A, Austen K. Exercise-induced anaphylaxis. J Allergy Clin Immunol 1984; 73:699-703.
20. Patterson R, Harris KE. Idiopathic anaphylaxis. Allergy Asthma Proc 1999; 20 (5):311-50.
21. Kumar MPS. Management of anaphylaxis in dental office – An update.Int J Pharm Bio Sci 2015; 6(3):401-407.
22. Balaji K, Jain AR. Knowledge, attitude and practice regarding anaphylaxis among dental students. IJSDR 2017;2(4):428-432.
23. Sriram K, Abilasha R. Awareness of allergic reactions to dental drugs and materials among patients, dentists and dental personnel - A cross sectional study. J Pharm Sci & Res 2016;8(9):1050-1057.
24. Vaishali S, Roy A. Awareness of anaphylaxis reaction to local anaesthesia drugs among dental students – A cross sectional study. IJPT 2017;9(1):29338-29344.
25. Dave PH, Dhanraj M, Rakshagan. Knowledge, awareness and practice among dental practitioners regarding the management of allergic reactions induced by dental prosthesis. IJCR 2017;9(5):50359-50361.
26. Varshita A, Nasim I. Allergic reactions to dental materials – A review. RJPT 2016;9(10):1819-1822.
27. Kumar RP. Latex allergy in clinical practice.Indian J Dermatol 2012;57(1):66-70.

Management of Opioid Analgesic Overdose-An Update

Mohammed Haikal Bin Sakaria[1],Anitha Roy[2],E.Roja[3],

Graduate Student[1],Assistant Professor[2],Department of Pharmacology[2],Tutor[3], Department Of Public Health Dentistry[3],

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Opioid pain relieving overdose is avoidable and conceivably deadly condition can comes about because of endorsing hones, insufficient comprehension on the patient's a piece of the dangers of medicine abuse, blunders in sedate organization and pharmaceutical manhandle. There are only few studies being done in the management of opioid analgesic overdose. Normal pharmacokinetic properties are often disrupted during an overdose and can prolong intoxication dramatically. So this review can throw light to the knowledge and management of opioid over dosage. The review is being done for studying the epidemiology of overdose, pathophysiology of opioid analgesics, clinical manifestation, diagnosis and management of over dosage of opioid analgesics.

Keyword*: opioid, overdose, pharmacokinetic, management, pathophysiology, epidemiology*

Introduction

Opioid analgesics overdose is a preventable and possibly deadly condition that outcomes from recommending rehearses, deficient comprehension on the patient's a piece of the dangers of prescription abuse, mistakes in medication organization, and pharmaceutical manhandle which then cannot be taken for granted.[1] Three elements are vital to a comprehension of opioid pain relieving harmfulness.[2] First and foremost, opioid analgesics overdose can have life-undermining harmful impacts in various organ frameworks. Second, ordinary pharmacokinetic properties are regularly upset amid an overdose and can delay intoxication significantly.[3] Third, the term of activity varies among opioid formulations, and inability to perceive such varieties can prompt to wrong treatment choices, at times with lethal outcomes.[4]

Pain is arguably the most common reason why patients seek treatment, especially in the emergency department (ED). The current physician wields many tools to relieve pain, the most potent of which are opioids. The term opiate particularly alludes to any substance that initiates rest, apathy, or trance, and it is utilized to allude to opioids or opioid subsidiaries.The word is derived from the Greek "narke" which means "numbness or torpor." It is common, however inaccurate, that the public uses the term narcotics for any illicit psychoactive substance.

In cultivation since approximately 1500 BC, pure opium is a mixture of alkaloids extracted from the sap of unripened seedpods of Papaver somniferum (poppy). Opiates, such as heroin, codeine, or morphine, are natural derivatives of these alkaloids. The term opiate is often used (albeit slightly incorrectly) to refer to synthetic opiate derivatives, such as oxycodone, as well as true opiates.

Even though opioids constitute a relatively small percentage of total overdoses encountered in the ED, still they merit particular attention because of the potential mortality/morbidity they cause when unrecognized and untreated, as well as the relative ease of reversing their effects. The notable importance of opioids in current prescribing patterns, as well as recreational uses,

mandates that physicians maintain a high index of suspicion when treating the patient who is unconscious for unknown reasons.

Epidemiology of Overdose

The quantity of opioid pain relieving overdoses is corresponding to the quantity of opioid solutions and the measurements prescribed.[5] Between 1997 and 2007, medicines for opioid analgesics in the United States expanded by 7%; the quantity of grams of methadone recommended over a similar period expanded by more than 12%.[6] In 2010, the National Poison Data System, which gets case portrayals from workplaces, doctor's facilities, and crisis offices, announced more than 107,000 exposures to opioid analgesics, which prompted to more than 27,500 admissions to human services facilities.[7] There is impressive cover between psychiatric infection and perpetual torment disorders; patients with depressive or nervousness issue are at expanded hazard for overdose, as contrasted and patients without these conditions, since they will probably get higher dosages of opioids.[8] Such patients are additionally more prone to get calming trancelike specialists (e.g., benzodiazepines) that have been firmly connected with death from opioid overdose.(9) moreover, information demonstrate that the successive remedy of opioid analgesics adds to overdose-related mortality among youngsters, who may discover and ingest operators in the home that were proposed for adults.[10]

Pathophysiology of Opioid Analgesics

Opioids increment action at least one G-protein–coupled transmembrane atoms, known as the mu, delta, and kappa opioid receptors, that create operational differing qualities from join variations, post-translational adjustment and platform of quality items, and the development of receptor heterodimers and homodimers.[11] Opioid receptors are enacted by endogenous peptides and exogenous ligands; morphine is the prototypical compound of the latter.[12] The receptors are generally disseminated all through the human body; those in the foremost and ventrolateral thalamus, the amygdala, and the dorsal-root ganglia intercede nociception.[13] With commitments from dopaminergic neurons, mind stem opioid receptors balance respiratory

reactions to hypercarbia and hypoxemia, and receptors in the Edinger–Westphal core of the oculomotor nerve control pupillary constriction.[14] Opioid agonists tie to receptors in the gastrointestinal tract to reduction gut motility.

The μ-opioid receptor is in charge of the dominance of clinical impacts created by opioids. Considers in knockout mice affirm that agonism of these receptors intercedes both absence of pain and opioid dependence.[15] Furthermore, the improvement of resilience, in which medicate measurements must be raised to accomplish a sought clinical impact, includes the dynamic failure of mu opioid receptors to engender a flag after opioid authoritative. Receptor desensitization, a basic occasion in the improvement of resistance, is a profoundly moderated handle that includes the uncoupling of the receptors from G-protein, and their resulting section into an intracellular compartment amid endocytosis. The receptors may then be come back to the layer in a procedure that resensitizes the cell to opioid authoritative. This dynamic procedure of endocytosis and reusing is hypothesized to constrain the resistance of mu opioid receptors for endogenous opioid ligands as they experience phasic discharge and quick leeway. Conversely, opioid analgesics, which are regulated tediously in long-acting details, hold on in the extracellular grid and flag through mu opioid receptors for delayed periods. Though endogenous local ligands encourage dynamic receptor cycling, opioid analgesics encourage resilience by constantly official and desensitizing the receptors as they limit receptor recycling.[16]

In any case, resistance of the pain relieving and respiratory depressive impacts of opioids is not exclusively identified with the desensitization of mu opioid receptors. Molded resilience creates when patients figure out how to relate the strengthening impact of opioids with ecological signs that dependably foresee tranquilize organization. Opioid use within the sight of these signs has weakened impacts; on the other hand, opioid use without these boosts or in new situations brings about elevated effects.[17]Tolerance of respiratory sadness seems to create at a slower rate than pain relieving resilience; after some time, this postponed resistance contracts the restorative window, incomprehensibly putting patients with a long history of opioid use at expanded hazard for respiratory depression.[18]

Clinical Manifestations of Overdose

The primary clinical appearance of opioid intoxication is respiratory discouragement. Despite the fact that the exemplary toxidrome of apnea, trance, and meiosis recommends the finding of opioid danger, these are not reliably present. Organization of helpful dosages of opioids in people without resilience to opioids causes a perceivable decrease in all periods of respiratory action, with the degree of the decay reliant on the managed dose.[19] At the bedside, in any case, the most effortlessly perceived variation from the norm in instances of opioid overdose is a decrease in respiratory rate finishing in apnea. A respiratory rate of 12 breaths for every moment or less in a patient who is not in physiologic rest emphatically proposes intense opioid inebriation, especially when joined by meiosis or daze. Meiosis alone is inadequate to gather the finding of opioid inebriation. Polysubstance ingestions may create regularly receptive or mydriatic students, as can harming from meperidine, propoxyphene, or tramadol. On the other hand, overdose from antipsychotic drugs, anticonvulsant specialists, ethanol, and other narcotic trancelike operators can bring about meiosis and trance like state, however the respiratory wretchedness that characterizes opioid poisonous quality is typically absent.[20,21]

Disappointment of oxygenation, characterized as an oxygen immersion of under 90% while the patient is breathing encompassing air and with ventilation sufficient to accomplish ordinary blood vessel carbon dioxide strain (halfway weight of carbon dioxide), is frequently created by pneumonic edema that gets to be distinctly obvious later in the clinical course. There are a few potential reasons for aspiratory edema. One likely cause is that endeavored motivation against a shut glottis prompts to a diminishing in intrathoracic weight, which causes liquid extravasation. Then again, intense lung damage may emerge from a component like that hypothesized for neurogenic aspiratory edema. In this situation, thoughtful vasoactive reactions to worry in a patient who has stirred after inversion of inebriation finish in spillage from aspiratory capillaries.[22]

Hypothermia may emerge from a tirelessly inert state in a cool domain or from confused endeavors by observers to turn around opioid inebriation by inundating a patient in icy water. What's more, people who have been lying stationary in an opioid-initiated daze might be liable to rhabdomyolysis, myoglobin uric renal disappointment, and the compartment disorder. Other lab variations from the norm incorporate lifted serum aminotransferase fixations in relationship with liver harm created by acetaminophen or hypoxemia. Seizures have been related with overdose of tramadol, propoxyphene, and meperidine.[23,24]

Symptoms

The symptoms for opiate poisoning may be a chronic problem, in which case the main complaint will be of constipation. There may be nausea, vomiting or just loss of appetite. There may be sedation and craving for the next dose. Moreover, acute toxicity presents with drowsiness that will be more severe if there is also alcohol involved, or involvement of other sedatives. There may be nausea or vomiting.

Signs [25]

One of the signs is respiratory depression may be apparent. Hypotension and tachycardia are possible. There are usually pinpoint pupils but this sign may be absent if other drugs are involved. Besides, the 'post-mortem sole incision' sign has been identified. This is an incision made in the sole by an acquaintance in the belief that the subsequent blood loss will reduce the likelihood of death in an individual who has taken an accidental overdose of an opiate.

Diagnosis of Overdose

The nearness of hypopnea or apnea, meiosis, and daze ought to lead the clinician to consider the finding of opioid pain relieving overdose, which might be construed from the patient's indispensable signs, history, and physical examination. In patients with extreme respiratory

misery, rebuilding of ventilation and oxygenation outweighs getting the historical backdrop of the present disease or playing out a physical examination or demonstrative testing.

After the patient's condition is balanced out, the clinician ought to ask about the utilization of all opioid analgesics, acetaminophen (counting items coformulated with acetaminophen), and illegal substances and figure out if the patient has had contact with anybody accepting pharmacologic treatment for constant torment or opioid dependence.[26] In playing out the physical examination, the clinician ought to assess the size and reactivity of the understudies and the level of respiratory exertion and search for auscultatory discoveries suggestive of pneumonic edema. The patient ought to be totally uncovered to take into consideration a careful look for fentanyl patches. Also, the clinician ought to palpate muscle assembles; the solidness, swelling, and delicacy that describe the compartment disorder (which comes about when lethargic patients lie on a muscle compartment for quite a while) warrant coordinate estimation of compartment weights. At last, the acetaminophen focus ought to be measured in all patients in light of the commonness of redirection and abuse of acetaminophen-containing opioids. Clinicians regularly ignore acetaminophen hepatotoxicity.[27]

Subjective examinations of pee for medications of manhandle (toxicology screens) once in a while influence choices about patient care and have little part in the quick assessment and administration of opioid inebriation, for a few reasons. To begin with, naloxone ought to never be withheld from a patient with apnea on the grounds that the consequences of subjective tests are inaccessible. Second, the administration of opioid overdose, regardless of the causative operator, changes nearly nothing. At last, standard poisonous screens, which recognize methadone, fentanyl, hydromorphone, and different mixes just occasionally, give minimal valuable clinical data. More current subjective screens that distinguish a more extensive scope of opioid analgesics may permit clinicians spend significant time in torment treatment, psychological well-being, or different territories of pharmaceutical to recognize patients who have strayed from recommended treatment regimens; more prominent diagnostic exactness, notwithstanding, does not change the administration of intense overdose. Quantitative measures

of medication fixations are futile in instances of overdose in light of the fact that patients who have been recommended raised dosages of opioid analgesics may have helpful serum focuses that enormously surpass research facility reference ranges.[38]

Management

- Do not postpone building up an unmistakable aviation route, satisfactory ventilation and oxygenation if awareness is impaired.[29]
- Give naloxone intravenously (IV) (0.4-2 mg for a grown-up and 0.01 mg/kg body weight for kids) if trance like state or respiratory misery is present.
- Give intramuscularly (IM) if no vein is accessible. Rehash the measurement if there is no reaction inside two minutes. Naloxone is an aggressive rival and huge measurements (4 mg) might be required in a seriously harmed persistent.
- Failure of an unmistakable sedative overdose to react to vast measurements of naloxone proposes that another focal sensory system (CNS) depressant, or cerebrum harm, is available.
- Observe the patient precisely for repeat of CNS and respiratory dejection. The plasma half-existence of naloxone is shorter than that of all opioid analgesics. Rehashed measurements might be required. Naloxone IM ought to be considered if the patient is debilitating to self-release, as it might help decrease the danger of respiratory capture when the IV naloxone wears off.
- If somebody takes an overdose of IV heroin it is critical to regulate naloxone at the earliest opportunity. Frequently this is finished by paramedics however a few people have pushed that clients ought to have a supply on the off chance that one of their number overdoses and treatment can be begun immediately. They are frequently hesitant to call for help.[30]
- IV mixtures of naloxone might be helpful where rehashed measurements are required. Naloxone 400 micrograms/ml is weakened with sodium chloride 0.9 % or glucose 5%.

Five ampoules of naloxone 400 micrograms/ml (2 mg) per 500 ml give 4 µg/ml. 66% of the bolus measurement expected to turn around inebriation given hourly as a consistent implantation regularly keeps up respiratory exertion without advancing sedative withdrawal. Implantations are not a substitute for continuous audit of the patient's clinical state.

- Give oral actuated charcoal, gave the aviation route can be ensured, if a generous sum has been ingested inside two hours.
- Naltrexone is prescribed by the National Institute for Health and Care Excellence (NICE) as a treatment choice for individuals who have been opioid-subordinate however who have quit utilizing opioids and who are exceptionally energetic to remain free from the medications in a restraint program. It is an aggressive sedative enemy that will hinder the impact of heroin. It ought to just be given to individuals who have been told about the issues related with treatment and with legitimate supervision. Treatment with naltrexone ought to be given as a major aspect of a bolster program to help the individual deal with their opioid dependence.[32]
- Choices incorporate vigilant holding up, with or without the utilization of intestinal medicines, entire entrails water system, endoscopic expulsion or surgery. A hazard advantage examination ought to be performed, mulling over whether the patient is symptomatic or asymptomatic and whether the treatment is probably going to increment or abatement the danger of bundle crack. Most patients can be overseen by careful holding up and released from doctor's facility when the bundle has been emptied with an ordinary solid discharge. Surgery ought to just be performed in body packers with indications of inebriation or ileus.[33]
- A new naloxone auto-injection device to treat opioid overdose.[34][35]

Summary

Opioid pain relieving overdose is an existence due to certain chronic cases that require opioid analgesics as the medication. Thus, since there is a lot of consuming opioid analgesics, this situation will lead to over dosage of opioid analgesics.[36] Therefore, medical experts have to look on this condition better because the effects of opioid analgesics overdose are too severe. Moreover, opioid analgesic poisoning is a life-threatening condition, and the antidote which is naloxone may have limited effectiveness in patients with opioid poisoning due to over dosage. [37]This unpredictable clinical course of intoxication requires empirical management of this potentially lethal condition so that it would not cause any troubles in future.[38][39]

Corresponding Author

E.Roja,

Tutor, Department Of Public Health Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Science,

Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077,Tamil Nadu, India.

Telephone Number- 9994174480

E-mail- itsrojaa@gmail.com

References

1. Okie S. A flood of opioids, a rising tide of deaths. N Engl J Med 2010;363:1981-1985

2. Opioid analgesic risk evaluation and mitigation strategies (REMS)Joint Meeting of the Anesthetic and Life Support Drugs Advisory Committee and the Drug Safety and Risk Management Advisory Committee. Silver Spring, MD: Food and Drug Administration, 2010.
3. Allen L, Kimura K, MacKichan J, Ritschel W, Committee for Pharmacokinetic Nomenclature of the American College of Clinical Pharmacology. Manual of symbols, equations & definitions in pharmacokinetics. J Clin Pharmacol, 1982;22:1S-23S
4. Larson AM, Polson J, Fontana RJ, et al. Acetaminophen-induced acute liver failure: results of a United States multicenter, prospective study. Hepatology, 2005;42:1364-1372
5. Paulozzi LJ, Budnitz D, Xi Y. Increasing deaths from opioid analgesics in the United States. Pharmacoepidemiol Drug Saf, 2006;15:618-627
6. Paulozzi L, Weisler R, Patkar A. A national epidemic of unintentional prescription opioid overdose deaths: how physicians can help control it. J Clin Psychiatry, 2011;72:589-592
7. Toblin RL, Paulozzi LJ, Logan JE, Hall AJ, Kaplan JA. Mental illness and psychotropic drug use among prescription drug overdose deaths: a medical examiner chart review. J Clin Psychiatry, 2010;71:491-496
8. Bailey JE, Campagna E, Dart RC. The underrecognized toll of prescription opioid abuse on young children. Ann Emerg Med 2009;53:419-424
9. Waldhoer M, Bartlett SE, Whistler JL. Opioid receptors. Annu Rev Biochem 2004;73:953-990
10. Lord JA, Waterfield AA, Hughes J, Kosterlitz HW. Endogenous opioid peptides: multiple agonists and receptors. Nature 1977;267:495-499.
11. Stein C. The control of pain in peripheral tissue by opioids. N Engl J Med 1995;332:1685-1690.
12. Lalley PM. Opioidergic and dopaminergic modulation of respiration. Respire Physiol Neurobiol 2008;164:160-167.

13. Mattes HW, Maldonado R, Simonin F, et al. Loss of morphine-induced analgesia, reward effect and withdrawal symptoms in mice lacking the mu-opioid-receptor gene. Nature 1996;383:819-823.
14. Whistler JL. Examining the role of mu opioid receptor endocytosis in the beneficial and side-effects of prolonged opioid use: from a symposium on new concepts in mu-opioid pharmacology. Drug Alcohol Depend 2012;121:189-204.
15. Ehrman R, Ternes J, O'Brien CP, McLellan AT. Conditioned tolerance in human opiate addicts. Psychopharmacology (Berl) 1992;108:218-224.
16. White JM, Irvine RJ. Mechanisms of fatal opioid overdose. Addiction 1999;94:961-972
17. Hoffmann RS, Goldfrank LR. The poisoned patient with altered consciousness: controversies in the use of a `coma cocktail.' JAMA 1995;274:562-569
18. Hoffmann JR, Schriger DL, Luo JS. The empiric use of naloxone in patients with altered mental status: a reappraisal. Ann Emerg Med 1991;20:246-252
19. Clark RF, Wei EM, Anderson PO. Meperidine: therapeutic use and toxicity. J Emerg Med 1995;13:797-802
20. Sporer KA, Dorn E. Heroin-related noncardiogenic pulmonary edema: a case series. Chest 2001;120:1628-1632
21. Talaie H, Panahandeh R, Fayaznouri M, Asadi Z, Abdollahi M. Dose-independent occurrence of seizure with tramadol. J Med Toxicol 2009;5:63-67
22. Kaiko RF, Foley KM, Grabinski PY, Central nervous system excitatory effects of meperidine in cancer patients. Ann Neurol 1983;13:180-185
23. Benomran F; Postmortem sole incisions - A new sign of heroin overdose? J Forensic Leg Med. 2008; 15(1):59-63.
24. Moon JM, Chun BJ. Fentanyl intoxication caused by abuse of transdermal fentanyl. J Emerg Med 2011;40:37-40
25. Khandelwal N, James LP, Sanders C, Larson AM, Lee WM. Unrecognized acetaminophen toxicity as a cause of indeterminate acute liver failure. Hepatology 2011;53:567-576.

26. Zell, Steven C., and Kenneth J. Kurtz. "Severe exposure hypothermia: a resuscitation protocol." Annals of emergency medicine 1985;14:4: 339-345.
27. Bowman S, Eiserman J, Beletsky; reducing the health consequences of opioid addiction in primary care. Am J Med. 2013;126(7):565-571.
28. Beauverd Y, Poletti PA, Wolff H, et al; A body-packer with a cocaine bag stuck in the stomach. World J Radiol. 2011;3(6):155-158.
29. Sheela, D. A Concept On The Development Of Buprenorphine Autoinjector For Self And Emergency Administration. International Journal Of Pharmacy And Pharmaceutical Sciences, [S.l.],2015;253-257, 0975-1491.
30. Roy, Anitha,R. Vijayaraghavan, "A comparative study of the effect of amikacin administered through autoinjector and manual injection on biochemical parameters in rats.Journal of Applied Pharmceutical Science.2016:6(02):109-114.
31. Sheela, D., Geetha, R., Mohan, S., & Vijayaraghavan, R. A Concept On The Development Of Buprenorphine Autoinjector For Self And Emergency Administration. International Journal OfPharmacy and Pharmaceutical Sciences,2015; 8(8), 253-257.
32. Sheela, D., Vijayaraghavan, R., & Senthilkumar, S. (2017, September). A study on the safety evaluation of buphrenorphine administered through an autoinjector compared with manual injection using haematological and biochemical variables in rats. Retrieved from google scholar December 13, 2017,
33. Devaraj, Sharmila Devi, and Prasanna Neelakantan. "Curcumin-pharmacological actions and its role in dentistry." Asian Journal of Pharmaceutical Research and Health Care.2014;6:1.
34. Ashwini, Shenai, D. Ezhilarasan, and R. Anitha. "Cytotoxic effect of Caralluma fimbriata against human colon cancer cells." Pharmacogn J 2017; 9(2): 204-207.

Management of Patients on Anti- Coagulant Therapy Undergoing Dental Procedures – A Review Article

Nandhini.S[1], D. Pradeep[2], N. Mahathi[3],
Graduate Student[1],Reader[2], Department of Oral and Maxillofacial Surgery,
Reader[3], Department of Oral and Maxillofacial Surgery,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Anticoagulation therapy is one of the most prevalent forms of treatment used in contemporary medicine. Thrombogenesis (clot formation) includes 2 principal processes: platelet aggregation and coagulation. The aim of this literature review is to evaluate the available evidence on the impact of anticoagulant medications on dental treatment and highlight certain patient management issues closely interrelated to various aspects of dental treatmentImpact of anticoagulants on patients undergoing dental treatment is very high.Mainly for cardiac patients, the physician's consent is mandatory before undergoing any dental procedures.INR value plays a major role play here which is now widely used for monitoring anticoagulant therapy and dosage planning.

Key Words*: platelet, coagulation, dental, anti-coagulant, thrombosis, fatal*.

Introduction

Millions of patients worldwide take medications that alter hemostasis in order to reduce the risk for thromboembolic events, such as strokes. Anticoagulation therapy is one of the most prevalent forms of treatment used in contemporary medicine. Thrombogenesis (clot formation) includes 2 principal processes: platelet aggregation and coagulation. These patients, however, present the most common potential bleeding problem that dentist's encounter.Oral anticoagulant therapy is prescribed for both prophylactic and therapeutic purposes.[1]Oral Anticoagulants reduce blood clotting which can help prevent deep vein thrombosis, pulmonary embolism, myocardial infarction and ischemic stroke. Frequently raised questions concern the safety and efficacy of the various anticoagulation regimens and their accompanying thromboembolic and bleeding risks relative to invasive dental procedures. The aim of this literature review is to evaluate the available evidence on the impact of anticoagulant medications on dental treatment and highlight certain patient management issues closely interrelated to various aspects of dental treatment.

A 2006 survey of hematologists and dentists revealed no consistent management of preoperative anticoagulation. Preoperative management ranged from the continuation of the regular dose of anticoagulant to reduction of the dose to its complete cessation. Another practice is to stop warfarin 5 days before the procedure and administer bridging anticoagulation, typically low-molecular-weight heparin, for patients at high risk of thromboembolism. This approach, which aims to avoid a residual anticoagulant effect at the time of the procedure, deals with dentists' perception of an increased risk of serious bleeding if warfarin is not interrupted. Dentists' concern about post-operative bleeding may be based, in part, on their observation that for patients not on anticoagulation, bleeding after dental procedures, such as extractions, can be excessive, given the highly vascular supporting structures. Prevention of oral bleeding is also desirable because it can be distressing for patients, presents challenges for the homebound elderly and may deter future dental care. Interruption of warfarin therapy, however, may increase the risk of thromboembolism, such as stroke, which can be associated with mortality and long-term morbidity.Further, bleeding after dental surgery is easily seen and usually self-limiting, and most often can be managed with local measures, such as biting on gauze. [2]

Finally, recent editorials and review shave suggested that dentists concerns about the risk of bleeding when procedures are done on patients taking anticoagulation havebeenover stated against this background, we did a systematic review of studies assessing outcomes for patients treated with anticoagulants who required elective dental procedures. We aimed to determine the risk of-bleeding for patients who continued warfarin therapy compared with that for those whose dose was reduced or interrupted.

Oral Anti Coagulation Medication

The most frequently used medications for oral anticoagulant therapy are: Anticoagulants with indirect action (coumarin derivatives)

a. Acenocoumarol- derivative of coumarin and vitamin K antagonist.

b. Warfarin sodium- it is also a derivative of coumarin.

c. Phenindione

The novel oral anticoagulants [NOACs] or directly acting oral anticoagulants [DOACs] are:

Direct Thrombin Inhibitors

a. Dabigatran- is a selective, reversible direct inhibitor.

b. Argatroban

Direct Factor XaInhibitors

a. Apixaban- is a potent, reversible, highly selective direct inhibitor.

b. Rivaroxaban

c. Edoxaban

Test for Anti Coagulation Assessment

The prothrombin time ratio (PTR), defined as the patient's prothrombin time (PT) was used to monitor anticoagulant therapy for many years. Because of the variability of Prothrombin Time (PT) values from different reagents, a system of standardizing the reporting of anticoagulation activity has been developed by the World Health Organization. In 1985, the International Committee on Thrombosis and Homeostasis requested that all lots of thromboplastin have an indication of their international sensitivity index (ISI) which allows standardization of the results from different laboratories by the introduction of the INR. [3,4]

INR = [PT test / PT normal]

INR- International Normalized Ratio

It is now widely used for monitoring anticoagulant therapy and dosage planning. The INR for a healthy patient is 1 and the therapeutic INR for those on anticoagulant therapy typically ranges from 2 to 4, depending on the reason for anticoagulation.

Risk of Thrombosis on Stopping Anti-Coagulants

In the past decade, it has become clear that routine discontinuation of oral anticoagulant therapy for dental procedures is not supported by the scientific literature, as it may put patients at unnecessary medical risk for thromboembolic events either from the cessation of anticoagulant therapy or because of rebound phenomenon unless serious bleeding is anticipated, the therapy should be continued.The risk of thrombosis associated with temporarily discontinuing anticoagulants prior to dental surgery is small but potentially fatal. Currently, most guidelines indicate that patients with an INR less than 3.5 can undergo minor oral surgery (e.g., simple single extraction) without any adjustment in anticoagulation. Withdrawal or temporary interruption of anticoagulant medication, could lead to thromboembolic events.[5]

Risk of Bleeding on Continuing Anti-Coagulants

The gums and their supporting structures are highly vascular and prone to bleeding when damaged. While visible bleeding in the mouth is not considered as dangerous as "silent" bleeding that occurs internally or at non-compressible sites, oral bleeding can be distressing for the patient and potentially life threatening. However, many primary care dental procedures are unlikely to cause bleeding that cannot be managed with local measures. These include minimally invasive procedures such as periodontal probing, scaling above the gum like, polishing, and orthodontic procedures. Invasive procedures such as local infiltration, scaling below the gums, root planning, biopsies, tooth extractions, minor periodontal surgery, cavity filling, endodontic procedures (root canals), and prosthodontic procedures (crowns, bridges, and implants) are also unlikely to cause

significant bleeding. Significant bleeding is more likely to occur with more invasive procedures such as extraction of impacted teeth and use of periodontal flaps.[6,7]

Patients with liver disease, kidney disease, hypertension, and gingival disease also have an increased risk of bleeding. The skill and experience of the dental practitioner and availability of haemostatic measures must also be considered when deciding whether to stop anticoagulants or anti-platelet agents.One of the most frequently cited studies regarding thromboembolic risks in dentistry are Wahl's literature review in which 5/493 patients who stopped anticoagulants for a dental procedure had serious embolic complications, including 4 deaths. This report has been criticized because the patients had stopped anticoagulation for longer than usually recommended and the relationship to coagulation status was unclear. However, considering that recurrent venous thromboembolism is fatal in ~6% of patients and arterial thromboembolism is fatal in ~20%, many consider the risks of thromboembolism outweigh the risks from bleeding from oral procedures.Thromboembolic risks vary with the reason for anticoagulation or anti-platelet therapy. The American College of Chest Physicians has published a stratification of thromboemobolicrisk.Dabigatran and rivaroxaban are not included, but risks would likely be similar to warfarin for similar indications.

The authors concluded that the risk of thromboembolism outweighed the risk of postoperative bleeding and patients whose INR is up to 4.0 do not have clinically significant bleeding post-operatively. The risks of bleeding that occurs in patients on continued anticoagulant therapy after dental surgical procedures can be easily controlled by local homeostatic measures.

Local Measures Used in Bleeding Control

Local Homeostasis

Local homeostasis after single or multiple teeth extractions can be achieved through: - Local pressure (biting on gauze); site packing with gelatin sponges; absorbable oxy cellulose; microcrystalline; collagen; additional suturing; Electro cauterization; topical thrombin powder.

Fibrin sealants induce clot formation at the site of the surgical wound. It mimics the last phase of blood clotting by conversion of fibrinogen to fibrin. The system has 2 components. The first consists mainly of fibrinogen and plasma proteins, and the second consists of thrombin and calcium chloride. When the 2 components are mixed, thrombin converts fibrinogen into fibrin so

that clotting is initiated and the mixture is solidified. Fibrin sealants for oral surgery in patients on oral anticoagulant therapy can be used to reduce postoperative hemorrhage in the therapeutic INR range of 1 to 5 in patients with a wide range of degrees of surgical trauma. 5% tranexamic acid mouthwashes used 4 times a day for 2 days (10ml in mouth for 2min) was reported to be effective in patients on anticoagulant therapy. This treatment modality is an attempt to reduce the amount of lysed fibrin and consequently the incidence of postoperative bleeding. It is conceivable that the mouthwash may have an effect only on the superficial clot and not on bleeding from the depth of the socket, an area not accessible to the mouthwash. [8]

Pain Control

Generally paracetamol is considered a safe analgesicdrug for patients taking anticoagulant medications and it maybe taken in normal doses if pain control is needed and no contraindicationexists.Recommendations of the British Committee for Standards in Hematology. Patients taking warfarin shouldnotbe prescribed nonselective NSAIDs as analgesicsfollowing dental surgery. These drugs inhibit platelet aggregation and may cause GI bleeding and peptic ulceration and/or perforation. Increased bleeding may occur during concomitant NSAID therapy with warfarin independently of an increase in INR.Patients taking Acenocoumarol should not be prescribed nonselective NSAIDs as analgesics following dental surgery, which potentate bleeding.

Association of new oral anticoagulants with other anticoagulants, platelet inhibitors(Aspirin, Clopidogrel, Ticlodipine, Prasugrel,Ticagrelor, and others), and non-steroidal anti-inflammatory drugs (NSAID) increases the bleeding risk. COX-2 inhibitors are unlikely to increase bleeding time following invasive dental procedures, although thereare no prospective studies in humans.Interactions with drugs frequently used or prescribed in dentistry.Macrolide antibiotics (i.e., erythromycin, clarithro-mycin, and possibly azithromycin) and sulphonamides havebeen implicated in causing significant episodes of bleedingin patients taking warfarin or acenocoumarol. Other drugs that may enhance the hypoprothrom-binemic effect of anticoagulant drugs include tetracyclines(especially doxycycline or tetracycline), and in particular the second and third generation cephalosporins, andlevofloxacin.Postulated mechanisms include an antibiotic-induced reduction in prothrombin activity(hypoprothrom-binemic) and a reduction in

gastrointestinal bacteria flora essential for vitamin K production which is subsequently used to produce various clotting factor.Dabigatran and rivaroxaban are reported to have comparatively few drug-drug interactions.The concomitant use of systemic azoleantimycotics(ketoconazole, Iitraconazole, voriconazole, andposaconazole) as well as HIV protease inhibitors is not recommended in patients being treated with rivaroxaban.Fluconazole is expected to have less effect on rivaroxaban exposure and can be co-administered withcaution.Although platelet aggregation was reported to beunaffected, concomitant use of some NSAIDs and rivaroxaban significantly increased bleeding time comparedwith rivaroxaban alone. [9,10]

Interaction with Drugs Frequently used in Dentistry

Macrolide antibiotics (i.e., erythromycin, clarithromycin, and possibly azithromycin) and sulphonamides have been implicated in causing significant episodes of bleeding in patients taking warfarin or acenocoumarol. Other drugs that may enhance the hypoprothrom- binemic effect of anticoagulant drugs include tetracyclines (especially doxycycline or tetracycline), and in particular the second and third generation cephalosporins, and levofloxacin. Postulated mechanisms include an antibiotic- induced reduction in prothrombin activity (hypoprothrombinemia) and a reduction in gastrointestinal bacteria flora essential for vitamin K production which is subsequently used to produce various clotting factors.Dabigatran and rivaroxaban are reported to have comparatively few drug-drug interactions. [11]The concomitant use of systemic azoleantimycotics (ketoconazole, itraconazole, voriconazole, and posaconazole) as well as HIV protease inhibitors is not recommended in patients being treated with rivaroxaban.

Fluconazole is expected to have less effect on rivaroxaban exposure and can be coadministered with caution. Although platelet aggregation was reported to be unaffected, concomitant use of some NSAIDs and rivaroxaban significantly increased bleeding time compared with rivaroxaban alone. There are potential weaknesses of our systematic re-view. First, we searched only the English-language literature. However, the degree of bias introduced by the exclusion of non-English literature is debatable. A study of number of disease areas showed that language-restricted meta-analyses, compared with language-inclusive meta-analyses, did not differ in their estimate-of the benefit or the effectiveness of an intervention.

Second, in our meta-analysis, 4 of 5 trials were of low methodological quality, scoring only 1 or 2 on a validated quality assessment scale. Only 1 study included a description of withdrawals and dropouts.Two19,21of the 5 trials did not report the indication forwarfarin therapy for their patient population, which determines the therapeutic range of the INR at which patients are normally maintained and gives a sense of the risk of bleeding and thromboembolism. Studies that include a majority of patients anticoagulated for atrial fibrillation or native valvulopathy maintained at lowerINRs (2.0–3.0) would be expected to show a lower incidence of bleeding than studies that include patients on anticoagulant therapy who have prosthetic heart valves and are therapeutically maintained at higher INRs (2.5–3.5). In addition to INR, surgical skill, complexity of thesurgical procedure, comorbid conditions such as liverdisease or blood dyscrasias, and the effect of combined therapy with antiplatelet and nonsteroidal anti-inflammatory agents can increase the risk of bleeding during a surgical procedure. Further, 5 studies included in this systematic review were conducted in a hospital setting. In 1 study,20th setting was not specified. It is possible that the results from this review maynot be generalizable to private practice.[12,13]

Our findings question the current practice of interrupting warfarin therapy for dental procedures. However, a number of pragmatic questions from everyday dental practice preclude an across-the-board recommendation continuewarfarin therapy around the time of dental procedures. For example, questions arise about management of patients with comorbid factors; the use of additional local measures and antifibrinolytic agents; and the need for referral to specialists, hospital care, or bridging therapy. Nonetheless, this is a clinical dilemma that is too prevalent and too important to defer because of insufficient evidence, with resulting continued ambiguous.

Summary

Dental treatment performed in patients receiving oral anticoagulant drug therapy is becoming increasinglycommon in dental offices.The aim of oral anticoagulant therapy is to reduce blood coagulability to an optimal therapeutic range withinwhich the patient is provided some degree of protection fromthromboembolic events. This is achieved at the cost of a minor risk of hemorrhage.Frequently raised questions concern the safety and efficacy of the various

anticoagulation regimens and their accompanying thromboembolic and bleeding risks relative to invasive dental procedures.

The aim of this literature review is to evaluate the available evidence on the impact of anticoagulant medications on dental treatment and highlight certain patient management issues closely interrelated to various aspects of dental treatment.For that purpose literature search in the electronic database of Medscape, PubMed-Medline, Science Direct, and EBSCO host, in the data base of Medical UniversityPlovdiv and specialized published books in general medicineand dentistry was made. A total of 33 publications between 1995 and 2013were identified: 12 review articles, 11 randomized controlledandnon-randomized studies, 6 guidelines and practicalguides, 1 meta-analysis and 3 specializedbooks.[14,15]

Conclusion

INR values should be obtained within 24 hours before the dental procedure. For patients with INR in the therapeutic range 2-4 or below, therapy need not be modified or discontinued for simple single dental extractions. More complicated and invasive oral surgical procedures for patients with an INR on the high end of the scale or greater than 3.5 should be referred to physician for dose adjustment or therapy alteration before invasive dental procedures. The risk of bleeding may be minimized by: use of oxidized cellulose or collagen sponges, fibrin sealants.[15]

Tranexamic acid mouthwashes used four times a day for 2days.Co morbid conditions as liver disease, bone marrow disorders, biliary tract obstruction, malabsorption, renal disease, and cancers such as leukemia may potentate anexisting bleeding problem. Increased inflammation of theoral tissues in patients on OAT can contribute to excessive bleeding even with minor procedures.These patients should NOT have a surgical dental procedure and should be referred to a dental hospital or hospital-based oral and maxillofacial surgery department.[16,17]

The use of concomitant medications, including antibiotics, antifungals, non-steroidal anti-inflammatory drugs(NSAIDs), and other platelet aggregation inhibitors may affect a patient’s ability after a routine dental procedure. For patients being treated with one of these new oral anticoagulants, the amount of “real-world” data and experience regarding the management of

patients undergoing dental procedures that are likely to involve significant bleeding is currentlylacking.Clearly, clinical studies are needed to determine the effects that the new oral DTIs and FXaIs have on bleeding and hemostasis after tooth extractions and other surgical and dentalprocedures. At this time patients taking dabigatran, rivaroxaban,orapixaban should be consulted with the treating physician prior dental surgical procedures.[18,19]

The aim of oral anticoagulant therapy is to reduce blood coagulability to an optimal therapeutic range within which the patient is provided some degree of protection from thromboembolic events.Impact of anticoagulants on patients undergoing dental treatment is very high.Mainly for cardiac patients, the physician's consent is mandatory before undergoing any dental procedures.INR value plays a major role play here which is now widely used for monitoring anticoagulant therapy and dosage planning.[20,21,22]

Corresponding Author

N. Mahathi,

Reader,Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077,Tamil Nadu, India.

Telephone Number-9840958141

Email Id- nerallamahathi@gmail.com

References

1. Firriolo EJ, Hupp WS. Beyond warfarin: the new generation of oral anticoagulants and their implications for the management of dental patients. Surg Oral Med Oral Pathol Oral Radiol, 2012; 113(4):431–441.
2. FirdusFareen, Revathy Gounder. Knowledge, Attitude, Practice among Dental Students Regarding Management of Patients Undergoing Anticoagulant Therapy - A Survey, Int. J. Pharm. Sci. Rev. Res., 2017:44(1)71-72.

3. Santhosh Kumar M P.Dental Management Of Patients On Antiplatelet Therapy: Literature Update,Asian Journal Of Pharmaceutical And Clinical Research; 2016;9(3) 30-36.
4. P. Kiruthika, M. Dhanraj, Ashish R. Jain. Effectiveness of local haemostatic agents following dental extraction: A systematic review. Journal of Advanced Pharmacy Education &Research: 2017;7(1):5-8; 2249-3379.
5. Broomhead RH, Mallett SV. Clinical aspects of coagulation. Anaesthesia&Intensive Care Medicine. 2010; 11(5):195–199.
6. PL Detail-Document, Managing Anticoagulant and Antiplatelet Drugs Before Dental Procedures. Pharmacist's Letter/Prescriber's Letter. 2011.
7. PopilievIl. Clinical and therapeutic guide of Cardiology. Med Pub House "Raikov" 2002;1:233-238
8. Manolov P, Belovejdov N, Monova D. Handbook of Drug Therapy. Pub house "Svetovit" 2007;1:19-41.
9. Sanz M. Screening for international normalized ratio in the dental office may provide useful information to prevent both hemorrhagic and thromboembolic events. J Evid Based Dent Pract.2012; 12(3):164-6.
10. Shamoun FE, Martin EN, Money SR. The novel anticoagulants: The surgeons' prospective. Surgery. 2013; 153(3):303–307.
11. Rosencher N, Steib A, Llau JV. Surgery and invasive procedures in patients on long-term treatment with direct oral anticoagulants: Thrombin or factor-Xa inhibitors. Recommendations of the Working Group on perioperativehaemostasis and the French Study Group on thrombosis and haemostasis Arch Cardiovasc Dis. 2011; 104(12):669-76.
12. Steffel J, Braunwald E. Novel oral anticoagulants: focus on stroke prevention and treatment of venous thrombo-embolism. Eur Heart J.2011; 32(16):1968–1976.
13. Thomas TF, Ganetsky V, Spinler SA. Rivaroxaban: An Oral Factor Xa Inhibitor. ClinTher. 2013; 35(1):4– 27.
14. Airoldi G, Campanini M. Apixaban. Italian Journal of Medicine. 2011;5(2):128–134.

15. Aframian DJ, Lalla RV, Peterson DE. Management of dental patients taking common hemostasis- altering medications. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2007;3: 103-11.
16. Carter G, Goss AN, Lloyd J, Tocchetti R. Current concepts of the management of dental extractions for patients taking warfarin. Aust Dent J. 2003; 48(2):89-96.
17. Aldous JA, Olson CJ. Managing patients on warfarin therapy: a case report. Spec Care Dentist. 2001; 21(3):109-112.
18. Kumar S, Dagli RJ, Mathur A, Jain M, Prabu D, Kulkarni S. Perceived sources of stress amongst Indian dental students. Eur J Dent Educ. 2009; 13(1):39–45.
19. Obrez A, Lee DJ, Organ-Boshes A, Yuan JC, Knight GW. A clinically oriented complete denture program for second-year dental students. J Dent Educ. 2009; 73(10):1194–201.
20. Haralur SB, Al-Malki AE. Student perception about efficacy of preclinical fixed prosthodontic training to facilitate smooth transition to clinical context. J Educ Health Promot. 2014; 3:73.
21. Rauf A, Baig L, Jaffery T, Shafi R. Exploring the trustworthiness and reliability of focus groups for obtaining useful feedback for evaluation of academic programs. Educ Health (Abingdon) 2014; 27(1):28–33.

Maxillary Sinus- A Review of Literature

SameerMohideenGani[1], AravindSivakumar[2],

Graduate Student[1], Senior Lecturer[2], Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstarct

The maxillary sinuses are a subset of the paranasal sinuses and can generally be thought of as the sinuses, or cavities, beneath the cheeks and on the sides of the nose. The maxillary sinuses are the most common region that paranasalsinus cancer arises, and most cancers in the maxillary sinuses are squamous cell carcinomas, meaning they arise from flat, thin cells in the epidermis lining the maxillary sinus. Malignant tumors of the paranasal sinus are uncommon, constituting less than 1% of all malignancies and 3% of all head and neck cancers. Nonsquamous cancers of the maxillary sinus are even rarer as is evident from the limited data available on the clinical characteristics and outcomes. Mucoepidermoid carcinoma accounts for 13% of all malignancies occurring in maxillary sinus.

Key Words: *Maxillary sinus, IA, CCRT, Carcinoma, Malignant neoplasm*

Introduction

Oral squamous cell carcinoma represents 90% to 95% of all malignant cancers of the oral cavity. It is classically regarded as an adult disease and has a high correlation with alcohol and tobacco consumption. Oral Squamous cell carcinoma occurs in several well established intraoral sites, including the floor of mouth, tongue (most common), gingiva, lips, and buccal mucosa. It might also present in the tooth-bearing segment of either the maxilla or the mandible, with bony involvement. The standard treatment in the early stage of maxillary sinus cancer is surgical resection followed by radiation therapy.

Computed and frontal tomograms were taken of 20 patients with carcinoma of maxillary sinus and were evaluated in order to compare the diagnostic accuracy of both the procedures. The diagnostic accuracy of CT in diagnosing the tumor was was greater than frontal tomography.

Maxillary Sinus

Malignant neoplasms of the paranasal sinuses and nasal cavities are rare ,comprising only 3% of all head and neck malignancies. Malignancies of the nasal cavity and paranasal sinuses constitute fewer than 1% of all malignancies and 3% of upper aerodigestive tract malignancies.[1] The majority of these tumors are in the maxillary sinus, and squamous cell carcinoma is the commonest histological type.[2] As a subset of maxillary sinus malignancies, non-squamous cell cancers of the maxillary sinus are rare .[3] Sinonasal malignancies occur twice as often in males as in females, and are most often diagnosed in patients 50 to 70 years of age.[4] Mucoepidermoid carcinoma arising from mucous glands of maxillary sinus are extremely rare and accounts for 13% of all malignancies occurring in maxillary sinus.[1]

Sinus means “pocket” in Latin. Humans have these air-filled spaces that surround the nasal cavity called paranasal sinuses. The four-paired paranasal sinuses include frontal and ethmoid sinuses between the eyes, sphenoid sinuses behind the ethmoid, and maxillary sinuses surrounding the nasal cavity. The maxillary sinuses are the largest of the paranasal sinuses. Although not proven, the biological roles of sinuses include decreasing the relative weight of skull, increasing voice resonance, providing a buffer against blows to face, insulating structures, and humidifying/heating inhaled air. (12)

The maxillary sinus is pyramidal in shape. The base of the pyramid is the medial wall of the sinus that is also the lateral wall of the nasal cavity, and its apex is pointed toward the zygomatic bone. The roof of the sinus is also the floor of the orbit. The average volume of a sinus is about 15 ml (range between 4.5 and 35.2 ml). The maxillary sinus maintains its overall size while the posterior teeth remain in function as the size expands with age, especially when posterior teeth are lost. This process is called pneumatization and is possibly the result of atrophy caused by reduced strain from occlusal function.

The membrane that lines the walls of the maxillary sinus is called Schneiderian membrane. It is multilayered and 0.13 mm to 0.5 mm in thickness. The layers include the respiratory epithelium

(pseudo-stratified ciliated columnar epithelium) that covers a loose, highly vascular connective tissue and periosteum. The healthy maxillary sinus is self-maintaining by postural drainage and actions of the ciliated epithelial lining, which propels bacteria toward the ostium. The ostium is a nonphysiologic drainage port high on the medial wall that opens into the nasal cavity between the middle and lower nasal conchae (hiatus semilunaris).

At its highest point, the ostium is located 30 mm above the floor and serves as an anatomic rationale for the sinus floor elevation procedure. Sinus elevation may improve the symptoms of sinusitis and congestion by bringing the floor of sinus closer to the drainage port, and the grafting procedure does not interfere with normal sinus function. (12)

The blood supply to the maxillary sinus is derived primarily from the maxillary artery. The posterior superior alveolar and infraorbital arteries anastomose in the bony lateral wall. (13) On average, the intraosseous anastomose (IA) occurs in 100% of the cases and 19 mm from the alveolar bone crest, whereas the extraosseous anastomose (EA) occurs in about 40% of the cases and 23 mm from the alveolar bone crest. There may be one or more septa dividing the maxillary sinuses, called Underwood's septa. (14,15,16) It occurs about 30% of the time and in the anterior region (most commonly between the second premolar and first molar) about 77% of the time. The mean height of the septae is 7.9 mm (range 0 to 17 mm). (14,15,16)

As mentioned above, the maxillary sinus provides a challenge in implant dentistry because of the reduced bone volume, which is due to alveolar bone resorption and pneumatization of the sinus cavity. Several ways to avoid the sinus cavity is to use a short implant, tilt the implant mesially or distally, use a long zygomatic implant, and/or shorten the dental arch with premolar occlusion. With the premolar occlusion, 50% to 80% of chewing capacity is maintained. (17) However, over the years, techniques have been developed to augment the sinus floor.

Philip Boyne is the first to report the elevation of the maxillary sinus floor for preprosthetic reasons. The maxillary sinus was augmented prior to a tuberosity reduction to increase the interarch distance and create a more symmetric maxillary arch for denture prosthesis. (18) Boyne also reported on the two-stage elevation of the maxillary sinus floor as a preparation for the placement of blade implants. He grafted the maxillary sinus with autogenous particulate iliac bone, and then placed blade implants three months later. (19)

Hilt Tatum suggested the crestal approach for sinus floor elevation with subsequent implant placement. A “socket former” was used to prepare the implant site and create green-stick fracture of the sinus floor. A root-formed implant was placed and allowed to heal in a submerged way. (20)

Robert Summers described another crestal approach — BAOSFE (Bone-Added Osteotome Sinus Floor Elevation) — using tapered osteotomes with increasing diameters. Adjacent bone was compressed by pushing and tapping as the sinus membrane was elevated. Autogeneous, allogenic, or xenogenic bone grafts were added to increase the volume below the elevated sinus membrane. Using this approach, Summers reported a 96% success rate at 18 months after loading 173 press-fit submerged implants. (21)

There are two major approaches to elevate the sinus floor: lateral window and transalveolar approach. The lateral window approach can be one- or two-stage techniques for implant placement; the transalveolar approach is a one-stage technique, mainly based on available residual bone and the possibility of achieving the primary stability of the implant.

Risk Factors

People who are exposed to mustard gas, isopropyl oils, volatile hydrocarbons, or metals like nickel and chromium (which occurs most commonly in the leather tanning, nickel mining and carpentry industries) have an increased risk of developing paranasal cancer including maxillary sinus cancer.

Furthermore, past exposure to an extremely long-acting radioactive chemical called Thorotrast that was used for radiographic studies in the 1960s is thought to increase the risk of maxillary sinus cancer. Chronic sinusitis may also increase the risk of developing the disease.

In addition, tobacco use increases the risk of developing the most common form of maxillary sinus cancer (squamous cell carcinoma) although tobacco's role in other types of paranasal and maxillary sinus cancer is less clear.

Discussion

Oral SCC is a disease entity with well established risk factors, including smoking and alcohol use. It can present on the tongue (most common), floor of the mouth, buccal mucosa, or gingiva and commonly presents as a nonhealing, exophytic or endophytic ulcer with associated local and regional pain. Otalgia, dysphagia, mobile teeth, and weight loss might also be present.

Oral SCC is typically associated with the mandible or the maxilla but will slowly invade the underlying tissues after onset. Invasive maxillary SCC will exhibit a multitude of clinical signs and symptoms, which might mimic facial pain syndromes, including . In the early stages of the disease, the patient will first complain of localized maxillary pain; later, symptoms might progress to mobility of teeth. This was noted in the discussed case. As the tumour invades the maxilla and infiltrates the maxillary sinus, the patient will experience nasal congestion due to direct-obstruction symptoms. The infraorbital nerve (branch of the maxillary division of the trigeminal nerve) will also be affected, resulting in sensory disturbances of the cheek.

Maxillary sinus carcinoma presents a therapeutic challenge to both the surgeon and the radiation oncologist. Because symptoms are vague and nonspecific, the majority of carcinomas are diagnosed as locally advanced disease [5]. Extension to contiguous structures including the orbit, ethmoid sinus, sphenoid sinus, nasal cavity, nasopharynx, pterygoid fossa, palate and cheek may occur and can be a potential problem in the surgical and/or radiotherapeutic management of this disease [5, 6]

Maxillary sinus malignancies are very difficult tumors to treat and traditionally have been associated with a poor prognosis. One reason for these poor outcomes is the close anatomic proximity of the nasal cavity and paranasal sinuses to vital structures such as the skull base, brain, orbit, and carotid artery.[7] This complex location makes complete surgical resection of sinonasal tumors a challenging and sometimes impossible task. In addition, mucoepidermoidcarcinoma of maxillary sinus tend to be asymptomatic at early stages, appearing more frequently at late stages once extensive local invasion has occurred.

The unfortunate combination of complex surrounding anatomy with late, advanced stage presentation leads to the frequent local recurrence and subsequent poor outcome associated with sinonasal malignancies. Overall 5-year survival for maxillary sinus MEC is 36%.[8] The early diagnosis is critical for better prognosis of this tumor. So also it suggests that MEC of maxillary sinus should be considered in the differential diagnosis of swellings in maxilla. The prognosis of maxillary sinus cancer is disappointing, despite aggressive treatments. For successful treatment outcomes, it is necessary to acquire complete surgical resection and to secure adequate resection margins.

However, maxillary sinus cancers usually are diagnosed at advanced stages, and the proximity of important organs such as the eyes and cranial nerves makes complete surgical resection difficult. In addition, functional impairments after surgical resection are the major cause of a decreased quality of life. Therefore, surgical resection with a curative intent should be considered as a primary treatment only in the early stages of the disease. In the advanced stages, multimodality treatment strategies should be arranged for prolongation of survival and improvement in the quality of life.

Maxillary sinuses can vary in size and shape from one individual to another, or even between the right and left sides in the same individual. In approximately half of the population, their length also varies. The floor of the maxillary sinus extends into the alveolar process between the roots of adjacent teeth, creating elevations and depressions called "extensions," with narrow cortical areas.(22) Through histological sections, it has been radiographically demonstrated that most of the roots projecting into the maxillary sinus were in fact surrounded by a thin layer of cortical bone, with perforations present in 14-28% of cases. (23) Under normal conditions, the relationship between the tooth and the floor of the maxillary sinus consists of either a thin layer of compact bone that provides support to the apical periodontal ligament fibers, to which it firmly adheres, or there is a direct relationship with the maxillary sinus mucosa. The inner lining of the maxillary sinus cavity is devoid of periosteum; therefore, in the absence of a thin layer of intervening bone, the periodontal tissues are in direct contact with the basal surface of the sinus mucosa. (24)

The roots of the upper premolars, molars, and occasionally the canine teeth have a close relationship with the maxillary sinus; in some cases, they may even protrude into it.(24,25,26). It has been demonstrated that the closer the tooth apex is to the floor of the maxillary sinus, the greater the impact on antral tissue. (27) This relationship can result in a variety of risks, especially for certain surgical procedures, such as tooth extraction and implant placement, or during endodontic or orthodontic treatments.(28) An accurate description of the relationship between the apices of the upper teeth and the lower wall of the maxillary sinus, as well as the thickness of the cortical bone between these structures, is essential for planning dental procedures.

Dental radiographs, such as panoramic radiography, consist of two-dimensional images and, as such, are inappropriate and/or of little use for accurate morphometric assessment of bone relationships. In cases where the panoramic radiograph reveals a possible relationship between a tooth that has undergone intervention and its contact with the adjacent maxillary sinus, evaluation by cone beam computed tomography (CBCT) can assist in dental treatment planning. This imaging modality allows for a thorough analysis of the anatomical relationship between the maxillary sinus and the roots of the upper teeth, thus overcoming the limitations of panoramic radiography, providing multiplanar views without magnification, distortion, or superimposition. 11 This technique is superior to multi-slice computed tomography due to its higher image resolution, reduced radiation exposure, and lower equipment cost. (29)

In apical periodontitis, a periodontal disease, treatment with implants and extraction of upper premolars and molars may increase the risk of pathological processes in the adjacent maxillary sinus. Of the odontogenic sinus diseases, apical periodontitis and periodontal disease account for 83% of all cases having dental origin. (30) The most frequent maxillary sinus pathologies are sinus mucosal thickening and mucous retention cysts, with a prevalence ranging from 8% to 29% and 2% to 36%, respectively. Another study has reported a prevalence rate of odontogenic maxillary sinusitis ranging from 10% to 86%.

Treatment

One of four different treatment modalities, including intra-arterial (IA)-ICT, intravenous (IV)-ICT, CCRT, and surgical resection, was selected as a primary treatment based on the TNM stage,performance status, age, and co-morbidity. ICT was administered through the IA or IV route. Superselective IA infusion of chemotherapeutic drugs was attempted via a series of processes. The contrast-enhanced tumor mass and tumor feeding vessels were confirmed via diagnostic angiographic procedures of the internal and external carotid arteries by means of transfemoral access. The internal maxillary artery was superselected with a microcatheter, and then the chemother-apeutic drug was administered via a microcatheter into the tumor-supplying artery.

The transfemoral catheter was removed on completion of the infusion. Cisplatin (100 mg/m^2) was administered via a microcatheter into the internal maxillary artery over two hours on day 1, and then 5-FU (1000 mg/m^2/day) was continuously infused from day 1 to day 5 over 120 hours through the IV route. A standard hydration and mannitol diuresis regimen were applied. The entire procedure was repeated 2–3 times every 3–4 weeks.

The IV-ICT was performed 2–3 times every four weeks as well. Cisplatin (100 mg/m^2) was administered intravenously over two hours on day 1, and 5-FU (1000 mg/m^2/day) was infused continuously from day 1 to day 5 over 120 hours through the IV route. All patients who received ICT were re-evaluated for tumor response with CT and/or MRI at least 4–6 weeks after the completion of ICT. The decision to perform surgery after ICT was based on the tumor response.

The chemotherapeutic agent used in the CCRT group was cisplatin. During radiation therapy, cisplatin (30 mg/m^2) was administered by a weekly schedule on days 1, 8, 15, 22, 29, 36, 43, and 50, or cisplatin (100 mg/m^2) was administered every 3 weeks on days 1, 22, and 43. All patients treated with CCRT were reevaluated for tumor response, and then the next treatment modality, surgical resection or salvage chemotherapy, was determined. The periodic follow-up was done at least 6–8 weeks after the completion of radiation therapy.

CCRT

CCRT is regarded as the more effective treatment because of the radiosensitizing efficacy of cisplatin compared with radiation therapy alone. Several studies have demonstrated that CCRT has a higher tumor control rate and survival rate in head and neck cancers than radiation therapy alone [9] Harrison *et al.* reported a three-year local control rate of 78% and a three-year survival rate of 42% in 12 patients with paranasal sinus carcinomas treated with cisplatin-based CCRT [10]In this study, seven patients with stage IV maxillary sinus cancer received CCRT as a primary treatment, and the objective response rate was 57%. However, the assessments of recurrence and survival rate were impossible because of small sample size and loss to follow-up.

IA Chemotherapy

The femoral artery was punctured under local anesthesia by Seldinger's method. The tip of the catheter was threaded into the external carotid artery. Fluorography was performed through amicro-catheter to determine the dominant tumor-supplying vessel with digital angiography. Cone-beam computed tomography was also used during angiography to ascertain the stained area. In cases which had multiple tumor-supplying vessels, the dose of CDDP and DOC was divided according to the stained tumor volume**.**

Conclusion

Invasive SCC can be difficult to diagnose in its early stages and might be misdiagnosed as facial pain syndromes (such as TN), which often present as a unilateral shooting pain and commonly affect branches of the trigeminal nerve. Treatment of TN is initially effective in up to 90% of patients but will dampen neural impulses,[11] disguising any alternate pathology. A careful history and extraoral and intraoral examination as well as appropriate investigations, such as a CT scan, should be undertaken before definitively diagnosing TN. If in doubt, early specialist referral to exclude an underlying malignancy is absolutely necessary.

Corresponding Author

Aravind Sivakumar,

Senior Lecturer,Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077,Tamil Nadu, India.

Email: aravind.sivakumar@gmail.com

Telephone Number: +91 8220552400

Reference

1. .Dulguerov P, Jacobsen MS, Allal AS, Lehmann W, Calcaterra T. Nasal and paranasal sinus carcinoma: Are we making progress? A series of 220 patients and a systematic review. Cancer. 2001;92:3012–29.
2. Osguthorpe JD, Richardson M. Frontal sinus malignancies. OtolaryngolClin North Am. 2001;34:269–81
3. Bhattacharyya N. Survival and staging characteristics for nonsquamous cell malignancies of the maxillary sinus. Arch Otolaryngol Head Neck Surg. 2003;129:334–7.
4. Carrau RL, Myers EN, Johnson JT. Paranasal sinus carcinoma-diagnosis, treatment, and prognosis. Oncology. 1992;6:43–50.
5. Paulino AC, Marks JE, Bricker P, Melian E, Reddy SP, Emami B. Results of treatment of patients with maxillary sinus carcinoma. Cancer 1998;83(3):457-465.
6. Manrique RD, Deive LG, Uehara MA, Manrique RK, Rodriguez JL, Santidrian C. [Maxillary sinus cancer review in 23 patients treated with postoperative radiotherapy]. ActaOtorrinolaringolEsp 2008;59(1):6-10.
7. Bhattacharyya N. Survival and staging characteristics for non-squamous cell malignancies of the maxillary sinus. Arch Otolaryngol Head Neck Surg. 2003;129:334–7.

8. da Cruz Perez DE, Pires FR, Lopes MA, de Almeida OP, Kowalski LP. Adenoid cystic carcinoma and mucoepidermoid carcinoma of the maxillary sinus: Report of a 44-year experience of 25 cases from a single institution. J Oral MaxillofacSurg. 2006;64:1592–7.
9. Monnerat C, Faivre S, Temam S, et al. End points for new agents in induction chemotherapy for locally advanced head and neck cancers. Ann Oncol. 2002;13:995–1006.
10. Waldron JN, O'Sullivan B, Gullane P, et al. Carcinoma of the maxillary antrum: a retrospective analysis of 110 cases. RadiotherOncol. 2000;57:167–73.
11. Chole R, Patil R, Degwekar SS, Bhowate RR. Drug treatment of trigeminal neuralgia: a systematic review of the literature. J Oral MaxillofacSurg. 2007;65(1):40–5.
12. Lindhe J, Lang N, Karring T, Elevation of the maxillary sinus floor. Clinical Periodontology and Implant Dentistry, Fifth Edition 2008;2:250.
13. Solar P, Geyerhofer U, Traxler H, Windisch A, Ulm C, Watzek G. (1999). Blood supply to the maxillary sinus relevant to sinus floor elevation procedures. Clinical Oral Implants Research 1999; 10(1), 34-44.
14. Underwood AS. An inquiry into the anatomy and pathology of the maxillary sinus. Journal of Anatomy and Physiology 1910; 44:354-369.
15. Ulm CW, Solar P, Gsellmann B, Matejka M, Watzek G. The edentulous maxillary alveolar process in the region of the maxillary sinus — a study of physical dimension. Journal of Oral and Maxillofacial Surgery 1995; 24:279-282.
16. Kim MJ, Jung UW, Kim CS, Kim KD, Choi SH, Kim CK, Cho KS. Maxillary sinus septa: prevalence, height, location, and morphology. A reformatted computed tomography scan analysis. Journal of Periodontology 2006; 77(5):903-908.
17. Kayser AF. Shortened dental arches and oral function. Journal of Oral Rehabilitation 1981; 8(5):457-462.
18. Boyne PJ. Restoration of osseous defects maxillofacial causalities. Journal of American Dental Association 1969; 78:767-776.
19. Boyne PJ, James R. Grafting of the maxillary sinus floor with autogeneous marrow and bone. Journal of Oral Surgery 1980; 38:613-618.
20. Tatum H. Maxillary and sinus implant reconstructions. Dental Clinics of North America 1986; 30(2):207-229.

21. Summers RB. (1994). A new concept in maxillary implant surgery: the osteotome technique. The Compendium of Continuing Education in Dentistry 1994; 15(2):152-162.
22. Waite DE. Maxillary sinus. Dent Clin North Am. 1971;15:349-68.
23. McGrowan DA, Baxter PW, James J. The maxillary sinus and its dental implications. 1st ed. London: Wright; 1993. p. 1-25.
24. Wehrbein H, Diedrich P. The initial morphological state in the basally pneumatized maxillary sinus - a radiologicalhistological study in man. FortschrKieferorthop. 1992;53: 254-62.
25. Eberhardt JA, Torabinejad M, Christiansen EL. A computed tomographic study of the distances between the maxillary sinus floor and the apices of the maxillary posterior teeth. Oral Surg Oral Med Oral Pathol. 1992;73:345-6.
26. Sharan A, Madjar D, Hashomer T. Correlation between maxillary sinus floor topography and related root position of posterior teeth using panoramic and cross-sectional computed tomography imaging. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2006;102:375-81.
27. Kilic C, Kamburoglu K, Pehlivan S, Ozen T. An assessment of the relationship between the maxillary sinus floor and the maxillary posterior teeth root tips using dental cone-beam computerized tomography. Eur J Dent. 2010;4:462-7.
28. Selden H. Endo-Antral syndrome and various endodontic complications. J Endod. 1999;25:389-93.
29. Hauman CH, Chandler NP, Tong DC. Endodontic implications of the maxillary sinus: a review. Int Endod J. 2002;35:127-41.
30. Howe R. First molar radicular bone near the maxillary sinus: a comparison of CBCT analysis and gross anatomic dissection for small bony measurement. Oral Surg Oral Med Oral Pathol Oral RadiolEndod. 2009;108:264-9.

Maxillary Sinus

Priyadharshni S[1], Saravana Kumar[2], E. Roja[3],
Graduate Student[1], Senior Lecturer[2], Department of Anatomy,Tutor[3], Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

The pyramid-shaped maxillary sinus (or Antrum of Highmore) is the largest of the paranasal sinuses and drains into the middle meatus of the nose. The image of the body of the zygomatic bone and the temporal process of the zygomatic bone may be superimposed on the sinus on a radiograph - less with paralleling technique. Radiographically just posterior to the posterior wall of the sinus one may see a teardrop radiolucency – the pterygomaxillary fissure. In adults with a full set of teeth, the maxillary sinus floor is the strongest of the bone walls surrounding the cavity. The anatomical relation between the maxillary sinus and the teeth is a complex one, due to the variable extension of the sinus. Antral mucosa is thinner and less vascular than nasal mucosa. The blood supply to the maxillary sinus is principally derived from the posterior superior alveolar artery and infra orbital artery, both being branches of the maxillary artery. There are significantly anastomoses between these two arteries in the lateral antral wall. The greater palatine artery also supplies the inferior portion of the sinus. However, because the blood supplies to the blood supplies to the maxillary sinus are from terminal branches of peripheral vessels, significant haemorrhage during the sinus lift procedure is rare. Nerve supply to the sinus is derived from the superior alveolar branch of the maxillary division of the Trigeminal nerve. The size of the sinus will increase with age if the area is edentulous. The extent of pneumatization varies from person to person from side to side. Nonetheless, this process often leaves the bony lateral and occlusal alveolus paper thin in the posterior maxilla. The maxillary sinus bony cavity is lined with the sinus membrane, also known as the schneiderian membrane. This membrane consists of ciliates epithelium like the rest of nasal epithelium through the ostium in the middle meatus. The membrane has a thickness of approximately 0.8mm.

***Key Words**: Sinus, bony septa, pathologic conditions, sinus lift, sinus membrane*.

Introduction

The maxillary sinus is the pneumatic space that is lodged inside the body of maxilla and communicates with the environment with the environment by the way of the middle nasal meatus. It is also known as the "Antrum of Highmore" The maxillary sinus is formed by pneumatization of the maxilla and is expanded to the body of the zygoma laterally and to the palatine bone posteriorly, to a one-side volume of 15 ml, though this varies widely from individual to individual. Maxillary sinus is the first of the PNS to develop. Early diagnosis is essential in order to avoid high morbidity and mortality associated with the destructive disease and to instigate treatment before irreversible condition arise. The inflammatory lesions that affect the paranasal sinuses receive the generic denomination sinusitis; maxillary sinus is the most commonly affected. This inflammation can have various origins, including the tooth. The maxillary sinus belonging to the nasal and oral cavity is the most susceptible of the all sinus to the invasion by pathogenic bacteria, either through their communication with the nasal cavity, or the product of odontogenic infection.

Anatomy of Maxillary Sinus

The maxillary sinus begins to develop between second and third month of pregnancy, with an evagination of nasal passage lateral wall mucosa. The maxillary sinus is a pyramid shaped cavity with its base adjacent to the nasal wall and apex pointing to the zygoma. The size of the sinus is insignificant until the eruption of permanent dentition. The average dimensions of the adult sinus are 2.5 to 3.5 cm wide, 3.6 to 4.5 cm tall, and 3.8 to 4.5 cm deep. It hasan estimated volume of approximately 12 to 15cm. Anteriorly, it extends to the canine and premolar area. The sinus floor usually has its most inferior point near the first molar region. The size of the sinus will increase with age if the area is edentulous. The extent of pneumatisation varies from person to person and from side to side(1). The close anatomical relationship of the maxillary sinus with the roots of maxillary molars and premolars renders this anatomical region susceptible to morbid situations resulting from damage to, and therapeutic intervention in the dentoalveolar environment.The loss of the teeth interrupts the cybernetic connections of the stomatognatic system, determining maxillary and mandibular structural changes that represent signs of inactivity atrophy due to functional unload. Modifications in the lateral area of the edentulous maxilla may affect

more than 70% of its height and width(2). The sinus may 'invade' the alveolar bone surrounding the roots of the posterior maxillary teeth, where it may pose a surgical hazard when extracting teeth in this area. The formation of septa (ie,Underwood's septa), both complete and incomplete, within the sinus is often noted(3).

Bony Septa

Maxillary sinus septa were first described by Underwood in 1910. They are walls of cortical bone within the maxillary sinus, their shape has been described as an inverted gothic arch arising from the inferior or lateral walls of the sinus, and may even divide the sinus into two or more cavities. Radiographic identification of these structures is important, since the design of the lateral window during sinus lift procedures is based on the presence and size of maxillary sinus septa(4). Inside the maxillary sinus, bony septa originating in the sinus floor are often found. They are called Underwood septa, they may divide the back part of the sinus into multiple compartments known as posterior recesses. They may even occasionally reach from the base to the upper sinus wall, creating two sinuses. Estimates of the prevalence of such septa have ranged from 16 to 58%.The formation of Underwood septa may be linked to the fact that teeth are lost at different times. The edentulous areas may resorb in a manner that results in a difference in level between the two adjacent portions of the sinus floor(5). Between 13 and 35.3% of maxillary sinuses have septa; to avoid complications during sinus lifting a meticulous study of the sinus is necessary, preferably by computed tomography, as panoramic radiography has been to shown to have a low sensibility and specificity in the identification of these structures.

According to Underwood(6), the maxillary sinus floor is frequently divided into three basins: a small anterior one over the premolar region; a large median one descending between the roots of the first and second molars, and a small posterior one corresponding to the third molar region. These three sections of the floor of the sinus, which are often marked off by ridges, rising sometimes to distinct septa, correspond to three de ned periods of tooth activity, separated by intervals of time: the anterior portion corresponds to the position of the eruption of the milk molars (between 8 months and 2 years); the middle portion corresponds to the eruption of the first and second permanent molars (from 5 to 12 years); and the posterior, corresponds to the eruption of the third molars (16 to 30 years). The origin of the septa is wholly dental, and is due to the persistence of the intervening partitions when the rest of the

bony floor sinks down between the dental roots during tooth eruption, leading to the location of septa between the roots of two adjacent teeth. Underwood observed the existence of another type of septa, indicating that it must have a different origin as it seemed to be unrelated with teeth.

Krennmair et al. (7) classified septa into primary and secondary: primary septa corresponding to those first described by Underwood, arising from the development of the maxilla; and secondary septa arising from irregular pneumatization of the sinus floor following tooth loss. Other authors (8,9,10) when observing maxillary sinus septa, classified them as primary septa if they were located superior to a maxillary tooth; and other septa, for septa located superior to an edentulous ridge, as they could be either primary or secondary septa, or a combination of both types.

Maxillary Sinus Membrane

The maxillary sinus is lined with a respiratory mucosa similar to that of the nose and other paranasal sinuses. It consists of a mucoperiosteum with 3layers: an epithelial covering, a lamina propria, and periosteum.The maxillary sinus membrane varies in thickness, but is generally 0.3 to 0.8 mm. The epithelial lining is a single layer of pseudo stratified columnar ciliated epithelium with few blood vessels and is thicker than that of the other paranasal sinuses. Numerous goblet cells are present within the mucosa and are the major source of mucous secretion. The highest density of these cells is located near the sinus ostium. The density of ciliated cells is high, ranging from 91.3% to 97.7%, except near the ostium, where the density is decreased by half(11). The lamina propria consists of a thin layer of connective tissue with few elastic fibres. It is thickest at the medial wall and contains more seromucous glands than the lateral wall. The periosteum is tightly bound to the overlying lamina propria, but is easily detachable from the underlying bone, making membrane separation during the sinus lift procedure relatively easy.Any factors that compromise mucous production, ciliary function, or patency of the ostium can increase the risk of sinusitis. As a result, with sinus elevation augmentation surgery, it is important to maintain membrane integrity to allow for confinement of the particulate graft and to decrease the risk of infection(12). The maxillary sinus bony cavity is lined with the sinus membrane, also known as theSchneiderian membrane. This membrane consists of ciliated epithelium like the rest of the respiratory tract. It is continuous with, and connects to, the nasal epithelium through the ostium in the middle meatus. The sinus floor tends to lower craniocaudally as the alveolar ridge is resorbed in the opposed direction. It is the

lack of vertical posterior maxillary bone that often necessitates the use of bone grafts or sinus-lifting procedures prior to implant rehabilitation. Progressive resorption of the posterior maxillary edentulous ridge follows a well-defined path that differs from that of the anterior regions and includes repeatable, predictable morphologic changes. Cawood and Howell's system for classifying the degrees of atrophy based on the morphologic differences in the residual ridge is extremely useful for presurgical diagnostic assessment, as the ridge appearance is connected to the horizontal and vertical size of bone available for implants.

Fungal Infections

A variety of different causative organisms are responsible for paranasal mycoses, Aspergillus and Mucor being the commonest. Paranasal mycoses manifests as two distinct entities, a benign or non invasive infection and the more serious invasive infection, which occurs in immunocompromised individuals(13). Early diagnosis is vital in these infections as delay in initiation of treatment can be life threatening due to propensity of the fungi to invade adjacent blood vessels and connective tissues produces thrombosis and ultimately necrosis of the hard and soft tissues. Although the fungi and spores of mucorales shows minimal intrinsic pathogenicity towards normal persons, they can initiate aggressive and fulminating infection in the patients whose resistance is lowered by immunocompression, diabetes, malignant disease, burns, trauma, and rarely steroids(14). The effective management of paranasal sinus fungal infection requires early diagnosis, histological classification and surgery and when appropriate chemotherapy.Treatment in invasive fungal sinusitis involves radical surgery in addition to intravenous amphotericin B/lipid complex of liposomal amphotericin B with or without flucytosine. The optimum dose and duration of treatment is still unclear. Endoscopic surgery has been found to be feasible and efficient, enabling excellent local control and less morbidity than traditional approaches. Sinus surgery must be performed as soon as feasible(15).

Pathologic Conditions of Maxillary Sinus

Diseases of the maxillary sinus are comparatively frequent, even in apparently young individuals with rates in excess of one in five individuals examined using the Waters projection (mucosal thickening 12.3%; cysts or polyps 7.2%; opacified sinus 3.3%)(16). For this reason, it is incumbent upon the dental practitioner to understand the panoramic

radiological features of disease and normal variations within the paranasal sinuses. Certainly the patient should not be referred to an ear, nose and throat specialist for every instance of antral mucosal thickening or mucous retention cyst, nor should the dentist ignore features that possibly reflect an early malignancy. The reputation of a practitioner is greatly enhanced given appropriate referrals that can make the difference between life and death. Failure to diagnose, on the other hand, can result in notoriety. Nortje et al (17) were among the first to comprehensively study the appearance on panoramic dental radiographs of pathological conditions affecting the maxillary sinuses, comparing inflammatory conditions of dental origin, iatrogenic disease/foreign bodies, non-odontogenic inflammatory conditions, cysts, benign neoplasms, malignant neoplasms and dysplasia affecting the maxilla. Primary malignancies affecting the maxillary sinus include squamous-cell carcinoma, adenoid cysticcarcinoma and adenocarcinoma(18). The maxillary sinus may also be affected secondarily by extension malignancies of the oral soft tissues or jaw, and also, although rare, is the site ofmetastases from distant sites(19). Owing to the radio-opacity, roots or whole teeth displaced into the sinus are readily apparent even when not centred within the image layer. These need to be differentiated from sinus bone nodules and antroliths (calcified stones arising in the antral lining) both of which entities could be mistaken for teeth or displaced roots(20). Foreign bodies, such as bullets, are clearly demonstrated; however care needs to be made to differentiate between clearly demarcated real images, and blurred magnified ghost images of foreign bodies or jewellery more distally and lower placed in or on the contralateral side of the face. Oroantral fistulas following dental extraction are only noticeable on panoramic radiography when large and within the panoramic image layer.

The growth of tumours within the maxilla is not concentric; hence, the site of origin is not necessarily the epicentre of the lesion. The maxillary sinus, constituted the path of least resistance for the growth of such maxillary lesions as cysts and benign neoplasms. Even very large benign tumours and cysts might be present without resulting in clinically noticeable jaw expansion. Hence, the panoramic radiograph is of value in detection of unsuspected disease.

Antral malignancies are usually insidious and produce clinical signs and symptoms relatively late, whentheprognosisisoftenquite poor. Panoramic radiographs have been found of utility in detection of antral carcinoma, particularly that affecting the posterior wall of the sinus(21). Caution should be used in that the panoramic radiograph is not the technique of choice for viewing the maxillary sinuses, however, it is incumbent on the dentist to evaluate the portion of the maxillary sinus shown in the panoramic radiograph made for other purposes. This might well be the first sign of disease and the only reason for pursuing further diagnostic

tests. Early detection of such sinister occurrences improves the prognosis for the unfortunate patient.

Maxillary Sinusitis

Sinusitis is a swelling of the inner lining of the sinuses The sinuses are the spaces between the bones in the face where air passes and where a fluid called mucus drains into the nose In sinusitis, the swelling blocks the openings in the sinuses through which mucus drains into the nose When mucus cannot drain properly, the pressure of the blocked fluid inside the sinuses can be painful.

Sinusitis can be acute (going on less than four weeks), sub acute (4–8 weeks) or chronic (going on for 8 weeks or more All three types of sinusitis have similar symptoms, and are thus often difficult to distinguish. Birt stated that otolaryngologists see scores of patients with vague discomfort in the forehead, between the eyes, and across the nose and cheeks.Patients invariably ascribe their symptoms to sinus disease, and are later surprised to discover that they are not infected. In fact, chronic sinusitis is not particularly common, and many headache patientswithautonomicfeatureswill probably have muscle tension headaches or migraines(22). Sinus headache is commonly diagnosed, and patients with headache often cite sinus pain and pressure as a cause of their headaches. A high frequency of diagnosis of sinus headache, which specialists consider to be relatively rare, among patients meeting International Headache Society (IHS) diagnostic criteria for migraine raises the possibility that migraine and perhaps other headache types are sometimes mistaken for sinus headache.

In some instances, chronic maxillary atelectasis can present with enophthalmos secondary to collapse of the maxillary sinus. For reasons that are unclear, the sinus component of the disease remains asymptomatic and is discovered only after thorough evaluation of the enophthalmos. Isolated sphenoid sinusitis is often misdiagnosed because of its rarity and varied clinical presentation. Presentation is often both subtle and suggestive of other intracranial etiologic sites, as determined by the anatomic relationships of the sphenoid sinus(23).

Sinusitis is among the most commonly encountered diseases of childhood and has been the major area of interest for many otolaryngologists, allergists, and paediatricians. Allergies and viral upper respiratory infections are among the most common predisposing factors of sinus disease. Every child with sinusitis is a candidate for an allergy evaluation.

Sinus Lift

The lack of bone volume can be treated with various bone grafting techniques before the implant placement. Boyne & James (1980) was the first to introduce maxillary sinus floor augmentation with autologous bone graft. This technique has been modified and improved by Tatum (1986) who introduced the lateral approach by fenestrating the buccal wall of maxillary sinus and lifting the Schneiderian membrane. This technique was modified by Wood and More in 1988.

The sinus floor augmentation procedure can be divided into two different techniques. The first of the two techniques is called the osteotomy technique and it is performed by the use of osteotomes to create a controlled fracture of the floor of the maxillary sinus. This method creates space by elevating the sinus membrane and provides room for the dental implant and bone grafting material. The advantage of this technique is that it is less invasive and thereby reduced surgical time and lower morbidity compared to other sinus lift techniques. This technique is suggested to be used when the vertical bone height is more than 4-6mm. The second technique is the lateral window technique and is performed by surgical preparing of the bone, lateral to the maxillary sinus, and thereby exposing the Schneiderian membrane which will be elevated. The bone graft material is carefully packed and placed on the sinus floor. [24] This technique is more invasive than the osteotome technique due to the fenestration of the lateral sinus wall. The lateral window technique is preferred when there is less than 6 mm residual bone height.

Direct Sinus Augmentation Technique (DSAT)

Those cases that has residual alveolar bone (RAB) height 5 mm or below was considered for the direct technique. Autogenous bone grafts was harvested by shaving the mandibular bone from external oblique ridge area or chin area. A bone mill was used to grind the bone shaving intofine particles. After adequate local anesthesia and preparation, a surgical incision was placed on the crest of the RAB at most appropriate area, with vertical releasing curvilinear incisions flaring into the vestibule. Full- thickness, subperiosteal labial, and palatal flaps were raised, reflected. Care was taken to keep the base of flap broad as well as adequate buccal and palatal tissue for closure. After elevation, the anterolateral wall of maxillary sinus was visualized. Care was taken to identify and protect infraorbital nerve, if encountered. The dimension of osteotomy was determined based on clinical and radiographic examinations as well as the extent of edentulous span. A buccal bone window was made on exposed wall of maxillary sinus using a postage stamp method. The bony wall was gently manipulated with

sinus membrane elevators without damaging Schneiderian membrane. The previously obtained graft material was then placed and packed. The implant was placed on same sitting with help of a stent which was positioned, then removed, and the site was checked for appropriate faciolingual and mesiodistal positioning. Any obvious abnormal crestal defects required slight modification of the position(25).

Indirect Sinus Augmentation Technique (ISAT)

Indirect sinus augmentation is done for cases with RAB height of 6-8 mm. The RAB to receive the implant was given local anesthesia and perforated using a small rounded drill. A pilot drill was placed in marked implant site to establish the axis of implant recipient site. Following the pilot drill, subsequently increasing diameter of drills were used to enlarge implant recipient site till the desired diameter corresponding to implant diameter was reached. The height of drill was maintained 2 mm short of sinus floor. The indirect sinus lift was done by insertion of correct caliber osteotome and working up through successively greater instrument diameters, until the sinus floor was fractured and elevated up. The sinus floor was carefully fractured, separated from the Schneiderian membrane avoiding damage to membrane using a surgical mallet with controlled force. If required, autogenous graft material was inserted within the socket. The material was displaced apically with help of larger-diameter instruments, thereby lifting the membrane and condensing graft material between the latter and sinus floor. The implant was then placed immediately in the prepared site. 3-0 Vicryl sutures were used to close the surgical wound. Antibiotic coverage, pain killers, and nasal decongestants were prescribed for 5 days. The patients were monitored on a periodic basis both clinically and radiologically(26).

Bone Healing

Bone healing after graft placement takes place in two phases: Repair with an inflammatory response and bone remodelling. In the first phase a blood clot is formed in the injured area where the outer area of the local bone becomes necrotic and the capillaries start to develop and further on migration of inflammatory cells e.g. lymphocytes, granulocytes and monocytes occur. This action restores blood flow and after 1-3 days an inflammatory response is active and granulation tissue is starting to form. The granulation tissue will mature to a collagen matrix and mesenchymal stem cells begin to differentiate into osteoblasts cells forming new bone. During the second phase, the bone remodel, and is replaced by a more mature lamellar

bone and a complete regeneration of a defect occurs when all bone is replaced with lamellar bone.

Conclusion

The maxillary sinus is one of the four paranasal sinuses. The maxillary sinus is the largest of the paranasal sinuses. The two maxillary sinuses are located below the cheeks, above the teeth and on the sides of the nose. The maxillary sinuses are shaped like a pyramid and each contain three cavities which point sideways, inwards, and downwards. Most of the maxillary sinuses have septa; to avoid complications during sinus lifting a meticulous study of the sinus is necessary, preferably by computed tomography, as panoramic radiography has been to shown to have a low sensibility in the identification of these structures.The sinuses are tiny air-filled holes found in the bones of the face which reduce skull weight, produce mucus, and affect the tone quality of a person's voice. The maxillary sinus drains into the nose through a hole called the ostia. When the ostia becomes clogged sinusitis can occur. The ostia of the maxillary sinus often clog because the ostia are located near the top of the maxillary sinus causing drainage to be difficult. Maxillary sinusitis or an infection of the maxillary sinus can have the following symptoms: Fever Pain or pressure in face near the cheek bones ,Toothache, Runny nose ,Sinusitis are the most common of maxillary sinus illnesses and is usually treated with prescription antibiotics.

Corresponding Author

E. Roja,

Tutor,Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai – 600 077,Tamil Nadu, India.

Email Id: itsrojaa@gmail.com

Contact Number: +91 99941 74480.

References

1. Alder ME,The maxillary sinus floor in theoral implantology. Romanian Journal of Morphology and Embryology 2008, 49(4):485–489.

2. Lang J. Clinical anatomy of the nose, nasal cavity and paranasal sinuses. New York: Thieme;1989. p.100
3. Kandpal h, aneesh M K, Seith A, Sharma S. symptomatic perineural extension of fungal sinusitis in an immunocompetent person: imaging features. Singapore Med J. 2008; 49(7): e 171
4. Robb PJ. Aspergillosis ofthe paranasal sinuses: a case report and historical prospective. J Laryngol Otol 1986; 100: 1071-7
5. Kim M. J., Jung U. W., Kim C. S., Kim K. D., Choi S. H., Kim C. K., Cho K. S., Maxillary sinus septa: prevalence, height, location, and morphology. A reformatted computed tomography scan analysis, J Periodontol, 2006,
6. Underwood AS. An Inquiry into the Anatomy and Pathology of the Maxillary Sinus. J Anat Physiol. 1910;44:354-69.
7. Krennmair G, Ulm CW, Lugmayr H, Solar P. The incidence, location, and height of maxillary sinus septa in the edentulous and dentate maxilla. J Oral Maxillofac Surg. 1999;57:667-71.
8. Velásquez-Plata D, Hovey LR, Peach CC, Alder ME. Maxillary sinus septa: a 3-dimensional computerised tomographic scan analysis. Int J Oral Maxillofac Implants. 2002;17:854-60.
9. Kim MJ, Jung UW, Kim CS, Kim KD, Choi SH, Kim CK. Maxillary sinus septa: prevalence, height, location, and morphology. A reformatted computed tomography scan analysis. J Periodontol. 2006;77:903-8
10. González-Santana H, Peñarrocha-Diago M, Guarinos-Carbó J, Sorní-Bröker M. A study of the septa in the maxillary sinuses and the subantral alveolar processes in 30 patients. J Oral Implantol. 2007;33:340-3.
11. Daley CL, Sande M. The runny nose. Infection of the paranasal sinuses. Infect Dis Clin.North Am, 1988;2(1):131–47.
12. Underwood AS. An inquiry into the anatomy and pathology of the maxillary sinus. J Anatomical Physiol, 1910;44:354-369.
13. Wallace SS, Froum SJ. Effect of maxillary sinus augmentation on the survival ofendosseous dental implants. A systematic review. Ann Periodontol. 2003
14. Tatum H Jr. Maxillary and sinus implant reconstructions. Dent Clin North Am. 1986;30:207-229.
15. MischCE. Maxillary sinus augmentation for endostealimplants: Organised alternative treatment plans. Int J Oral Implantol.1987;4:49-58.

16. SavolainenS,EskelinM,Jousimies-Somer H, Ylikoski J. Radiological findings in the maxillary sinuses of symptomless young men. Acta Otolaryngol Suppl 1997;529:153-157.
17. NortjÈ CJ, Farman AG, Joubert JJ de V. Pathological conditions involving the maxillary sinus: their appearances on panoramic Dental radiographs.Brit J Ora Surg 1979;17:27-32.
18. Kim GE, Chung EJ, Lim JJ, Keum KC, Lee SW, Cho JH, Lee CG, Choi EC. Clinical significance of neck node metastasis in squamous cell carcinoma of the maxillary antrum. Am J Otolaryngol 1999;20:383-390.
19. KoscielnyS.Theparanasalsinusesas metastatic site of renal cell carcinoma. Larungorhinootologie 1999;78:441-444.
20. D.Shiva kumar et al. BioMedRx: An International Journal 2013;1(8):798-802.
21. Ramani, Cholesterol granuloma of maxillary sinus, International journal of oral and maxillofacial surgery. 2006; 35(11):1063–1065.
22. Dinesh ram raja, Antrum of highmore - its implications in dentistry, International Journal of Pharma and Bio Sciences ISSN,2008;54:5975 – 6299.
23. Sankaranarayanan S, chandrasekar T, srinivasa Rao P, Rooban T, RanganathanK.Maxillary Non-non-Hodgkinslymphoma.J Oral maxillofacpathol, 2005;9:34-6.
24. Tina Banihashem, Florida Zulfijaj, Maxillary floor augmentation with different bone grafting materials for dental implant placement, a systematic review, Academia RegiaOdontologica, 2013.
25. De Vicente JC, Hernandez-Vallejo G, Brana-Abascal P, Pena I., Maxillary sinus augmentation with autologous bone harvested from the lateral maxillary wall combined with bovine-derived hydroxyapatite; clinical and histologic observations, Clin Oral Implants Res., 2012;21(4): 22-55.
26. S.M. Balaji, Direct v/s Indirect sinus lift in maxillary dental implants, Annals of Maxillofacial Surgery, 2013; 3(2):148-153.

Mechanisms of Apoptosis

Benazir HussainM[1], Sivakumar M[2]

Graduate Student[1], Senior Lecturer[2], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

Abstract

Programmed cell death plays critical roles in a wide variety of physiological processes during fetal development and in adult tissues. In most cases, physiological cell death occurs by apoptosis as opposed to necrosis. Defects in apoptotic cell death regulation contribute to many diseases, including disorders where cell accumulation occurs (cancer, restenosis) or where cell loss ensues (stroke, heart failure, neurodegeneration, AIDS). In recent years, the molecular machinery responsible for apoptosis has been elucidated, revealing a family of intracellular proteases, the caspases, which are responsible directly or indirectly for the morphological and biochemical changes that characterize the phenomenon of apoptosis. Diverse regulators of the caspases have also been discovered, including activators and inhibitors of these cell death proteases. Inputs from signal transduction pathways into the core of the cell death machinery have also been identified, demonstrating ways of linking environmental stimuli to cell death responses or cell survival maintenance. Knowledge of the molecular mechanisms of apoptosis is providing insights into the causes of multiple pathologies where aberrant cell death regulation occurs and is beginning to provide new approaches to the treatment of human diseases.

Key Words*: apoptosis, caspases, death domain, death effector, card protein*

Introduction

Apoptosis is a morphological phenomenon. As viewed with the assistance of the light (or, preferably, the electron) microscope, the characteristics of the apoptotic cell include chromatin condensation and nuclear fragmentation (pyknosis), plasma membrane blebbing, and cell shrinkage. Eventually, the cells breaks into small membrane-surrounded fragments (apoptotic bodies), which are cleared by phagocytosis without inciting an inflammatory response. The release of apoptotic bodies is what inspired the term “apoptosis” from the Greek, meaning “to fall away from” and conjuring notions of the falling of leaves in the autumn from deciduous trees.[1]

Apoptosis is caused by Caspases

Activation of a family of intracellular cysteine proteases which cleave their substrates at aspartic acid residues, known as caspases for *C*ysteine *Asp*artyl-specific Prote*ases*. These proteases are present as inactive zymogens in essentially all animal cells, but can be triggered to assume active states, generally involving their proteolytic processing at conserved aspartic acid (Asp) residues. During activation, the zymogen pro-proteins are cleaved to generate the large (~20 kd) and small (~10 kd) subunits of the active enzymes, typically liberating an N-terminal prodomain from the processed polypeptide chain. The active enzymes consist of heterotetramers composed of two large and two small subunits, with two active sites per molecule.[2] Analysis of the structures of the active sites of these enzymes, experiments with combinatorial peptide libraries, and other data suggest that caspases recognize the Asp residues they cleave within the context of tetrapeptide motifs, where the most proximal (N-terminal) residue recognized is designated P4 (position #4) and target Asp is P1 (position #1), and where cleavage occurs at the peptidyl bond distal (C-terminal) to the targeted Asp. This information about the structures and mechanisms of caspases has been exploited for developing small-molecule inhibitors, which are finding their way into clinical trials for stroke, liver failure, inflammatory diseases, and a wide variety of other ailments.[3]

The observation that caspases cleave their substrates at Asp residues and are also activated by proteolytic processing at Asp residues makes evident that these proteases collaborate in proteolytic cascades, whereby caspases activate themselves and each other. In humans and mice, approximately 14 caspases have been identified. They can be subgrouped according to either their amino acid sequence similarities or their protease specificities. From a functional perceptive, it is useful to view the caspases as either upstream (initiator) caspases or

downstream (effector) caspases.[4] The proforms of upstream initiator caspases possess large N-terminal pro-domains, which function as protein interaction modules, allowing them to interact with various proteins that trigger caspase activation. In contrast, the proforms of downstream effector caspases contain only short N-terminal prodomains, serving no apparent function. Downstream caspases are largely dependent on upstream caspases for their proteolytic processing and activation. Accordingly, the sequence of the cleavage sites separating the large and small subunits of the zymogen forms of the effector caspases generally match the preferred tetrapeptide specificities of the upstream initiator caspases. Similarly, examination of the cleavage sites of multiple cellular proteins, which have been identified as caspase substrates and which are known to undergo processing during apoptosis, reveals (in most instances) coincidence with the preferred tetrapeptide sequences cleaved by the effector caspases.[5] These substrates of effector caspases include protein kinases (often separating the autorepressing regulatory domains from catalytic domains) and other signal transduction proteins, cytoskeletal and nuclear matrix proteins, chromatin-modifying (eg, polyADPribosyl polymerase) and DNA repair proteins, and inhibitory subunits of endonucleases (CIDE family proteins).

Caspase Activation Mechanisms

A diversity of mechanisms exists for activating initiator caspases, thus setting the wheels of the apoptotic machinery in motion. However, fundamentally, the biochemical mechanisms appear to be remarkably similar and can be explained by a single model, known as the induced proximity model.[6] The induced proximity model is predicated on the empirical observation that the zymogen forms of unprocessed caspases are not entirely inactive but rather possess weak protease activity (measured in some cases at ~1% of the fully active enzymes). When brought into close apposition through protein interactions, the zymogens can *trans*-process each other, producing the fully active proteases.

Though many pathways for activating caspases may exist, only two have been elucidated in detail. One of these centers on tumor necrosis factor (TNF) family receptors, which use caspase activation as a signaling mechanism, thus connecting ligand binding at the cell surface to apoptosis induction.[7] The other involves the participation of mitochondria, which release caspase-activating proteins into the cytosol, thereby triggering apoptosis. The death receptor and mitochondrial pathways for caspase activation are sometimes referred to as the extrinsic and intrinsic apoptosis pathways, respectively, though this is an oversimplification.

Also, though commonly viewed as separate pathways and capable of functioning independently, cross-talk can occur between these pathways at multiple levels, depending on the repertoire of apoptosis-modulating proteins expressed.[8]

Protein Domains Associated with Apoptosis Regulation

The proteins that directly control the intrinsic, extrinsic, and other less understood caspase activation pathways often exist as families that can be recognized based on their amino acid sequence and/or structural similarity.[9] Moreover, interactions among these proteins are commonly mediated by domains that are intimately associated with apoptosis regulation, including caspase-associated recruitment domains (CARDs), death domains (DDs), death effector domains (DEDs), Bcl-2 homology (BH) domains of Bcl-2 family proteins, baculovirus inhibitor of apoptosis proteins (IAP) repeat (BIR) domains of IAP family proteins, and NB-ARC domains representing the nucleotide-binding oligomerization domains of CED-4/Apaf-1 family proteins. A summary of the proteins that constitute the known members of these families of apoptosis regulators follows, along with information about their mechanisms and some examples of their relevance to diseases.

Death Domain Proteins

The DD is a protein interaction module consisting of a compact bundle of six α-helices. DDs bind each other, probably forming oligomers of unknown stoichiometry. Specificity for partner selection among DDs is dictated by differences in surface residues. Several of the members of the TNF family of cytokine receptors contain DDs in their cytosolic regions, including TNFR1, Fas (Apo1), DR3 (Apo2), DR4 (TrailR1), DR5 (TrailR2), DR6 in humans, mice, and probably other mammals. The p75 nerve growth factor receptor (p75-NGFR) also contains a modified (type II) DD and has been reported to induce apoptosis under some circumstances.[10] The TNF family receptors, TNFR1, DR3, and DR6, are known to bind an adapter protein, Tradd, via its homologous DD. The DD of Tradd is capable of binding certain other DD-containing proteins, including the adapter protein Fadd. The Fadd (Mort1) protein contains two protein interaction modules, a DD and a DED. Fadd links TNF family death receptors to caspases, using its DD to bind Tradd or to interact directly with the cytosolic DD of the TNFR family member Fas, and employing its DED to bind DED-containing caspases (see below). Experimental evidence has demonstrated the presence of Fadd within the receptor complexes of all known DD-containing members of the TNF family except p75-NGFR. Thus, this protein plays a central role in linking caspases to TNF family

death receptors, a notion borne out by gene ablation studies in mice, which have demonstrated an inability of TNFR1, Fas, and other death receptors tested thus far to induce apoptosis in the absence of Fadd.[11]

An analogous mechanism for recruiting pro-caspases to death receptor complexes has been revealed in the case of Raidd (Cradd). This adapter protein contains a DD in combination with a CARD, allowing it to bind the corresponding CARD found within the prodomain of pro-caspase-2.[12] Based on the aforementioned Fadd knockout studies, however, it remains questionable how important this alternative pathway for caspase activation is.

Additional DD-containing proteins have been implicated in apoptosis, such as the DAP kinase, which modulates apoptosis induction by TNF family death receptors through mechanisms that are not understood.[13] This kinase has been implicated in suppression of metastasis. Several cytoskeleton-associated ankryin family proteins contain DDs, but their relevance to apoptosis remains uncertain. Given evidence, however, that caspase-8 activation is triggered by suspension of adherent epithelial cells, an event that disturbs the cytoskeleton, it is intriguing to speculate a possible role in the phenomenon of anoikis (ie, apoptosis induced by depriving cells of integrin-mediated attachments to extracellular matrix).[14] Avoidance of anoikis represents an important aspect of tumor invasion, metastasis, and angiogenesis. It is also fundamental to correct positioning of cells during development, possibly accounting for the embryonic lethality of Fadd and caspase-8 gene ablation in mice.

DDs, however, are not always involved in caspase activation or apoptosis induction. In fact, DDs have been found in proteins involved in two other receptor signaling systems, namely Toll family receptors and UNC family receptors. Nevertheless, some of the signal transduction pathways in which non-caspase-activating DD proteins are involved at least indirectly regulate apoptosis through effects on NF-κB, suppressing rather than inducing apoptosis. For example, the RIP protein binds Tradd and activates kinases that induce degradation of IkB, thus releasing NF-κB so that it can translocate to the nucleus and fulfill its function as a transcription factor.[15] Among NF-κB-inducible genes are several that block apoptosis, including anti-apoptotic Bcl-2 family members Bfl-1 (A1) and Bcl-X, and IAP family member cIAP2 and possibly cIAP1 and XIAP. The dual function of Tradd, as a partner for both caspase-activator Fadd and NF-κB activator RIP, causes many of the TNF family receptors to nullify their own apoptosis-inducing activity. Thus, TNFR1, DR3, and DR6 are uncertain apoptosis inducers unless NF-κB induction is inhibited.[16] In contrast,

Fas and the Trail receptors DR4 and DR5 only rarely activate NF-κB, probably because these receptor complexes contain Fadd but not Tradd. In fact, it is because Trail receptors (DR4 and DR5) do not induce NF-κB-mediated pro-inflammatory responses *in vivo* that the Trail ligand is under consideration for clinical use in the treatment of cancer, whereas NF-κB-inducing TNF-α proved to be unacceptable.[17]

Multiple mechanisms for modulating signaling by TNF family death receptors probably exist. One that acts directly on DDs involves the silencer of death domains protein (SODD), also know as BAG4.[18] SODD contains a C-terminal BAG domain that allows it to bind Hsc70/Hsp70 family molecular chaperones. An N-terminal domain in SODD mediates its interactions with the DDs of TNFR1 and DR3. Overexpression of SODD prevents TNFR1 and DR3 from spontaneously aggregating and signaling in the absence of ligand. Though unproven, it has been suggested that recruitment of Hsp70/Hsc70 to these death receptors induces conformational changes in the DD that prevent self-oligomerization until the receptors are appropriately triggered by cognate ligands.[19]

Another resistance mechanism has been attributed to FAP, a protein tyrosine phosphatase that uses PDZ domains to interact with the C-termini of Fas and p75-NGFR, suppressing apoptosis signaling by these receptors through uncertain mechanisms.[20] Overexpression of FAP-1 in tumors has been associated with resistance to Fas-induced apoptosis.

Death Effector Domain (DED) Proteins

The structure of the DED is similar to the DD, comprised of 6 α-helices. DEDs are found in the initiator caspases, caspase-8 and -10 in humans (a caspase-10 orthologue has yet to be identified in mice). The prodomain regions of pro-caspase-8 and -10 contain two tandem DEDs, which are responsible their interactions with the DED of Fadd, and thus mediate their recruitment to death receptor complexes. Multiple DED-containing modulators of apoptosis have been identified. Flash, for example, is a large protein containing two DED-like domains which reportedly enhances caspase-8 activation by Fas. DEDD (DEFT) is another DED-containing protein which reportedly enhances Fas-induced apoptosis. DEDD contains an N-terminal DED and a C-terminal histone-like domain.[21] DEDD is present in the cytosol, but during Fas-induced apoptosis it translocates to nuclei in a caspase-dependent manner,

localizing to nucleoli and possibly shutting off ribosomal RNA gene transcription. DEDD associates with Fadd, and to some extent pro-caspase-8, via DED-mediated interactions.[22]

In contrast to Flash and DEDD (DEFT), which enhance Fas-induced apoptosis, the DED-containing protein Flip (also known as Flame, CASH, Clarp, MRIT, Casper, I-Flice, Usurpin) is capable of suppressing caspase-8 activation by Fas and other death receptors. Flip shares extensive amino acid sequence similarity with pro-caspase-8 and -10, containing two N-terminal DEDs followed by a pseudo-caspase domain that lacks critical residues required for protease activity, including the catalytic cysteine.[23] Flip associates with pro-caspase-8 and also competes with pro-caspases-8 and -10 for binding to Fadd, thus squelching death receptor signaling. Interestingly, some tumors have been reported to contain inappropriately elevated levels of Flip, rendering them resistant to apoptosis induction by Fas-expressing CTLs.[24] Though controversial, Flip-mediated resistance to Fas may even permit tumor cells to tolerate expressing FasL, using this death ligand as a weapon against neighboring normal cells and triggering apoptosis of immune cells. Interestingly, some viruses also encode DED-containing proteins analogous to Flip that similarly suppress apoptosis induced by Fas. By thwarting Fas-induced apoptosis, viruses presumably can avoid CTL-mediated eradication of the host cells they infect, allowing more time for viral replication.

Decreased expression of the DED-containing apoptosis suppressor, Flip (Usurpin), occurs in myocardial tissue damaged by ischemia-reperfusion injury, suggesting increased sensitivity to apoptosis induction via the death receptor pathway.[25]

CARD Family Proteins

Several pro-caspases contain N-terminal CARDs in their prodomains, including caspases 1, 2, 4, 5, and 9 in humans and caspases 1, 2, 9, 11, and 12 in mice. (To date, orthologues of human caspases 4 and 5 have not been observed in mice and conversely orthologues of murine caspases 11 and 12 have not been found in humans.) Roles for CARD-carryingcaspases in diseases have been revealed through gene ablation studies in mice. For example, caspase-1 knockout mice exhibit marked resistance to endotoxin-induced sepsis.[26] Caspase-1, as well as caspase-2 and caspase-11, knockout mice also suffer less tissue loss in stroke models. Inhibition of caspase-1 also slows progression in a mouse model of Huntington’s disease. In addition, cells from caspase-12 knockout mice are resistant to apoptosis induced by amyloid β-peptide, a finding of potential relevance to Alzheimer’s disease. In this regard, caspase-12 appears to be associated with the endoplasmic reticulum

(ER) and becomes specifically activated by ER stress, thus linking ER damage to a caspase activation pathway independently of the mitochondrial (cytochrome c) and death receptor (TNF family) pathways.[27]

The overall structure of the CARD is similar to DDs and DEDs, comprised of 6 α-helices.Homotypic interactions among CARD-carrying proteins play important roles in caspase activation throughout animal evolution. One of the paradigms used for caspase activation is embodied in CED-4/Apaf-1 family proteins. These proteins contain a CARD domain in combination with a nucleotide-binding oligomerization domain, known as a NB-ARC (NACHT) domain for *N*ucleotide-*B*inding domain homologous to *A*paf-1, *C*ED-4 and plant *R* gene products. The N-terminal CARDs of Apaf-1 in humans and CED-4 in the nematode *Caenorhabditiselegans* mediate interactions with the CARDs of specific initiator caspases, pro-caspase-9 and pro-CED-3, respectively.[28] Oligomerized Apaf-1 and CED-4 activate caspases by the induced proximity method.

With the CED-4 protein of *C. elegans*, binding and activation of the caspase pro-CED-3 is spontaneous. In contrast, the human and *Drosophila* Apaf-1 proteins contain an additional regulatory domain, comprised of several WD repeat domains, which renders them dependent on cytochrome c.[29] Though less is known about the fly protein, biochemical analysis of human Apaf-1 using *in vitro* reconstituted systems with purified components indicates that cytochrome c, in combination with dATP, induces oligomerization of Apaf-1 molecules, followed by binding to and activation of pro-caspase-9. Several mechanisms for suppressing caspase activation by Apaf-1 have been identified, including expression of a shorter noncatalytic isoform of pro-caspase-9 that competes with full-length caspase-9 for binding Apaf-1, phosphorylation of human caspase-9 by the kinase Akt, overexpression of heat shock proteins, and alkaline pH.[30]

IAP Family Proteins

The IAPs represent a family of evolutionarily conserved apoptosis suppressors. IAPs are found in the genomes of mammals, insects, and certain animal viruses. All members of this family, by definition, contain at least one copy of a so-called BIR (baculovirusiap repeat) domain, a zinc-binding fold important for their anti-apoptotic activity. In addition to 1 to 3 copies of a BIR domain, many IAP family proteins also contain other domains, including RING zinc-fingers, CARDs, Ubiquitin-conjugating enzyme (E2s) domains, or putative nucleotide-binding domains. Interestingly, the RINGs of IAPs have recently been implicated

in interactions with the cellular components of the ubiquitination machinery, thus controlling turnover of these proteins and possibly other proteins with which they associate. Also, the BIR-containing protein, Apollon (Bruce) contains a domain with ubiquitin-conjugating enzyme (E2) activity, further suggesting links of BIR family proteins to the cellular ubiquitination machinery.[31] Though the overall relevance to apoptosis of this protein remains uncertain, antisense-mediated down-regulation of Apollon reportedly can sensitize tumor cell lines to apoptosis induced by anti-cancer drugs. In this regard, it bears noting that the mere presence of a BIR domain does not necessarily indicate anti-apoptotic activity. For example, BIR-containing proteins, which regulate mitosis and meiosis but have no apparent effect on cell death regulation, have been studied in yeast and *C. elegans*.[32]

Though IAP family proteins may possess other functions, several of them have been shown to bind and potently inhibit activated caspases. Among the caspases inhibited by human IAP family members XIAP, cIAP1, and cIAP2 are the effector caspases-3 and -7, as well as the initiator caspase-9. Suppression of the effector caspases maps to the N-terminal half of the protein in the region encompassing the first two BIR domains, BIR1 and BIR2, whereas the third BIR domain is required for suppression of caspase-9. Importantly, IAPs are selective caspase inhibitors and lack activity against many members of the caspase family of cell death proteases. This stands in marked contrast to the baculovirus p35 protein, an apoptosis suppressor that displays broad activity against caspases but for which no cellular homologue has been identified.[33]

Human IAPs can arrest apoptosis induced via either the intrinsic (mitochondrial) or extrinsic (death receptor) pathways, probably because they target effector caspases common to both pathways. In contrast, insect IAPs may inhibit primarily initiator caspases within the mitochondria-dependent pathway, possibly reflecting the later evolution of the death receptor (TNF family) pathway.[34]

Bcl-2 Family Proteins

The mitochondria-dependent pathway for apoptosis is governed by Bcl-2 family proteins. Bcl-2 family proteins are conserved throughout metazoan evolution, with homologues found in mammalian, avian, fish, and amphibian species, as well as in invertebrates such as *C. elegans*, *Drosophila*, and marine sponges. Several types of animal viruses also harbor Bcl-2 family genes within their genomes. Both pro- and anti-apoptotic Bcl-2 family proteins exist, and many of these proteins physically bind each other, forming a complex network of homo-

and heterodimers.[35] The relative ratios of anti- and pro-apoptotic Bcl-2 family proteins dictate the ultimate sensitivity or resistance of cells to various apoptotic stimuli, including growth factor/neurotrophin deprivation, hypoxia, radiation, anti-cancer drugs, oxidants, and Ca^{2+} overload. Not surprisingly, then, alterations in the amounts of these proteins have been associated with a variety of pathological conditions, characterized by either too much (cell loss) or too little (cell accumulation) cell death. These diseases include cancer, autoimmune disorders such as lupus (where a failure to eradicate autoreactive lymphocytes occurs), immunodeficiency associated with HIV infection, and ischemia-reperfusion injury during stroke and myocardial infarction, among others.

Based on their predicted (or experimentally determined) three-dimensional structures, Bcl-2 family proteins can be broadly divided into two groups. One subset of these proteins is probably similar in structure to the pore-forming domains of bacterial toxins, such as the colicins and diphtheria toxin. These α-helical pore/channel-like proteins include both pro-apoptotic proteins (Bcl-2, Bcl-X_L, Mcl-1, Bfl-1, Bcl-W, and possibly Boo), as well as pro-apoptotic proteins (Bax, Bak, Bok, and Bid). Most of these protein in this subcategory can be recognized by conserved stretches of amino acid sequence homology, including the presence of Bcl-2 homology (BH) domains BH1, BH2, BH3, and sometimes BH4. However, this is not uniformly the case, as the Bid protein contains only a BH3 domain but has been determined to share the same overall protein fold with Bcl-X_L.[39] Where tested, these proteins, including Bcl-2, Bcl-X_L, Bax, and Bid, have all been shown to form ion-conducting channels in synthetic membranes *in vitro*.

The other subset of Bcl-2 family proteins appears to have in common only the presence of the BH3 domain, including Bad, Bik, Bim, Hrk, Bcl-G_S, p193, and APR (Noxa). These proteins are all pro-apoptotic in their function, and their cell death-inducing activity depends on their ability to dimerize with anti-apoptotic Bcl-2 family members, typically functioning as *trans*-dominant inhibitors of proteins such as Bcl-2 and Bcl-X_L.[40] In this regard, the BH3 domain has been shown to mediate dimerization among Bcl-2 family proteins. This domain consists of an amphipathic α-helix ~16 amino acids in length that inserts into a hydrophobic crevice on the surface of anti-apoptotic proteins such as Bcl-X_L. Thus, mutations in the BH3 domain of proteins such as Bad, Bik, Bim, Bcl-G_S, and Hrk that abolish their ability to bind other Bcl-2 family member also abrogate their capacity to induce apoptosis.

Conclusion

Advances in elucidating the molecular mechanisms of apoptosis and its regulation have laid the foundation for a deeper understanding of the pathophysiology of many diseases. More importantly, this work has revealed strategies for therapeutic intervention in a wide range of ailments, including cancer, autoimmune disorders, immunodeficiency, inflammation, ischemic heart disease, stroke, and neurodegenerative diseases. Small-molecule inhibitors of caspases, for example, are now in early clinical trials, with more to come in the near future. Anticancer therapies based on antisense oligonucleotide-mediated suppression of *BCL-2* expression have advanced to Phase III trials. Biological agents that control apoptosis, such as Trail-ligand (Apo2L), will also soon enter clinical trials for patients with refractory metastatic cancers. This progress in translating knowledge about apoptosis mechanisms into the clinical arena suggests rich opportunities for new and more effective treatments for many of the medical illnesses for which adequate therapies currently do not exist.

Corresponding Author

Sivakumar M

Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha University,Saveetha Institute of Medical and Technical Science

162, Poonamallee High Road,Chennai-600077, Tamil Nadu, India.

Telephone Number: +919443602627

Email: drsivaomfs@gmail.com

References

1. Kerr JF, Wyllie AH, Currie AR: Apoptosis: a basic biological phenomenon with wide-ranging implications in tissue kinetics. Br J Cancer 1972, 26:239-257

2. Alnemri ES, Livingston DJ, Nicholson DW, Salvesen G, Thornberry NA, Wong WW, Yuan J: Human ICE/CED-3 protease nomenclature. Cell 1996, 87:171.

3. Thornberry N, Lazebnik Y: Caspases: enemies within. Science 1998, 281:1312-1316

4. Cryns V, Yuan Y: Proteases to die for. Genes Dev 1999, 13:371

5. Nicholson DW: ICE/CED3-like proteases as therapeutic targets for the control of inappropriate apoptosis. Nat Biotechnol 1996, 14:297-301

6. Lakshmi T, Ezhilarasan D, Vijayaragavan R, Bhullar SK, Rajendran R. Acacia catechu ethanolic bark extract induces apoptosis in human oral squamous carcinoma cells. Journal of advanced pharmaceutical technology & research. 2017 Oct;8(4):143.

7. Salvesen GS, Dixit VM: Caspases: intracellular signaling by proteolysis. Cell 1997, 91:443-446

8. Thornberry N, Rano T, Peterson E, Rasper D, Timkey T, Garcia-Calvo M, Houtzager V, Nordstrom P, Roy S, Vaillancourt J, Chapman K, Nicholson D: A combinatorial approach defines specificities of members of the caspase family and granzyme B. J Biol Chem 1997, 272:17907-17911

9. Salvesen GS, Dixit VM: Caspase activation: the induced-proximity model. Proc Natl Acad Sci USA1999, 96:10964-10967

10. Wallach D, Varfolomeev EE, Malinin NL, Goltsev YV, Kovalenko AV, Boldin MP: Tumor necrosis factor receptor and Fas signaling mechanisms. Annu Rev Immunol 1999, 17:331-367

11. Yuan J. Transducing signals of life and death. Curr Opin Cell Biol 1997, 9:247-251

12. Parathan KK, Kumar M, Kannan R, Chitrambalam P, Volga H. Cardiac amyloidosis in a patient with multiple myeloma. Journal of evolution of medical and dental sciences-jemds. 2015 Oct 8;4(81):14248-52.

13. Green DR, Reed .Mitochondria and apoptosis. Science 1998, 281:1309-1312

14. Varfolomeev E, Schuchmann M, Luria V, Chiannilkulchai N, Beckmann J, Mett I, Rebrikov D, Brodianski V, Kemper O, Kollet O, Lapidot T, Soffer D, Sobe T, Avraham K, Goncharov T, Holtmann H, Lonai P, Wallach D: Targeted disruption of the mouse caspase 8 gene ablates cell death inducation by the TNF receptors, Fas/Apo1, and DR3 and is lethal prenatally. Immunity 1998, 9:267-276

15. Juo P, Kuo CJ, Yuan J, Blenis J: Essential requirement for caspase-8/FLICE in the initiation of the Fas-induced apoptotic cascade. Curr Biol 1998, 8:1001-1008

16. Kuida K, Haydar TF, Kuan C-Y, Gu Y, Taya C, Karasuyama H, Su MS-S, Rakic P, Flavell RA: Reduced apoptosis and cytochrome c-mediated caspase activation in mice lacking caspase 9. Cell 1998, 94:325-337

17. Hakem R, Hakem A, Duncan GS, Henderson JT, Woo M, Soengas MS, Elia A, de la Pompa JL, Kagi D, Khoo W, Potter J, Yoshida R, Kaufman SA, Lowe SW, Penninger JM, Mak TW: Differential requirement for caspase 9 in apoptotic pathways in vivo. Cell 1998, 94:339-352

18. Chinnaiyan AM, O'Rourke K, Tewari M, Dixit VM: FADD, a novel death domain-containing protein, interacts with the death domain of Fas and initiates apoptosis. Cell 1995, 81:505-512

19. Boldin MP, Varfolomeev EE, Pancer Z, Mett IL, Camonis JH, Wallach D: A novel protein that interacts with the death domain of Fas/APO1 contains a sequence motif related to the death domain. J Biol Chem1995, 270:7795-7798

20. Muzio M, Chinnaiyan AM, Kischkel FC, O'Rourke K, Shevchenko A, Ni J, Scaffidi C, Bretz JD, Zhang M, Gentz R, Mann M, Krammer PH, Peter ME, Dixit VM: FLICE, a novel FADD-homologous ICE/CED-3-like protease, is recruited to the CD95 (Fas/APO-1) death-inducing signaling complex. Cell1996, 85:817-827

21. Boldin MP, Goncharov TM, Goltsev YV, Wallach D: Involvement of MACH, a novel MORT1/FADD-interacting protease, in Fas/APO-1- and TNF receptor-induced cell death. Cell 1996, 85:803-815

22. Muzio M, Stockwell BR, Stennicke HR, Salvesen GS, Dixit VM: An induced proximity model for caspase-8 activation. J Biol Chem 1998, 273:2926-2930

23. Zou H, Henzel WJ, Liu X, Lutschg A, Wang X: Apaf-1, a human protein homologous to C. elegans CED-4, participates in cytochrome c-dependent activation of caspase-3. Cell 1997, 90:405-413

24. Zou H, Li Y, Liu X, Wang X: An APAF-1 cytochrome c multimeric complex is a functional apoptosome that activates procaspase-9. J Biol Chem 1999, 274:11549-11556

25. Saleh A, Srinivasula S, Acharya S, Fishel R, Alnemri E: Cytochrome c and dATP-mediated oligomerization of Apaf-1 is a prerequisite for procaspase-9 activation. J Biol Chem 1999, 274:17941-17945

26. Li P, Nijhawan D, Budihardjo I, Srinivasula S, Ahmad M, Alnemri E, Wang X: Cytochrome c and dATP-dependent formation of Apaf-1/Caspase-9 complex initiates an apoptotic protease cascade. Cell1997, 91:479-489

27. Stennicke HR, Jurgensmeier JM, Shin H, Deveraux Q, Wolf BB, Yang X, Zhou Q, Ellerby HM, Ellerby LM, Bredesen D, Green DR, Reed JC, Froelich CJ, Salvesen GS: Pro-caspase-3 is a major physiologic target of caspase-8. J Biol Chem 1998, 273:27084-27090

28. Rodriguez A, Oliver H, Zou H, Chen P, Wang X, Abrams J: Dark is a Drosophila homologue of Apaf-1/CED-4 and functions in an evolutionarily conserved death pathway. Nat Cell Biol 1999, 1:272-279

29. Huang B, Eberstadt M, Olejniczak ET, Meadows RP, Fesik SW: NMR structure and mutagenesis of the Fas (APO-1/CD95) death domain. Nature 1996, 384:638-641

30. Liepinsh E, Ilag LL, Otting G, Ibanez CF: NMR structure of the death domain of the p75 neurotrophin receptor. EMBO J 1997, 16:4999-5005

31. Bredesen D, Ye X, Tasinato A, Sperandio S, Assa-Munt N, Rabizadeh S: P75NTR and the concept of cellular dependence: seeing how the other half die. Cell Death Differ 1998, 5:365-371

32. Hsu H, Xiong J, Goeddel DV: The TNF receptor 1-associated protein TRADD signals cell death and NF-kappaB activation. Cell 1995, 81:495-504

33. Chinnaiyan AM, O'Rourke K, Yu G-L, Lyons RH, Garg M, Duan R, Xing L, Gentz R, Ni J, Dixit VM: Signal transduction by DR3, a death domain-containing receptor related to TNFR-1 and CD95. Science1996, 274:990-992

34. Pan G, Bauer JH, Haridas V, Wang S, Liu D, Yu G, Vincenz C, Aggarwal BB, Ni J, Dixit VM: Identification and functional characterization of DR6, a novel death domain-containing TNF receptor. FEBS Lett 1998, 431:351-356

35. Yeh W, De La Pompa J, El-Deiry W, Lowe S, Goeddel D, Mak T: FADD essential for embryo development and signaling from some, but not all, inducers of apoptosis. Science 1998, 279:1954-1958

36. Zhang J, Cado C, Chen A, Kabra N, Winoto A: Fas-mediated apoptosis and activation-induced T-cell proliferation are defective in mice lacking FADD/Mort 1. Nature 1998, 392:296-299

37. Sprick MR, Weigand MA, Rieser E, Rauch CT, Juo P, Blenis J, Krammer PH, Walczak H: FADD/MORT1 and caspase-8 are recruited to TRAIL receptors 1 and 2 and are essential for apoptosis mediated by TRAIL receptor 2. Immunity 2000, 12:599-609

38. Kischkel FC, Lawrence DA, Chuntharapai A, Schow P, Kim KJ, Ashkenazi A: Apo2L/TRAIL-dependent recruitment of endogenous FADD and caspase-8 to death receptors 4 and 5. Immunity 2000, 12:611-620

39. Duan H, Dixit VM: RAIDD is a new 'death' adaptor molecule. Nature 1997, 385:86-89

40. Ahmad M, Srinivasula SM, Wang L, Talanian RV, Litwack G, Fernandes-Alnemri T, Alnemri ES: CRADD, a novel human apoptotic adaptor molecule for caspase-2 and FasL/tumor necrosis factor receptor-interacting protein RIP. Cancer Res 1997, 57:615-619

Micro-Nutrition – A Review

E.Shubha Poorani [1], Sirisha [2], Sreedevi Dharman [3]
Graduate Student [1],Assistant Professor [2],Department of Microbiology,
Senior Lecturer [3],Department of Oral Medicine,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Micronutrients play animportant part in metabolism and in maintaining tissue function. An adequate intake is therefore necessary, but provision of excess supplements to people who do not need them may be harmful. Single micronutrient deficiency states are comparatively easily recognised and treated. Subclinical deficiency, often of multiple micronutrients, is themost difficult to recognise, and laboratory assessment is often complicated by the acute phase response. Clinical benefit is most likely in those people who are severely depleted and at risk of complications, and is unlikely if this is not the case. There is a little evidence for supplements leading to a reduction in the incidence of infections of the elderly population, in coronary artery disease, or in the malignant disease. The best evidence for benefit is in the critical illness, and in children in developing countries who consume a deficient diet. More clinical trials are required along with good clinical outcomes to optimise the intake of prevention and treatment of disease.

Key Words*: Micronutrients, Supplements, Disease*

Introduction

The growing interest in the importance of the micronutrients (essential trace elements and vitamins) in optimising health, and in prevention or treatment of disease. This stems partly from the increase in knowledge and understanding of the biochemical functions of these nutrients, but also from the extensive but less well founded commercial claims for such substances. It is important that doctors and other health professionals are aware of the evidence for the nutritional essentiality of these sub- stances, and for the situations where an increased intake may lead to clinical benefit.

This review will therefore consider current knowledge of the requirements in health, those people at risk of an inadequate intake, and the conditions where supplements may be clinically required. The review will focus only on the generally accepted essential inorganic micronutrients (trace elements) and organic micronutrients (fat soluble and water soluble vitamins) for which deficiency states, with biochemical, physiological, or structural changes, have been clearly reported—such states occur after pro- longed consumption of a diet lacking the single nutrient under consideration, and are uniquely corrected by addition of the nutrient back into the diet. Other nutrients for which there are no confirmed deficiency states, but where supplements are often taken, such as v-3 fatty acids, glucosamine, coenzyme Q, will not be considered[1].

Functions

Cofactors in metabolism—follow components are every now and again associated with regulating catalyst action or are an indispensable piece of chemical prosthetic gatherings—for instance, zinc is a cofactor for more than 100 compounds, though selenium is required as selenocysteine inside the protein glutathione peroxidase.

N Coenzymes in metabolism—numerous vitamins or metabolites of vitamins are required to have a dynamic influence inside complex biochemical reactions, for instance, riboflavin and niacin in the electron transport chain, or folic corrosive as a major aspect of methyl assemble exchange. These responses are basic to delegate digestion and guarantee usage of the real supplements to give vitality, proteins and nucleic acids.

N Genetic control—zinc "fingers" are transcription control factors that predicament to DNA and direct interpretation of receptors for steroid hormones and different variables.

N Antioxidants—a significant part of the well known enthusiasm for the micronutrients comes from the acknowledgment that a large number of the micronutrients have hostile to oxidant properties. Oxidative digestion unavoidably prompts age of responsive oxygen species (ROS) or "free radicals", which can possibly bring on additional oxidative responses, particularly to those parts of the phone in a moderately decreased state, for example, cell layers or nucleic acids.[1] The potential to cause harm is restricted by instruments that incorporate direct extinguishing of oxidant action by tocopherols (vitamin E) or carotenoids (vitamin An), or protein frameworks to discard the results of oxidation—super-oxide dismutase (either zinc/copper or manganese subordinate) and glutathione peroxidase (selenium subordinate)[2].

Reference Nutrient Intakes of Micronutrients

Numerous nations have created recommendations for admission of micronutrients in the typical eating routine. These have been founded on the watched admissions in the solid populace, combined with little quantities of point by point supplement adjust studies, and research facility assessments of blood and tissue status related with specific levels of admission. Qualities have been set for the admission of every micronutrient beneath which a clinical inadequacy state is progressively likely, or above which a lethality state is probably going to create. Despite the fact that these are applicable for populaces, the trouble is to decide how sufficient is the admission for a specific individual. This is particularly the case for specific micronutrients where it is perceived that there might be extra advantages as far as tissue function if the intake is to some degree more important than that required, just to keep an insufficiency state from creating[2,3].

FinancialImpacts

Numerous gatherings in the populace have a poor admission of supplements because of an intricate association amongst social and monetary conditions. In this way individuals from a poorer foundation may well take less crisp products of the soil . Such gatherings may profit by some type of micronutrient supplement, albeit coordinate examinations are missing of arrangement of supplements instead of enhancing dietary admission[4,5].

Population Groups with Known Poor Intake or Increased Requirements

Certain gatherings inside the populace are known to have a poor or lacking admission. For instance, youths and adolescents may have a deficient admission of drain and different wellsprings of calcium,[6] and elderly individuals in nursing homes and private care have a lacking vitamin D intake[5]. Furthermore, regardless of there being a satisfactory supply of most micronutrients in the sustenance accessible in nursing homes, there is a high occurrence of biochemical inadequacy for most micronutrients, including zinc, press, vitamin C, and riboflavin[5]. There are numerous conceivable reasons for inadequate admission or retention, including anorexia, failure to cut up nourishment, or poor nourishment introduction.

Besides, certain gatherings have expanded prerequisites—eager moms require expanded folate both before origination and in the primary trimester,[2] smokers require extra vitamin C,[3] and individuals who are recuperating from an intense disease or after surgery likely have numerous requirements[7].

It would be altogether fitting for gatherings of this sort to take supplements to guarantee their aggregate admission, including their eating routine, was sufficient.

Other Biochemical Effects—Uncertain Clinical Consequences

As such a significant number of the micronutrients are engaged with parts of the cancer prevention agent barrier systems, it has been expected that imperfect cancer prevention agent movement will prompt oxidative harm to tissues or to parts of the cell, with conceivably genuine outcomes.

Oxidative Harm and Coronary Artery Disease

On account of the solid connection between oxidised low thickness lipoprotein and advancement of coronary vein malady, together with the lower frequency of coronary conduit illness in nations where there is a high admission of cancer prevention agents, it has been normal that an expanded admission of cell reinforcements would lessen the rate and complexities of coronary illness. Some little investigations did at first recommend advantageous impacts from taking vitamin E supplements[8,9].

Notwithstanding, more as of late various extensive scale trials have neglected to show such an advantage, for instance, the HOPE study[10] considered 9541 patients with high danger of coronary illness, who got 400 IU vitamin E in addition to ACE inhibitors and demonstrated no advantage from vitamin E supplements. The GISSI study[11] in Italy neglected to demonstrate any advantage from the utilisation of vitamin E in 324 patients after myocardial dead tissue, in spite of the fact that patients benefitted from an expanded admission of polyunsaturated unsaturated fats.

Additionally, the Medical Research Council/British Heart Foundation study[12] of 20 333 high hazard UK grown-ups demonstrated that an admission of vitamin E (600 mg), vitamin C (250 mg), and b carotene (20 mg), over a five year time frame had no critical advantage on frequency or movement of coronary illness.

An exhaustive survey of concentrates by the American Heart Association reasoned that cardiovascular sickness lessening can be accomplished by long haul utilisation of an all around adjusted eating routine together with standard physical movement, and that there was no extra advantage from utilisation of micronutrients at levels surpassing these acquired from such a diet[13].

Moreover, Miller and coworkers[14]. have distributed a meta-examination of 19 clinical trials inspecting the part of vitamin E, either alone (nine investigations) or together with different cell reinforcements (10 considers) in a sum of 135 000 members. They presumed that high admission of vitamin E at dosages of .400 IU/day or more conveyed no advantage, and without a doubt may expand all reason mortality. They assessed that the expanded hazard may begin at measurements as low as 150 IU/day. Supplements of even lower dosages than this may however convey a peripheral advantage. An especially essential component of this meta examination is the measurements impact of vitamin E. It has for some time been trusted that the higher the measurements of vitamin E the better would be any potential advantage, and that there were no unsafe impacts related with a high dosage. This meta examination makes it clear that high measurements of any supplement are probably going to be related with hurtful symptoms. On the off chance that valuable impacts are gotten by remedy of a sub-clinical lack, at that point the dosage expected to do as such should be painstakingly settled.

Oxidative Harm and Neoplastic Sickness

It has additionally been expected that an expanded admission of cell reinforcements would be valuable in lessening the rate of different types of growth, by diminishing oxidation actuated transformations in DNA. In fact this is the most well-known reason given in the USA for utilisation of such supplements[15]. Apart from the investigations of selenium, including that said above, and an examination on liver growth in China40 different investigations have been disillusioning. Specifically, an investigation over a five to eight year time of 29 133 Finnish male smokers who got b carotene (20 mg), a tocopherol (50 mg), or both, demonstrated a 18% higher frequency of lung disease in the b carotene gathering, though a tocopherol had no effect[16]. An examination from the USA of 18 314 smokers, previous smokers, and asbestos labourers who got 30 mg b carotene and 25 000 IU vitamin A [17]over a four year term, additionally demonstrated that the supplemented aggregate had a higher relative danger of lung malignancy of 1.28.

Different investigations have given clashing confirmation. The attendant wellbeing study in USA of 88 758 ladies demonstrated that folate admission or supplements for under 15 years did not altogether lessen the danger of colon disease, but rather more prominent than 15 years' supplementation reduced risk[18]. The advantage of folate is by all accounts most prominent in those taking in excess of two mixed beverages for each day[19]. However, in a similar gathering of ladies there was a higher frequency of non-Hodgkin's lymphoma in those supplemented[20]. Other investigations have demonstrated that long haul vitamin E (more prominent than 10 years) may decrease bladder malignancy mortality[21].

The worry about supplements and disease was com-beat in a further meta-investigation that demonstrated that with the conceivable special case of selenium, cell reinforcement supplements did not lessen the danger of gastric or intestinal malignancies, but instead, in the event that anything, to some degree expanded that hazard by around 6%.47 This prompted the Lancet featuring in an article—"The prospect that vitamin pills may do no great as well as execute their clients is a frightening hypothesis given the immense amounts that are utilized as a part of certain communities"[22].

In any case, strangely, and on a more positive note, a current investigation of the eating routine of the individuals who participated in the lung tumor think about performed in

Finland[23] demonstrated that when a cell reinforcement record was utilised that joined all cell reinforcements in sustenances eaten by the members, the subjects in the most noteworthy quintiles of cancer prevention agent admissions had a lower rate of lung cancer[24]. So no doubt when taken in nourishments cell reinforcements may have a gainful impact, though high dosage filtered supplements might be unsafe.

There have additionally been some intriguing discoveries from the SU.VI.MAX contemplate on 13 000 grown-ups, matured over 60 years, from the overall public in France[25]. A supplement of similarly low measurements of individual cancer prevention agents (vitamin C 120 mg, vitamin E 30 mg, b carotene 6 mg, selenium 100 mg, zinc 20 mg) was examined over a middle follow up time of 7.5 years. Albeit no vital contrasts were found in all out malignancy occurrence, ischaemic coronary illness, or all reason mortality, a noteworthy distinction was seen amongst male and female reactions. The sex stratified investigation indicated bring down tumor frequency and all reason mortality in men who took the supplement yet not in ladies. A further examination of the information recommended this may be a consequence of the poorer benchmark status of b carotene in men, which was dynamically rectified amid the time of supplementation. A further intriguing outcome from this examination was that the men getting supplements had a lower occurrence of prostate tumor if PSA toward the start of the examination was typical, however an expanded frequency if PSA at benchmark was raised[26]. The essentialness of these outcomes in connection to advancement of prostate growth requires advance examination.

In rundown, the circumstance in regards to cell reinforcement supplements and tumor is as of now confounded. There would appear to be little uncertainty that an eating routine rich in cell reinforcements is probably going to limit the danger of specific kinds of disease, however the circumstances where particular supplements might be valuable requires considerably additionally work to focus on the populaces and the admissions of micronutrients that might be helpful, and to restrict the likelihood that these might be destructive.

Oxidative Harm and Eye Infection

The Age Related Eye Disease Study (AREDS) Group[27,28] detailed the outcomes from a huge forthcoming twofold visually impaired fake treatment controlled investigation crosswise over 11 focuses in the USA. The supplements were of vitamin C (500 mg) in addition to

vitamin E (400 IU) in addition to b carotene (15 mg), or zinc (80 mg) in addition to copper (2 mg), or both of these. 4629 patients were followed up for a mean time of 6.3 years. The essential conclusions from this examination were right off the bat that there was no critical distinction being developed or movement of age related waterfall. Be that as it may, there was a critical diminishment in the movement of age related macular degeneration, the combi-country of cell reinforcements together with zinc being best and prompting a lessening of around 25%.

Non-Specific Functional Effects

Immune Function

As a significant number of the micronutrients are engaged with parts of the cancer prevention agent barrier systems, it has been expected that imperfect cancer prevention agent movement will prompt oxidative harm to tissues or to parts of the cell, with conceivably genuine outcomes.

Infections in Youngsters

Zinc deficiency is common in kids in creating nations where loose bowels is additionally a vital issue. In six of nine trials, zinc supplementation essentially lessened the rate of loose bowels, and in five of these there was a lower occurrence of pneumonia[29]. In addition, in intense the runs trials, zinc supplemented kids had a 15% lower prob-capacity of proceeding with the runs on a given day, and in persevering the runs trials, there was a 24% lower likelihood of proceeding with the runs.

Vitamin A supplements in populaces with poor vitamin A status, have been appeared to lessen mortality from loose bowels in group studies, and passings from pneumonia in measles studies[30].

A conceivable cooperation amongst zinc and vitamin A status has been investigated by Rahman and colleagues[31]. In an examination on 800 youngsters (matured 12– 35 months) in Bangladesh, a two week supplement of zinc, vitamin A, both, or fake treatment was given to kids who were then followed up for a half year. Consolidated zinc and vitamin A synergistically decreased the commonness of constant loose bowels and looseness of the bowels. Curiously, zinc alone was related with a noteworthy increment in intense lower respiratory contamination, yet this unfriendly impact was decreased by collaboration amongst

zinc and vitamin A. In an investigation of tuberculosis in Indonesia, supplementation with zinc and vitamin A prompted substantially before determination of radiological changes and time to sputum negativity[32].

In an extensive late examination in India on private school-kids with biochemical confirmation of poor status for a few micronutrients, a multi-micronutrient supplement did not lessen the occurrence of normal youth contaminations, but rather reduced the term of such illnesses[33].

HIV Disease and AIDS

Weight reduction is a typical issue in HIV, and patients are every now and again found to have anomalies of plasma mineral and follow component focuses, particularly of zinc, selenium, and magnesium[34]. There are various connecting factors, including loss of hunger, diminished assimilation, diarrhoea and urinary misfortunes, and the impacts of redistribution from plasma to tissues because of the reaction to infection[35].

Of uncommon importance is the part of follow components and different micronutrients in cell reinforcement guard. Zinc and copper are basic for cytoplasmic superoxide dismutase, manganese for the mitochondrial protein, and selenium is a piece of the prosthetic gathering of glutathione peroxidase. Loss of cancer prevention agent action will prompt expanded actuation of atomic factor-kB (NF-kB), which is a key controller of HIV replication[36]. There is prove that decrease in plasma selenium parallels the loss of CD4+ cells, and that low levels of selenium in youngsters is identified with speedier sickness movement and to mortality. In spite of the fact that supplementation may prompt biochemical change, demonstrating that this prompts enhanced clinical result remains controversial[37]. In any case, one examination suggests that supplementation will postpone movement of the seriousness of HIV[38].

Selenium Insufficiency and Harmfulness of Disease

In spite of the fact that not straightforwardly identifying with resistant capacity, a different line of examination has potential results on the occurrence of contamination. This is the finding that when an ordinarily kindhearted strain of Coxsackie B3 infection is infused into selenium lacking mice, the infection changes to a more harmful shape that may cause serious cardiomyopathy[39]. The viral genome was found to have transformed in six districts when

the infection was refined toward the finish of the examination. A similar gathering has likewise discovered that flu infection causes more serious lung malady in selenium insufficient mice, presumably additionally because of genome transformation—they distinguished 29 nucleotide changes in the M1 lattice protein, an inward popular protein[40]. The importance of these discoveries to human irresistible illness stays to be built up, yet in the event that there are similar impacts in people, there are suggestions for enhancement of selenium status.

Contaminations in the Elderly Populace

There has additionally been a conviction that elderly individuals when all is said in done, who frequently have poor micronutrient status and weakening resistant capacity, would especially profit by supplements. This field of work has experienced severely the current disclosures with respect to adulteration of work by Dr Ranjit Chandra—his production in the Lancet was one of only a handful couple of concentrates to offer confidence to the speculation that supplements would decrease irresistible malady in an elderly population[41]. This examination has now been discredited[42].

One critical examination included systematised elderly patients in France, where 725 patients more than 65 years of age partook in a two year twofold visually impaired fake treatment controlled trial, where they got either follow components (20 mg zinc/100 mg selenium), vitamins (vitamin C 120 mg, b carotene 6 mg, a tocopherol 15 mg) or both of these[43]. The outcomes were suggestive, however did not achieve measurable noteworthiness, of a diminishment in respiratory diseases over the two year time frame in the gathering getting follow components, with halfway outcomes for the vitamin gatherings. This outcome was however especially striking in one individual nursing home concerning consolidated respiratory and urogenital contaminations, where in the 80 patients considered, the gathering getting follow components alone had a noteworthy decrease in diseases, while the gatherings accepting vitamins, either alone or together with follow components did not[44]. Specifically noteworthy in the fundamental examination was the finding that sero-change to flu immunization was altogether better in the follow component gather alone, and essentially more awful in the gathering accepting vitamins. This observing critically requires to be examined in future examinations, as it would have suggestions on whether the elderly populace would profit more from focused particular supplements instead of a general multivitamin and follow component supplements. We have however been not able

recogniseany change in sero-transformation by one month's supplementation with an adjusted follow component and vitamin preparation[45].

Taken by and large, a meta-examination demonstrated that there is pretty much nothing if any advantage from a multivitamin and mineral supplement, as far as occurrence of diseases in an elderly population,[46] and this has as of late been affirmed in a vast free living populace in Scotland[47].

Bone Capacity

Postmenopausal ladies specifically are in danger of osteoporosis, and in spite of the fact that the ailment isn't caused by absence of calcium and vitamin D, sufficient arrangement of these smaller scale supplements is advantageous in keeping up and surely expanding bone mass[48]. There is a positive connection between's zinc admission and bone mineral thickness in moderately aged pre-menopausal women[49]. A controlled trial of copper supple-mentation in moderately aged ladies demonstrated no misfortune in bone mineral thickness (BMD) in the copper supplemented amass contrasted and a noteworthy decline in BMD in the control

Group[50]. A little trial of calcium, zinc, manganese, and copper supplements demonstrated a beneficial outcome on spinal BMD in post-menopausal women[51]. Furthermore, there is developing confirmation of the significance of ideal vitamin K allow in carboxylation of bone proteins and complexation of calcium, to expand bone mass[52]. Particular proof to advance dietary admission is as yet missing, and pending the consequences of future trials, the present best counsel is to guarantee an eating regimen high in products of the soil to guarantee sufficient admissions of all vitamins and follow elements[53].

Conclusion

Micronutrients play a central part in metabolism and in the maintenance of tissue function. An adequate intake is therefore necessary but provision of excess supplements to people who do not need them may be harmful. Clinical benefit is most probable in those people who are severely depleted and at risk of complications, and is unlikely if this is not the case. Much more research are needed to characterise better markers of micronutrient status both in terms of metabolic effects and antioxidant effects, so that at risk patients can be identified and supplementation modified accordingly. Large scale trials of different doses of micronutrients

are required with precise outcome markers to optimise the intakes in different groups of patients as well as in the general population.

Corresponding author

Sreedevi Dharman

Senior Lecturer, Department of Oral Medicine

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162,Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Email ID: sanjamrut@gmail.com

Telephone Number: 9841009003

References

1. Evans P, Halliwell B. Micronutrients: oxidant/antioxidant status. Br J Nutr 2001;85:67–74.

2. Food and Nutrition Board IoM. Dietary reference intakes for thiamin, roboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin, and choline. Washington, DC: National Academy Press, 1998;13:58-86.

3. Food and Nutrition Board IoM. Dietary reference intakes for vitamin C, vitamin E, selenium, and carotenoids. Washington, DC: National Academy Press, 2000;9:186-324

4. Hoare J, Henderson L, Bates CJ. The national diet and nutrition survey:adults aged 19–64. London: The Stationery Office, 2004;9:523-530.

5. Finch S, Doyle W, Lowe C. National diet and nutrition survey: peopleaged 65 and over. London: The Stationery Office, 1998;98:133-134.

6. Weaver C. Adolescence:the period of dramatic bone growth. Endocrine 2002;17:43–8.

7. Shenkin A. Adult micronutrient requirements. In: Payne-James J, Grimble G, Silk D, eds. Artificial nutrition support in clinical practice. London: GMM, 2001;193–212.

8. Fang JC, Kinlay S, Beltrame J. Effect of vitamins C and E on progressionof transplant-associated arteriosclerosis: a randomised trial. Lancet2002;359:1108–13.

9. Heart Outcomes Prevention Evaluation Study Investigators. Effects oframipril on cardiovascular and microvascular outcomes in people with diabetes mellitus: results of the HOPE study and MICRO-HOPE substudy. Lancet 2000;355:253–9.

10.Gruppo Italiano per lo Studio della Sopravvivenza nell'Infarto miocardico. Dietary supplementation with n-3 polyunsaturated fatty acids and vitamin E after myocardial infarction: results of the GISSI-Prevenzione trial. Lancet 1999;354:447–55.

11. Heart Protection Study Collaborative Group. MRC/BHF heart protection study of antioxidant vitamin supplementation in 20,536 high-risk individuals: a randomised placebo-controlled trial. Lancet 2002;360:23–33.

12.Kris-Etherton PM, Lichtenstein AH, Howard BV. Antioxidant vitamin supplements and cardiovascular disease. Circulation 2004;110:637–41.

13.Miller ER III, Pastor-Barriuso R, Dalal D. Meta-analysis: high-dosage vitamin E supplementation may increase all-cause mortality. Ann Intern Med 2005;142:37–46.

14. American Institute for Cancer Research. Vitamin supplements don't help prevent cancer. Washington, DC: AICR,1999;15:523-6.

15. Li W, Zhu Y, Yan X. [The prevention of primary liver cancer by selenium in high risk populations]. Zhonghua Yu Fang Yi Xue Za Zhi 2000;34:336–8.

16.The effect of vitamin E and beta carotene on the incidence of lung cancer and other cancers in male smokers. The Alpha-Tocopherol, Beta Carotene Cancer Prevention Study Group. N Engl J Med 1994;330:1029–35.

17.Omenn GS, Goodman GE, Thornquist MD. Effects of a combination of beta carotene and vitamin A on lung cancer and cardiovascular disease.N Engl J Med 1996;334:1150–5.

18.Giovannucci E, Stampfer MJ, Colditz GA. Multivitamin use, folate, and colon cancer in women in the nurses' health study. Ann Intern Med 1998;129:517–24.

19.Jacobs EJ, Connell CJ, Patel AV. Multivitamin use and colon cancer mortality in the cancer prevention study II cohort (United States). Cancer Causes Control 2001;12:927–34.

20.Zhang SM, Giovannucci EL, Hunter DJ. Vitamin supplement use and the risk of non-Hodgkin's lymphoma among women and men. Am J Epidemiol 2001;153:1056–63.

21.Jacobs EJ, Henion AK, Briggs PJ. Vitamin C and vitamin E supplement use and bladder cancer mortality in a large cohort of US men and women. Am J Epidemiol 2002;156:1002–10.

22.Bjelakovic G, Nikolova D, Simonetti RG. Antioxidant supplements for prevention of gastrointestinal cancers: a systematic review and meta-analysis. Lancet 2004;364:1219–28.

23. Forman D, Altman D. Vitamins to prevent cancer: supplementary problems. Lancet 2004;364:1193–4.

24. Wright ME, Mayne ST, Stolzenberg-Solomon RZ. Development of a comprehensive dietary antioxidant index and application to lung cancer risk in a cohort of male smokers. Am J Epidemiol 2004;160:68–76.

25. Hercberg S, Galan P, Preziosi P. The SU.VI. MAX study: a randomized, placebo-controlled trial of the health effects of antioxidant vitamins and minerals, Arch Intern Med 2004;164:2335–42.

26. Meyer F, Galan P, Douville P. Antioxidant vitamin and mineral supplementation and prostate cancer prevention in the SU.VI.MAX trial. Int J Cancer 2005;116:182–6.

27. Age-Related Eye Disease Study Research Group. A randomized, placebo- controlled, clinical trial of high-dose supplementation with vitamins C and E and beta carotene for age-related cataract and vision loss: AREDS report no 9. Arch Ophthalmol 2001;119:1439–52.

28. Age-Related Eye Disease Study Research Group. A randomized, placebo- controlled, clinical trial of high-dose supplementation with vitamins C and E, beta carotene, and zinc for age-related macular degeneration and vision loss: AREDS report no 8. Arch Ophthalmol 2001;119:1417–36.

29. Black RE. Zinc deficiency, infectious disease and mortality in the developing world. J Nutr 2003;133:1485–9S.

30. Glasziou PP, Mackerras DE. Vitamin A supplementation in infectious diseases: a meta-analysis. BMJ 1993;306:366–70.

31.Rahman MM, Wahed MA, Fuchs GJ. Synergistic effect of zinc and vitamin A on the biochemical indexes of vitamin A nutrition in children. Am J Clin Nutr 2002;75:92–8.

32. Karyadi E, West CE, Schultink W. A double-blind, placebo-controlled study of vitamin A and zinc supplementation in persons with tuberculosis in Indonesia: effects on clinical response and nutritional status. Am J Clin Nutr 2002;75:720–7.

33. Sarma KV, Udaykumar P, Balakrishna N. Effect of micronutrient supplementation on health and nutritional status of schoolchildren: growth and morbidity. Nutrition 2006;22:S8–14.

34. Cunningham-Randles S. Trace element and mineral nutrition in HIV infection and AIDS. In: Bogden JD, Kleavey LM, eds. Clinical nutrition of the essential trace elements and minerals. Clifton: Humana Press, 2000:333–52.

35. Shenkin A. Trace elements and inflammatory response: implications for nutritional support. Nutrition 1995;11:100–5.

36. Conner EM, Grisham MB. Inflammation, free radicals, and antioxidants. Nutrition 1996;12:274–7.

37. Irlam JH, Visser ME, Rollins N. Micronutrient supplementation in children and adults with HIV infection. Cochrane Library. Chichester: Wiley, 2005;19:CD003650

38. Fawzi WW, Msamanga GI, Spiegelman D. A randomized trial of multivitamin supplements and HIV disease progression and mortality. N Engl J Med 2004;351:23–32.

39. Beck MA, Shi Q, Morris VC. Rapid genomic evolution of a non-virulent coxsackievirus B3 in selenium-deficient mice results in selection of identical virulent isolates. Nat Med 1995;1:433–6.

40. Beck MA, Levander OA, Handy J. Selenium deficiency and viral infection. J Nutr 2003;133:1463–7S.

41. Chandra RK. Effect of vitamin and trace-element supplementation on immune responses and infection in elderly subjects. Lancet 1992;340:1124–7.

42.Carpenter KJ, Roberts S, Sternberg S. Nutrition and immune function: a 1992 report. Lancet 2003;361:2247–8.

43. Girodon F, Galan P, Monget AL. Impact of trace elements and vitamin supplementation on immunity and infections in institutionalized elderly patients: a randomized controlled trial. MIN. VIT. AOX geriatric network. Arch Intern Med 1999;159:748–54.

44. Girodon F, Blache D, Monget AL. Effect of a two-year supplementation with low doses of antioxidant vitamins and/or minerals in elderly subjectson levels of nutrients and antioxidant defense parameters. J Am Coll Nutr 1997;16:357–65.

45. Allsup SJ, Shenkin A, Gosney MA. Can a short period of micronutrient supplementation in older institutionalized people improve response to influenza vaccine? A randomized, controlled trial. J Am Geriatr Soc 2004;52:20–4.

46.El Kadiki A, Sutton AJ. Role of multivitamins and mineral supplements in preventing infections in elderly people: systematic review and meta-analysis of randomised controlled trials. BMJ 2005;330:871–4.

47.Avenell A, Campbell MK, Cook JA. Effect of multivitamin and multimineral supplements on morbidity from infections in older people (MAVIS trial): pragmatic, randomised, double blind, placebo controlled trial. BMJ 2005;331:324–9.

48.Sub Group on Bone health CoMAoFP. Nutrition and bone health: with particular reference to calcium and vitamin D. Report on health and social subjects no 49. London: The Stationery Office, 1998;49:1-24.

49. Reid DM, New SA. Nutritional influences on bone mass. Proc Nutr Soc 1997;56:977–87.

50.Eaton-Evans J, McIlrath EM, Jackson EV. Copper supplementation and the maintenance of bone mineral density in middle-aged women. J Trace Elem Exp Biol 1996;9:87–94.

51. Strause L, Saltman P, Smith KT. Spinal bone loss in postmenopausal women supplemented with calcium and trace minerals. J Nutr 1994;124:1060–4.

52.Vermeer C, Shearer M, Zitterman A. Beyond deficiency:potential benefits of increased intakes of vitamin K for bone and vascular health. Eur J Nutr 2006;43:325–35.

53.Nieves J. Osteoporosis:the role of micronutrients. Am J Clin Nutr 2005;81:1232–9S.

Mineral Trioxide Aggregate versus Biodentin as Pulpotomy Agent in Paediatric Dentistry.

Preethi Pavithra.R [1], Ganesh Jeevanandan [2], Asha Ramesh [3]
Graduate student [1], Senior Lecturer [2],Department of Pedodontics
Senior Lecturer [3],Department of Periodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University,Chennai ,India

Abstract

The greatest threats to developing teeth especially in field of dentistry are dental caries and traumatic injury. A primary goal of all restorative treatment is to maintain pulp vitality so that normal root development.If pulpal exposure occurs, then a pulpotomy procedure aims to preserve pulp vitality to allow. Pulpotomy technique basically consists of removing the coronal pulp and fixing the radicular pulp with a medicament. It is the most widely accepted clinical procedure for treating primary teeth with coronal pulp inflammation caused by caries with no involvement of the radicular pulp. Vital pulpotomy is considered as a one-stage procedure with the objective of preserving the vitality, function of the remaining radicular portion of pulp and maintain it asymptomatic without adverse clinical signs or symptoms such as sensitivity, pain, or swelling,So the ideal requisites of any pulpotomy material should be bactericidal, harmless to pulp and surrounding structures, promote healing of remaining radicular pulp without interfering with the physiologic root resorption and not possess any toxicity.calcium hydroxide has been the material of choice for pulpotomy procedures. Recently, an alternative material called mineral trioxide aggregate (MTA) has demonstrated the ability to induce hard-tissue formation in pulpal tissue. The need for more and more new materials is never ending especially in the field of dentistry. Various materials have been formulated, tested and standardized to obtain maximum benefit for good clinical performance. latest bioactive calcium-silicate based material (biodentine),Mineral trioxide aggregate.Hence to analysis the difference between bioactive calcium-silicate based material (biodentine),Mineral trioxide aggregate this review is done.

Key Words: Pulpotomy,Mineral trioxide aggregate,Biodentin,Restoration,primary molars.

Introduction

The preservation of pulpally involved deciduous tooth in a healthy state until the time of normal exfoliation remains to be a great challenges for Pedodontists.Pulpotomy technique consists of removing the coronal pulp and fixing the radicular pulp with medicament. It is commonly usedclinical procedure to preserve the primary teeth with coronal pulp inflammation caused by caries with no involvement of the radicular pulp [1].Restorative materials compatible with physiologic root resorption that determine this choice.A pulpotomy is based on the concept that the inflammation and reduced vascularization, caused by bacterial invasion, are confined to the coronal pulp, while the root pulp remains vital an ideal pulpotomy material should be bactericidal, promote root pulp healing, provide a relatively stable environment for the dentin-pulp complex, stimulate dentin-complex regeneration and not disturb the physiologic process of root resorption [2]. A scientific noise has been generated about several materials some of which have been popular pulpotomy medicaments. Concerns have been raised about the toxicity and potential carcinogenicity of these materials, and alternatives have been proposed to maintain the partial pulp vitality[3].

Over the years, various types of dental materials have been developed to serve different purposes and to be applied to multiple scenarios in the field of dentistry. Mineral trioxide aggregate (MTA), was initially introduced as material for root perforation repair [4].It is primarily composed of tricalcium silicate, tricalciumaluminate, tricalcium oxide, and bismuth oxide [5]. It is formulated of fine hydrophilic particles that solidify in the presence of moisture or blood [6]. MTA was approved by the U.S. Food and Drug Administration for endodontic use in 1998 .Mineral trioxide aggregate (MTA) is a biocompatible material which provides a biological seal. MTA has been proposed as a potential medicament for various pulpal procedures like pulp capping with reversible pulpitis, apexification, repair of root perforations, etc [7] overcoming the disadvantages of MTA Biodentin has several advantages. Hence the present study was done to evaluate the efficacy of MTA and Biodentin as a pulpotomy medicament.

Mineral Trioxide Aggregate

Mineral trioxide aggregate (MTA), is commonly used material for root perforation repair. Over the years its use has expanded to include versatile applications in the field of pediatric dentistry [8]. MTA is a unique material with various advantages. It has been used successfully by pediatric dentists in a variety of clinical applications. However, its drawbacks especially its high cost, discoloration potential, difficulty in handling, and long setting time cannot be overlooked. Despite MTA,noveltricalcium silicate based materials that overcome MTA's key limitations, they are competing to be the next potential dentin substitutes for the various clinical application in which MTA has been used [9]. MTA is a trimineral aggregate, which is composed of tricalcium silicate, tricalicum aluminate, tricalcium oxide, silicate oxide and bismuth oxide material contains calcium oxide (50-75% by weight) and silicon dioxide (15–25%), then they are mixed, they result in the production of tricalcium silicate, dicalcium silicate, tricalcium aluminate and tetracalciumaluminoferrite [10].

The flexural strength is the material's capability to withstand deformation. It is highly recommended to place a wet cotton pellet on top of the placed MTA for the first 24 hours to increase the flexural strength then remove it to avoid the subsequent decrease in the flexural strength 72 hours after MTA receives moisture [11].

The push-out strength is the material's ability to withstand dislodgement. It is a crucial factor for MTA since soon after repair, the material can be dislodged by the tooth function. It was found that the push-out strength of MTA is lower than that of the intermediate restorative material (IRM) or Super ethoxybenzoic acid (EBA)[12].

Both the maximum compressive and push-out strength of MTA was found to be reached several days after mixing [13]. Its properties like sealing ability and marginal adaptation of MTA, was found to be better than the various other restorative materials used for root-end filling (attributed to the MTA expansion in its setting reaction [14]. The retentive strength of MTA, however, was found to be significantly inferior to GIC or zinc phosphate cement as a luting cement [15]. The AAPD recommended the use of MTA for pulpotomies of primary teeth with normal pulps or reversible pulpitis when caries removal results in pulp exposure or after a traumatic pulp

exposure.Clinical trials have revealed that MTA's performance is equal or superior to formocresol (FC), ferric sulfate, and might be considered as one of the favored pulpotomy materials.The introduction of MTA for pulpotomy overcame the drawbacks of FC such as its potential toxicity, caustic nature, and tissue irritation and inflammation upon contact with soft tissue.Farsi et al found that pulpotomized primary molars treated with MTA had significantly more success than those treated with FC [16].Sushynski et al conducted a randomized controlled trial and concluded that MTA demonstrated significantly better radiographic outcomes as a pulpotomy medicament compared to diluted FC which also exhibited a higher frequency of internal root resorption in the FC-treated molars. A recent systematic review and meta-analysis comparing MTA and FC used for primary molar pulpotomy, found that MTA was more effective and showed higher success rate than FC at six to 24-months follow-ups.However, Marghalani et al systematic review and meta-analysis comparing long-term (24 months) clinical and radiographic success of MTA and FC as a pulp-dressing material in pulpotomy treatment in primary molars showed comparable success rate [17].

Types of MTA

There are two basic forms of MTA based on color, gray (GMTA) and white (WMTA). MTA was initially presented as gray Pro-root MTA, however, the potential for discoloration of GMTA lead to the development of WMTA in 2002.

They principally differ in that WMTA was found to have lower quantities of iron, aluminum, and magnesium oxides than GMTA.The reduced ferrous oxide (FeO) was suggested by Asgary and colleagues to be the likely cause of the lighter color of WMTA. Other differences between WMTA and GMTA were reported in the literature. GMTA's crystal size was found to be about 8 times larger than WMTA's crystal size.GMTA also has significantly longer setting time; less radiopacity; solubility; and pH value than WMTA.Concerning the compressive strength, there are conflicting results with one study reporting significantly less compressive strength,while two other studies reported more compressive strength of WMTA compared to GMTA.Furthermore, regarding their antimicrobial behavior, it was reported that GMTA achieved similar antimicrobial action against particular microorganisms at lower concentrations than

WMTA.However, Asgary and Kamran,reported similar antibacterial properties for both types of MTA [18].

Other variations include incorporating resin in the MTA mix to be used as a root canal sealing cement. The purpose was to improve material flow, dentine bonding, and setting time and so to reduce micro-leakage. An example is the MTA Fillapex.However, it was found that adding resin to the materials results in the reduction of the desired free $Ca(OH)_2$ (essential for continued root formation in immature permanent teeth).

The NeoMTA (NuSmile, Huston, USA), on the other hand, is a pure MTA and does not contain resin. It is marketed as a cost effective MTA intended to be used for pediatric pulp therapy. The powder is provided in a lined vial that is re-sealable which facilitates the use of only the needed amount of MTA in each treatment thereby enhancing costeffectiveness. The liquid used for mixing is a gel which makes it easier to mix and apply. In addition, it has a non-staining formulation and a fast setting time. All these features are desirable while delivering treatment to the pediatric patient [19]. Another comparable material is the Neo MTA Plus ,it also possesses a powder-gel formula and is promoted to be used in pulpotomies because it does not stain the tooth structure by replacing bismuth oxide with tantalum oxide for opacity.

Biodentin

Calcium silicate based materials have gained popularity in recent years due to their resemblance to mineral trioxide aggregate (MTA) and their applicability in cases where MTA is indicated. Although various calcium silicate based products have been launched to the market recently, one of these has especially been the focus of attention and the topic of a variety of investigations. is material is the “Biodentine” calcium silicate based product which became commercially available in 2009 and that was specically designed as a “dentine replacement” material [20]. Biodentine has a wide range of applications including endodontic repair (root perforations, apexication, resorptivelesions, and retrograde lling material in endodontic surgery) and pulp capping and can be used as a dentine replacement material in restorative dentistry [21]. The

material is actually formulated using the MTA-based cement technology and the improvement of some properties of these typesofcements, such as physical qualities and handling

Composition of Biodentine states that the powder component of the material consists of tricalcium silicate, dicalcium silicate, calcium carbonate and oxide, iron oxide shade, and zirconium oxide [22]. Tricalcium silicate and dicalcium silicate are indicated as main and second core materials, respectively, whereas zirconium oxide serves as a radioopacity, on the other hand, contains calcium chloride as an accelerator and a hydrosoluble polymer that serves as a water reducing agent. It has also been stated that fast setting time, one unique characteristics of the product, is achieved by increasing particle size, adding calcium chloride to the liquid component, and decreasing the liquid content. The setting period of the material is as short as 9–12 minutes [23].It has shorter setting time is an improvement compared to other calcium silicate materials. Some authors have indicated that there are few studies on the properties of newly developed materials such as Biodentine.The material is characterized by the release of calcium when in solutionTricalcium silicate based materials are also de ned as a source of hydroxyapatite when they are in contact with synthetic tissue [24].

When materials' influences are to be evaluated in terms of pulpal response during vital procedures, in vivo study designs are helpful and animal and human teeth are generally preferred to demonstrate the effects of pulp capping agents [25].These should further be supported by clinical trials to establish a clear picture regarding the general characteristics of the materials. MTA, which is generally considered a gold standard, has been investigated in various human and animal experimental models. On the other hand, studies comparing MTA with Biodentine in terms of vital pulp treatment behavior are rather limited.study to demonstrate the induction of e effective dentinal repair was the one by Tran et al [26] where the material was applied directly on mechanically exposed rat pulps. In their study where Biodentine was compared to MTA and calcium hydroxide in terms of reparative dentine bridge formation, they noted that the structure induced by Ca(OH)2 contained several cell inclusions, also called tunnel defects as previously reported by Cox et al. in 1996.These defective regions were regarded as undesirable areas facilitating the migration of the microorganisms towards the pulp and predisposing the tooth to an endodontic infection. On the contrary, the dentine bridge formation induced by Biodentine

showed a pattern well-localized at the injury site unlike that caused by calcium hydroxide that exhibited an expanding structure in the pulp chamber. e quality of the formed dentine was also much more favorable compared to calcium hydroxide and an orthodentin organization was noted in which dentine tubules could be clearly visualized [27]. Moreover, cells secreting the structure well exhibited DSP expression as well as osteopontin expression, which are critical regulators of reparative dentine formation.

Pulpotomy is another vital pulp treatment method in which Biodentine is advocated to be used. is method is widely used in pediatric dentistry and involves the amputation of pulp chamber and the placement of a material for the preservation of the radicular pulp tissue's vitality. is methodology is specifically useful and preferred when the coronal pulp tissue is mainly involved and a direct pulp capping is not a suitable option [28].

Comparison between MTA and Biodentin

The placement of a root-end filling material during periapical surgery is a procedure of paramount importance to seal the root canal.Several materials have been tested as root-end filling materials, but none of them has been demonstrated to be free from limitation [29].Mineral trioxide aggregate (MTA), a calcium silicate-based endodontic material possesses excellent biocompatibility and sealing ability; thus, it is considered a promising material for various endodontic applications such as pulp capping, pulpotomy,apical barrier formation in teeth with open apexes, repair of root perforations, and as a root canal filling material, besides a root-end filling material.Despite its good physical and biologic properties, some clinicians still claim to have difficulties in handling this material after its preparation to fill a retroprepared root cavity.Several new calcium silicate-based materials have recently been developed, aiming to improve some MTA drawbacks such as its difficult handling property and long-setting time (ProRoot MTA) [30]. Biodentineis among these materials and is claimed to be used as a dentine restorative material in addition to endodontic indications similar to those of MTA.

Compressive Srength of MTA and Biodentin

Biodentine has more prominent biomineralization ability than MTA, with wider calcium and silicon rich layer at material-dentine interface. The setting time is one of the most clinically relevant factors. A long-setting duration may cause clinical problems because of the cement's inability to maintain shape and support stresses during this period.The setting time of MTA-Angelus mixed with water was 8.5 min [31]. In contrast, Biodentine exhibited shorter setting time (6.5 min) though not statistically significant. Accelerated setting reduces the risk of dislodgement and contamination of MTA-like cements when used as root-end filling material.An important feature of a root-end filling material is its handling property [32]. MTA-Angelus is grainy and has a poor consistency, making it difficult to manipulate in clinical situations. In contrast, Biodentine was relatively easier to handle and on thorough amalgamation it rolled into a dough-like consistency that could be easily condensed. Compressive strength is an indicator of the setting process and strength of the material.According to the results of this study, the compressive strength of MTA-Angelus was 41 MPa at 24 h that increased to 76.8 MPa at 28 days. These results are similar to those reported by Torabinejad et al. (40 MPa at 24 h and 67.3 MPa after 21 days). Several studies have reported modifications of MTA to overcome its shortcomings and improve its physical properties.Biodentine exhibited compressive strength of 170 MPa at 24 h that increased substantially to 304 MPa after the material was placed in moisture for 28 days. This value of compressive strength is close to that reported for human dentine (297 ± 24 MPa).The compressive strength of Biodentine was significantly higher than that of MTA at all time intervals. These factors reduce the chances of bacterial contamination. Biodentine like MTA has an ability to initiate and continue the mineralization process. Biodentine has a limitation that it cannot be used in presence of moisture unlike MTA [33].

Biocompatibility of MTA and Biodentin

Biocompatibility of a dental material is a major factor that should be taken into consideration specifically when it is used in pulp capping, perforation repair or as a retrograde filling. During the aforementioned procedures, the material is in direct contact with the connective tissue and has the potential to affect the viability of periradicular and pulpal cells. Cell death under these

circumstances occurs due to apoptosis or necrosis [34]. Therefore, it is essential that toxic materials are avoided and materials promoting repair or that are biologically neutral are preferred during procedures in which the material is directly in contact with the surrounding tissue [35]. Though the information accumulated so far regarding the biocompatibility of Biodentine is rather limited, the available data generally is in favor of the material in terms of its lack of cytotoxicity and tissue acceptability. Han and Okijicompared Biodentine and white ProRoot MTA in terms of Ca and Si uptake by adjacent root canal dentine and observed that both materials formed tag-like structures. They observed that dentine element uptake was more prominent for Biodentine than MTA.

Comparative Strength of MTA and Biodentin

The high mechanical strength of Biodentine may be attributed to the elimination of aluminates that leads to weakening and fragility of the set material as reported by manufacturer. With physical properties superior to those of MTA, especially in terms of setting time and compressive strength, it exhibits the same characteristics of sealing ability, with controlled (size and spatial organization) formation of calcium salts.

Bond Strength

Considering that Biodentine is recommended for use as a dentine substitute under permanent restorations, studies were performed that assess the bond strength of the material with di erent bonding systems. Odaba ş et al. evaluated the shear bond strength of an etch-and-rinse adhesive, a 2-step self-etch adhesive and a 1-step self-etch adhesive system to Biodentine at di erent intervals. No signicant di erences were found between all of the adhesive groups at the same time intervals (12 minutes and 24 hours). When different time intervals were compared, the lowest bonding value was obtained for the etch-and- rinse adhesive at a 12-minute period, whereas the highest was obtained for the 2-step self-etch adhesive at the 24-hour period [36].

Another area of use of Biodentine, specicallyfrom an endodontic point of view, is the repair of perforations, which is likely to be encountered in clinical practice. It is essential that a

perforation repair material should have sufficient amount of push-out bond strength with dentinal walls for the prevention of dislodgement from the repair site [36].

Radiographic Feature of MTA and Biodentin

Radiopacity is an important property expected from a retrograde or repair material as these materials are generally applied in low thicknesses and they need to be easily discerned from surrounding tissues.On radiographic observance, it was noted that initial periapical healing was better in tooth filled with biodentine. However, long-term periapical healing of MTA filled tooth was better. This may due to the fact that MTA has superior marginal adaptation.Superior marginal adaptation of MTA over biodentine may influence sealing ability and clinical success.It is important to note that quality of healing was assessed purely on radiographic changes on monthly basis [37].

Discussion

The main disadvantages of MTA include its high cost, which has been one of its major limitation, its discoloration potential, difficulties in handling the material, prolonged setting time, presence of toxic elements in its composition, difficulty to remove after setting, and it does not have a solvent .Unfortunately, the use of MTA as a dressing for pulpotomies in molars for filling pulp chambers of immature teeth and as an apical barrier has resulted in the discoloration of the crown. The reason of discoloration is debatable, but it has been linked mainly to the interaction between bismuth oxide and the collagen of the tooth tissue and sodium hypochlorite, which is usually used in root canal therapy

In concern of the toxic elements in MTA, mainly in its arsenic content several investigations were conducted and found that the amount of arsenic released is very low.In addition, other factors further minimize the arsenic release from the material into the tissue, such a s the small amounts of the MTA that are actually used clinically, its insoluble nature and the FeO present in its composition with its subsequent stabilizing effect on the arsenic content [38].

In an effort to overcome these disadvantages other tricalcium silicate based materials have been introduced to the market. Bioaggregate (Verio Dental, Vancouver, Canada) and Biodentine (Septodont, Saint-Maur-des-Foss es, France) are tricalcium silicate based materials that have been developed to overcome the drawbacks of MTA while maintaining its desired characteristics and clinical applications.Compared to MTA, Biodentine is a cost effective material that is easier to handle, has a faster setting time (set at about 10-12 minutes), is less soluble, and results in a better seal. It also has alternative radiopacifiers to overcome the discoloration related to bismuth oxide present in MTA [39].

In a study to assess the color stability of MTA Plus compared to Neo MTA plus and Biodentine when used in pulpotomies in immature permanent teeth in the presence of sodium hypochlorite solution, it was found that MTA Plus showed discoloration while Neo MTA Plus and Biodentine did not show discoloration [40].

Conclusion

Vital pulpotomy is considered as a one-stage procedure with the objective of preserving the vitality, function of the remaining radicular portion of pulp and maintain it asymptomatic without adverse clinical signs or symptoms such as sensitivity, pain, or swelling. So the ideal requisites of any pulpotomy material should be bactericidal, harmless to pulp and surrounding structures, promote healing of remaining radicular pulp without interfering with the physiologic root resorption and not possess any toxicity.It may be concluded the sealing quality of Biodentine and commercially available MTA cement (MTA-Angelus) is comparable. The enhancement in handling properties of Biodentine may make it more convenient for use in various clinical applications.

MTA is a unique material with various advantages. It has been used successfully by pediatric dentists in a variety of clinical applications. Despite the many advantages of MTA, its drawbacks especially its high cost, discoloration potential, difficulty in handling, and long setting time cannot be overlooked. With the emergence of other novel tricalcium silicate based materials in

the market, such as Biodentine, that overcome MTA's key limitations, they are competing to be the next potential dentin substitute for the various clinical application in which MTA has been used. However, with the recent introduction of new improved MTA products, MTA-based materials are likely to remain at the heart of good pediatric dental practice for many years to come.

Corresponding author

Asha Ramesh

Senior Lecturer,Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University

Chennai-600077

Mobile: +91 9841722113.

Email: doc.ash4587@gmail.com.

Reference

1. Niranjani K, Prasad MG, Vasa AA, Divya G, Thakur MS, Saujanya K. Clinical Evaluation of Success of Primary Teeth Pulpotomy Using Mineral Trioxide Aggregate®, Laser and BiodentineTM-an In Vivo Study. Journal of clinical and diagnostic research: JCDR. 2015 Apr;9(4):ZC35.
2. Nathan JE. Behavioral management strategies for young pediatric dental patients with disabilities. Journal of Dentistry for Children. 2001 Mar 1;68(2):89-101.
3. Malkondu Ö, Kazandağ MK, Kazazoğlu E. A review on biodentine, a contemporary dentine replacement and repair material. BioMed research international. 2014;2014.
4. Zhou HM, Shen Y, Wang ZJ, Li L, Zheng YF, Häkkinen L, Haapasalo M. In vitro cytotoxicity evaluation of a novel root repair material. Journal of endodontics. 2013 Apr 1;39(4):478-83.

5. Han L, Okiji T. Uptake of calcium and silicon released from calcium silicate–based endodontic materials into root canal dentine. International endodontic journal. 2011 Dec 1;44(12):1081-7.
6. Luo Z, Li D, Kohli MR, Yu Q, Kim S, He WX. Effect of Biodentine™ on the proliferation, migration and adhesion of human dental pulp stem cells. Journal of dentistry. 2014 Apr 1;42(4):490-7.
7. Duarte MA, de Oliveira Demarchi AC, Yamashita JC, Kuga MC, de Campos Fraga S. Arsenic release provided by MTA and Portland cement. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics. 2005 May 1;99(5):648-50.
8. De-Deus G, de Souza MC, Fidel RA, Fidel SR, de Campos RC, Luna AS. Negligible expression of arsenic in some commercially available brands of Portland cement and mineral trioxide aggregate. Journal of endodontics. 2009 Jun 1;35(6):887-90.
9. Mortazavi M, Mesbahi M. Comparison of zinc oxide and eugenol, and Vitapex for root canal treatment of necrotic primary teeth. International Journal of Paediatric Dentistry. 2004 Nov 1;14(6):417-24.
10. Jeevanandan G. Obturation of a Retained Primary Maxillary Second Molar Using BiodentineTM: A Case Report. Journal of clinical and diagnostic research: JCDR. 2017 Jan;11(1):ZD34.
11. Nayak G, Hasan MF. Biodentine-a novel dentinal substitute for single visit apexification. Restorative dentistry & endodontics. 2014 May 1;39(2):120-5.
12. Aminozarbian MG, Barati M, Salehi I, Mousavi SB. Biocompatibility of mineral trioxide aggregate and three new endodontic cements: An animal study. Dental research journal. 2012 Jan;9(1):54.
13. Walker MP, Diliberto A, Lee C. Effect of setting conditions on mineral trioxide aggregate flexural strength. Journal of endodontics. 2006 Apr 1;32(4):334-6.
14. Gancedo-Caravia L, Garcia-Barbero E. Influence of humidity and setting time on the push-out strength of mineral trioxide aggregate obturations. Journal of endodontics. 2006 Sep 1;32(9):894-6.

15. Valois CR, Costa ED. Influence of the thickness of mineral trioxide aggregate on sealing ability of root-end fillings in vitro. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology and Endodontics. 2004 Jan 1;97(1):108-11.
16. Danesh G, Dammaschke T, Gerth HU, Zandbiglari T, Schäfer E. A comparative study of selected properties of ProRoot mineral trioxide aggregate and two Portland cements. International Endodontic Journal. 2006 Mar 1;39(3):213-9.
17. Shie MY, Huang TH, Kao CT, Huang CH, Ding SJ. The effect of a physiologic solution pH on properties of white mineral trioxide aggregate. Journal of endodontics. 2009 Jan 1;35(1):98-101.
18. Fridland M, Rosado R. Mineral trioxide aggregate (MTA) solubility and porosity with different water-to-powder ratios. Journal of endodontics. 2003 Dec 1;29(12):814-7.
19. Hachmeister DR, Schindler WG, Walker WA, Thomas DD. The sealing ability and retention characteristics of mineral trioxide aggregate in a model of apexification. Journal of Endodontics. 2002 May 1; 28(5):386-90.
20. Padavala S, Aarthy MG. MTA-An Elixir for Root Perforations-A Short Review. Research Journal of Pharmacy and Technology. 2017;10(5):1577-9.
21. Arruda RA, Cunha RS, Miguita KB, Silveira CF, De Martin AS, Pinheiro SL, Rocha DG, Bueno CE. Sealing ability of mineral trioxide aggregate (MTA) combined with distilled water, chlorhexidine, and doxycycline. Journal of oral science. 2012;54(3):233-9.
22. Dorileo MC, Bandeca MC, Pedro FL, Volpato LE, Guedes OA, Villa RD, Tonetto MR, Borges AH. Analysis of metal contents in Portland Type V and MTA-based cements. The Scientific World Journal. 2014;2014.
23. De Bruyne MA, De Bruyne RJ, De Moor RJ. Capillary flow porometry to assess the seal provided by root-end filling materials in a standardized and reproducible way. Journal of Endodontics. 2006 Mar 1;32(3):206-9.
24. Subramanyam D, Vasantharajan M. Effect of Oral Tissue Fluids on Compressive Strength of MTA and Biodentine: An In vitro Study. Journal of clinical and diagnostic research: JCDR. 2017 Apr;11(4):ZC94.

25. Camilleri J, Grech L, Galea K, Keir D, Fenech M, Formosa L, Damidot D, Mallia B. Porosity and root dentine to material interface assessment of calcium silicate-based root-end filling materials. Clinical oral investigations. 2014 Jun 1;18(5):1437-46.
26. Mortazavi M, Mesbahi M. Comparison of zinc oxide and eugenol, and Vitapex for root canal treatment of necrotic primary teeth. International Journal of Paediatric Dentistry. 2004 Nov 1;14(6):417-24.
27. Gjorgievska ES, Nicholson JW, Apostolska SM, Coleman NJ, Booth SE, Slipper IJ, Mladenov MI. Interfacial properties of three different bioactive dentine substitutes. Microscopy and Microanalysis. 2013 Dec;19(6):1450-7.

28. 28)Atmeh AR, Chong EZ, Richard G, Festy F, Watson TF. Dentin-cement interfacial interaction: calcium silicates and polyalkenoates. Journal of dental research. 2012 May;91(5):454-9.
29. O'sullivan SM, Hartwell GR. Obturation of a retained primary mandibular second molar using mineral trioxide aggregate: a case report. Journal of Endodontics. 2001 Nov 1;27(11):703-5.
30. Tagger M, Katz A. Radiopacity of endodontic sealers: development of a new method for direct measurement. Journal of Endodontics. 2003 Nov 1;29(11):751-5.
31. Bolla N, Naik BD, Kavuri SR, Velagala LD. Obturation of a retained primary mandibular second molar with missing successor using gutta-percha: A case report. J Indian Dent Assoc. 2011;5:194-5.
32. Malkondu Ö, Kazandağ MK, Kazazoğlu E. A review on biodentine, a contemporary dentine replacement and repair material. BioMed research international. 2014;56-59.
33. Wang X, Sun H, Chang J. Characterization of Ca3SiO5/CaCl2 composite cement for dental application. dental materials. 2008 Jan 1;24(1):74-82.
34. Doyon GE, Dumsha T, von Fraunhofer JA. Fracture resistance of human root dentin exposed to intracanal calcium hydroxide. Journal of endodontics. 2005 Dec 1;31(12):895-7.

35. Soares CJ, Santana FR, Silva NR, Preira JC, Pereira CA. Influence of the endodontic treatment on mechanical properties of root dentin. Journal of Endodontics. 2007 May 1;33(5):603-6.
36. Leiendecker AP, Qi YP, Sawyer AN, Niu LN, Agee KA, Loushine RJ, Weller RN, Pashley DH, Tay FR. Effects of calcium silicate–based materials on collagen matrix integrity of mineralized dentin. Journal of endodontics. 2012 Jun 1;38(6):829-33.
37. Kubota K, Golden BE, Penugonda B. Root canal filling materials for primary teeth: a review of the literature. ASDC journal of dentistry for children. 1992;59(3):225-7.
38. Nowicka A, Lipski M, Parafiniuk M, Sporniak-Tutak K, Lichota D, Kosierkiewicz A, Kaczmarek W, Buczkowska-Radlińska J. Response of human dental pulp capped with biodentine and mineral trioxide aggregate. Journal of endodontics. 2013 Jun 1;39(6):743-7.
39. Zhou HM, Shen Y, Wang ZJ, Li L, Zheng YF, Häkkinen L, Haapasalo M. In vitro cytotoxicity evaluation of a novel root repair material. Journal of endodontics. 2013 Apr 1;39(4):478-83.
40. Han L, Okiji T. Uptake of calcium and silicon released from calcium silicate–based endodontic materials into root canal dentine. International endodontic journal. 2011 Dec 1;44(12):1081-7.

Muscle Dystrophy - Review

Abiraamasri BL[1], DhanalakshmiRavikumar[2]

Graduate Student[1], Senior Lecturer[2,] Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Muscular dystrophy (MD) is a group of muscle diseases that weaken the musculoskeletal system and hamper locomotion. Dystrophies are characterized by progressive skeletal muscle weakness, defects in muscle proteins, and the death of muscle cells and tissue. In the 1860s, descriptions of boys who grew progressively weaker, lost the ability to walk, and died at an early age became more prominent in medical journals. In the following decade, French neurologist Guillaume Duchenne gave a comprehensive account of thirteen boys with the most common and severe form of the disease, which now carries his name—Duchenne muscular dystrophy (DMD). It soon became evident that the disease had more than one form. The other major forms are Becker, limb-girdle, congenital, facioscapulohumeral, myotonic, oculopharyngeal, distal, and Emery-Dreifuss muscular dystrophy. The main cause of the Duchenne and Becker types of muscular dystrophy is the muscle tissue's cytoskeletal impairment to properly create the functional protein dystrophin and dystrophin-associated protein complex.

Key words: *Muscular dystrophy, musculoskeletal system, Duchenne muscular dystrophy, cytoskeletal impairment, muscle cell death.*

Introduction

Muscular dystrophy (MD) is a group of muscle diseases that weaken the musculoskeletal system and hamper locomotion.[1,2] Dystrophies are characterized by progressive skeletal muscle weakness, defects in muscle proteins, and the death of muscle cells and tissue.[3]In the 1860s, descriptions of boys who grew progressively weaker, lost the ability to walk, and died at an early age became more prominent in medical journals. In the following decade, French neurologist Guillaume Duchenne gave a comprehensive account of thirteen boys with the most common and severe form of the disease, which now carries his name—Duchenne muscular dystrophy (DMD).It soon became evident that the disease had more than one form. The other major forms are Becker, limb-girdle, congenital, facioscapulohumeral, myotonic, oculopharyngeal, distal, and Emery-Dreifuss muscular dystrophy.[4] Duchenne and Becker muscular dystrophies, being caused by a mutation of a gene located on the X chromosome, predominantly affect males, although females can sometimes have severe symptoms as well. Most types of MD are multi-system disorders with manifestations in body systems including the heart, gastrointestinal system, nervous system, endocrine glands, eyes and brain.[4]Apart from the nine major types of muscular dystrophy listed above, several MD-like conditions have also been identified. Normal intellectual, muscular, behavioral, bowel and sexual function is noticed in individuals with other forms of MD and MD-like conditions.[5,6] Children with muscular dystrophy require complex and long-term health services in multiple settings by a variety of professionals. To protect and promote the well being of this population, it is important to assess and monitor their receipt of care. MD-affected individuals with susceptible intellectual impairment are diagnosed through molecular characteristics but not through problems associated with disability.[7] However, a third of patients who are severely affected with DMD may have cognitive impairment, behavioral, vision and speech problems.[8,9]. This review explains in detain about the individual muscle dystrophy and the gene involved in it.

Signs and Symptoms

Progressive muscular wasting

Poor balance

Drooping eyelids

Atrophy

Scoliosis (curvature of the spine and the back)
Inability to walk
Frequent falls
Waddling gait
Calf deformation
Limited range of movement
Respiratory difficulty
Joint contractures
Cardiomyopathy
Arrhythmias
Muscle spasms
Gowers' sign [10]

Etiology

These conditions are generally inherited, and the different muscular dystrophies follow various inheritance patterns. However, mutations of the dystrophin gene and nutritional defects (with no genetics history) at the prenatal stage are also possible in about 33% of people affected by DMD.[11] The main cause of the Duchenne and Becker types of muscular dystrophy is the muscle tissue's cytoskeletal impairment to properly create the functional protein dystrophin and dystrophin-associated protein complex.

Dystrophin protein is found in muscle fibre membrane; its helical nature allows it to act like a spring or shock absorber. Dystrophin links actin (cytoskeleton) and dystroglycans of the muscle cell plasma membrane, known as the sarcolemma (extracellular). In addition to mechanical stabilization, dystrophin also regulates calcium levels.[11]

Diagnosis

Histopathology of gastrocnemius muscle from patient who died of pseudohypertrophic muscular dystrophy, Duchenne type. Cross section of muscle shows extensive replacement of muscle fibers by adipose cells.The diagnosis of muscular dystrophy is based on the results of muscle biopsy, increased creatine phosphokinase (CpK3), electromyography, electrocardiography and

DNA analysis.A physical examination and the patient's medical history will help the doctor determine the type of muscular dystrophy. Different types of muscular dystrophy affect specific muscle groups.

Often, there is a loss of muscle mass (wasting), which may be hard to see because some types of muscular dystrophy cause a buildup of fat and connective tissue that makes the muscle appear larger. This is called pseudo hypertrophy.

Management

There is no known cure for muscular dystrophy, although significant headway is being made with antisense oligonucleotides.[12] Physical therapy, occupational therapy, orthotic intervention (e.g., ankle-foot orthosis), speech therapy and orthopedic instruments (e.g., wheelchairs, standing frames and powered mobile arm supports) may be helpful. Inactivity (such as bed rest, sitting for long periods) and bodybuilding efforts to increase myofibrillar hypertrophy can worsen the disease.There is no specific treatment for any of the forms of muscular dystrophy. Physiotherapy, aerobic exercise, low intensity anabolic steroids, prednisone supplements may help to prevent contractures and maintain muscle tone. Orthoses (orthopedic appliances used for support) and corrective orthopedic surgery may be needed to improve the quality of life in some cases. The cardiac problems that occur with Emery-Dreifuss muscular dystrophy and myotonic muscular dystrophy may require a pacemaker. The myotonia (delayed relaxation of a muscle after a strong contraction) occurring in myotonic muscular dystrophy may be treated with medications such as quinine, phenytoin, or mexiletine, but no actual long-term treatment has been found.Occupational therapy assists the individual with MD to engage in activities of daily living (such as self-feeding and self-care activities) and leisure activities at the most independent level possible. This may be achieved with use of adaptive equipment or the use of energy conservation techniques. Occupational therapy may implement changes to a person's environment, both at home or work, to increase the individual's function and accessibility. Occupational therapists also address psychosocial changes and cognitive decline, which may accompany MD, as well as provide support and education about the disease to the family and individual. [13]High dietary intake of lean meat, seafood, pulses, olive oil, antioxidants such as leafy vegetables and bell peppers, and fruits like blueberry and cherry is advised. Decreased intake of refined food, trans

fats, and caffeinated and alcoholic beverages is also advised, as is a check for any food allergies.[14]

After diagnosis, medical care may include services in neurology, nutrition, gastroenterology, respiratory care, cardiac care, orthopedics, psychosocial, rehabilitation, and oral care.

Prognosis

The prognosis for people with muscular dystrophy varies according to the type and progression of the disorder. Some cases may be mild and progress very slowly over a normal lifespan, while others produce severe muscle weakness, functional disability, and loss of the ability to walk. Some children with muscular dystrophy die in infancy while others live into adulthood with only moderate disability. The muscles affected vary, but can be around the pelvis, shoulder, face or elsewhere. Muscular dystrophy can affect adults, but the more severe forms tend to occur in early childhood.

Types

Gene Description

Becker Muscular Dystrophy 300376 BMD

Becker muscular dystrophy (BMD) is a less severe variant of Duchenne muscular dystrophy and is caused by the production of a truncated, but partially functional form of dystrophin.[4] Survival is usually into old age.[15] Affects only boys (with extremely rare exceptions)

Congenital Muscular Dystrophy

Multiple at the age of onset: symptoms include general muscle weakness and possible joint deformities; disease progresses slowly; shortened life span.[16]

Congenital muscular dystrophy includes several disorders with a range of symptoms. Muscle degeneration may be mild or severe. Problems may be restricted to skeletal muscle, or muscle degeneration may be paired with effects on the brain and other organ systems. A number of the forms of the congenital muscular dystrophies are caused by defects in proteins that are thought to have some relationship to the dystrophin-glycoprotein complex and to the connections between

muscle cells and their surrounding cellular structure. Some forms of congenital muscular dystrophy show severe brain malformations, such as lissencephaly and hydrocephalus.[4]

Duchenne Muscular Dystrophy 310200 DMD

Duchenne muscular dystrophy (DMD) is the most common childhood form of muscular dystrophy; it generally affects only boys (with extremely rare exceptions), becoming clinically evident when a child begins walking. By age 10, the child may need braces for walking and by age 12, most patients are unable to walk.[17] Life span ranges from 15 to 51.[18] In the early 1990s, researchers identified the gene for the protein dystrophin which, when absent, causes DMD. The amount of dystrophin correlates with the severity of the disease (i.e., the less dystrophin present, the more severe the phenotype). Since the gene is on the X chromosome, this disorder affects primarily males, and females who are carriers have milder symptoms. Sporadic mutations in this gene occur frequently, accounting for a third of cases. The remaining two-thirds of cases are inherited in a recessive pattern.

Dystrophin is part of a complex structure involving several other protein components. The "dystrophin-glycoprotein complex" helps anchor the structural skeleton (cytoskeleton) within the muscle cells, through the outer membrane (sarcolemma) of each cell, to the tissue framework (extracellular matrix) that surrounds each cell. Due to defects in this assembly, contraction of the muscle leads to disruption of the outer membrane of the muscle cells and eventual weakening and wasting of the muscle [18]. The lower prevalence in 2006–2010 may reflect delayed diagnosis of DBMD among cases. It also may reflect a change in parental reproductive choice; however, a separate analysis observed that reproductive patterns of mothers after knowledge of family history of DBMD tended to be similar to those without such family history. The similar estimates for all cases and non-Hispanic white cases reflect the sizable proportion of non-Hispanic white individuals in the future efforts will include expanding surveillance among minority populations. It also may reflect racial/ethnic differences in time to diagnosis and access to care, random fluctuations, or true differences in prevalence of DBMD across these racial/ethnic groups. Conversely, the increased prevalence among Hispanic individuals may reflect suspected underreporting in census estimates for Hispanic children, ages 5 to 9 years. Additionally, our definitions for DMD and BMD, determined from age of onset of signs and

symptoms, may have also biased the true prevalence of each phenotype. Duchene and Becker muscular dystrophy (DBMD) may occur in multiple sons within a family and is associated with variable manifestations among brothers. Common medical complications include loss of ambulation, scoliosis, and cardiomyopathy.

Birnkrant analyzed pulmonary and cardiac function in sibling pairs with Duchenne and Becker muscular dystrophies and found discordant pulmonary outcome among 3 of 7 pairs and discordant cardiac outcome in 3 of 6. They concluded that this variation could have implications for using genotype information to predict the clinical course of DBMD and response to treatments. The observed variability in expression between brothers with the same mutation underscores this point. Although the clinical outcome of older brothers predicted those of their younger brothers for loss of ambulation, we did not find it to be predictive for development of scoliosis or cardiomyopathy in this dataset with the small sample sizes for these milestones. Furthermore, there were sibships for which the differences in reaching clinical milestones were striking, as much as a 6 year, 9 month difference for ceased ambulation, a 3 year 10 month difference for scoliosis ≥ 20°, and an 11 year 11 month difference for cardiomyopathy. This natural variation in clinical outcome among boys with identical dystrophin gene mutations and similar modifying gene profiles suggests that trials of therapeutic interventions will likely require a large number of subjects to detect a significant effect size.

Distal Muscular Dystrophy 254130 DYSF

Distal muscular dystrophies' age at onset: 20 to 60 years; symptoms include weakness and wasting of muscles of the hands, forearms, and lower legs; progress is slow and not life-threatening.[15]

Miyoshi myopathy, one of the distal muscular dystrophies, causes initial weakness in the calf muscles, and is caused by defects in the same gene responsible for one form of LGMD (Limb Girdle Muscular Dystrophy).[4]

Emery-DreifussMuscular Dystrophy 310300, 181350 EMD

Emery-Dreifuss Muscular Dystrophy patients normally present in childhood and the early teenage years with contractures. Clinical signs include muscle weakness and wasting, starting in

the distal limb muscles and progressing to involve the limb-girdle muscles. Most patients also suffer from cardiac conduction defects and arrhythmias that, if left untreated, increase the risk of stroke and sudden death.

There are three subtypes of Emery-Dreifuss Muscular Dystrophy, distinguishable by their pattern of inheritance: X-Linked, autosomal dominant and autosomal recessive. The X-linked form is the most common. Each type varies in prevalence and symptoms. The disease is caused by mutations in the LMNA gene, or more commonly, the EMD gene. Both genes encode for protein components of the nuclear envelope. However, how these mutations cause the pathogenesis is not well understood.[18]

FacioscapulohumeralMuscular Dystrophy 158900 DUX4 FSHD

Facioscapulohumeral muscular dystrophy (FSHD) initially affects the muscles of the face, shoulders, and upper arms with progressive weakness. Symptoms usually develop in the teenage years. Some affected individuals become severely disabled. The pattern of inheritance is autosomal dominant, but there are a significant number of spontaneous mutations. Seminal research published in August 2010 documents that two defects are needed for FSHD, which for the first time provides a unifying theory for the underlying genetics of FSHD. The first is the deletion of D4Z4 repeats and the second is a "toxic gain of function" of the DUX4 gene.[4, 19-20]Facioscapulohumeral muscular dystrophy (FSHD) occurs both in males and females.

Limb-Girdle Muscular Dystrophy

Limb-girdle muscular dystrophy is also called LGMD. Affects both boys and girls. LGMDs all show a similar distribution of muscle weakness, affecting both upper arms and legs. Many forms of LGMD have been identified, showing different patterns of inheritance (autosomal recessive vs. autosomal dominant). In an autosomal recessive pattern of inheritance, an individual receives two copies of the defective gene, one from each parent. The recessive LGMDs are more frequent than the dominant forms, and usually have childhood or teenage onset. The dominant LGMDs usually show adult onset. Some of the recessive forms have been associated with defects in proteins that make up the dystrophin-glycoprotein complex.[4] Though a person normally leads a normal life with some assistance, in some extreme cases, death from LGMD occurs due to cardiopulmonary complications.[21]

MyotonicMuscular Dystrophy 160900, 602668 DMPK, ZNF9

Myotonic muscular dystrophy is an autosomal dominant condition that presents with myotonia (delayed relaxation of muscles) as well as muscle wasting and weakness.[22] Myotonic dystrophy varies in severity and manifestations and affects many body systems in addition to skeletal muscles, including the heart, endocrine organs, eyes, and gastrointestinal tract.

Myotonic muscular dystrophy type 1 (DM1), also known as Steinert disease, is the most common adult form of muscular dystrophy. It results from the expansion of a short (CTG) repeat in the DNA sequence of the DMPK (myotonic dystrophy protein kinase) gene. Myotonic muscular dystrophy type 2 (DM2) is much rarer and is a result of the expansion of the CCTG repeat in the ZNF9 (zinc finger protein 9) gene. While the exact mechanisms of action are not known, these molecular changes may interfere with the production of important muscle proteins.[23]

Oculopharyngeal Muscular Dystrophy 164300 Pabpn1

Oculopharyngeal MD's age at onset: 40 to 70 years; symptoms affect muscles of eyelids, face, and throat followed by pelvic and shoulder muscle weakness, has been attributed to a short repeat expansion in the genome which regulates the translation of some genes into functional proteins.[24]

Lijing Quyang 2012, concluded that among children with special health care needs, those children having muscular dystrophy have lower family socioeconomic status than children without muscular dystrophy and also had more negative health care experiences. The negative healthcare experiences appear to be explained by the association with lower socioeconomic status. Adjusting for multiple factors, muscular dystrophy was not associated with lower likelihood of a medical home. Children with muscular dystrophy incurred substantially more negative financial and employment impacts on family members than did other children in the survey, independent of household socio-demographic characteristics. There is a need to identify effective strategies to help families and caregivers cope with muscular dystrophy [25].

Conclusion

This review provides a brief insight on the etiology, types, genes involved, management and prognosis of muscle dystrophies. Genes explained in detail helps the clinician to arrive at an appropriate diagnosis.

Corresponding Author

Dhanalakshmi Ravikumar,

Senior Lecturer,Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai-600077, Tamil Nadu, India

Email id: dhana9677@gmail.com

Phone Number:+91 9597011442.

Reference

1. R H Brown Jr., J R Mendell. Harrison's Principles of Internal Medicine. 200;17; 2527.
2. Muscular Dystrophy Campaign Retrieved 9 April. Emery AE. "The muscular dystrophies". Lancet2002;02; 687–695.
3. Motlagh B, MacDonald JR, Tarnoplosky MA. Nutritional inadequacy in adults with muscular dystrophy. Muscle Nerve. 2005; 31(6): 713-8.
4. Lehman R.M. & McCormack G.L., Neurogenic and Myopathic Dysfunction, In L. Pedretti and M Early Occupational Therapy Skills for Physical Dysfunction Louis MO: Mosby, 2001;5:802-803.
5. Congenital Muscular Dystrophy (CMD)". MDA. [Retrieved 27 April 2012.]
6. Emedicine re EDMD Retrieved 30 July 2007.Kolkata, Gina (19 August 2010).
7. Reanimated 'Junk' DNA Is Found to Cause Disease". New York Times.[Retrieved 29 August 2010.]
8. Lemmers, Richard; Patrick J. van der Vliet, Rinse Klooster, Sabrina Sacconi, Pilar Camaño, Johannes G. Dauwerse, Lauren Snider, Kirsten R. Straasheijm, "A Unifying

Genetic Model for 9) Facioscapulohumeral Muscular Dystrophy". Science, 2010;19: 1650–1653.

9. Kanamori M, Morton NE, Fujiki K, Kondo K. Genetic epidemiology of Duchenne muscular dystrophy in Japan: classical segregation analysis. Genet Epidemiol. 1987; 4(6):425–432.
10. Yasuda N, Kondô K. No sex difference in mutations rates of Duchenne muscular dystrophy. J Med Genet. 1980; 17(2):106–111.
11. Morton NE, Chung CS. Formal genetics of muscular dystrophy. Am J Hum Genet. 1959; 11(4):360–379.
12. Jenkins, Simon P.R. Sports Science Handbook:I - Z. Brentwood, Essex: Multi-Science Publ. Co, 2005;37:17-24.
13. Turner, C; Hilton-Jones D."Themyotonic dystrophies: diagnosis and management". J NeurolNeurosurg Psychiatry, 2010; 81: 358–367.
14. Ballo R, Viljoen D, Beighton P. Duchenne and Becker muscular dystrophy prevalence in South Africa and molecular findings in 128 persons affected. S Afr Med J. 1994; 84:494–497.
15. H.R. 717--107th Congress (2001): MD-CARE Act, GovTrack.us (database of federal legislation), (accessed Jul 29, 2007). Public Law 107-84, PDF as retrieved from NIH website.
16. Darin N, Tulinius M. Neuromuscular disorders in childhood: a descriptive epidemiological study from western Sweden. NeuromusculDisord. 2000; 10(1):1–9.
17. Prot J. Genetic-epidemiological studies in progressive muscular dystrophy. J Med Genet. 1971; 8(1):90–95.
18. RichaGoel, Gheena S., Thiruvengadam Chandrasekhar, PratibhaRamani, Herald J. Sherlin, Anuja Natesan, Priya Premkumar, Amino acid profile in oral submucous fibrosis: A high performance liquid chromatography (HPLC) study.Journal of Clinical and Diagnostic Research 2014;8(12);44-45.
19. Nakagawa M, Nakahara K, Yoshidome H, et al. Epidemiology of progressive muscular dystrophy in Okinawa, Japan. Classification with molecular biological techniques.Neuroepidemiology. 1991; 10(4):185–191.

20. Peterlin B, Zidar J, Meznaric-Petrusa M, Zupancic N. Genetic epidemiology of Duchenne and Becker muscular dystrophy in Slovenia. Clin Genet. 1997;51(2):94–97.
21. Norwood FLM, Harling C, Chinnery PF, Prevalence of genetic muscle disease in Northern England: in-depth analysis of a muscle clinic population. Brain. 2009; 132(pt 11):3175–3.
22. Darin N, Tulinius M. Neuromuscular disorders in childhood: a descriptive epidemiological study from western Sweden. NeuromusculDisord. 2000; 10(1):1–9.
23. Romitti P, Puzhankara S, Mathews K, Centers for Disease Control and Prevention (CDC). Prevalence of Duchenne/Becker muscular dystrophy among males aged 5–24 years—four states, 2007. MMWR Morb Mortal Wkly Rep. 2009;58(40):1119–1122.
24. Swaminathan B, Shubha G N, Shubha D, Murthy A R, Kiran Kumar H B, Shylashree S, Gayathri N, Jamuna R, Jain S, Purushottam M, Nalini A. Duchenne muscular dystrophy: A clinical, histopathological and genetic study at a neurology tertiary care center in southern India. Neurol India 2009;57:734-8.
25. Rajalathiltha P, Vali S, Molecular pathogenesis of OSMF- A collagen metabolic disorder J Oral Pathol Med 2005 35:321-28.

A Questionnaire Survey to Evaluate the Awareness Among the Dentists of the Appropriate Usage of Topical And Local Anesthesia in Pediatric Patients

Umme Salma Durbar[1], Geo Mani[2], EMG Subramanium[3], GurumoorthyKaarthikeyan[4]

Graduate Student[1], Senior Lecturer[2], Department of Pedodontics, Professor[3], Department of Pedodontics, Professor[4], Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical SciencesSaveetha Univeristy,Chennai, India

Abstract

Introduction: Anesthetic injection is the dental procedure that produces the greatest negative response in children. Pain and anxiety can reduce the efficacy of anesthesia in pediatric patients. In the arena of pediatric dentistry, successful delivery of local anesthesia is an essential component of positive treatment outcomes. For many individuals, the experience of pain with dental procedures can lead to their future avoidance of much needed oral health care due to dental fears and anxiety. A child's early positive experiences can lead to a lifetime of healthy perceptions and attitudes towards dental health. The aim of this study was to assess the knowledge and use of topical and local anesthetic agents in pediatric oral procedures amongst the dentists.

Materials and Method: A closed- ended questionnaire comprising 12 questions based on type, procedural use, effectiveness, and adverse reactions noted to various topical and local anesthetics in pediatric patients was given to 150 dental practitioners. Data was analyzed by computing the percentage response for each question.

Results: The results show that 50% of the dental practitioners treating pediatric patients always used topical anesthesia prior to local anesthesia. 76%used topical in spray form. 69% of the practitioners used 4.4mg/kg dosage of LA. 55% of the respondents preferred the use of a 25 gauge needle.

Conclusion: Thisstudy concludes that dental practitioners treating pediatric patients consider topical anesthesia to be effective when used prior to LA and preferred short length needle with 25 gauge.

Key Words:*Local anesthesia, topical, Treatment, pediatric, Needle*

Introduction

Behavior management in pediatric dentistry comprehends various essentials ranging from creating a pain-free environment to reception of treatment by a child [1].The goal of providing “painless” dental care has been actively sought after for decades. In pediatric dentistry, successful delivery of local anesthesia is an essential component of positive treatment outcomes. According to the American Academy of Pediatric Dentistry, the prevention of pain during dental procedures can nurture the relationship of the patient and dentist, build trust, allaying fear and anxiety, and promoting a positive dental attitude [2]. In order to instilla positive attitude towards dental care among the patients, proper injection technique, topical anesthetics, dosage and time should be followed[3,4,5]. Many advances have been made in topical anesthetics, types of anesthesia, volume, location, and pre-operative patient management that have been used to improve overall patient care[6,7].

Local anesthesia causes temporary loss of sensation in one part of the body without depressing the level of consciousness with the use of a topically-applied or injected agent [8]. Local anesthetics act within the neural fibers to inhibit the ionic influx of sodium for neuron impulse[9,10]. This helps to prevent transmission of pain sensation. The two general types of local anesthetic formulations are: Esters (eg, procaine, benzocaine, tetracaine) and Amides (eg, lidocaine, mepivacaine, prilocaine,articaine)[11]. The local anesthetic blocks the action of a specialized gate called the sodium channel. The nerve signals cannot be transmitted when the sodium channel of a nerve is blocked. The only location at which the local anesthetic molecules have access to the nerve membrane is at the nodes of Ranvier, where there is an abundance of sodium channels. The interruption of a nerve signal in a myelinated nerve occurs when nerve depolarization (the nerve signal) is blocked at three consecutive nodes of ranvier.Local

anesthetics create a chemical road block between the source of pain and the brain by interfering with the ability of a nerve to transmit electricalsignals or action potentials [10].

Epinephrine, levonordefrin, norepinephrine are vasoconstrictors which can be added to local anesthetics to constrict blood vessels in the area of injection. Theyhelp to prolong the anaesthetic action by decreasing the rate of absorption of the local anesthetic into the blood streamandthereby lowers the risk of toxicity [12].A local anesthetic without a vasoconstrictor also can be used for shorter treatment needs but should be used with caution to minimize the risk of toxicity of the anesthetic agents.

Because of lower weight when compared to adults, young children are more likely to experienceanesthetic overdose or toxicity. Most adverse drug reactions occur within 5-10 minutes of injection. Overdose of local anesthetics are caused by high blood levels of anesthetic as a result of an inadvertent intravascular injection or repeated injections. Local anesthetic overdose outcomes depression of the central nervous system. Early subjective symptoms of the central nervous system include dizziness, anxiety and confusion and may be followed by diplopia, tinnitus, drowsiness and circumoral numbness or tingling. Objective signs include muscle twitching, tremors, talkativeness, slowed speech and quivering. Although allergic reactions to injectable amide local anesthetics are rare, patients may exhibit a reaction to thebisulfite preservative added to anesthetics containing epinephrine. Patients with a sulfa allergy should not receivearticaine. Patients may also exhibit allergic reactions to benzocaine topical anesthetics [13,14].

Injection prick is perceived as painful procedure and most children are fearful about the needle prick rather than the dental procedure itself.Topical local anaesthetics (TLA) are frequently used in dental practice to reduce acute and chronic pain and facilitate atraumatic dental treatment. TLA induces temporary loss of sensation up to a depth of 2-3mmon the applied surface [15,16]. Topical anesthetics produce temporary loss of sensationbyreversibly blocking the nerve conduction near their site of administration in a limited area. Nerve impulse conduction is blocked by decreasing nerve cell membrane permeability to sodium ions, possibly by competing with calcium-binding sites that control sodium permeability. Decreased depolarization and an increased excitability threshold occurs due to change in permeability that ultimately prevents the nerve action potential from forming. [17,18,19]TLA is an important prerequisite for many

pediatric dental procedures such as needle penetration, scaling and root planing, mucosal punch biopsies, application of orthodontic appliances and rubber dam clamps, postsurgical pain etc. [20]. The application of a topical anesthetic minimizesthe distress caused during administration of local anesthesia. Topical anaesthesia is effective when they are in contact with the applied site for a minimum duration of 2 minutes,1and lower duration of application might not produce desired clinical effect. Topical anesthetic agents are obtainable in gel, liquid, ointment, patch and aerosol forms.It's disadvantages are the taste may be disagreeable to patient and the length of application time may increase apprehension of approaching procedure in the pediatric patient.Benzocaine or lidocaineare the common topical anesthetics used in dentistry.

Local anesthesia forms the backbone of pain control techniques in dentistry and has a major role in dentistry for children. There is a constant search for ways to find a more comfortable and pleasant means of achieving local anesthesia before dental procedures to avoid the invasive and often painful nature of the injection. Fear of syringes and needles has been reported as one of the major causes of apprehension and anxiety in pediatric patients. Hence, pediatric dentists are on a constant search of tools for painless administration of topical and local anesthesia [21]. Thus, the aim of the present study was to evaluate the knowledge and awareness amongst the general dental practitioners about the appropriate use of topical and local anesthetic agents in pediatric patients.

Materials and Method

A questionnaire was designed for the dentists regarding the type, procedural use, effectiveness, and adverse reactions noted to various topical and local anesthetics in pediatric patients. The survey sampled one hundred and fifty general dental practitioners in Chennai city,India. The survey consisted of 12-items in multiple choice/answer format. The survey consisted of several questions related to the usage of local and topical anesthetics, the adverse reaction to the anesthetics, the needle size and gauge used,dosage and waiting time after the application of topical anesthetic before injecting. The survey also asked the clinicians to provide demographic data such as the name, age, sex and year of study. Data was analyzed by computing the percentage response for each question.

Results

Question	Response	MaxResponse	Response	MinResponse
Use of topical anesthesia	Always	50%	Never	3%
Type of topical anesthesia	Spray	76%	Ointment	3%
Time waited after application of topical anesthesia	31-60sec	40%	<10sec	12%
Effectiveness of topical anesthesia	Effective	60%	Poor	3%
dislike to topical anesthesia	Taste	76%	Colour	1%
Adverse reaction to TLA	0	71%	4-6%	3%
Times TLA works	75%	45%	100	3%
Dosage of LA	4.4mg/kg	69%	10mg/kg	2%
Factor considered during dosage	Body weight	74%	Others	5%
Gauge of needle	25 gauge	55%	30 gauge	9%

Length of needle	Short	71%	Long	29%
Time taken to inject	31-60sec	41%	<10sec	7%

Table 1 Responses of the practitioners to the questionnaire

The results show that 50% of the dental practitioners treating pediatric patients always used topical anesthesia prior to local anesthesia and 3% never used. For the question regarding the type of topical anesthesia used, a majority of the respondents (76%) used spray form and only about 3% used it in the form of ointment. When the respondents were asked about the waiting time before injecting, 12% waited only for <10sec and 40% waited for31-60 seconds. When asked about the effectiveness of topical anesthetics, 60% perceived them effective and 3% perceived them poor.When questioned concerning the different properties of topical anesthetic, according to the dental practitioners most of their patients did not like the taste (76%). The adverse drug reactions to topical anesthetics in pediatrics seen in the last year of practice were also examined. Most of the practitioners (71%) reported that their patients did not experience any adverse reactions to the topical anesthetic, while only 3% of the practitioners reported that 4-6% of their patients experienced an adverse drug reaction. When practitioners were asked the percentage of times they felt that the topical anesthetic worked, 45% felt that the topical worked 100% of the times and 3% felt that it never worked. 69% of the practitioners used 4.4mg/kg dosage of LA while 2% said it is 10mg/kg (figure 1).

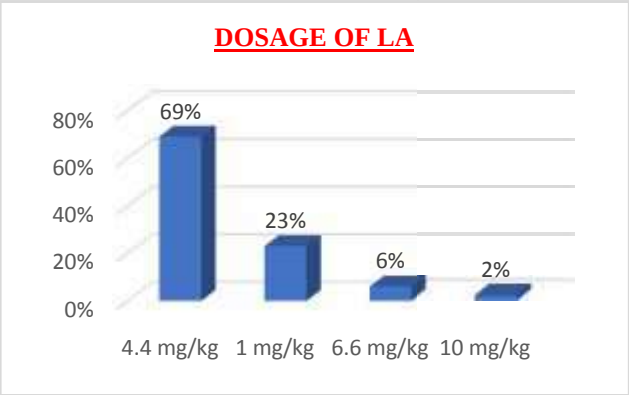


Figure 1: Dosage of LA(%).

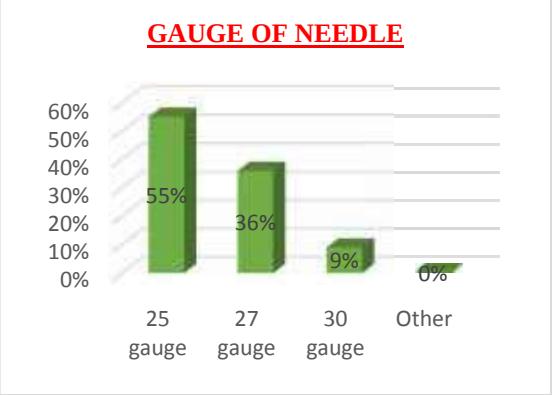
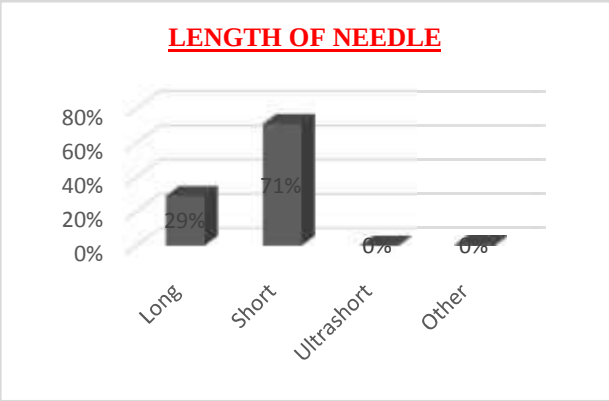


Figure 2: Gauge of needle used(%)

When questioned which criteria practitioners used to determine the local anesthetic dosage, 74% of the respondents reported using the exact body weight when determining the dosage of local anesthetic for each pediatric patient. The commonly used gauge sizes by the practitioners showed that 55% of the respondents preferred the use of a 25 gauge and 9% used a 30 gauge (figure 2). The length of needle used for pediatric patients was short length needles (figure 3).



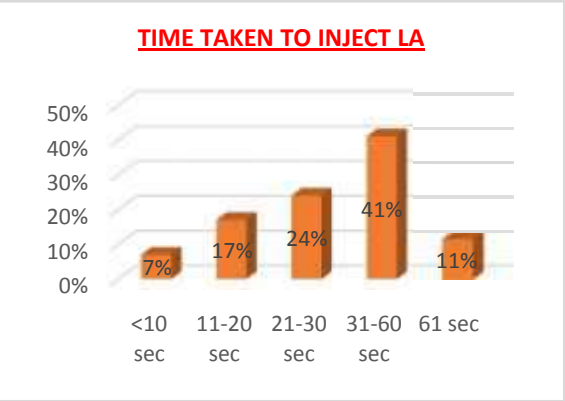


Figure 3: Length of needle used(%)

Figure 4: Time taken to give LA(%)

When questioned regarding the amount of time taken to inject a full cartridge of local anesthetic, 7% took less than 10 secs and 41% took 31-60 secs (figure 4).

Discussion

The questionnaire was distributed among dental practitioners which included 83% females and 17% males between age groups of 20-27 years. Majority of the dentists always use topical anesthesia when treating pediatric patients. Topical anesthesia when used appropriately can improvepatients behaviors and attitudes and provide a safe and positive treatment outcome [22].The type of topical anesthesia commonly used is the spray form. Many options to deliver anesthesia have developed over the last several decades. Most of the practitioners use the spray form of topical anesthesia for pediatric patients. Electronic dental anaesthesia (EDA), precooling, vibration, iontophoresis, and sonophoresis are some alternative techniques that can be used for producing topical/surface anaesthesia.

As for the waiting time after application of topical anesthetics, the recommend waiting time is 10-30 seconds before injecting depending on the form of the preparation. Earlier studies recommended 60 seconds or longer before injection to assure maximum efficacy of topical anesthetics [23]. Others recommend a waiting time of approximately 30 seconds [24].Stern and Giddon showed that, application of a topical anaesthetic to mucous membrane for 2 to 3 minutesleads to profound soft tissue analgesia [25].

Most practitioners responded that the current topical anesthetics they were using were effective, while about 3% thought they were inadequate.Most practitioners responded that their patients disliked the taste of the topical anesthetics.

In this survey, very few practitioners reported any adverse reactions from the local and topical anesthetics. Majority of the practitioners felt that the topical anesthetics work when applied prior to the local anesthesia. Many local anaesthetics used effectively via injection prove ineffective when applied topically (e.g., articaine, mepivacaine, prilocaine, procaine), because, the concentrations necessary to produce anaesthesia via topical application are high, with significantly increased overdose and local tissue toxicity. Higher concentration also increases the risk of toxicity, locally and systemically if the drug is efficiently absorbed. Allergic reactions are more common with ester type when compared to amide type. The risk of overdose with amide topical anaesthetics is greater than that with the esters and increases with the area of application of the topical anaesthetic. Because topical anaesthetic formulations do not contain vasoconstrictors and local anaesthetics have vasodilatory properties, vascular absorption of some topical formulations is rapid, and blood levels may quickly reach those achieved by direct IV administration. Preservatives, such as methylparaben, and other ingredients may also cause allergic reactions. Toxic reactions to local anaesthetics are more common after topical than via any other route of administration. It is the amount of drug administered that is important with regard to toxicity and topical agents are usually available in concentrations much greater than are found in injected formulations. Local anesthetic toxicity can be prevented by careful injection technique, watchful observation of the patient, and knowledge of the maximum dosage based on weight. Practitioners should aspirate before every injection and inject slowly [26].

With the question of the factor used to determine the dosage of local anesthetic, majority of the practitioners chose using body weight as an important factor while calculating the dosage. Malamed recommended the use of exact body weight to prevent anesthetic overdose.

Needle selection should allow for profound local anesthesia and adequate aspiration. Larger gauge needles provide for less deflection as the needle passes through soft tissues and for more reliable aspiration [27]. The depth of insertion varies not only by injection technique, but also by the age and size of the patient. The survey showed that a high percentage of pediatric dentists are using needles with a diameter (25 gauge) smaller than recommended. Malamed recommends the

use of a 25-gauge needle for all injections in highly vascular areas and 27-gauge needle can be used for all other injection techniques provided the aspiration percentage is exceedingly low [28]. Trapp and Davies have reported positive aspiration through 23-, 25-27- and 30-gauge needles. However, the question as to which the appropriate needle gauge isremains, and it is universally agreed upon that the anesthetic solution must be injected slowly, and the dentist should observe the patient carefully for any unexpected reactions [29].Malamed also recommends the length of the needle to be long for all techniques requiring penetration of significant thickness of soft tissue. Short needles may be used for injections that do not require penetration of significant depths of soft tissue. If using a smaller diameter is justified to prevent visual excitation of the patient, it must be noted that the diameter difference is almost negligible to the unaided eye. Needles should not be bent if they are to be inserted into soft tissue to a depth of greater than five millimeters or inserted to their hub for injections to avoid needle breakage[30].

As for the time taken to inject a full cartridge of anesthetic, a slow injection is important for two reasons: 1) for the safety of the patient and 2) to avoid the solution from tearing the soft tissues into which it is being injected. Rapid injection causes an immediate discomfort that last a few seconds followed by a prolonged soreness long after the numbing effect of the local anesthetic has subsided [31].Malamed recommends at least 60 seconds for a full 1.8ml of cartridge as this rate of deposition will not produce tissue damage either during or after anesthesia, nor will it cause a serious reaction in an event of accidental intravascular injection.48%of the surveyed pediatric dentists were injecting a full cartridge with a time of less than sixty seconds.

Conclusion

The findings of this study demonstrate that dental practitioners treating pediatric patients consider that topical anesthesia are effective when used prior to LA and are most commonly using spray form as the preferred type of topical anesthesia. Topical and local anaesthetics are most important armamentarium for pediatric dentists to make dental procedures atraumatic for the children.

Corresponding Author

GurumoorthyKaarthikeyan,

Professor, Department of Periodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,SaveethaUniveristy, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India.

Email:drkarthik79@yahoo.co.in,

Telephone Number: +91 9841222027

References

1. NehaBansal,SonaliSaha, Jaiswal and FirozaSamadi. Pain Elimination during Injection with Newer Electronic Devices: A Comparative Evaluation in Children. International Journal of Clinical Pediatric Dentistry, May-August 2014;7(2):71-76.
2. Dean JA, Avery DR, McDonald RE. Local anesthesia and pain control for the child and adolescent. In: Dentistry for the Child and Adolescent. 9th ed., St Louis, Mo: Mosby; 2011:241-52.
3. Wilson S. Local anesthesia and oral surgery in children. In Casamassimo PS, Fields HW, McTigue DJ, Nowak A, eds. Pediatric Dentistry Infancy Through Adolescence, 5th ed., St. Louis, Mo: Elsevier; 2013:398-410.
4. Malamed SF. Basic injection technique. In: Handbook of Local Anesthesia. 6th ed., St. Louis, Mo: Mosby; 2013: 157-68.
5. Martin MD, Ramsay D, Whitney C, Fiset L, Weinstein P. Topical anesthesia: differentiating the pharmacological and psychological contributions to efficacy. Anesthesia Prog. 1994;41(2):40-47.
6. Kincheloe J, Mealiea W Jr, Mattison G, Seib K. Psychophysical measurement on pain perception after administration of a topical anesthetic. Quintessence Int. 1991;22(4):311-315.

7. A Naurin Salma, Mahesh Ramakrishnan. Use of anesthesia in pediatric dentistry: A cross-sectional survey.Int J of PedodonticRehabilitation. 2016;1(1):5-9.
8. Ogle OE, Mahjoubi G. Local anesthesia: Agents, techniques, and complications. Dent Clin North Am 2012; 56(1):133-48.
9. Robyn Gmyrek, Maurice Dahdah. Local and Regional Anesthesia. Medscape. Jul 07, 2015
10. Moore PA, Hersh EV. Local anesthetics: pharmacology and toxicity. Dent Clin North Am 2010;54(4):587-99.
11. A. L. Sisk. Vasoconstrictors in local anesthesia for dentistry. AnesthProg. 1992; 39(6): 187–193.
12. Sunny Priyatham and SivakumarNuvvula. Intraoral topical anaesthesia in pediatric dentistry: review. Int J Pharm Bio Sci 2016; 7(4):346 – 353.
13. Peedikayil FC, Vijayan A. An update on local anesthesia for pediatric dental patients. Anesthesia, essays and researches. 2013;7(1):4-5.
14. Harvey M, Elliott M. Transcutaneous electrical nerve stimulation for pain management during cavity preparationsin pediatric patient. ASDC J Dent child 1995;62(1):49-51.
15. Gayathri R Menon, Dr. Dhanraj,Dr.RevathyGounder. Knowledge, awareness and practice among dental practitioner regarding various topical anaesthetic agents used in dental clinics. International Journal of Current Research2017;9(4):49490-49492.
16. Meechan JG. Intraoral topical anesthesia. Periodontology 2000. 2008;46(1):56-79.
17. Kundu S, Achar S. Principles of office anesthesia: part II. Topical anesthesia. Am Fam Physician. 2002 Jul 1. 66(1):99-102.
18. Milgrom P, Coldewell SE, Getz T, Weinstein P, Ramsay DS. Four Dimensions of Fear ofDental Injections. JADA 1997;128(6):756-766.
19. McGee D. Local and Topical Anesthesia. Roberts JR, Hedges J. Clinical0 procedures in emergency medicine. 4th ed. Philadelphia: WB Saunders; 2004. 533-551.

20. Nayak R, Sudha P. Evaluation of three topical anesthetic agents against pain: A clinical study. Indian Journal of Dental Research 2006; 17(4): 155-60.

21. Lawrence Shults.A Survey of the Usage of Topical Anesthesia Among Dentist. VCU Scholars Compass. May 14, 2010.

22. JasdevBhalla, John G. Meechan, À Herenia P. Lawrence,Helen A. Gradand Daniel A. Haas. Effect of Time on Clinical Efficacy of Topical Anesthesia. AnesthProg2009;56(2):36-41 2009.

23. McDonald, RE and Avery, D: Local Anesthesia for the Child and Adolescent. In Dentistry For The Child and Adolescent. 7th Ed., RE McDonald and D Avery, St. Louis: CV Mosby Co., 2000, pg 283-284.

24. Kohli K, Ngan P, Crout R, Linscott CC.A survey of local and topical anesthesia use by pediatric dentists in the United States.Pediatr Dent. 2001 May-Jun;23(3):265-9.

25. Stern I, Giddon DB. Topical anesthesia for periodontal procedures. AnesthProg 1975;22(4):105-8.

26. Kravitz ND. The use of compound topical anesthetics: A review. J Am Dent Assoc 2007;138(10):1333-9.

27. Luke J. Haseler, Randy R. Sibbitt, Wilmer L. Sibbitt, Adrian A. Michael, Charles M. Gasparovic and Arthur D. Bankhurst. Syringe and Needle Size, Syringe Type, Vacuum Generation, and Needle Control in Aspiration Procedures. CardiovascInterventRadiol. 2011; 34(3): 590–600.

28. Kenneth L. Reed, Stanley F. Malamedand Andrea M. Fonner. Local Anesthesia Part 2: Technical Considerations. AnesthProg2012;59(3):127-137.

29. Hersh EV. Local anesthetics in dentistry: clinical considerations, drug interactions, and novel formulations. Compendium (Newtown, Pa.). 1993 Aug;14(8):1020-2.

30. Blanton PL, Jeske AH. Dental local anesthetics: Alternative delivery methods. J Am Dent Assoc2003;134(2):228-34.

31. Malamed, SF: Basic Injection Technique. In Handbook of Local Anesthesia. 4th Ed., SF Malamed, St. Louis: Mosby Co., 1997, pp 134 and 140.

A Survey of Prevalence of Various Classes of Kennedy's Classification

Hansini B. Ajantha[1], Pon Kirubha Robin[2], Radhika Arjunkumar[3]

Graduate Student[1],Senior Lecturer[2],Department of Prosthodontics,Reader[3],Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The design of prosthetic replacement depends upon the patterns of partial edentulism. The aim of classification of partially edentulous arches is to facilitate the communication of prosthesis designs and treatment options among professional colleagues, students and technicians. The aim of this study was to determine the prevalence of various classes of Kennedy's classification in patients reporting to Saveetha dental college.

Materials and Methods: 170 patients were recruited in this study from February 2015 to May 2015 of age group 30 to 60. Kennedy's classification was utilized after applying Applegate's rules to record the partial edentulism.

Results: 101 male patients and 69 female patients participated in this study. Out of this, 71 were bite cases and 99 were single arch cases. Partial edentulism in the maxillary arch was found in 22.3% patients, while 35.9% patients had partial edentulism in mandibular arch. The remaining 41.8% of patients had missing teeth in both arches. Kennedy's class I was found to be most common, while Kennedy's class IV the least common.

Conclusion: A higher frequency of tooth loss due to dental caries and negligence of oral health among the patients in this study is quite alarming and is suggestive of need to create awareness among dental health care system for timely prevention of diseases and factors leading to tooth loss. By bringing about awareness of tooth loss and available treatment options, the dental needs of the community could be met leading to an overall improvement in their quality of life.

***Key Words**: Applegate's rules, Kennedy's classification, Maxilla, Mandible, Partial edentulism*

Introduction

Teeth are the key entities of stomatognathic system for mastication, phonation and aesthetics [1, 2]. Many options are available for replacing missing teeth like removable partial dentures, fixed partial dentures and implant-supported prosthesis[3]. Conservative treatment modalities used to treat partial edentulism in patients, such as dental implants also happen to be the most expensive. This happens to limit their availability to lower socioeconomic groups[4]. Hence, it should not be much of a surprise that conventional removable prosthodontic treatment modalities continue to outnumber implant tooth replacements in general dental practice. Removable partial prosthesis is a versatile, cost effective and reversible treatment method for partially edentulous patients of any age. The design of prosthetic replacement depends upon the patterns of partial edentulism. The aim of classification of partially edentulous arches is to facilitate the communication of prosthesis designs and treatment options among professional colleagues, students and technicians. The classification is also helpful in recording history of patients[5]. Different Prosthodontists had proposed many classification systems[6]. Each classification system is associated with its own merits and demerits. The ultimate goal of any classification system and is, improved communication and consistency within the dental professionals. Different classifications may also have associated treatment principles that assist in treatment standardization and teaching. Kennedy's system of classification is most widely used[7]. The Kennedy classification system's main differentiating factor is its ability to quickly and clearly demonstrate the location of tooth support in relation to edentulous spaces. Due to its simplicity, it does not make judgments regarding condition or position of the remaining teeth. There are many instances, in which Kennedy's classification is difficult to apply. For such special situations Applegate's rules are applied[8]. The objective of this study was to determine the prevalence of various patterns of partial edentulism, using Kennedy's classification system, of patients reporting to Saveetha Dental College and Hospitals.

Materials and Methods

This study was undertaken in Saveetha Dental College Hospitals from February 2015 to May 2015. After approval from institutional ethical review Committee, the data for this study was compiled from the patients fulfilling the inclusion criteria. Name, age, gender, cause of tooth loss, periodontal health and Kennedy's class along with modification spaces were recorded. Informed consent was taken from all patients. History was taken, which was followed by detailed clinical examination for partial edentulism. Whether only anterior or only posterior or both anterior and posterior teeth were missing was recorded. If assessment of missing anterior teeth was in question, anterior teeth were considered missing, if the patient's prosthesis consisted of a prosthetic anterior tooth. This avoided misrepresentation of large diastema's or spaces closed due to tooth migration. The total number of missing teeth was recorded. Fixed prosthodontic pontics and closed spaces were not considered missing teeth. Kennedy's classification system modified by Applegate was used in this study.

Results

A total of 170 patients with partial edentulism were examined, in which 101 were male patients and 69 were female patients. Out of this, 71 were bite case and 99 were single arch cases, resulting in examination of 241 dental arches in total. Partial edentulism in maxillary arch was found in 22.3% patients, while 35.9% patients had partial edentulism in mandibular arch. The remaining 41.8% of patients had missing teeth in both arches (Table 1). Caries are found to be the most common cause of tooth loss in patients with edentulism (72.4%) followed by periodontal diseases (17%). The distribution is given in Fig.1. Kennedy's class I (44.3%) was found to be most common, while Kennedy's class IV (11%) the least common (Fig 2). Male predilection is higher in all Kennedy's classification than female (Fig 4).

Table 1: Distribution of Kennedy's classification according to dental arches

DENTAL ARCH	MALE	FEMALE	TOTAL n (%)
MAXILLA	20	18	38 (22.3)
MANDIBLE	33	28	61 (35.9)
BITE CASE	48	23	71 (41.8)

TOTAL	101	69	170

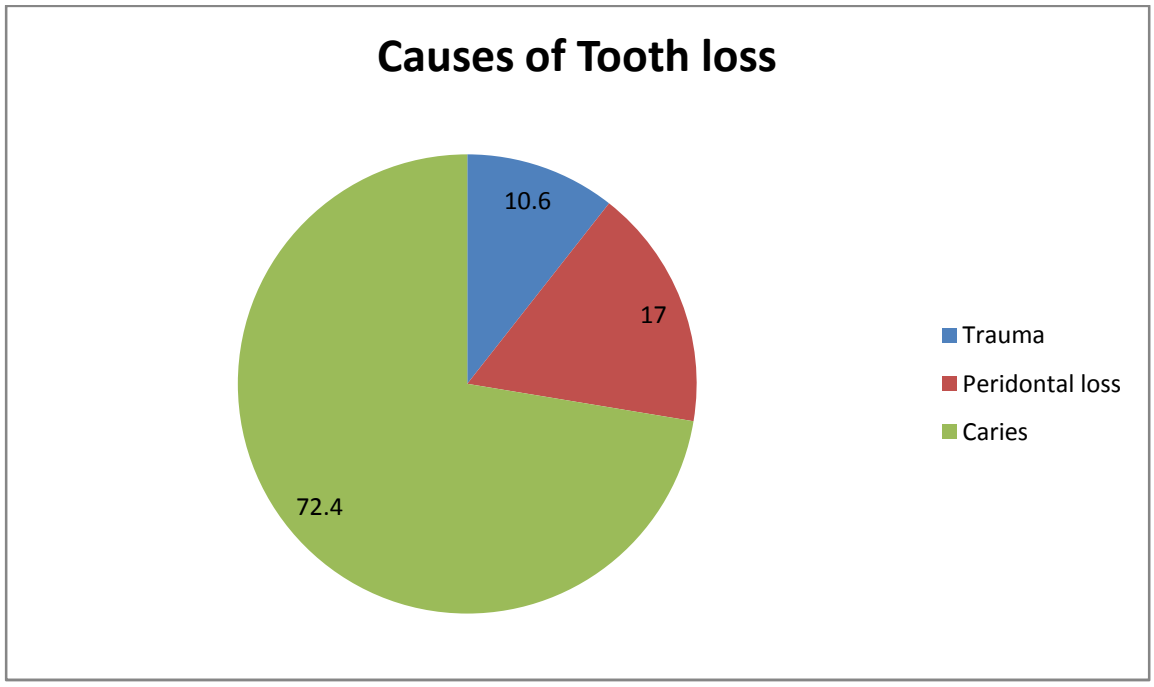


Figure 1: Pie chart showing Causes of tooth loss

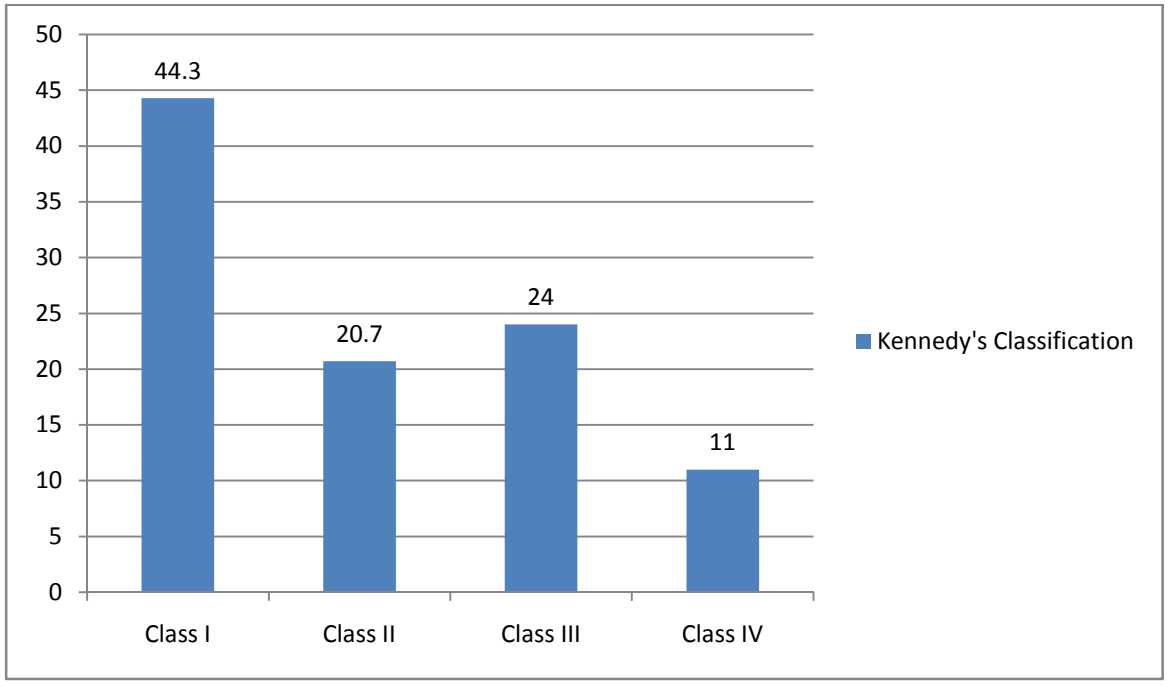


Figure 2: Bar chart showing Incidence of various classes of Kennedy's classification (in %)

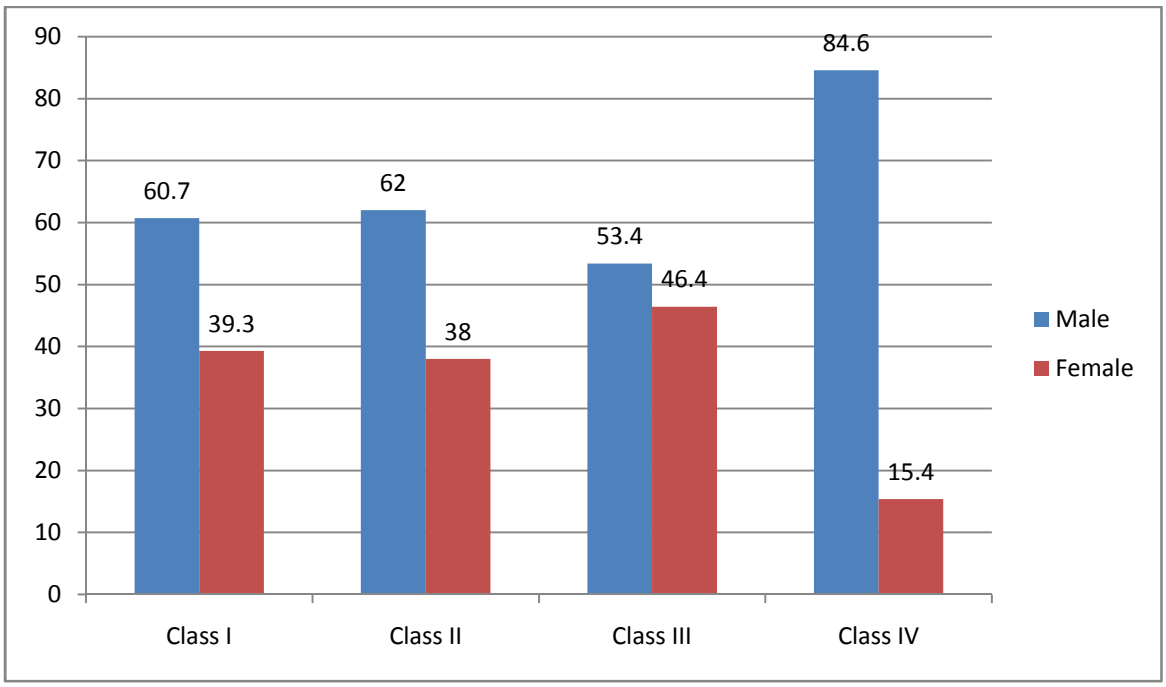


Figure 3: Bar chart showing Distribution of Kennedy's classification according to gender (in %)

Discussion

The presence or absence of teeth acts as a good indicator of the oral health of an individual in general. Oral health is an important component of both overall health and quality of life, as oral health and general health are interdependent. Oral disease creates a major public health burden worldwide and receives inadequate attention in many low- and middle-income countries [World Health Organization (WHO)] [9]. Systemic conditions have oral manifestations that increase the risk of oral disease, which in turn is a risk factor for various systemic diseases. Tooth loss is recognized as an individual risk factor for a range of systemic conditions, such as hypertension, stroke, cardiovascular disease, diabetes and coronary heart disease. Tooth loss affects the quality of life of an individual resulting in reduced efficiency and function of the masticatory system and the appearance of the individual. By reduced chewing ability in individuals with tooth loss, nutritional deficiencies may be seen, and intakes of certain foods could probably be reduced. There is strong evidence that a diet high in fruits and vegetables improves systemic immunity and protects our body against obesity, diabetes, cardiovascular disease, and many other

cancers[10]. The recommended intake of fruits and vegetables is 400 gm/day. Thus, tooth loss will ultimately lead to the poor dietary intakes which in turn causes poor nutritional status. Masticatory performance and biting force are greater in dentate individuals followed by partially edentate and completely edentulous individuals[11]. Most of the patients evaluated in this study had missing posterior teeth, and in such partially edentulous subjects, biting force and masticatory ability is very much lower compared to dentate individuals, which was the prime concern in replacing the missing posterior teeth. Enlargement of the tongue, reduction in the vertical dimension, interocclusal space and lower facial height of the individual are few common effects of tooth loss. The reasons for tooth loss may be due to dental caries, periodontal disease, traumatic accidents, impaction, orthodontics, supernumerary, preparation for radiotherapy, or even due to congenital and developmental disorders[12]. The two most common causes of tooth loss in Asian population are dental caries and periodontal disease[13]. Secondary reasons for tooth loss are restricted access to dental services, health systems, and lack of oral health care[14]. These individuals with a high percentage of tooth loss need prosthetic restorations to restore the form, function, and aesthetics.

Several methods have been proposed to classify the partially edentulous arches on the basis of the potential combinations of teeth to ridges. At present, Kennedy's classification is probably the most widely accepted one. Kennedy divided all partially edentulous arches into four main types. In his classification, edentulous areas, other than those determining the main types, were designated as modification spaces. The Kennedy classification is as follows[15]

Class I. Bilateral edentulous areas located posterior to the remaining natural teeth.

Class II. A unilateral edentulous area located posterior to the remaining natural teeth.

Class III. A unilateral edentulous area with natural teeth remaining both anterior and posterior to it.

Class IV. A single but bilateral (crossing the midline) edentulous area located anterior to the remaining natural teeth.

The results of this study showed that the frequency of partial edentulism is higher in mandible than maxilla in our population and Class 1 is the most common and Class 4 is the least common. This is in concordance with the study carried out by Curtis et al[16], at University of California, school of dentistry. Anderson et al[17] investigated the prevalence of the classification of removable partial dentures at Birmingham Dental Hospital in the United Kingdom. Out of a total

of 417 removable partial dentures, 208 (49.9%) were Kennedy Class I, 76 (18.2%) Class II, 101 (24.2%) Class III and 32 (7.6%) Class IV. This was the earliest study of Kennedy classification prevalence. Basker et al and Davenport et al[18] conducted survey in three commercial dental laboratories in the United Kingdom, resulting in examination of 80 maxillary and 44 mandibular cast metal framework removable partial dentures. Kennedy Class III (69%) was the most frequent maxillary removable partial denture while Class II was the most frequent in the mandibular arch (43%). Interestingly, the study was repeated 10 years later for comparison purposes[19]. This time five commercial dental laboratories were surveyed for a total of 330 frameworks examined. Kennedy Class III (55%) was still the most common maxillary removable partial denture, though the frequency decreased. Kennedy Class I was the most common mandibular removable partial denture found. These differences may have been due to chance or possible changes in tooth loss and treatment selection. Bergman et al[20] did a long-term follow-up consisting of 33 removable partial denture patients in Sweden. Two cases were Class II and a single case was Class III. Of the 30 Class I removable partial dentures, 25 were mandibular. Wetherill et al and Smales et al[21] conducted a survey in which, a total of 150 RPDs was thereby assessed. A distinct difference between maxillary and mandibular arches was seen. 60 out of 86 mandibular removable partial dentures were Class I. 29 Class II and 21 Class III maxillary removable partial dentures were found from the total of 64. Schwalm et al[22] conducted a survey in which 161 patients were examining in which the Class I RPD was the most common followed by Class III, II and IV respectively. Filiz keyf et al[23] analyzed the distribution of partial edentulous patients who sought treatment for removable partial denture in clinics using Kennedy classification. This study showed Class I had a large distribution in the mandible, while class II in maxilla. In contrast to our study, Lana A. Shinawi et al[24] investigated the frequency of different pattern of partial edentulism in 200 patients in King Abdul Aziz University, showed that Kenney's class III was most predominant. A R Jain et al[25] studied the prevalence of partial edentulism in South Indian rural population. The results of this study showed that Kennedy's class III was most prevalent in both maxilla and mandible. Al Dawairi et al[26] in a study investigated the frequency of different pattern of partial edentulism of 200 patients in Jordan. In this study Kennedy's class III pattern of partial edentulism was most commonly encountered in both maxilla (47%) and mandible (45%). Sadig and Idowu et al[27] carried out a study in Saudi population on 422 partially dentate patients in which they concluded that Kennedy's class III in

both maxilla and mandible were common. Maqsood et al[28] in study of partial edentulism based on Kennedy's classification carried out on 395 patients in Isra Dental College outpatient department in Hyderabad, showed that Kennedy's class IV was most prevalent in both arches. A study carried by Hassan et al[29] at Armed Forces Institute of Dentistry, Pakistan on 1000 partially edentulous patients showed that Kennedy's class III was most common in maxilla (60.9%) and mandible (46.8%).

Conclusion

The result of the distribution of various classes of Kennedy's classification is almost similar to most of the other studies. The higher frequency of posterior tooth loss is suggestive of a greater need to create awareness in patients about oral hygiene maintenance and early restorative management of carious lesions. The higher frequency of class 4 in male patients requires emphasis on safety measures for trauma prevention and advances in aesthetic dentistry.

Corresponding Author

Radhika Arjunkumar

Reader,Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Mobile- +91-9884281681

Email id- radhikaarjunkumar@gmail.com

References

1. Mitchell L, Mitchell D, McCaul L. Oxford Handbook of clinical dentistry. New York: Oxford Edi 5:2009, 294.

2. Stratton RJ, Weibelt FJ. An atlas of removable partial denture design. Chicago, Illinious: Quintessence publishing Co. 3: 1998; 27-30.
3. Charyeva OO, Altynbekov KD, Nysanova BZ. Kennedy classification and treatment options. A study of partially edentulous patients being treated in a specialized prosthetic clinic. J Prosthodont 2012; 21(3):177-80.
4. Devishreea RA, Arumughamb M, Jain AR. The Role of Social Economic Status on Dental Caries and its Prevention among Outpatients Visiting Private Dental College Hospital. Journal of Pharmaceutical Sciences and Research. 2018 Feb 1; 10(2):369-71.
5. Arthur N, Christopher R, Rober S. Classification system for partial edentulism. J Prosthodont 1999; 8(1):27-39.
6. Rasoul A, Ahmedian L, Sharif E. A simplified classification system for partially edentulism. A theoretical explanation. J Ind Prosthod Soc 7:2007; 85-87.
7. Alan B, David T. McCraken's Removable Partial Prosthodontics. Elsevier.Edi 12: 2010; 19-26.
8. Applegate DC. The rationale of partial denture choice. Journal of Prosthetic Dentistry 1960; 10(5): 891-907.
9. Adegboye AR, Fiehn NE, Twetman S, Christensen LB, Heitmann BL. Low calcium intake is related to increased risk of tooth loss in men. J Nutr 2010 Oct;140(10):1864-1868.
10. Jagadeesan M, Rotli SB, Danabalan M. Oral health status and risk factors for dental and periodontal disease among rural women in Pondicherry. Indian J Community Med 2000 Jan-Mar; 25(1); 31-38.
11. Bradbury J, Thomason JM, Jepson NJ, Walls AW, Mulvaney CE, Allen PF, Moynihan PJ. Perceived chewing ability and intake of fruit and vegetables. J Dent Res 2008 Aug;87(8): 720-725.
12. Oginni FO. Tooth loss in a sub-urban Nigerian population: causes and pattern of mortality revisited. Int Dent J 2005 Feb; 55(1):17-23.
13. Ong G, Yeo JF, Bhole S. A survey of reasons for extraction of permanent teeth in Singapore. Community Dent Oral Epidemiol 1996 Apr; 24(2):124-127.
14. George B, John J, Saravanan S, Arumugham IM. Prevalence of permanent tooth loss among children and adults in a suburban area of Chennai. Indian J Dent Res 2011;22:364

15. Henderson D, McGivney GP, Castleberry DJ. McCrackenÕs removable partial prosthodontics. CV Mosby. St.Louis, Toronto, Princeton Edi 7:1985, 21-126.
16. Curtis DA, Curtis TA, Wagnild GW, Finzen FC. Incidence of various classes of removable partial dentures. J Prosthet Dent 67: 1992,664-667.
17. Anderson JN, Lammie GA. A clinical survey of partial dentures. Br Dent J 1952;92:59-67.
18. Basker RM, Davenport JC. A survey of partial denture design in general dental practice. J Oral Rehabil 1978;5:215-222.
19. Basker RM, Harrison A, Davenport JC. Partial denture design in general dental practice – 10 years on. Br Dent J 1988:165:245.
20. Bergman B, Hugoson A, Olsson C. Periodontal and prosthetic conditions in patients treated with removable partial dentures and artificial crowns. A longitudinal two-year study. Acta Odont Scand 1971;29:621-38.
21. Wetherell JD, Smales RJ. Partial denture failures: A long term clinical survey. J Dent 1980;8:333-40.
22. Schwalm CA, Smith DE, Erickson JD. A clinical study of patients 1 to 2 years after placement of removable partial dentures. J Prosthet Dent 1977;38:380-91.
23. Filiz keyf, Frequency of the Various Classes of Removable Partial Dentures and Selection of Major Connectors and Direct/Indirect Retainers. Turk J Med Sciences. 31 :(2001), 445-449.
24. Lana A. Shinawi. Prevalence and pattern of tooth loss. Life Science Journal 2012; 9(4): 1-7.
25. Ashraf J, Jain AR, Ariga P, Nallaswamy D. Prevalence of partial edentulousness and treatment needs in rural population of south India. World J Dent 2017; 8(3):213-217.
26. Al-Dawairi ZN. Partial edentulism and removable denture construction: A frequency study in Jordanians. Eur J Prosthodont Restor Dent 2006; 14: 13- 17.
27. Sadig WM, Idowu AT. Removable partial denture design. A study of selected population in Saudi Arabia. J Cont Dent Prac 2002; 3: 1-11.
28. Soomro MA, Maqsood S, Afaq A. Partial edentulism based on Kennedy's classification. J Medical Forum. 2011; 1: 1-5.

29. Naveed H, Aziz M, Hassan A. Patterns of partial edentulism among Armed Forces personnel. Pak Oral and Dent J 2011; 31(1): 217-21.

A Survey of Soft Carbonated Drink Consumption among Adolescents

S.Pooja[1],Pradeep Kumar[2],Geo Mani[3],

Graduate Student[1],Senior Lecturer[2],Department of Public HealthDentistry,Senior Lecturer[3], Department of Pedodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The consumption of soft drinks has increased dramatically over past several decades. Dentists are becoming more concerned about over consumption of soft drinks which results in dental disease. All soft drinks are acidic which corrodes the teeth by eroding its enamel. The high measure of sugar expended through soda pops prompt the advancement of microscopic organisms that assault the teeth along these lines exasperating dental issues.

Material and Method: A questionnaire with 7 questions was made on oral health hazards on taking aerated soft drinks among adolescents was conducted between Dec to March 2016.

Results: This study showed 65% females of age group 10-19 years and 60% males of age group 10-19years had the habit of consuming carbonated soft drinks.

Conclusion: The findings of the present study indicate a high rate of soft drink consumption among adolescents. In order to diminish the use of carbonated drinks by population it is necessary to provide appropriate training for the adolescents about the adverse effects of soft drinks and the oral health knowledge.

Key Words: *soft drinks, adolescents, survey, carbonated*

Introduction

A soft drink is a beverage that is carbonated and it has become a highly visible with increase energy intake and body weight (1).Generally soft drinks are composed of filtered water, artificial additives and refined sugar. They usually lack nutritional value and add up calories

and it makes you gain weight. It has been found that dental erosion is considered as a clinical problem in the oral health of school children and young adults and acidic drinks are thought to be an important cause(2).High consumption of soft drinks and other sugary drinks are associated with a number of health problems, including overweight and obesity, type 2 diabetes, osteoporosis and dental caries(3).High soda pop consumption is linked with widespread demineralisation of enamel and extensive caries in pits and fissures and in the inter proximal areas(4).The soda pops are thought to make harm the teeth in view of two properties – to start with, the low pH and corrosiveness of a few beverages can cause disintegration on the polish surfaces and besides the fermentable starch in drinks is processed by plaque miniaturized scale living beings to produce natural acids in the dental plaque and spit, bringing about demineralisation and prompting dental caries(5). People are devouring sodas relatively consistently without knowing the evil impacts and the mischief of sodas on their dental and general wellbeing. Thus this study aimed to assess the level of knowledge and attitude towards oral health hazards on taking carbonated soft drinks among adolescents(6). Causative components for tooth disintegration are isolated into extraneous and inborn classes. Inborn disintegration happens from automatic gastrointestinal unsettling influences, for example, gastroesophageal reflux ailment (GERD) and from intentional disgorging of gastric acids showed by anorexic as well as bulimic people(7). Outward tooth disintegration causes incorporate natural elements, medicaments, way of life, and eating regimen. In present day social orders the outward factor is ending up more critical, because of the expanded utilization of corrosive beverages as soda pops, don drinks, organic product squeezes and natural product teas(8).Dental examination found that crescent-shaped lesions were present on the cervical region of the buccal and labial surfaces of the teeth of the patients(9). The individual manner of drinking acidic soft drinks has been said to affect how long the teeth are in contact with the erosive challenge and therefore influence the pattern of destruction caused by them. The drinking method strongly affects tooth-surface pH and thereby the risk for dental erosion. Six different methods of drinking were tested in a randomized order by Johansson et al. : holding; short-sipping; long-sipping; gulping; nipping; and sucking(10).Patient should avoid toothbrushing enamel which has been softened by exposure to black cola this may cause additional material loss due to tooth brush abrasion Kids ought to be urged to drink acidic beverages rapidly and utilize a straw with the goal that the fluid is pushed to the back of the mouth.Also, avoid buying re-sealable bottles and discourage sipping over a long period of time. The liquid fruits can be used as a natural

alternative to synthetic beverages(11). Hence this study aimed to bring awareness about carbonated drinks among adolescents.

Materials and Methods

This is an institutional based study conducted to determine the level of knowledge and attitude towards oral health hazards on taking carbonated soft drinks among adolescents.A self-administered questionnaire survey was conducted among patients between the age group of 10-19years. This survey was conducted between Dec to March 2016. A convenience sample of 150 adolescents were the target population .In this study, a questionnaire was prepared, which consists of 7 questions related to knowledge of oral health hazards on taking carbonated soft drinks among adolescents .The aims and objectives were explained to all the adolescents before starting the study. Questions were manually checked for completion of data. All data were entered in MS Excel spreadsheet, and the percentage was calculated.

Results

The study was conducted between males and females of age group 10-19years. Of these 87 were males and 63 were females. This study showed 65% females of age group 10-19 years and 60% males of age group 10-19years had the habit of consuming carbonated soft drinks. It was found that 30% of females consumed carbonated drinks in daily basis and 20% of males consumed carbonated drinks in daily basis. Regarding the question awareness of consumption of soft drinks it was found that 28% females of age group 10-19years, 30% males of age group 10-19years were aware of consumption of soft drinks would erode tooth enamel. When asked about any sensitivity on taking aerated soft drinks

it was found that 60% females and 65% males experienced sensitivity in taking aerated soft drinks. Thus the awareness programme regarding the oral health hazards of carbonated soft drinks are less, overall only 65% of adolescents are aware of it.

Table 1: Questionnaire

FEMALE:

AGE GROUP: 10 – 19 years

Questions	Sample	%
1. Consumption of soft drinks among patients in varying dental office	41(yes) 22(no)	65% 34%
2.Frequency of consumption of soft drinks	25(daily basis) 21(weekly basis) 17(monthly basis)	39% 33% 26%
3. Necessity of drinking a soft drink	23(taste) 40(refreshment)	36% 64%
4. Ill effects of consuming soft drinks	20(yes) 43(no)	30% 68%
5. Awareness of acids present in soft drinks	29(yes) 34(no)	46% 53%
6.awareness of consumption of soft drinks would erode tooth enamel	22(yes) 41(no)	30% 65%
7.experienced any sensitivity on taking aerated soft drinks	38(yes) 25(no)	60% 39%

MALE:

AGE GROUP: 10 – 19 years

Questions	Sample	%
1. Consumption of soft drinks among patients in varying dental office	53(yes) 34(no)	60% 39%
2.Frequency of consumption of soft drinks	28(daily basis), 18(weekly basis), 5(monthly basis)	32% 20% 5%
3. Necessity of drinking a	34(taste)	39%

soft drink	53(refreshment)	60%
4. Ill effects of consuming soft drinks	40(yes) 47(no)	45% 54%
5. Awareness of acids present in soft drinks	41(yes) 46(no)	47% 52%
6.awareness of consumption of soft drinks would erode tooth enamel	26(yes) 61(no)	30% 70%
7.experienced any sensitivity on taking aerated soft drinks	57(yes) 30(no)	65% 34%

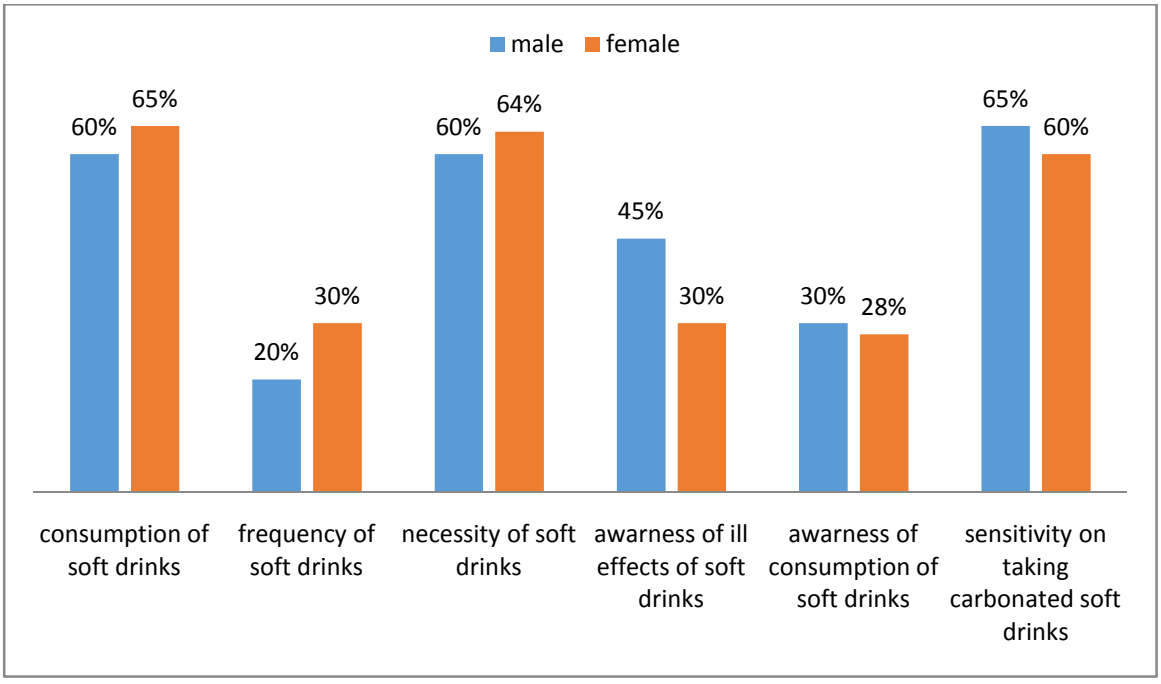


Figure 1: Carbonated drink consumption among adolescents

a)65% of female and 60% of male consumed soft drinks **b)**30% of female and 20% of had the habit of drinking soft drinks frequently **c)**64% of female and 60% of male were aware of the necessity of soft drinks **d)**45% of male and 30% of female were aware of the ill effects of soft drinks **e)**30%of malesand 28% of females were about of consumption of

soft drinks **f)**65% of males and 60% of females had sensitivity on taking carbonated soft drinks.

Discussion:

Soft drinks are now considered to be the most important etiological factors for enamel erosion(12). It is well known that disease loves acid, and particularly true of dental caries' common problem that is associated with consumption of a large number of soft drinks is the increased acid levels throughout the body. Dental cavities are often associated with carbonated beverag(13). This association is important because the amount of sugars that are consumed is important in forming caries, which is when a cavity affects only the enamel, the outer protective layer of a tooth(14). Soft drinks have been extensively investigated over a long period of time and are one of the principle factors in the etiology of extrinsic dental erosion(15).

Study was conducted to assess the knowledge of oral health hazards on taking carbonated soft drinks among patients in varying dental office. Our study was conducted between female and male of age group 10-19years. Among the female of age group 10-19years was found that 65% preferred carbonated soft drinks. Among the male of age group 10-19years was found that 60% preferred carbonated soft drinks. Another study by Gupta,et.al 2015 was found that 73% of students preferred carbonated drinks. An examination by priyadharshini et al indicated 78.5% of the young people among chennai populace are dependent on carbonated refreshments. Among the age group of 10-19years female was found that 30% consumed soft drinks in daily basis and among male of age group 10-19years was found that 20% consumed soft drinks in daily basis. Another study by Dehdari, et al Nov 2011 showed the result of frequency of using drinks is daily for 8.1% of students, 5-6 times per week for 10.8%, 3-4 times per week for 22.1%.

When asked about the ill effects of consuming soft drinks among females it was found that 30% were aware. When asked among male it was found that 45% were aware of it among age group of 10-19years.Another study by Heller kEet.al Nov 2001 showed a result of 78.47 % students had read about ill effects of carbonated drinks. In our present study it was found that 28% of females and 30% of males were aware that consumption of soft drinks would erode

tooth enamel and cause caries. Heller et al, using 1988–1994 U.S. data, determined that there were significant increases in caries rates in adults aged 25–34 with increased consumption of soft drinks.

About 60% of females and about 65% of males of age group 10-19years experienced sensitivity on taking aerated soft drinks. Study by Gupta,et.al 2015 showed only 48% of the students felt discomfort within few minutes of consuming soft drinks. A study byMahumuthaAffshanasuggest that erosion is as prevalent in chennai a,one adolescents. Possible reason may include Sampling variation or regional variations. Hence our present study found that females are consuming soft drinks at higher rate when compared to males also found that adolescents are at higher risk of consuming carbonated soft drinks

Conclusion:

The findings of the present study indicate a high rate of soft drink consumption among adolescents. In order to diminish the use of carbonated drinks by population it is necessary to provide appropriate training for the adolescents about the adverse effects of soft drinks and the oral health knowledge. Most of them preferred to drink aerated soft drinks in spite of being aware of the ill-effects of carbonated drinks. Hence the oral knowledge, attitude and practice among the population is still below the satisfactory level.

Corresponding Author

Geo Mani,

Senior Lecturer,Department of Pedodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai -600 077,Tamil Nadu, India.

Email Id: geosdch@gmail.com

Telephone number: 7358433246

Reference

1. Seow, W. K., and K. M. Thong. "Erosive effects of common beverages on extracted premolar teeth." *Australian Dental Journal* 50.3 (2005): 173-178.

2. Anna Rangan, Debra Hector, Jimmy Louie. Soft drinks, weight status and health: health professional update, *NSW Cluster of Public Health Nutrition*,2009:1-25.
3. Kaplowitz GJ. An update on the dangers of soda pop. *Dental assistant.* 2011 Jul 1;80(4):14.
4. Agostinelli G, Brown JM, Miller WR. Effects of normative feedback on consumption among heavy drinking college students. *Journal of drug education.* 1995 Mar;25(1):31-40.
5. Dehdari T, Mergen T. A survey of factors associated with soft drink consumption among secondary school students in Farooj city, 2010. *Journal of Jahrom University of Medical Sciences.* 2012 Jan 1;9(4):33-9.
6. Kishore, S.; Aggarwal, P.; Muzammil, K.KAP Study About Carbonated Drinks Among Medical and Nursing Students at Teaching Hospital, *JK Science;*2009,Oct-Dec 11(4),196-201.
7. Gandara BK, MSD and Edmond L Truelove MS. Diagnosis and management of dental erosion. *The Journal of Contemporary Dental Practice.* 1999 Dec 25;1(1):1-7.
8. Shipley S, Taylor K, Mitchell W. Identifying causes of dental erosion. *General dentistry.* 2005;53(1):73-5.
9. Ludwig DS, Majzoub JA, Al-Zahrani A, Dallal GE, Blanco I, Roberts SB. High glycemic index foods, overeating, and obesity. *Pediatrics.* 1999 Mar 1;103(3):e26-e26.
10. Cavadini C, Siega-Riz AM, Popkin BM. US adolescent food intake trends from 1965 to 1996. *Archives of disease in childhood.* 2000 Jul 1;83(1):18-24.
11. Erickson PR, Alevizos DL, Rindelaub DJ. Soft drinks: hard on teeth. *Northwest dentistry.* 2001;80(2):15-9.
12. Shreya S, Priya V. A study on the Effect of Aerated Drinks on Enamel Surface of the Tooth. *Research Journal of Pharmacy and Technology.* 2015 Aug 1;8(8):1137.
13. Khan F, Young WG, Law V, Priest J, Daley TJ. Cupped lesions of early onset dental erosion in young southeast Queensland adults. *Australian dental journal.* 2001 Jun 1;46(2):100-7.
14. Johansson AK, Lingström P, Imfeld T, Birkhed D. Influence of drinking method on tooth-surface pH in relation to dental erosion. *European journal of oral sciences.* 2004 Dec 1;112(6):484-9.

15. Naziya KB, Kumar RP, Arumugham IM, Sakthi DS. Measuring the buffering capacity of commercially available soft drinks in India: An in vitro study. *Journal of Advanced Pharmacy Education & Research*| Apr-Jun. 2017;7(2).
16. Gupta R, Solanki A, Sharma S, Gumber P, Sharma A, Upadhyay R.A knowledge, attitude and practices of soft drinks among adolescent students and their dental health: A Questionnaire study. Concern. 2015;1:1-5.
17. Tiwari RV, Megalamanegowdru J, Gupta A, Agrawal A, Parakh A, Pagaria S, Sahu A. Knowledge, attitude and practice of tobacco use and its impact on oral health status of 12 and 15 year-old school children of Chhattisgarh, India. *Asian Pac J Cancer Prev.* 2014 Jan 1;15:10129-35
18. Dehdari T, Mergen T. A survey of factors associated with soft drink consumption among secondary school students in Farooj city, 2010. *Journal of Jahrom University of Medical Sciences.* 2012 Jan 1;9(4):33-9.
19. Heller KE, Burt BA, Eklund SA. Sugared soda consumption and dental caries in the United States. *Journal of Dental Research.* 2001 Oct;80(10):1949-53.
20. Gupta R, Solanki A, Sharma S, Gumber P, Sharma A, Upadhyay R.A knowledge, attitude and practices of soft drinks among adolescent students and their dental health: A Questionnaire study. *Concern.* 2015;1:1-5
21. Affshana M. Acid Erosion of Teeth in a Pediatric Population. *Journal of Pharmaceutical Sciences and Research.* 2015 Jul 1;7(7):435.

A Survey on Awareness of Protein Energy Malnutrition in Below Poverty Line People

Thejeswar EP [1], Brundha M P [2], Thummala Bhagya Lakshmi [3]

Graduate Student [1],Senior lecturer[2],Department of General Pathology

Senior Lecturer [3],Department of Pedodontics and Preventive Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: India has provided with a number of nutrition analysing programs, but malnutrition remains highly widespread in the poor states of the country. Malnutrition results due to the imbalance in needs of the body to the intake of nutrients. It may be due to poverty, lack of awareness and illiteracy.The aim of the study is to analyse the awareness of protein malnutrition among below poverty line people, by questionnaire method.

Material and method: The present study was undertaken for below poverty line people from rural areas in Chennai district, Tamil Nadu, India. A questionnaire consisting of 10 questions were framed based on knowledge about protein-energy malnutrition and they were given to 300 below poverty line people of Chennai.

Result: This study showed that (74%) people aware of protein-energy malnutrition, and family members were the most common source of information on protein-energy malnutrition.

Conclusion: Protein malnutrition might have a serious impact on health especially in growing children as well as in adults. Creating awareness about the importance of proteins among people who are under below poverty line becomes a significant factor of study.

Key Words: *Protein energy malnutrition, Marasmus, Kwashiorkor, Poverty, children.*

Introduction

India has provided with a number of nutrition analysing programs, but malnutrition remains highly widespread in the poor states of the country. Malnutrition results due to the imbalance in needs of the body to the intake of nutrients. It may be due to poverty, lack of awareness and illiteracy[1]. In the present study, Protein Energy Malnutrition (PEM) in the below poverty line people from rural areas was measured. Moderate and severe malnutrition was observed in the various categories of age.In additional occupation and annual income were analysed in the study and it was found that these conditions may be the main reason for the Protein-energy malnutrition. A wide variety of development actions are needed to improve the food security and nutrition among the population[2]. Nutritional problems in India have their roots in poverty and inequality[3]. Poverty which restrains an individual or family to establish command over "Commodity Bundles with Enough Food" and inequality among other things places a disproportionate burden of ill health and under-nutrition on women and children[4]. Malnutrition and its associated disease conditions can be caused by eating too little, eating too much, or eating an unbalanced diet that lacks necessary nutrient[5]. Under-nutrition is defined as failure to consume adequate energy, protein, and micronutrients to meet the basic requirements for body maintenance, growth, and development[6]. Malnutrition is one of the most devastating problems worldwide and is intensively linked with poverty[7-8]. This paper mainly focuses on the malnutrition and health status of below poverty line population. The oppression of people socially and culturally means they have less access to everything, including food, resources, healthcare, community support, and information. Hence, significant steps should be taken to educate and to make them aware against the terrible problems of malnutrition and thereby providing proper awareness for below poverty line people in the rural areas is the significant factor of this research.

Materials and Methods

The present study was undertaken for below poverty line people from rural areas in Chennai district, Tamil Nadu, India. A questionnaire consisting of 10 questions were framed based on knowledge about protein-energy malnutrition and they were given to 300 below poverty line people of Chennai. This was a cross-sectional Study; hence it has a self-developed questionnaire, consisting of both open and close-ended questions related to various aspects of knowledge about protein-energy malnutrition. Study population between age group 20-50 years were selected. The aim and purpose of the study were clearly explained and discussed with them population prior to the survey. The respondents were interviewed with the questions and corresponding answers were marked.

The questionnaire was in two parts. The first part had questions on demographic information of the respondents such as Name, age, gender, etc. The second part contained questions such as basic knowledge

on protein-energy malnutrition, the source of knowledge, causes, and risk factors as well as about treatment of it. The questionnaire was framed in such a way for easy understanding and filling by respondents.

Results

This questionnaire is based on the awareness of protein-energy malnutrition, which includes causes, signs, symptoms, investigation, prevention and treatment. Out of 300 responders, 174 people were male, while remaining 126 people were female. This study showed that (74%) people aware of protein-energy malnutrition, and family members were the most common source of information on protein-energy malnutrition. People think that dietary deficiency is the major cause of protein-energy malnutrition and knowledge about the risk factors were mostly identified as low socio-economic conditions. People were not aware of different types and laboratory investigations for protein-energy malnutrition. Provision of immunization was found to be an important factor in managing a child. This study also showed that most people were aware of symptoms associated with protein-energy malnutrition but not aware of its risk factors.

Discussion

When enquired about the source of knowledge, the Family was the most common source of knowledge for protein-energy malnutrition (34%), followed by Friends (26%), Media (16%), Relatives (10%), office (6%), Doctors (6%), while rest (2%) were not aware of it. The dietary deficiency was the most common cause of protein-energy malnutrition (44%), followed by social inequality (34%), Drought (8%), while (14%) population selected all the above. When questioned about the risk factors of protein-energy malnutrition, (44%) were aware of the risk factors, followed by ignorance of parents about the importance of child's nutrition (30%), child abuse (10%), and infections like diarrhoea (6%); whereas rest (4%) people said they don't know about it. This study also showed that (60%) people do not know the types of protein-energy malnutrition, while (16%) and (14%) people know about marasmus and Kwashiorkor respectively. Provision of immunization was the most common way of managing child with protein-energy malnutrition, followed by correction of specific nutrient diseases, treating super-imposed infection. According to them the Extreme weakness and fatigue were the main symptom of Marasmus, followed by low body weight, chronic diarrhoea, Dryness of skin and peeling of skin and rest (30%) people doesn't know any symptoms of marasmus. The symptoms of kwashiorkor were known to grow or gain weight, loss of muscle mass, fatigue, Diarrhoea, and oedema. The healthy and nutritional diet (66%) is the most common treatment of choice for marasmus, followed by intravenous fluids (16%), oral rehydration solution (10%), Electrolyte imbalance (4%), whereas (2%) people were not aware of it. Increased vitamin and mineral supplements (36%) and increased carbohydrate and protein intake (32%) are the most common treatment for the Kwashiorkor, followed by Electrolyte imbalance (12%), Hypo-glycaemia (10%), while (10%) people do not know about it. Several reports from Germany, the United Kingdom, the United States, and France as recently as the last

decade reported the prevalence of acute malnutrition in hospitalized paediatric patients to be between 6.1%and 24%[9-11]. Global nutritional assessment in children admitted for elective surgery in a tertiary referral paediatric hospital in Toronto and found that (51%) of children were malnourished (36%) had moderate malnutrition, and (15%) had severe malnutrition. Despite differences in methods of assessing malnutrition, these studies clearly document a significant prevalence of malnutrition even in the developed world, particularly in hospitalized paediatric patients. In 2008, Pawellek and colleagues, using Waterlow's criteria, reported 24.1% of patients in a tertiary paediatric hospital in Germany to be malnourished (<90th percentile weight for height), of which 17.9% were mild, 4.4% moderate, and 1.7% severely malnourished[12]. The prevalence of malnutrition varied depending on their underlying medical conditions and ranged from 40% in patients with neurologic diseases, to 34.5% in those with infectious disease, 33.3% in those with cystic fibrosis, 28.6% in those with cardiovascular disease, 27.3% in oncology patients, and 23.6% in those with gastrointestinal disease[13]. Patients with multiple diagnoses were most likely to be malnourished (43.8%). The prevalence and degree of acute PEM in hospitalized paediatric patients are similar to those observed by Hendricks and colleagues almost a decade ago using the same criteria[14]. One of the difficulties in being able to compare prevalence between studies and Centres is the lack of consensus on a uniform definition of malnutrition and its grades of severity. A recent review highlighted the issues of lack of uniform screening tools, poor nutritional data collection, and early identification of those at risk of developing PEM[15]. Worldwide, only 36 countries account for 90% of all stunted children when countries with stunting prevalence of at least 20% were considered. India alone has 34% of the world's stunted children because of its large population, although there is significant variation between its states[16].

Primary protein-energy malnutrition is caused by lack of access to adequate nutrient intake and usually affects children and elderly persons[17]. The functional and structural abnormalities associated with primary protein-energy malnutrition are often reversible with nutritional therapy. However, prolonged primary protein-energy malnutrition can cause irreversible changes in organ function and growth[18].

Secondary protein-energy malnutrition is caused by illnesses that alter appetite, digestion, absorption, or nutrient metabolism and can be divided into three general, but often overlapping, categories:

(1) Diseases that affect gastrointestinal tract function,

(2) Wasting disorders, and

(3) Critical illness.

Gastrointestinal disease can cause protein-energy malnutrition by pre-mucosal (maldigestion), mucosal (malabsorption), or post-mucosal (lymphatic obstruction) defects. The nutritional status of patients with protein-energy malnutrition caused by gastrointestinal tract dysfunction can often be restored to normal if adequate nutritional support can be provided by dietary manipulations, enteral tube feeding, or parenteral nutrition. Wasting disorders, such as cancer, acquired immunodeficiency syndrome (AIDS), and

rheumatologic diseases, are characterized by involuntary loss of body weight and muscle mass in the setting of a chronic illness. More than 85% of the students consume balanced diet among a dental school[19].

These patients often experience wasting because of

(1) Inadequate nutrient intake related to anorexia and possibly gastrointestinal tract dysfunction and

(2) Metabolic abnormalities caused by alterations in regulatory hormones, cytokines, and systemic inflammation.

The alterations in metabolism are responsible for the greater loss of muscle tissue observed in these patients than in those with pure starvation or semi starvation. It was proved that on consuming dates boiled in milk in an empty stomach helps to increase the haemoglobin level[20]. Restoration of muscle mass is unlikely with nutritional support unless the underlying inflammatory disease is corrected. Weight gain that occurs after nutritional support is started is usually caused by increases in fat mass and body water, without significant increases in muscle tissue. Patients with critical illness exhibit marked metabolic alterations, manifested by increased energy expenditure, altered endogenous glucose production and lipolytic rates, and protein breakdown. About 47% of the people prefer to get self-help from the internet to maintain their their nutritional status[21]. Therefore, protein and energy requirements are increased in critically ill patients. However, providing aggressive nutritional support may ameliorate but does not prevent net lean tissue losses without correction of the underlying illness or injury.

It has been claimed that eating balanced diet is beneficial for living healthy. However, some individuals in our society could not stuck with only this limit because chocolate and candies, for example are the main part of their life, which make them extremely happily. Various factors like social, economic status, age, sex, social class, dietary habits are some of the major etiological factors for malnutrition[22]. As a consequence, diet means for many kind of entertainment and happiness. Eventually, without specific food, they may feel tired and unhealthy[23]. To conclude, the balanced diet has brought plenty of positives effects, so once the individual maintains on this nutritious diet, the potential cause of having any disorder would be rare. Nevertheless, those people, who are against eating balanced diets, might understand the value of this diet in future.

Conclusion

This study showed that overall awareness of protein-energy malnutrition was found to be (**74%**) population in which (**66%)** people were mostly aware of health and nutritional diet as a treatment of choice for marasmus. Consequently, unawareness of protein-energy malnutrition was found to be in (**26%**) population. Even though overall awareness was good for protein-energy malnutrition, proper counselling and education must be given to the risk factors, prevention, Treatment, Symptoms of it. Public health programs, programs from NGOs and medical camps must be conducted to educate the people about protein-energy malnutrition.

Acknowledgement

We would like to express our gratitude to all the respondents for spending their valuable time to answer our questionnaire that helped us to successfully complete our study.

Corresponding Author

Thummala Bhagya Lakshmi

Senior Lecturer, Department of Pedodontics and Preventive Dentistry

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

162, Poonamallee High Road, Chennai – 600077, Tamilnadu, India.

Email ID: Bhagya.thummala@yahoo.co.in

Telephone Number: 9642432345.

References

1. World Health Organization. The World Health Report. Reducing Risks, Promoting Healthy Life. Geneva, Switzerland: World Health Organization; 2002.
2. Barret H, Browne A. Health Hygiene, maternal education: Evidence from Kenya. SocSci Med1996;431:579-1590.
3. Dettwyler KA. Breastfeeding and weaning in Kakamega: cultural context and hard data. Soc Sci Med 1987;24:633-644.
4. Madzingira N. Malnutrition in children under five in Zimbabwe: effect of socioeconomic factors and disease. Soc Bio 1995;42(3):239-46.
5. Svedberg P. Undernutrition – gender bias in sub-Saharan Africa. J DevelStud1996;32:933-943.
6. Serventi M, Dal Lago AM, DN Kimaro. Early cessation of breastfeeding as a major cause of serious malnutrition in under twos: a hospital-based study – Vihiga district, Kenya. EAfrMed J1995;72:132-34.
7. UNICEF The state of the World's children. Oxford University Press, Oxford 1998.
8. Subramanian M, Subrahmanyam G. Determinants of protein energy malnutrition among rural preschool children. Journal of Evolution of Medical and Dental Sciences 2013;9(3):157-62.
9. Joosten KFM, Hulst JM. Prevalence of malnutrition in pediatric hospital patients.Curr Opin Pediatr 2008;20:590–6.
10. Pawellek I, Dokoupil K, Koletzko B. Prevalence of malnutrition in paediatric hospital patients. Clin Nutr 2008;27:72–76.

11. Hendricks KM, Duggan C, Gallagher L, et al. Malnutrition in hospitalized pediatric patients. Current prevalence. Arch Pediatr Adolesc Med 1995;149:1118–22.
12. Secker DJ, Jeejeebhoy KN. Subjective global nutritional assessment for children.Am J Clin Nutr 2007;85:1083–89.
13. Pawellek I, Dokoupil K, Koletzko B. Prevalence of malnutrition in paediatric hospital patients. Clin Nutr 2008;27:72–74.
14. Hendricks KM, Duggan C, Gallagher L, et al. Malnutrition in hospitalized pediatric patients. Current prevalence. Arch Pediatr Adolesc Med1995;149:1118–22.
15. Joosten KFM, Hulst JM. Prevalence of malnutrition in paediatric hospital patients.Curr Opin Pediatr2008;20:590–596.
16. Black RE, Allen LH, Bhutta ZA. Maternal and child undernutrition: global and regional exposures and health consequences. Lancet2008;371:243–260.
17. Sahbanathul M. Prevalence of Protein Energy Malnutrition Among Children at Slums of North Chennai. Int j of sci research2013;2(10)221-227.
18. Oyedeji GA. Socioeconomic and cultural background of hospitalised children in Ilesha. Niger J Paediatr1985;12:11-12.
19. Aparnaa M, Vishnupriya V, Gayathri R, Mohan SK. Intake of Balanced Diet among Dental Students-An In Campus Survey. Journal of Pharmaceutical Sciences and Research2016;8(6):453-457.
20. Krishnaa PK, Priya AJ. Effect of Dates in Milk on Haemoglobin Level of Blood. Journal of Pharmaceutical Sciences and Research2017;9(9):1536-38.
21. Bano AM, Vishnupriya V, Gayathri R. Nutritional awareness among Adolescents. Asian Journal of Research in Chemistry2016;9(7):898-900.
22. Lakshmi C, Maragathavalli G. Prevalence of nutrition anaemia among patients reporting to Saveetha dental college. Int J Adv Res 2017;5(3),44-49.
23. National Institute for Health and Clinical Excellence. Behaviour Change at Population, Community and Individual Levels. London: Department of Health, 2007.

A Survey On Complete Denture Patient's Satisfaction

Harini. N.S[1]., Pon Kirubha Robin[2], Sankari.M[3]
Graduate student [1],Senior Lecturer [2],Department of Prosthodontics
Professor [3],Department of Periodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Aim: The aim of study is to determine the level of satisfaction of the complete denture patients.

Objective: To assess the satisfactory level of complete denture patients after the treatment, to analyse the drawbacks and to enhance the dental treatment in future.

Background: Complete denture is one of the challenging dental procedure in prosthodontics which when done perfectly, will make the smile and hearts of the patients to utmost beauty. Thus, analysing the satisfactory level of the patients is of most value so that any discomfort for the patient can be corrected immediately which will again improve the status and bonding between the patient as well as the doctor.

Material and methods :50 edentulous patients with their maxillary and mandibular complete dentures in use who were seeking prosthetic treatment for relining of their old dentures or the fabrication of new ones were selected for the study. They were surveyed about their satisfaction with the treatment provided with their complete denture. The patients were also informed that their answers will be confidential. Then, the patients photograph along with their oral cavity have been captured for reference. It has been informed to the patients that their photographs captured is only collected for a personal data. The survey ended by collecting the patients' signature with date for the survey reference.

Conclusion: It is been concluded that patients wearing complete denture are mostly satisfied with the treatment but some are not satisfied. This may be due to the personality traits and the behaviour and attitude of the dentist. Moreover, we can also see that there is some amount of

dissatisfactory rate among the patients due to multiple number of visits, outcome of the denture and due to facial expressions. These can be corrected by the dentists by giving an excellent denture with good retention, stability and support and also with most esthetic prosthesis and with minimal visits which should have shorter appointments too which may enhance the satisfactory level for both the patients and dentists. Thus, it is expected that all the treatments provided to the complete denture patients in the future must be satisfactory.

Key Words:*Complete denture, satisfaction, patient, dentist, treatment*

Introduction

The prevalence of edentulism has been decreased during the last three decades especially in the developing countries[1]. However, the interesting fact is the level of edentulism is still high in the developing countries.[2]This is because more people worldwide are advancing into the old age and thus a growing number of edentulous patients are expected [3].Edentulism has a deep impact on the quality of life, which affects an individual's physiological, biological, social and psychological state [4].It can also cause a state of depression which occurs due to disturbances in speech, esthetics, mastication, and a feeling of inferiority because an important part of the person has been lost [5].Thus the treatment of choice for many cases is still the placement of conventional complete dentures [6].However, a considerable proportion of denture wearers are dissatisfied with their complete dentures[7].Several studies have investigated that factors that may affect the patients' satisfaction with their complete dentures may include denture technical quality, condition of residual ridges and patients' gender, age, previous denture experience, and personality [8-9]. Some studies also suggest that patients must always be handled with care or else they might not be satisfied [10].The loss of all the teeth is a traumatic event in a person's life. Subsequent adaptation to complete dentures is influenced considerably by the ability of the person to come to terms with the edentulous condition. Satisfaction with dentures is thought to be influenced primarily by the person's personality [11].Clinicians need to assess the values of a wide range of factors prior to arriving at a decision that represents optimal care for their patient. These factors include clinical factor patient values, the available research evidence, clinical guidelines, their previous clinical experience and medico-legal implications [12]. Conventional complete denture prostheses have been widely used for the replacement of missing dentition [13]. Simpler methods of complete denture fabrication and technique

helps to reduce appointments and offer better compliance to the patients with respect to time, logistic and expenses [14]. Compilation of the various denture techniques in complete denture fabrication shows the evolution of denture techniques that led to reduced number of appointments, without compromising the outcome. Reduced appointments offer better compliance to the patients with respect to time, logistics and expenses [15].Over the past century, the technique of complete denture fabrication has evolved. Today, the most likely technique used in complete denture fabrication is the five appointment complete denture, which is the most suitable method of denture construction[16].Complete denture teaching, about four decades ago is composed of lectures, followed by clinical work, and later on preclinical complete denture curriculum was established[17]. Preclinical work includes demonstrations for the purpose of effective performance by the students[18]. Now, living in the fast-paced world, advanced learning method has been implemented such as video lectures with demonstrations and visual aids have been introduced[19]. Provided with advanced learning method with technology in dental education, studies show that there are still significant ineptness in complete denture fabrication even after graduation. This study sparked the need to improve competency of complete denture fabrication in undergraduates. Using better clinical materials, improving training methods and by making the technique simpler, competency in fabricating complete dentures can be accomplished. This survey conveys the proportion of the patients wearing the complete denture whether they are satisfied or not satisfied[20].Thus analysing this data will give us a clean knowledge on patients, their attitude, their knowledge towards dentistry and the skills of the dentist, and their knowledge towards dentistry.

Materials and methods

50 edentulous patients with their maxillary and mandibular complete dentures in use who were seeking prosthetic treatment for relining of their old dentures or the fabrication of new ones were selected. They were surveyed about their satisfaction with the treatment provided with their complete denture. The patients were also informed that their answers will be confidential. Then, the patients photograph along with their oral cavity have been captured for reference. It has been informed to the patients that their photographs captured is only collected for a personal data. The survey ended by collecting the patients' signature with date for the survey reference. This survey is now brought up by concluding with the results which are conveyed along with the statistics. It has also been discussed about the various

causes which affect the dentures such as halitosis, adaptation etc, and are briefly discussed in detail. This survey also depicts that the preparations of the complete dentures and the way of treatment towards the patients must be enhanced in such a way that the results are satisfactory and it is also satisfied to the patients.

Results:-

The results obtained were that almost 70% of the patients are satisfied with their complete dentures while the remaining are not satisfied. The statistics for the results are given below:

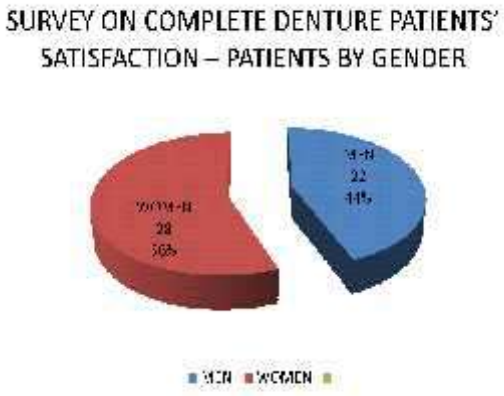
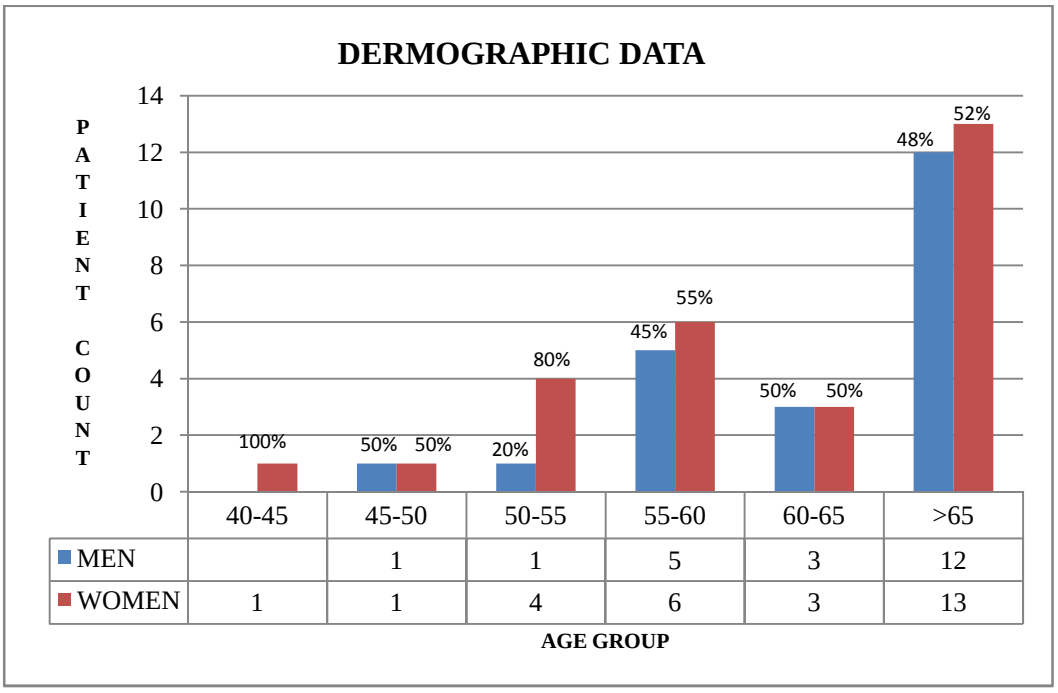
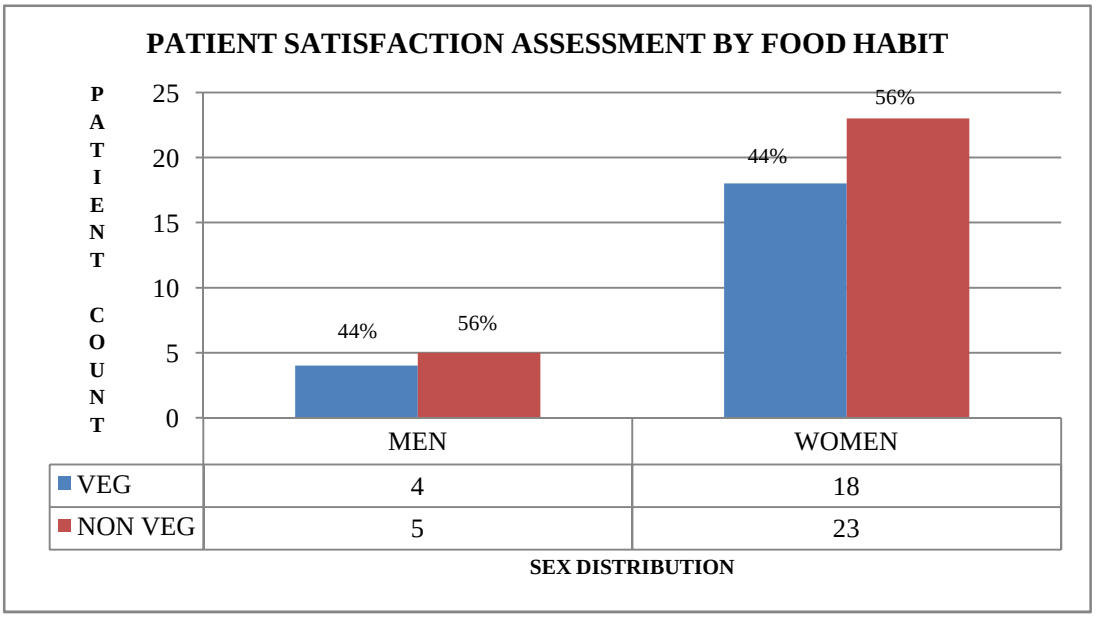


Figure-1 Gender

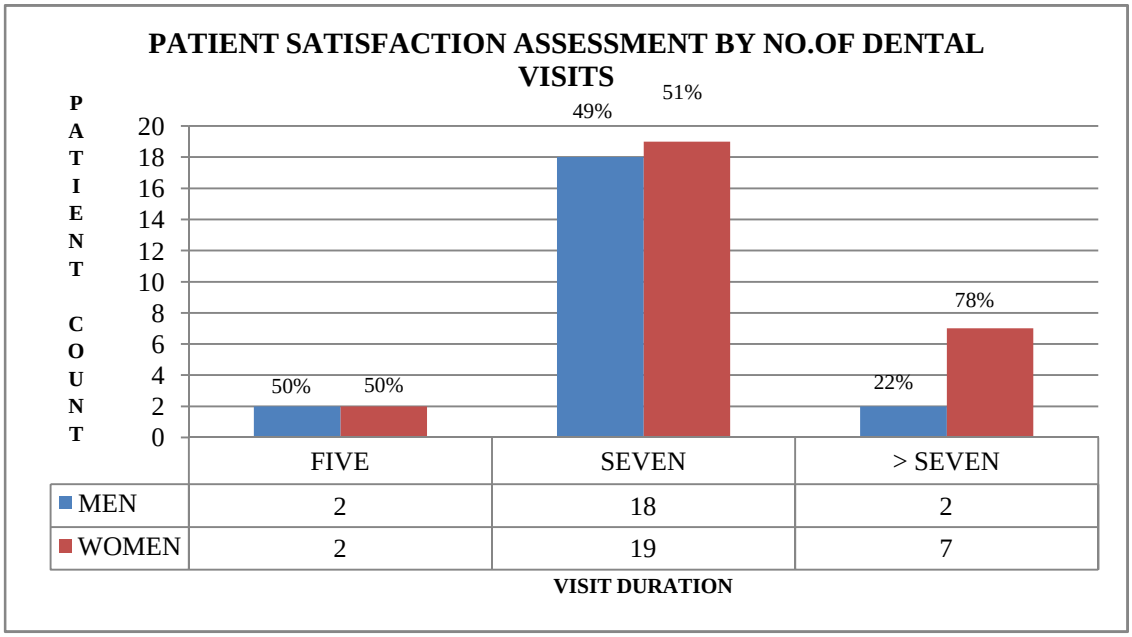
Graph -1 Age group



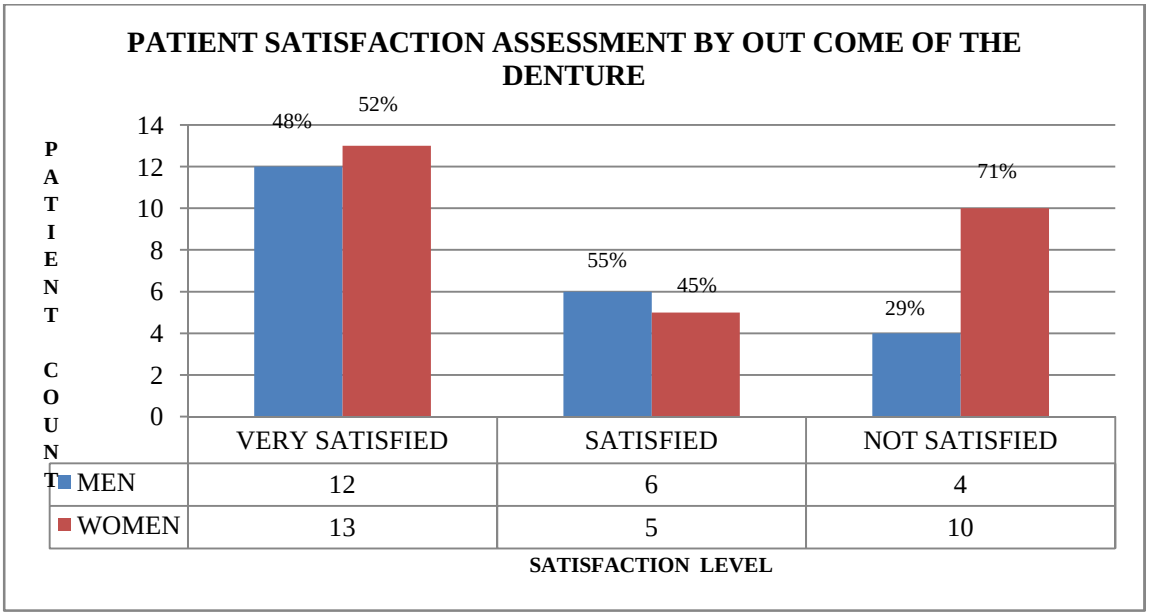
Graph 2 Food habit



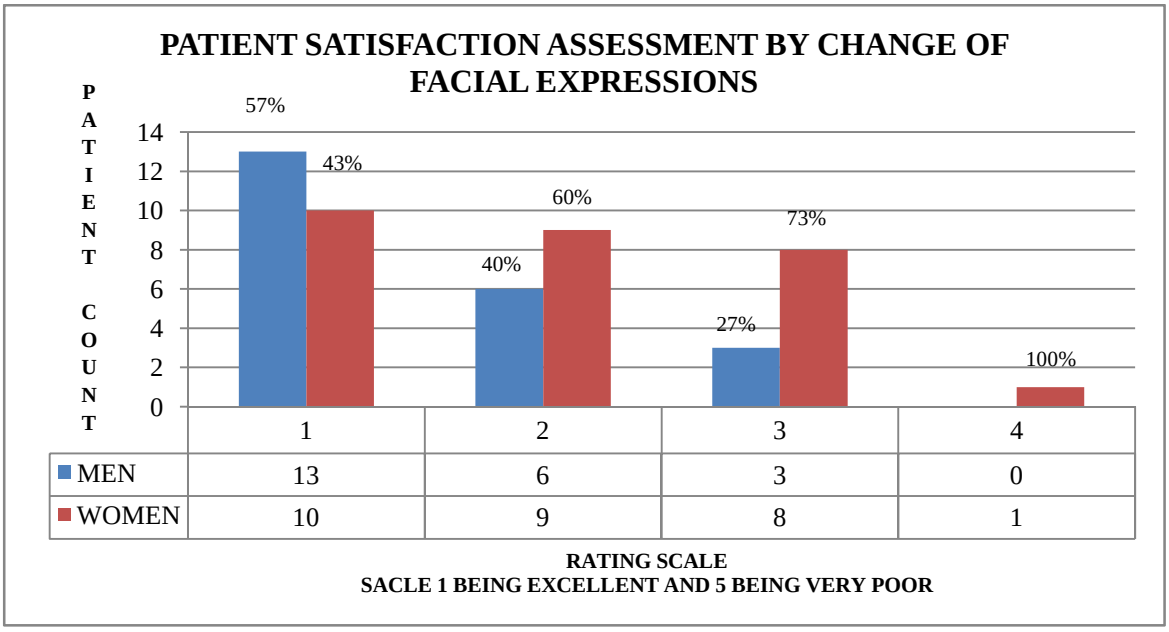
Graph 3 Number of visits by the patients



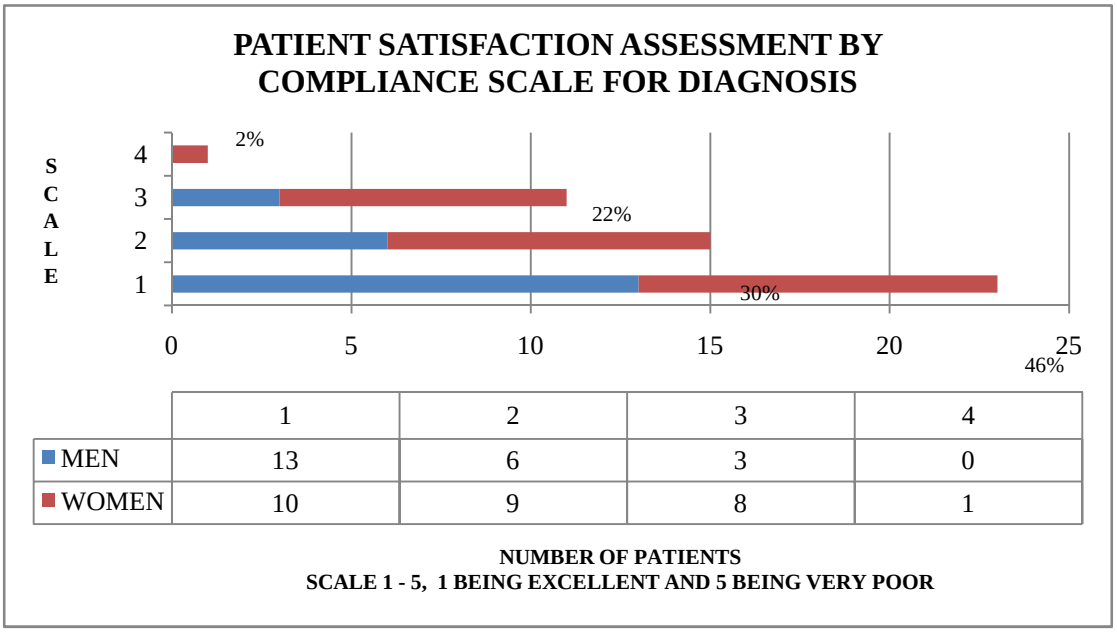
Graph 4 Patient's satisfaction level



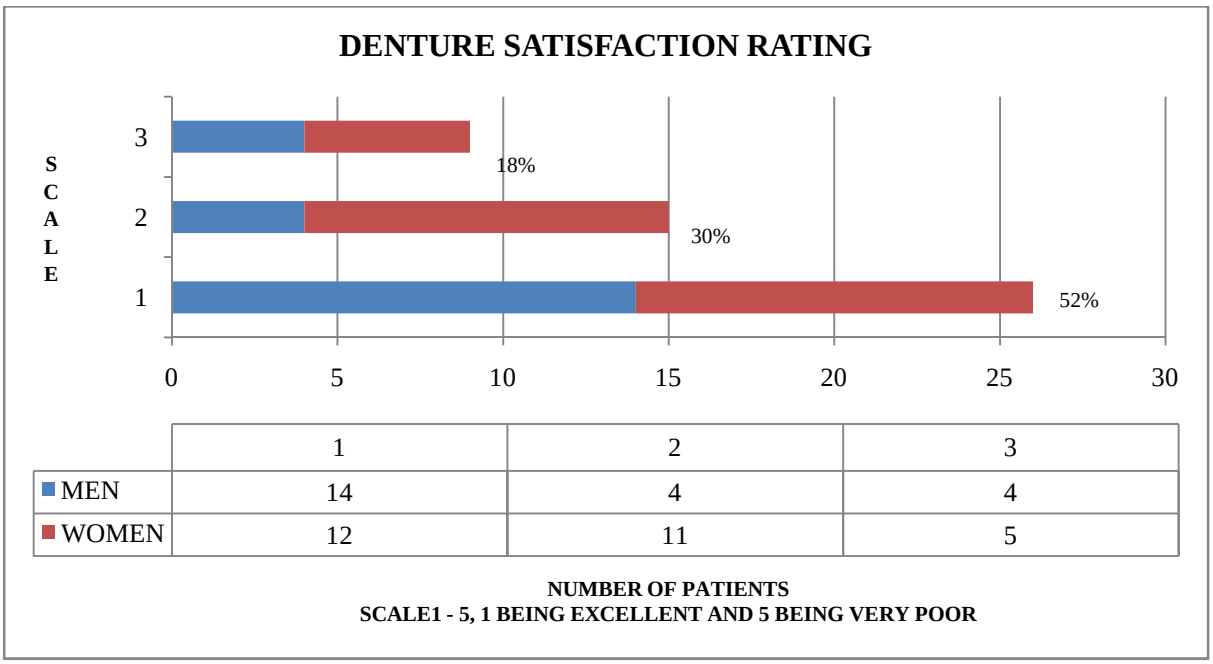
Graph-5 Facial expressions of the patients



Graph-6 Diagnosis satisfaction rating



Graph -7 Denture satisfaction rating



	1	2	3
MEN	14	4	4
WOMEN	12	11	5

NUMBER OF PATIENTS
SCALE1 - 5, 1 BEING EXCELLENT AND 5 BEING VERY POOR

Figure -2 Questionnaire page 1

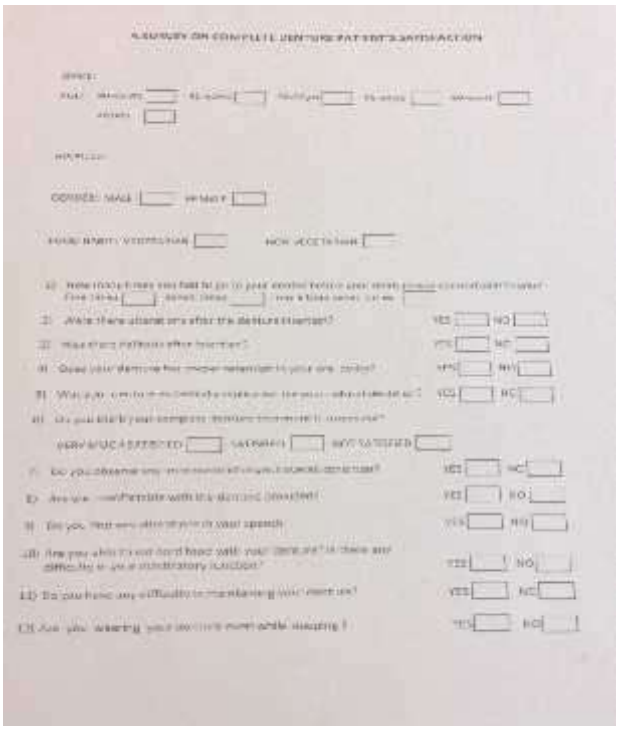
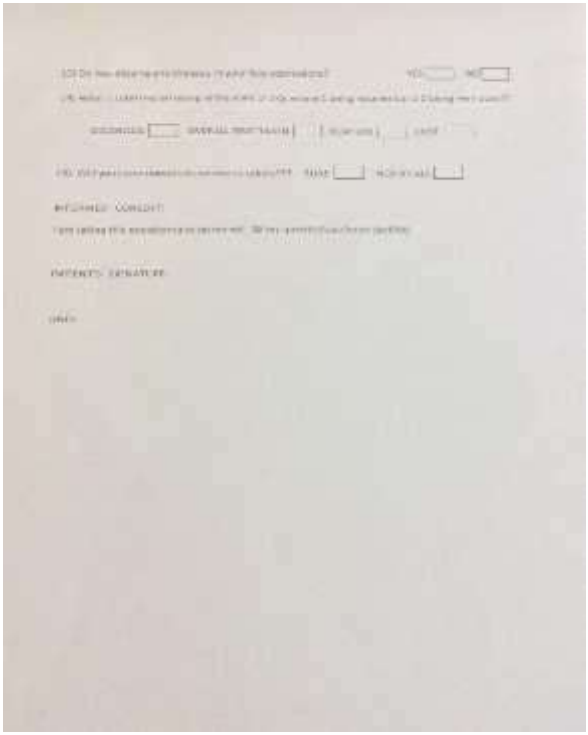


Figure -3 Questionnaire page 2



Discussion

Based on the facts of the survey and the above analysis clearly proves that women are more exhibited for the treatment of complete dentures than men. Further people with non vegetarian food habits are more likely to lose their teeth and going for complete denture. People in the age group of 55-60 and >65 are the ones who are affected mostly. Further, the number of visits is mostly 7 and >7 visits which needs to be addressed considering the age factor of the patients. While 50% of the patients are very much satisfied with their dentures provided, about 38% of the patients are not satisfied of which most of the women which is 71% of the total non satisfied category. Due attention and care to be improved while handling women patients for enhancement of satisfaction levels[11]. From the survey it is clear that most of the patients has visited their doctors seven times or more until their denture is comfortable to them. This means that lack of attention and concentration is the only cause for more number of visits[12]. Dentures also causes ulcerations and halitosis if it is not maintained properly. If the dentures are not maintained in a hygienic way, then microorganisms like bacteria, etc accumulates on the dentures and causes ulcerations[13]. Due to this there is progressive caries and this causes halitosis. Maintenance of the dentures is also important and it is not a difficult way. Placing the dentures in a compact and clean container which is filled with water cleans the dentures. Again, while replacing the dentures has to be replaced with fresh water[14-15].Thus hygiene also plays a very important role in maintenance of the dentures, where this also affects the outcome as well as the dental treatment. Patterns of tooth loss and retention are also changing. One of the key factors affecting the outcome of the treatment is the impression procedure involved in the fabrication of complete denture prosthesis. Various impression philosophies have been proposed over years by various authors, out of which the selective-pressure impression technique is most widely accepted by all since it produces a denture of good outcome[21]. Nine visit technique is the most time consuming complete denture fabrication method and was popular in 1950's. From this technique, complete denture fabrication started to evolve to simpler techniques. The nine visit denture technique consists of nine appointments, where the first two are diagnostic appointments and the remaining appointments are treatment appointments [22]. All these are techniques to improve the quality of the complete denture prosthesis which will even more satisfy the patients. Thus higher the quality, more the satisfactory rate is

assured.

Conclusion

It is been concluded that patients wearing complete denture are mostly satisfied with the treatment but some are not satisfied. This may be due to the personality traits and the behaviour and attitude of the dentist. Moreover, we can also see that there is some amount of dissatisfactory rate among the patients due to multiple number of visits, outcome of the denture and due to facial expressions. These can be corrected by the dentists by giving an excellent denture with good retention, stability and support and also with most esthetic prosthesis and with minimal visits which should have shorter appointments too which may enhance the satisfactory level for both the patients and dentists. Thus, it is expected that all the treatments provided to the complete denture patients in the future must be satisfactory.

Corresponding Author

M.Sankari
Professor,Department of Periodontics
Saveetha Dental College,Saveetha Institute of Medical And Technical Sciences,
Saveetha University, Chennai, India
162, Poonamalle high Road,Chennai -600077, Tamil Nadu, India.
Email.msankari@gmail.com
Telephone No.:9840285905

References

1) Bilhan H, Erdogan O ,Ergin S, Celik M, Ates G, Geckili O (2012) *Complication rates and patient satisfaction with complete dentures.*J Adv Prosthodont 4, 109-115

2) Steele JG, Treasure E,Pitts NB, Morris J, Bradnock G(2000); Total tooth loss in the united kingdom in 1998 and implications for the future. Br Dent J. 2000 Dec 9;189(11):598-603.

3) Beck CB, Bates JF, Basker RM, Gutteridge DL,Harrison A (1993*) A survey of the dissatisfied denture patient. Eur J Prosthodont Restor* Dent2, 73-78.

4) Inoue M,JohnMT, Tsukasaki H, Furuyama C, Baba K (2011) *Denture quality has a minimal effect on health-related quality of life in patients with complete dentures.*J Oral Rehabil 38, 818-826.

5) Douglass CW, Watson AJ (2002) *Future needs for fixed and removable partial dentures in the United States* .J Prosthetdent 87, 9-14.

6) Zitzmann NU , Marinello CP (2006) *Survey of treatment seeking complete dentures wearers, concerning tooth loss, retention, behaviour and treatment expectations.*Schweiz Monatsschr Zahnmed 116,229-236

7) Karacaer O Dogan OM, Tincer T, Dogan A (2001*) Reinforcement of maxillary dentures with silane-treated ultra high modules polyethylene fibres.*J Oral Sei 43,103-107.

8) Silverman MM (2001) *The speaking method in measuring vertical dimension* J Prosthet Dent 85,427-431.

9) Basker RM , Davenport JC (2002*) The Prosthetic treatment of the edentulous patients, fourth edition, Blackwell, Oxford,*269-311.

10) Fenton AH (2004)*Selecting and arranging prosthetic teeth and occlusion for the edentulous patients. In: Prosthodontic treatment for edentulous: complete dentures and implant-supported prostheses*, 12th edition, Zarb GA, Bolender CL, Eckert SE, Fenton AH, Jacob RF, Mericske-Stern R eds, C.V.Mosby, St.Louis, 298-329.

11) Mary Smith, B.D.S., L.D.S.R.C.S., MS.* The Ohio State University, College of Dentistry, Columbus, Ohio; *Measurement of personality traits and their relation to patient satisfaction with complete dentures, removable prosthodontics;*146,132-146

12) Vaishali.S. and Dr. Marian Anand Bennis, Department of Prosthodontics, Saveetha Dental College, 162, P.H. Road, Chennai, Tamilnadu 600077, *attitudes towards prosthodontic clinical decision-making for edentulous patients among dental students*, International Journal of Current Research Vol. 9, Issue, 06, pp.51871-51875, June, 2017.

13) *Vallimalar, A. and Dr. Suresh, V., 162, Poonamalle High Road, Saveetha Denta College, Girls Hostel, Chennai-600077, India*; an investigation into the patient satisfactory level of wearing a mandibular complete denture;*International Journal of Recent Advances in Multidisciplinary Research Vol. 02, Issue 07, pp. 0530-0533, July, 2015

14) Nurul Syamimi binti Mohd Azlan Sunil1 and *Dr. Preetham Nittla;*review on various complete denture techniques;*Bachelor of Dental Surgery (year 1), Saveetha Dental College, Chennai-600077. 2. Professor, Department of Prosthodontic, Saveetha Dental College and Hospitals, No.162, PH road, Chennai- 600077; Article DOI: 10.21474/IJAR01/3878 DOI URL: http://dx.doi.org/10.21474/IJAR01/3878.

15) Petropoulos, V.C. and B. Rashedi,*Current concepts and techniques in complete denture final impression procedures.*Journal of Prosthodontics, 2003. 12(4): p. 280-287

16) Clark, R., D. Radford, and M. Fenlon,*The future of teaching of complete denture construction to undergraduates in the UK: is a replacement denture technique the answer?* British dental journal, 2004. 196(9): p. 571-575.

17) Clark, R.,*The future of teaching of complete denture construction to undergraduates.*British dental journal, 2002. 193(1): p. 13-14.

18) Faraone, K., P. Garrett, and E. Romberg, *A blended learning approach to teaching pre- clinical complete denture prosthodontics.*European Journal of Dental Education, 2013. 17(1): p. e22-e27.

19) Iwaki, M;*Live Broadcast Lectures on Complete Denture Prosthodontics at Tokyo Medical and Dental University: Comparison of Two Years.* Journal of dental education, 2013. 77(3): p. 323-330.

20) Kawai, Y; *Do traditional techniques produce better conventional complete dentures than simplified techniques?* Journal of dentistry, 2005. 33(8): p. 659-668.

21) Ashish R Jain; Dhanraj.M; Department of Prosthodontics, Saveetha Dental College and Hospitals, Saveetha University, Chennai, India; *A Clinical Review of Spacer Design for Conventional Complete Denture.* Biol Med (Aligarh); 2016. 8(5): p. 2-5.

22) Zarb, G.A., *Boucher's Prosthodontic Treatment for Edentulous Patients*. 1997: Mosby 13th edition.

A Survey on Consequences of Self-Medication Versus Prescribed Medication

Thejeswar EP [1], Anitha Roy [2], Thummala Bhagya Lakshmi [3]
Graduate Student, Professor,Department of Pharmacology
Senior Lecturer,Department of Pedodontics and Preventive Dentistry
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Aim: To compare the pattern of self-medication and prescribed medication among the population in Chennai, India and to tabulate results and thereby creating awareness on population.

Materials and Methods:A questionnaire consisting of 15 questions were framed based on knowledge and usage of self-administered drugs as well as prescribed drugs. They were given to 100 people aged between 20-50 years without any gender difference. This study has a self-developed questionnaire, related to various aspects of self-medication and prescribed medication. A pre-tested interviewed administered questionnaire, with 17 questions was distributed to participants.

Results: Among 100 respondents, 72 respondents practice self-medication, while only 28 people choose prescribed the medication by a registered medical practitioner. Adverse reactions, lack of knowledge about dose, the frequency of administration and chances of taking the wrong medicine were the major drawbacks of self-medication reported. The frequently reported illness that prompted self-medication included Headache, Cough, Fever, Diarrhoea, and Acidity.

Conclusion: Usage of OTC drugs was highest among common population. Time consumption for consultation, financial crisis, availability of OTC drugs, the predictability of doctor's general prescription were the commonly mentioned reasons for self-medication. Hence, proper Education and awareness must be created to help patients decide on proper medications.

Key Words: *Awareness, Anti-biotics,Over the counter(OTC), Prescribed Drugs, Self-medication.*

Introduction

Self-medication is the use of drugs with therapeutic intent but without any professional advice or prescription. It has also been defined as the use of non-prescription medicines on their own initiative[1]. While on the other hand prescribed medication is the use of drugs with the help of medical supervision and prescription. Drug retail shops frequently serve as the public's first point of contact with the healthcare system[2].In India, pharmacists and pharmacy attendants play an important role in fostering self-medication among the public[3]. Self-medication can cause resistance to bacteria and may lead to the emergence of multiple resistant organisms that would be difficult to treat[4-7]. Studies on self-medication patterns and the prevalence of doctor prescribing in India shows an increasing number of patients developing resistance to the commonly used antibiotics. The most commonly available over the counter (OTC) medicines in Chennai are analgesics, non-steroidal anti-inflammatory drugs (NSAIDs), antihistaminic, vitamin supplements, tonics and cough and cold remedies. Although these medications are considered safe, their excessive use can lead to serious adverse effects.

In India, the Drugs and Cosmetics Act, 1940 (DCA), the Drugs and Cosmetics Rules, 1945 (DCR) regulates the import, manufacture, distribution, and sale of drugs and cosmetics. The “OTC” (over the counter) has no legal implications in India[8]. Hence “OTC Drugs” means drugs legally allowed to be sold “Over the Counter” by pharmacists, i.e. without the prescription of a Registered Medical Practitioner. Prescription-only drugs are listed in Schedules H and X of the Drug and Cosmetics Rules. Few drugs which are scheduled under G do not need the prescription to purchase but require the following mandatory text on the label: Caution: It is dangerous to take this preparation except under medical supervision[9]. In several studies it has been found that inappropriate self-medication can generally cause serious health hazards such as adverse drug reactions, and drug dependence[10].

Self-medication is now increasing as a component of self-care[11]. Unlike other aspects of self-care, self-medication involves the use of drugs and these drugs have the potential to do good as well as harm. Self-medication can save the time, may be economical but due to its improper use of correct dose, side-effects and interactions can lead to serious implications[12]. Therefore, the study was taken up to analyze the population at risk, in extend to various aspects of self-medication and prescribed medication and create awareness of the adverse effects among the participants.

Materials and Methods

A questionnaire consisting of 15 questions were framed based on knowledge and usage of self-administrated drugs as well as prescribed drugs and they were given to 100 people. This was a cross-sectional study and has a self-developed questionnaire, consisting of both open and closed end questions related to various aspects of self-medication and prescribed medication which were most commonly employed. The study population consisted of the common population of Chennai, between age group 20-50 years. People were explained about the aim and purpose of the study and concept of self- medication and prescribed medication were clearly explained and discussed with the them prior to the survey. The respondents were interviewed with the questions and corresponding answers were marked.

The questionnaire was in two parts. The first part had questions on demographic information of the respondents such as Name, age, gender, etc. The second part contained questions on core issues which dealt on direct questions such as reasons for self-medication, most common drugs used for self-medication, type of ailment treated through self-medication, factors influencing discomforts of self-medication practices and also knowledge about drug resistance. The questionnaire was framed in such a way for easy understanding and filling by respondents. The questionnaire was also backed by a moral interview. Some of the questions asked during the interview section were: do you think self-medication is a good practice? Do you think education will help reduce the incidence of self-medication?

The aims of our study were to obtain baseline data on self-medication and doctor prescribing drugsin Chennai city, obtain information on the factors influencing self-medication and Doctor prescribing medication. And to create awareness about the drug interactions, that can lead to drugs in implications and adverse effects, due to self-medication.

Data obtained from the questionnaire were coded, given sequential numbers and entered into the Statistical Package for Social Sciences (SPSS), version 16, software. The analysis was carried out using descriptive statistics and Prevalence of self-medication along with prescribed medication, among population were measured in percentages. Descriptive analysis was employed to present bar charts for appropriate results.

Results

One hundred respondents were covered during this entire study. The age distributions of the responders were between the age of 20 to 50 years. Out of one hundred responders (59) were male and the rest (41) were females. These 100 responders were from various professions from Chennai district selected

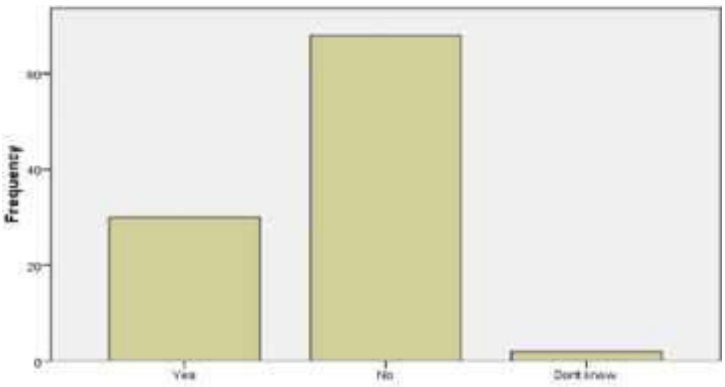
randomly for this survey. Coming to the results of administrating self-medication drugs in comparison to doctor's prescription drugs, when enquired about whether self-medication is good practice or not, (68%) responders accepted it's not good, while remaining (30%) responders said it is notbad, followed by 2% who doesn't know about it.[Graph 1]When we enquired them about Source of drug information, they responded that they gained knowledge from Doctors (48%), Internet (24%), friends (14%), Media (7%), Relatives (4%), while remaining (3%) learned from academic knowledge.[Graph 2]

According to the (28%) responders fever and cold are major reasons that demand them visiting doctors, followed by continuous headache (27%), and diarrhoea (24%). When they were enquired about completing the course of drugs that doctors prescribed (63%) responders said yes, while remaining (35%) does not finish the course of drugs which doctors prescribe is noted. (62%) responders said that they will visit the doctor for the second time, if the problem persists, while (38%) responded that, they will buy the same drugs over the counter, which was earlier prescribed by the doctors. From this survey we identified that confidence in self-medication (41%) was the main reason for administrating self-medication followed by, the financial crisis (35%), and time consumption (15%).

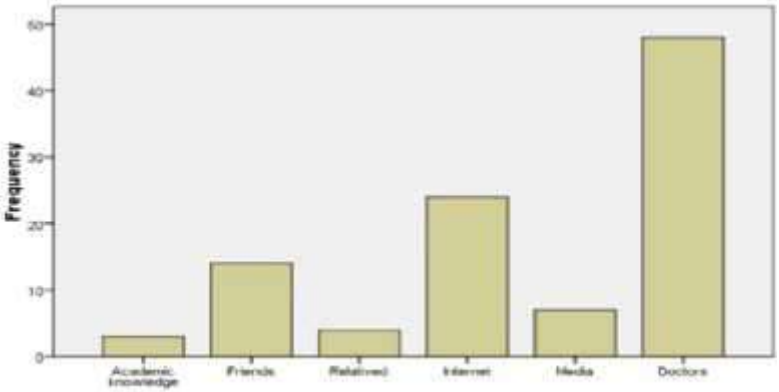
We also noticed (66%) responders were aware that drugs taken through self-medication have many disadvantages, while (33%) came up with a thought, it doesn't have any sort of adverse effects while remaining (1%) doesn't have any idea about it.[Graph 3] Even though self-medication has many side effects (54%) responders do not experience any side effects, while (31%) had severe side effects due to wrong self-medication, remaining (15%) were not sure about it. [Graph 4] When enquired about the safety of physician-prescribed medication in sequence to self-medicated ones, (93%) responders were absolutely sure that prescribed drugs are safe, while (7%) individuals said even prescribed drugs may not be safe always.

Interestingly (79%) responders said that they didn't experience any hospitalization due to adverse effects of self-medication, while (14%) went on hospitalization for further treatment due to its side effects, while (7%) individuals were not sure about it. According to the responders, the most commonly used over the counter drugs were Antibiotics (21%), Analgesics (21%), Anti-diarrheal (17%) followed by Anti-histamines (12%).[Graph 5] We also found that (63%) responders are not aware that Anti-biotic must be taken for complete dose due to its resistance, while just (32%) responders know about it, followed by (5%) individuals who don't even have any clue about it. We also identified the most common discomforts that people experience while administrating self-medication, they were Diarrhoea (32%), Allergies and rashes

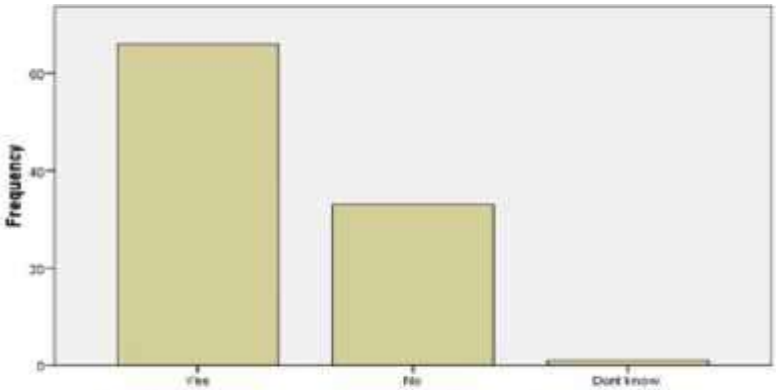
(21%), Constipation (18%), Nausea /Vomiting (14%), Headache (12%), and remaining (3%) doesn't have any discomforts.[Graph 6] when we enquired about their knowledge about drugs used for common problem, (47%) people responded that they have good knowledge, (36%) responded they have the average knowledge, while remaining (17%) have no idea about any drugs or drug names. Even after (47%) individuals said, they have good knowledge about drugs still (89%) individuals have no clue about combination drugs like H2 Blockers for NSAIDs, while only (11%) people knew about it and responded it's to counteract gastric irritation.



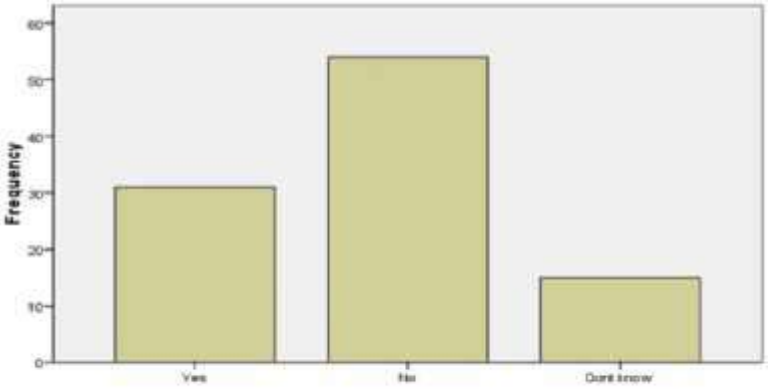
Graph 1 : Is self-medication a good practice?



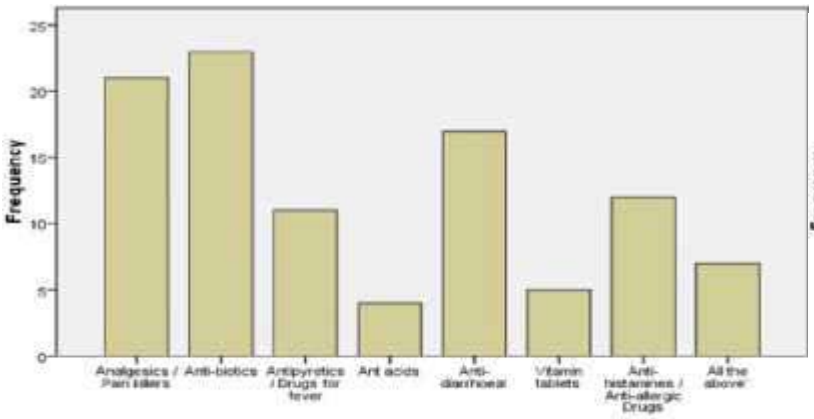
Graph 2 : Source of drug information?



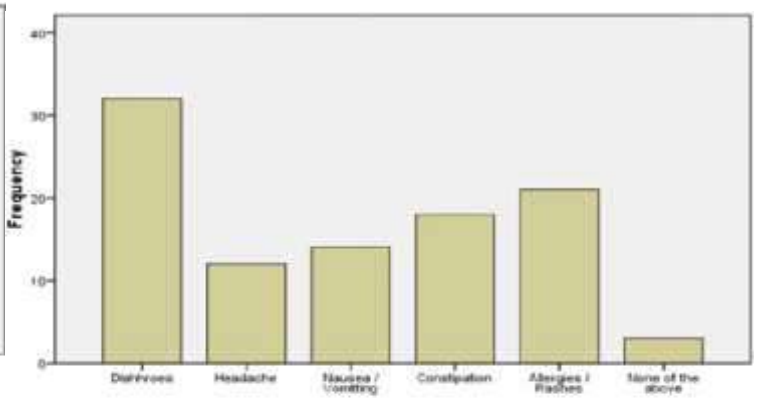
Graph 3 : Are you aware that drugs taken through self-medication have many disadvantages?



Graph 4 : Have you experienced adverse effects due to over the counter drugs?



Graph 5 : Most commonly used over the counter drug?



Graph 6 : Most common discomforts administrating self-medicated drug?

Discussion

Our study shows that majority of the responders were aware of OTC drugs and self-medication is widely practiced among them when compared to prescribed medication. Confidence in self-medication, a financial crisis etc, were some of the reasons for using OTC drugs. Anti-Biotics and Analgesics were the drugs most commonly used for self-medication. Male sex and age between 25- 40 years were associated with increased self-medication. Time consumption for consultation, the Financial crisis, availability of OTC drugs, predictability of doctor's general prescription were the commonly mentioned reasons for Self-medication. Diarrhoea and headache were the most common reasons for self-medication. We found Drugs especially anti-biotics were not taken for the proper course, which can lead to serious resistance in future. Education to help patients decide on the appropriateness of self-medication is required.

Self-medication is defined as the use of medication, whether modern or traditional, for self- treatment without the advice of physician (expert in the medical profession) either for diagnosis, and prescription. Of treatment. Experience from prior illness; advice from pharmacist, relatives, and friends play a major role in the self-medication. Out of total 100 responders, 59 were male and 41 were female. There was no statistically significant difference between males and females in regard to self-medication. A similar result was noted by Shahbaz Baig[13].According to one study, the most common reason of self-medication was not being in need, for a doctor in diseases widely seen and the most common form of obtaining medication was directly from a pharmacist without the prescription[14].

Prevalence of self-medication has been found among 81.5% individuals in a rural area in Maharashtra. More male patients used self-medication compared to females, contrary to data from Western reports[15]. A questionnaire-based cross-sectional study showed that prevalence of self-medication was about 88% among study participants. The frequency of self - medication is highly variable in different parts of the world; as

low as 45% in Turkey to as high as 94% in Hong Kong[16].

Some authors reported that previous experience was one of the major reason for self-medication besides unavailability of doctors and transport, ability to self-manage, an urgency to treat, assumption of better knowledge, lack of time and cost of treatment were the other contributing factors. But our study proves that they are not only the reason, but also, the confidence in self-medication drugs (41%), the financial crisis (35%), and time consumption (15%) were also the main reason for administrating self-medication.

In our study we enquired them about Source of drug information; they responded that they gained knowledge from Doctors prescription (48%), Internet (24%), friends (14%), Media (7%), Relatives (4%), while remaining (3%) learned from academic knowledge. Similarly, Advertisement in the newspaper and media as main sources followed by chemist shops. Therefore, it is necessary that pharmacist should take responsibility to avoid selling drugs without prescriptions.

Pharmacists play a valuable role in identifying, solving and preventing drug-related problems for achieving optimal patient outcomes and quality of life. Ambulatory based pharmacists have the responsibility to appropriate, effective and economical use of all medications, especially those therapies patients are self-selecting. Pharmacists should guide their customers to consult the physician before taking any medication by self[17-19].

Whenever a drug is prescribed to a person, he/she should be given instructions to the person's comprehensive levels so that it will be helpful for them to understand the impacts of it and also encourage them to adopt alternative methods to relieve their symptoms instead of using self-mediation there by preventing the ill effects of self-medication such as drug dependence, drug abuse, etc[20].

The economic barriers and familial competition has the potential to prompt an individual to indulge in self-medication[21].

The prevalence of medicine storage in room leads to practice of self medication. Analgesics and antibiotics are being the most common types of medicine present in residence[22].

self-medication was practiced with a range of drugs from the conventional anti-pains to antibiotics. Although the practice of self-medication is inevitable; drug authorities and health professionals need to educate students about the pros and cons of self-medication[23-24].

By taking inappropriate drugs the results can be two-fold. These drugs does not cure the actual pathology. It produces unwanted side - effects which affects the patient[25].

An antimicrobial is an agent that kills microorganisms or inhibits their growth. Antimicrobials can be grouped according to the microorganisms they act primarily against. They are ones which help in building up resistance[26].

Doctors and pharmacists play a very important role in creating awareness about self-medication by

educating the patients. Hence it is suggested that the public education is mandatory on the type of illness for self-diagnosis and its medication, along with an implementation of stringent rules and regulations on their use. It is also essential to highlight the dangers of OTCs on their misuse.

Conclusion

Our study shows that majority of the responders were aware of OTC drugs and self-medication is widely practiced among them when compared to prescribed medication. Confidence in self-medication, a financial crisis where the some of the reasons for using OTC drugs. Anti-Biotics and Analgesics were the drugs most commonly used for self-medication. Male sex and age between 25- 40 years were associated with increased self-medication. Time consumption for consultation, the Financial crisis, availability of OTC drugs, predictability of doctor's general prescription were the commonly mentioned reasons for Self-medication. Diarrhoea and headache were the most common reasons for self-medication. We found Drugs especially anti-biotics were not taken for the proper course, which can lead to serious resistance in future. Education to help patients decide on the appropriateness of self-medication is required. Self-medication is an alarming sign for society. It was analyzed that most of them were not aware of the drug, dosage, the frequency of administration and adverse reactions. While very little people were aware of these medications. Drugs, especially antimicrobials, which were not taken for the proper duration, can lead to serious resistance. Moreover, there are many drugs which are new to the market and many are banned due to side effects. Hence, proper education and awareness must be created to help patients decide on proper medications, which can save the lives from unnecessary complications.

Acknowledgement

We would like to express our gratitude to all the respondents for spending their valuable time to answer our questionnaire that helped us to successfully complete our study.

Corresponding Author

Thummala Bhagya Lakshmi

Senior Lecturer,Department of Pedodontics and Preventive Dentistry

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

162,Poonamallee High Road,Chennai – 600077,Tamilnadu, India.

Email ID: bhagya.thummala@yahoo.co.in

Telephone Number:+91 9642432345

References

1. Jamison AJ, Kielgast PJ, Hoek AJ, Reinstein JA. Responsible self-medication. Joint Statement by the International Pharmaceutical Federation and World Self-Medication Industry 1999;16.
2. Kafle KK, Madden JM, Shrestha AD, Karkee SB, Das PL, Pradhan YM, Quick JD. Can licensed drug sellers contribute to safe motherhood? A survey of the treatment of pregnancy-related anaemia in Nepal. Social science & medicine 1996;42(11):1577-88.
3. Kamat VR, Nichter M. Pharmacies, self-medication and pharmaceutical marketing in Bombay, India. Social science and medicine 1998;47(6):779-94.
4. World Health Organization.WHO global strategy for containment of antimicrobial resistanceWHO/CDS/CRS/DRS/2001.2.2001.
5. Fadare JO, Tamuno I. Antibiotic self-medication among university medical undergraduates in Northern Nigeria. Journal of public health and Epidemiology 2011;3(5):217-20.
6. Aswapokee N, Vaithayapichet S, Heller RF. Pattern of antibiotic use in medical wards of university hospital, Bangkok, Thailand. Reinject Dis 1990;(1):136-141.
7. Okeke NI, Laminar A, Edelman R. Socioeconomic and Behavioral Factors Leading to Acquired Bacterial Resistanceto Antibiotics in Developing Countries. Emerge Infect Dis 1999;5(1):18-27.
8. KP Osemene, Lamikanra A. Faculty of Pharmacy, Obafemi Awolowo University, Ile-Ife, Osun State, Nigeria, Tropical Journal of Pharmaceutical Research 2012;11(4):683-689.
9. Sean M, Christian J, Carol J. Illicit use of prescription pain medication among college students. Drug and Alcohol Dependence 2005;77(1):37-47.
10. Kiyingi KS, Lauwo JAK. Drugs in the home: danger and waste. World Health Forum 1993;14:381-84.
11. Kamat VR, Nichter M. Pharmacies, self-medication and pharmaceutical marketing in Bombay, India. Soc Sci Med1998;47(6):779-94.
12. Hughes CM. Benefits and risks of self-medication. Drug Saf2001;24:1027-37.
13. Shahbaz B. Self Medication Practices. Professional Med2012;19(4):513-521.
14. Anuradha D, Hanmant S, Shilpa K. Department. of Pharmacology, People's College of Medical Sciences and Research Centre Bhopal, MP, Int J Pham Bio Sci2014;5(4):289-293.
15. Shyam K, Priyanka K, Mili M. Prevalence and Pattern of Self-medication Practices in Rural Area of Barabanki. Indian Journal of Clinical Practice 2014;25(7).
16. Aljinović-Vucić V, Trkulja V, Lacković Z. The content of home pharmacies and self-medication practices in households of pharmacy and medical students in Zagreb. Croat Med J2015;46(1):74-80.

17. Wilbur K, Salam SE, Mohammadi E. Patient perceptions of pharmacist roles in guiding self-medication of over-the-counter therapy in Qatar. Patient Prefer Adherence 2010;4:87–93.
18. Montastruc JL, Bagheri H, Geraud T, Lapeyre-Mestre M. Pharmacovigilance of self-medication. Therapie1997;52:105–10.
19. Habeeb GE, Gearhart JG. Common patient symptoms: Patterns of self-treatment and prevention. J Miss State Med Assoc 1993;34:179–81.
20. Sigloria I, Mangalagowri P. Asses the prevalance of self-medication among people in selected area. Int J Pharm Bio Sci 2017;8(1):276-280.
21. Binti F. Roy A. A questionnaire study on usage of self medication of paracetamol among dental undergraduate students of private hospital. Int J Pharm 2017;9(1):14-15.
22. Yuvaraj S, Priya AJ, Sriram N. Effect of Non-SteroidalAnti-Inflammatory Drugs in Pregnancy: A Systematic Review. Research Journal of Pharmacy and Technology 2015;8(6):787-89.
23. Auta A, Banwat S, Sariem C, Shalkur D, Nasara B, Atuluku M. Medicines in pharmacy students' residence and self-medication practices. J Young Pharm2012;4:119-23.
24. Roy A, Shivaani S. Knowledge and awareness of antibiotic resistance among dental students. Int j Sci 2017;6(4):1343-45.
25. Svitlana AS. Study on Medicines Delivered By Pharmacists Relating to Dental Pain, Without Prescriptions. Journal of Pharmaceutical Sciences and Research 2016;8(9):1077-79.
26. Mariona P. Lakshmi T. Survey on Knowledge, Attitude and Practice of Antimicrobial Resistance among Dental Students. Int J Pharm Sci 2017;43(2):5-7.

A Survey on Evaluation of Preparedness for Medical Emergencies at Dental Office

D. Shruthi[1], Pradeep Kumar[2], Antonette Rhea[3]

Graduate Student[1], Reader[2], Department of Public health Dentistry[2], Senior Lecturer[3,] Department of Prosthodontics[3]

Saveetha Dental College, Saveetha institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Medical emergencies can occur at any time in the dental office.Every health care providers should be aware of recognizing and handling medical emergencies in their respective workplace. All should be trained and frequently updated regarding aiding patient in emergency situations. The aim of this study is to determine the knowledge and preparedness of a dental practitioner to manage medical emergencies at their dental office.

Material and Methods: A self-administered questionnaire comprising of 10 questions was designed relating to medical emergencies that may occur in dental office, its prevention and management and was distributed to the practicing dentists in and around Chennai.

Result: A total of 150 dentists participated in this study. Of these 48 were BDS graduates and 102 were MDS graduates. Almost 134 have their working place in urban areas, 12 of them have working place in rural areas and 4 of them have their working place in both urban and rural areas. This study shows almost 97% of surveyed dentists enquire medical history including medication and allergy. Only 60% of dentists record vital signs of their patients. The study shows that only 72.6% were trained for management of medical emergencies in dental office and 94.6% of them responded that training to dentist for management of medical emergencies is important. It is found that only 68% of participants have been reported to be confident in handling medical emergency situation and about 27% have been faced with medical emergency for past 12 months

Conclusion: Dentists being members of the healthcare profession, should be prepared to deal with medical emergencies which may arise at their workplace. A better knowledge of medical emergencies is essential for further development and professional care for people with various medical complications. This will ensure the provision of better and safer dental healthcare services for the population.

Key Words: *dental office, adverse effects, drugs, vital signs, knowledge*

Introduction

A medical emergency is generally termed as an acute injury or illness that poses an immediate risk to a person's life or long-term health. Medical emergencies can occur at any time in the dental office.[1] Medical emergencies can be alarming to any clinician but these situations and can be prevented if proper preparations are made.[2] Effective management of medical emergencies in the dental office are ultimately the dentist's responsibility. Lack of training and inability to cope with medical emergencies can lead to tragic consequences and sometimes legal action.[3] Fear and anxiety in patient seeking dental treatment may cause measurable metabolic changes, which make these patients prone to such emergencies. The chance of medical emergency increases with administration of anesthesia and medication, virtually all of which are known to have adverse reactions [4]. Most dental procedures are performed under local anesthesia.[12] The primary prevention by giving prescription may influence their patient's health.[13] Anaphylaxis is defined as a serious allergic reaction that is rapid in onset and may cause death.[14] The worst manifestations are cardiovascular collapse, bronchospasm and laryngeal edema.[15]. General Adverse effects include localized reactions like minimal to moderate redness was reported and central nervous system manifestations.[16] A good step in making ready for clinical emergencies is the acquisition of emergency supplies. The circle of neighboring physicians should choose emergency drugs and device that reflect the spectrum of their office's expected emergencies.[17] The most crucial element of nearly all medical emergencies in the dental workplaces is to save patient, or accurate, provide adequate oxygenation to the brain and heart.[18] Therefore, the control of all clinical emergencies ought to ensure that oxygenated blood is being introduced to those important organs. That

is constant with basic CPR, with which the dentist must be in a position.[19] A successful prevention relies upon at the assessment of a patient's history and physical situation as well as suitable treatment adjustments.[20] The aim of this study is to determine knowledge and preparedness of dentists to manage medical emergencies at their dental offices and assess the availability of medical emergency drugs and equipments.

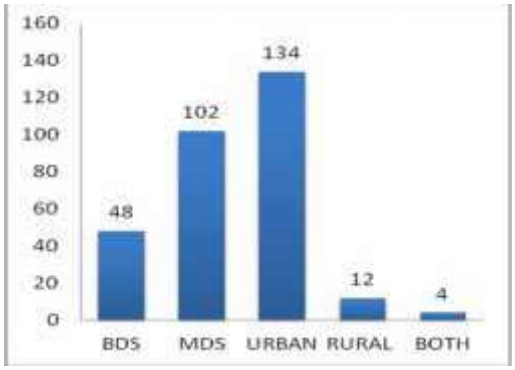
Material and Methods

This study was conducted to determine the awareness on medical emergency in dental office among practicing dentists. This study was conducted among dentists practicing in and around Chennai. A valid questionnaire of 10 was designed which was inclusive of both open and close ended questions relating to medical emergencies that may occur in dental office, its prevention and management. All questions were given two options (Yes / No**)**. The questionnaire was distributed among dentists in and around Chennai, who were willing to participate in the study. This is self reported questionnaire which takes 10 minutes for completion. The participant of the study includes dentists who were undergraduates, postgraduate with clinics in urban or rural areas or clinics in both urban and rural areas. Questionnaires were manually checked for completion of data. All data were entered in data entry form. The analysis of data was done and percentage calculation of the collected data was done with simple mathematical calculations.

Result

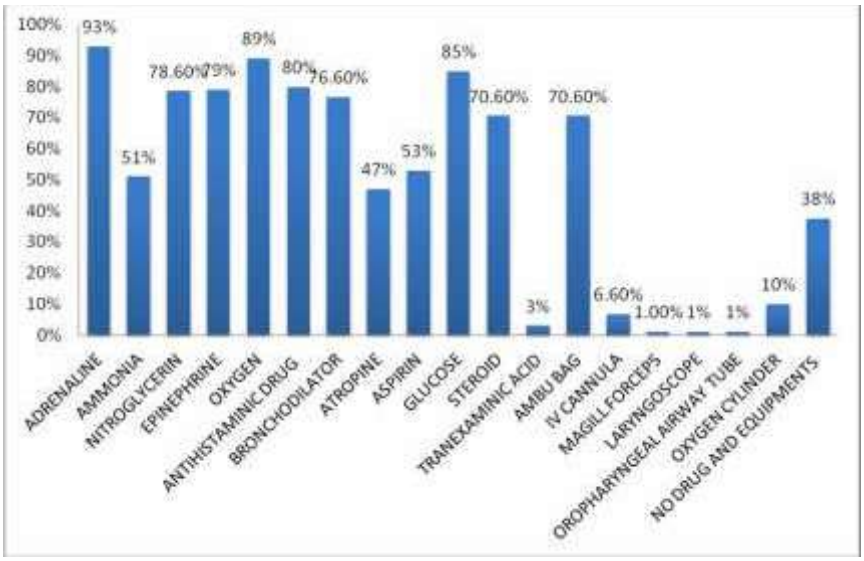
A total of 150 dentists participated in this study. Of these 48 were BDS graduates and 102 were MDS graduates. Almost 134 have their working place in urban areas, 12 of them have working place in rural areas and 4 of them have their working place in both urban and rural areas (graph 1). This study shows almost 97% of surveyed dentists enquire medical history including medication and allergy. Only 60% of dentists record vital signs of their patients. The study shows that only 72.6% were trained for management of medical emergencies in dental office and 94.6% of them responded that training to dentist for management of medical emergencies is important. It is found that only 68% of participants were reported to be confident in handling medical emergency situation and about 27% have faced with medical emergency in their dental office for past 12 months (graph 2).

Graph 3 shows that among surveyed dentists 41 of them have no emergency drugs and equipments and only 109 have been reported to have emergency drugs and equipments. Adrenalin, glucose are the most commonly stocked drugs and 70% of them have AMBU bag and 38% of them have oxygen cylinders. Other drugs and equipments reported are ammonia (51%), nitroglycerin (78.6%), epinephrine (79%), oxygen (89%), antihistaminic drugs such as phenramine maleate, diphenhydramine, chlorpheniramine (80%), bronchodilator such as salbutamol, albuterol (76.6%), atropine (47%), aspirin (53%), steroids such as hydrocortisone, dexamethasone (70.6%), tranhexaminic acid (3%), magill

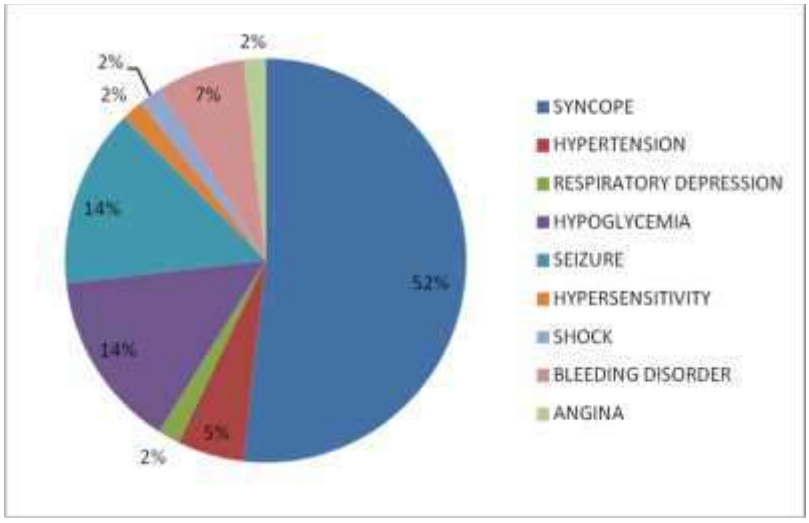


Graph 1: Distribution of participants based on graduation and working place

forceps, laryngoscope, oropharyngeal tube (1%), I.V cannula (6.6%). Chart 1 shows that the most common medical emergency reported in dental office faced by participants is syncope (52%). Other medical emergencies faced in dental office reported are hypertension (5%), respiratory depression (2%), hypoglycemia (14%), seizure (14%), hypersensitivity (2%), shock (2%), bleeding disorder (7%), and angina (2%).



Graph 2: Knowledge of management of medical emergencies among dentist



Graph 3: Availability of medical emergency drugs and equipments

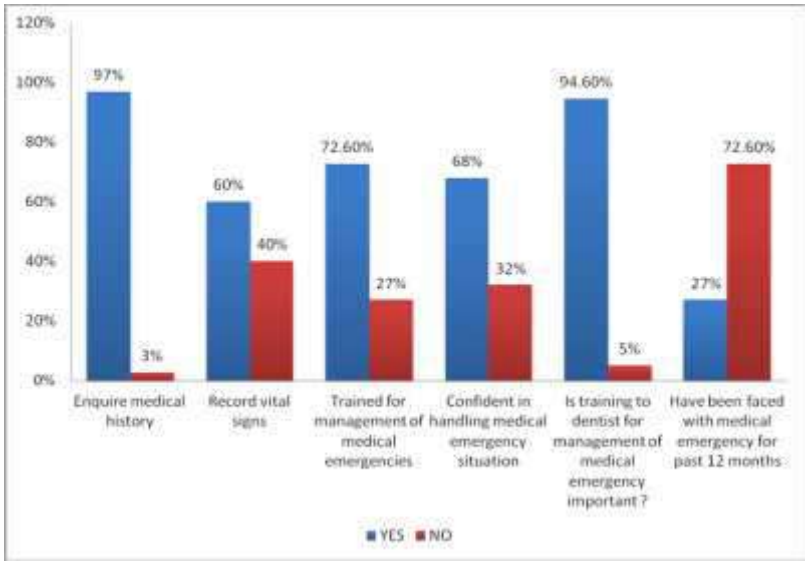


Chart 1: Medical emergencies reported by dentists in their dental office

Discussion

This study was conducted to assess the knowledge and preparedness of dentists to manage medical emergencies at their dental office. In this study, it is determined that only 72.6% of surveyed dentists are trained for management of medical emergencies, which is comparatively higher than the one reported by Gupta et al in which only 42.1% dentists were reported to be trained.[4] It is found that 94.6% of dentists responded that training to dentists for management of medical emergency is important, this is comparatively less than Praveen et al who reported 97.1% felt training is important.[2] In this study it is found that 97% of surveyed dentists enquire about medical history including medication and allergy which is similar to that of Shwetha et al.[5] About 60% of dentists record vital signs but most of them record only blood pressure and pulse in needed situation. It is important to enquire about medical history, record vital signs because it may give a clue for possibility of occurrence of medical emergency at dental office. It is found that only 27% of them have experienced medical emergency during their practice. The experience reported only within 12 months duration was enquired as it may be easy for the dentists to recall the situation. Among the surveyed dentists only 68% of them are confident in managing medical emergency situations in dental office which is higher than compared to the survey done by Maisa et al. According to their survey only 41% of the surveyed dentists were confident in handling medical emergency situation.[6] Even though 72.6% of the dentists were reported to be trained only 68% of them are confident in handling medical emergency situation. This may be due to improper training to the dentists, short duration programs, etc. The 27% of dentists who have experienced medical emergency situation at their dental office were enquired about the common medical emergency situation they experienced.

The most commonly reported medical emergency was syncope of 52% , seizure disorder and hypoglycemia 14%, and other emergency situations such as hypersensitivity, shock, angina, respiratory distress, hypertension, bleeding disorders were reported to be occurring less than 10%. Similar results were obtained in study done by Praveen et al [2]. In study

done by Laurent et al[7] and Gbotolorun OM et al,[8] syncope was reported to be the common medical emergency situation encountered by dentists.

On enquiring about availability of medical emergency drugs, among the surveyed dentists, about 38% of them reported to have no drugs and equipments available in their dental office. This is higher when compared to Chapman's study in which 14% of dentists reported of not having any emergency drugs and equipments.[9] Adrenalin and glucose are the most commonly stocked drug and 70% of them have AMBU bag, 38% of them have oxygen cylinders. Other drugs and equipments reported are ammonia (51%), nitroglycerin (78.6%), epinephrine (79%), oxygen (89%), antihistaminic drugs such as pheniramine maleate, diphenhydramine, chlorpheniramine (80%), bronchodilator such as salbutamol, albuterol (76.6%), atropine (47%), aspirin (53%), steroids such as hydrocortisone, dexamethasone (70.6%), tranexaminic acid (3%), magill forceps, laryngoscope, oropharyngeal tube (1%), I.V cannula (6.6%). According to Amirchaghmaghi et al, nitroglycerin was reported to be commonest drug and syringe and I.V cannula were reported to be commonest equipment available in dental office.[10]

Even though the incidence level is reported to be increased when compared to studies done previously, the reported levels are still low and should be improved. It is important for all dentists to attain knowledge about management of medical emergency. It is suggested that training to dentists about management of medical emergency should be considered seriously. Many awareness programs, training sessions, long duration training programs should be done. The training must be provided in such a way that the dentists are confident in handling any medical emergency situation during their practice. Proper training, better knowledge of management of medical emergencies will provide confidence for a dentist to handle it. By properly equipping the office, educating their assistants and staffs, the dentist can ensure that their patients receive essential care in office emergencies.[11] This will provide a fortified dental care for the population.

Conclusion

To conclude, Dentists being members of the healthcare profession, should be prepared to deal with medical emergencies which may arise at their workplace. Dentistry in India has made tremendous progress in different subspecialties of dentistry. A better knowledge of

medical emergencies is essential for further development and professional care for people with various medical complications. This will ensure the provision of better and safer dental healthcare services for the population.

Corresponding Author

Antonette Rhea,

Senior Lecturer, Department of Prosthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email Id: arhea.a@gmail.com

Phone Number: +91 91766 45343

Reference

1. Sue Protzman; Jeff Clark, MS, REMT-P; Wilhemina Leeuw, MS, CDA. Management of Medical Emergencies in the Dental Office. at dentalcare.com Continuing Education Course, Revised January 24, 2012:47-54.

2. Praveen S. Jodalli, Anil V.Ankola. Evaluation of knowledge, experience and perceptions about medical emergencies amongst dental graduates (Interns) of Belgaum City, India. J Clin Exp Dent. 2012;4(1):14-8.

3. Mandakini Mohan, Himani Sharma, Abhishek Parolia, Ankur Barua. Knowledge, Attitude and Perceived Confidence in Handling Medical Emergencies among Dental Practitioners in Dakshina Kannada, India. OHDM 2015; 14(1):27-31.

4. Tanupriya Gupta, M.R. Shankar Aradhya, Anup N. Preparedness for medical emergencies among dentists in Udupi and Manglore, India. The journal of contemporary dental practice 2008; 9(5)3-10.

5. Shweta Kumarswami, Anish Tiwari,Mayank Parmar, Manisha Shukla, Amit Bhatt and Mittal Patel. Evaluation of preparedness for medical emergencies at dental offices: A survey. J Int Soc Prev Community Dent. 2015; 5(1): 47–51.

6. Maisa O. Al-Sebaei, Moayyad A. Alkayyal, Abdulelah H. Alsulimani, Othman S. Alsulaimani, Weam T. Habib. The preparedness of private dental offices and polyclinics for medical emergencies A survey in Western Saudi Arabia. Saudi Med J 2015;36(3):335-340.

7. Florian Laurent, Pascal Augustin, Scott T Youngquist, Nicolas Sega. Medical emergencies in dental practice. Med Buccale Chir Buccale 2014; 20:3-12.

8. Gbotolorun OM, Babatunde LB, Osisanya O, Omokhuale E. Preparedness of government owned dental clinics for the management of medical emergencies: a survey of government dental clinics in Lagos. Nig Q J Hosp Med. 2012; 22(4):263-7.

9. P. J. Chapman. Medical emergencies in dental practice and choice of emergency drugs and equipment: A survey of Australian dentists. Australian Dental Journal 1997; 42(2):103-8.

10. Amirchaghmaghi M, Sarabadani J and Delavarian Z. Preparedness of Specialist Dentists about Medical Emergencies in Dental Office-Iran. Aust. J. Basic & Appl. Sci.2010; 4(11): 5483-5486.

11. Seth L. Toback, MD, Washington, Pennsylvania. Medical Emergency Preparedness in Office Practice, American Family Physician 2007; 75(11):1679-84.

12. Sharmraaj Subramaniam, Prasanna Neelakantan. Local anesthesia in dentistry-Clinical Considerations. Int. J. Drug Dev. & Res., October -December 2013; 5 (4): 30-36.

13. Nor Syakirah Binti Shahroom, T. Lakshmi, Anitha Roy. Knowledge of drug prescription among dental and medical student in India – an online survey. Journal of Advanced Pharmacy Education & Research , Apr-Jun 2017; 7(2): 2249-3379

14. Keerthana Balaji, Ashish.R.Jain, MD.ACU.Varma. Knowledge and practice regarding anaphylaxis among dental students. IJSDR, April 2017 ; 2 (4): 30-36

15. S. Vaishali, Anitha Roy. Awareness of Anaphylaxis reaction to local anesthetic drugs among dental students - A cross sectional study. IJPT, April-2017; 9 (1):29338-29344.

16. Yen Lai Kee, Prasanna Neelakantan. Local Anesthetics in Dentistry – Newer Methods of Delivery. IJPCR, January-March, 2014; 6 (1):4-6.

17. Johnston CL, Coulthard MG, Schluter PJ, Dick ML. Medical emergencies in general practice in south-east Queensland: prevalence and practice preparedness. Med J Aust. 2001;175:99–103.

18. Varma L S, Pratap K, Padma T M, Kalyan V S, Vineela P. Evaluation of preparedness for medical emergencies among dental practitioners in Khammam town: A cross-sectional study. J Indian Assoc Public Health Dent 2015;13:422-8.

19. Gupta T, Aradhya MR, Nagaraj A. Preparedness for management of medical emergencies among dentists in Udupi and Mangalore, India. J Contemp Dent Pract 2008;9:92-9.

20. Shampaine GS. Patient assessment and preventive measures for medical emergencies in the dental office. Dent Clin North Am 1999; 43(3):383-400.

A Survey on Knowledge of Clinical Criteria for evaluation of Dental Restorations among Dentists

Yendodu Varshitha[1], Sandhya Raghu[2], Arvind. M[3]
Graduate Student[1],Reader[2],Department of Conservative of Endodontics,Professor[3],
Department of Oral Medicine and Radiology
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: Federation DentaireInternationale have formulated criteria for quality assessment of restorations and serve as a guidelines to determine if a restoration needs refurbishment, repair or replacement. Refurbishment is referred as a minimal intervention such as polishing or contouring when no additional material is placed. Repair is a minimal intervention that requires additional material to be placed with or without a minimal preparation in the restoration or dental tissues. Clinical criteria will group the aesthetic, biological and functional categories of restoration for clinical evaluation procedure.

Materials and Methods: A pre-piloted questionnaire was distributed amongst a group of 100 dentists randomly selected from all the departments of the dental college. Dentists were surveyed in relation to their knowledge about FDI criteria and advantages in assessing a restoration using FDI criteria.

Result: 96% of participants were aware of FDI criteria for evaluating the restorations and 4% knew both Ryge and FDI criteria. We found that more that half population are not trained for assessing the restorations. 80% population believe that criteria should be followed for evaluation of restoration which gives a positive scope for this system.

Conclusion: The FDI clinical criteria and scoring system is very efficient system for evaluation of direct and indirect restorations. With the limitations of the study, we conclude that by proper training and calibration this criteria can be applied not only by the researchers but also by dental students and general practitioners for quality assurance purpose of replacement andrepair of dental restorations.

Key Word: *FDI criteria, Dental restorations, Replacement, Quality, Training*

Introduction

Dental restorations are the one most commonly done treatment in dentistry. Assessment of the dental restoration is important for quality assurance in dental office[1]. Significant step has to be taken to improvise the knowledge about the dental restoration. In 2007, a new clinical criteria were approved by theFDI to assess restoration quality. This article on restoration evaluation was published in the journal of adhesive dentistry[2] and also in the journal of clinical oral investigation[3] for the evaluation of dental restoration. This standard criteria should be utilised when the restorative materials and operative techniques are to be investigated clinically. Restored material should be assessed whether it can be maintained or undergo refurbishment, repair or any replacement. One study has compared the FDI criteria and the traditional United States Public Health Service (USPHS) which is also called as Ryge criteria for the evaluation of restorations in deciduous teeth[4]. In that study it was concluded that FDI is more sensitive for identifying difference in deciduous composite resin restorations[5]. In 2008 a tool called e-calib was introduced to facilitate both training and calibration of new FDI criteria. The cases will be selected randomly with high quality photographs. Scoring in percentage is done to evaluate correct response. This helps a practitioner to improvise their knowledge of assessing the restorations. A good quality restoration requires many clinical considerations. FDI criteria gives many strategies to identify the cause of failure[6,7]. Applicationof FDI criteria improves the standards of clinical practice and dental care[8-11].

FDI Criteria

Aesthetic Criteria

1. Surface luster
2. Staining: (a) surface and (b) margin
3. Colour match and translucency
4. Aesthetic anatomical form

Functional Criteria

1. Fracture of material and retention
2. Marginal adaptation
3. Occlusal contour and wear

4. Approximate anatomical form: (a) contact point and (b) contour
5. Radiographic examination, where applicable
6. Patient's view

Biological Criteria

1. Postoperative sensitivity and tooth vitality
2. Recurrence of caries, erosion, abfraction
3. Tooth integrity
4. Periodontal response
5. Adjacent mucosa
6. Oral and general health

For all three groups, the following gradings are used for evaluation:

1. Clinically excellent/very good
2. Clinically good
3. Clinically sufficient/satisfactory
4. Clinically unsatisfactory
5. Clinically poor

Table 1: Questionnaire

Age:
Sex:
Speciality:
Years of experience:
1.Are you aware of clinical criteria for dental restorations
a)Yes b) No
2. Among the following which clinical criteria have you read
a)FDI Clinical criteria
b) Ryge criteria
c) none of the above
3. Do we need strict/universally accepted clinical criteria for evaluation of
dental restorations.
a)Yes b)No

If yes why?
a)Easy communication
b)Standardisation
c)Easy follow up
d)Treatment plan
4. Have you undergone training for evaluation of dental restoration
a) yes
b) no
5.Clinical evaluation of restored material should be done
a) on the day of restoration
b) after one week
c) after 6,12 and 18 months
6. Assessment of the restoration is done through
a) black and white photographs
b)Colour photographs
c)Radiographs
7. The clinical assessment influence the treatment plan
a)strongly agree
b)Agree
c) disagree
d) strongly disagree
8. Is it possible to train the undergraduate students to assess restoration based on the FDI criteria.
a) yes
b) no
9.Are you practising FDI criteria

If yes, reasons for practising the FDI criteria.
a) reliable
b) quality
c) easy
d) increase restoration longevity
e)treatment plan
f) faster than other criteria
10. Are you practising FDI criteria
If no, reasons for not practicing the FDI criteria
a)Time consuming
b)patient cooperation
c)lack of knowledge and training
d)not interested

Scoring the Dental Restoration

Restoration is judged as clinically as unacceptable, then the reason has to be recorded for the failure and it must be decided whether the restoration goes for repair or replacement. Not all restorations with score 4 be replaced. Localised defects with sufficient clinical access can be repaired like gaps which can be sealed, adding new material to chipped fractures, and veneering of stained areas of the restorations, etc. Repaired restorations are scored as relative failure and replaced restorations are scored as absolute failure. Most commonly score 5 clinical results gives bad results than score 4, but that is not inevitable. Score 4, and consequently the possibility for repair, depends more on whether it is accessible for repair or not. A repair is considered to be minimally invasive approach that implies the addition of restorative material after the defect is explored with or without involvement of material placement. Refurbishment is referred as a minimal intervention which leads to contouring , polishing or the application of glaze or adhesives. To take into consideration the extent of a clinical defect or observation in relation to the entire restoration or to record the exact location of the defect, there is a semi quantitative Clinical Evaluation which is recommended. Final rating of a particular restoration is only evaluated after completion of the assessments of the final scores for aesthetic, biological, functional aspects of restoration. More severe the score it will prevail. Whenever a restoration receives a score of 4 or 5 independent of the

specific criteria below, it should be recorded as a failure, but it is not necessary that all failures call for replacement of the entire restoration.

Results

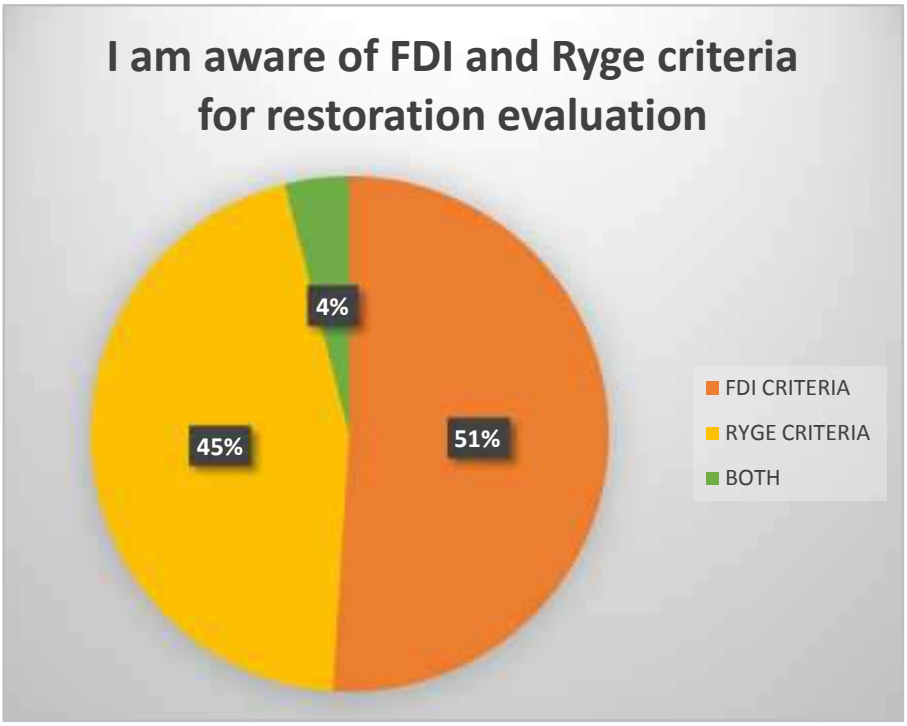


Figure 1: Awareness of restoration evaluation

Figure 2: Training for evaluation of dental restoration

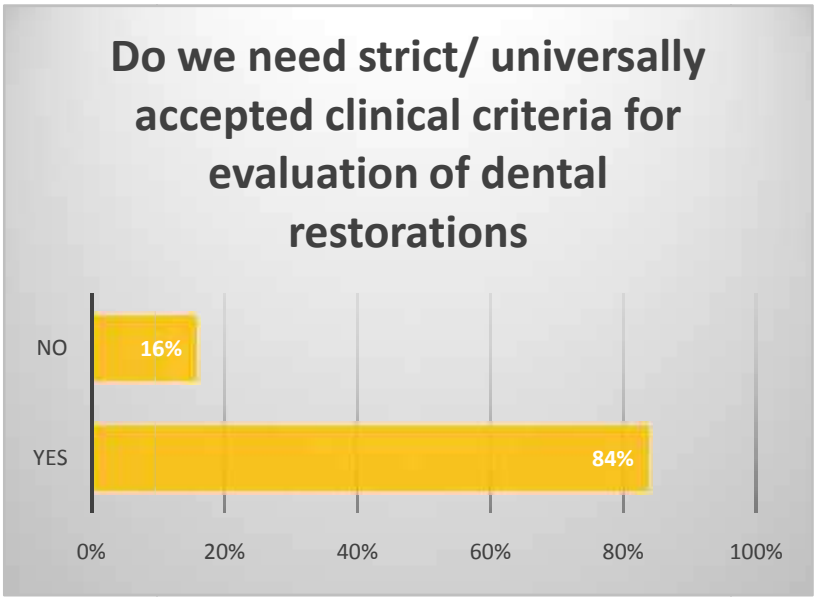


Figure 3: Requirement of criterias for evaluating a restoratio

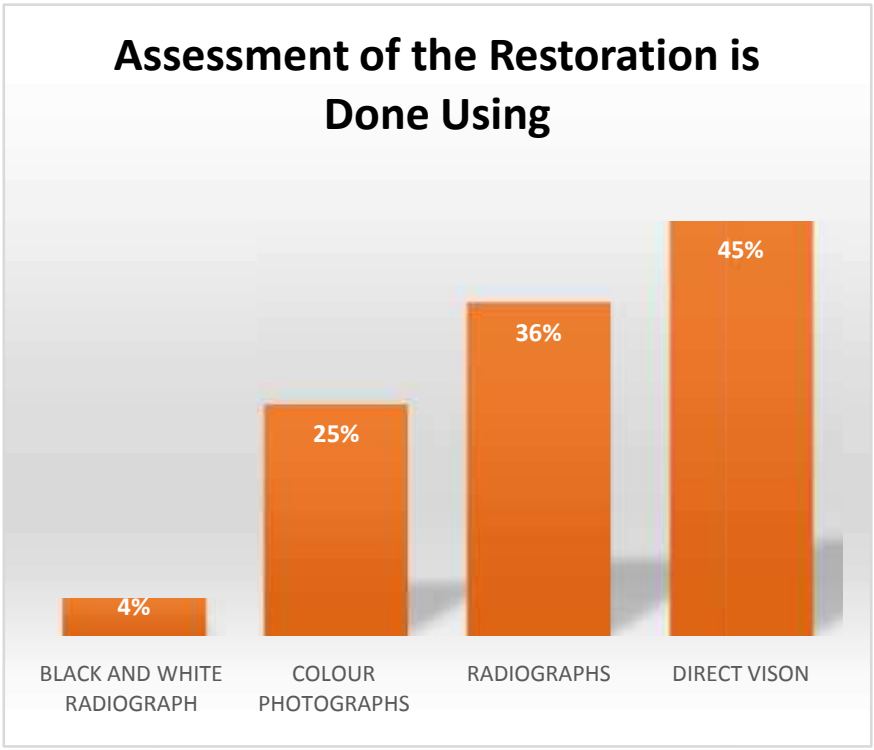


Figure 4: Restoration Assessment

Figure 5: Duration left to evaluate a restoration

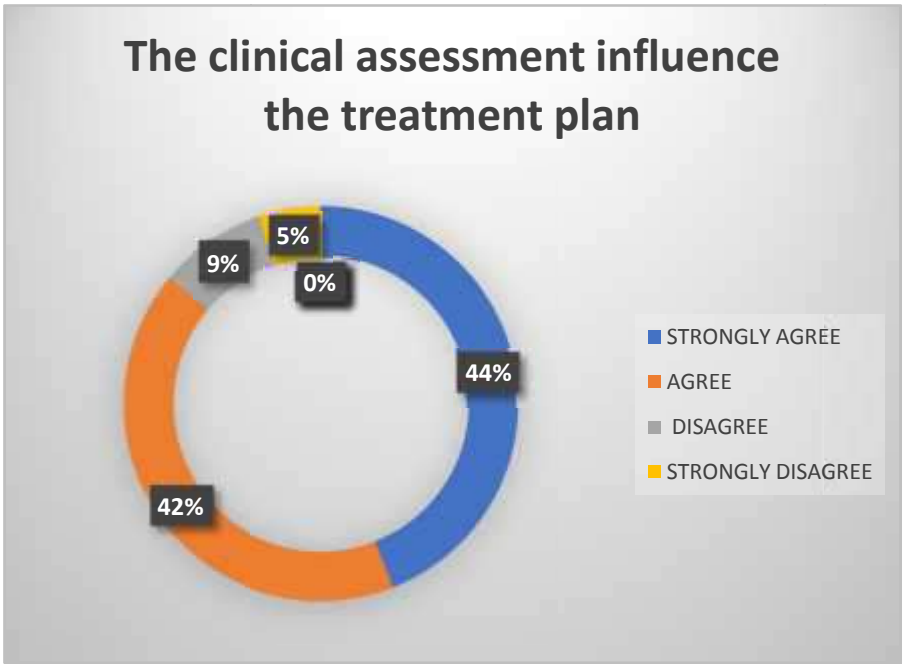


Figure 6: Clinical assessment on influencing treatmentplan

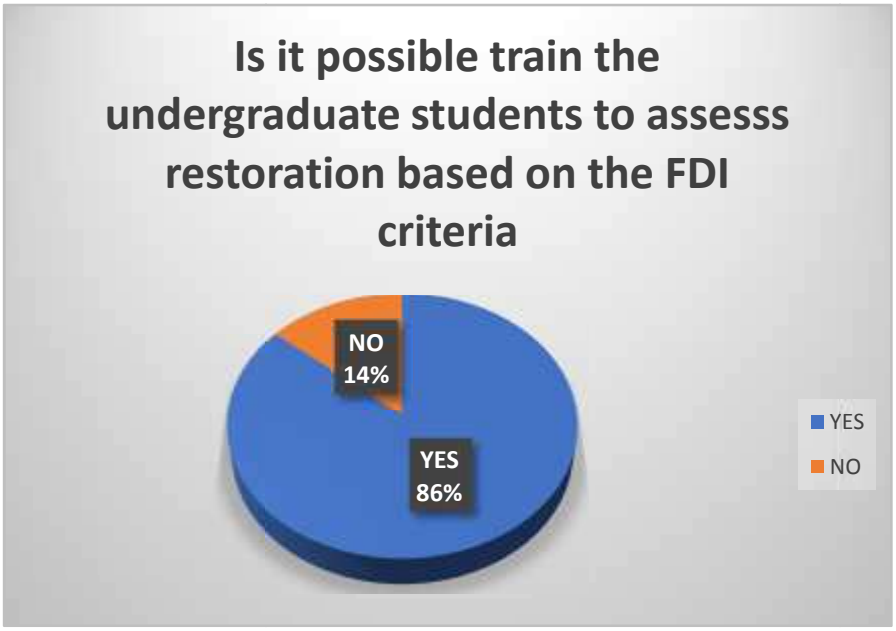


Figure 7: Possibility of traing an undergraduate

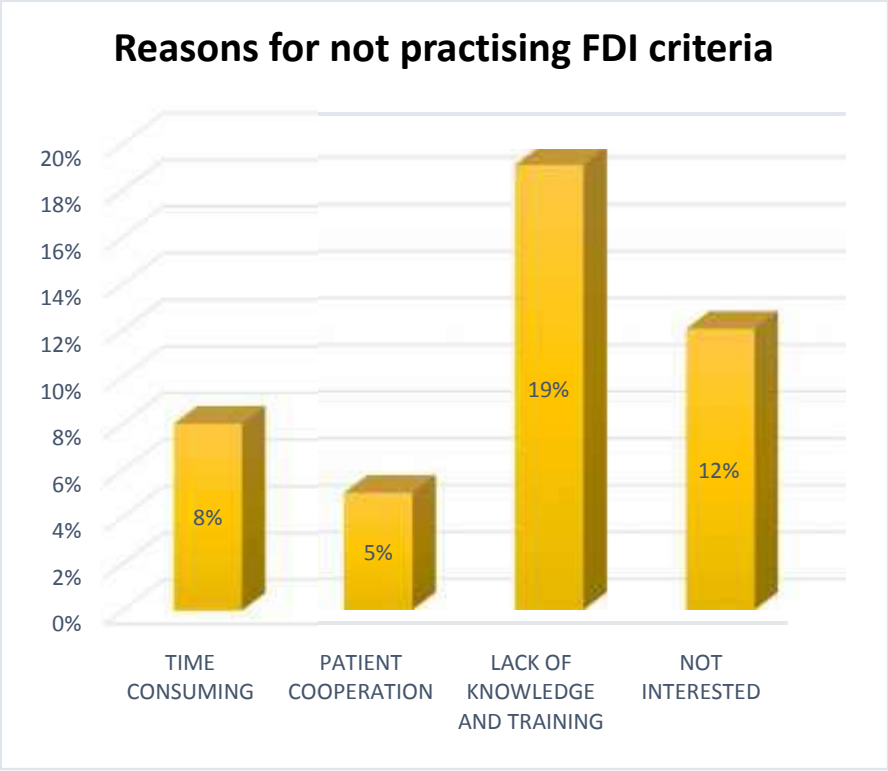


Figure 8: Reason for not practising criteria

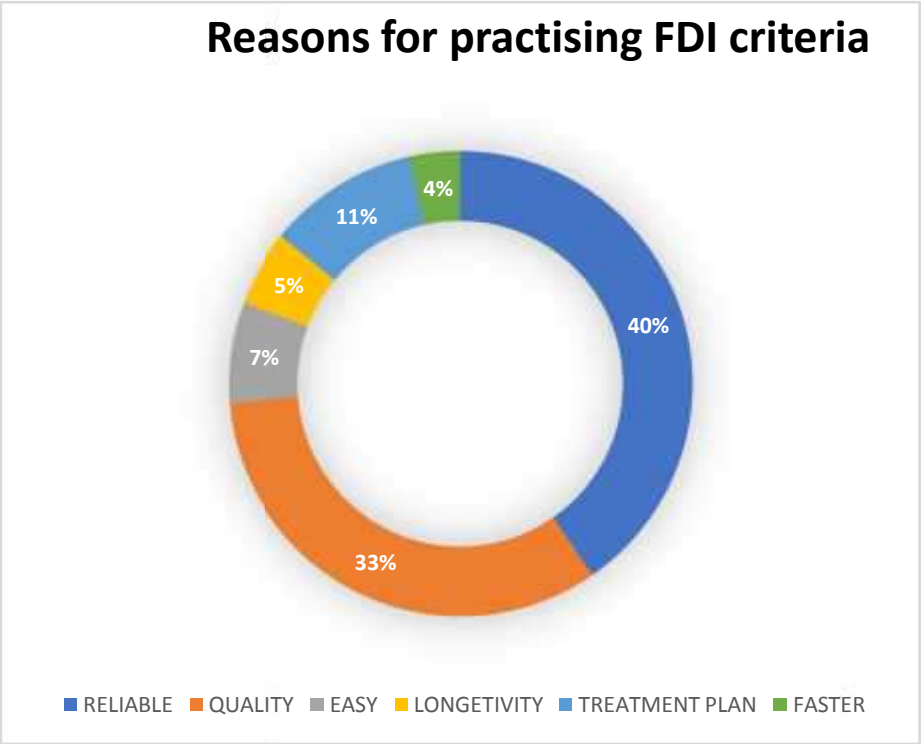


Figure 9: Reason for not practising criteria

Total respondents n=100 completed the questionnaire. Among 100 dentists 53 are females and 47 are males. Minimum year of experience of dentists has been surveyed to be 1 year and maximum is 8 years.63% of the dentists have 3 years of experience.

51%of respondents were aware of FDI criteria, 45% with Ryge and only 4% were well versed with both (Figure1). Among 100 dentists 58 were trained to assess the restoration (Figure2). 84% of participants favoured universally accepted clinical criteria for evaluation(Figure 3) . Most of the dentists shared their opinion that evaluation of dental restoration should be done after 6,12 and 18 months only(Figure5). 86% of dentists reported that it is possible to train the undergraduate students to assess the restorations(Figure7).

45% of the dentists prefers assessment of restoration through direct vision, 35% through radiographs,25% through colour radiographs and 4% through black and white photographs(Figure4). 86% of respondents feel that clinical assessment influence the treatment plan(Figure6). Reliability of FDI criteria is responsible for dentist adapting it for restoration evaluation.

Discussion

Dental caries is the most common oral health disease in developing countries[11]. The conservative approach of treating dental caries is dental restorationsMany studies have demonstrated that major work of the dentist is re-restoration of previously restored teeth. The estimations of annual expenditure of replacement dentistry were 5000 million dollars in USA[12,13].600 million Euros in Netherland[14], 100 million Great Britain pounds in UK[15]. To avoid premature replacement of restoration, FDI clinical criteria and scoring system for the evaluation of both direct and indirect restoration have been introduced.

This study is to evaluate the knowledge of dentists in assessing the dental restoration. Knowledge of clinical criteria is important, which plays a role in treatment plan and in turn success of the procedure. Moreover the dental material used and the technique can also be assessed. 86% of participants were aware of FDI criteria for evaluating the restorations and 4% knew both Ryge and FDI criteria. We found that more than half respondents are not trained for assessing the restorations. 80% respondents strongly want criteria to be followed for evaluation of restoration which gives a positive scope for this system. Most of the dentists answered that restoration is assessed through direct vision which is true but even colour photographs can help in assessing which is seen in e-calib trainings but 36% and 4% population failed to give correct answer by choosing radiographs and black and white photographs which cannot currently help in assessment. We found that about 86% of participants completely agree that standard clinical criteria helps in treatment plan.

Recent studies in 2016 by Thomas marquillier et al. shows that 16.3% of the studies used FDI criteria and the percentage of using this criteria has increased from 4.5% in 2010 to 50% in 2016 and the most employed criteria was marginal adaptation and the least in the study was surface luster[16]. Another study by Reinhardhickle reported that a grouping of esthetic, functional and biological categories have simplified the clinical evaluation procedures[17]. On other hand a study by s Deepak et al. stated that FDI criteria is not sensitive enough to determine the minor changes like proximal contact tightness[18] instead there are better instruments which was designed by loomans et al. at university of technology at Delft, Nertherlands[19]and other instrument by investigators at university of Tokushima, Korea[20]. It is surprising to know that even though FDI criteria has lot of advantages it remains as a hurdle to practice it with ease. Some of the dentists feels it is time consuming or have limited

knowledge. The dentists also responded that FDI criteria can be easily taught to students.

Conclusion

The FDI clinical criteria and scoring system is very efficient system for evaluation of direct and indirect restorations. It is a well- structured and flexible criteria, which can be selected and adjusted according to the needs of the investigator. With the limitations of the study, we conclude that by proper training and calibration this criteria can be applied not only by the researchers but also by dental students and general practitioners for quality assurance purpose of replacement and repair of dental restorations.

Corresponding Author

Sandhya Raghu,

Reader,Department of Conservative of Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077, Tamil Nadu, India

Mobile Number: +91 9884610410

Mail id: drsandhyaendo@gmail.com

References

1. Anusavice KJ. Criteria for selection of restorative materials: properties vs technique sensitivity. Quality evaluations of dental restorations. 1989:15-56.

2. Hickel R, Roulet JF, Bayne S, Heintze SD, Mjör IA, Peters M, Rousson V, Randall R, Schmalz G, Tyas M, Vanherle G. Recommendations for conducting controlled clinical studies of dental restorative materials. Clinical Oral Investigations. 2007 Mar 1;11(1):5-33.

3. Piva F, Coelho-Souza FH. A deciduous teeth composite restoration clinical trial using two methods.InJ Dent Res Abstract 2009 (No. 3241).

4.VKnibbs PJ. Methods of clinical evaluation of dental restorative materials.Journal of oral rehabilitation. 1997 Feb 1;24(2):109-23.

5. Elderton RJ. The causes of failure of restorations: a literature review. Journal of dentistry. 1976 Nov 1;4(6):257-62.

6. Elderton RJ. The quality of amalgam restorations.Assessment of the quality of dental care. 1977:45-81.

7. Downer MC, O'Brien GJ. Evaluating health gains from restorative dental treatment.Community

dentistry and oral epidemiology. 1994 Aug 1;22(4):209-13.

8. Shugars DA, Bader JD. Appropriateness of care. Appropriateness of restorative treatment recommendations: a case for practice-based outcomes research. The Journal of the American College of Dentists. 1992;59(2):7-13.

9. Bader JD, Shugars DA. Variation, treatment outcomes, and practice guidelines in dental practice. Journal of dental education. 1995 Jan;59(1):61-95.

10. Friedman JW, Atchison KA. The standard of care: an ethical responsibility of public health dentistry. Journal of public health dentistry. 1993 Sep 1;53(3):165-9.

11. Poorani ES, Chandana CS. Prevalence of dental caries among Chennai population. J Pharm Sci Res. 2015;7(10):895-6.

12. Mjor IA, Gordan VV. Failure, repair, refurbishing and longevity of restorations. Operative dentistry. 2002 Sep 1;27(5):528-34.

13. Maryniuk GA. Replacement of amalgam restorations that have marginal defects: variation and cost implications. Quintessence International. 1990 Apr 1;21(4).

14. Bronkhorst E. Interne gegevensonderzoeklijngezondheidszorg. VakgroepCariologie en Endodontologie. Nijmegen: KatoliekeUniversiteit. 1988.

15. Sheldon T, Treasure E. Dental restoration: what type of filling.Effective Health Care. 1999 Jul;5(2):1-2.

16. Thomas M, Sophie D, Florence C, Kerstin G, Jean-Christophe M, Pierre M, Matthieu P, Brigitte G, Elisabeth D. The use of FDI criteria in clinical trials on direct dental restorations: A scoping review. Journal of dentistry.2017 Oct 18.

17. Hickel R, Peschke A, Tyas M, Mjör I, Bayne S, Peters M, Hiller KA, Randall R, Vanherle G, Heintze SD. FDI World Dental Federation: clinical criteria for the evaluation of direct and indirect restorations—update and clinical examples. Clinical oral investigations. 2010 Aug 1;14(4):349-66.

18. Deepak S, Nivedhitha MS. Proximal contact tightness between two different restorative materials–An in vitro study. Journal of Advanced Pharmacy Education & Research| Apr-Jun. 2017;7(2).

19. Loomans BA, Opdam NJ, Roeters FJ, Bronkhorst EM, Burgersdijk RC. Comparison of proximal contacts of Class II resin composite restorations in vitro. Operative dentistry. 2006 Nov;31(6):688-93.

20. Oh SH, Nakano M, Bando E, Shigemoto S, Kori M. Evaluation of proximal tooth contact tightness at rest and during clenching. Journal of oral rehabilitation. 2004 Jun 1;31(6):538-45.

A Survey on Rubberdam Usage among Students during Dental Treatment

Yendodu Varshitha[1], Iffat Nasim[2], Arvind. M[3]

Graduate Student[1], Reader[2], Department of Conservative and Endodontics, Professor[3], Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The use of rubber dam is an excellent means of providing infection control during dental treatment by reducing bacterial contamination of prepared cavities, root canal system and reducing the transmission of infections between dentist and patient. Rubber dam is recommended for various restorative and endodontic procedures.This study was conducted to evaluate the attitude towards the use of rubber dam in clinical practice by students of a private dental school.The rubber dam is considered to be the greatest benefit to patients safety.

Materials and Method: A prepiloted questionnaire was prepared and distributed to 120 juniorstudents in private dental college. The questionnaire included various aspects on rubber dam use in dental procedures like indications, advantages difficulty in application. Statistical analysis done by using chi-square test and used for comparison of qualitative data ($p<0.05$) was estimated.

Results: 79.3% males accepted that rubber dam is adequate usage in dental school training and 20.7% opinionated that training is inadequate. 81.7% females accepted it is adequate and 18.3% accepted it is not.

Conclusion: The response to the questionnaire was mixed amongst the participants which was statistically significant. It is very much necessary to increase the awareness amongst practitioners to the benefits of a rubber dam usage by means of continuing education. Rubber dam will make dentistry much faster, easier and safer for both practitioners and patients.

Key Words:*Rubber dam, Clamp, Dental school, Restoration, Clinical standard*

Introduction

Rubber dam has been considered as an ideal method to control infection in the oral environment. It was introduced in dentistry in 1864 by S.C. Barnum, a Newyorkcity dentist. To provide isolation, to create an aseptic area and to prevent the ingestion of irrigants rubber dam application has been in practice for more than 150 years[1]. It offers various advantages such as dry, clear operative field, improved access and visibility, soft tissue protection and retraction during operative procedures, prevention of infection transfer and aspiration of instrument and materials and also for the patients who are sensitive to gag reflex[2,3]. Although universally accepted, dentists fail to use it in their daily practice[4]. . It has been widely recommended in developed countries[5,6]. It is consider to be an essential component in modern restorative dentistry[7,8]. Many dental schools emphasizes all students to use to the rubber dam during all operative procedures, the matter of concern is whether they strongly adopt using rubber dam throughout their dental practice. In spite of all recommendation its wide range of functions is often overlooked by dental practitioners[10,11,12]. Literature shows rubber dam in pediatric dentistry has been on use for more than 100 years[13]. The present questionnaire based survey aimed to assess students perspective on the use of rubber dam in routine dental practice.

Materials and Methods

A prepiloted questionnaire was prepared and distributed to 120 interns and post graduate students of private dental collage, Chennai. The questionnaire included various aspects on rubber dam use in dental procedures like indications, advantages difficulty in application. Statistical analysis done by using chi-square test and significance ($p<0.05$) was estimated.

Table 1: Questionnaire

1. Gender
male female
2.Training in dental school on the use of rubber dam
a) Adequate b) Inadequate
3. Greatest advantage offered by the rubber dam
a) Provision of isolation and an aseptic working area

b) Preventive of swallowing or aspirating materials
c) Preventing the ingestion of irrigants
4. What is the major factor that makes rubber dam application difficult?
a) Selection of clamp and its adaptation
b) Placement of rubber dam
c) Placement of the frame
5. Rubber dam is not a helpful adjunct because
a) I experience difficulty during application
b) I believe that it consumes time
c) I believe patients do not like it.
6. Is patient explained about need to use rubber dam?
a) Yes
b) No
7. Patient opinion on rubber dam application
a) Doctors benefit
b) Their benefit
c) Both
8. Patient experience with rubber dam
a) Pleasant
b) Uncomfortable
c) Painful
9. Do you ask your patient whether they have latex allergy prior to rubber dam usage
a) Yes
b) No
10. Patient preference on rubber dam to be during next dental treatment
a) Yes
b) No
11. Which procedure you feel it is mandatory to use rubber dam
a) Amalgam filling
b) Composite resin filling

c) Root canal treatment
d) All the above
12. Your most common reason not to use rubber dam for restorative procedure is
a) Time and cost
b) Inconvenience
c) Patients refusal
13. Do you feel that the rubber dam has any effect on your restorative procedures
a) Yes
b) No
14. A higher clinical standard is possible when restorations are placed under a rubber dam
a) Yes
b) No
15. Restorations placed under the rubber dam have greater longevity than those placed without
a) Yes
b) No
16. You believe rubber dam prevents contamination
a) Yes
b) No
17. Do you use rubber dam in pediatric patients
a) Yes
b) No
18. Time taken to apply rubber dam
a)Seconds b)Minutes
19.Do you use of rubber dam in private practice following graduation
a) I strongly believe that it is helpful tool
b) I only use it because I am obliged to
20. Following graduation
a) I intend to use the rubber dam during all the procedures required
b) I intend to use only during restorative procedures
c) I intend to use it only during root canal treatment

d) I will never use it.

Results

Total respondents were 120 students. Among 120 students there were 29 males and 91 females. Among them 79.3% males accepted that rubber dam is adequate usage in dental school training and 20.7% opinionated that training is inadequate. Among females 81.7% accepted it is adequate and 18.3% accepted it is not.

Table 2: Answers given by the students regarding the adequacy of rubber dam

Training in dental school on the use of rubber dam	Gender						P-VALUE
	Male		Female		Total		
	N	%	N	%	N	%	
Adequate	23	79.3	75	82.4	98	81.7	0.706
Inadequate	6	20.7	16	17.6	22	18.3	
Total	29	100.0	91	100.0	120	100.0	

In terms of advantage offered by rubber dam about 63.3% with statistical significance(P=0.037) mentioned that provision of isolation and an aseptic working area as a top ranked benefit and 29.2% of students mentioned that prevention of swallowing or aspirating materials as another advantage. 88.3% students agreed that rubber dam prevents contamination but 11.7% said it is not helpful in preventing contamination.

Table 3: Answers given to the questions based on utilization of rubber dam usage

Questions	Options	Total		P- VALUE
		N	%	
Greatest advantage offered by the rubber dam	Provision of isolation and an aseptic working area	76	63.3	0.037
	Prevention of swallowing or aspirating materials	35	29.2	
	Preventing the ingestion of irrigants	9	7.5	
	Total	120	100.0	P-VALUE 0.995
Believe rubber dam prevents contamination	Yes	106	88.3	
	No	14	11.7	
	Total	120	100.0	

Regarding difficulties faced during rubber dam usage, 44.2% students reported that selection of clamp and its adaptation as their difficulty and 41.7% students felt difficulty in placement of rubber dam. Only 14.2% had difficulty in placement of the frame .40% students had an opinion that rubber dam is not a helpful adjunct because patients do not like it. 38.3% felt that it consumes time (p=0.009).45.8% students did not use the rubber dam due to patients refusal.

Table 4: Opinion of students based on the difficulties faced during Rubber Dam usage

Questions	Options	Total		P- VALUE
		N	%	
Major factor that makes rubber dam application difficult	Selection of clamp and its adaptation	53	44.2	0.771
	Placement of rubber dam	50	41.7	
	Placement of the frame	17	14.2	
	Total	120	100.0	
Rubber dam is not a helpful adjunct because	Difficulty during application	26	21.7	0.009
	It consumes time	46	38.3	
	Patients do not like it	48	40.0	
	Total	120	100.0	
Most common reason not to use rubber dam for restorative procedure is	Time and cost	26	21.7	0.304
	Inconvenience	39	32.5	
	Patients refusal	55	45.8	
	Total	120	100.0	

73.3% students (p=0.022) felt that patient should be explained about rubber dam usage. 42.5% of respondents felt patients feel rubber dam is used for both doctors and patients benefit, 35% opinioned it is only for dentists benefit. 62.5% students were of opinion that patient find wearing rubber dam to be uncomfortable. 73.3% of students questioned the patients regarding latex

allergy prior to rubber dam usage. 70.8% (p=0.002) students reported that patients will never prefer wearing the rubber dam next time.

Table 5: Answers given by the students regarding Rubber Dam usage on Patients

Questions	Options	Total		P-VALUE
		N	%	
Patient explained about need to use rubberdam.	Yes	88	73.3	0.022
	No	32	26.7	
	Total	120	100.0	
Patient opinion onrubber dam application	Doctors benefit	42	35.0	0.730
	Their benefit	27	22.5	
	Both	51	42.5	
	Total	120	100.0	
Patient experience with rubber dam	Pleasant	10	8.3	0.451
	Uncomfortable	75	62.5	
	Painful	35	29.2	
	Total	120	100.0	
Do you ask patients whether they have latex allergy prior rubber dam usage	Yes	88	73.3	0.187
	No	32	26.7	
	Total	120	100.0	

Patient preference on rubber dam to be used during next Dental treatment	Yes	35	29.2	0.002
	No	85	70.8	
	Total	120	100.0	

Majority of the students told that they do not use rubber dam on pediatric patients (78.3%). 50% of students reported that they use rubber dam because it is a helpful tool remaining do it as an obligation. Following graduation 44.2% students preferred rubber dam only for restorative procedures and 22.5% for all the dental procedures. 15% students said that they will use it only during root canal treatment. 18.3% said they will never use rubber dam following their graduation.

Table 6: Student's opinion about Rubber Dam usage

Questions	Options	Total		P- VALUE
		N	%	
Use of rubber dam in pediatric patients	Yes	26	21.7	0.883
	No	94	78.3	
	Total	120	100.0	
Use the rubber dam in the clinic because	It is helpful tool	60	50.0	0.831
	Obliged to	60	50.0	
	Total	120	100.0	
Use of rubber dam in private practice Following	Intend to use the rubber dam during all the procedures required	27	22.5	

graduation	Intend to use only during restorative procedures	53	44.2	0.217
	Intend to use it only during root canal treatment	18	15.0	
	Will never use it	22	18.3	
	Total	120	100.0	

According to 84.2% students, higher clinical standard is possible when restorations are placed under rubber dam while 15.8% did not agree for the same.

Table 7: Students opinion on effect of rubber dam usage on restorations

Questions	Options	Total		P- VALUE
		N	%	
Feel that the rubber dam has any effect on your restorative procedures	Yes	93	77.5	0.451
	No	27	22.5	
	Total	120	100.0	
A higher clinical standard is possible when restorations are placed under a rubber dam	Yes	101	84.2	0.777
	No	19	15.8	
	Total	120	100.0	
Restorations placed under	Yes	95	79.2	

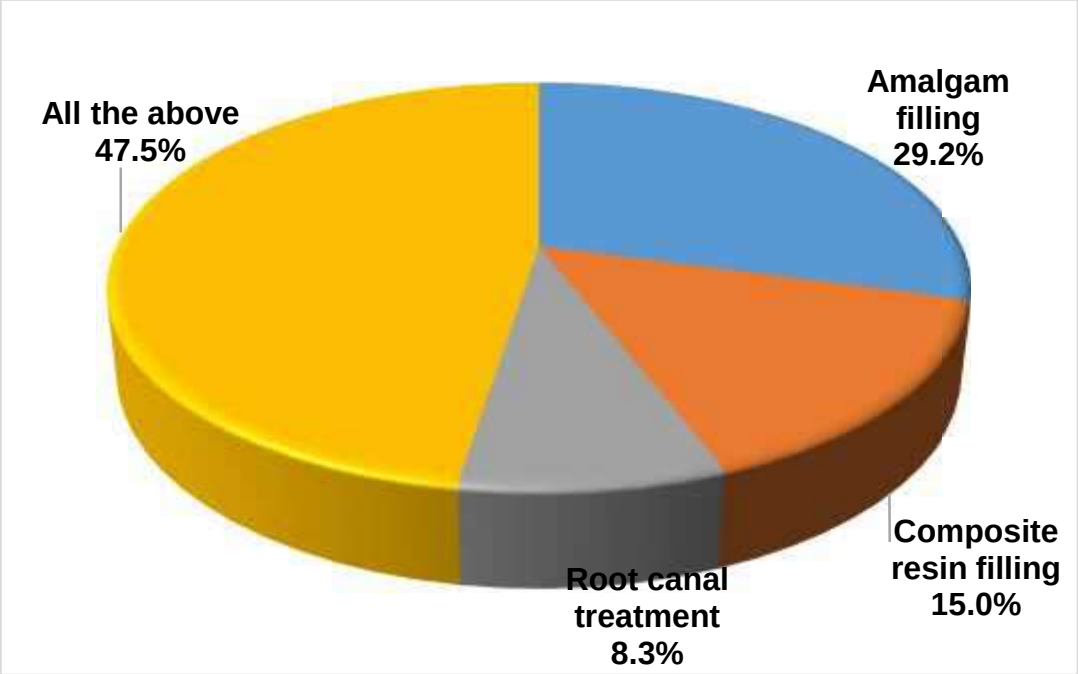
the rubber dam have greater longevity than those placed without	No	25	20.8	0.615
	Total	120	100.0	

47.5% students shared their opinion that rubber dam application is mandatory for all the procedures while 29.2% students reported that it is mandatory only for amalgam restorations

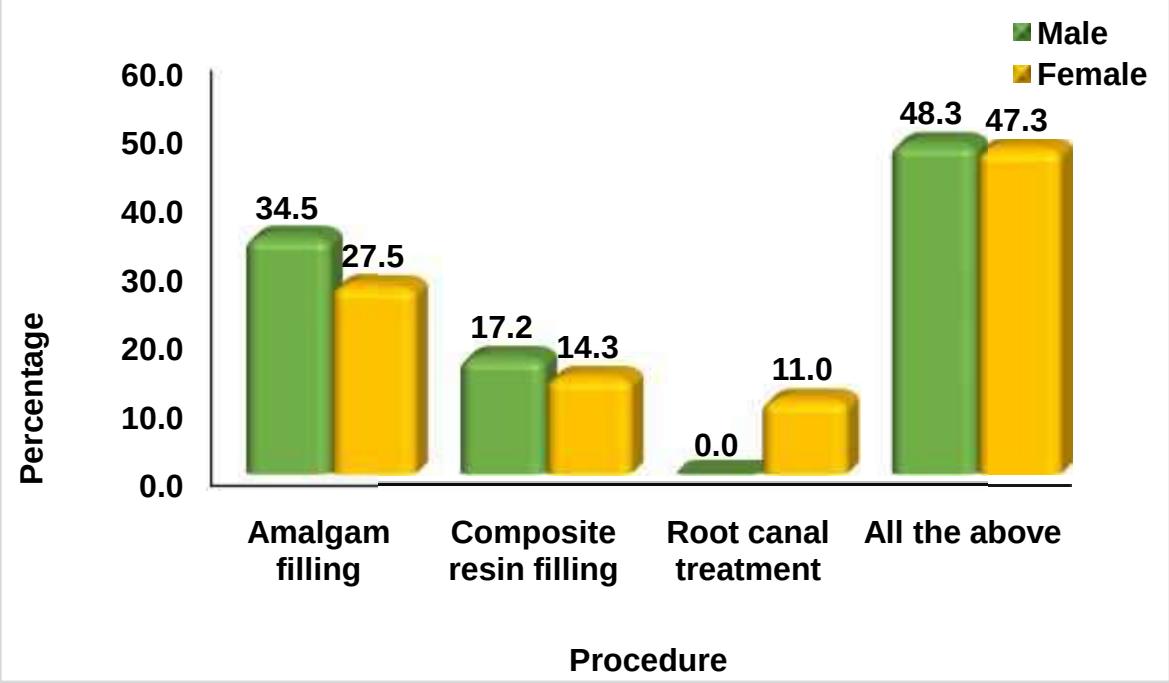
Table 8: Answers given by students on procedure that require rubber dam

Procedure which is mandatory to use rubber dam	Total		P- VALUE
	N	%	
Amalgam filling	35	29.2	0.279
Composite resin filling	18	15.0	
Root canal treatment	10	8.3	
All the above	57	47.5	
Total	120	100.0	

Graph 1: Shows procedure which is mandatory to use rubber dam



Graph 1: Shows procedure which is mandatory to use rubber dam



The mean time taken for rubber dam application was 7.72 minutes for males and 10.55 minutes for females with a statistical significance(p=0.021).

Table 9: Time taken by students for the application of rubber dam

	Gender	N	Mean time	Std. Dev	t-Value	P-Value
Time taken to apply rubber dam (Min)	Male	29	7.72	4.300	2.331	0.021
	Female	91	10.55	6.050		
	Total	120	9.87	5.789		

Discussion

Conducting a survey on a topic without external influence is quite difficult as stated by Hill and Rubel[9]. As students are in their learning phase they may be tempted to give what is perceived as correct answer instead of an honest answer.Majority of the participants thought the rubber dam usage training in dental school is adequate. It is surprising to know that even though rubber dam has a lot of advantages it remains as a topic of controversy. It was disappointing to note that few students reported that rubber dam is not a useful adjunct in preventing contamination. As rubber dam is an excellent tool for isolation, the reason for such response may be due to incorrect rubber dam application. The majority of students find the selection of clamp and its adaptation difficult. The reason behind it may be presence of inadequate tooth structure posing difficulty in placement of regular clamp or they were not having clamps for different teeth. Adequate knowledge about availability of various clamps in the market and selection of clamp according to the available tooth structure may help to resolve this problem. Pertaining to difficulty in placement and selection of clamp, repeated application may help to resolve this problem. Other problemsnoticed in rubber dam acceptance were patient refusal. Adequate patient education and motivation could help in overcoming this problem. Many students were not using rubber dam in pediatric patients, the reason being patients are uncooperative to this device. As rubber dam improves the quality of dental materials, proper education should be given to the parents for its

acceptance. It is rather highly disappointing to know that half of the student population think that they are obliged to use it and few opinionated that they will not be using this device further in their practice. It is accepted that there is a strong learning curve in rubber dam application and its usage, the advantage of using should be overweighed in comparison to difficulties encountered in its usage.

In a study from Belgium, about 64.5% of practitioners do not use rubber dam in their daily practice, while only a very few that is 3.4% believed that rubber dam to be a standard procedure[14]. Whitworth et al.stated that thepatients dislike towards rubber dam can be strongly determined by the cliniciansattitude. Stewardsonand McHugh et al. also stated that the experience of the dentist and the efficiency and technique regarding the usage of rubber dam in the patients oral cavityshould be achieved by frequent practice. In most of the studies practitioners are not been asked of latex allergy to the patient prior the application of rubber dam which suggests that more attention should to be directed towards the possibility of latex allergy prior to application of the rubber dam as it causes intra oral complications .

Mala et.al,stated that theincrease percentage of students who did not use rubber dam for child patients (89.1%) [15]. This difficulty however needs to be considered from a pedodontic point of view, probably in a future study focusing on this group of patients. Percentages of students with this difficult facing the rubber dam were increased than that of those who reported by Mala et al. Marshall and Page (1990) in their study used Patient discomfort as main reason for not using rubberdam[16]. But the limitation of spending extra time in placing the dam is compensated with better working conditions offered by the dam including controlling the saliva contamination and eliminating the need to frequently change cotton rolls as well as limiting the movements of the patient’s tongue and lips. As it is already evident that rubber dam may reduce the incidence of post-treatment disease during root canal treatment[17].Saranyapackiriet al reported that 37.4% believe it is a useful tool and45.8% students felt that patient’s do not like it, there by implyingin its disincentive use in future dental practice (26.2%)[18].In other study on non caries cervical lesions, Haripriya et al stated that, it is evident that majority of the practitioners are not aware of isolation methods for restoring NCCLs and those who are aware also do not imply them in their clinical practice. Therefore, knowledge about conventional methods of isolation and newer methods of gingival retraction should be imparted[19].

Conclusion

As rubber dam is considered as a standard tool in terms of proper oral health delivery care, it judicious use cannot be neglected by the students. By increasing the awareness among the patients and by heightening educational awareness among studentsthe ultimate goal of standard method of isolation can be achieved and practiced widely.

Corresponding Author

Arvind. M,

Professor, Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

Email Id: arvindmuthukrishnan@yahoo.com

Mobile Number: +91 9444303303

References

1. Rule RW Rubber dam: its use and adjustment. Pac Dent Gas. 1931; 39, 541-546.

2. Cochran MA, Miller CH, Sheldrake MA The efficiency of the rubber dam as a barrier to the spread of microorganisms during dental treatment. J Am Dent Assoc.1989;119,141-144.

3. Lynch CD, McConnell RJ The use of microabrasion to remove discolored enamel: a clinical report. J Prosthet Dent.2003;90, 417-419.

4.Ireland L. The rubber dam.Its advantage and application. Tex Dent J. 1962;80,6-15.

5. Rugg-Gunn AJ, Welbury RR, Toumba J British Society of Paediatric Dentistry: a policy dicument on the use of amalgam in pediatric dentistry. Int J Paediatr Dent.2001; 11, 233-238.

6. Fayle SA, Welbury RR, Roberts JF. British Society Of Paediatric Dentistry: a policy document on the management of carries in primary dentition.2001;Int J Paediatr Dent 11,153-157.

7.Small BW. The rubber dam: a step toward clinical excellence. Compendium Continuing Educ Dent 2002;23(3):276-80,282.

8. Terry DA. An essential component to adhesive dentistry: the rubber dam. Pract Proc Aesthetic Dent 2005;17(2):106,108.

9. Hill EE, Rubel BS Do Dental Educators Need To Improve Their Approach To Rubber Dam Use? Journal of Dental Education 2008;72:1177-81.

10. Ahmad IA. Rubber dam usage for endodontic treatment: A review. International Endodontic Journal 2009;42:963-72.

11. Bjorndal L, Reit C. The adoption of new endodontic technology amongst Danishgeneral dental practitioners. International Endodontic Journal 2005;38:52-8.

12. Gergely EJ. Desmond Greer Walker Award. Rubber dam acceptance.British Dental Journal 1989; 167: 249-52.

13. Jinks GM. Rubber dam technique in pedodontics. Dent Clin North Am1966*:* 327–340.

14. J. M. Whitworth, G. V. Seccombe, K. Shoker, and J. G. Steele,“Use of rubber dam and irrigant selection in UK general dental practice,” International Endodontic Journal, vol. 33, no. 5, pp.436–441, 2000.

15. S. Mala, C. D. Lynch, F. M. Burke, and P. M. H.Dummer,“Attitudes of final year dental students to the use of rubber dam,”International Endodontic Journal, vol. 42, no. 7, pp. 632–638,2009.

16. D.J.Kleier and K.Shibilski,“Management of the latex hypersensitive patient in the endodontic office,” Journal of Endodontics,vol. 25, no. 12, pp. 825–828, 1999.

17. E.Kosti and T.Lambrianidis,“Endodontic treatment in cases of allergic reaction to rubber dam,”JournalofEndodontics,vol.28,no. 11, pp. 787–789, 200

18. Saraniyapackiri, Rubber Dam- its present and future use among dental students. Int J Pharm Bio Sci 2016 July ; 7(3): (B) 838 – 842.

19. Haripriya, Knowledge, attitude, and practice survey on isolation techniques and its importance in restoring non-carious cervical lesions among general practitioners in Chennai., Journal of Advanced Pharmacy Education & Research.,2017.

20. Fayle SA, Welbury RR, Roberts JF. British Society Of Paediatric Dentistry: a policy document on the management of carries in primary dentition.2001;Int J Paediatr Dent 11,153-157.

A Survey on the Preference of Spectacles or Lens in Myopic Patients

LeyaMeriam Mathews [1], M.P.Brundha [2], Shantha Sundari [3]

Graduate Student [1],Senior Lecturer [2],Department of General Pathology

Professor and Head [3],Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: Nearsightedness, or myopia, is the most common refractive error of the eye, and it has become more prevalent in recent years. Myopia occurs when the eyeball is too long, relative to the focusing power of the cornea and lens of the eye. This causes light rays to focus at a point in front of retina, rather than directly on its surface. Symptoms include squinting, eye strain, distant vision not clear, and headaches .Treatment options include use of prescription glasses, contact lens, and corneal refractive therapy.

Materials and Method: To survey the myopic population using spectacles and contact lens, on certain practical issues. A comparative analysis of the use of spectacles and contact lens in terms of frequency, regularity, comfort and complications if any

Results: Most of the participants wore prescription glasses over contact lens for obvious reasons of comfort and better vision. However, contact lens was aesthetically preferred over prescription glasses and also helped give a clear vision, due to less chances of fogging.

Conclusion: The survey, meant to appraise personal opinions of myopic patients, on their use of visual aids, willhelp to understand various aspects of a very commonly used device in day to day life, the spectacle and contact lens. The study will reveal the efficacy and short comings posed by both, in the attempt to correct the inevitable.

Keywords: *Myopia, short -sight, contact lens, spectacles,aesthetics*

Introduction

Myopia also known as near-sightedness or short sight is one of the common refractive error of the eye and ocular disease in Asia.It is the state of refraction where the parallel rays of light are brought to focus in front of retina [1]. Therefore, a distant object tends to be out of focus. The onset of myopia is observed between the age of 5 and 15 years [2].Myopia is normally signified by a blurry distant vision and at times, people with high myopia, the near sight is also affected. On examination, it is often observed that, the optic nerve is tilted with pigmentation on the macula. Squinting, headaches are other symptoms observed. It occurs when the eyeball is too long, relative to the focusing power of the cornea and lens of the eye. This causes the light rays to focus at a point in front of the retina.Severe cases of myopia can even lead to blindness because of its associated ocular co morbidities of retinal detachment, macular degeneration and glaucoma [3].The cause of myopia is still debated as being pathological, congenital or acquired. However, other factors which could cause myopia are high sugar levels in diabetic patients, visual stress and even a family history of myopia could put an individual at high risk.People with myopia have several options to correct their vision. Spectacles, contact lens, laser surgery,vision therapy for people with stress related myopia and corneal refracted therapy.For most people with myopia, spectacles are primary choice for correcting distantvision. Generally, a single vision lens is required to provide a much clearer and better vision. However, certain studies show that children now opt for contact lenses as choice of treatment. Contact lenses like spectacles, provide correction of vision, but cannot be worn full time.The objective of this analysis was to find the amount of time and level of comfort felt, when the subjects wore their mode of correction and, to assess the efficacy and short comings of each

Materials and Methods

The study was conducted among the South Indian population, in Saveetha Dental College, Tamil Nadu, India, during the month of May 2017. .The subjects were selected at random, among those who attended Saveetha Dental College, Out- patient department, with visual aids. Fifty participants who accepted to participate in the study,in the age group 15 - 30 years were included in the study.

A questionnaire was prepared and distributed. Participants were questioned based on their preference to spectacles or lens on issues like level of comfort, age, advantages, disadvantages and their knowledge on other treatment options available. Subjects were asked if the prescribed mode of correction helped to correct myopia.

The collected data was then analyzed and compared accordingly.

Results

The data collected from the participants was evaluated and the following observations were made.As shown in Table 1,more number of the participants preferred prescription glasses over contact lens to correct myopia. With increase in age, the number of people whoused glasses and lens alternatively on a daily basis also increased. It was also noted that only few participants chose contact lens as first choice of treatment to correct myopia when compared to spectacles

Table I: Age based distribution of spectacle and lens users

Age group	Only spectacles used	Only contact lens used	Both	Number who used Spectacle the most	Number who used contact lens the most
15-20 years	0	0	7	5	2
20-25 years	7	2	8	10	7
25-30years	13	3	10	20	6
TOTAL	20	5	25	35	20

A few number of theparticipants used both spectacles and contact lens to treat myopia. However,when askedabout the age they had begun to use Glasses / Contact lens, data(Table II) showed that participants, when less than 10 years of age preferred using spectacles. Contact lens was used on the rise from late teenage towards young adults beyond 18 years age.

Table II: Age of commencement of Treatment

Age Group	Spectacles	Contact Lens
6-12years	6	0
13-18years	5	6
19-24years	7	9
25-30years	7	10

Among the advantages listed, aesthetics, vision and level of comfort were questioned. Data collected showed that among lens user's aesthetics and better vision (80%) was the major advantage, while comfort andvision (60%)was the primary advantage among spectacle users. (Figure 1)

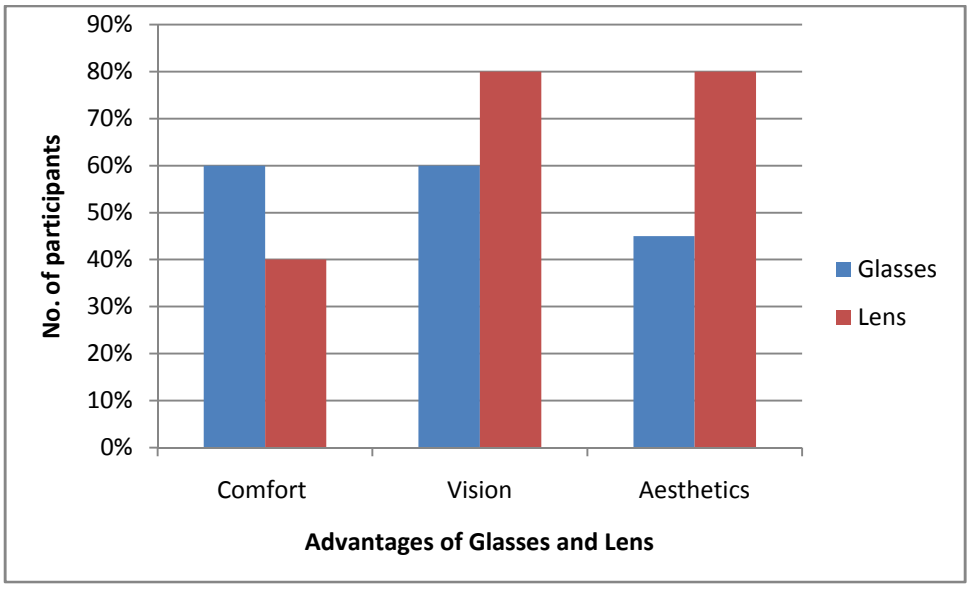


Figure 1: Comparison of advantages

However, the disadvantages listed about lens users, were also more compared to prescription glass users (Figure 2). About 80 -70% complained about irritation and the time consumption for wear, while using lens. Lens also required excessive care when compared to glasses, to avoid any damage or infection. Less aesthetics and fogging were more observed in prescription glasses. Most of the participants did not find many difficulties while using spectacles

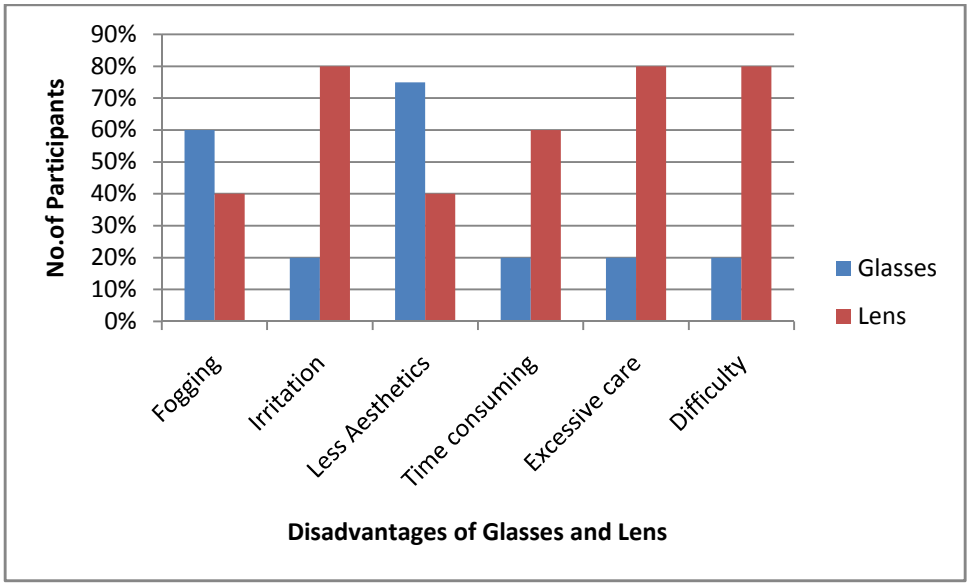


Figure2: Comparison of Disadvantages.

Knowledge about other treatment options was also asked during the survey. Laser and eye exercises were the most listed treatment options. Latest treatment options include corneal refractive treatment and Orthokeratology. (Table 3)

Table 3 Alternative ways for correction of Myopia

Age Group	Eye Exercise	Laser	Others
15-20years	5	2	0
21-26 years	7	7	3

27-30 years	10	8	8

Discussion

Myopic patients are provided with refractive correction, especially young patients, to help them have a clear distant vision .School work, sports and other activities benefit from optimal vision. Studies have also shown that myopia can further have social, economic and educational consequences, if not corrected on time [4].

The underlying pathogenesis behind myopia is still debatable whether it is acquired, pathological or congenital [5]. Studies show a possible chance that, myopia could begenetically acquired. Zadnik et.al conducted a study focusing on the refractive errors of both the parents and children. All the components of refraction of both the parents and children were measured and it was observed that children with myopic parents tend to have longer eyes than children without myopic parents. In myopia, the image is formed in front of the retina because the cornea or lens curvature is too strong or the eye is too long Hence, this factor could be a possible predisposition to become myopic later in life [6, 7]. Myopia has also been related to various ocular co morbidities like pathologies in retina, pigmentary degeneration, optic nerve crescent, bilateral rhegmatogenous retinal detachment [8].

In this study, most of the participantswore prescription glasses over contact lens for obvious reasons of comfort and better vision. It was also observed, that those participants who usedlens for a much shorter time in a day attributed it due to the excessive care needed to maintain duringwear and time consumption needed for wearing. However,contact lens was aesthetically preferred over prescription glasses and also helped give a clear vision,due to less chances of fogging.

The studies related to self-perception and physical appearance was quite interesting. It appearedthat, among thosewho used both spectacles and contact lens, those who were conscious of their looks, preferred to use lens for longer hours, in spite of being knowledgeable, that spectacles helped to retard myopic progression better. In fact, certain studies indicate that

physical appearance or self – esteem was significantly improved among those who felt good about their contact lenses [9].

Age is another factor which comes to light while judging between contact lens and spectacles. Studies show that optometrists recommend spectacles at a primary age than contact lens. Most importantly, the maturity of the child and other criteria like motivation to take care of it, personal hygiene also determined why at a primary age, spectacles is more preferred [10].

People with low myopia, tend to lose spectacles as they do not wear it whole time. Fogging of the glasses and less aesthetic appearance are other disadvantages listed out while wearing spectacles. They also do not help much in myopic correction.

From this study, it has been observed that the better choice of the treatment to control myopia is spectacles. Spectacles can be given at a primary age to children, at the age of 11, as a source of primary myopic correction. Spectacles also do not offer complete vision correction when compared to lens, but definitely more comfortableand easy to maintain. Studies show that though, spectacles were less aesthetic, they providedcorrection over a range of refractory errors, reduce retinal defocus thereby providing a corrective refractive treatment [11, 12].

Reasons cited for preferring contact lens for correction of myopia, have been better vision, ocular health, less discomfort and also boost up the self - esteem.But studies show that contact lens are not effective in retarding myopic progression[13] .Corneal curvature flattening, which could also be responsible for a myopic progression have been noted while using contact lens [14,15]. Change in axial length rather than refractive error is typically used as the outcome measure because of corneal flattening produced by the lenses. Pilot studies have shown that the use of contact lenses have also increased the progression of myopia .The longitudinal Orthokeratology research in children was a pilot study conducted in Hong Kong which showed that lenses slowed axial elongation and the progression of myopia. [16,17]. In conditions like astigmatism , which causes blurred vision due to irregular curvature of the cornea, spherical lenses was not very effective in providing a better treatment [18].

As per recent studies, it was observed that on anaverage, a person wore spectacles for 13.5 hours and lens for 11 hours but, neither contact lens nor spectacles provided total correction of vision

with time. Currently, the only mode of total correction is corneal refractive therapy, without the use of corrective device [19].

The search for an effective intervention to slow down the progression of myopia continues to be hampered due to a lack of clear understanding of the pathogenesis of myopia .Effective therapy should be more focused on environmental factors, early age of onset, retinal degenerative factors in the future. Other treatment options like orthokeratology, corneal refractive therapy, atropine drops have also been tried and better results have been yielded to correct myopia [20, 21].

Conclusion

As per the data analysis, it was observed that spectacles were more preferred over contact lens among patients who attended the out-patient department at Saveetha Dental College. At a primary age, prescription glasses were recommended by ophthalmologists, with thematurity of the child in mind, the less amount of care needed for maintenance and personal hygiene. Contact lens, although aesthetically favorable, was preferred less due to the demand for excessive care, irritation and discomfort .Studies show that both prescription glasses and contact lens did not aid in providing a complete effective treatment and other treatment options like corneal refractive treatment, Orthokeratology should be further explored.

Corresponding Author

Shantha Sundari

Professor ,Department of Orthodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,

Saveetha University,

162, Poonamallee High Road,Chennai- 600077,Tamil Nadu, India.

Email: shanthakkss@gmail.com

Telephone Number: +91 9444443640

References

1. Curtin, B.J., Karolin, D.B. Axial length measurements and fundus changes of the myopic eye (I. The posterior fundus).Transactions of the American Ophthalmological Society1970; 68:312–334.
2. Saw SM, Tong L, Chua WH, Chia KS, Koh D, Tan DT, Katz J. Incidence and progression of myopia in Singaporean school children. Investigative Ophthalmology and Visual Science. 2005 Jan; 46(1):51-7.
3. Revalo JF, Ramirez E, Suarez E, Antzoulatos G, Torres F, Cortez R, Morales-Stopello .Rhegmatogenous retinal detachment after laser-assisted in situ keratomileusis (LASIK) for the correction of myopia. Journal of Cataract and Refractive Surgery 2001 May; 27(5):674-80.
4. Aslam A. Contact lenses and spectacles: a winning combination. Optician, 2013; 246:6425 26-28.
5. P J Foster1, 2, and Y Jiang1, Epidemiology of myopia Eye (Lond). 2014 Feb; 28(2): 202–208.
6. Douglas R Fredrick, Myopia, 2002 May 18; 324(7347): 1195–1199.
7. Zadnik K, Satariano WA, Mutti DO, Sholtz RI, Adams AJ , The effect of parental history of myopia on children's eye size. JAMA. 1994 May 4; 271(17):1323-7.
8. Cheng SC, Lam CS, Yap MK. Prevalence of myopia-related retinal changes among 12-18 year old Hong Kong Chinese high myopes. Ophthalmic Physiology Opt. 2013 Nov; 33(6):652-60.
9. Fonn D, Gauthier CA, Pritchard N. Patient preferences and comparative ocular responses to rigid and soft contact lensesOptometry and Vision Science: December 1995.; 72: 857–863.
10. Cho P, Cheung SW, Edwards M. The longitudinal orthokeratology research in children (LORIC) in Hong Kong: a pilot study on refractive changes and myopic control. Current Eye Research 2005; 30: 71–8

11. Walline JJ, Jones LA, Mutti DO, and Zadnik K .A randomized trial of the effects of rigid contact lenses on myopia progression Archivesof Ophthalmology. 2004 Dec; 122(12):1760-6
12. Jensen H. Myopia progression in young school children: A prospective study of myopia progression and the effect of a trial with bifocal lenses and beta blocker eye drops. Acta Ophthalmologica. 1991; 200:1–79.
13. Lisa A Jones-Jordan, Monica Chitkara, Bradley Coffey, A comparison of spectacle and contact lens wearing times in the Archive study, Ohio State University College of Optometry, Columbus, USA . Clinic and Exp Optometry , May 2010; 93(3):89-93
14. Fulk GW, Cyert LA, Parker DE, and West RW the effect of changing from glasses to soft contact lenses on myopia progression in adolescents. Ophthalmic Physiology Opt. 2003 Jan; 23(1):71-7.
15. Cho P, Cheung SW, Edwards M.The longitudinal Orthokeratology research in children (LORIC) in Hong Kong: a pilot study on refractive changes and myopic control. Curr Eye Res 2005 Jan; 30(1):71-80.
16. Sevda Aydin Kurna, Tomris Şengör, Murat Ün, and Suat Aki. Success rates in the correction of astigmatism with toric and spherical soft contact lens fittings. Clin Ophthalmol. 2010; 4: 959–966
17. Saw SM, Katz J, Schein OD, Chew SJ, and Chan TK: Epidemiology of myopia. Epidemiology Rev 1996, 18, 175-187
18. Horner DG, Soni PS, Salmon TO, Swartz TS Myopia progression in adolescent wearers of soft contact lenses and spectacles. Optometry Visual Science. 1999 Jul; 76(7):474-9.
19. Walline JJ, Jones LA, Sinnott L, Chitkara M, Coffey B, Jackson JM, Manny RE, Rah MJ, Prinstein MJ, ACHIEVE Study Group. Randomized trial of the effect of contact lens wear on self-perception in children. Optometry and Vision Science. 2009 Mar 1;86(3):222-32.
20. Shih YF, Hsiao CK, Chen CJ, Chang CW, Hung PT, Lin LL .An intervention trial on efficacy of atropine and multi-focal glasses in controlling myopic progression. Acta Ophthalmol Scand. 2001 Jun; 79(3):233-6.

21. Barr JT1, Rah MJ, Jackson JM, Jones LA. Orthokeratology and corneal refractive therapy: a review and recent findings. Eye Contact Lens. 2003 Jan;29:33-38

An Endodontic Practice Profile Amongst General Practitioners In Chennai- A Questionairre Survey

Nandhini.S[1], D.Delphine Priscilla[2], V.Jayanth kumar[3]
Graduate Student[1],Senior Lecturer[2], Department of Conservative Dentistry,Reader[3], Department of Oral Medicine and Radiology,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: The practice of dentistry shows a vast difference owing to the place of practice that is., between private practice and institutional one as well as between the general practitioner and the specialists. Endodontic treatment itself offers a wide gamut of options to a clinician in the way a simple root canal can be carried out. Use of rubber dam, irrigants, prescription of antibiotics, choice in Single and multiple-visit treatment and interappointment dressings are the aspects of one most commonly carried out procedure that is., root canal treatment in dentistry

Materials and Method: This study was a questionnaire-based survey of samples of dentists in Chennai. The self-administered questionnaire contained 15 close-ended questions with multiple choice options. The data collected included demographic details of respondents, root canal preparation techniques, irrigants and intra canal medicaments used, the number of appointments, method of working length determination, root filling techniques, cements used, and the scope of treatment performed.

Result: The usage of rubber dam is very low among the general practitioners. Most of the dentists follow radiograph with instrument in the canal as the best option for working length determination. Out of three root canal instrumentation techniques like crown down, push and pull and step back, the third one(56%) is the mostly followed technique. Hero shaper(51%) is the choice of Ni-Ti rotary instrument used by the dentists of Chennai followed by M Two. Stainless steel hand files(69%) are mostly used to prepare canals followed by nickel titanium hand files(21%) and rotary nickel titanium files(10%).Lateral compaction(91%) is the

commonly used obturation technique.Autoclave sterilization(20%) is mostly followed by the dentists followed by glass bead and chemical sterilization(12%).The most commonly used sealer is ZOE sealer (72%).

Conclusion: During past decade many innovative concepts, techniques and instruments have been introduced in practice. And standard of care for endodontics can be improved by increasing general dental practitioner interest. This study concludes that standard guidelines and new technologies are to be implemented by the practitioners in Chennai.

Key Words: *Endodontic practice, general practitioners, root canal, medicaments, apex locator.*

Introduction

The practice of dentistry shows a vast difference owing to the place of practice that is., between private practice and institutional one as well as between the general practitioner and the specialists. Endodontic treatment itself offers a wide gamut of options to a clinician in the way a simple root canal can be carried out. Use of rubber dam, irrigants, prescription of antibiotics, choice in Single and multiple-visit treatment and interappointment dressings are the aspects of one most commonly carried out procedure that is., root canal treatment in dentistry.With the introduction of new materials, devices and techniques, quality of endodontic treatment has increased leading to higher endodontic success rate.[1,2] With the development of these technologies, many teeth with guarded prognosis that used to be extracted in the past can be salvaged by surgical or non-surgical endodontic treatment. Endodontic treatment includes all clinical procedures designed to maintain the teeth in a functional state in the dental arch. The challenges of preserving the dental arch free of pulp and periapical diseases have contributed to numerous innovations in contemporary endodontic practice. Research data on attitude of general dental practitioners towards endodontic therapy is rare.[3] Several studies have investigated the attitudes of general dental practitioners towards various aspects of endodontic treatment in England,UK, Belgium, Nigeria, Sudan, Australia, Denmark, USA, Sweden, Turkey, Jordan and Iran. But there has been no survey of the current status of endodontic practice in India. So, the primary purpose of this survey was to determine the attitude of general dental practitioners towards endodontic

treatment and current use of new endodontic technology and materials by them.[4,5] Changes in the field of endodontics have been so rapid in recent years that current instruction in root canal treatment (RCT) bears little resemblance to that of 10 years ago. The consequence is that dentists are faced with numerous materials and techniques, while dental schools try to tailor their curriculum accordingly.In order to ensure acceptable treatment outcomes, quality guidelines for endodontic practice have been published.The aim of the study is to investigate and compare the status of endodontic practice among Indian endodontists that will help suggest a standardized treatment guideline.

Materials and Methods

The target population of this investigation was the general dental practitioners in Chennai.A sample of 100 questionnaires with 15 closed ended questions weredistributed to them in their working place.Convenience sampling was used and samples were chosen to be 100 arbitrarily.Question categories included different aspects of endodontic treatment, including root canal therapy stages, materials, choice of instruments, isolation methods, use of canal irrigants, use of intracanal medicaments and choice of obturation technique.Percentages were then calculated, based on the number of respondents to each question.The data was analyzed using descriptive statistics.[6,7,8]

Table 1: Questionnaire

1.Gender
A) male b)female
2 .Do you use rubber dam in your endodontic treatment ?
a)always
b) occasionally
c) never
3.If its more than one visit ,what intra canal medicament do you give ?

a) CaOH

b)Formocresol

c) iodoform

4.Method of working length determination

A) apex locator

B) pre operative radiograph

C)Radiograph with instrument in the canal

5.Method of root canal instrumentation

A) crown down

B) step back

C) push and pull

6.Which one do you prefer the most ?

a) step back

b) crown down

7.Which is your choice of Ni Ti rotary instrument ?

a)Profile

b) hero 642

c) pro taper

8.Length of time of use of rotary NI TI ?

A) 0 to 12 months

B) 13 to 24 months

C)35 months

D) more than 36 months

9.How do you retrieve fractured instrument?

A) ultra sonic

B) by pass the instrument

C) apical surgery

10.Which irrigating solution you use?

A) sodium hypochlorite

B) Normal Saline

C) hydrogen peroxide

11.which type of obturation technique you commonly use?

A) lateral compaction B) warm compaction c) single cone

12.Type of RCT complication which u think is more common

A) irrigant related b) perforation c) over instrumentation.

13.How do you sterilise your endodontic files ?

A) glass bead sterilisation

B) autoclave

C) chemical sterilisation

14.How do you manage emergency ?

A) analgesics and antibiotics

B) pulpectomy

C)pulpectomy and medication

15.The sealer that you use mostly is

A) ZOE

B) endomethasone

C)sealapex

Results

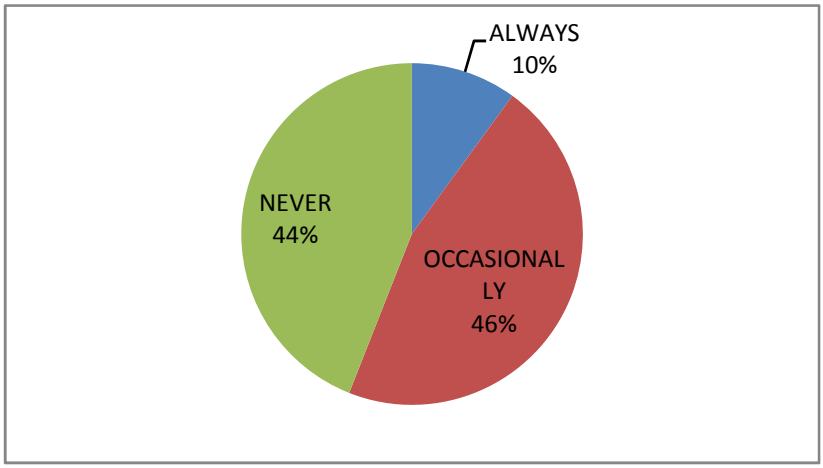


Figure 1: Frequency of rubber dam usagein clinical practice

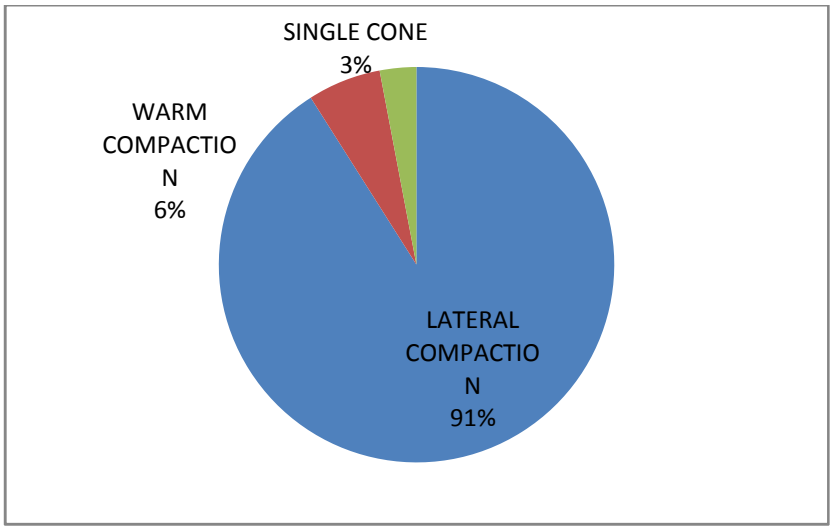


Figure 2:Type of obturating technique used

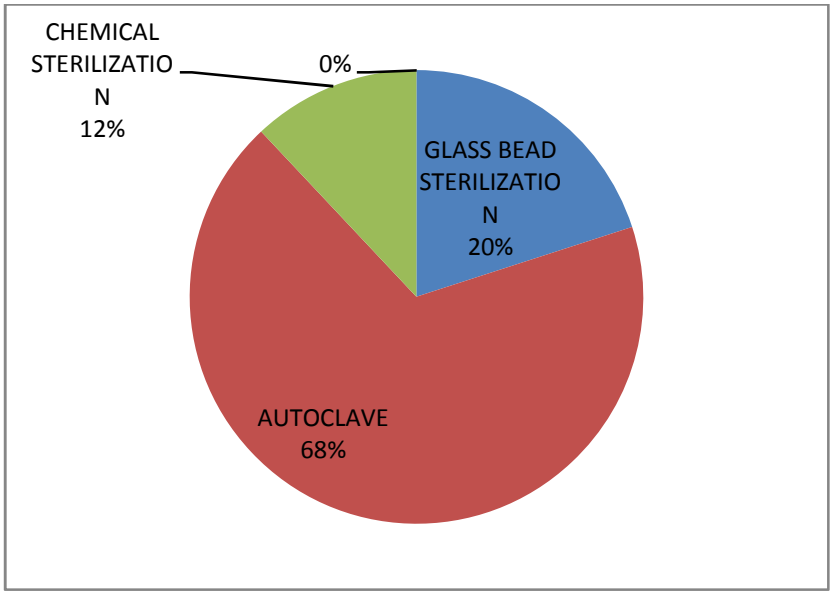


Figure 3:Sterilization of endodontic files

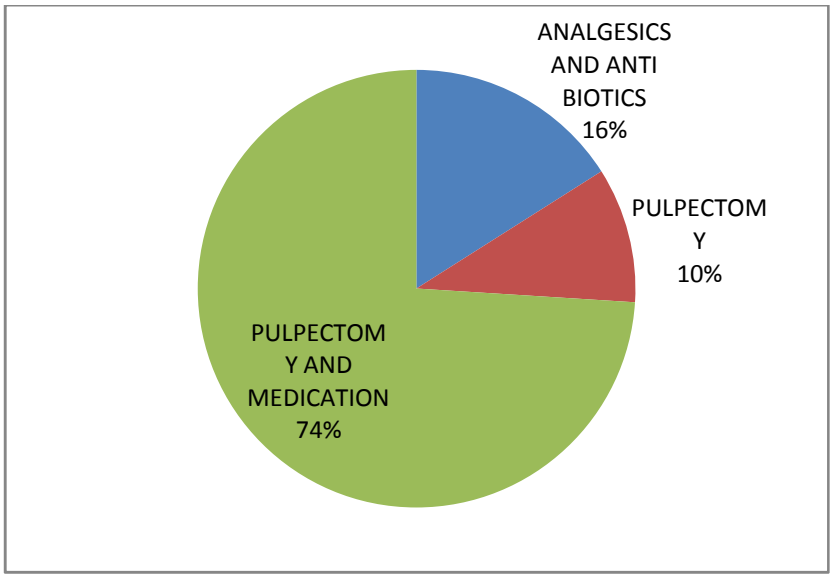


Figure 4: Mode management of endodontic emergency

This study investigated the endodontic practice profile.Out of 100 dentists here,only very few use rubber dam.The mostly used intracanal medicament is CaOH (74%) followed by formocresol and iodoform.Most of the dentists follow radiograph with instrument in the canal as the best option for working length determination. Out of three root canal instrumentation techniques like crown down,push and pull and step back, the third one(56%) is the mostly followed technique.Hero shaper(51%) is the choice of Ni-Ti rotary instrument used by the dentists of Chennai followed by M Two.Stainless steel hand files(69%) are mostly used to prepare canals followed by nickel titanium hand files(21%) and rotary nickel titanium files(10%).Most of the dentists bypass the fractured instrument(46%).Ultrasonic method(17%) and apical surgery(22%) are followed by very few dentists.Lateral compaction(91%) is the commonly used obturation technique then comes warm compaction(6%) and single cone(3%).Obturating beyond apex (42%) is the common root canal complication.Autoclave sterilization(20%) is mostly followed by the dentists followed by glass bead and chemical sterilization(12%).The most commonly used sealer is ZOE sealer (72%).Resin based(10%), calcium hydroxide based (9%) and bioceramic sealers(9%) are also used.

Discussion

A dentists' attitude, knowledge and practice can be determined in an effective manner by conducting a questionnaire survey. Face to face surveys have response rates higher than electronic or mailed questionnaires. The respondents in the current study were dentists who were able to provide valid and valuable information on the current status of endodontic practice. The major limitations and weaknesses of this study include the inflexible nature of the study design. As opposed to direct observations, it is difficult to deal with specific contexts in the current questionnaire-based study. Moreover, some questions that may be more appropriate to respondents may have been missed in an attempt to standardize the questions.[9,10,11]

This study investigated the endodontic practice profile.Out of 100 dentists here,only very few use rubber dam.Because its time consuming and patient discomfort rate is very high. All endodontic procedures should be performed with a rubber dam, which should be considered standard care. The purpose of rubber dam protection in dentistry on whole cannot be overempasized. Although rubber dam isolation is taught as mandatory during training, its importance appears to be ignored by most of the Endodontists in India.The mostly used intracanal medicament is CaOH (74%) followed by formocresol and iodoform. For intracanal medicament, calcium hydroxide usage was the most popular followed by formocresol, which was similar to that practiced by dentists in HK and endodontists of India. Calcium hydroxide has been determined as suitable for use as an intracanal medicament as it is stable for long periods, harmless to the body, and bactericidal in a limited area. It also induces hard tissue formation and is effective for stopping inflammatory exudates. Besides, intracanal dressing is also for a blockade against coronal leakage from the gap between filling materials and cavity wall. On the other hand, formocresol has a tissue fixative property and also is distributed to the whole body from the root apex and so might induce various harmful effects including allergies. Furthermore, as these medicaments are potent carcinogenic agents, there is no indication for these chemicals in modern endodontic.Most of the dentists follow radiograph with instrument in the canal as the best option for working length determination.With use of either electronic apex locator or radiographs, the respondents preferred to be short of the apex, while Lee et al. in their survey showed that 30-40% of respondents instrumented to the level of apex reading on the electronic apex locator regardless of the clinical situation[12,13,14].

Periapical radiographs are important in endodontics for diagnosis, determine the number, location, shape, size, and direction of roots and root canals, estimate and confirm the length of root canals before instrumentation, localize hard-to-find, or disclose unsuspected, pulp canals by examining the position of an instrument within the root, aid in locating a pulp that is markedly calcified and/or receded, determine the relative position of structures in the facial–lingual dimension, confirm the position and adaptation of the primary filling point and aid in the evaluation of the final root canal filling.This method can be useful in patients who need not to be exposed to repeated radiation because of mental, medical or oral conditions.Moreover, electronic apex locators areparticularly useful when the apical portion of the canalis obscured by anatomic structures, such as impactedteeth, tori, the zygomatic arch, excessive bone density,overlapping roots or shallow palatal vaults. Irrigation has been performed in conjunction with endodontic therapy for many years. One of the primary reasons for irrigating the root canal system is to ensure cleanliness of the canals prior to obturation. This cleanliness involves both elimination of microorganisms and removal of organic matter.Out of three root canal instrumentation techniques like crown down,push and pull and step back, the third one(56%) is the mostly followed technique. A crown-down approach provides certain advantages including early organic debris removal, the creation of a large reservoir for irrigating solutions, a straighter access to the apical region of curved canals, and greater precision with regard to the exact working length and apical size.20 Only 23 % of the GDPs used crown-down technique while 90% still followed step-back technique.Hero shaper(51%) is the choice of Ni-Ti rotary instrument used by the dentists of Chennai followed by M Two.Stainless steel hand files(69%) are mostly used to prepare canals followed by nickel titanium hand files(21%) and rotary nickel titanium files(10%). Non-rotary manual endodontic files were commonly used by the endodontists surveyed. Recently introduced rotary NiTi files are able to produce a uniformly tapered canal configuration without canal transportation.[13,14,15]

However, unpredictable instrument separation remains a deterrent to their popularity. Moreover, cost of rotary files also restrained their use in the developing countries like India. Majority of respondents only replaced their instruments when signs of distortion and bluntness were obvious, which is likely to result in a higher risk of instrument separation in the canal. Most of the dentists bypass the fractured instrument(46%).Ultrasonic method(17%) and apical surgery(22%) are followed by very few dentists.Lateral compaction(91%) is the

commonly used obturation technique then comes warm compaction(6%) and single cone(3%).The preferred technique for filling the root canals in the current study was the lateral condensation technique and this has been reported as the most universally accepted technique.5 It is also the preferred technique among undergraduate dental students.[16,17,18]Obturating beyond apex (42%) is the common root canal complication.Autoclave sterilization(20%) is mostly followed by the dentists followed by glass bead and chemical sterilization(12%).The most commonly used sealer is ZOE sealer (72%).Resin based(10%), calcium hydroxide based (9%) and bioceramic sealers(9%) are also used.

There are some limitations of the present study which are worth mentioning. Since, the study used a face to face questionnaire it was not possible to survey a large sample size. The survey didn't attempt to differentiate the participants based on their specialty and training. Hence, it could be assumed that not all the dentists would perform all procedures listed in the questionnaire frequently. This could result in their response to be based on knowledge and not on routine practice.

Conclusion

This study investigated and compared the status of endodontic practice among Indian endodontists. It showed that dentists are following the basic protocols and techniques in accordance with recognized international standards. But still there are certain differences that suggest a need for standardization of treatment guidelines for the Indian specialist.[19,20] These findings would help reach a consensus to set an Indian endodontic treatment protocol/guideline by the Indian Board of Endodontists similar to one done by European Society of Endodontists, American Association of Endodontists and the Canadian Academy of Endodontists.Duringpast decade many innovative concepts,techniques and instruments have been introduced in practice.And standard of care for endodontics can be improved by increasing general dental practicionerinterest.This study concludes that standard guidelines and new technologies are to be implemented by the practicioners in chennai.[21]

Corresponding Author

JayanthKumar.V,

Reader, Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University,Chennai, India.

Email Id:doctorjayanth@gmail.com

Mobile Number: +91 8056135807

Reference

1. Bergenholtz G, Spanberg L. Controversies in Endodontics,*Crit Rev Oral Biol Med* 2004;15:(99-114).

2. Chan AW, Low D, Cheung GS, Ng RP. Questionnaires survey of endodontic practice profile among dentists in Hong Kong.,*Hong Kong Dent J* 2006;3:80-87.

3.Udoye CI, Jafarzadeh H. Rubber dam use among a subpopulation of Nigerian dentists, *J Oral Sci* 2010;52:245-49.

4. European Society of Endodontology. Consensus report of the European Society of Endodontology on quality guidelines for endodontic treatment.,*Int Endod J* 1994;27:115-24.

5.Jenkins SM, Hayes SJ, Dummer PMH. A study of endodontic treatment carried out in dental practice within the UK,. *IntEndod J*.2001;34(1):16–22.

6. Chan AWK, Low DCY, Cheung GSP, Ng RPY. A questionnaire survey of endodontic practice profile among dentists in Hong Kong. *Hong Kong Dent J*.2006; 3(2):81-87.

7.Al-Omari WM. Survey of attitudes, materials and methods employed in endodontic treatment by general dental practitioners in North Jordan.*BMC Oral Health.*2004;4(1):34-89.

8.Slaus G, Bottenberg P. A survey of endodontic practice amongst Flemish dentists,*IntEndodJ.* 2002;35:759-767.

9.Vadhera N, Makkar S, Kumar R et al. Practice profile among endodontists in India: A nationwide questionnaire survey,*Ind J Oral Sci*. 2012; 3(2):90-93.

10.Ahmed MF, Elseed Al, lbrahim YE: Root canal treatment in general practice in Sudan,*IntEndod J.* 2000;33:316-319.

11. ShohrehRavanshad, SaiedSahraei, and Akbar Khayat,Survey of Endodontic Practice amongst Iranian Dentists Participating Restorative Dentistry Congress in Shiraz, November 2007,*Iran Endod J*. 2008 Winter; 2(4): 135–142.

12.ShohrehRavanshad and Mohammad Reza Nabavizade,Endodontic Treatment of a Mandibular Second Molar with Two Mesial Roots: Report of a Case,*Iran Endod J.* 2008 Fall; 3(4): 137–140.

13. M. F. Ahmed ,A. I. Elseed ,Y. E. Ibrahim, Root canal treatment in general practice in Sudan,*J British EndodonticSoceity*, 2670;33:396-366.

14. Cartwright A. Professionals as responders: Variations in and effects of response rates to questionnaires, 1961-77,*Br Med J.* 1978;2(6149):1419–21.

15. Brazier JE, Harper R, Jones NM, O'Cathain A, Thomas KJ, Usherwood T, et al. Validating the SF-36 health survey questionnaire: New outcome measure for primary care, *BMJ*. 1992;305(6846):160–66.

16. Pineda F, Kuttler Y. Mesiodistal and Buccolingualroentgenographic investigation of 7,275 root canals,*JOral Surg Oral Med Oral Path*. 1972;33:101-10.

17. SmadiL,Comparison between two methods of working length determination and its effect on radiographic extent of root canal filling: a clinical study,*BMC Oral Health*. 2006;6:4;789-990.

18. Sharma CMC, Arora MGV. Determination of Working Length of Root Canal, *MJAFI.* 2010;66(3):231-4.

19. Pasqualini D, Bianchi CC, PaolinoDSet al. Computed micro-tomographic evaluation of glide path with nickel-titanium rotary PathFile in maxillary first molars curved canals, *J Endod.* 2012;38(3):389-93.

20. Tortini D, Colombo M, Gagliani M. Apical crown technique to model canal roots. A review of the literature.*Minerva Stomatol.* 2007;56(9):445-59.

21. MidhunMoha1, Prof. Dr. V. Susila Anand.Accuracy of Different Methods of Working Length Determination in Endodontics Dr. G. IOSR *Journal of Dental and Medical Sciences*

(IOSR-JDMS) e-ISSN: 2279-0853, p-ISSN: 2279-0861.Volume 12, Issue 4 (Nov.- Dec. 2013).

22.Jamed David Raj,Endodonticirrigants: A comprehensive review, *Journal of Pharmaceutical Sciences and Research*, 7(1):5-9.

Analysing the Reason for Lack Of Interest towards Orthodontic Treatment Among School Children –Survey

Suvitha.S [1], A.V.Arun [2], Balakrishna R N [3]
Graduated Student [1], Professor [2], Department of Orthodontics
Senior Lecturer [3], Department of Oral and Maxillofacial Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction : Many students have malocclusion in our population but the accurate percentage of students with malocclusion has not been determined properly. Hence dentists are not aware of how much percentage of people requires treatment. This study is to estimate the various reasons that why school children with malocclusion have not started orthodontic treatment, particularly in Chennai population and also how many people requires orthodontic treatment.

Aim and Objectives: The survey is to analyse the reason why school student are not talking orthodontic treatment between 15-20 years of age particularly in Chennai.

Materials and Methods: 517 school students from Chennai region who are between ages of 15-20 who have not undergone any orthodontic treatment or extraction will be examined intraoral to detect the malocclusion. The examination for malocclusion was made according to DAI as described in WHO Oral Health Survey Basic Methods, 1997. Data collected were statistically analyzed.

Result: The analyzed data shows about 68% of students in the sample are aware about their irregular dentition and 32% were not aware of their malocclusion since they don't have any functional problems. Only 10% of students were aware about various orthodontic treatments and remaining 90% of people are not aware of any orthodontic treatments that would help them to correct their malocclusion in both functionally and esthetically.

Conclusion: This study reveals lack of awareness in school children about orthodontic treatment. Hence a proper awareness and treatment protocol should be given to children and their parents. This enables them to think in better way for opting orthodontic treatment in future.

Key Words: Malocclusion, Orthodontic treatment, Dental aesthetic index, Aesthetics, Awareness.

Introduction

Malocclusion was defined as a deviation of teeth or a malrelationship of the dental arches over the level of what is accepted as normal.[1] even though malocclusion is not life-threatening,[2] it may be regarded as a public health problem because of its high prevalence and preventive treatment availability.[3] Malocclusions occupies the third highest prevalence with in oral diseases, second only to the tooth decay and periodontal disease, hence ranked third among world-wide dental public health precedence.[4]

Malocclusion occurs as a result of orofacial deformities and various etiological factors,[4] which result in various complications such as psychosocial problems related to impaired dentofacial aesthetics, disruptions of oral function, such as mastication, swallowing and speech and greater susceptibility to trauma and periodontal disease.[1] Several studies have emphasised on its impact on quality-of-life.[5,6] Since the public equates good dental appearance with success in many pursuits and societal forces define the norms for acceptable, normal and attractive physical appearance, persons with malocclusion may develop a feeling of inferiority bout their dental appearance and may feel shame in social situations this even leads to loss of their career promotion. Several methods are available which can be used to evaluate, describe and classify occlusion. Malocclusions are evaluated by dental aesthetic index (DAI), since its development in 1986; the dental aesthetic index (DAI) has proven to be simple and rapidly applied. [7] Some existing reports have demonstrated the high accuracy and validity of this index, which also compares favourably with other indices. [8, 9] It gives a mathematical value that links clinical and esthetic components to produce a single score hence it is called as a cross cultural index. This index can be used for various communities and populations without any necessary for modification. [10] According to World Health Organization (WHO), these main oral diseases should be described to periodic epidemiological surveys. The epidemiological information's on orthodontic treatment need was the interest of dental public health programs, clinical treatment, screening for treatment precedence, resource planning and third party funding.[4] estimating the value of distribution of malocclusion in childhood can make steps to prevent such a disorder and its complications and make it possible to decreasing the complexity of costly orthodontic treatment. Later, this information might help to minimize or eliminate future treatment need.

Malocclusion is one of the main reasons why a person would like to take orthodontic treatment. It's also related to the esthetic concern.Smile is an attractive thing in human appearance, a well-balanced smile is one of the important treatment goals of modern

orthodontic therapy, extensive studies on facial features have resulted in the establishment of norms that orthodontists use as guidelines to evaluate facial forms to direct therapy [11]. But many time we surprised to see that so many people in a general population with unaesthetic smile and severely misalignedteeth who have still not opted for taking up orthodontic treatment. If esthetic is the only reason for taking up orthodontic treatment, most of these people would have taken up orthodontic treatment. So this leads to the question, is that any other reason why patients who obviously have malocclusion and smile which is unaesthetic are not willing to take up orthodontic treatment. This study aims at finding out the reasons for not taking up orthodontic treatment which will help the orthodontist in future to realize the problems faced by patients and find solutions for the same.

Materials and Method

Information was collected by a single examiner. The examiner visited the school on particular dates according to schedule. The examination for malocclusion was made according to DAI as described in WHO Oral Health Survey Basic Methods, 1997.[12] Survey was conducted among the school students of velammal matriculation higher secondary school, ponneri - Chennai. Eligible children were identified and given informed consent forms to get them signed from their parents/guardians who were also notified by the school teachers on request of the investigator. A total of 517 female students of age group between 14-18 years were included in the study. A pre-structured questionnaire consisting of few questions with multiple options were given to the children to elucidate about their misaligned dentition and reason why they are not taking up orthodontic treatment.

Initially Dental Aesthetic Index (DAI) was developed in the United States of America and incorporated into the International Collaboration Study of Oral Health Outcomes by the World Health Organization (WHO, 1989) as an international index, identifies occlusal traits and mathematically derives a single score. [13] It plays an important advantage in public health programmes, paves a platform for the dentist to explain the quality of life given by the orthodontic treatment.

The DAI was based on socially defined aesthetic standards. It conjoint the gap between the aesthetic and clinical aspects of occlusion by mathematically produces single score. It has some limitations such as it couldn’t identify cases with deep overbite that impinge on gingival or palatal tissue. The DAI was developed as a screening tool for permanent dentitions; it may be not applicable for mixed dentition stage accompanied with changes in future dental appearance. [14] The DAI was evidentially proved to be reliable, simple, valid,

and user-friendly. [15, 16] Hence, it can be used and adapted globally and cross- culturally without any modification. [17, 18]

The following questionnaire format is to analyze the reason why people are not taking up orthodontic treatment,

- Name Age Sex Mobile No.
- Are you aware of your irregular teeth?
- Have you visited a dentist regarding your teeth alignment?
- Are you aware of the various treatments options that are available to correct your teeth alignment? ☐ Yes ☐ No
- Why have you not started treatment for teeth alignment?
- _____
  _____
  _____
  _____
  _____
  _____
  _____
  _____ Your Signature

Result

Questions	**Yes Percentage**	**No Percentage**
Awareness of irregular teeth	68%	32%
Visited to a dentist regarding teeth alignment	45%	55%
Awareness of various orthodontic treatment option	10%	90%

The analyzed data shows about 68% of students in the sample are aware about their irregular dentition and 32% were not aware of their malocclusion since they don't have any functional problems. Only 10% of students were aware about various orthodontic treatments and remaining 90% of people are not aware of any orthodontic treatments that would help them to correct their malocclusion in both functionally and esthetically.

Reasons

S.No:	REASON	PERCENTAGE
1	Fear of post operative pain	6
2	Fear of extraction	13
3	Crowding of back tooth region, have aesthetic smile	2
4	Fear of teeth mobility	7
5	Lack of time	10
6	Scared that my facial structure might change	3
7	Because of 12^{th} board exams	10
8	Not aware of misaligned teeth	12
9	Dentist suggests that start treatment after 18yrs of age	1
10	Facial appearance with braces will be bad	8
11	Parents are not willing for treatment	2

12	Fear of pain and inability to take food	5
13	Crowding is not affecting my aesthetic	10
14	Expensive	5
15	Uncomfortable to wear braces	6

Pre-structured questionnaire were given to 517 students of velammal matriculation higher secondary school, ponneri. The data collected from those questionnaires were analyses and the following results were concluded. About 6% of students have fear of post operative pains, 13% percentage of students have fear of extraction, 2% has crowding of posterior teeth hence they have good esthetics and are not willing for orthodontic treatment, 7% of students have fear of post operative tooth mobility, 10% of student says lack of time for treatment,3% of student reason out that their facial structure would change, 12% of students are not aware of their misaligned teeth,10 % of student says that they would start treatment after their schooling, 8% of students feel that they would have bad appearance with braces, 2% of students parents are not willing for treatment, 5% of student feels that they can't take proper food during treatment duration, 10% of student says that crowding
Is not affecting their aesthetics, 5% of students feel that orthodontic treatment is expensive, and 6% of students feel uncomfortable to wear braces.

Discussion

The inheritance of malocclusion is developed by genetic and environmental factors, but with a greater significant on the effect of oral environment in particularly the orofacial tissues such as positioning of tongue.[19-21] study shows large tongue with increased length of lingual frenum are always associated with class three malocclusion, while class I and II shows less significant variation in length of lingual frenum.[22]This study shows that about more than 60% of children were aware of their own teeth mal-alignment. Remaining 40% of student has malocclusion but they don’t notice before and so they are not aware of their misaligned teeth. Only those who were aware of malocclusion were included in this study. Students who were undergone orthodontic treatment, cleft lip and cleft palate were excluded in the survey. Students who are aware of malocclusion are not taking orthodontic treatment for Several reasons such as post operative complication, fear of extraction, malocclusion of posterior tooth , postoperative mobility, changes in facial structure, lack of time due to board exams, dentist suggested to start treatment after 18 years of age, unaesthetic appearance during

treatment period, parents are not aware and no willing for their kids orthodontic treatment, inability to take proper foods, misaligned to is looking good and it's not affecting my esthetics, expensive and discomfortable in speech while wearing orthodontic brackets.

People always enquiries about treatment options, before they go for the treatment or giving a try for those options. Likewise before planning for orthodontic treatment they would have heard some myth statements about orthodontic treatment. Like Postoperative mobility, improper food intakes are some reasons would have made scary for the children and their parents; some children are scared of extraction that is because of poor dental experience in childhood due to anxiety. Hence a proper awareness and treatment protocol should be given to children and their parents. This enables them to think in better way for opting orthodontic treatment in future.

Conclusion

This study reveals lack of awareness in school children about orthodontic treatment.

Most children with malocclusion have not started their treatment due to ignorance of treatment protocols, extraction, periodontal problems, lack of knowledge about malocclusion and other influencing factors like literacy rate and socio-economic status. Malocclusion has an impact on the social and the psychological behavior of an individual; hence knowledge on how individuals perceive and react to malocclusion in a community is necessary for effective orthodontic treatment and care.

Corresponding Author

Balakrishna R N

Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id:balakrishdr8394@gmail.com

Mobile Number: 8939896729

References

1. Mtaya M, Brudvik P, Astrøm AN. Prevalence of malocclusion and its relationship with socio-demographic factors, dental caries, and oral hygiene in 12- to 14-year-old Tanzanian school children. Eur J Orthod. 2009; 31:467–76.

2. Karaiskos N, Wiltshire WA, Odlum O, Brothwell D, Hassard TH. Preventive and interceptive orthodontic treatment needs of an inner-city group of 6- and 9-year-old Canadian children. J Can Dent Assoc. 2005; 71:649.

3. Marques LS, Pordeus IA, Ramos-Jorge ML, Filogônio CA, Filogônio CB, Pereira LJ, et al. Factors associated with the desire for orthodontic treatment among Brazilian adolescents and their parents, BMC oral health.2009;9:34.

4. Brito DI, Dias PF, Gleiser R. Prevalence of malocclusion in children aged 9-12 years old in the city of Nova Friburgo, Rio de Janeiro state, Brazil. Rev Dent Press OrtodOrtop Facial. 2009; 14:118–24.

5. Marques LS, Ramos-Jorge ML, Paiva SM, Pordeus IA. Malocclusion: Esthetic impact and quality of life among Brazilian school children. Am J OrthodDentofacialOrthop.2006; 129:424–7.

6. Bernabé E, Flores-Mir C, Sheiham A. Prevalence, intensity and extent of oral impacts on daily performances associated with self-perceived malocclusion in 11-12-year-old children. BMC Oral Health.2007; 7:6.

7. Baca-Garcia A, Bravo M, Baca P, Baca A, Junco P. Malocclusions and orthodontic treatment needs in a group of Spanish adolescents using the dental aesthetic index. Int Dent J. 2004; 54:138–42.

8. Marques CR, Couto GB, Orestes Cardoso S. Assessment of Orthodontic treatment needs in Brazilian school children according to the dental aesthetic index, Community Dent Health.2007;24:145–8.

9. Camilleri S, Mulligan K. The prevalence of malocclusion in Maltese school children as measured by the index of orthodontic treatment need. Malta Med J. 2007; 19:19–23.

10. Esa R, Razak IA, Allister JH. Epidemiology of malocclusion and orthodontic treatment need of 12-13-year-old Malaysian school children. Community Dent Health.2001; 18:31–6.

11. Sridharan S, Samantha C. Comparison of perception of smile by orthodontists and other specialty dentists: A questionnaire study. International Journal of Orthodontic Rehabilitation. 2016 Jul 1; 7(3):92.

12. World Health Organization. World Health Organization Oral Health Surveys–Basic Methods, 4th ed. Geneva: WHO; 1997.

13. Ashari A, Mohamed AM. Relationship of the Dental Aesthetic Index to the oral health-related quality of life. The Angle Orthodontist. 2015 May 27; 86(2):337-42.

14. Ansai T, Miyazaki H, Yamashita Y, Takehara T, Jenny J, Cons NC. Prevalence of Malocclusion in High School Students in Japan According to the Dental Aesthetic Index. Community Dent Oral Epidemiology 1993; 21:303-305.

15. Jenny J, Cons NC. Comparing and contrasting two orthodontic indices, the Index of Orthodontic Treatment Need and the Dental Aesthetic Index. Am J Orthodontic- facial Orthod. 1996 110:410–416.

16. Beglin FM, Firestone AR, Vig KW, Beck FM, Kuthy RA, Wade D. A comparison of the reliability and validity of 3 occlusal indexes of orthodontic treatment need. American Journal of Orthodontics and Dentofacial Orthopedics. 2001 Sep 1; 120(3):240-6.

17. Tessarollo FR, Feldens CA, Closs LQ. The impact of malocclusion on adolescents' dissatisfaction with dental appearance and oral functions. Angle Orthod.2012; 82: 403–409.

18. Peterson P, Bourgeois D, Ogawa H, Estupinan-Day S, Ndiaye C. The global burden of diseases and risks to oral health. Bull World Health Org. 2005; 83:661–669.

19. Horowitz EP, Oxbourne RH, de George FC. Cephalometric study of craniofacial variations in adult twins. Angle Orthod.1960; 30:1–5.

20. Defabianis P. Ankyloglossia and its influence on maxillary and mandibular development. (A seven year follow-up case report) FunctOrthod.2000; 17:25–33.

21. Ruffoli R, Giambelluca MA, Scavuzzo MC, Bonfigli D, Cristofani R, Gabriele M. Ankyloglossia: a morphofunctional investigation in children. Oral Dis. 2005; 11:170–4.

22. Meenakshi S, Jagannathan N. Assessment of lingual frenulum lengths in skeletal malocclusion. Journal of clinical and diagnostic research: JCDR. 2014 Mar; 8(3):202.

Analysis of Knowledge about the Hospital Warning Symbols among the Clinical Dental Assistants in a Private Dental College in Chennai – A Comparative Study

Sharanya.H [1], Brundha M. P [2], D.Sri Sakthi [3]
Graduate Student [1],Senior Lecturer [2],Department of General Pathology
Reader [3],Department of Public Health Dentistry
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: Hospital Safety Signs Are The Main Means Of Communicating Health And Safety Informations. There Are Specific Colors, Shape And Patten Of The Safety Signs. They Include The Use Of Illuminated Signs And Hand Signals . These Symbols Are Critical To Understand Without The Prime Knowledge . Health Care Workers Come Across These Signs Every Day In Their Working Environment. It Is Very Important To Make Them Aware And Train Them In Interpreting Theses Symbols Correctly.

Materials And Methods : A Questionnaire Containing 20 Frequently Encountered Hospital Signs Were Given To 40 Clinical Dental Assistants Pre And Post The Training Sessions. The Difference In Knowledge Before And After The Sessions Were Statistically Analysed. The Questionnaire Was Tested For Its Face And Content Validity.

Results : The Total Number Of Participants Were 40,With A Mean Age Of 22.2 Years. During Pre-Training, Majority Had Scored Below 10, 23(57.50%), Post Training Scores Improved Drastically And Majority Scored Between 10 – 15, 24(62.19%).

Conclusion: Based On The Aims And Objectives Of The Study, It Can Be Concluded That Knowledge Scores Of The Clinical Assisstants Improved Post Training. Majority Of Them Had Fair Knowledge Post Training. Hence Periodic Training And Calibration Can Improve Their Awareness And Attitude Towards Interpreting The Hospital Warning Symbols.

Keywords: *Hospital Warning Symbols, Dental, Private Dental*

Introduction

Hospitals Are Those Institutions Which Have Existed Since Time Immemorial In One Form Or The Other And Have Become More Complex In The Present Time Frequented By People From Every Walk Of Life Without Any Distinction Between Sex, Age, Caste And Religion . Recently, There Is A Significant Increase In The Dental And Medical Teaching Hospitals And Correspondingly There Has Been Tremendous Increase In The Amount Of Biomedical Waste Generated By The Hospitals.

Waste Generated In A Teaching Hospital Is Similar To That Generated By Other Hospitals Which Includes A Large Component Of General Waste And A Smaller Proportion Of Hazardous Waste. Dental Professionals And Clinical Assistants Who Work In The Clinic Are At A Greater Risk For Acquiring Cross-Infection While Treating Patients. This Is Evident From The Fact That Most Of The Human Pathogens Have Been Isolated From Oral Secretions. Hospitals Use Instruments And Materials That Are Directly Exposed To Blood And Saliva And Are Therefore Potential Sources Of Infection. Hence It Is Most Important To Acquire Knowledge For Clinical Assistants To Deal With These Biomedical Wastes Generated In The Hospitals [1].

A Study Of The Knowledge, Attitude, And Practice On Usage Of Personal Protective Equipment Was Carried Out And An Attempt Was Made To Create Awareness Among The Workers About Its Importance. Personal Protective Equipment Is Designed To Protect The Skin And The Mucous Membranes Of The Eyes, Nose, And Mouth Of Dental Healthcare Personnel From Exposure Toblood Or Other Potentially Infectious Material.Occupational Safety And Health Administration Mandates That These Ppe In Specified Circumstances Reduces The Risk Of Exposure To Blood Borne Pathogens.

Personal Protective Equipment (Ppe) Is Used To Create A Protective Barrier Between A Worker And Hazards In The Workplace. Ppe Includes Such Equipment As Chemical Resistant Gloves, Safety Shoes, Hard Hats, Safety Glasses, Respirators, And Clothing Such As Gowns And Aprons. Cleaners And Sweepers Play Important Roles For Keeping The Area Clean. Their Works Entail Removing Of Debris From Area Collecting Solid Waste, Disposing And Recycling Waste Material. Consequently, They Have Higher Chances To Be Exposed To Numerous Risk Factors; Therefore, Their Occupational Safety And Health Hazard Became Crucial. There Is Little Evidence About The Sweeping Practices, Perceptions And Knowledge On Their Occupational Safety And Health Hazards[2].

We All Encounter Symbols Day To Day In Life. Symbols And Pictorial Representation Convey Messages Easily And Are Retained In Pictographic Memory Than Text Explanations And They Do Not Have Any Langiage Barrier. Warning Symbols Can Be Found Anywhere, Hospitals, Factories, Health Centers, Or Other Public Places. These Symbols Come In Varied Colours And Backgrounds And Are Considered Essential For Safety At Work And Other Places.

Safety Sign Is An Information Or Instruction About Health And Safety At Work Place On A Sign Board, A Colour, An Illuminated Sign Or Acoustic Signal ,Verbal Or Hand Signal. .The Sign Board Is A Combination Of Shape ,Colour And Symbol Or Pictographs Made Visible By Adequate Lighting And Which May Have Supplementary Text. The Basic Principle For Understanding Safety Symbols Remains The Same Like Colour And Shape Of Outline Symbol But Some Of The Icons Have Changed [3].

Warning Signs Should Be Used To Make People Aware Of A Nearby Danger. For Example, A Flammable Liquid Store Or A Laboratory Where Radioactive Substances Are In Use Should Have An Appropriate Warning Sign Near The Entrance. Signs Warning Of A Particular Hazard Consist Of A Black Band In The Shape Of An Equilateral Triangle. The Background Within The Band Should Be Yellow With The Pictographic Representing The Type Of Hazard In Black Placed Centrally On The Sign [4].

Mandatory Signs Should Be Used To Indicate Actions That Must Be Carried Out In Order To Comply With Statutory Requirements. Symbols Are Indicated In White On A Blue-Coloured Circular As A Background.Mandatory Signs Specify An Instruction That Must Be Carried Out To Vehicles, Workers, Visitors To Sites And Any Other People That Might Need To Know The Instruction. These Can Be Used In Construction Sites, Building Sites ,Hospitals (Protective Eye Wear), And Mine Sites . Sign Wording Is Printed In Black To Give Clear Instructions. Often Mandatory Signs Can Be Used In Conjunction With Other Signs, Traffic Lights And Bollards As Visual Communication To Workers, Drivers, Pedestrians, Visitors And People Regarding Risks[5].

The Radiation Hazard Can Be Particularly Relevant For Young Patients And Especially Children, Whose High Biological Susceptibility And Long Life Expectancy Tend To Increase The Likelihood Of The Effects Of Not Only Cancer But Also Other Non-Cancerous Dis-Eases. In This Respect, Evidence Exists That Imaging Parameters For Paediatric Examinations Are Frequently Not Adjusted To The Smaller Sizes Of Children Compared With Adults, Resulting In An Unnecessarily High Radiation Exposure[6].

The Use Of Radiation In Medical Applications Continues To Increase Worldwide. According To The Latest The United Nations Scientific Committee On The Effects Of Atomic Radiation (Unscear) Reports About 4 Billion X-Ray Examinations Are Performed Annually In The World . Ionizing Radiations Is One Of The Most Common And Robust Diagnostic Tools For Many Diseases. Beside Their Advantages, As With Any Medical Interventions, There Are Some Potential Risks In The Application Of Ionizing Radiations .Ionizing Radiations Have Been Reported As Carcinogen[7]. The Risk Of Radiation-Induced Cancers Varies With The Type Of Radiation, Exposure Time, Exposed Tissue And Patient Age.

Therefore, It Is Necessary To Consider The Principles Of Occupational Radiation Protection For All Staffs Of Diagnostic Imaging Departments. As Low As Reasonably Achievable (Alara) And The 10-Day Rule Are Important Radiation Protection Principles Which Set To Avoid Unnecessary Radiation Exposure Of The Staff [8].

A Laboratory Is A Place Where Dangerous Events Can Occur, From High Voltage Stations To Biohazards To Corrosive Substances. Many People Are Unaware Of The Dangers Lurking In A Laboratory. To Alert About The Hazards Such As Biohazards And High Voltage Stations, There Are Hazard Signs Placed In Laboratories [9]. Day To Day We Will Commonly Find Hazard Signs In A Laboratory, Alerting Us To Various Hazards We Are Expected To Find. However, Many People May Not Be Able To Identify What These Hazard Symbols Mean. To Help People Get Aware Of The Various Meanings Associated With Each Sign, This Study Was Concentrated In Educating And Making The Clinical Assistants Who Work In Hospitals And Laboratory To Create Awareness Among Different Hospital Warning Symbols Used In Hospitals [10].

Materials and Method

Study Design Descriptive Study Design

Study Area Study Was Conducted Among Clinical Dental Assistants Of Saveetha Dental College.The College Has A Steady Patient Inflow Of Approximately 1000 Out Patients Per Day. Highly Trained Auxiliary Personnel Help In Clinical And Non Clinical Duties Of The Patient. It Is Highly Imperative That, These Auxiliary Persons Get Adequately Trained And Are Aware About The Bio Hazardous Environment They Work, Day In And Day Out.

Study Population A Total Of 40clinical Assisstants Aged Between 24-27 Years Were Included In The Study According To There Knowledge And The Perception Level The Various Hospitals Warning Symbols Were Selected. Pre-Training And Post-Training Evaluation Were Done.Clinical Assistants Who Were Not Able To Identify The Certain Symbols Were Given Effective Training In Such A Way That They Could Identify The Symbols During Post Training Evaluation.

Inclusion Criteria

All Clinical Assistants Employed Under Saveetha Dental College Are Included In The Study

Exclusion Criteria

***** Clinical Assistants Who Were Not Interested To Participate In The Study Were Excluded.

* Clinical Assistants Who Were Not Available During Scheduled Time Of Data Collection.

Ethical Clearance

Prior To The Start Of The Study,Ethical Clearance Was Obtained From The Scientific Review Board And Institution Ethics Committee Of Saveetha Dental College.

Scheduling

Data Collection Was Scheduled For A Period Of One Month From November To December 2016.

Survey Instrument

A Template Consisting Of 20 Various Hospital Warning Symbols Like Biohazard, Eye Protection,Flammable,Radiation,ProtectiveGloves,High Voltage Hot Surface,LowTemperature,Laser,Corrosive, Noise, Dangerous Chemical,Explosives,MedicalWaste,HarmfulGoods,FireAlaram,Toxic Gases, Slide Door Left ,Slide Door Right And Emergency Exit Was Designed.

Survey Proceedings

The Questionnaire Was Given Pre And Post Training To Evaluate The Difference In Knowledge Scores. The Training Sessions Were Given To The Particpants For A Period Of 3 Days, 20 Minutes Sessions Each. The Faculty Who Was Training The Clinical Assisstants Were Calibrated Before The Sessions.

Personal Training

Method Of Training Used Was Case Study Method (Off-The-Job Method).The Type Of Training Used In Our Current Study Was Induction Or Orientation Training.Induction Training Is A Systematic Training Plays An Important Role In A Part Of The Organisation Overall Planning Process And Is In Line With Its Goals.Induction Training Provided The

Clinical Assistants With A Better Knowledge And Perception And Response Rate In Indentifing The Warning Symbols Than Those Response Which Got During Per Training.Hence The Training Given Was Seemed To Be More Effective.

Statistical Analysis

Descriptive Statistics Was Done Using Frequency And Percentages And Inferential Statistics Done Using ChisquareTest To Test Whether The Difference In Pre And Post Treatment Scores Were Statistically Siginificant. Siginificance Level Was Set At 5%.

Results

All The Clinical Dental Assistants Working In Saveetha Dental College And Hospitals Participated In The Study And The Response Rate Was 100%.A Total Of 40 Female Assistants, With A Mean Age Of 22.2 Years(18.25 Years) Were Enrolled In The Study. The Pre And Post Training Scores Were Grouped As <10 (Poor), 10-15 (Fair) And 15 To 20 (Good) Respectively.

Majority Of The Pre Training Scores Belonged To The Poor Category, 23(57.5%). The Scores Improved Post Training. Majority Of Them Belonged To The Fair Category, 24(62.19%). Figure 1 Depicts The Comparison Of Knowledge Scores Pre And Post Training.

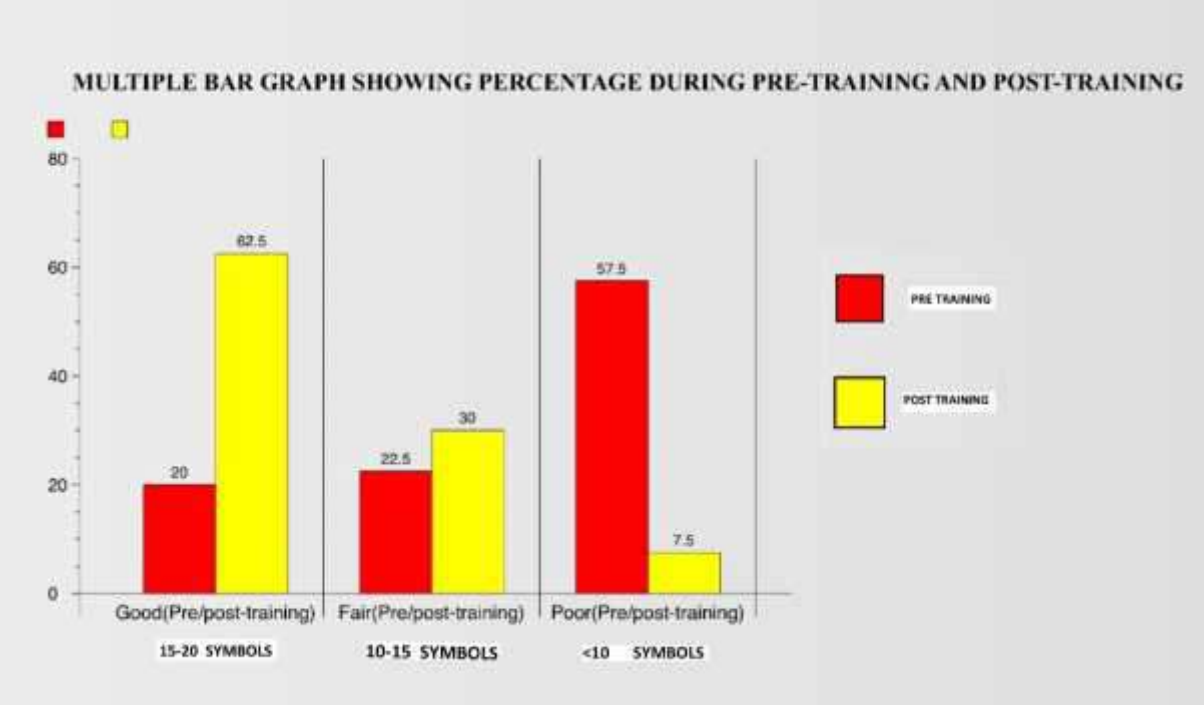


Figure 1: Pre And Post Training Knowledge Scores Comparison Among The Study Participants

Discussion

The Current Study Was Conducted On 40 Dental Clinical Assistants. The Response Rate Was 100% And There Was No Attrition In The Study. As Projected In The Results, The Knowledge Scores Drastically Improved After Training. A Large Proportion Of (62.5%) Of The Participants In This Study Demonstrated Good Knowledge Of Radiation Hazards Symbol And Mandatory Signs And Symbols Used In Hospitals. This Result Was Consistent With That Obtained In A Study Conducted In Three Hospitals In Port Harcourt,Nigeria,In Which(58.7%)Of Radiographers Were Aware Of Dangers Of Ionising Radiation And The Symbol Pertaining To It [11].

On The Contrary The Study Conducted By Iral D' Souze Et Al, The Respondent Had Average Awareness About Eye Protection (Ppe) (52.3%).This Result Was Compared To The Current Study Were The Study Participants Had Good Knowledge 82.8% Regarding Personal Protective Equipment Used In Hospitals And Laboratories [12].

According To TsegayeSewent Et Al, The Study Shows That Subjects Involved In The Study Has Poor Knowledge 32.7% Regarding Flammable Symbols And There Risk In Day To Day Life.This Result Was Compared To Our Study Were The Knowledge Level Was Increased To 73.7% [13].

The Study Conducted By Harvi Et Al., Showed That Majority Of The Participants (72.2%)Were Able To Analyse The Use And The Purpose Of Wearing Gloves (Ppe) In Hospitals,Laboratories And Health Care Centers. When This Results Were Compared To Our Current Study It Showed That Participants Enrolled In Our Study Had More Knowledge 92.5% Regarding Personal Protective Equipment Used In Hospitals [14].

According To J.A Hoops The Study Revealed That Subjects Encoded In The Study Had Poor Knowledge 42.6% Regarding Electrical Hazards Used In Hospitals And Electrical Board Centers .The Study Was Compared With J.A Hoops Et Al Showed Significant Increase In Awareness About High Voltage Symbols In Hospitals And Electricity Board Centers [16].

The Study Conducted By S.Mounika Et Al Says That Participants Involved In The Study Has Average Knowledge 50.9% Regarding The Hot Surface Warning Symbols Used In Laboratories,Hospitals And Workshops.The Results Were Compared To Our Current Study There Was No Significant Difference,Hence The Study Shows Awareness Rate About 47.2% [17].

According To T.L Fisher, Et Al., The Study Revealed That The Subjects Have Poor Knowledge Regarding The Chemical Hazard 37.5% .Hence When Is Was Compared To Our Study There Was Little Increase In The Knowledge Level Of The Participants [18].

According To Swapnill Et Al ,The Study Showed That The Workers Had Good Knowledge 78.3% Regarding Awareness Of Laser In Dentistry And Medical Field .The Results Were Compared To Our Current Study There Was No Significant Difference,Hence The Study Shows Awareness Rate About 75.3% Regarding The Laser Warning Symbols Used In Hospitals And Health Care Centers [19].

The Study Conducted By Robync,Et Al., Showed That Participants Had Poor Knowledge 37.8% Regarding Corrosive .Where Has In Our Current Study There Was Significant Increase In The Knowledge And The Awareness Level Of The Participants Enrolled,Which Was About 72.3% [20].

A Study Done On Radiation Protection By Svenson And Petersson Among Swedish Dental Practitioners Revealed Their Knowledge, Attitude, And Practices In The Field As Dentist Having Higher Level Of Knowledge which Was 84.2%Hence The Current Study Also Revealed That Subjects Have A Good Knowledge As Same As Svenson Et Al 80.5% .Hence There Was No Significant Difference Found Between Them [21].

In Another Study, Among Invasive Cardiologists Whereas Almost All The Participants (93%) Had The Good Knowledge Regarding Lead Apron, And Other Radiation Protective

Measures Including Thyroid Collar And Lead Eye Glasses; This Was Attributed To The Fact That While Lead Apron Was Available, The Other Devices Were Available To Less Than A Third Of Participants.When Results Were Compared With Our Study,The Subjects Had Average Knowledge In Radiation Protection [22].

The Overall Results Of A Study Conducted On Clinical Assistants Indicated That These Had Good Awareness And Perception Level (62.19%) About Warning Symbols Used In Hospitals As Compared To Another Study In Which Most Of The Students Were Not Having Adequate Knowledge Regarding Hospital Warning Symbols . Reports Of A Study Conducted On Dental Staff [Dentists, Dental Auxiliaries And Attenders] Revealed That Staff Had Good Attitude Towards Biohazard When Compared To The Findings Of Another Study In Which Many Dentists Had The Knowledge About Waste Management But They Lacked In Attitude And Practice . Also, There Were Poor Levels Of Knowledge And Awareness About Biohazard , Legislation And Management In Two Other Studies Conducted In Delhi And Rajasthan .

During The Pre Training ,The Most Easily Identifiable Symbols Among 20 Were Flammable, Corrosion , Eye Protection , Hand Protection And Radiation Hazards. The Non Ionising Radiation Was Mistakenly Identified As Wifi Symbol. Laser Symbol Was Mistaken For Sun, Also Low Temperature Was Mistaken For Snow Flake. But All These Changed In The Post Training Session. In This Present Study Majority Of The Pre Training Scores Belonged To The Poor Category, 23(57.5%). The Scores Improved Post Training. Majority Of Them Belonged To The Fair Category, 24(62.19%).Despite The Appreciable Results Obtained Post Training, If Theory Needs To Be Put In To Practice, Continuous Training And Calibration Of These Health Care Workers Needs To Be Done.

Conclusion

Based On The Aims And Objectives Of The Study It Can Be Concluded That Mandatory Training And Periodic Calibration Is Required For All The Health Care Workers Who Are At Potential Risk Of Getting Exposed To Bio Hazardous Materials.

Corresponding Author

D.Sri Sakthi

Reader,Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India
Mobile - +91 – 9962787531
Email: Drsrisakthiphd@Gmail.Com

Reference

1.Charles M. Sources And Effects Of Ionising Radiation. J RadiolProt. 2001;21:83–85.
2.National Council On Radiation Protection And Measurement. Ionizing Radiation Exposure Of The Population Of The United States: Recommendation Of The National Council On Radiation Protection And Measurements. Report No 160.Ncrp. Bethesda Md2009.
3.Shiralkar S, Rennie A, Snow M, GallandRb, Lewis Mh, Gower-Thomas K. Doctors' Knowledge Of Radiation Exposure: Questionnaire Study. Bmj. 2003;327:371–72.
4.Lee Ci, Haims Ah, Monico Ep, Brink Ja, Forman Hp. Diagnostic Ct Scans, Assessment Of Patients, Physicians And Radiologist Awareness Of Radiation Dose And Possible Risks. Radiology. 2004;231:393–98.
5.International Commission On Radiological Protection (Icrp). Recommendations Of The Icrp: Icrp Publication 103. Annals Of The Icrp. 2007;37(2-4)
6)Keiijeers Gb, Britton Cj. Doctors' Knowledge Of Patient Radiation Exposure From Diagnostic Imaging Requested In The Emergency Department. Med J Aust.2010;193(8):450–53.
7.Briggs-Kamara Ma, Okoye Pc, Omubo-Pepple Vb. Radiation Safety Awareness Among Patients And Radiographers In Three Hospitals In Port Harcourt. Am J Sciind Res. 2013;4(1):83–88.
8.FamurewaOc, AyoolaOo, OgunsemoyinAo, OnayadeAa. Radiation Protection And Dose Awareness Among Doctors In A Nigerian Teaching Hospital: A Preliminary Study. West Afr J Radiol. 2013;20:37–40.
9.Booshehri M, Ezoddini-Arkakani F, Nozari H. Evaluation Of Dentists' Awareness About Personnel And Patients National Protection In Yadz Dental Office. Health. 2012;4:490–92.
10.Ramanathan S, Ryan J. Radiation Awareness Among Radiology Residents, Technologists, Fellows And Staff: Insights Imaging. 2015;6(1):133–39.
11.Brown N, Jones L. Knowledge Of Medical Imaging Radiation Dose And Risk Among Doctors. J Med Imaging RadiatOncol. 2013;57(1):8–14.

12.Kapoor D, Nirola A, Kapoor V, Gambhir Rs. Knowledge And Awareness Regarding Biomedical Waste Management In Dental Teaching Institutions In India-A Systematic Review. Journal Of Clinical And Experimental Dentistry. 2014 Oct;6(4):E419.13) D'souza Ai, D'cunha S, Suresh S. A Study On The Awareness On Occupational Health Safety Among Health Care Professionals In Radiology Department In A Selected Hospital. Rguhs Journal Of Medical Sciences. 2013 Apr 1;3(2):85-90.

14. Asewunet T, Kebede W, Wondafrash B, Workalemau B, Abebe G. Survey Of Safety Practices Among Hospital Laboratories In Oromia Regional State, Ethiopia. Ethiopian Journal Of Health Sciences. 2014;24(4):307-10

15.Dr.Brundha, R. Harshini .Awareness About Personal Protective Equipments In Hospital Workers (Sweepers And Cleaners) In Saveetha Dental College And Hospitals– Int. J. Pharm. Sci. Rev. Res., 40(1), September – October 2016; Article No. 07, Pages: 28-29

16.HoppsJa. The Electric Shock Hazard In Hospitals. Canadian Medical Association Journal. 1968 May 25;98(21):1002.

17. S.Mounika.,. Analysis Of Knowledge About The Hospital Warning Symbols Among The Undergraduate Dental Students From Saveetha Institute Of Medical And Technical Sciences - A Comparative Study.J/ Pharm. Sci. And Res. Vol. 8(8), 2016, 856-858

18. National Research Council. Guide For The Care And Use Of Laboratory Animals. National Academies Press; 2010 Dec 27.

19. KadamSr, NaviwalaGa, Panchal Am, NishtalaSs, PardeshiVr. Dental Laser Education And Knowledge Among Students From Dental Colleges Of Mumbai And Pune City: A Questionnaire Study. Journal Of Indian Association Of Public Health Dentistry. 2017 Oct 1;15(4):368.

20.GildenRc. Pesticides Use In Hospitals: Health Protection, Health Hazards, And Viable Alternatives. Nursing Administration Quarterly. 2010 Oct 1;34(4):320-6.

21.Sewunet T, Kebede W, Wondafrash B, Workalemau B, Abebe G. Survey Of Safety Practices Among Hospital Laboratories In Oromia Regional State, Ethiopia. Ethiopian Journal Of Health Sciences. 2014;24(4):307-10.

22. Ogunnowo Be, Anunobi Cc, Onajole At, Odeyemi Ka. Awareness Of Occupational Health Hazards And The Practice Of Universal Safety Precautions Among Mortuary Workers In South West Nigeria. Nigerian Quarterly Journal Of Hospital Medicine. 2010;20(4):192-6.

Assessment about Reasons for Extraction of Primary Tooth among Dental Practitioners- A Survey

Savitha P[1], Mahesh Ramakrishnan [2], Pradeep.S [3]

Graduate Student [1],Reader [2],Department of Pedodontics

Senior Lecturer[3], Department of Conservative Dentistry and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: Extraction of primary tooth in a populations provide information about the prevalence of dental care, the availability of dental care, and attitudes towards extraction. To investigate the reasons for primary teeth extraction and the tooth type most frequently extracted in population.

Materials and Method:A questionnaire with 10 structured questions were prepared and distributed among 100 dentist practitioners to obtain information. The question were designed to determine the level of knowledge about the reasons and tooth type being extracted.

Result:the most common reason for extraction of teeth is due to dental caries among other reasons and the common type of teeth extracted due to dental caries is molars. And also observed that 13% of cases primary tooth extraction is due to over retention and tooth type is canine, 11% due to mobility which is its time for exfoliation, and 9% of the cases the extraction of primary tooth is due to trauma. In some cases extraction is done for orthodontic purpose which accounts for 5% of cases, 55% of cases extraction is due to dental caries.

Conclusion: Since tooth extraction can cause difficultly to pediatric patients, the result is that to identity the various reasons of extraction of teeth and eliminate the cause prior to it and thus minimising tooth loss and it's expected adverse consequences.

Key Words*: Dental caries, Orthodontic purpose, Eliminate, Tooth loss, Retention*

Introduction

Oral health is an important component of both overall health and quality of life [1].Human beings have two successive sets of teeth primary and permanent, therefore they are better known as diphodonts [2]. Primary teeth are considered equally important as the permanent teeth. Primary tooth helps in chewing of food, speech, and esthetics and also acts as template for permanent teeth to assume proper position in dental arch [3]. Extraction of primary toothin a populations provide information about the prevalence of dental care, the availability of dental care, and attitudes towards extraction [4]. Preservation of both primary and permanent teeth in children is of great importance; this is because teeth are important for stimulating the development of the dental arches, maintaining normal occlusal relationship, and playing a role in speech development [5].Extraction of primary or permanent teeth is performed for several reasons, including caries, periodontal disease, orthodontic treatment, traumatic injures, treatment failure, tooth impaction or eruption. Caries is the most common reason for extractions in a majority of the younger age groups. Early loss of primary dentition can result in delayed eruption, drifting, tilting or malpositioning of permanent teeth, thus causing malocclusion. It may affect esthetics, mastication and speech. Loss of several teeth in children can results in psychological disturbances [6].

Extraction of deciduous canine or molars may leads to mesial drift of teeth behind the space and distal drift of anterior teeth, with resultant displacement of permanent teeth and centerline disturbance [7].Although the presence of dental caries in children has reduced considerably in recent years, caries continues to affect the children in the population. Other causes may include trauma, malposition, congenital disorders, and arch length deficiencies causing resorption of primary teeth [8]. The loss of primary teeth also predisposes crowding, rotation and impaction in the permanent teeth [9]. Three factors are important,

- Degree of crowding is directly related to rate and extent of space loss after deciduous tooth extraction.
- Type of tooth loss. Loss of one primary canine may cause centerline shift. Loss of a deciduous molar, especially the second may allow mesial drift of the first permanent molar.
- Age of the child. The earlier a tooth is lost, the greater the tendency for drifting of tooth [10].

Traumatic injuries can be considered as one of the common causes for a tooth to be extracted since traumatic injuries usually occur among young children immediately after tooth eruption [11]. It is important to understand the factors which cause such loss and relative contributions of caries, periodontal diseases, trauma and orthodontic consideration to develop the strategies for future the reduction in loss of deciduous tooth [12]. The retained deciduous teeth are teeth that are did not shed at the normal shedding time and showed insufficient root resorption for normal shedding six months after the loss of the corresponding tooth in the same mouth and usually associated with double primary teeth, hypodontia affecting permanent successors, and subsequent to trauma or sever infection of primary teeth; so extraction of these teeth is indicated to allow proper space for permanent tooth to erupt if it is present [13]. A better knowledge of the reasons for extractions can be a help to optimize dental care and treatment planning and thereby avoid painful events and the possible development of dental anxiety for children.

The purpose of this study was to investigate knowledge, attitude about the principles reasons for primary tooth extractions and tooth type most frequently extracted.

Materials and Method

This study was conducted by assessing the responses to 10 pre-tested multiple choice questions regarding the reasons for extraction among dental practitioners. The questionnaire was used to conduct the survey among dental practitioners, a total of one hundred and fifty dental practitioners were recruited in the study. The questionnaire based study was conducted among dental practitioners to obtain information about their knowledge, attitude about the reasons for extraction of primary tooth and the tooth type that being extracted during their dental practice.

The questionnaire comprised of 10 structured question used to collect the data concerning about their knowledge about the causes for extraction of primary tooth.

1. What's the ideal function of Primary teeth?
    a. Mastication
    b. Aesthetics
    c. normal development of occlusion in the permanent dentition
    d. All of the above.
2. Does paediatric patient seek dental clinic, most commonly for extraction?
    a. Yes. b. No
3. If yes, what is the most common factor that influence the extraction of primary tooth?
    a. Dental caries. b. Trauma. c. Mobility d. Over retention
    e. Orthodontic purpose. f. Other causes.
4. What do you think of other causes for extraction of primary tooth?
    a. Periodontal diseases
    b. Parents request.
    c. Some medical condition.
    d. Failure of pulp therapy
    e. All of the above
5. What is the most common type of tooth being extracted for dental caries?
    a. Incisor.
    b. Canine.
    c. Molar
6. What is the most common type of tooth being extracted due to trauma?
    a. Maxillary Incisors.
    b. Mandibular incisors
7. What is the most common type of tooth being extracted due to mobility?
    a. Maxillary Incisor.
    b. Mandibular incisors
8. What is the most common type of tooth being extracted due to prolonged retention of primary tooth?
    a. Incisors
    b. Canine
    c. Molars.

9. Do you think economic problem are also one of reasons for extraction of primary tooth?

   a. Yes. b. No

10. What is the most common arch that being extracted?

   A. Maxilla. b. Mandible

Results

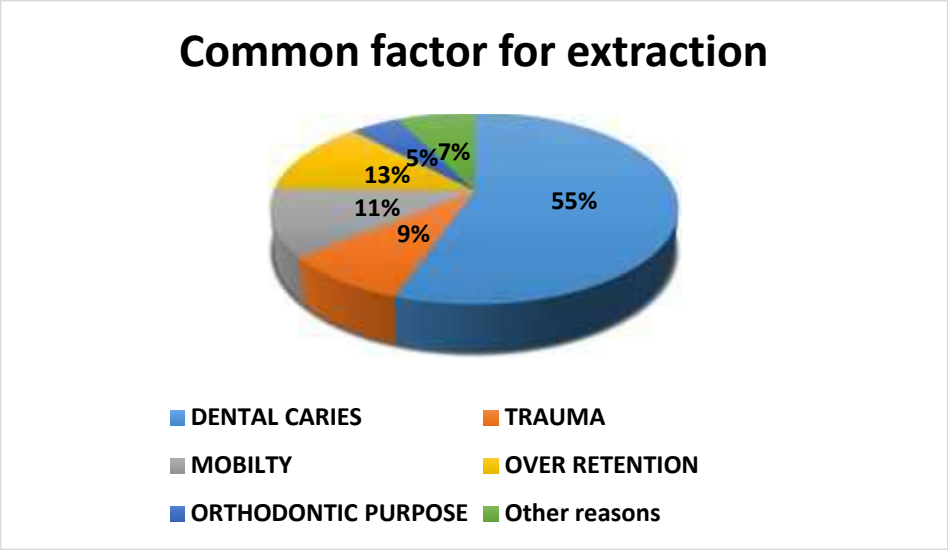


Figure 1: Common Reasons for Extraction of Primary Tooth

Figure 1: Illustrates about the extraction of deciduous tooth due to many reasons but possible reasons are due to dental caries [55%], over retention of primary tooth [13%], mobility due to exfoliation [11%], trauma [9%], other reasons [7%], for orthodontic purpose [5%]. Dental caries is the most common reason due to poor oral hygiene practices and habits.

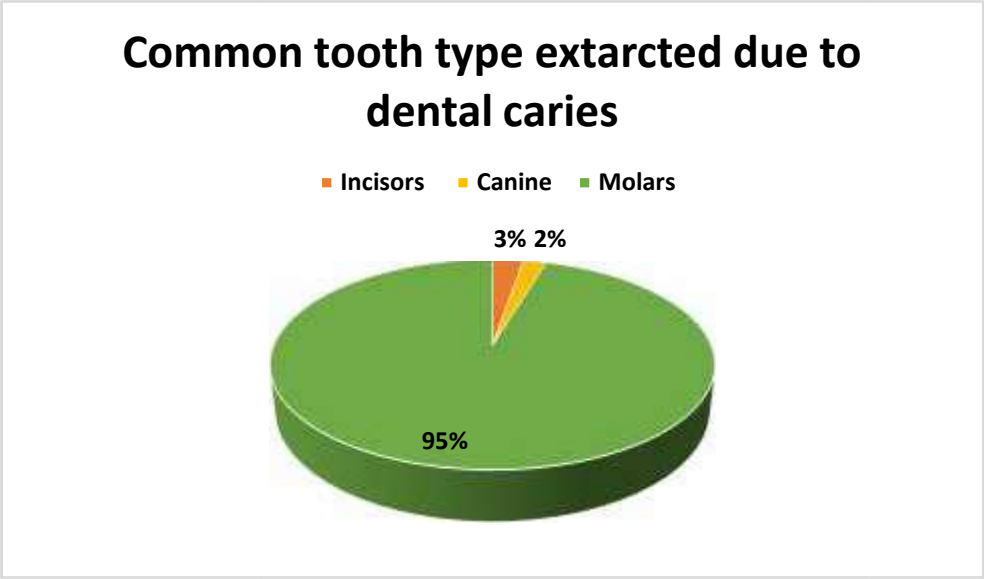


Figure 2: Common tooth type extracted due to dental caries.

Figure 2: Illustrates the most common tooth types extracted due to dental caries were molars [95%], incisors [3%], and canine [2%].The teeth at highest risk for carious lesions are the permanent first and second molars due to length of time in oral cavity and presence of complex surface anatomy.

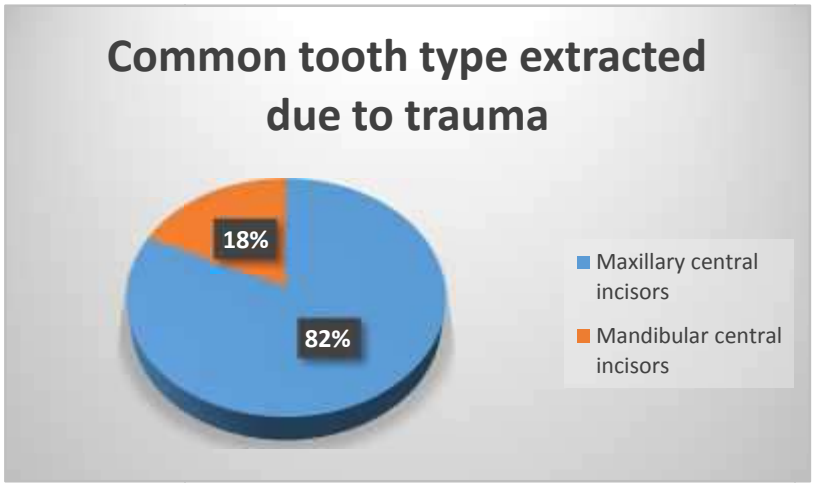


Figure 3: Common tooth type extracted due to trauma

Figure 3:Illustrates the most common tooth type extracted due to trauma maxillary incisors [82%] and mandibular incisors [18%]. Traumatic injuries can be considered as one of the common causes for a tooth to be extracted since traumatic injuries usually occur among young children immediately after tooth eruption.

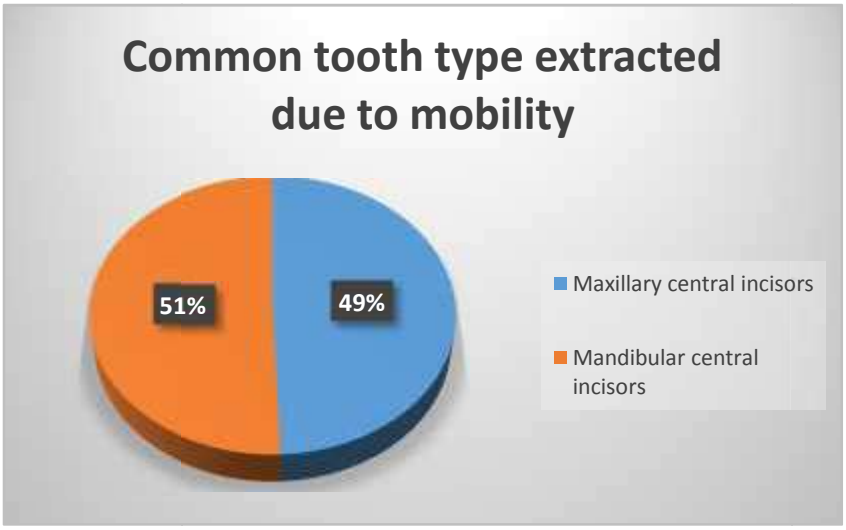


Figure 4: Common tooth type extracted due to mobility

Figure 4: Illustrates about the most common tooth type extracted due to mobility were maxillary incisors [49%] and mandibular incisors [51%]. Tooth extracted because of its mobility; time for exfoliation, thus the primary teeth will ultimately be replaced again with permanent teeth.

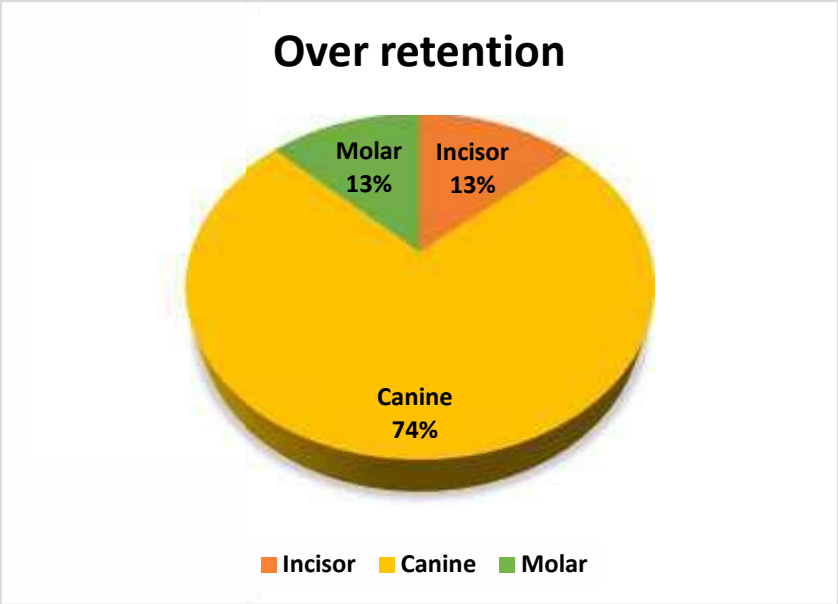


Figure 5: Illustrates about common over retained deciduous tooth.

Figure 5: Illustrates about the most common over retained primary tooth were canine [74%], incisors [13%], and molars [13%].Prolonged retention of primary teeth. Primary teeth may be retained for a variety of reasons, the most common being developmental absence of the permanent successor. Even when the permanent tooth is present it may fail to erupt leaving the deciduous tooth in situ. This can be a result of crowding, ankylosis of the primary tooth or the presence of supernumerary or other problems.

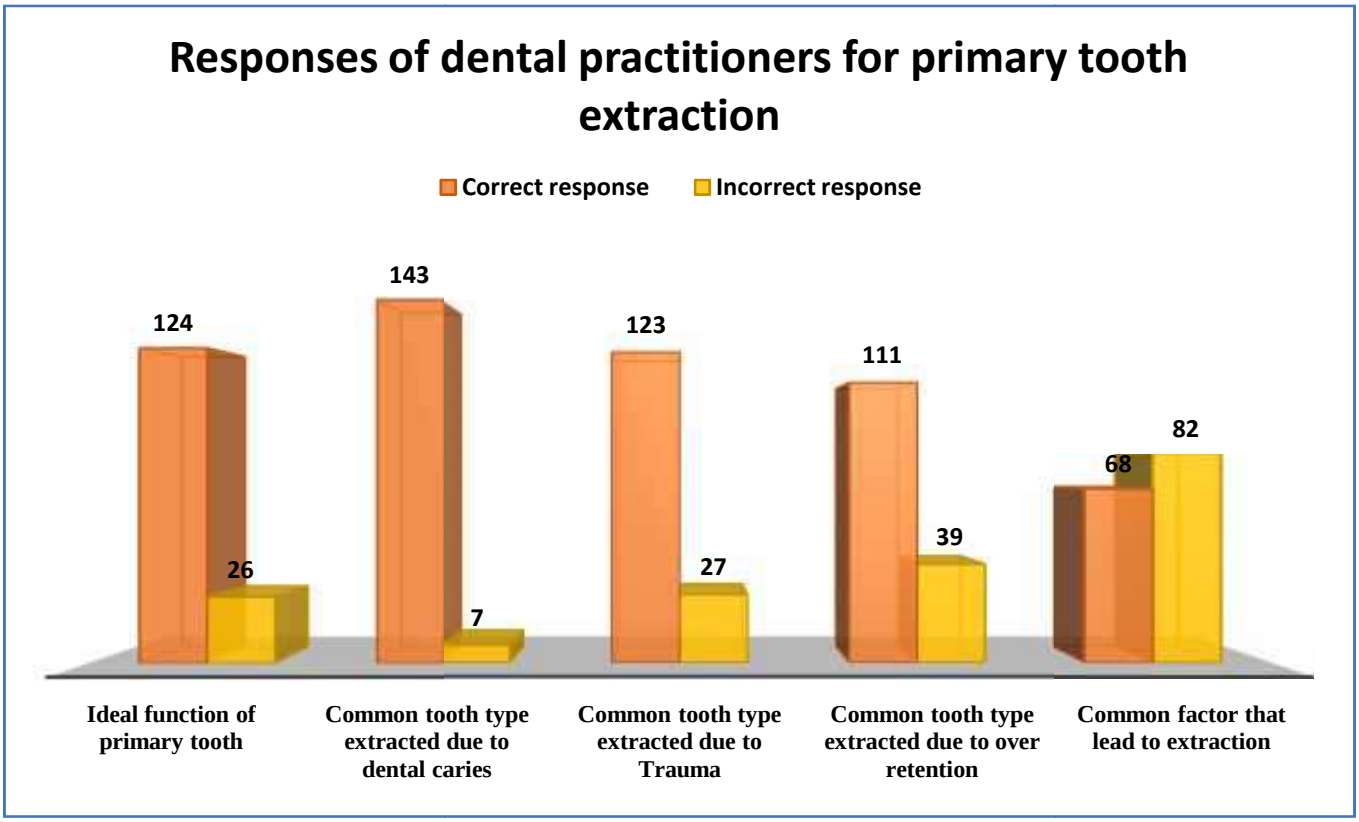


Figure 6: Illustrates about responses of dental practitioners for primary tooth extraction

On reviewing 150 questionnaire which is filled by the dental practitioners, it is observed that the most common reason for extraction of teeth is due to dental caries among other reasons and the common type of teeth extracted due to dental caries is molars. And also observed that 13% of cases primary tooth extraction is due to over retention and tooth type is canine, 11% due to mobility which is its time for exfoliation, and 9% of the cases the extraction of primary tooth is due to trauma. In some cases extraction is done for orthodontic purpose which accounts for 5% of cases, 55% of cases extraction is due to dental caries.

Discussion

Extraction of tooth is commonly a treatment consideration in the orthodontic management since the primary aims of orthodontic treatment in the mixed dentition are to correct dental arch

irregularities, occlusal and jaw relation abnormalities and to eliminate functional interferences; these may be classified as preventive which means the elimination of factors that may lead to malocclusion or interceptive that implies to the corrective measures which may be necessary to prevent a potential irregularity from progressing into a more sever malocclusion [14]. Tooth loss is considered as a crucial indicator of oral health status of the individual and the community at large [15,16]. Studies have shown that tooth loss strongly mirrors quality-of-life and nutrition intake of those affected and signal the onset of serious morbidity and disability [17,18]. Extraction of primary tooth in a population can provide information regarding the availability of dental care, the prevalence of dental disease, and attitudes toward tooth loss. There were many differences in the reasons for the extraction of different tooth types. Since very few extractions were due to periodontal disease, patient requests, or general medical reasons, these categories were come under other reasons. A better knowledge of the reasons for extraction can help to optimize dental care and treatment planning.

In our study, the most common factor for extraction are dental caries [55%], over retention of primary tooth [13%], mobility due to exfoliation [11%], trauma [9%], other reasons [7%], for orthodontic purpose [5%]. The most common tooth types extracted due to dental caries were molars [95%], incisors [3%], and canine [2%]. The tooth types extracted due to trauma weremaxillary incisors [82%] and mandibular incisors [18%]. The tooth type extracted due mobility were maxillary incisors [49%] and mandibular incisors [51%]. The over retained primary tooth were canine [74%], incisors [13%], and molars [13%]. For orthodontic purpose, the most common tooth types extracted were primary canine.

The factors which influencing the primary tooth extractions were many, which includes economic reasons which is due to the tooth could have been saved but the patient found the treatment is too expensive and also due to patient/parent request and periodontal diseases due to loss of function, periodontal abscess, pain and general medical reasons that is prophylactic extractions [19].

Several studies shown the data that the type of food consumed, feeding practices, snacking habits and pattern of dental visits have an important role in increasing caries level. The delayed visits to the dental clinic for routine checkup, and if the attendance is only promoted by symptoms; carious teeth are often treated by radical treatment which is extraction. Also, the lack of

awareness of the importance of daily brushing plays an important factor in increasing the prevalence of dental caries [21].

The reasons for primary teeth extraction varied between the different age groups.Mohitbansal et al., 2017 reported that primary maxillary central incisor was found to be the most affected tooth due to trauma [20]. It is similar to our study, maxillary central incisors [82%] were most common tooth type affected due to trauma than mandibular central incisors [18%]. Kuthyet al., 1994 reported that orthodontic considerations are more frequently the reason for extraction of deciduous canines, which is similar to our study. [21].

In our study it was found that primary maxillary and mandibular first molars were more affected due to caries, similar to Mohitbansal et al., 2017. Premature loss of a deciduous molar tooth due to caries also results in not only loss of function, but also can lead to increased possibility that the other teeth may drift [22]. This may influences the occlusion normal development and creates an increased need for orthodontic treatment [16].

People living in developing countries, and concomitantly, of lower socio- economic status have a lacunae in oral health awareness mirrored in their practice of oral hygiene habits [23]. Dental care has been systematically organised to improve dental health attitudes. This development has improved dental health and changed the dental caries patterns affecting them [24]. The high percentage of deciduous teeth loss could be due to the lack of knowledge among parents or a belief that the primary teeth will ultimately be replaced again. Prevention of caries in primary teeth must begin early in life to identify those at risk.Preservation of primary teeth is one of the major concerns for dentists. These teeth serve as a jewel which increases the smile and hence the self-esteem of the children.This shows the presence of poor maintenance of oral cavity in children.

Conclusion

Oral diseases have been a persistent public health problem globally, with almost every individual experiencing oral health problems at least once in their lifetime .Dental caries is observed to be the most frequent reason for extraction of teeth, this shows that there is poor oral hygiene maintained by children. Children usually face difficulty while extracting the tooth and also there

are post operative complication such as drifting off adjacent teeth, rotation and other causes. In order to prevent complications, it is applicable to create awareness and prevent tooth loss. It's important to understand how tooth loss occurs and take steps to prevent it through education, early diagnosis and regular dental care. The awareness should be created about their oral hygiene practices and habits. Parental oral health care education is essential to establish good oral health maintenance. Knowledge about oral health care among parents play an important role in maintaining oral hygiene and reducing the level of decayed tooth among children.

Corresponding Author

S. Pradeep,

Senior Lecturer,Department of Conservative Dentistry and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

Telephone Number: +91 9710404482

E-mail ID: pandu.pradeep@gmail.com.

Reference

1. Javed Ashraf, Ashish R Jain, Padma Ariga, Deepak Nallaswamy. Prevalence of Partial Edentulousness and Treatment needs in Rural Population of South India. World Journal of Dentistry, 2017;8:213-217.
2. Hulland SA, Lucas JO, Wake MA. Eruption of the primary dentition in human infants: A prospective descriptive study. Pediatr Dent. 2000;22:415-421.
3. Mouadian WE, Wehr EJD, Crall JJ. Disparities in children’s oral health and access to dental care. JAMA. 2000;284:2625-2631.
4. Whittle J G, Whittle KW. Five-year-old children: change in their decay experience and dental health behaviors over years. Comm Dent Health 1995;12:204-207.

5. Chen M. Oral health status and it's inequality among education groups. J Int health Serv.2002; 32:139-161
6. Klingberg, G., Berggren, U. and Noren, J.G: Dental fear in an urban Swedish children population: prevalence, concomitant factors. Community Dent Health 1994;11:208-214.
7. RavnJJ . Occlusion in the primary dentition in three year old children. Journal of the American Dental Association 1975; 89: 599-606.
8. Owen DG. The incidence and nature of space closure following the premature extraction of deciduous teeth: A literature review. Am J Or-thod. 1971;59:37-49
9. Pedersen J, Stensgaard K, MelsenB. Prevalence of malocclusion in relation to premature loss of primary teeth. Community Dent Oral Epidemiol. 1978;6:204-9.8.
10. Foster TD [1980] A textbook of orthodontics, 3rd edn. Blackwell, Oxford, p138
11. Kargul B, Tanbogal I. Dental trauma in Turkish children, Istanbul. J Dent Traumatol. 2003; 19: 72–77.
12. QuteishTaani DS. Reasons of tooth extraction in adult population of Jordan. J Oral Rehabil 2003;30: 110-112.
13. Bjerklin K, Bennett J**.** The long term survival of lower second primary molars in subjects with agenesis of the premo- lars. J Eur. of Orthod. 2000; 22: 245– 255.
14. Chestnutt IG, Binnie VI, Taylor MM.Reasons for tooth extraction in Scotland. J Dent. 2000; 28: 295–297
15. Ashiwaju MO, Folayan MO, Sote EO, Isikwe MC. Pattern of tooth extraction in children attending tertiary health care centers in Nigeria: A prospective study. J Clin Pediatr Dent 2011;36:107-110.
16. Petersen PE, Bourgeois D, Ogawa H, Estupinan-Day S, Ndiaye C. The global burden of oral diseases and risks to oral health. Bull World Health Organ 2005;83:661-269
17. Al-Bluwi GS. Epidemiology of dental caries in children in the United Arab Emirates. Int Dent J 2014;64:219-228.
18. Brennan DS, Spencer AJ, Roberts-Thomson KF. Tooth loss, chewing ability and quality of life. Qual Life Res 2008;17:227-235.
19. Kay EJ, Blinkhorn AS. The reasons underlying the extraction of teeth in Scotland. Br Dent J. 1986;160:287-290.

20. Mohitbansal, nidhigupta, preetygupta, vikramarora, sahilthakar. Reasons for extraction in primary teeth among 5-12 years school children in haryana, India. J clin Exp Dent. 2017 Apr; 9[4]: e545-e549.
21. Kuthy RA, Antkowiak MF, Clive JM. Extractions prior to comprehensive orthodontic treatment in the mixed dentition. Pediatr Dent. 1994; 16[3]: 211 – 16.
22. Lin YT, Lin WH, Lin YTJ. Immediate and six-month space changes after premature loss of a primary maxillary first molar. J Am Dent Assoc. 2007;138;362-8.
23. Mohammed Ahad, Gheena. Awareness of Tooth Brushing Techniques and Proper Oral Hygiene among School Children. J. Pharm.Sci &Res. Vol. 7[6], 2015, 367-372.
24. Thanish Ahamed. S. Awarness of oral hygiene among children in chennai. Research J. Pharm. and Tech 2016; 9[8]:1055-1058.

Assessment about the Oral Health Awareness among Undergraduate Medical Students.

Savitha P[1], Nithya.J [2], Pradeep.S [3]

Graduate Student [1],Senior Lecturer [2],Department of Oral Pathology

Senior Lecturer [3],Department of Conservative Dentistry and Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: people of all walks of life. General health is incomplete with the maintenance of oral health. Good oral health care practice is an effective and preventive measure for maintaining good general heath. Oral health is an essential for good health throughout life. Poor oral health and untreated oral conditions and diseases can have a significant impact on quality of life.

Aim: The aim of the study was to assess oral health awareness among undergraduate medical students.

Materials and Method: The study was conducted among 100 undergraduate medical students. The pre-tested questionnaire with 20 structured questions were prepared and distributed to medical students to obtain information. The questionnaire comprised of questions based on the attitude, awareness towards the oral health among undergraduate medical students.

Results: In the present study, the awareness about oral health was found to be good among medical students. This survey found that 48% of students brush their teeth twice a day and 49% of students had regular dental visit. Out of 100 student's majority of them were answered for main cause of tooth decay were due to high sugar content, reduction in saliva level, reduction in fluoride level and some of them were answered due to high sugar content only.

Conclusion: The present survey shows good oral health awareness among undergraduate medical students, and final year students were found to have better knowledge compared to first, second and third year undergraduate students.

Key Words:*Knowledge, Attitude, Oral health, Hygiene, Fluoride level.*

Introduction

Health is a universal basic right and need for people of all walks of life. General health is incomplete with the maintenance of oral health [1]. Good oral health care practice is an effective and preventive measure for maintaining good general heath. Oral health is an essential for good health throughout life. Poor oral health and untreated oral conditions and diseases can have a significant impact on quality of life [2]. The oral hygiene care procedures are major component in children's health educational programs and incorporate a complex of activities, targeted to reduce and limit dental plaque accumulation. Majority of the population approaches medical practitioners for their oral health problems at primary health centre level. Hence, general practitioners should have an appropriate knowledge about oral health and also should play an active role in oral health promotion [3].The most common oral diseases, dental caries and periodontal disease, are considered to be behavioral diseases because adoption of healthy oral habits is crucial in controlling them. To a great extent, their prevention and control depend on a person's lifestyle and behavior [4].

Oral health can affect the most basic human needs, including the ability to eat and drink, swallow, maintain good nutrition, smile, and communicate. [5]. Medical practitioners should play an active role in oral health promotion [6]. Good knowledge of oral diseases is crucial in medical practice due to the following reasons (a) Periodontal diseases are associated with multiple systemic conditions of medical interest (b) A large number of systemic diseases have oral manifestation. (c) Many drugs are associated with oral adverse drug reactions and (d) majority of the population approach medical practitioners for their oral health problems [7].Dental caries and periodontal diseases can be effectively prevented by plaque removal, which serves as a standard to evaluate oral hygiene. Poor oral health has been proven to have unfavorable effects on general health.Hence, early screening and proper referral by these professionals may benefit to improve the access to oral health problems and to reduce the associated morbidity and mortality. The need of the hour is for general practitioners to have adequate knowledge about oral health as they are the one to whom majority of the population approach[8].The practices that help in prevention of oral diseases include tooth brushing; tooth flossing; use of fluoridated toothpaste for brushing and mouth rinsing and lower consumption of sugary foods and beverages. Periodic dental visits are a practice that helps in early detection and

prevention of dental diseases. Several factors influence compliance with oral hygiene practices among the children. Poor oral hygiene occurring due to increasing plaque and calculus deposits with increasing age has been diagnosed. Personal and professional plaque removal and professional calculus removal have been extensively available for prevention of gum diseases. In order to achieve oral health benefits, toothbrush and other oral hygiene aids should be used daily in a correct manner. Dental plaque will usually adhere on the surface of teeth. Exploration of literature reveals that only few studies have assessed oral health awareness among undergraduate medical students. So, the present study was conducted with an aim to assess oral health awareness among undergraduate medical students.

Materials and Method

The pretested questionnaire was used to conduct the survey in medical college, a total of 100 students were recruited in the study. The questionnaire-based study was conducted among students of Saveetha medical college which is located in Chennai, India. The data collected concerned the oral hygiene awareness and attitude, dental complaints and previous visits to dentists. The target population comprised of 100 students, female and male was 53 and 47 respectively.

The questionnaire comprised of 20 pre-tested questions. The questionnaire comprised of questions based on the attitude, awareness towards the oral health among undergraduate medical students. Participants knowledge was gathered about tooth cleaning, brushing and dental problems were asked for practices of oral health. One of the factors that is responsible for someone attitude and behavior is knowledge. Knowledge can be described as level of understanding of an individual towards facts, information, skills and many more.

A self made ended questionnaire written in English language was given to each one of the undergraduate medical student. The questionnaire included information related to name, age, and sex. The questionnaire was further categorized to evaluate the awareness of oral hygiene among medical students.

1. Proper oral health care practice is an effective and preventive measure for maintaining good general health.

   A) Agree B) Disagree C) don't know

2. If agree, what are the diseases which are related to oral health

   A) Heart disease and stroke

   B) Respiratory disease

   C) Gastrointestinal problems

   D) All of the above

   E) None of the above

3. How often do you visit a dentist?

   A) Once a year

   B) Once in 6 months

   C) Infrequently

   D) More often

4. What is the reason for visiting a dentist?

   A) Tooth ache

   B) Regular checkup

   C) Control

5. How often do you brush your teeth?

   A) Once a day

   B) Twice a day

   C) More than twice a day

6. Do you think you know how to brush your teeth properly?

   A) Yes B) No C) Don't knows.

7. How long do you use the same tooth brush?

   A) Less than 3 months

   B) 3 – 6 months

   C) More than 6 months

8. What else do you use besides your tooth brush and paste?

   A) Mouth wash

B) Interdental brush

C) Dental floss

D) Tooth brush only

9. Use of fluoridated tooth can prevent tooth decay?

A) Agree

B) Disagree

C) don’t know

10) What is the main cause for tooth decay?

A) High sugar content food products

B) Reduction in saliva level

C) Reduction in fluoride level

D) All of the above

11) Which among the following brushing technique do you use while brushing?

A) Horizontal strokes

B) Vertical strokes

C) Circular strokes

D) Both vertical and horizontal strokes

12) In hospitalized patients, what is the common dental complaint do you come across?

A) Bleeding gums

B) Ulcers

C) Oral halitosis

D) Don’t know

13) What does bleeding gums means?

A) Gums are unhealthy

B) Ulcerated

C) Gums are infected

D) Don’t know

14) What are yellow or greenish yellow deposits near the gums?

A) Food deposits

B) Plaque

C) Calculus

D) Don’t know

15) What are the oral health’s problems occur to person with tobacco chewing habits?

A) Mouth ulcers

B) Oral cancers

C) All of the above

D) Don’t know

16) Cheek and lip biting may increase the chances for oral health?

A) Agree

B) Disagree

C) don’t know

17) Smoking or using tobacco products increase the chances for tooth loss?

A) Agree

B) Disagree

C) don’t know

18) Do you think oral cancer can affect general health?

A) Agree

B) Disagree

C) don’t know

19) What do you think for cause of oral cancer?

A) Usage of betel nut and betel quid

B) Usage of tobacco

C) Smoking

D) Don’t know

20) Do you think use of tongue cleaner would damage the taste buds?

A) Agree

B) Disagree

C) Don’t know

Result

In the present study, the awareness about oral health was found to be good among undergraduate medical students.

Proper oral health care practice is an effective and preventive measure for maintaining good general health, the results among undergraduate medical were 100% agreeable that oral health plays an essential element in health of an individual, poor oral health and conditions can have a significant impact on quality of life.

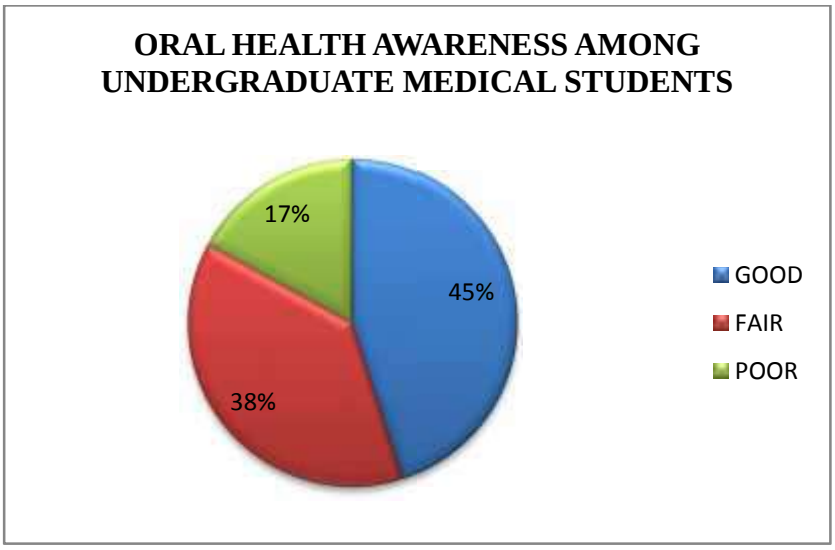


Figure 1: Awareness about oral health

Figure 1 shows that, only 45% were found to have good oral health awareness, 38% of students were found to have fair oral health awareness and 17% were found to have poor oral health awareness. Out of 100 student's final year medical students have good oral health awareness when comparing to third year students. And the third year students have fair oral health awareness comparing to first and second year students. The probable reasons could be more clinical exposure, more experience and knowledge of final year undergraduate students when compared to second and third year students.

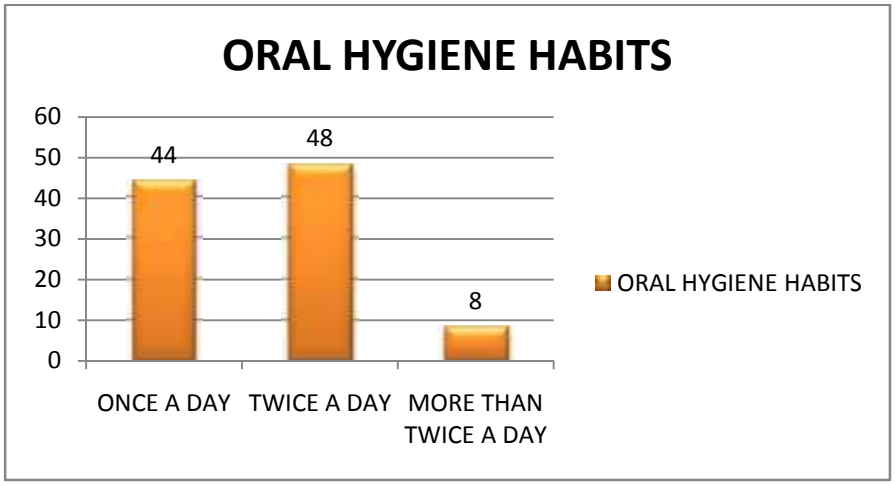


Figure 2: Oral hygiene habits

Figure 2 shows, 48 students have oral hygiene habits of brushing twice a day and 44 of them brush once a day and 8 of students brush more than twice a day due to some reasons. Brushing at least twice a day has shown a decrease in dental caries incidence [9].

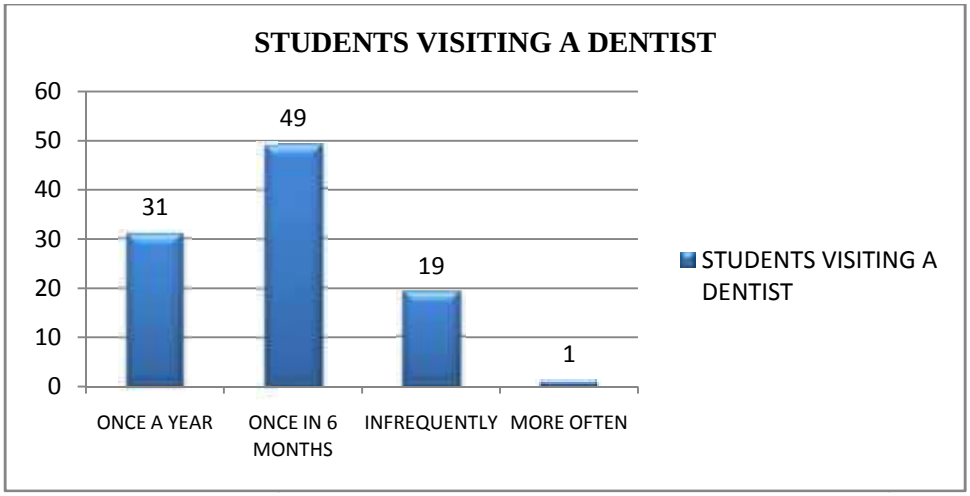


Figure 3: Regular dental visit

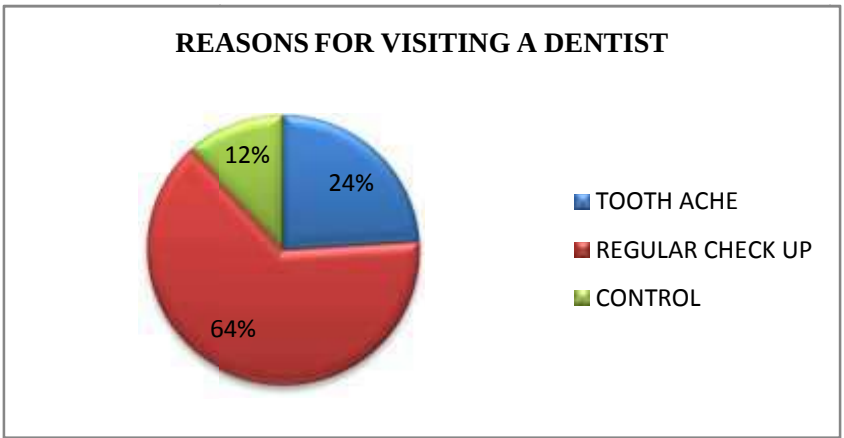


Figure 4: Reasons for dental visit

Figure 3 and 4 shows, 49 students were visiting dentist once in 6 months for their regular checkup and some of the due to tooth ache or some other reasons, 31 of them visiting once a year and 19 of them infrequently visiting a dentist. Medical students should be aware of the oral health problems and should understand the causative agents. In order avoid such oral health problems; they should have periodic visiting to dental hospitals. In here 64% of students visiting dental hospital for regular periodic checkup, 24% students due to dental caries and tooth ache and 12% visited dental hospital for control measures of oral diseases.

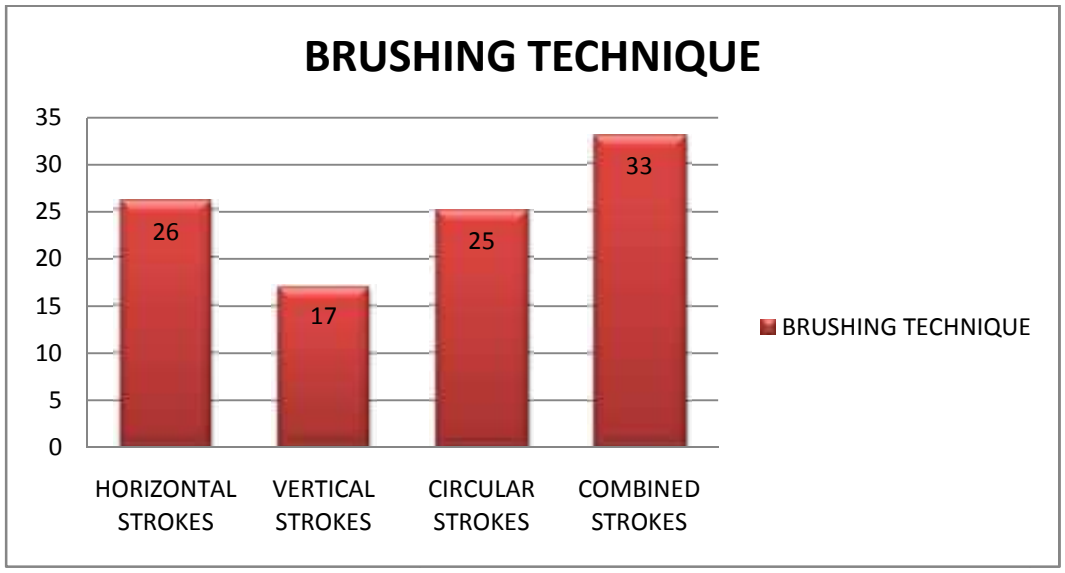


Figure 5: Brushing technique

Figure 5 shows, most of them were using combined strokes as brushing technique, and 26% of them were using horizontal strokes, 25% of them were using circular strokes and 17%of were using vertical strokes as their brushing technique.

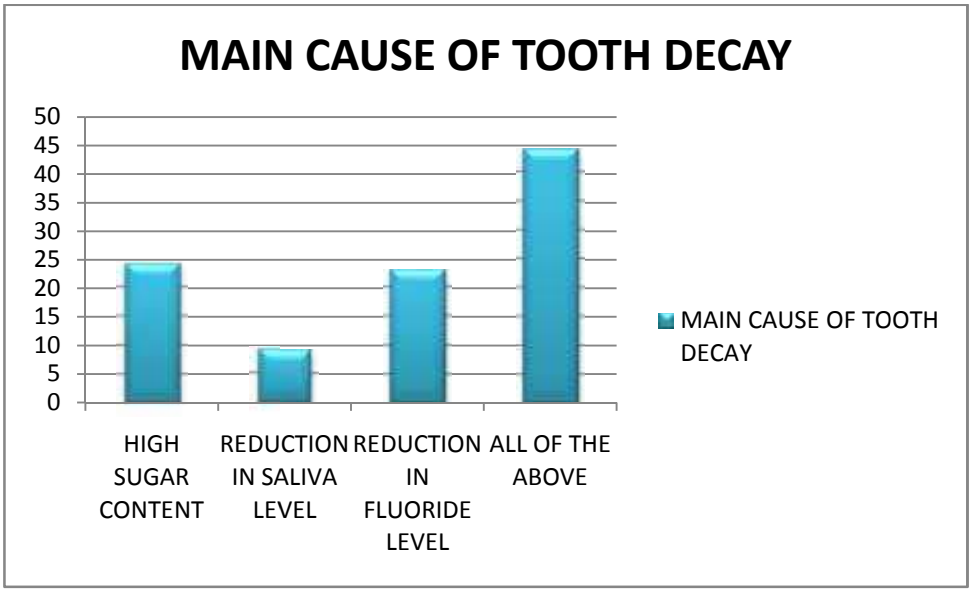


Figure 6: Main cause of tooth decay

Figure 6 illustrates that most of the medical students, 24% of them were answered as main cause of tooth decay due to consuming high sugar content products, 9% of them told reduction in saliva level, 23% were answered reduction in fluoride level and 44% of them mentioned all the above reasons were the reasons for the main cause for tooth decay.

Discussion

People living in developing countries, and concomitantly, of lower socio- economic status have lacunae in oral health awareness mirrored in their practice of oral hygiene habits[10]. Dental care has been systematically organised to improve dental health attitudes. This development has improved dental health and changed the dental caries patterns affecting them[11]. For preserving oral health and for preventing oral diseases oral self care such as tooth brushing and flossing are important [12]. Regular dental visit is an important aspect of maintaining good oral health [13].

Pattern of oral health behavior in medical graduates, their beliefs, and attitudes play an important role in knowledge they impart to the general public and their community [14]. Oral health is an essential component of general health and overall well-being of an individual. Oral cavity and its surrounding structures that are free of any diseases is indicative of good oral health. This not only makes a person look and feel good, it is equally relevant in maintaining oral

functions [15, 16]. Moreover, medical students are far more likely to encounter underserved and vulnerable populations than dental students [17].

In this study, 45% were found to have good oral health awareness, 38% of students were found to have fair oral health awareness and 17% were found to have poor oral health awareness. Out of 100 student's final year medical students have good oral health awareness when comparing to third year students. And the third year students have fair oral health awareness when comparing to second and first year students. The probable reasons could be more clinical exposure, more experience and knowledge of final year undergraduate students when compared to second and third year students.

In this questionnaire-based study, out of 100 student's majority of them were answered for main cause of tooth decay were due to high sugar content, reduction in saliva level, reduction in fluoride level and some of them were answered due to high sugar content only.

Many of the first-year undergraduate medical students were found to have average oral health awareness when compared to second and third year undergraduate medical students in which most of them had poor oral health awareness.

The reason could be medical students are exposed to dental subjects in their second academic year. First year undergraduate medical students were not included in the study as they do not have any clinical exposure and experience. It is recommended that oral health awareness of the undergraduate medical students can be improved by incorporating basic information about oral health in their academic curriculum, conducting various inter-disciplinary workshops and conferences, increasing the clinical exposure of the students to oral findings as most of the systemic diseases manifests in the oral cavity, special study modules or electives in oral health and disease should be created by involving the dental faculty, emphasizing the importance of oral health. Thus, oral health care needs to be addressed by the combined efforts of dental and medical professionals and should be integrated into comprehensive health-promoting strategies and practices [18,19]. This has been attributed to the positive self care attitudes for internal psychological reasons to improve their appearance and self-esteem[20].

As prospective doctors - they should have sufficient awareness about oral health as oral health problems are cumulative by nature, more amenable to prevention and difficult to treat.

Early identification of the oral diseases and referral to a specialist not only prevents a patient from pain, agony, functional and esthetic problems, but also from death in some conditions.

Conclusion:

The present survey shows good oral health awareness among undergraduate medical students, and final year students were found to have better knowledge compared to first, second and third year undergraduate students. Medical practitioners should play an active role in oral health promotion. Proper knowledge of oral diseases is crucial in medical practice. The knowledge they are going to acquire at present will be reflected in the future during their practice. Awareness makes maintenance of optimum oral health important in one's life. Medical students adapt the knowledge acquired at undergraduate level which influences their orientation of medical practice and patient care after their graduation. Implementation of oral health programs among medical students will not only improve their personal oral health care, but also potentially influences the motivation of patients to follow the preventive oral health measures. It also forms an essential basis for promoting the importance on primary oral health care.

Corresponding Author

S. Pradeep

Senior Lecturer,Department of Conservative Dentistry and Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India.

Telephone Number: +91 9710404482

Email: pandu.pradeep@gmail.com

Reference

1. Amina Mehrin Bano, G. Karthikeyan. The Knowledge and the Extent of Execution of Oral Hygiene in Undergraduate Dental Students. Research J. Pharm. and Tech. 9(6): June 2016.

2. Kishor KM. Public health implications of oral health-inequity in India. J Adv Dent Res 2010; 1:1-0.
3. Usman S, Bhat SS, Sargod SS. Oral health knowledge and behavior of clinical medical, dental and paramedical students in Mangalore. J Oral Health Commun Dent 2007; 1:46-8
4. Shristi nadar, SP Saravana Dinesh. A questionnaire study about oral hygiene awareness among orthodontic patient. International journal of orthodontic rehabilitation 2016; 7(3):97-100.
5. Doshi D, Baldava P, Anup N, Sequeira PS. A comparative evaluation of self-reported oral hygiene practices among medical and engineering university students with access to health-promotive dental care. J Contemp Dent Pract 2007; 8:68-75
6. Patel A. Awareness of oral health among medical practitioners in Sangamner City - A cross-sectional survey. Int J Clin Dent Sci 2010; 1:26-9.
7. Baseer MA, Alenazy MS, Alasqah M, Algabbani M, Mehkari A. Oral health knowledge, attitude and practices among health professionals in King Fahad Medical City, Riyadh. Dent Res J (Isfahan) 2012; 9:386-92.
8. Ramirez JH, Arce R, Contreras A. Why must physicians know about oral diseases? Teach Learn Med 2010; 22:148-55.
9. Nguyen L, Hakkien U, knuttila M, Jarvelin MR. Should we brush twice a day? Determinants of dental health among young adults in Finland. Health Econ 2008 Feb; 17(2):267-287.
10. Mohammed Ahad,Gheena. Awareness of Tooth Brushing Techniques and Proper Oral Hygiene among School Children. J. Pharm.Sci &Res. Vol. 7(6), 2015, 367-372.
11. Thanish Ahamed. S. Awareness of oral hygiene among children in Chennai.Research J. Pharm. and Tech 2016; 9(8):1055-1058.
12. Subhasri. A, Dr. T. N. Uma Maheshwari. Knowledge and Attitude of Oral Hygiene Practice among Dental Students. Research J. Pharm. and Tech 2016; 9(11): 1840-1842.
13. Ankur G shah. Evalution of oral hygiene awareness, oral health practices and dental health problems among the undergraduate medical students of India. International journal of preventive and clinical dental research, 2017;4(2):109-113.

14. Kombayashi T, Kwan SY, Hu DY, Kajiwara K, Sasahara H, Kawamura M. A comparative study of oral health attitudes and behavior using the Hiroshima University- dental behavioral inventory (HU-DBI) between dental students in Britain and China. J Oral Sci 2005 Mar; 47(1):1-7.
15. Abdollahi M, Radfar M. A review of drug-induced oral reactions. J Contemp Dent Pract 2003; 4:10-31
16. Puy CL. The role of saliva in maintaining oral health and as an aid to diagnosis. Med Oral Patol Oral Cir Bucal 2006;11: E449-55
17. Kaur S, Kaur B, Ahluwalia SS. Oral Health Knowledge, Attitude and Practices amongst Health Professionals in Ludhiana, India. Dentistry 2015; 5:315.
18. A-Rabiei S, Mohebbi SZ, Patja K, Virtanen JI. Physicians' knowledge of and adherence to improving oral health. BMC Public Health 2012; 12:855.
19. Baseer MA, Alenazy MS, AlAsqah M, AlGabbani M, Mehkari A. Oral health Knowledge, attitude and practices among health professionals in King Fahad Medical City, Riyadh. Dent Res J 2012; 9:386-92
20. Al Kawas S, Fakhruddin KS, Ur Rehman B. A comparative study of oral health attitudes and behaviour between dental and medical students; the impact of dental education in United Arab Emirates. J Int Dent Med Res 2009; 2:6-10.

Assessment of Impaction Pattern of Mandibular Third Molars:-A Radiographic Survey

S.Jayakeerthana[1],R.Navaneethan[2],Nabeel Nazar[3],
Graduate Student[1],Reader[2], Department of Orthodontics,Reader[3], Department Of Oral and Maxillofacial Surgery,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Third molars are known as wisdom teeth as they erupt after 18 years of age. In a lot of individuals they fail to erupt onto the oral cavity owing to a variety of reasons, the most common being lack of space in the dental arch.The aim of this study is to assess the pattern of Mandibular third molar impactions using the Orthopantomograph (OPG's)**.**

Material and Methods: OPG's of 150 patients (91 female and 59 male)who were between 18-25 years of age and had impacted mandibular third molars was assessed for gender, side of the mandible having greater frequency of impaction and the pattern of impaction of the third molars.

Results: Bilateral impaction (26.66%) was more common than and particular left (23.33%) or right (19.33%) unilateral impactions.The mesioangular pattern of impaction was more common in both male and female and was followed by distoangular, vertical and horizontal patterns.

Conclusion: The study showed that there was no gender predilection in the presence of impacted mandibular third molars and that the mesioangular pattern of impaction was more common.

Key Words: *Mandibular third molars, impacted teeth, Orthopantomograph, Mesioangular Impaction, Gender predilection.*

Introduction

Impaction is defined as a completely or partially unerupted tooth and positioned against another tooth, bone or soft tissue,so that its further eruption would be unlikely(1). Third molars are also known as wisdom teeth as they erupt in the late adolescence or even in adulthood. Since they are the last teeth to erupt they are frequently impacted due to the lack of space in the dental arch. Impacted wisdom teeth are teeth which do not fully erupt into the mouth because of blockage from other teeth (impaction).

Owing to the fact that at least one impacted third molar can be traced in 33% of the general population (2,3), impacted third molars especially the mandibular third molars, constitute a common cause of pain and inflammation in the oral region. The high prevalence of their impaction has been attributed to a remarkable variety of factors, among which are inadequate retromolar space, unfavorable path of eruption, malposition of the tooth germ and hereditary reasons (4).

Impacted third molars are associated with numerous complications, such as pericoronitis, periodontal pathology or root resorption of the adjacent tooth, caries, cystic or neoplastic lesions, orthodontic or prosthetic problems and temporomandibular joint symptoms (5,6). Majority of patients who undergo a surgical extraction of an impacted third molar suffer from pain, swelling, trismus and general oral discomfort during the first postoperative days. The other less frequent complications arealveolitis, infection, hemorrhage and nerve injury (7).The aim of the study is to assess the pattern of impaction of the mandibular third molars.

Various classifications have been given on impacted teeth such as WINTER'S classification, PELL AND GREGORY'S classification, KILLEY AND KAY, ARCHER'S classification of impacted maxillary teeth, etc. Winter's classification is classified based on the inclination of the impacted tooth to the long axis of the second molar into distoangular, mesioangular, horizontal, vertical and transverse. This classification is used for the study as it is simple and easily understandable.(16) The aim of this study is to evaluate the positionof impacted third molars and also to evaluate the most common sex affected using the Winters classification. (17)

Impacted third molars can also be classified according to their angular relationship to the adjacent second molar. Angulation of the impacted third molar can be determined by evaluating the angle formed between the intersected longitudinal axes of the impacted third molar and the adjacent second molar, as described by Winter, either visually or by using an orthodontic protractor.

Materials and Methods

The impaction pattern of the mandibular molars was studied using Orthopantomograph (OPG). OPG’s of 150 patients who had impacted mandibular third molars were used for the study. The patients included in the study were between 18-25 years of age, with a mean age of 21.5 years.The pattern of impaction is derived by measuring the angles formed between the lines intersecting the long axis of the second and third molar. The angle formed is used to determine the mesial, distal, horizontal and vertical inclination in relation to the second molar.

The radiographs were interpreted for the following:

a) Unilateral or bilateral impaction of the mandibular third molar.
b) Pattern of impaction of the mandibular third molar.
c) Gender difference.

Results:

The results have been tabled in the tables 1-6.

Table 1: Gender distribution

Gender	N	Percentage
Male	59	39.33
Female	91	66.66
Total	150	100

Table 2: Distribution of mandibular impaction in the sample

Side Affected	Frequency	Percentage
Left Side Only	35	23.33
Right Side Only	29	19.33
Both Sides	40	26.66
Erupted	46	30.66
Total	150	100

Table 3: Gender distribution pattern of impaction on right quadrant in female patients

Right Mandibular Third Molar	N	Percentage
Absence	29	31.9
Mesioangular Impaction	29	31.9

Distoangular Impaction	19	20.9
Vertical Impaction	6	6.5
Horizontal Impaction	7	7.6
Total	91	100

Table 4: Gender distribution Pattern of Impaction on right quadrant in male patients

Right Mandibular Third Molar	**N**	**Percentage**
Absence	17	28.81
Mesioangular Impaction	20	33.89
Distoangular Impaction	8	13.56
Vertical Impaction	5	8.47
Horizontal Impaction	9	15.25
Total	59	100

Table 5: Gender distribution pattern of impaction on left quadrant in female patients

Left Mandibular Third Molar	**N**	**Percentage**
Absence	21	31.5
Mesioangular Impaction	35	23.3
Distoangular Impaction	12	18
Vertical Impaction	10	15

Horizontal Impaction	12	18
Total	90	100

Table 6: Gender distribution pattern of impaction on left quadrant male patients

Left Mandibular Third Molar	N	Percentage
Absence	25	37.5
Mesioangular Impaction	22	33
Distoangular Impaction	7	10.5
Vertical Impaction	4	6
Horizontal Impaction	9	13.5
Total	67	100

Discussion

Out of a total number of 150 OPG's used in the study, 59 were of males (33.33%) patients and 91 were of females (66.66%) patients (Table 1). A significant difference was seen in unilateral and bilateral distribution of third molar impaction. Unilateral impaction was found in 64 radiographs which accounted for 43% of the total and bilateral impaction was seen in 40 people which accounted for 26.6% of the sample (Table 2).

The mesioangular pattern of impaction was more common and was followed by distoangular, vertical and horizontal patterns. Third molar impactions were more common in the right quadrant (34% percent for the males and about 39% for the females) than in the left quadrant (23.3% in females and 33% in males). There's no sexual predominance seen in impaction pattern or agenesis (Tables 3-6). These findings are similar other studies done in different populations(8,9,10,11).

The main factor for third molar impaction is the lack of space for eruption. The early man ate coarse uncooked food that resulted in attrition of teeth on the occlusal and proximal surfaces of the teeth. This attrition and tendency of mesial drifting of the teeth provided the space required for the eruption of the third molars. Whereas, in modern man, the attrition of teeth

does not take place due to cooked and soft textured food and hence as a result the space required for third molars eruption by attrition and mesial drifting of teeth is lost (12-15).

Other factors that bring about impactions include the growth of the face, size of jaw, tooth size and food habits, overlying bone or soft tissue or with the host having systemic diseases etc. A properly positioned third molar erupts between the ages 18 and 24. The other complications seen with late eruption of the third molar and third molar impaction include incisor crowding, resorption of roots of adjacent tooth, inflammatory process and TMJ dysfunction.

The distribution of angulation and depth of impaction in the impacted lower third molars seen in this study is similar to that noted by Kramer and Williams. (18-19) They reported that 75% of impacted lower third molars were in mesio-angular and horizontal angulation. The angulation of an impacted tooth against the second molar has potential clinical implications, as outlined by Yamaoka et al. (20,21)

Conclusion

The following are the conclusions derived from the study,

- Bilateral impaction of mandibular third molar was more common than unilateral impaction
- Mesioangular impaction was the most common type of impaction and it was more common in males than in females followed by horizontal and vertical impaction.
- Agenesis was more common in females than in males.

Corresponding Author
R.Navaneethan,
Reader,Department of Orthodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162, Poonamallee High Road, Chennai-600077, Tamil Nadu, India
Mobile: +91-9962787531
Email: ethan.nava@gmail.com

Reference

1. Kapoor V.*Textbook of oral and maxillofacial surgery*. New Delhi, Arya (Medi) Publishing House. 1996;1: 45-71.
2. Rosa AL, Carneiro MG, Lavrador MA, Novaes AB. Influence of flap design on periodontal healing of second molars after extraction of impacted mandibular third molars. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2002; 93: 404–7.

3. Arta SA, Kheyradin RP, Mesgarzadeh AH, Hassanbaglu B. Comparison of the Influence of Two Flap Designs on Periodontal Healing after Surgical Extraction of Impacted Third Molars. *J Dent Res Dent Clin Dent Prospect*. 2011;5:1–4.
4. Juodzbalys G, Daugela P. Mandibular Third Molar Impaction: Review of Literature and a Proposal of a Classification. *J Oral Maxillofac Res.* 2013;4: (e1)1-12.
5. Farish SE, Bouloux GF. General technique of third molar removal.*Oral Maxillofac Surg Clin North Am.* 2007;19:23–43.
6. CetinkayaBO, Sumer M, Tutkun F, Sandikci EO, Misir F. Influence of different suturing techniques on periodontal molars after extraction of impacted mandibular third molars. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod*. 2009; 108:156–61.
7. Charan Babu HS, Reddy PB, Pattathan RK, Desai R, Shubha AB. Factors influencing lingual nerve paraesthesia following third molar surgery: a prospective clinical study. *J Maxillofac Oral Surg.* 2013;12:168–72.
8. Santhosh Kumar M.P and ShamaraAysha. Angulations Of Impacted Mandibular Third Molar: A Radiographic Study in Saveetha Dental College. *J. Pharm. Sci. & Res*. 2015; 7(11): 981-983.
9. Sonti Sri Harsha. Incidence of Mandibular Third Molar Impaction in Patients Visiting a Private Dental College. *IOSR Journal of Dental and Medical Sciences (IOSR-JDMS).* 2014: 13(1): 01-02.
10. Obuekwe ON, Enabulele JE. Gender variation in pattern of Mandibular Third Molar Impaction. *J Dent Oral Disord Ther*. 2017; 5(2):1-4.
11. Kramer RM, Williams AC. The incidence of impacted teeth: A survey at Harlem Hospital.*Oral Surg Oral Med Oral Pathol*. 1970; 29(2): 237-241.
12. Bolender CJ. Attritional occlusion, the basis of the Begg technic. *Orthod Fr.* 1985; 56(2):409-16.
13. Arne Bjork, Elli Jensen and MogensPalling.Mandibular growth and third molar impaction. *Acta OdontologicaScandinavica* 1956; 14(3): 231-272.
14. Obiechina AE., Arotiba JT, Fasola AO. Third molar impaction: evaluation of the symptoms and pattern of impaction of mandibular third molar teeth in Nigerians. *Odonto-Stomatologie Tropicale.*2001; 24(93):22-25.
15. Hattab FN, Rawashdeh MA, Fahmy MS. Impaction status of third molars in Jordanian students. *Oral Surg Oral Med Oral Pathol Oral Radioland Endod*. 1995; 79(1): 24-29.
16. M.P. Santhosh Kumar et al /J. Pharm. Sci. & Res. Vol. Angulations Of Impacted Mandibular Third Molar : A Radiographic Study in Saveetha Dental College 2015, 7(11), 981-983,
17. Sldv R.M.K., Das M.C., Vijayaraghavan R., ShanmukhaI.Incidence of agenesis, impactions, angular positions and pathologies related to third molar teeth, National Journal of Physiology, Pharmacy and PharmacologyDepartment of Research, Saveetha University, Chennai, Tamil Nadu, India; Department of Pharmacology, NRI Medical College, Chinakakani, Andhra Pradesh, India; Department of Pharmacology, SCS Pharmacy College, Harapanahalli, Karnataka, India. 2017; 7(5):(486-490) .
18. Neelakantan P., Sharma S., Shemesh H., Wesselink P.R.Influence of Primary and Secondary Closure of Surgical Wound After Impacted Mandibular Third Molar

Removal on Postoperative Pain and Swelling-A Comparative and Split Mouth StudyJournal of Endodontics)Saveetha Dental College and Hospitals, Saveetha University, Chennai, India; Academic Center for Dentistry Amsterdam (ACTA), Amsterdam, Netherlands. ,2015;41(7):1108-1111.

19. Subathra Devi V., GnanavelB.K.Prophylactic removal of third molars. Procedia Engineering() Department of Civil Engineering, Saveetha School of Engineering, Saveetha University, Thandalam, Chennai, Tamil Nadu, India; Department of Mechanical Engineering, Saveetha Engineering College, Thandalam, Chennai, Tamil Nadu, India. 2014; 9:95-104
20. Danda A.K., Krishna Tatiparthi M., Narayanan V., SiddareddiA.Influence of Primary and Secondary Closure of Surgical Wound After Impacted Mandibular Third Molar Removal on Postoperative Pain and Swelling-A Comparative and Split Mouth Study,Journal of Oral and Maxillofacial Surgery(); Department of Oral and Maxillofacial Surgery, Saveetha University, Chennai, India. 2010; 68(2):309-312.
21. Balasubramaniam P., NabeelNazarM.Complications following mandibular third molar surgeryInternational Journal of Clinical Dentistry.Department of Oral and Maxillofacial Surgery, Saveetha University, Chennai, TN, India.2015, 7(4):(337-346.

Attitude and Knowledge of Oral Health Care Professionals in Treating Mentally Challenged Patients in Hospital Setting - A Cross Sectional Study

Hansini B. Ajanta[1],Pavithra Priyadarshoni .S[2], Laliytha K.B[3]

Graduate Student[1],Tutor, Department of Public Health Dentistry[2], Senior lecturer, Department of Oral Medicine[3].

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Mentally challenged patients being treated by oral health care professionals is becoming common these days. As these patients should be treated with utmost patience and care, it is understandable that concern exists about the practitioner's attitude, confidence and skill levels in recognising and treating this group of patients.The purpose of this study is to assess the attitude, knowledge and confidence of oral health care professionals in treating mentally challenged patients.

Materials and Methods:A questionnaire based cross-sectional study is conducted among 430 oral health care professionals of Saveetha dental college. A questionnaire consisting of 15 validated questions was personally distributed to the oral health care professionals and their responses were collected.

Results:The attitude of oral health care professionals us good whereas they exhibit average knowledge and low confidence level in treating these group of patients.

Conclusion:Educating and training oral health care professionals should be emphasised in dental schools, so that they can manage these patients confidentially and skilfully in their general practice.

Key Words: *Mentally Challenged Patients, Attitude, Awareness, Knowledge, Oral health care professionals*

Introduction

Mental disorders affect more than 25% of all people at some time during their lives.People tend to have strong beliefs about the mentally ill, and many of these concepts are based on prevailing local systems of belief. Social stigma and negative attitudes can affect the quality of life for people with mental illness. Lack of accurate information about mental illness, lack of contact with individuals with mental illness and lack of familiarity might be one of the most important reasons of these. Educating people may have positive impact on this prejudice. [1], [2]Such negative attitudes may be detrimental not only to the patient care but also to the society's attitude to psychiatric disorders if they are present in the health professionals [3]. The knowledge of the attitude and awareness of the undergraduate medical students toward psychiatry, mental health and psychiatric disorders is of utmost importance as these individuals are going to be involved in the care of these patients either directly or indirectly during the later years of their careers. Psychiatry as a discipline is felt to be given a prime importance at the undergraduate levelwhich is reflected in the number of lectures in psychiatry andnumber of hours of psychiatry clinical posting. The present study aims at understanding the attitude of the oral health care professionals toward psychiatry, mental health, psychiatric disorders and their treatment.

Materials and Methods

A questionnaire based cross-sectional study is conducted among 430 oral health care professionals,which includes undergraduate interns, postgraduate students and teaching faculty of Saveetha dental college from November 2016 to December 2016. Ethical committee clearance was obtained. A questionnaire consisting of 15 validated questions was personally distributed to the oral health care professionals.Subjects were explained about the aims and objectives of the study being conducted in detail. Informed consent was taken after ensuring confidentiality of the information gathered in the questionnaires being used and clarifying that it would only be used for the purpose of the research intended. Their responses were collected and statistical analysis was done.

Research Proforma

1) GENDER: M/F

2) AGE: years

3) a) STUDENT

- UG
- PG

b) FACULTY

If yes, speciality ----------

4) Years of experience in clinical practice: years

5) Are you aware that mentally challenged patients come under the category of patients with disability?

a) Yes b) No

6) Are you aware of the spectrum of mental disorders?

a) Yes b) No

7) Are you aware of the percentage of patients with mental disability regarding dental treatment in your locality?

a)Yes b) No

8) Does your immediate circle (family/ friends) include anybody with mental disability?

a)Yes b) No

9) Are you interested in providing oral care for mental disability group patients?

a)Yes b) No

10) Are you interested to be a part of the community health services to render treatment for mental disability patients?

a)Yes b) No

11) As a dentist by qualification are you confident in delivering basic oral care in your clinic for these group of patients?

a)Yes b) No

12) Are you confident to manage any complications which may arise while treating these patients?

a)Yes b) No

13) Are you comfortable in referring these patients to concerned specialists for oral care?

a)Yesb) No

14) Does your course curriculum teach/ train to manage patients with mental disability?

a)Yes b) No

If yes, specify

- UG

- PG

15) If given an opportunity to work with these group of patients over a fixed period for training, will you render your dental services?

a)Yes b) No

Results

A total of 430 oral health care professionals participated in this study, 117 males and 313 females, out of which 139 were interns (under graduates), 170 were post graduates and 121 were teaching faculties. They were categorised under three age groups, 21 to 25 yearsold-229 candidates, 26 to 30 years old-107 candidates and above 30 years old-94 candidates. (Table 1)

Table 1: General characteristics of the candidates

Characteristic	N
Age	
21-25	229
26-30	107
>30	94
Gender	
Male	117
Female	313
Education	
Interns(UG)	139
PG	170
Faculty	121

In this study, 74% were aware that mentally challenged patients come under the category of patients with disability, 52% were aware of the spectrum of mental disorders, only 25% were aware of the percentage of mentally challenged patients requiring dental treatment in their locality, 7% has people with mental disability in their circle, 61% were interested in providing oral care for these group of patients, 66% were interested in being a part of

community health services rendering treatment for these group of patients, 27% were confident in delivering basic oral care for these group of patients, 26% were confident to manage any complications while treating these group of patients, 80% were comfortable in referring these patients to concerned specialists, 73% were willing to render their dental services, over a fixed period of training and 20% were trained to manage these patients in their course. (Table 2)

Table 2: Attitude and Knowledge of Oral Health Care Professionals in Treating Mentally Challenged Patients

S.NO.	STATEMENT	RESPONSE	FREQUENCY (%)
1.	Mentally challenged patients come under the category of patients with disability	Yes	74
		No	26
2.	Awareness of the spectrum of mental disorders	Yes	52
		No	48
3.	Awareness of the percentage of mentally challenged patients requiring dental treatment in their locality	Yes	25
		No	75
4.	Immediate circle (family/ friends) include anybody with mental disability	Yes	7
		No	93
5.	Interest in providing oral care for these group of patients	Yes	61
		No	39
6.	Interest in being a part of community health services rendering treatment for these group of patients	Yes	66
		No	34
7.	Confidence in delivering basic oral care for these group of patients	Yes	27
		No	73
8.	Confidence to manage any complications while treating these group of patients	Yes	26
		No	74
9.	Comfortable in referring these patients to concerned specialists	Yes	80
		No	20
10.	Willingness to render their dental services, over a fixed period of training	Yes	73
		No	27

11.	Trained to manage these patients in their course.	Yes	20
		No	80

Limitations

This study was not without any limitations. Sample size was quite largecompared to other studies done related to attitude of health care professionals in treating mentally challenged patients. Furthermore, the participants were selected from only one institute, generalization of the results is difficult. Cross-sectional nature of the study and absence of any control groups might not have served our purpose of assessing the attitude and ascertaining the importance of undergraduate education in Psychiatry. Some observed differences on groupwise and sex-wise comparison may be attributed solely to the cohort effect.

Discussion

Stigma appears in the students who lack knowledge, adopting negative attitudes and avoiding certain group of people, usually with mental illness [4]. Despite of the burdens of the mental illness, some societies continue to hold deep-rooted, culture sensitive and negative beliefs about mental illnesses. The theory of classical labeling focus on stereotyping and rejection of others which may lead to social exclusion and demoralization [5]. This group of people usually show the impairment in cognitive, emotional and behavioral aspects. Due to this the patients are significantly less willing to interact with general population and sometimes it creates loneliness to patient or it may leads to violent behavior towards others who are less willing to interact with them [6]. Due to this stigma and discrimination the people with mental illness face difficulties in social relationships, experience social isolation, social withdrawal, social distance, homelessness, unemployment and institutionalization [7]. The patients with mental illness are considered as labeled persons. The concept of stigma has been divided into four components: labeling, stereotyping, separation, status loss and discrimination. Labeling involves categorizing the people in different groups. Stereotyping involves the association of labels with negative attributes. Separation is itself used to justify the association of negative attributes to labeled persons. Finally these three leads to loss of status and an experience of discrimination by the labeled person. The four components are interconnected with each other [8]. To overcome this stigma and discrimination from decades is to support and change the tools of institutionalization. Lack of knowledge and awareness

among the students is associated with the negative attitudes towards mental illness in the community. Various health professionals have to conduct awareness camps at initiative levels like schools and universities. Attitude is a hypothetical construct that represents an individual's like or dislike for a living or non living thing, which may be positive, negative or neutral. It originates from judgments and has affective, behavioral and cognitive components. [9] It also determines orientation towards a particular environment. [10] So, orientation of the oral health care professionals towards management of psychological problem in physically ill patients during medical training shapes their attitude towards mental illnesses as a disease. In the debate about changing social attitude, the issue is whether a positive change in attitude also leads to an improvement in anti stigmatizing behaviour. Theories about behavioural change and learning vary as to whether knowledge gained through extrinsic reinforcement, eg.Grades or positive remarks [11]. In social learning theories or cognitive theories, attitude change is seen as dependent on personal factors, environmental factors and attributes of the behaviour itself [12]. In the ecological learning model, behaviour change is effective if it occurs on multiple levels: interpersonal factors, group factors, institutional factors, community factors and public policy [13]. Long lasting attitude change is thus a complicated matter. Previous results from research in the area of attitudes towards mental illness found somewhat divergent results. Some researchers claim that changing prejudicial attitudes towards persons with mental illness have a direct influence on discriminatory behaviour [14]. Broad educational approaches aimed at changing inaccurate stereotypes about mental illness have shown to be effective in producing a short term improvement in attitudes [15].

The current study made use of a questionnaire based survey methodology to assess the attitude of oral health care professionals.The attitude of oral health care professionals us good whereas they exhibit average knowledge and low confidence level in treating these group of patients. A similar study done by Neeli Uma Jyothi et al showed neutral attitude of the participants in treating these patients. A study done by Peter Baghurst et al and Lisa et alshowed the attitude towards mental disability changed after the students attended a training program for treating mental disabilities [16] [17].Encouraging and actively involving the students in educational programs, organising camps and creating awareness towards managing mentally challenged patients should be done[18].As more dental schools utilise community based clinical experiences to increase student's exposure, it is important that these experiences provide exposure to these group of patients.Other similar studies by Chou and Maket al, Chung et al, Wolff et alhave shown that, general practitioners or oral health

care professionals who have previously treated these group of patients have shown more positive attitude towards mental illness [19] [20] [21]. A study done by Loan P. et aloral health care professionals did not think their undergraduate dental education had prepared them well to treat mentally challenged patients [22].Major efforts need to be made to improve dental education concerning these patient groups.A study done by Raymond et al, showed an average comfort level and willingness to treat special care needs patients and vulnerable populations [23].It is obvious that providing knowledge and skills basis will improve dentists attitude towards treating these patients and will give more confidence. Such positive attitudes may then allow them to think about overcoming obstacles that may keep them from treating these patients.

Conclusion

The oral health care professionals had multiple lacunae in their knowledge toward psychiatry, psychiatric disorders, psychiatric patients and psychiatric treatment. The possible reasons could be many. But a potential contributory fact could be the neglect of psychiatry as a discipline at the undergraduate level. The lesser number of hours assigned to psychiatry at this level could add to the cause. Subjects such as psychiatry are underrepresented during this training. It has been felt that psychiatry remains a neglected subject during the undergraduate training [22]. Thus, there is a need to relook into the curricula of the undergraduates and possibly a few solutions could be reached at. Overall attitude towards mental illnessamong the oral health care professionals is good whereas they exhibit average knowledge and low confidence level in treating this group of patients.A further study with oral health care professionals from different institutional backgroundis needed to get a detail nationwide picture that canbe implemented in future academic and professionalpractice.

Corresponding author

Pavithra Priyadarshoni S.,

Tutor, Department of Public Health Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id- drpavithraphd88@gmail.com

Mobile Number:+9176222104

References

1. Neeli Uma Jyothi,MounicaBollu, SK Faizan Ali, D Sri Chaitanya, S Mounika. A Questionnaire Survey on Student's Attitudes towards Individuals with Mental Illness.J. Pharm. Sci. & Res. 2015; 7(7):393-96.
2. BWM Siu, KKW Chow, LCW Lam, WC Chan, VWK Tang, WWHChui. A Questionnaire Survey On AttitudesAnd Understanding Towards Mental Disorders. East Asian Arch Psychiatry. 2012; 22:1824.
3. Raj SC. Management of oral care among people with intellectual disability. Research Journal of Pharmacy and Technology. 2015 Aug 1; 8(8):1068.
4. Jennifer Hill; Attitudes toward Mental Disorders among CollegeStudents**:** Uw-L Journal of Undergraduate Research Viii (2005) 1-8.
5. Gilbert, P., Bhundia, R Mitra, R., Mcewan, K., Irons, C. & Sanghera, J. (2007). Cultural Differences in Shame-Focused Attitudes towards Mental Health Problems in Asian and Non-Asian Student Women. Mental Health, Religion & Culture, 10, 127-141.
6. Reddy KV, Sharma A. Prevalence of oral health status in visually impaired children. Journal of Indian Society of Pedodontics and Preventive Dentistry. 2011 Jan 1; 29(1):25.
7. Gyllensten,A.,Svensson,B.,Bjorkman,T.,Hansson,L.,Leufstadius,C.,Bejerholm,U.,Ostman,M.,Eklund,M.,Brunt,D.,Sandlund,M.AttitudesIn Healthcare Students Towards Mental Illness-A Pre- And PostMulticentre University Program Survey. The Internet Journal OfAllied Health Sciences And Practice.July 2011.Volume 9 Number 3.
8. Ghanean, H., Nojomi, M. And Jacobsson, L. (2015) A CommunityStudy On Attitudes To And Knowledge Of Mental Illness In Tehran.Open Journal Of Psychiatry, 5, 26-30.
9. Burra P, Kalin R, Leichner P, Waldron JJ, Handforth JR, Jarrett FJ, Amara IB. The ATP 30-a scale for measuring medical students attitudes to psychiatry. Med Educ. 1982 Jan;16(1):31-8.
10. Khan SA, Yousafzai AU, Mehir RK, Inam-ul-Haq. Attitude of medical students towards psychiatry in NWFP. J AyubMed Coll Abbottabad. 2008 Jul-Sep; 20(3):44-6.

11. Korthagen FAJ. Situated learning theory and the pedagogy of teacher education: Towards an integratedview of teacher behavior and teacher learning. Teacher and Teacher education. 2010;26:98-106.
12. Bandura A. Self efficiency mechanisms in psychological activation and health promoting behaviours. In: Adaptation learning and effect. Edited by Matton IO, Mathysse S, Barchas J. New York: Raven press; 1986.
13. Hill LH, Clover DE. Environmental adult education: Ecological learning, Theory and practice new directions for Adult and Continuing education, No. 99(Fall). San Francisco: Jossey-Bass; 2003.
14. Corrigan PW, Green A, Lundin R, Kubiak MA, Penn DL. Familiarity with and social people who have serious mental illness. Psychiatr serv. 2001;52:953-58.
15. Corrigan PW, Gelb B. Three programs that use mass approaches to challenge the stigma of mental illness. Psychiatric services. 2006;57:393-8.
16. Peter Baghurst. Mental health of young people in Australia. Australian and New Zealand journal of psychiatry. 2002;10: 1440-1514.
17. Lisa M. Delucia. Dental students attitude towards care of individuals with intellectual disabilities. Journal of dental education. 2009; 445-53.
18. Natalie Khin. Questionnaire development for the management of mental disorders in general practice. nzfp2003;30(2): 405-15.
19. Chung KF. How are psychotic symptoms perceived?. Australian and New Zealand journal of psychiatry.1997; 36:756-61.
20. Chou KL, Mak KF. Attitude towards mental illness among Hong Kong Chinese. International journal of social psychiatry.1996; 44: 215-24.
21. Wolff G, Craig T, Leff J. Community knowledge of mental illness and attitude towards mentally ill people.British Journal of psychiatry. 1996; 168:191-98.
22. Loan P Dao. General dentists and special needs patients: Does education matter? Journal of dental education. 2005; 1107-15.
23. Raymond A Kutty. Student's comfort level in treating vulnerable populations and future willingness to treatment.; Journal of dental education. 2005; 1307-14.
24. Feldmann TB. Medical students' attitudes toward psychiatry and mentaldisorders. Acad Psychiatry 2005;29:354-6.

Attitude and Perception of Undergraduate Dental Students towards Endodontics as a Specialty

S. Mohamed Jaqfer Sadiq[1], Jayalakshmi Somasundaram[2], Aarthi Vignesh[3],
Graduate Student[1],Senior Lecturer[2],Department of Conservative Dentistry and Endodontics,
Senior Lecturer[3], Department of Oral and Maxillofacial Surgery,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Students attitude toward choosing a specialty subject for post graduate studies is of great importance for the education system. The scope of endodontics has broadened vastly in recent years.CBCT, MRI, Doppler's, PCR in diagnosis and the treatment planning for oral lesions of pulp and periapical origin, Nonsurgical approach for root canal therapy, microsurgical procedures for removal of pathologic tissues with minimal intervention are the various types of advancement introduced in Endodontics.

Materials and Methods: A questionnaire study survey consisting of 10 multiple-choice questions was compiled and distributed among 100 undergraduate dental clinical students studying in Saveetha dental college. A pilot study was conducted to check the feasibility and reliability of the questionnaire.The main domains appraised were preclinical and clinical training in endodontics, their opinion and knowledge in endodontics as post-graduate specialty.

Result: Total number of undergraduate students participated in the study were 100. 92% of the students participate think endodontics as one of the major branch in dentistry concerned about estheticswhereas the remaining 8% disagreed to it.

Conclusion: This study concluded that most of the undergraduate students are interested in persuading endodontics as a specialty for post-graduation. Thus it needs more emphasis on preclinical endodontic training and clinical training for management of all pulpal and periapical lesions with different modalities at the undergraduate level.

Key Words: *Endodontics, Post-Endodontic rehabilitation, 3-D Endodontics, Rotary instrumentation.*

Introduction

Students attitude toward choosing a specialty subject for post graduate studies is of great importance for the education system. The scope of endodontics has broadened vastly in recent years.CBCT, MRI, Doppler's, PCR in diagnosis and the treatment planning for oral lesions of pulp and periapical origin, Nonsurgical approach for root canal therapy, microsurgical procedures for removal of pathologic tissues with minimal intervention are the various types of advancement introduced in Endodontics.(1) The specialty also includes endodontic implants; bleaching of discoloured teeth, retreatment of previously endodontically treated teeth with special instruments, post-endodontic rehabilitation of teeth with more preservation of tissues and the management of traumatically injured teeth.(2) But unfortunately, endodontics appears to be similar with root canal treatment among the undergraduate students.This reality necessitates dental students to be satisfactorily equipped with knowledge as well as experience in endodontic procedures prior to working independently. (3)The UG students it is necessary for the UG students to understand what should be the extent of endodontic training, so that they will be able to render basic endodontic treatment (4). In this way, it is the duty of dental schools to set up their understudies to attempt at any rate straightforward root trench medicines of unsurprising quality on graduation. [5] In India the undergrad (UG) preparing in endodontics is restricted to conclusion of endodontic sores, straightforward imperative mash treatment and root channel treatment for foremost teeth. The UG understudies and the assistants are thought about fledgling, so it is fundamental that instructors comprehend what ought to be the degree of endodontic preparing that must be conferred to the understudies previously they graduate, with the goal that they will have the capacity to render essential endodontic treatment. This examination planned to assess the fourth year understudies and assistant's observation and state of mind toward learning, in rendering endodontic treatment, picking endodontics as a strength for post-graduation and general supposition of the claim to fame.(6,7)There are numerous references in the dental literature regarding the quality and outcome of endodontic treatments carried out by dental students; however, there is scarce information regarding the way students perceive the branch of endodontology and their level of self-confidence about various aspects of endodontic treatment with respect to their future practice.(8)The aim of

this survey was to gather information about the general opinion of dental students in Saveetha dental college regarding endodontic treatment, to analyze their perception of this significant branch of dentistry and how they self-evaluate their confidence level in endodontic treatment.The aim of the study is to evaluate the dental students' perception and attitude toward learning, in rendering treatment, choosing endodontics as a specialty for post-graduation.

Materials and Methods

A questionnaire study survey consisting of 10 multiple-choice questions was compiled and distributed among 100 undergraduate dental clinical students studying in Saveetha dental college.A pilot study was conducted to check the feasibility and reliability of the questionnaire.The main domains appraised were preclinical and clinical training in endodontics, their opinion and knowledge in endodontics as post-graduate specialty.All answers were kept confidential, and no individual students were identified. The students were not allowed to re-participate in the survey once completed.

Result

Total number of undergraduate students participated in the study were 100. 92% of the students participate think endodontics as one of the major branch in dentistry concerned about esthetics whereas the remaining 8% disagreed to it. The analysis for question no 2 showed 22% of them opted cone beam computed tomography in pulp all disease diagnosis as the best suited advancement in endodontics and 28% felt sonic irrigation for cleaning and shaping as best while 50% of them found optical occurrence of tomography in diagnoses of pulpal disease as the best.Analysis of question no 3 revealed that 78% of the students were not sure of microsurgical endodontics though 18% responded yes. 74% of the students thought microsurgical endodontics created a revolution and 6% gave a negative response while 20% had no idea about microsurgical endodontics means. 44% students suggested that rubber dam isolation is good but time consuming and 40% taught its unnecessary as it may hurt the patientand 16% mentioned it is really important to isolate rubber dam for safety measures. 52% of the students responded that endodontics makes lot of money when opted as post graduation and 12% felt that endodontic can be specialised for the advancements it has seen. And 36% of the students refused to take endodontics as their post graduation. 78% of the students suggested that use of rotary instruments are not thought in an effective manner

and also 4-6% found it not helpful. 64% of the students responded yes that undergraduates just must be trained to treat molar RCT and 34% feel it has high risk of perforations. 88% of the undergraduates suggested that they were confident to identify and refer patients with advanced endodontictreatment.

Discussion

With rapid changes taking place in treatment techniques and materials in the field of endodontics, it is important that UG students are equipped with understanding and preclinical experience while using these instruments and materials before treating patients. There is a wide variation in the way the endodontics is taught and the level of competencies developed in UGs in various institutions across the world.(9,10) It also recommended that endodontic educational aids such as models, 3-D interactive tooth atlas which combines sound, text, stills and video with interactive learning should be used as educational adjuvants. (11)Studies have suggested that endodontic computer simulation programs results in significant gain in knowledge and can successfully replace traditional learning strategies.(12) The undergraduate students are not much convinced that the use of rubber dam is effective and efficient in endodontics were as in other studies they have shown students preferring rubber dam more convenient and effective.(13)

In the USA, it was discovered that a dental undergrad's "possession of special skills or talent”was the principle explanation behind needing to practice as opposed to any money related factor, [14] and once more, Orthodontics was the most favoured subject. This investigation additionally reasoned that sexual orientation was not a vital determinant when it came to picking strength, which is predictable with past examinations [15,16]. In any case, later research [16] did among fourth year dental understudies at another school inside the USA found that sexual orientation had an effect on the decision of claim to fame, and that guys were seven times more inclined to progress into an Oral and Maxillofacial Surgery field, while females were four times more slanted to think about Pediatric Dentistry. This investigation found that 'pleasure in giving consideration' was the major impacting factor for picking any postgraduate instructive heading. Discoveries of the Annual American Dental Education Association [17,18] whose exploration included senior dental understudies from every dental school over the USA likewise announced Orthodontics to have been the most mainstream claim to fame for dental understudies from 1995 to 2004, trailed by Oral and

Maxillofacial Surgery and Pediatric Dentistry. As anyone might expect these three claims to fame were likewise the most usually offered as postgraduate courses by both American and Canadian dental school.[19,20]

A survey by Al-Jewai et al. presumed that PC helped learning is proposed to help conventional learning strategies, yet not to supplant them. [21] Only 53.2% of the respondents trust that they have been prepared in preclinical endodontics utilizing removed teeth models. 71% of the understudies trusted that extraordinary preclinical preparing will assist them with managing patients certainly. Dhima et al. assessed dental understudies recognition on dental fortes and components that can assume an essential part in understudies choice to seek after claim to fame preparing or vocation decisions and inferred that happiness regarding giving consideration in a strength or field was recognized as the absolute most vital factor in picking a claim to fame profession. [22] 84.3% of the members in the review recommended that they would have some expertise in endodontics if given an opportunity, which is a positive sign for the claim to fame.

In contrast to past examinations [23], this present investigation found that sexual orientation had an impact on choice to practice, and essentially, twice the same number of females contrasted with guys was undecided on specialization. This is justifiable as female dental practitioners, notwithstanding working and contemplating, are regularly dedicated to raising youngsters, with, all things considered 71% of female dental specialists announced as having kids [24]. Childcare needs will impact the future work examples of female dental specialists [25]. Also, more female than male dental understudies inside the UK hope to remove additional time from their future professions to focus on childcare [26].

87% of students associated endodontics with root canal therapy. But there is a paradigm shift in management of pulpal and periapical diseases with emphasis on vital pulp therapy, regenerative endodontics, microsurgical procedures, post-endodontic restorations, non-vital bleaching for esthetics, endodontics as a part of interdisciplinary treatment modalities , thus suggesting need to emphasis on these treatment modalities during teaching of this specialty. [27]93% of the students suggested that there should be a change in the teaching methodology. Respondents also recommended that endodontic educational aids such as models, 3-D interactive tooth atlas which combines sound, text, stills and video with interactive learning

should be used as educational adjuvants. Studies have suggested that endodontic computer simulation programs results in significant gain in knowledge and can successfully replace traditional learning strategies. [28],[29] According to Urbankova, eight hours of computerized dental simulator training conducted early in the preclinical operative dentistry improved student performance which could also be true for endodontic preclinical training. [30]

Flare-ups are unfortunate circumstances that may emerge over the span of endodontic treatment, requiring an unscheduled visit in some cases.[31] It is additionally obvious that, flare-ups don't specifically impact the result of the endodontic methodology, yet are fairly distressful circumstances coming about because of the disturbance of the harmony between the host safeguard component and chafing operators.[31] One reason for the event of between arrangement flare-ups might be procedural mistakes amid the execution of endodontic treatment, for example, expulsion of intracanal content incidentally into the periradicular tissues. It can be guessed that flare-ups might be experienced all the more oftentimes in the understudies facility, conceivably because of inability of understudies enabling them to make some procedural blunders, for example, overinstrumentation or expulsion of irrigants and intracanal flotsam and jetsam.

Conclusion

This study concluded that most of the undergraduate students are interested in persuading endodontics as a specialty for post-graduation. Thus it needs more emphasis on preclinical endodontic training and clinical training for management of all pulpal and periapical lesions with different modalities at the undergraduate level. This study shows that a large number of undergraduates had an intention to specialise, with Restorative Dentistry and Orthodontics being the most popular intended subjects, although many were still undecided at this stage.Endodontics is a vibrant, evolving and interesting clinical specialty. Intense endodontic training at UG level prepares the graduates to provide good basic patient care in clinical practice studies comprising other dental schools will be helpful in precisely determining the extent of instillation of adequate skills in endodontology and major missing areas that need further improvement.

Corresponding Author

Aarthi Vignesh,

Senior Lecturer, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077, Tamil Nadu, India.

Email Id: draarthiomfs@gmail.com

Phone Number: +91 8056985855

References

1. Ingle JI, Bakland LK, Baumgartner JC. Ingle's Endodontics 6. Hamilton, Ontario: BC Decker; 2008;6:345-48.
2. Pileggi R, O'Neill PN. Team-based learning using an audience response system: An innovative method of teaching diagnosis to undergraduate dental students. J Dent Educ 2008;72:1182-8.
3. Holmes DC, Diaz-Arnold AM, Williams VD. Alumni self-perception of competence at time of dental school graduation. J Dent Educ. 1997;61:465–72.
4. Murray FJ, Blinkhorn AS, Bulman J. An assessment of the views held by recent graduates on their undergraduate course. Eur J Dent Educ. 1999;3:3–9.
5. Friedlander L, Anderson V. A new predoctoral endodontic module: Evaluating learning and effectiveness. J Dent Educ 2011;75:351-9.
6. Reader CM, Kleier DJ, College C, Bujanda-Wagner S. Anatomical artificial teeth for teaching preclinical endodontics. J Dent Educ 1994; 58:229-32
7. Nassri MR, Carlik J, da Silva CR, Okagawa RE, Lin S. Critical analysis of artificial teeth for endodontic teaching. J Appl Oral Sci 2008;16:43-9.
8. Urbankova A. Impact of computerized dental simulation training on preclinical operative dentistry examination scores. J Dent Educ 2010;74:402-9.
9. Reader CM, Kleier DJ, College C, Bujanda-Wagner S. Anatomical artificial teeth for teaching preclinical endodontics. J Dent Educ 1994; 58:229-32.
10. Nassri MR, Carlik J, da Silva CR, Okagawa RE, Lin S. Critical analysis of artificial teeth for endodontic teaching. J Appl Oral Sci 2008;16:43-9.
11. Fouad AF, Burleson JA. Effectiveness of an endodontic diagnosis computer simulation program. J Dent Educ 1997;61:289-95.

12. Plasschaert AJ, Cailleteau JG, Verdonschot EH. The effect of a multimedia interactive tutorial on learning endodontic problem-solving. Eur J Dent Educ 1997;1:66-9.
13. Urbankova A. Impact of computerized dental simulation training on preclinical operative dentistry examination scores. J Dent Educ 2010;74:402-9.
14. Al-Jewair TS, Qutub AF, Malkhassian G, Dempster LJ. A systematic review of computer-assisted learning in endodontics education. J Dent Educ 2010;74:601-11.
15. Dhima M, Petropoulos VC, Han RK, Kinnunen T, Wright RF. Dental students' perceptions of dental specialties and factors influencing specialty and career choices. J Dent Educ 2012;76:562-73.
16. Saeed, S.; Jimenez, M.; Howell, H.; Karimbux, N.; Sukotjo, C. Which factors influence students' selection ofadvanced graduate programs? One institution's experience. J. Dent. Educ. 2008; 72: 688–697.
17. Scarbecz, M.; Ross, J.A. The relationship between gender and postgraduate aspirations among first- and fourth-year students at public dental schools: A longitudinal analysis. J. Dent. Educ. 2007;71:797–809.
18. Dhima, M.; Petropoulos, V.C.; Han, R.K.; Kinnunen, T.; Wright, R.F. Dental Students' Perceptions of DentalSpecialties and Factors Influencing Specialty and Career Choices. J. Dent. Educ. 2012; 76: 562–573.
19. Weaver, R.G.; Chmar, J.E.; Haden, N.K.; Valachovic, R.W. Annual ADEA Survey of Dental School Seniors:2004 Graduating Class. J. Dent. Educ. 2005, 69, 595–619.
20. American Dental Association. 2010–2011 Survey of dental education. Acad.Programs Enrol. Grad. 2012; 6:1–78.
21. Weaver R.G., Chmar J.E., Haden N.K., Valachovic R.W. Annual ADEA Survey of Dental School Seniors: 2004 Graduating Class. J. Dent. Educ. 2005;69:595–619.
22. American Dental Association 2010–2011 Survey of dental education. Acad. Programs Enrol. Grad. 2012;6:1–78.
23. Gallagher J.E., Patel R., Wilson N.H.F. The emerging dental workforce: long-term career expectations and influences. A quantitative study of final year dental students' views on their long-term career from one London Dental School. BMC Oral Health. 2009;9:-6831-35.
24. Hunter M.L., Harrhy L.E., Morgan M.Z. The United Kingdom's specialist workforce in paediatric dentistry: Current and future trends. Br. Dent. J. 2010;208:559–562.

25. Drugan C.S., Chestnutt I.G., Boyles J.R. The current working patterns and future career aspirations of specialist trainees in dentistry. Br. Dent. J. 2004;196:761–765.
26. Nixon P.J., Benson R.E. A survey of demand for specialist restorative dental services. Br. Dent. J. 2005;199:161–163.
27. Fisher N.L., Wilson R., Bartlett D. The perceived barriers of vocational dental practitioners in London to specialisation. Br. Dent. J. 2007;203:25–27.
28. Baharvand M., Moghaddam E.J., Pouretemad H., Alavi K. Attitudes of Iranian Dental Students toward Their Future Careers: An Exploratory Study. J. Dent. Educ. 2011;75:1489–1495.
29. I'Rashid, H.H.; Ghotane, S.G.; Abufanas, S.H.; Gallagher, J.E. Short and long-term career plans of final yeardental students in the United Arab Emirates. BMC Oral Health 2013;23:13.
30. Widström, E.; Birn, H.; Haugejorden, O.; Martinsson, T. Dental students' views on their education and studycircumstances in Nordic countries. Swed. Dent. J. 1990; 14: 123–129.
31. Katrova, L.G. Gender impact on the socioprofessional identification of women dentists in Bulgaria.J. Dent. Educ. 2004; 68:19.

Attitude of Adolescent Females towards Orthodontic Treatment – A Questionnaire Based Study

Maria Kurian [1], A.V. Arun[2], Sreedevi Dharman[3]

Graduate Student[1] ,Professor[2],Department of Orthodontics

Senior Lecturer[3], Department of Oral Medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Background: Motivating patients to seek orthodontic treatment affect adherence to treatment suggestions, treatment outcomes, and patient satisfaction. Prejudiced notions about treatment and various fears have created a resistance or hesitation towards orthodontic treatment in women. The aim of this study is to assess the mind set and overall attitude of female adolescent patients towards orthodontic correction of malocclusion.

Materials and methods: A questionnaire was prepared and distributed to 50 female adolescent patients with malocclusions visiting Saveetha Dental College and Hospital. All questionnaires were anonymously collected and the data was kept confidential and not used for anything apart from the study purpose.

Results: It was seen that the female adolescent patients were unwilling for braces as they were scared or that they found it embarrassing, expensive, painful and has long treatment duration.

Conclusion: The attitude of female adolescent patients with malocclusions for orthodontic treatment is studied so as to be able to convince them for orthodontic treatment.

Keywords*: Attitude, Females, Orthodontics*

Introduction

Orthodontic treatment is the process of aligning the teeth or correcting other irregularities, which thus improves health, function, appearance, and social well-being [1,2].Malocclusion is an occlusion where there is a mal-relationship between both the arches in either of the planes or there are irregularities in tooth position which extend beyond the normal limits. The etiology of malocclusion can be genetic or environmental and or a combination of both along with various local factors such as oral habits, tooth anomalies etc. Malocclusion for the most part makes sentiment disgrace for their facial appearance and may likewise give sentiment bashful in their society. The results of the orthodontic treatment are anticipation of tissue destruction, change in physical capacity and appearance. The malocclusion is seen to affect oral health by causing increase in the incidence of caries and also temporo-mandibular disorders. The many benefits provided by orthodontic treatment include, the prevention of damage to the tissues, improvement in aesthetics and functioning of the teeth. The readiness to pursue orthodontic treatment is influenced by various factors such as the desire to look good, improving self-esteem and improving his or her self -perception of dental appearance [3]. Females are generally more concerned about the esthetics of their teeth. There is a lack of research on the motivation of adults seeking orthodontic treatment, [4]especially in women. Both positive and negative motivations are correlated with adherence [5] treatment.Unacceptable dental appearance has been found to exert a negative impact on self-image, career advancement, and peer-group acceptance. This in turn will have an adverse influence on an individual's level of social interactions. Such esthetic concerns and adverse influence on psychosocial well-being are the primary factors for decision to start orthodontic treatment [6].Changes in morphogenesis and physiology of dentofacial structure over time and an increased concern for dental appearance and orthodontic treatment with age have become apparent rather than inadequate decisions and a provision of care during childhood and adolescence [7]. Orthodontists traditionally have considered oral health and function as the principal goals of treatment. However, recently, there has been growing acceptance of esthetics and its psychosocial impact as an important treatment benefit [8].The benefits of orthodontic treatment are prevention of tissue damage, improvement in esthetics, and physical function[9]. In orthodontic clinical practice, as well as in otherdental specialties, treatment success does not depend solely on factors such as proper diagnosis, biocompatibility, and skill of the dentist but also depends on the patient’s cooperation [10].A

compliant orthodontic patient may be described as a patient who practices good oral hygiene, follows appropriate diet, maintains appliance without breaking, keeps regular appointments, and follows instructions of the doctor. The cooperation of the patient helps in achieving treatment goal more expeditiously. The compliant patient may be identified by personal and demographic characteristics such as; age, sex, personality type, social class etc. Since sex is an imperative attribute variable, it is frequently reported in many studies [11].If the patient is not cooperative and does not follow the instructions given by the dentist may lead to unsatisfactory outcomes of the treatment [12,13]. Adolescence is the phase where orthodontic treatment is most recommended. It is also is a period in which importantphysical, social, and emotional changes appear [14]. The cooperation of the adolescent patients can be altered based on extrinsic factors (social stereotypes,gender, educational level, family) and the intrinsicfactors (personality, temperament) [15,16]. A clinician must precisely know and explain to patient how much time is needed for such adaptation to occur. Due to insufficient information on the treatment and lack of communication between the orthodontist and the patient, treatment was discontinued by the patient in many instances.Assessment of patients' expectations is central to understanding oral health needs, patient satisfaction with treatment, and ultimately the perceived overall quality of health systems. Mismatch between patient desires and the service received is related to decreased satisfaction. The overall aim of orthodontic care should be good treatment results and satisfied patients but at a reasonable cost. To reach this goal it is important that the quality of care is continuously and systematically evaluated and documented by means of professional clinical assessments and patient questionnaires or interviews. Patients with inappropriately high expectations may be dissatisfied with the optimal care and those with inappropriately low expectations may be satisfied with deficient care.Orthodontic treatment is highly technique-sensitive and timeconsuming clinical procedure. Patient's understanding in its technical aspect and compliance in appliance maintenance are important for management and successful outcome of the treatment [17]. The unwillingness of female adolescent patients with malocclusions for orthodontic treatment is studied so as to be able to educate them on the long term effects of malocclusion. Hence being able to convince them for opting to go with the orthodontic treatment.

Materials and Methods

A questionnaire based study was conducted at Chennai to determine the attitude of female adolescent patients towards orthodontic treatment. Data was collected using a structured, self-administered questionnaire which was designed after reviewing recent articles, developments and also similar questionnaires that are based on the objectives of the study. The questionnaire was divided into three parts. The first part included questions based on demographic data, the second part consisted of questions related to their knowledge on malocclusions and the final part consisted of an open question asking them to state out their reason for not undergoing orthodontic treatment. The questionnaire was distributed to 50 female adolescent patients with malocclusions. All questionnaires were anonymously collected and the data was kept confidential and not used for anything apart from the study purpose.

Results

From the survey, it was found that 54% of the adolescent females stated that malocclusion is the irregular positioning of teeth, 24% of the females thought malocclusion to be a state where there are missing teeth and 22% thought it to be a condition having broken teeth (Figure.1). 48% stated thumb sucking to be the etiology of malocclusion, 24% suggested accidents, and 22% said improper brushing habit to be the cause and 6% suggested sticky food to be the etiology (Figure.2). 46% of them stated that decayed teeth are not an outcome of malocclusion. Similarly, 28% suggested discoloured teeth are not an outcome, 20% said that food accumulation is not an outcome and 6% stated that compromised aesthetics is not an outcome of malocclusion (Figure.3). When asked on how one could correct malocclusion, 54% suggested braces, 18% suggested removing the tooth and 4% suggested surgery (Figure.4). Only 48% of them were aware that they had a malocclusion whereas, the remaining 52% were not aware (Figure.5). On asked as to why they were not correcting the malocclusion, 40% stated that it was embarrassing to wear braces, 18% stated that the treatment was too expensive, 12% stated that the treatment was that of a long duration, 8% had no interest to do the treatment, 8% stated it would painful, another 8% had concerns that food would get stuck to the braces, 4% were scared to undergo treatment and 2% stated that they received no complaints on how they looked (Figure.6).

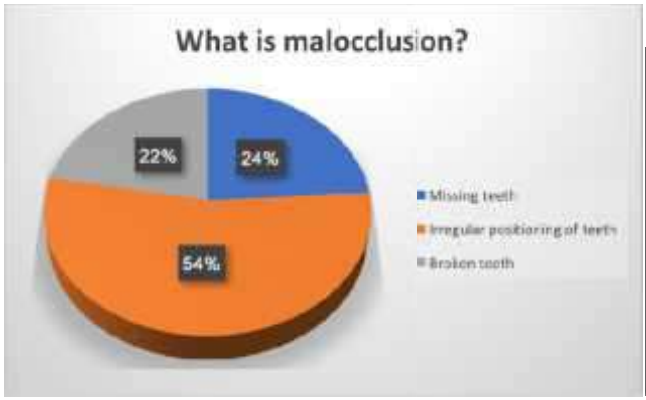


Figure.1 – The response given by the patients when asked what they knew about the term malocclusion.

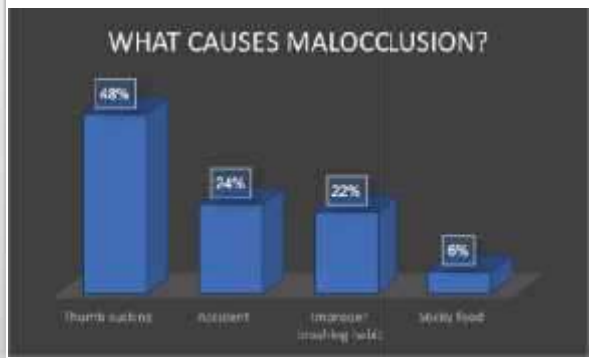


Figure.2 – Their response when asked about the etiology of malocclusion.

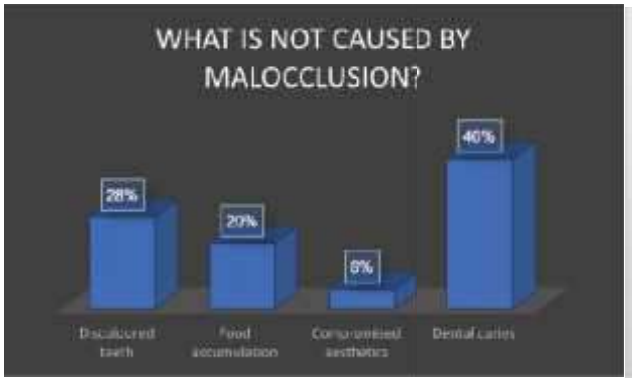


Figure.3 – Their response when asked what were not outcomes of malocclusion.

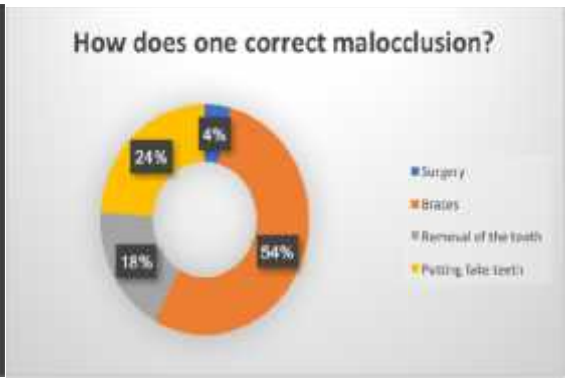


Figure.4 – Their response when asked on the methods of management of malocclusion

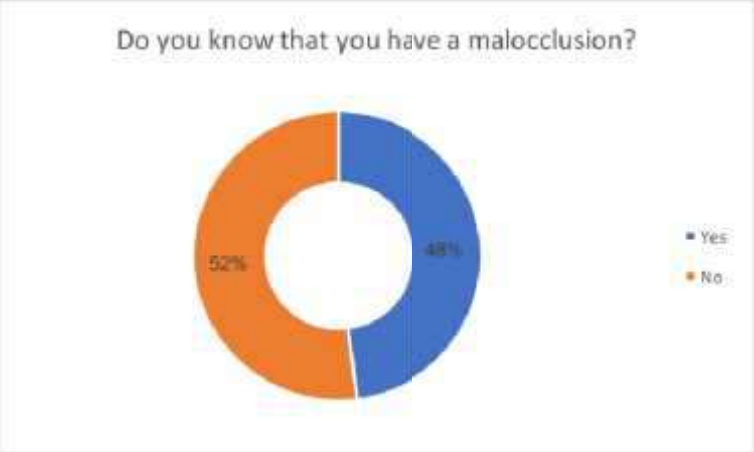


Figure.5- Their response when asked if they were aware of their malocclusions.

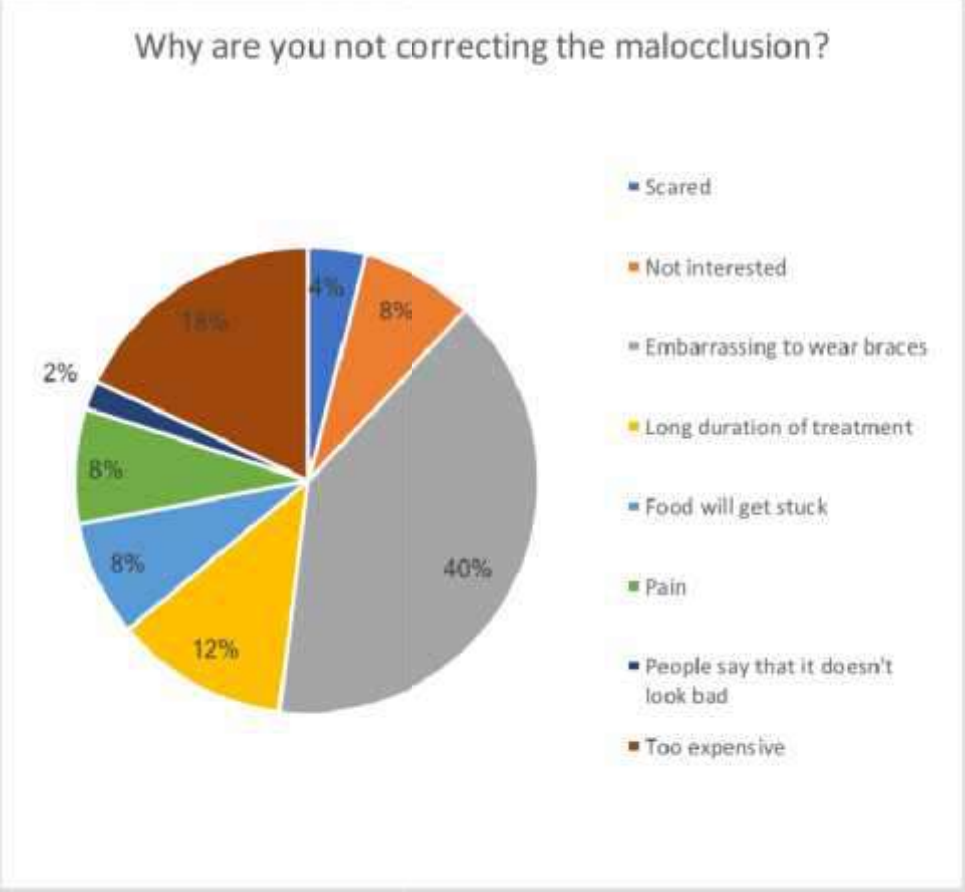


Figure.6 – Their response when asked for the reasons for not correcting their malocclusion.

Discussion

Many factors such as age, gender, opinion of dental appearance, socioeconomic status and educational status can influence the patients desire to undergo orthodontic treatment. However, it all depends on how the patients perceive orthodontics. Perception is defined as the way in which something is regarded, understood, or interpreted. In orthodontics, perception deals with the way this field of dentistry is seen by the people. Some consider the correction of malocclusion a necessity to achieve proper esthetics while few consider it as an unimportant health condition and few are concerned with problems regarding difficulty in speech, mastication, etc. In this age and era, where aesthetics play a significant role in each and every walk of life, be it at a personal or professional level, there has appears to be a massive surge in the demand for orthodontic services. Egolf et al [18] studied factorsassociated with compliance. These factors were considered as a combination of personality type, negative motives and positive motives. The present study assessed the reason for the patients not going forward with orthodontic treatment. The assessment on attitude of the patient was related to perception on treatment service, cost and also personal factors such as embarrassment. However, many orthodontists have difficulty to deal with behavioral compliance of their patients. Thus, orthodontists seem to deal with the complex issue of motivating and improving the compliance of their patients during the appointments. These involve educating the patients and/or parents about the importance of compliance. Moreover, the orthodontists need to verbally praise for compliant behavior. Treating the patients with respect, establishing a good communication, and informing the patients about the progress of treatment offered to them are rewarded as well. However, an orthodontic patient differ psychologically from each other in the perception of the way their practicing orthodontist contacts and communicates with them. Understanding the behaviors and attitudes of the patient can provide valuable information for the orthodontist prior to developing treatment plan and orthodontic treatment[19]. The study on pain experience during orthodontic treatment among Nepalese patients was analyzed by Kafle and Rajbhandari [20].Attitudes and perceptions towards dental appearance differ among populations and among individuals [21]. Various studies identified female orthodontic patients as more cooperative than males [4,5,10]. The present study showed similar finding in many parameters that female Nepalese patients possessed better understanding and perceptions towards ongoing orthodontic treatment. Siddegowda and

Rani [22] found a decent amount of knowledgeabout orthodontist and mal-positioned teeth, and relatively less awareness about the varieties of orthodontic treatment among children in Indian schools by means of an epidemiological survey. The orthodontic patients were educated on oral hygiene, food restriction, and regularity of appointments. Anorthodontist's interpersonal behavior and role in patient motivation is another aspect of successful orthodontic treatment. The orthodontist should give positive feedback and communicate with the patient on the issues of patient cooperation and their inconveniences. Orthodontists should explain about the appliance including retainers, and advice on oral hygiene, dietary control and appliance maintenance methods [23].A successful orthodontic treatment is largely dependent on the knowledge and skills of the clinician and the cooperation of patients and also that of the parents, in case of children and adolescent patients. Major considerations regarding patient cooperation are regularity in keeping appointments, compliance in wearing elastics, headgear or wearing removable appliances, refraining from chewing hard and tenacious substances that are likely to distort the arch wires, and remove bonded brackets and maintenance of oral hygiene. Laxity in following these instructions may lead not only to compromised treatment but also to slow progress of treatment, loss of chair time, increased number of visits to the orthodontist and frustration for the clinician, patient, and parents [24]. Personality traits, pain perception and attitude of the patient toward orthodontic treatment can be important factors influencing cooperation of the patient and knowledge of their interrelationship will help to gain maximum cooperation from the patient and thereby optimal treatment results.Patients' main reason for seeking orthodontic treatment is to correct dento-facial disharmony. But males have different expectations of orthodontic treatment than females. Males focus more on social well-being while females concentrate more on their improved appearance.Discomfort and pain are common during orthodontic treatment. Approximately, 90% to 95% of orthodontic patients are reported to experience pain during the course of orthodontic treatment. In orthodontics, the main cause of pain is the application of forces to induce tooth movement. Pain is also influenced by motivation, gender, and personality factors. Pain and discomfort can be one of the important discouraging factors for orthodontic treatment[25]. It is very important to understand the personality of the patient, pain perception, and attitude toward treatment so as to customize the treatment protocol to achieve optimum patient compliance and patient satisfaction. Pretreatment assessment of personality is one of the important factor for achieving patients' compliance, even though due

emphasis has not been given in the literature to personality assessment before, during and after orthodontic treatment. Objective criteria are lacking for delineation of personality, the available instruments are complex and orthodontists are not familiar with them[26]. About half the number of patient thinks that wearing the braces do not look good. About two third of the patients think that orthodontic treatment is expensive. In general, the attitude of female adolescent patients towards orthodontic treatment is moderate. It is important for practicing orthodontists to inform patients about the retainers, appliance maintenance and build inter-personal rapport with the patients. The importance of patients' perceptions regarding orthodontic treatment cannot be underestimated as it is the patients who receive treatment and need to gain satisfaction from improved esthetics andfunction.It had been hypothesized that increased experience with and availability of orthodontic service should be translated into differences in esthetics rating and perception of treatment need.The decision making process that a person undertakes when judging his/her own dental esthetic satisfaction may be broken down into several steps. First to know about the awareness about their own dentition, after this step, second their self-satisfaction, and finally their attitude toward orthodontic treatment. We may postulate that females had very good knowledge about dental esthetics compared to the males. The subjects who had malocclusion and did not report to the orthodontic clinic seems to believe that teeth do not affect their esthetic value and this appeared to be more of an ignorance that teeth does significantly affect facial appearance and lack of knowledge was the main factor that kept away from treatment[27].Regarding self-satisfaction about their teeth, the study indicates that the subjects did make fairly accurate self-evaluation of their own malocclusions. The unsatisfied subjects with their dental esthetics had a positive attitude toward orthodontic treatment. The satisfied subjects with their dental esthetics were aware of the attractiveness of their teeth[28].The practitioners shall improve the patient attitude by shortening the waiting time of patients at waiting room, adequately spend time on procedure and charge the treatment cost more reasonably.

Conclusion

The unwillingness of female adolescent patients with malocclusions for orthodontic treatment is studied so as to be able to educate them on the long term effects of malocclusion. Hence being

able to convince them for opting to go with the orthodontic treatment. Majority of the adolescent females were not aware that they had a malocclusion. Those who were aware did not want it because of the embarrassment it would cause and also because they found it very expensive. By teaching them about the merits and demerits of the treatment, one can positively motivate the patient to undergo orthodontic treatment.

Corresponding Author

Sreedevi Dharman

Senior Lecturer, Department of Oral Medicineand Radiology

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Science

162, Poonamallee High Road,Chennai 600077,Tamil Nadu,India.

E-mail: sanjamrut@gmail.com

Telephone number: +919841009003

References

1. Liu Z, McGrath C, Hägg U. The impact of malocclusion/orthodontic treatment need on the quality of life: a systematic review. The Angle Orthodontist. 2009 May;79(3):585-91.
2. Zhang M, McGrath C, Hägg U. The impact of malocclusion and its treatment on quality of life: a literature review. International Journal of Pediatric Dentistry. 2006 Nov 1;16(6):381-7.
3. Bhullar MK, Nirola A. Malocclusion Pattern In Orthodontic Patients. Indian Journal of Dental Sciences. 2012 Oct 2;4.
4. Pabari S, Moles DR, Cunningham SJ. Assessment of motivation and psychological characteristics of adult orthodontic patients. American Journal of Orthodontics and Dentofacial Orthopedics. 2011 Dec 1; 140(6):e263-72.

5. Egolf RJ, BeGole EA, Upshaw HS. Factors associated with orthodontic patient compliance with intraoral elastic and headgear wear. American Journal of Orthodontics and Dentofacial Orthopedics. 1990 Apr 1; 97(4):336-48.
6. Hamamci N, Başaran G, Uysal E. Dental Aesthetic Index scores and perception of personal dental appearance among Turkish university students. The European Journal of Orthodontics. 2009 Jan 6;31(2):168-73.
7. Urtane I, Pugaca J, Liepa A, Rogovska I. The severity of malocclusion and need for orthodontic treatment in correspondence with the age. Stomatologija. 2006; 8(2):35-8.
8. Bellot-Arcís C, Montiel-Company JM, Pinho T, Almerich-Silla JM. Relationship between perception of malocclusion and the psychological impact of dental aesthetics in university students. Journal of clinical and experimental dentistry. 2015 Feb; 7(1):e18.
9. Kamath MK, Arun AV. Perception of orthodontics. International Journal of Orofacial Research. 2017 Jan 1; 2(1):5.
10. Arnrup K, Broberg AG, Berggren U, Bodin L. Treatment outcome in subgroups of uncooperative child dental patients: an exploratory study. International journal of pediatric dentistry. 2003 Sep 1; 13(5):304-19.
11. Reichmuth M, Greene KA, Orsini MG, Cisneros GJ, King GJ, Kiyak HA. Occlusal perceptions of children seeking orthodontic treatment: impact of ethnicity and socioeconomic status. American Journal of Orthodontics and Dentofacial Orthopedics. 2005 Nov 1; 128(5):575-82.
12. Cucalon III A, Smith RJ. Relationship between compliance by adolescent orthodontic patients and performance on psychological tests. The Angle Orthodontist. 1990 Jun; 60(2):107-14.
13. Slakter MJ, Albino JE, Fox RN, Lewis EA. Reliability and stability of the orthodontic patient cooperation scale. American journal of orthodontics. 1980 Nov 1;78(5):559-63.
14. Kaliyaperumal KI. Guideline for conducting a knowledge, attitude and practice (KAP) study. AECS illumination. 2004 Jan; 4(1):7-9.
15. Bhattarai P, Shrestha RM. Comparative Study of Duration of Orthodontic Treatment among Nepalese Adolescent and Adult Patients. Orthodontic Journal of Nepal. 2013 Dec 18; 1(1):28-30.

16. Bos A, Hoogstraten J, Prahl- Andersen B. A comparison of dental health care attitudes in the Netherlands in 1985, 1995, and 2001. Community dentistry and oral epidemiology. 2003 Jun 1; 31(3):207-12.
17. Shyagali TR, Bhayya DP. Evaluation of orthodontic treatment expectations of Indian parents and their children. Pakistan Orthodontic Journal. 2014; 6(1):19-26.
18. Kreit LH, Burstone C, Delman L. Patient cooperation in orthodontic treatment. The Journal of the American College of Dentists. 1968 Oct; 35(4):327-32.
19. Gravely JF. A study of need and demand for orthodontic treatment in two contrasting National Health Service regions. British journal of orthodontics. 1990 Nov 1; 17(4):287-92.
20. Reichmuth M, Greene KA, Orsini MG, Cisneros GJ, King GJ, Kiyak HA. Occlusal perceptions of children seeking orthodontic treatment: impact of ethnicity and socioeconomic status. American Journal of Orthodontics and Dentofacial Orthopedics. 2005 Nov 1; 128(5):575-82.
21. Priyanka.S, A Survey on Awareness of Orthodontic Treatment Among Female Population Reporting to Dental Clinic, JMSCR, 2016, 4(8): 12222-24.
22. Starnbach HK, Kaplan A. Profile of an excellent orthodontic patient. The Angle Orthodontist. 1975 Apr; 45(2):141-5.
23. Doyley RA. Elder Patient-Physician Communication: Satisfaction in the Medical Encounter in One Rural and One Suburban Family Practice Clinic (Doctoral dissertation, Fielding Graduate University).
24. Ramsay DS. Patient compliance with oral hygiene regimens: A behavioral self- regulation analysis with implications for technology. International Dental Journal. 2000 Dec 1; 50(S6_Part1):304-11.
25. DiMatteo MR, McBride CA, Shugars DA, O'Neil EH. Public attitudes toward dentists: a US household survey. The Journal of the American Dental Association. 1995 Nov 1; 126(11):1563-70.
26. Phillips C, Broder HL, Bennett ME. Dentofacial disharmony: motivations for seeking treatment. The International journal of adult orthodontics and orthognathic surgery. 1997; 12(1):7.

27. Peck BM, Ubel PA, Roter DL, Goold SD, Asch DA, Mstat AS, Grambow SC, Tulsky JA. Do unmet expectations for specific tests, referrals, and new medications reduce patients' satisfaction? Journal of general internal medicine. 2004 Nov 1; 19(11):1080-7.
28. Rasool G, Nausheen A, Hassan N, Bashir S, Raza HA. Orthodontic treatment and patient's expectations. Pakistan Orthodontic Journal. 2012;4(2):44-7.

Attitude of Dental Students toward their Oral Hygiene- A Cross Sectional Study

Sahana .K[1], Gheena.S[2], L.Leelavathi[3]
Graduate Student[1], Senior Lecturer[2], Department of Oral Pathology, Senior Lecturer[3], Department of Public Health Dentistry
Saveetha Dental College,SaveethaInstitute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Oral health awareness has an important role in the general health and well being of the individual. As dental students, we have an enormous role to play in maintaining the public health .Hence it is very important to have a sound knowledge and importance of Oral hygiene measures and their importance.The aim of this study was to know the views of dental students towards their oral health and over all public oral health development.

Materials and Method: A pretested structured questionnaire was used to assess the attitude of dental students towards their oral health. The awareness about their oral health, self-evaluation and their oral hygiene practices were assessed.A sample of 100 students was evaluated and their response was recorded , tabulated and analyzed.

Results:The frequency of the dental student's brushing interval and duration has been highlighted through the study that a Majority(58 percent) of the students brush twice a day, the rest brush either only morning(39 percent) or only night(3 percent). The brushing duration has an equal proportion(50 percent). About 69 percent of the students showed concern about having bleeding gums and about 75 percent of them do a self appraisal of their teeth.

Conclusion: From the study done we can conclude that the dental students were very much concerned about the damages that were caused on their oral cavity. They were also aware of the need to maintain a proper oral hygiene and the methods to be followed for the same. A positive outcome about the knowledge of the students have been concluded from the study

Key Words:*attitude, dental students, questionnaire, oral hygiene, awareness*

Introduction

Health is a universal human need for all cultural groups. General health cannot be attained or maintained without oral health. The mouth is regarded as the mirror of the body and the gateway to good health. [1] Oral health education begins from footsteps of awareness. Today, various dental health education programs have been conducted in schools and other settings; however, these efforts will not succeed until people are not aware of the importance of oral health and positive attitude toward treatment modalities.

Oral hygiene is the method by which the tooth and its surrounding structures are clean in order to prevent any disease. The factors that influence the oral hygiene are generally the life style, culture,foodhabits,location[1]. In the upcoming generations the need for a good oral hygiene is felt and hence the dental health professionals are insisted to take adequate measures to decrease the risk of oral diseases[2,3]. Since the dental students have exposure to the dentistry world, conducting a survey would help in finding out the their attitude towards developing a better oral hygiene for themselves as well as the community. Hence, a cross sectional study was conducted to evaluate the knowledge on oral health and practices of the dental students.

Material and Method

A pre-tested structured questionnaire were given to 100 students of Saveetha Dental College and Hospitals, Chennai who were undergoing the undergraduate programme. The feedback of the students regarding the questionnaire were gathered and the data collected were entered for further evaluation. Evaluation of the data was done and the output was obtained.

Results

A cross sectional study was conducted among 100 dental students to evaluate their knowledge on oral health and practices.

From the questionnaire it is portrait that a large proportion(73 percent) of students think that it is very required to visit the dentist followed by a proportion(22 percent) of students who think it is required at times and 5 percent even think it not required.

Table 2 reveals that the reasons for the visit being regular check ups tops the list with 54 percent followed by the reason of pain occurrence with 34 percent, then comes the reason of parental force with 7 percent and the last reason of never visiting has 5 percent.

Table 3 illustrates the Self evaluation of the dental students. About 69 percent of the students show their concern of having bleeding gums, where as 31 percent of them are not concerned about having bleeding gums.

It is clear that a majority(75 percent) of students worry about discolouration of the teeth and the rest(25percent) are not worried.
About 75 percent of the students look at the mirror and do a self appraisal of their teeth and 25 percent of them do not do.

Table 4 demonstrates the various Oral hygiene practices followed by a dental student. Roughly 78 percentage of the dental students feel that they use the correct brushing technique while brushing and the rest 22 percentage of them feel they are not using the correct technique.It is shown that about 63 percentage of the students use proper toothbrush and paste.The usage of floss and mouth wash is limited to 28 percentage and 72 percentage does not use them on a regular basis.

78 percent of the students do a regular tongue cleaning where as the rest 22 percent fail to do so. The frequency of rinsing the mouth after meal is 73 percent and the rest 27 percent lack it.

The frequency of the dental student's brushing interval and duration has been highlighted through the study that a Majority(58 percent) of the students brush twice a day, the rest brush either only morning(39 percent) or only night(3 percent). The brushing duration has an equal proportion(50 percent).

Table 5 explains the Awareness of the dental students on the various aspects of the dental health. The implication of data from the above table is that 76 percent of the students are aware of the terminology of the sticky deposits

Graph 2 illustrates the frequency of the student's reason for preserving the tooth. About three fourths of thee study participants want to preserve their tooth for aesthetic appearance, 16 percent of them want to preserve it for masticatory purpose and the rest 12 percent preserve it in order to avoid false teeth.

Table 1: Visiting dentist.

		Percent
	Often	32.0
	Rare	68.0

Table 2: Reasons for visiting dentist

		Percent
	Regular check ups	54.0
	Parental force	7.0
	When pain occurs	34.0
	Never	5.0

Table 3:Distribution of study participants based on Self Evaluation of their oral hygiene

QUESTIONS\ RESPONSE	YES [IN PERCENTAGE]	NO [IN PERCENTAGE]
Worry about bleeding gums	69	31
Worry about coloured teeth	75	25
Worry about bad breathe	79	21
Self appraisal of teeth	75	25
Concerned about bad oral hygiene	80	20
Concerned about sticky deposits	70	30

Table4:Distribution of study participants based on Oral HygienePractises

QUESTIONS\ RESPONSE	YES [IN PERCENTAGE]	NO [IN PERCENTAGE]
Using correct brushing technique	78	22
Using toothbrush and paste	63	37
Using floss and mouth wash	28	72
Regular tongue cleaning	72	28
Rinse your mouth after meal	73	27

Table 5:Distribution of study participants based on Awareness on Oral Hygiene Practices

QUESTIONS\ RESPONSE	YES [IN PERCENTAGE]	NO [IN PERCENTAGE]
Terminology of sticky deposits	76	24
Consumption of Soft drinks and carbohydrates affects	50	50
Damage tooth structure due to prolonged brushing	83	17
Awareness of procedures done	81	19
Regular follow ups with dentist	63	37
Preaching about oral hygiene to others	87	13

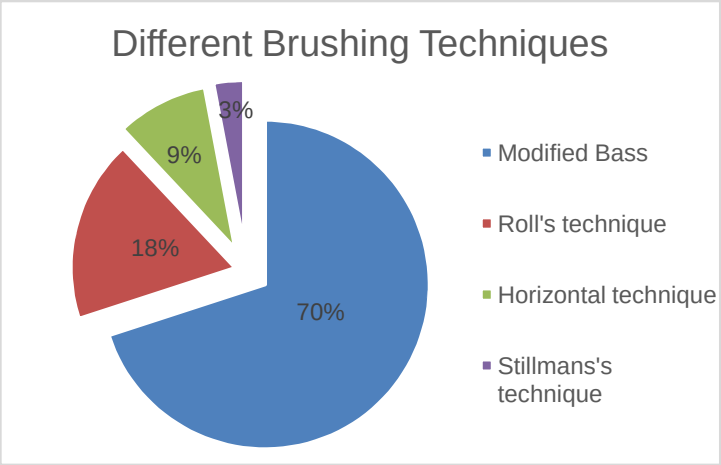
Table 6: Problems of improper oral hygiene

		Percent
	Dental caries	74.0
	Periodontal disease	13.0
	Unaesthetic appearance	9.0
	Malocclusions	4.0

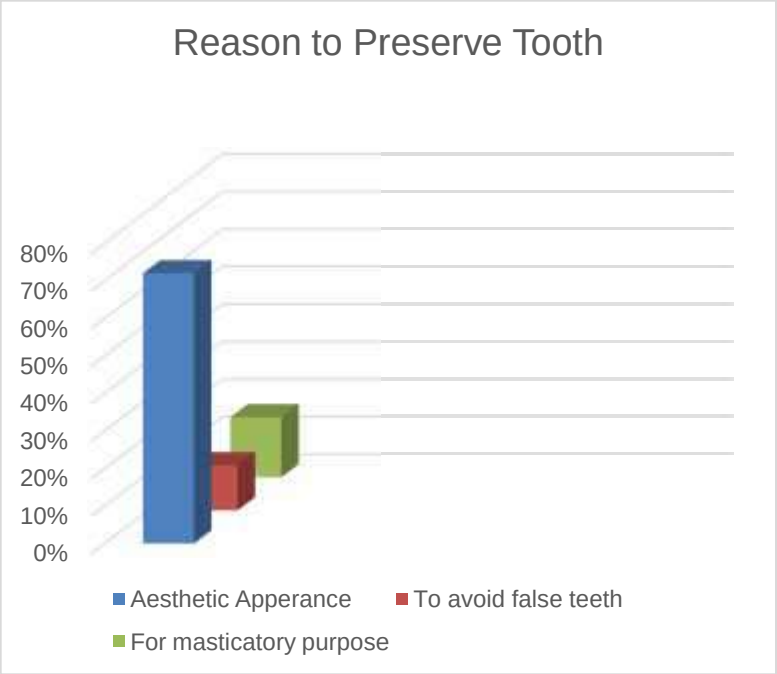
Table 7:Frequency in Changing in toothbrush

		Percent
	1-3 months	70.0
	>3 months	13.0
	When bristles wear out	15.0
	Never	2.0

Graph 1: Different brushing techniques used by the study participants



Graph 2: Reasons for preserving tooth



Discussion

From the study done, the dental students are very much concerned about the damages that are caused on the oral cavity like bleeding gums, discolouration of the teeth, presence of sticky deposits, intake of soft drinks and carbohydrates and tooth structure damage due to prolonged brushing. Evaluation of their understanding helps in knowing their self awareness and also their interest in taking care of themselves before preaching it to others.

Nearly three fourths of the study participants rarely visit the dentist and only a minority of them visit regularly thus explaining the fact that a majority of them are aware of the precautionary measures to preserve their teeth and avoid visiting the dentist.

More than half of the study participants were concerned about bleeding gums which infer that the dental students were aware of the various problems that can cause bleeding gums and are concerned about gum care.

Majority of the students did self appraisal of the teeth, which leads to a conclusion that the majority of the students are aware of the need to maintain a good oral health.We can also infer that the dental students follow the correct duration while brushing while varience is accepted and the brushing twice is also followed meticulously by them.[15]

Majorityof the students are really concerned about having a bad oral hygiene and show their concern towards this sticky deposit .Thus implying that being a dental student the concern for maintaining a good oral health and their awareness about the various problems caused due to having a bad oral hygiene is of utmost concern.

The usage of dental floss and mouth wash was limited to nearly one fourth of the students which is similar to that of a study conducted by FahmidabintiAbd Rahman et al.[9,16] From the above findings the usage of toothbrush and paste is prevalent among the dental students as it acts as mechanical and chemical removal of deposits on the oral cavity respectively.[17,18] But the need for the awareness of the usage of floss and mouth wash is felt here.

Most of the students rinsed their mouth after a meal which was on contradictory to a study by Sandhya et al where most of the study group didn't rinse their mouth after a meal.[7] This helps us arrive at a conclusion that the importance of mouth rinsing after meal is known to the students in order to keep their oral cavity free of remnants of food particles.[20]

The modified bass technique was used by a majority of the students, and hence it implies that most of the were aware of the correct brushing technique.

Being dental students they are aware of the different terminologies used in dentistry which is very essential for communicatory purpose with the fellow dentists. The frequency of students who are concerned about the intake of soft drink and carbohydrates and those who are not concerned are equalwhere in a study by Gopinath et al revels 59.4 percent of the population where concerned about the carbohydrate and sugar coated food items.[8] Weinferred from the study that the dental students are aware of the problems caused due to the consumption of soft and carbohydrates and try to avoid it but still their desires does not allow them to stay completely away from it.

A majority of the studentsbeing dentist thought that prolonged brushing damages the tooth structure, as the bristles can erode the tooth surface when used along with chemicals(tooth paste). The awareness of the dental students to the dental procedures done to them by their dentist is 81 percent similar to that of Jembulingamet al.[6] As a dental students they are

more keen on knowing the different procedures done to them and the significance of each of it.

majority of the study pollutants go for regular follow ups and that the knowledge about the need to go to the dentist for regular follow up is felt by the dental students.[13,14] The portrayal of the frequency of the students who preach about maintaining oral hygiene was also observed. Majority of the dental students preach about maintain a good oral hygiene. As a dental student they know the advantages and disadvantages of having a oral hygiene and hence we can infer that most of them preach it for the welfare of others. [11]

Knowledge about the various problems caused by improper oral hygiene is known to the dental students and the dental caries formation is the main concern of it. As a majority of the study participants think that dental caries arise due to improper oral hygiene where as the periodontal disease, unaesthetic appearance and malocclusions are few other problems caused due to improper oral hygiene.[19]

Majority(70 percent) of the students change their tooth brush every 1-3 months, where as in a similar study by Pradeep Kumar et al only 40% of the population changed their brush every 1-3 months. [10] This helps us infer that changing the toothbrush is necessary and the students are aware of it.

We can infer from the study that the students prefer aesthetic appearance over any other reason for preserving the tooth. [12,19]

Conclusion

Dental's students have widespread knowledge about the damages caused to the oral tissues due to etiopathological contributing factors like sticky food, soft drinks intake and carbohydrate and tooth structure damage due to prolonged brushing Since they have a vast knowledge about the various aspects of dentistry they were able to maintain a good oral hygiene by brushing twice a day for a duration of 1-2min with toothbrush and paste using he correct brushing technique- modified bass technique. Rinsing the mouth after meal, changing the tooth brush regularly and tongue cleaning also added up for the maintenance of a good oral hygiene along with the prevention of bad breath. This reduced their visit to dentist significantly. It is also portraited that an improper oral hygiene results in dental caries, and

hence the visiting the dentist for regular check ups is required in order to preserve their tooth for aesthetic appearance and masticatory purposes. Being a dental student the urge to know the procedures done and to go for regular follow ups during treatment plans can makes the communication between the dentist and the patients easier as there will be a easy understanding of the terminologies used by the dentist. By doing all this a dental student was also able to preach about oral hygiene to others. Thus the attitude of dental students helps in proper orientation of their oral hygiene.

Corresponding Author

L. Leelavathi

Senior Lecturer, Department of Public Health Dentistry

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162,Poonamallee High Road,Chennai 600077, Tamil Nadu, India

Mobile Number: +91 9443702882

Mailid:karleela81@gmail.com

Reference

1. Sischo L, Broder HL. Oral-Health related Quality of Life: What, Why, How, and Future Implications. J Dent Res 2011;90 (11); 1264-1270.
2. Kolawole KA, Ayeni OO, Osiatuma VI. Psychosocial impact of dental aesthetics among university undergraduates. Int. Orthod J 2012; 10: 96-109
3. Jain G, KawadkarA, Moidin T, Singhal V, Chouksey A:Assessment of Oral Hygiene Habits among the Dental Students of Bhopal, Madhya Pradesh: A Questionnaire Survey: *Journal of Orofacial Research, October-December 2013;3(4):249-252*
4. Al-Ansari J, Honkala E, Honkala S (2003) Oral health knowledge and behavior among male health sciences college students in Kuwait. *BMC Oral Health* 2003 3(1): 2-5
5. Dagli RJ, Tadakamadla S, Dhanni C, Duraiswamy P, Kulkarni S Self reported dental health attitude and behavior of dental students in India. *J. Oral Sci.* 2008; 50 (3): 276–272.

6. Jembulingamsabarathinam, dr.Geomani ,dr.deepagurunathan - Awarness and attitude about dental treatments among middle school students of chennai,Int J Pharm Bio Sci 2016 Oct ; 7(4): (B) 637 – 641
7. A. Sandhya,Dr.Dhanaraj,Dr. Sangeetha -A Survey on Mouth Rinsing Habits in Young Adult Population ,Int J of sci and Res 2017 Apr ; 6(4): 1715-1717
8. Gopinath V. Oral hygiene practices and habits among dental professionals in Chennai. Indian J Dent Res. 2010;21:195–200
9. FahmidabintiAbd Rahman-A Questionnaire Study on Oral Hygiene Among Dental Students in Chennai, India,J of dental and med sci.2016 June ;15(6): 58-64.
10. R. Pradeep Kumar,ShristiNadar-Oral Health Knowledge, Attitude, and Practice of Patients Visiting a Private Hospital in Chennai .2015; 14(6): PP 12-15.
11. Ghasemi H, Murtomaa H, Vehkalahti MM, Torabzadeh H. Determinants of oral health behaviour among Iranian dentists. Int Dent J 2007; 57:237-42.
12. Tseveenjav B, Vehkalahti M, Murtomaa H. Oral health and its determinants among Mongolian dentists. Acta OdontolScand2004 ;62:1-6.
13. Komabayashi T, Kwan SY, Hu DY, Kajiwara K, Sasahara H, Kawamura M. A comparative study of oral health attitudes and behaviour using the Hiroshima University - Dental Behavioural Inventory (HU-DBI) between dental students in Britain and China. J Oral Sci 2005;47:1-7.
14. Richards W, Ameen J. The impact of attendance patterns on oral health in a general dental practice. Br Dent J 2002;193:697-702.
15. Hobdell M, Petersen PE, Clarkson J, Johnson N. Global goals for oral health 2020. Int Dent J 2003;53:285-8.
16. Merchant A, Pitiphat W, Douglass CW, Crohin C, Joshipura K. Oral hygiene practices and periodontitis in health care professionals. J Periodontol2002;73:531-5.
17. Petersen PE. The world health report 2003: continuous improvement of oral health in the 21 st century - the approach of the WHO global oral health programme. Community Dent Oral Epidemiol 2003;31:3-23.
18. Telivuo M, Murtomaa H, Lahtinen A. Observations and concepts of the oral health consequences of tobacco usage of Finnish periodontists and dentists. J Clin Periodontol1992;19:15-8.
19. Marcus SE, Drury TF, Brown LJ, Zion GR. Tooth retention and tooth loss in the permanent dentition of adults: united states, 1988-1991. J Dent Res 1996;75:684-95.

20. Curtis D.A, Lind S.L, Setia G, Finzen F.C, Dental Students Self Assessment of Preclinical Examinations. Journal of Dental Education. March 2008;72(3):45-47

Attitude of Pedodontists towards Pedodontic Speciality Education: A Survey

Boopana .K[1], Magesh Ramakrishnan[2], Geo Mani[3]

Graduate Student[1], Senior Lecturer[2], Department of Pedodontics,
Senior Lecturer[3], Department of Pedodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: To study the attitude of pedodontist with regards to specialty education. The main objective was to correlate the attitude of pedodontists towards their specialty education. Pedodontic specialty education programs are based on university certificate and the hospital certificate.

Materials and Methods: This method of study is a questionnaire with the 5 bipolar descriptive used to indicate degree of satisfaction perceived towards inquiry areas developed from guidelines for specialty education. The major topics covered was patient education, behavior management, restorative dentistry, pulp therapy , interceptive orthodontics.

Result: The purpose of this study was to establish a database analysis of pedodontists attitude towards specialty education.

Conclusion: The survey indicated positive satisfaction levels in relation to behavior management, restorative dentistry, pulp therapy, traumatic injury and interceptive orthodontics, reduced satisfaction levels in the areas of prevention/patient education and child sedation, and significantly lower satisfaction levels for pedodontists in treatment of the special child, hospital dentistry, and conducting experimental and clinical research.

***Key Words**: Pedodontist, specialty education, Attitude, dentists, aids*

Introduction

The pedodontic specialty education programs are based on the university certificate and the hospital certificate. A basic curriculum and instruction level broadly representative of the specialty is required in each setting [1,2]. In a 1996 survey by Bentle and his coworkers, pedodontists indicated " to little" instruction during their training in orthodontics, dentistry for the handicapped , general anesthesia , premedication , research experience , treatment planning and patient education. The study concentrated on conceptions of an ideal gradual program and provided no comparisons between the program settings. The purpose of this study was to establish a data based analysis of the pedodontists attitude towards the speciality eduction and to determine how these perceptions differ by program setting [4].

Data based attitudinal surveys provide quantitative and qualitative information of performance by the determination of correlation magnitudes between variables [5]. Attitudinal surveys account for a substantial portion of education research, and have been frequently used in dental education[6,7,8]. While not useful as as direct measure of use and effect, the data can be used as a comparative basis for self-analysis and future planning[9,10,11]. The Aim of this survey is to understand the attitude of Pedodontists towards the speciality education.

Materials and Methods

A questionnaire was given to 35 pedodontists across various universities in chennai like the Saveetha Dental College and hospitals, Sri Venkateshwara Dental College and hospitals, Sri Ramachandra Dental College and hospitals, Chettinadu Dental College and hospitals, Priyadharshini Dental College and hospitals. A 5- point Liker-type attitude scale with bipolar descriptive was sued to indicate the degree of satisfaction perceived towards inquiry areas developed from guidelines for the specialty education. The major topic areas were prevention/patient education, behavior management, restorative dentistry, pulp therapy, traumatic injury, interceptive orthodontics, dentistry for the special child, sedation, hospital dentistry, and academic dentistry. The MS EXCEL was used for the statistical analysis of the data and it is represented as a bar graph.

The questionnaire that was distributed to the pedodontists is

Note: For each of the following statements, the respondent indicated the degree of satisfaction felt towards his experiences during pedodontic practice.

Key: highly satisfied -5

Mildly satisfied -4

Neutral - 3

Mildly dissatisfied – 2

Highly dissatisfied – 1

Name/Optional

1. Years of experience –
2. The ability to educate and counsel the child and parent to accept and maintain optimum oral health care through.
   a) Use of audiovisual aids
   b) Treatment plan presentation
   c) Rationale of plaque control
   d) Design of preventive program
   e) Fluoride supplementation
3. The ability to guide the behavior of the child to accept oral health care utilizing the following skils or knowledge.
   a) Voice control
   b) Hand over mouth exercise
   c) Restraints
   d) Positive reinforcement
   e) Basic child psychology
4. Mastery of restorative procedures required during the growth period from birth to adolescence.
   a) Stainless steel crowns

b) Polycarbonate crowns
c) classII alloys on deciduous teeth

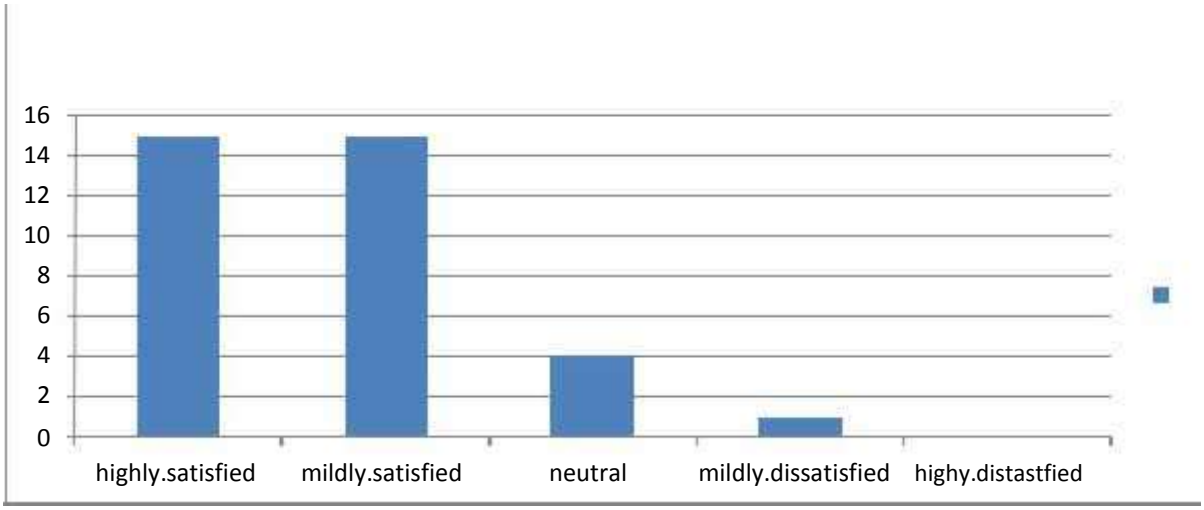
5. Ability to diagnose and treat developing occlusion utilizing interceptive procedures until final development of permanent dentition :
   a) Removable appliance
   b) Techniques of guidance of eruption
   c) Cephalometric analysis
   d) Correction of ectopic eruptions
   e) Minor tooth movement with the fixed appliance
   f) Cross bite diagnosis and correction
6. Knowledge and ability to render competent oral care for the special child with pediatric or medical considerations.
   a) Diagnosis and treatment of herpetic and aphthous ulcerations
   b) Precautions in positive cardio vascular history
   c) Hemophilia – dental approach treatment
   d) Epilepsy and dental treatment
   e) Mentally retarded patients.
7. Knowledge and competency in the utilization of pharmacologic methods of patient management in pediatric dentistry
   a) Oral sedation
   b) Biomedical aspects of narcotics
   c) Intramuscular sedation techniques
   d) Emergency drugs and techniques
   e) Intravenous sedation techniques
8. Ability and knowledge in hospital oral health care:
   a) Biomedical aspects of general anesthetics
   b) Knowledge of hospital protocol
   c) Experience in administering general anesthesia
   d) Operating room experience as dental operator
9. Knowledge and experience in academic pedodontics
   a) Ability to evaluate literature.

b) Teaching experience in didactic and clinical environment
c) Developing and conducting experimental and or clinical research
d) Working knowledge of basic pedodontic literature.

Results

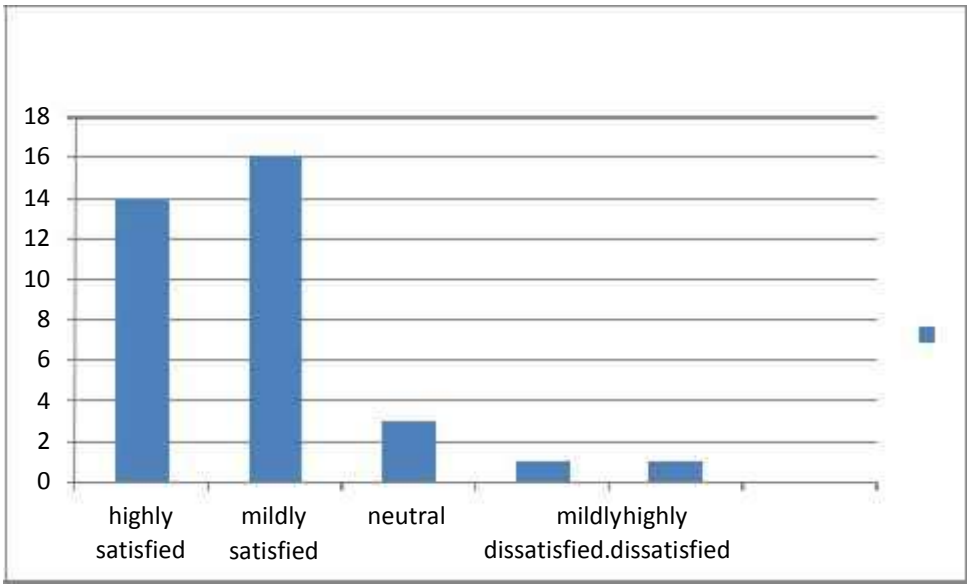
All the questions in the questionnaire were answered by the pedodontists. 86.96% of the pedodontists showed positive results towards the specialty education. The data collected is represented as a bar graph.

Table 1: Ability to educate and counsel the child and parent to accept and maintain optimum oral health care through:



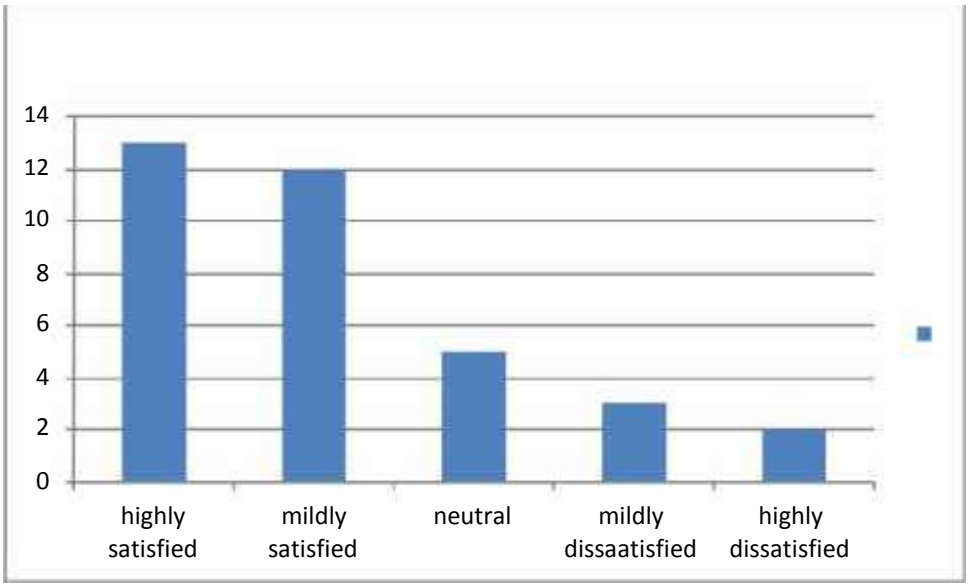
84-86% of the pedodontists were highly and mildly satisfied with the use of audiovisual aids, treatment plan presentation, rationale of plaque control, fluoride supplementation. While 22-24% of the pedodontists showed neutral response to the question and 2-4% of the pedodontists were mildly dissatisfied.

Table 2: Ability to guide behavior of the child to accept oral health care utilizing the following skills or knowledge:



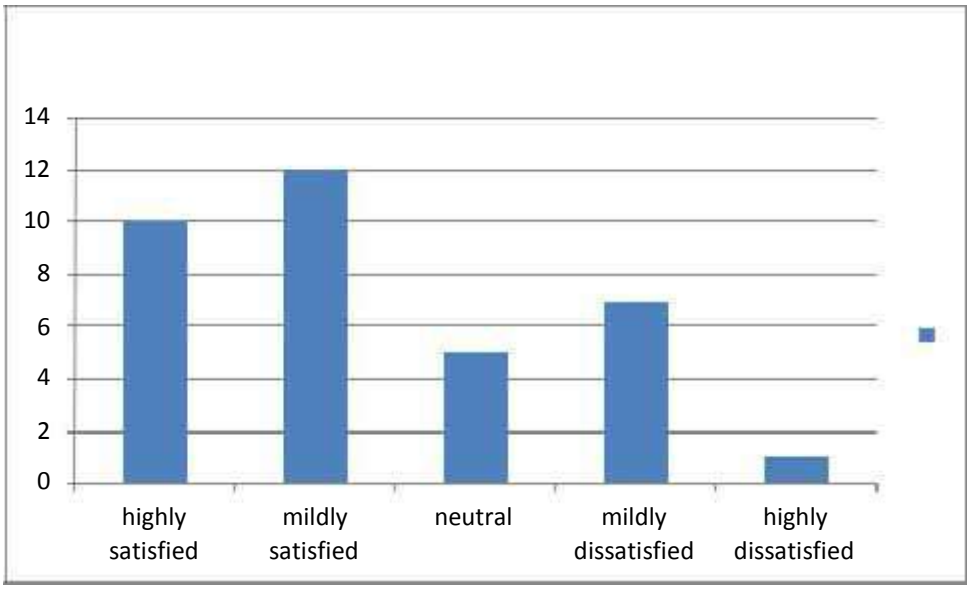
83-85% of the pedodontists showed highly satisfied and mildly satisfied response for the techniques like voice control, hand over mouth exercise, restraints, positive reinforcement, and basic child psychology. 11-15% of the pedodontists were neutral to these techniques and about 8-9% of the pedontists showed mildly and highly dissatisfied response to it.

Table 3: Mastery of the restorative procedures required during the growth period from the birth to the adolescence:



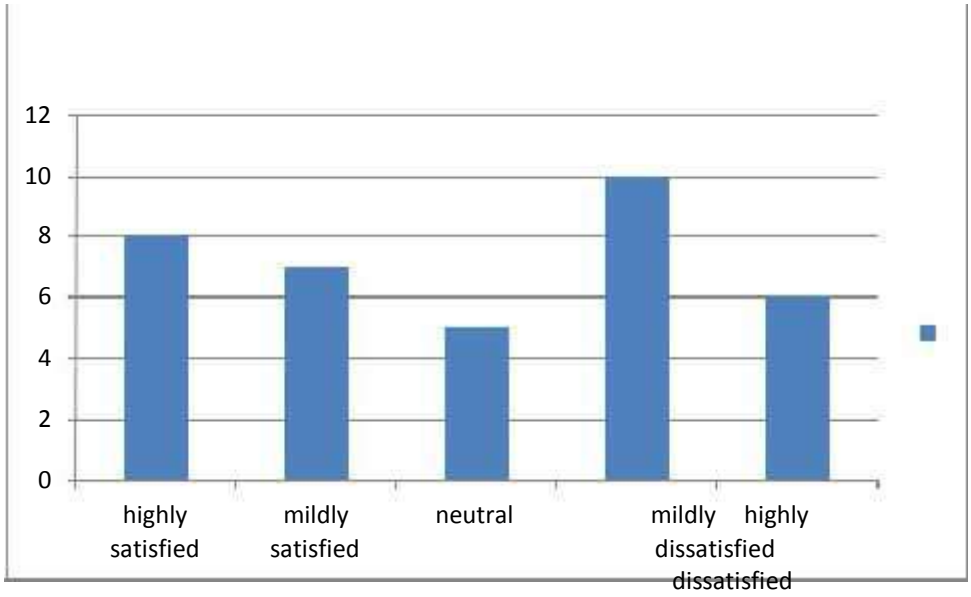
About 82-84% of the pedodontists showed positive response for the restorative procedures like stainless steel crowns , polycarbonate crowns, class II alloys on deciduous teeth, acid etch resin techniques, properties of resin materials, radiographic diagnosis , 18-22% showed mild and highly dissatisfied response to the restorative procedures .

Table 4: Ability to diagnose and treat developing and treat occlusion utilizing interceptive procedures until final development of permanent dentition.



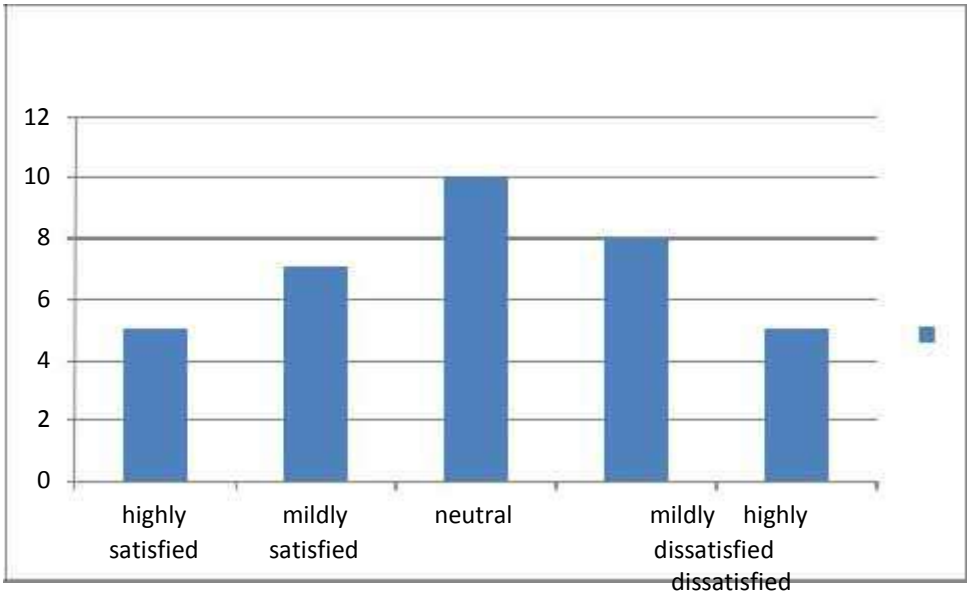
About 68-72% of the pedodontists show highly and mildly satisfied response to the interceptive procedures like removable appliance, techniques of guidance of eruption, cephalometric analysis, correction of ectopic eruptions, minor tooth movement with fixed appliance, crossbite diagnosis and correction. 29% of the pedontists showed mildly dissatisfied response to the interceptive procedures.

Table 5: Knowledge and ability to render competent oral care for the special child with pediatric or medical considerations.



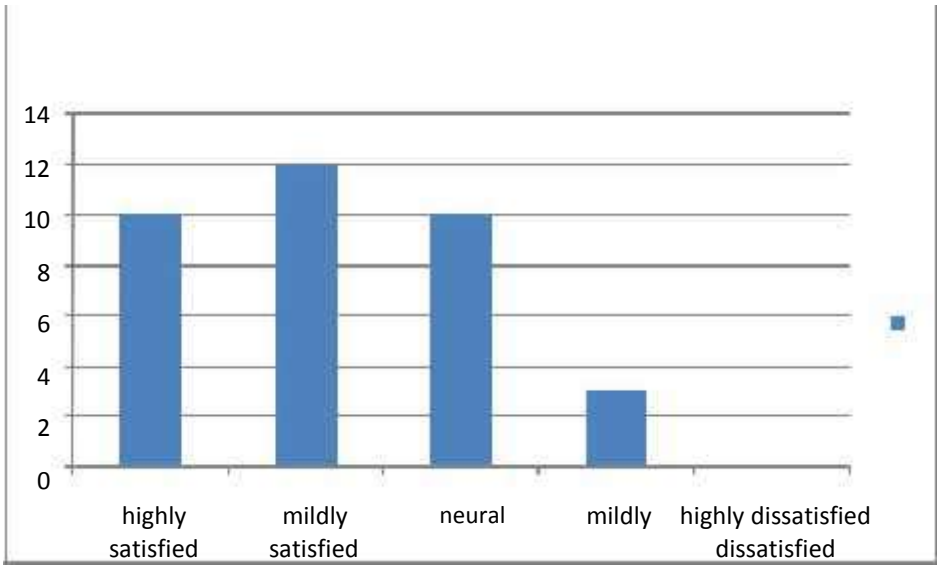
32-38% of the pedontists showed highly satisfied and mildly satisfied results in the diagnosis and treatment of herpecric and aphthous ulcerations and nearly 61%-70% of the pedodontists showed mildly dissatisfied and highly dissatisfied results in precautions in positive cardio vascular history , hemophilia- dental approach to treatment, epilepsy and dental treatment, mentally restarted patient.

Table 6: Knowledge and competency in the utilization of pharm logic methods of patient managementinpediatricdentistry.



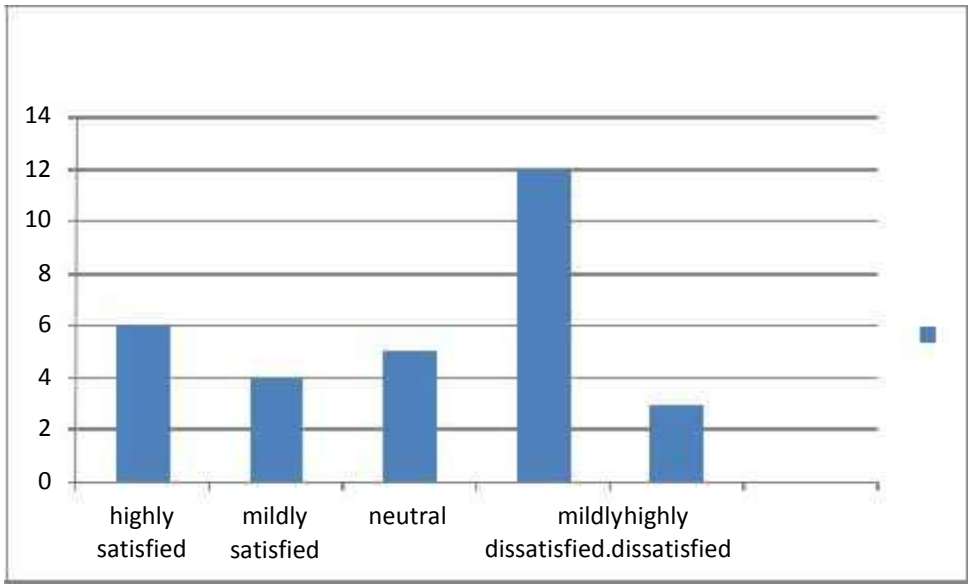
32-37% percent of the pedodontists showed mild and highly satisfied with the utilization of the pharmacological method , while nearly 56-68% of the pedodontists showed mildly dissatisfied and neutral response to methods like oral sedation, biomedical aspects of narcotics, intramuscular sedation techniques, emergency drugs and techniques, intravenous sedation techniques.

Table 7: Ability and knowledge in the hospital oral health care:



83-86% of the pedodontists showed highly and mildly satisfied reports in the oral health care aspects like biomedical aspects of general anaeatetics, knowledge of hospital protocol and experience in admistering the general anesthesia. While remaining 20-22% of the pedodontists show neutral response and mild dissatisfied response to the oral health care.

Table 8: Knowledge and experience in academic pedodontics



Nearly 35-40% of the pedodontists showed highly satisfied and mildly satisfied in the academic pedodontics in topics like ability to evaluate literature, working knowledge of basic pedodontic literature. And nearly 65-71% of the pedodontists are mildly and highly dissatisfied with the teaching experience in the didactic and the clinical environment and mainly in conducting experimental and or clinical research.

Discussion

The indicated differences in degree of satisfaction may not reflect differences between the programs, but may be due to the varied aims and expectations of the respondents choosing the different programs. Attitudinal surveys are affected by respondents' feelings, beliefs, knowledge, and predisposition towards a subject. In a similar manner, the non-respondents may represent a population different in attitude towards their specialty education. In consideration of these facts,

survey methodology incorporates various design measures to minimize bias and increase the usefulness of the obtained data. Specific measures incorporated in this study were multiple inquiry items, Likert-type attitudinal scale, population control, response anonymity, transmittal letter, and significance levels. The survey indicates pedodontists are generally satisfied with their educational experiences related to behavior management, restorative dentistry, pulp therapy, traumatic injury and interceptive orthodontics. The reduced satisfaction level in prevention/patient education suggests a general desire for increased experience in these areas during specialty education. The survey also indicates less satisfaction in the areas of academic pedodontics like the developing and conducting experimental and or clinical research, and also in special child and any kind of pediatric considerations like the epilepsy in dental treatment, mentally retarded patients.

The reduced satisfaction levels related to sedation indicate pedodontists in general desire increased experience in child sedation during their training. University degree programs may require particular improvement in providing such experiences due to significantly over satisfaction levels than other groups[14,16,17]. Combined with similar reduced satisfaction levels concerning treatment of the special child and hospital dentistry, particular consideration by university degree programs may be warranted towards treatment of the more unusual or difficult-to-manage child. Academically related topics such as literature review and teaching experience appear to receive satis- factory emphasis in the various program types. However, the perceived attitudes of the pedodontists indicate hospital and university certificate programs might benefit from increased research experience[21]

Conclusion

A survey was designed to evaluate the degree of satisfaction perceived by pedodontists towards their specialty education. The data-based inquiry items were correlated to the various program settings. The respondents indicated*:*

1. Positive satisfaction levels in relation to behavior management, restorative dentistry, pulp therapy, traumatic injury, and interceptive orthodontics.
2. Reduced satisfaction levels in the areas of prevention/patient education and child sedation.

3. Significantly lower satisfaction levels for pedodontists in treatment of the special child, hospital dentistry, and conducting experimental and clinical research.

Although no definitive and specific recommendations can be derived from the data, the findings may serve as a reference base for future studies and individual program self-analysis. Prospective students of pedodontics may find the information useful in evaluating and matching pedodontic programs with individual.

Corresponding Author

Geo Mani

Senior Lecturer, Department of Pedodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai -600 077, Tamil Nadu, India.

Email Id: geosdch@gmail.com,

Telephone Number: 7358433246

Reference

1. Knebel E, Greiner AC, editors. Health professions education: A bridge to quality. National Academies Press; 2003 Aug 1.

2. Bell RA, McTigue DJ. Attitude Toward Pedodontic Specialty Education: A Survey of Pedodontists. Pediatr Dent. 1979 Dec;1(4):215-0.

3. Bennett IC, Mink JR, Hall DS. Attitudes of pedodontists to their graduate education. Journal of dentistry for children. 1967 Mar;34(2):87.

4. Borg WR, Gall MD. Educational Research: An Introduction. New York: David McKay Company. Inc. 1971;19:71.

5. Vernon DT. Reviews: Oppenheim, AN Questionnaire Design and Attitude Measurement. New York: Basic Books, 1966. Pp. iv+ 351. $5.95. American Educational Research Journal. 1968 Jan;5(1):128-30.

6. Well RA, McTigue DJ. Attitude Toward Pedodontic Specialty Education: A Survey of Pedodontists. Pediatr Dent. 1979 Dec;1(4):215-0.

7. Walker JD. Favorable and unfavorable behaviors of the dental faculty as evaluated by dental students. Journal of dental education. 1971 Oct;35(10):625-31.

8. Ripa LW. Hospital-related activities in pedodontic specialty training program. Journal of dental education. 1973 Mar;37(3):9-12.

9. Nammalwar RB, Rangeeth P. Knowledge and attitude of pediatricians and Family Physicians in Chennai on Pediatric Dentistry: A survey. Dental research journal. 2012 Sep;9(5):561.

10. Hashim Nainar SM, Straffon LH. Targeting of the year one dental visit for United States children. International journal of paediatric dentistry. 2003 Jul 1;13(4):258-63.

11. Walker JD, Till MJ, Parkins FM. Survey of pedodontic graduate student interests: 1975-1976. ASDC journal of dentistry for children. 1976;43(2):114-8.

12. Massler M. The effective clinical teacher. Journal of dental education. 1977 Oct 1;41(10):613-7.

13. Tamisiea PE, McKercher TC, Mattson JS, Blankenau RJ, Trumm GA. Emphasis and quality of a dental education: a survey of graduates. Journal of dental education. 1977 Sep 1;41(9):575-7.

14. Emling RC, Fritz LS. Dental student and faculty perceptions of teacher characteristics. Journal of dental education. 1978 Feb;42(2):78-82.

15. Bell RA, McTigue DJ. Attitude Toward Pedodontic Specialty Education: A Survey of Pedodontists. Pediatr Dent. 1979 Dec;1(4):215-0.

16. Tittle CR, Hill RJ. Attitude measurement and prediction of behavior: An evaluation of conditions and measurement techniques. Sociometry. 1967 Jun 1:199-213.

17. Chilton NW. Design and analysis in dental and oral research. Greenwood; 1982.

18. Walker JD. Favorable and unfavorable behaviors of the dental faculty as evaluated by dental students. Journal of dental education. 1971 Oct;35(10):625-31.

19. Reddy KV, Sharma A. Prevalence of oral health status in visually impaired children. Journal of Indian Society of Pedodontics and Preventive Dentistry. 2011 Jan 1;29(1):25.

20. Saravanan S, Madivanan I, Subashini B, Felix JW. Prevalence pattern of dental caries in the primary dentition among school children. Indian Journal of Dental Research. 2005 Oct 1;16(4):140.

21. Gurunathan D, Somasundaram S, Kumar SA. Casein phosphopeptide-amorphous calcium phosphate: a remineralizing agent of enamel. Australian dental journal. 2012 Dec 1;57(4):404-8.

Attitudes of General Dental Practitioners towards Biopsy Procedures - A Survey

N. Vidulasri[1], Gheena[2],Caroline Jacob[3],
Graduate Student[1], Reader[2], Department of Oral Pathology[2],
Senior Lecturer[3], Department of Periodontics[3],
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: Biopsies are one among the diagnostic procedures in detection of malignancy, but are not conventionally performed in dental practice due to the fear of medico-legal complications, unfamiliarity with the technique and the misconception of it being a predominantly specialist procedure. This survey was therefore aimed to explore the knowledge, attitudes and practices of general dental practitioners towards biopsy procedures.

Materials and Methods:A self-designed questionnaire was administered to 50 dentists comprising of private practitioners and dental surgeons working in public and private institutions. The first part of the questionnaire included the demographic details of the dental practitioner while the second part explored the knowledge, attitude and practices towards oral tissue biopsies.

Results: All general dental practitioners (GDPs) (100%) believed it was important to perform biopsies for the diagnosis of oral lesions. 82% maintained that it should be done for all premalignant, malignant and cystic lesions, while 18% suggested that it should be performed only in premalignant and malignant lesions,26% performed the biopsy on their own. Regarding the knowledge of various biopsy methods, 78% were aware of all biopsy techniques. Reasons quoted for not performing biopsy were the lack of experience (38%), lack of confidence (12%) and inadequate patient cooperation(12%). 96% dentists felt the need to update their knowledge regarding oral lesions and biopsy procedures with the preferable use of information.

Conclusion: The GDPs enrolled in this study were adequately aware of oral screening and biopsy procedures but felt reluctant to perform them, which suggests that dental education programmes are needed for GDPs in oral pre-cancer/cancer detection as well as screening and diagnostic procedures.

Key Words:*Tissue specimen, oral lesions, histopathology, differential diagnosis*

Introduction

Biopsy technique is a diagnostic procedure which involves removal of tissue from affected area in the living organism for microscopic examination and providing appropriate diagnosis.(1)The diagnoses of many of the oral lesions require additional investigative procedures, out of which biopsies plays an important role.Biopsy and subsequent tissue examination helps in diagnosing the histological characteristics, level of differentiation, and the extent of the suspected lesion.Although dental practitioners may arrive at the clinical diagnosis of a lesion with various diagnostic tools, it is conventionally confirmed by histopathological examination.(2) Oral biopsies are indicated in:

- white lesions(leukoplakia, lichen planus, leukodema)
- red lesions (erythroplakia, atrophic lichen planus)
- vesiculobullous lesions (pemphigus, pemphigoid),
- soft tissue lesions (fibroma, mucocele),
- lesions on gingiva (pyogenic granuloma, gingival enlargement), periapical cyst(3)

Dentists are the first to encounter such changes in the oral cavity, they should take the responsibility to counsel, diagnose and effectively treat the condition in its initial stages.(4) Dental professionals should detect and recognize oral lesions and inform the patient accordingly so as to provide early diagnosis and treatment. Though certain patients are not convinced with the biopsy procedures for some reasons, it should be worth convincing the patient by informing the benefits of an early diagnosis of an lesion.General dentalpractitioners (GDP) must therefore be well-versed in performingsimple oral biopsies to diagnose oral lesions, preservingtissue specimens and generatingthe subsequent report.(5,6)Despite the benefits of performing a biopsy, it is observedthatthepractice of oral biopsy is not as widespread in dental practice predominantly due to the unfamiliarity of the procedure and its documentation.(7) Early identification of oral cancerous lesions reduces rates of morbidity, mortality and mutilation, increases the quality of life and lowers treatment costs(8). Incidence of oral cancer is rising in most countries, with squamous cell carcinoma (SCC) accounting for 95% of oral cancers, and it is associated with avoidable etiological risk factors.(9,10)Awareness, clinical findings and the experience of dentist form the basis for successful judgment and diagnosis of a particular condition(11).Oral lesions may be overlooked due to lack of awareness of the disease or the presence of subclinical changes in a clinically innocuous oral mucosa(12).However, in spite of the wide range of information

available from routine oral biopsy and histopathological examination, biopsy procedures remain unpopular among GDPs.Therefore, theaim of the study was to explore the knowledge, attitudes and practices of general dental practitioners towards biopsy procedures.

Materials and Methods

A self-designedquestionnaire was administered to a convenient sample size of 50 dentists comprising of private practitioners and dental surgeons working in public and private institutions. The first part of the questionnaire included demographic details such as the age, sex and professional qualifications of the GDP while the second part explored the knowledge, attitudes and practices towards the diagnosis of oral lesions and performance of oral biopsies. The questionnaire comprising of total 21 questions, was distributed among the practitioners.
The questions were devised to obtain information on:

- Awareness about importance of biopsies in oral lesions
- knowledge and practical skills in diagnosing lesions, knowledge about biopsy of oral lesions
- Knowledge of tissue handling and preservation of the samples
- Reasons for not performing the procedures
- Proximity to an Oral Pathologist

Results

Among the 50 general dental practitioners, 23 males and 27 females participated in the survey. All GDP(100%) believed it wasimportant to perform biopsies for the diagnosis of oral lesions.76% (n=38) of GDP did not perform biopsies in their clinical practice except for the remaining 24% (n=12) as seen in Fig.1.

On asked about the question regarding lesions that required biopsy, 82% maintained that it should be done for all premalignant, malignant and cystic lesions, while 18% suggested that it should be performedonlyin premalignant and malignant lesions.(Fig. 2)On asked about what they do for lesions requiring biopsy,48% (n=24) of GDPemployed a specialist to perform the biopsy, while 24% (n=12) referred their patients to a higher centre. The remaining 26%(n=13) performed the biopsy on their own (2%)claimed that they referred their patients to super speciality centers and 24% said that they would either call a specialist or refer them to a higher center based on the condition of the lesion.(Fig. 3)

Regardingthequestion about their knowledge of various biopsy methods as in Fig 4,

78%(n=39) were aware of allbiopsy techniques. However, only 14%(n=7) were awareof excisional biopsy, 6% were aware of only incisional biopsiesand 2% were only aware of fine needle aspiration.

The common reasons quoted for not performing biopsy was that 38% said it was due to the lack of experience, 12% of GDPs said it was due to lack of confidence to interpret the results for the particular lesion and 12% reported that it was due to inadequate patient cooperation for the biopsy procedures. The questionnaire also sought the opinion regarding the preservation of the specimen after removal of tissue, of which 62%(n=31) believed that preservation in formalin was the most ideal technique while 38%(n=19) agreed that the tissue sample could be preserved both in saline and formalin as seen in Fig 6.

Majority of GDPs (96%)dentists felt the need to update their knowledge regarding oral lesions and biopsy procedureswith the preferable use ofinformation via journals, the internet and workshops which is seen in Fig. 6

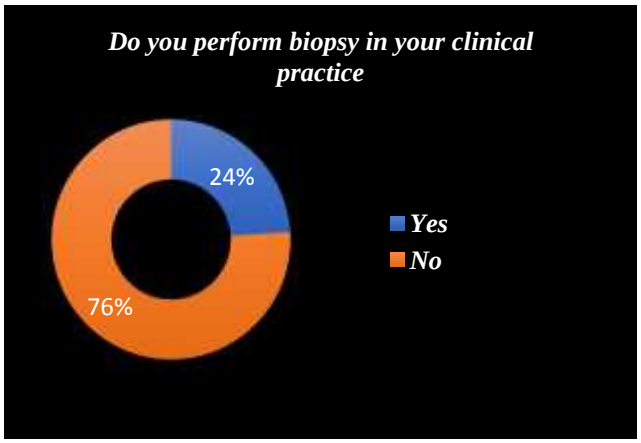


Figure 1: Percentage of dentists who perform biopsies.

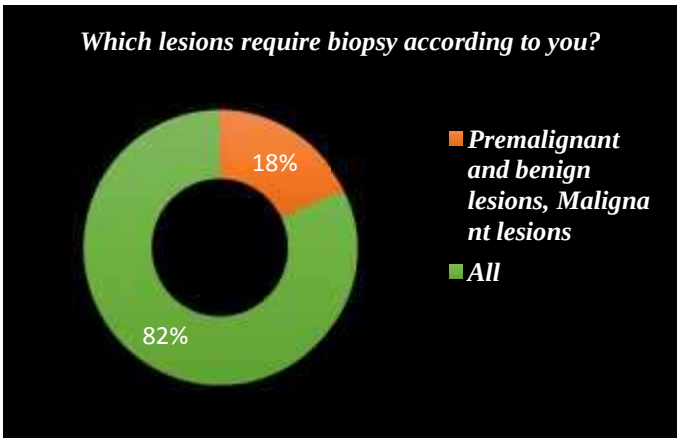


Figure 2:Percentage of Lesions that require biopsy.

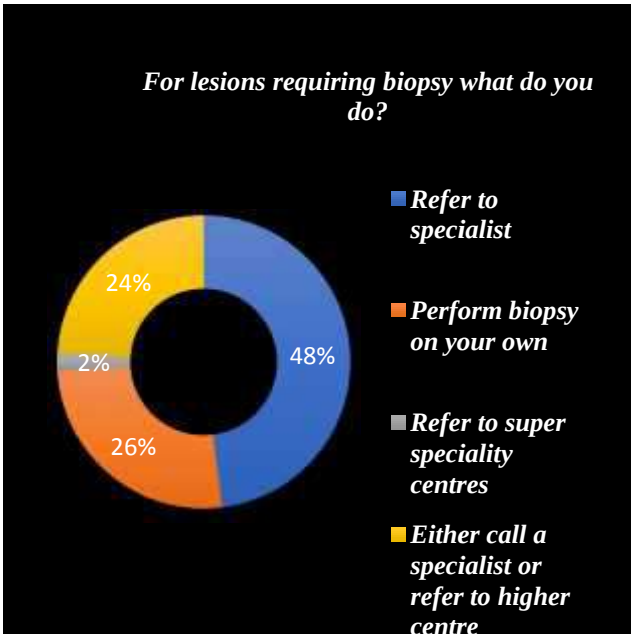


Figure 3:Percentage of dentists who prefer to perform biopsy or when referred to specialist

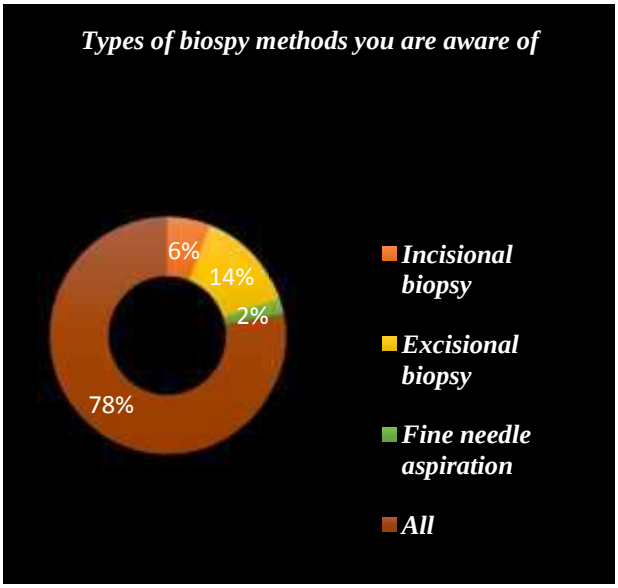


Figure 4: Percentage of awareness of various biopsy techniques

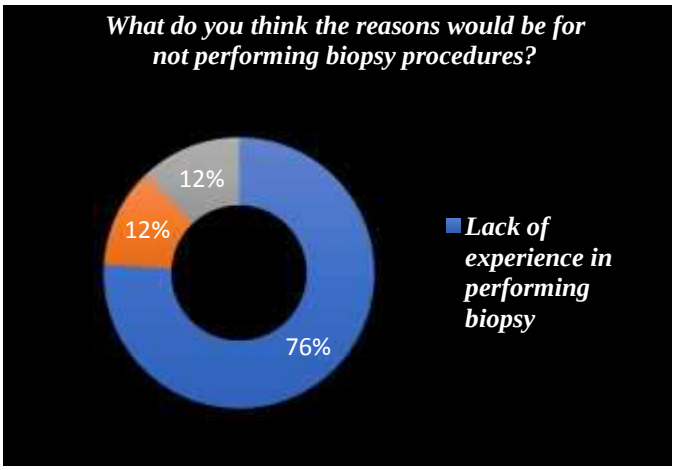


Figure 5: Representation of reasons for avoiding biopsy procedures

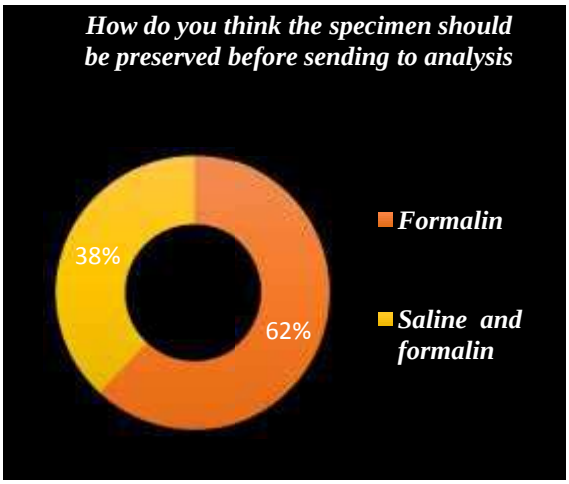


Figure 6: Representation of biopsy sample preservation

Discussion

Biopsies play a prime significance in diagnosing oral lesions (4) and there have been conflicting suppositions concerning if GDPs can perform biopsies. Certain GDP think that performing incisional and excisional biopsiesfor suspicious lesions will help in early and accurate diagnosis (4-6)while others contend that those suspicious lesions should have to be instantly referred.(5,6)Although most dentists prefer to refer biopsy cases to a specialist or a

referral centre, most believed that routine biopsies are well within the scope of a GDP as this would provide direct access to prompt management.Most GDPs who took part in a study (91.4%) believed that biopsy is a vital surgical procedure for an accurate diagnosis with commonly observed lesions that include cysts, benign and premalignant lesions, and malignant tumors. In addition, lesions in the oral cavity are much more accessible compared with lesions in other sites.In a study (4), 97% of students agreed that family history plays an important role in oral cancer and thus the importance of performing a biopsy was unanimously agreed upon by all dentists in order to arrive at a conclusive diagnosis of various oral lesions.Boyle (5) suggested thatthe degree of qualification had little to do with the ability to perform a biopsy. In this survey, 26% respondents performed biopsies on their own which was higher than those reported by Cowan et al. (7) who observed about 12% in Northern Ireland and 15% by Diamanti et al. (8) in Manchester. A study by Warnakulasuriya and Johnson (9) found that 21% of dental specialists in United Kingdom and Seoane et al. (10) revealed that 24.5 % GDPs perform biopsies in Northwest Spain. While in Norway, Berge (11) found that 56% of dental specialists endeavored to perform a biopsy.In the current study 48% of GDPseither called a specialist or referred the patient to a higher centre. This was lesser than those reported by Wan and Savage (12) in Brisbane, where it was seen that 76.2% of GDP referred their biopsy cases to a specialist. This could be due to a few variables like the dread of medico-legal complexities, newness to the biopsy strategy, absence of confidence in individual analytic abilities, misguided judgment that suggests the need for expert opinion or that the GDP may not be prepared to advise the patient that he has disease. 78% of GDP's however, were familiar about excisional, incisional and FNAC types of biopsies. The rest were aware of only one or other of the biopsy techniques. This clarifies the requirement for the GDP to be more compatible with biopsy strategies and their signs and contraindications. This would help them to settle on the sort of biopsy required in singular cases (13,14). The most commonly used fixatives were formalin, glutaraldehyde, and alcohol.(15) Astudy by Murgodstated that after the removal and prior to sending for analysis,(53.73%) practitioners, rightly knew that it had to be sent in formalin; while (29.85%) believed that it could be stored in saline and 11.94%believed it could be preserved in alcohol (16).In this study,62% believed that the tissue sample should be preserved in formalin while 38% stated that either saline or formalin could be used for preservation.This is an important aspect in biopsy procurement forclinicians as tissues that are not safeguarded legitimately produce artefacts which hamper diagnosis.(17-19).Histopathological

examination of 967 biopsy specimens revealed that, 43% of clinical diagnosis made by dental surgeons were incorrectin a study by Franklin and Jones (17) who estimated that 85% of dentists in their region did not send biopsies for histological analysis. The rationale for this could be that the excisional biopsy of lesions they consider to be clinically apparent such asmucoceles, fibromas or periapical granulomas may only be considered for treatment. Thus, altered tissue after removal from the oral cavity should be sent for histopathological examinationto arrive at a final diagnosis.(18)In doubtful conditions the patient should be referred to a specialist with expertise in the diagnosis and management of oral lesions.In a study it was observed that 89% dentists did not think histopathologic analysis was required for all biopsied lesions and also stated that 48.09% of GDPs do not examine mucosal lesions on routine basis ignoring the fact that early detection has better prognosis for patient.(19)Incipient lesions are easily detected in the oral cavity because of accessibility of oral cavity for examination and detection. Both the patient and the professional are associated with causes underlying a delay in definitive diagnosis.(20) In many cases patients are unaware of the presence of early asymptomatic lesion or they just ignore the condition by taking self medication.

In a study it was found that 91.4% of dentists appreciated the diagnostic importance of oral biopsy while only (19.8%) indicated that they perform an oral biopsy in their daily practice. (21).This aspect was also noticed by Wan and Savage (22), where 58.1% of GDPs did not feel competent to undertake any biopsies mainly due to lack of experience and practical skills, Diamanti (23) reported 25% of GDP’s said they did not feel confident to perform biopsies while Greenwood et al. (24) found that only 21% of GDPs were ready in carrying out biopsy techniques.

Anurada (25) suggested that 90% of GDP’s felt the need to update their knowledge regarding various pathological lesions and biopsy procedures. In certain studies it was stated that when GDPs were asked how they intended to update their knowledge, 59% of them preferred to attend workshops or CDE programmes, 22% of them by attending conferences, 10% of them through internet and 9% of them through journals (26-28). However in our study, 96% ofdentists felt the need to update their skill-set regarding oral lesions and biopsy procedures, which they believed could be enhanced byaids such as journals, online content, conferences and workshops. From several studies it was concluded that there is a need for including clinical abilities workshops when instructing on oral biopsy techniques, as it is a

supplementary but essential educational resource and supervised clinical procedure to be performed on real patients. (29,30)

Conclusion

With the exponential growth of dental science, dentists need to update their practices according to the best available scientific evidences. It is suggested that the frequent use of biopsy in dental practice will reduce the number of successful lawsuits brought for delay or failure to diagnose.The role of general dental practitioners and histopathologist in the evaluation of tumour specimens has become complex in the last 10- 20years and will continue to develop in the decade ahead, thus proper diagnostic aids should serve the purpose of diagnosis such conditions.Dental practitioners are not well-informed about the diagnostic importance and need for biopsy procedures.There may be a fear factor working in the minds of dentists about losing the patient who has been presented with a chief complaint unrelated to his mucosal lesion, if in case a biopsy procedure is advised or performed, or even if the patient is referred to a specialist for managing such a lesion.Knowledge about recent methodology in diagnosis, treatment and precaution measures in oral cancer is very essential for improving the quality of life in patients with oral cancer .In order to help GDPs detect suspicious oral lesions, continuing dental education programs pertaining to the practical aspects of oral cancer should be conducted.

Corresponding Author

Caroline Jacob,

Senior Lecturer, Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai600 077, Tamil Nadu,India.

E-mail: caroline_jacob@ymail.com

Telephone Number: +91 9840035702

References

1. Murgod V, Angadi PV, Hallikerimath S, Kale AD, Hebbal M, Attitudes of general dental practitioners towards biopsy procedures.JClinExp Dent. 2011;3(5):418-23.
2. Kumar MPS, AK Harshini.Knowledge and awareness about oral cancer among

undergraduate dental students. Asian J Pharm Clin Res 2016; 9(4):167-67.

3. López-Jornet P, Camacho-Alonso F, Molina-Miñano F. Knowledge and attitudes about oral cancer among dentists in Spain. J EvalClinPract. 2010; 16 (1):129–133.
4. Mota-Ramírez A, Silvestre FJ, Simó JM. Oral biopsy in dental practice. Med Oral Patol Oral Cir Bucal. 2007; 12(7):504-10.
5. Boyle PE. Who should take the biopsy? Oral Surg Oral Med Oral Pathol. 1955; 8 (2):118-122.
6. Porter SR, Scully C. Early detection of oral cancer in the practice.Br Dent J.1998;185 (2):72-3.
7. Cowan CG, Gregg TA, Kee F. Prevention and detection of oralcancer: the views of primary care dentists in Northern Ireland. Br Dent J. 1995;179 (9): 338-42.
8. Shubhapoorani E,Brundha MT. Preservative and Fixative Methods of Brain Biopsy-Review, J. Pharm. Sci. & Res. 2015, 7(6), 336-338.
9. Kumaraswamy KL, Vidhya M, Rao PK, Mukunda A. Oral biopsy: Oral pathologist's perspective. J Cancer Res Ther 2012;8 (2):192-8.
10. Shabihkhani M., Lucey G.M., Wei B., Mareninov S., Lou J.L, Vinters V., Singer E.J., Cloughesy T.F., Yong W.H.. The Procurement, Storage, and Quality Assurance of Frozen Blood and Tissue Biospecimens in Pathology, Biorepository, and Biobank Setting. Journal of Clinical Biochemistry 2014;47(4): 258-266.
11. Warnakulasuriya KA, Johnson NW. Dentists and oral cancer prevention in the UK: opinions, attitudes and practices to screening for mucosal lesions and to counselling patients on tobacco and alcohol use: baseline data from 1991. Oral Dis. 1999; 5 (1): 10-4.
12. Devi M, Vijayalakshmi D, Dhivya K, Vezhavendha N. A study on awareness of early detection and screening of potentially malignant oral disorders and oral cancer. J NTR Univ Health Sci 2017;6 (1):35-8.
13. Seoane J, Valera-Centelles PI, Ramírez JR, Cameselle-Teijeiro J, Romero MA. Artifacts in oral incisional biopsies in general dental practice: a pathology audit. Oral Dis. 2004; 10 (2):113-7
14. Berge TI. Oral surgery in Norwegian general dental practice-a survey. Extent, scope, referrals, emergencies, and medically compromised patients. Acta Odontol Scand. 1992; 50 (1):7-16.

15. Wan A, Savage NW. Biopsy and diagnostic histopathology in dental practice in Brisbane: usage patterns and perception of usefulness. Aust Dent J. 2010; 55 (2) : 162-9.
16. Diamanti N, Duxbury AJ, Ariyaratnam S, Macfarlane TV. Attitudes to biopsy procedures in general dental practice. Br Dent J.2002; 192 (10): 588-92.
17. Kondori I, Mottin RW, Laskin DM. Accuracy of dentists in the clinicaldiagnosis of oral lesions. Quintessence Int 2011;42 (7):575-7.
18. Franklin CD, Jones AV. A survey of oral and maxillofacial pathology specimens submitted by general dental practitioners over a 30-year period. Br Dent J 2006; 200 (8): 447-450.
19. Avon SL, Klieb HB. Oral soft-tissue biopsy: An overview. J Can DentAssoc 2012;78:75
20. Kaur R, Sircar K, Singh S, Rastogi V. Noninvasive Diagnostic Aids in Te Screening of Oral Cancer – A Review. JOHS2012;3(1):9-13.
21. Bataineh AB, HammadHM,Darweesh IA. Attitude toward oral biopsy among general dental practitioners: Awareness and practice.J Orofac Sci 2015;7(1): 19-26
22. Baykul T, Yilmaz HH, Aydin U, Aydin MA, Aksoy M, Yildirim D. Early diagnosis of oral cancer. J Int Med Res 2010;38(3):737-49.
23. Scully C. Oral cancer aetiopathogenesis; Past, present and future aspects. Med Oral Patol Oral Cir Bucal 2011;16(3):306-11.
24. Radoi L, Paget-Bailly S, Guida F, Cyr D, Menvielle G, Schmaus A. Family history of cancer, personal history of medical conditions and risk of oral cavity cancer in France. BMC Cancer 2013;13(1):560.
25. Ramani P, Thomas G, Ahmed S.Use of Rt- PCR in Detecting Disseminated Cancer Cells after Incisional Biopsy among Oral Squamous Cell Carcinoma Patients, J Cancer Res Ther 2005 ;1(2):92-7.
26. Anandani C, Metgud R, Ramesh G, Singh K.Awareness of General Dental Practitioners about Oral Screening and Biopsy Procedures in Udaipur, India. Oral Health Prev Dent. 2015;13(6):523-30.
27. Sunil A, Mohan A, Mathew J, Mukunda A, Nair A.Attitudes of General Dental Practitioners toward Biopsy Procedures, Oral and Maxillofacial Pathology Journal, 2017;8(1):9-15

28. Nazar SA,Umamaheswari TN.Awareness Of Early Detection And Prevention Of Oral Cancer among Dentists - A Review. IOSR-JDMS 2014, 13(1): 10-12
29. Anuja N, Sherlin HJ, Anandan S, Mani NJ, Malathi N. Subclinical changes of oral mucosa in Hansen's disease - a histopathological and immunohistochemical study. Biology and Medicine, 2011, 3(5): 31-42
30. Seoane J,Varela-Centelles P, Esparza-Gómez G, Cerero-Lapiedra R, Diz P. Simulation for training in oral cancer biopsy: A surgical model and feedback from GDPs. Med Oral Patol Oral Cir Bucal. 2013;18 (2):246-50.

Awareness about Dental Implants among Patients Attending Dental Treatment

Sameer Mohideen Gani[1], Marian Anand Bennis[2], Aravind Sivakumar[3]

Graduate Student[1],Senior Lecturer[2], Department of Prosthodontics, Senior Lecturer[3], Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Since the introduction of implants by Brånemark in the 1960s, oral implants have become a reliable treatment option for the replacement of missing teeth. The physical and chemical properties of implant materials are well-reported and documented factors that influence the clinical outcome and the prognosis of implant therapy. These properties include the microstructure of the implant, its surface composition and characteristics, as well as design factor. An ideal implant material should be biocompatible, with adequate toughness, strength, corrosion, wear and fracture resistance. The design principles of the implant should be compatible with the physical properties of the material. Materials used for the fabrication of dental implants can be categorized according to their chemical composition or the biological responses they elicit when implanted. From a chemical point of view, dental implants may be made from metals, ceramics or polymers.The objective of this study is to understand the awareness about dental implants among patients attending dental treatment.

Materials and Methods: This is a survey based study. This cross sectional survey has been conducted among patients visiting the outpatient department of Saveetha Dental College and Hospitals. A questionnaire was designed, regarding pattern of awareness about dental implants for the patients to assess the knowledge of the patients who are attending dental treatment. This questionnaire was distributed among 250 patients. The data was collected, tabulated and analyzed.

Result: Out of 100%, 59.2% of patients think its necessary to replace the missing teeth, 20.4% think if replacement is needed if space is available and other 20.4% think there is no need to go for an replacement.46.4% people think only rich people should go for an implant, while other 33.6% think its suitable for anyone who has a missing teeth. Out of 250 patients 65 patients (26%) are very confident about the success of an implant, 94 patients (37.6%) patients are confident about the success rate. Another (25.2%) 63 patients are not sure about the success of an implant, while 28 patients (11.2%) are very doubtful about the success rates.

Conclusion: Majority of patients had a thought that implants are teeth replacement options for the rich and not middle class individuals whereas 33.6% of patients thought that implants can be placed for any individual with a missing tooth and sufficient bone density and does not concern the economic status of the individual.

Most of the patients were willing and eager to know more about implants as teeth replacement options whereas 34.8% of patients did not show any significant interest.

Key Words: *Dental implants, Zirconium, Overdentures, Phonetics, rehabilitation.*

Introduction

The aim of dentist is to restore the basic functions of the teeth and oral cavity. Dental implant is an artificial root that is surgically inserted into the jawbone to support a single tooth replacement, fixed partial, complete denture or maxillofacial prosthesis.Modern dentistry aims to restore the patient's normal function, aesthetics, speech and health of the patient attending dental treatment. Dental implantology has transformed into an emerging field in dentistry. It has become increasingly important [1],[2] as majority of patients treated with implant-supported prosthesis have reported improvement in their quality of life, assurance, self-confidence [3] including psychological benefits and moreover conservation of the tooth structure adjacent to the teeth to be replaced.[4] Due to its high success rates and predictability, its clinical implication is increasing.

Implant supported prosthesis has proven advantages like increased masticatory efficiency, maintenance of the bone, improved function, phonetics, aesthetics, etc. The interest in

aesthetically flawless teeth, and also in a closely natural substitute such as an implant supported overdenture has improved.Previously dental implants were used for the treatment of edentulous patients which are primarily associated with improved denture retention, stability, functional efficiency, and quality of life. But currently for the past decade, dental implants are widely accepted as a prosthetic treatment of completely or partially edentulous patients.

Implant treatment is an increasingly popular treatment option with a high success rate. Recently, it has become the focus of the patients' interest hence for dentist, it is vital to assess their level of knowledge with regards to dental implants and whether their perception of dental implants does in fact reflect reality in order to guide patients who do not have the education or background knowledge to make an informed decision between implant supported dentures and removable dentures.[7],[8],[9],[10] Overall aspiration for improved oral health related quality of life has become a truth after the arrival of dental implants. Replacement of missing teeth with implant supported prosthesis has been accepted and rated as a positive experience by patients who have undergone implant treatment.[9],[11] Many studies have been done in different parts of the world with regards to the awareness of dental implants as a treatment option. An overwhelming majority of patients with severely compromised local host bone can be offered implant-supported rehabilitation with a very good prognosis and improved esthetics, phonetics and function.[2],[9],[12],[13],[14],[15],[16],[17] Thus, the aim of the study was to assess the awareness of the patients regarding implant-retained prosthesis as an option for tooth replacement and the knowledge about tooth replacement as a whole including source of information and attitude towards it.

Materials and Methods

This is a survey based study. This cross sectional survey has been conducted among patients visiting the outpatient department of Saveetha Dental College and Hospitals. A questionnaire was designed with questions about the need for replacement of a missing tooth and their willingness to accept the implants as a choice of replacement. The questionnaire was distributed among 250 patients. The data was collected, tabulated and analyzed.

Result

This survey was done among patients attending the outpatient department of Saveetha dental college. Out of the 250 members who attended this survey 131 9(52.4%)were male and 119(47.6%) were female. 50.4% of patient falls under the age group of 20-25 years, 31.2% of patient falls under 18-20 years and 18.4% comes under 25-30 years.

Table 1: Mode of Awareness

Education	**Total (%)**
Upto high school	**19.2%**
Uneducated	**52.8/%**
Masters	**11.6%**
Upto university or above	**16.4%**

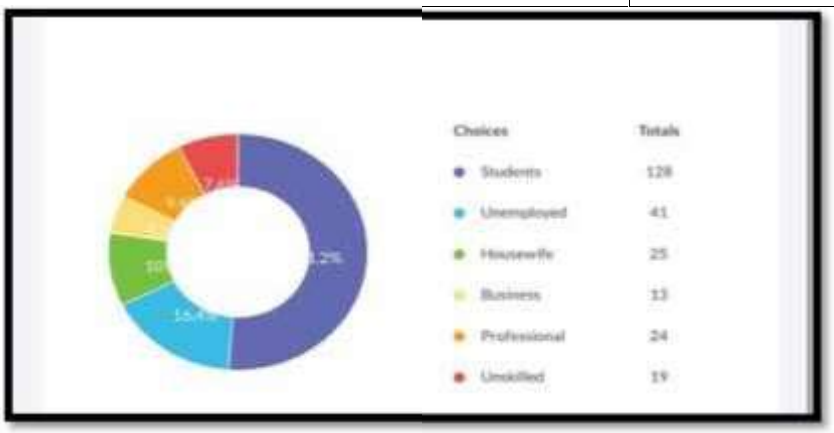


Figure 1: Commonly Asked People

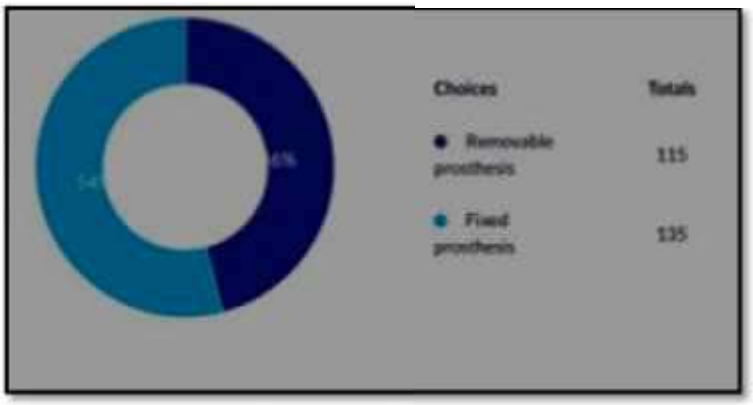


Figure 2: Patient's Preference

Out of 100%, 59.2% of patients think its neceesary to replace the missing teeth, 20.4% think if replacement is needed if space is available and other 20.4% think there is no need to go for anreplacemnet.

Table 2: Mode of Replacement

Methods of Replacement	**Total %**
Crown	28.6%
Bridge	17.2%
Complete denture	24.4%
Removable denture	29.8%

54% patients are comfortable with fixed prosthesis and the remaining 46% are comfortable with removable prosthesis. Out of 250 patients, 167 patients (66.8%) are aware of the term implant and the remaining 83 patients(33.2%) are not aware of the term implant. 40.8% of patients know about implants through dentists 26.4% through friends, 18.4% through books and 14.4% through newspaper. 48% people think its a good method of replacement, while 34.4% are not sure and 17.6% says its not at all a good method of replacement. Dentists have informed about the implants to about 46.4% of the patients while 43.46% patients have not been informed. When asked to all the patients personally whether they are ready to get an implant placed for their missing tooth/teeth,44.4% said yes, 40.8% were doubtful and 14.8% not at all.

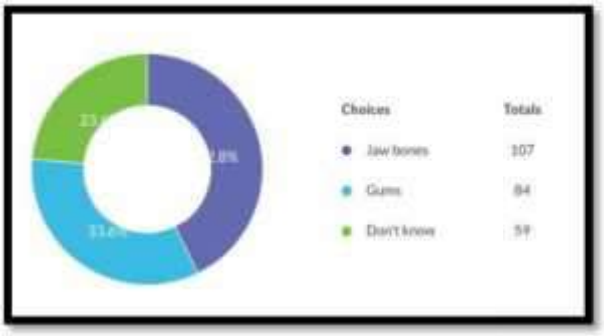
Table 3: Advantages of Implant

Advantages of Implant	Total %
Fixed replacement	21.3%
Looks good	24.4%
Good functioning capacity	23.6%
All of the above	30.7%

Talking about the disadvantages 38% said it was time consuming, 28.4% said they have to undergo surgery, 23.6% said it was costly.

46.4% people think only rich people should go for an implant, while other 33.6% think its suitable for anyone who has a missing teeth. Out of 250 patients 65 patients (26%) are very confident about the success of an implant, 94 patients (37.6%) patients are confident about the success rate. Another (25.2%) 63 patients are not sure about the success of an implant, while 28 patients (11.2%) are very doubtful about the success rates.

45.2% of patients are willing to know more about dental implants and the remaining 34.8% are not willing to know.



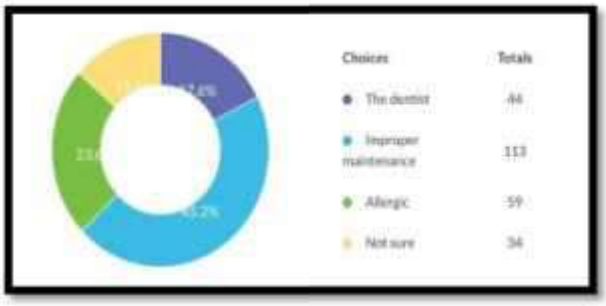


Figure 3: Site of placement Figure 4: Failure of implant

Discussion

Tooth loss may be traumatic and upsetting, and it is regarded as a serious life event that requires significant social and psychological readjustment. The nonacceptance of edentulousness and the individual's feelings about dentures, which have been the traditional way of replacing missing teeth, are important for the acceptance of new dentures.Traditionally, missing teeth are replaced by removable partial dentures, fixed partial dentures (bridges) and complete dentures in cases of complete edentulousness. The need to replace lost teeth with a near-natural successor has encouraged rapid research and advancement in the field of dental implants, especially in advanced economies.

Dental implant is an artificial root that is surgically inserted into the jawbone to support a single tooth replacement (crown), fixed partial or complete denture or maxillofacial prosthesis.It is an ideal option for people with good oral health who have lost a tooth or teeth due to injuries, periodontal diseases, failure of endodontics, etc. It is also used for the treatment of edentulousness and is associated with improved denture retention, stability and functional efficiency and, thus, improving the quality of life of the patient.

This survey was aimed at assessing the awareness about dental implants as a tooth replacement option among patients seeking dental treatment. A total of 250 patients participated in the above survey among which 128 of them were students, 41 patients were unemployed or retired elderly people, 25 were homemakers, 13 business persons, 24 professional workers and 19 were skilled workers. It is seen that majority of patients seeking dental treatment for replacement of missing teeth were students ranging from 18-24 years of age. This can be attributed to the young age and unaesthetic appearance due to loss of teeth.

When the patients were asked about the necessity of replacing missing teeth, a high proportion of patients (59.2%) thought it was very important to replace missing teeth as teeth play an important role in the overall well being of an individual as they take part in mastication, speech and other important functions. 20.4% patients answered that teeth need to be replaced only if they cause difficulty in food intake and if sufficient space is available. An equal proportion of patients also think that tooth replacement is totally unnecessary. The latter mostly belonged in the old age/ elderly category of people.

Majority of patients were comfortable with fixed prosthesis for replacement of their missing teeth while 40% of patients preferred removable dentures as they thought removable dentures to be cost efficient compared to fixed prosthesis.

167 patients were aware about implants as a replacement option for their missing teeth. However 83 patients were not aware about the term implant. Most of the patients who were aware about implants had gained their knowledge about the same through their dentist or through friends or newspaper advertisements. Most of the patients who knew about implants through their dentists were ready to get an implant placed whereas few patients were apprehensive. 14% of patients did not prefer implants due to their cost and technique sensitivity.

Majority of patients had a thought that implants are teeth replacement options for the rich and not middle class individuals whereas 33.6% of patients thought that implants can be placed for any individual with a missing tooth and sufficient bone density and does not concern the economic status of the individual.

Most of the patients were willing and eager to know more about implants as teeth replacement options whereas 34.8% of patients did not show any significant interest.

Corresponding Author

Aravind Sivakumar,

Senior Lecturer,Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai- 600077,Tamil Nadu, India.

Telephone Number: +91 8220552400

Email: aravind.sivakumar@gmail.com

Reference

1. Pommer B, Zechner W, Watzak G, Ulm C, Watzek G, Tepper G. Progress and trends in patients' mindset on dental implants. I: level of information, sources of information and need for patient information. Clinical oral implants research. 2011;22(2):223-9.
2. Lindh T, Gunne J, Tillberg A, Molin M. A meta-analysis of implants in partial edentulism. Clin Oral Impl Res, 1998;9:80-90.
3. Aglietta M, Siciliano VI, Zwahlen M, Brägger U, Pjetursson BE, Lang NP, Salvi GE. A systematic review of the survival and complication rates of implant supported fixed dental prostheses with cantilever extensions after an observation period of at least 5 years. Clinical oral implants research. 2009;20(5):441-51.
4. Den Hartog L, Huddleston Slater JJ, Vissink A, Meijer HJ, Raghoebar GM. Treatment outcome of immediate, early and conventional single-tooth implants in the aesthetic zone: a systematic review to survival, bone level, soft-tissue, aesthetics and patient satisfaction. Journal of clinical periodontology. 2008 ;35(12):1073-86.
5. Sonoyama W, Kuboki T, Okamoto S, Suzuki H, Arakawa H, Kanyama M, Yatani H, Yamashita A. Quality of life assessment in patients with implant-supported and resin-bonded fixed prosthesis for bounded edentulous spaces. Clinical oral implants research. 2002 ;13(4):359-64.
6. Narby B, Bagewitz IC, Söderfeldt B. Factors explaining desire for dental implant therapy: analysis of the results from a longitudinal study. International Journal of Prosthodontics. 2011;24(5).
7. Narby B, Kronström M, Söderfeldt B, Palmqvist S. Changes in attitudes toward desire for implant treatment: a longitudinal study of a middle-aged and older Swedish population. International Journal of Prosthodontics. 2008;21(6):205-246.
8. Brunski JB. In vivo bone response to biomechanical loading at the bone/dental-implant interface. Advances in dental research. 1999 ;13(1):99-119
9. Chowdhary R, Mankani N, Chandraker NK. Awareness of dental implants as a treatment choice in urban Indian populations. International Journal of Oral & Maxillofacial Implants. 2010;25(2):290-92.

10. Zimmer CM, Zimmer WM, Williams J, Liesener J. Public awareness and acceptance of dental implants. International Journal of Oral & Maxillofacial Implants. 1992 ;7(2)-117-120.
11. Bergendal T, Engquist B. Implant-supported overdentures: a longitudinal prospective study. International Journal of Oral & Maxillofacial Implants. 1998;13(2):223-229.
12. Friberg B, Gröndahl K, Lekholm U, Brånemark PI. Long-term follow-up of severely atrophic edentulous mandibles reconstructed with short Branemark implants. Clinical implant dentistry and related research. 2000;2(4):184-9.
13. Adell R, Eriksson B, Lekholm U, Branemark PI, Jemt TA. Long term follow-up study of osseointegrated implants in the treatment of totally edentulous jaws. Int J Oral Maxillofac Implants,1990;5:347-59.
14. Steenberghe DV, Quirynen M, Naert I, Maffei G, Jacobs R. Marginal bone loss around implants retaining hinging mandibular overdentures, at 4-, 8-and 12-years follow-up. Journal of clinical periodontology. 2001;28(7):628-33.
15. Satpathy A, Porwal A, Bhattacharya A, Sahu PK. Patient awareness, acceptance and perceived cost of dental Implants as a treatment modality for replacement of missing teeth: A survey in Bhubaneswar and Cuttack. International Journal of Public Health Dentistry. 2011;2(1):1-7
16. Lambrecht JT, Cardone E, Kühl S. Status report on dental implantology in Switzerland in 2006. A cross-sectional survey. Eur J Oral Implantol 2010;3:71-4.

Oral Potentially Malignant Disorders among Dental Patients

Priyadharshni S[1], Vivek Narayan[2], E. Roja[3]
Graduate Student[1], Professor[2], Department of Oral Medicine and Radiology,Reader[3], Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Most PMDs are asymptomatic, and the main aim of treatment is to prevent and/or to detect cancer development early. Etiological factors can be identified and patients can be warned of the potential of malignancy. Abstinence from tobacco and alcohol, even after many years of use, significantly reduces the risk of developing cancer. The knowledge of susceptibility to cancer among the general population must be imparted by awareness programs and publications in media. Oral PMDs may be considered a blessing in disguise as they can help in early diagnosis and prevention of oral malignancies which can decrease the burden of cancer prevalence, especially in the youth and working class of the society.
Result: According to this survey, awareness of oral potential malignant disorders was low among patients. It was found that television, internet had played a significant role in patients who were aware of oral precancerous conditions. The aim of this survey is to assess the awareness about oral potentially malignant disorders among dental patients.

Materials and Method: In order to assess the awareness about oral potentially malignant disorders(PMDs), a self -interviewed questionnaire was used to collect information from 100 patients of different age groupsattending the dental hospital, in Saveetha Dental College,Chennai. The questionnaire was designed and included relevant questions to ascertain information on awareness, and sources of information aboutoral precancerous condition.

Result: A total of 100 questionnaires were distributed. The study population consistedof 41female and 59 male patients of different age group.42% of the individuals out of 100 were aware of potentially malignant disorders(PMDs) whereas, 58% did not know about it.

Conclusion: According to this survey, awareness of oral potential malignant disorders was low among patients.

Key Words: *Pre cancer, awareness, lesions, mucosa,patients.*

Introduction

Potentially malignant disorders (PMD) of oral cavity were classified as 'lesions' and 'conditions' by WHO in 1978(1). A precancerous lesion is a morphologically altered tissue in which oral cancer is more likely to occur that its apparently normal counterpart whereas a precancerous condition is a generalized state associated with a significantly increased risk of cancer. However, World Health Organization decided to use the term "potentially malignant disorders (PMD)" to convey that not all disorders described under this term may transform into cancer rather this is a family of morphological alterations among which some may have an increased potential for malignant transformation. These lesions are not only site specific predictors of malignancy but also indicate an increased risk of future malignancies elsewhere in oral mucosa(2).

In the past decades little progress has been made in defining oral leukoplakia. In 1978, the World Health Organisation de ned leukoplakia as "a white patch or plaque that cannot be characterized clinically or pathologically as any other disease" (3). In that communication it was noted, that the term leukoplakia was unrelated to the absence or presence of epithelial dysplasia. In 2005, the World Health Organisation defined leukoplakia as "a white plaque of questionable risk having excluded (other) known diseases or disorders that carry no increased risk for cancer". Both the 1978 and the 2005 definition are worded in a somewhat negative way. Besides, when accepting the view that lichen planus is a potentially malignant disorder, then this disease actually falls within the 2005 WHO definition of leukoplakia.

The prevalence of leukoplakia for all ages is approximately one per cent, with an increasing prevalence in adults. The male-female ratio varies in different parts of the world. Smoking is the most common etiologic factor. Nevertheless, leukoplakia may occur in non-smokers as well. Cessation of smoking habits may result in regression or even disappearance of the leukoplakia in a matter of a few months.The prevalence of oral lichen planus is in general accepted to be approximately 1 per cent. This chronic disorder mainly affects middle-aged

people. The etiopathogenesis is still poorly understood. There is no effective treatment and there are no preventive measures either(4).

When the incidence of oral cancer is set at 5 per 100.000 population per year, then an annual risk of malignant transformation in oral leukoplakia patients of 2% is a four hundred times increased risk. Prevalence of erythroplakia are only available from studies in South- and South East Asia and are as low as 0.02%(5). The annual malignant transformation rate is actually unknown but is much higher than in leukoplakia. There are numerous reported parameters that allegedly predict future malignant transformation of oral leukoplakia. These parameters include previously diagnosed cancer in the head and neck region, older age, female gender, absence of smoking habits, duration of the leukoplakia, clinical subtype (homogeneous versus non-homogeneous), large seize, and oral subsite such as borders of the tongue and floor of the mouth. The use of toluidine blue staining may help to identify high-risk leukoplakias with poor outcome. Other predicting factors include the presence of C. albicans, the presence and severity of epithelial dysplasia, and, in addition, numerous molecular markers, such as aberrant expression of p16INK4a and Ki-67, chromosome instability, and loss of heterozygosity at 9p and mutated TP53(6). Some of the predicting factors mentioned above carry a certain degree of subjectivity. For instance, it may difficult to objectively the clinical homogeneous sub- type. Some clinicians use this adjective only in case of thin , smooth and homogeneously white lesions, while others may apply this adjective also in homogeneously white and homogeneously verrucous lesions. The histopathological assessment of the presence of epithelial dysplasia and the degree of dysplasia is another source of substantial subjectivity(7).

Material and Method

In order to assess the awareness about oral potentially malignant disorders(PMDs), a self interviewed questionnaire was used to collect information from 100 patients of different age groups (convenience sampling) attending the dental hospital, in Saveetha Dental College,Chennai. The questionnaire was designed and included relevant questions to ascertain information on awareness, and sources of information about oral precancerous condition.

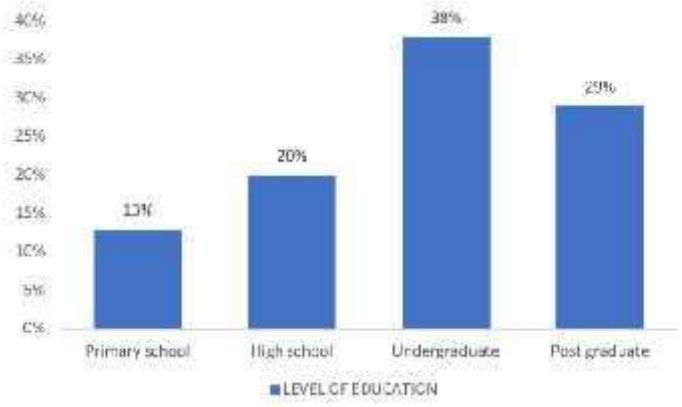
Results

A total of 100 questionnaires were distributed. The study population consistedof 41femaleand 59 male patients of different age group(Tab.1). In terms of education, most of the respondents are undergraduate(38%) , 29% are post graduate individuals(Tab.2),althoughalmost all the individuals had some form of education.

Table 1: Different age group participated in the survery.

SEX	NUMBERS
Male	59
Female	41
Total	100

Table 2: Level of Education of people participated in the survey.



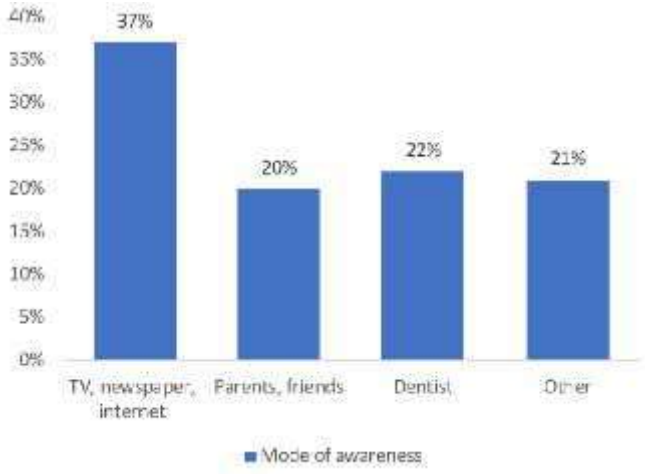
The participants were asked if they had any adverse habits, 43% of the individuals agreed that they have adverse habits which includes smoking, tobacco, pan chewing and 57% of the respondents did not have any adverse habits. 42% of the individuals out of 100 were aware of potentially malignant disorders(PMDs) whereas, 58% did not know about it. The participants were asked if they had any white or red patch in oral cavity and 21% of them hadpatches in their oral cavity. More than 66% of the respondents said that PMD is preventable, however 34% felt it is not preventable.(Tab.3)

Table 3: Knowledge about potentially malignant disorders

PARAMETER	YES	NO
Adverse habits	43%	57%
Awareness about PMDs	42%	58%
Presence of PMDs	21%	79%
PMD preventable	66%	34%
PMD leads to oral cancer	79%	21%

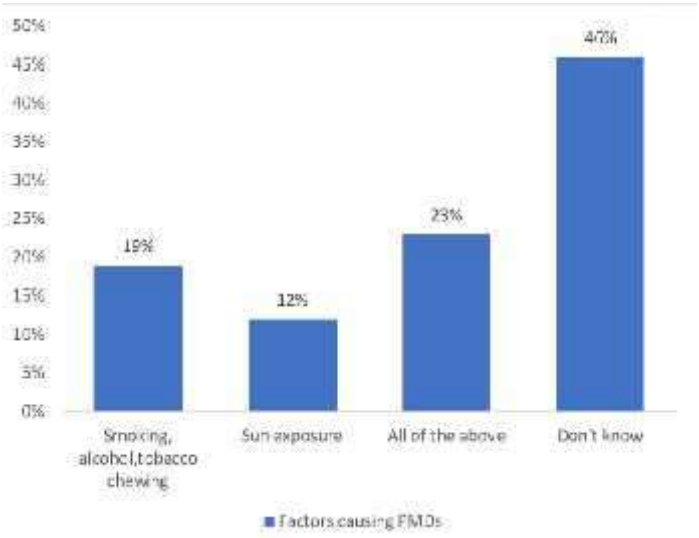
42% of the participants were aware of PMDs through Television,newspaper, internet(37%) and through parents(20%), dentist(22%) and other ways(21%).(Tab.4)

Table 4: Mode of awareness



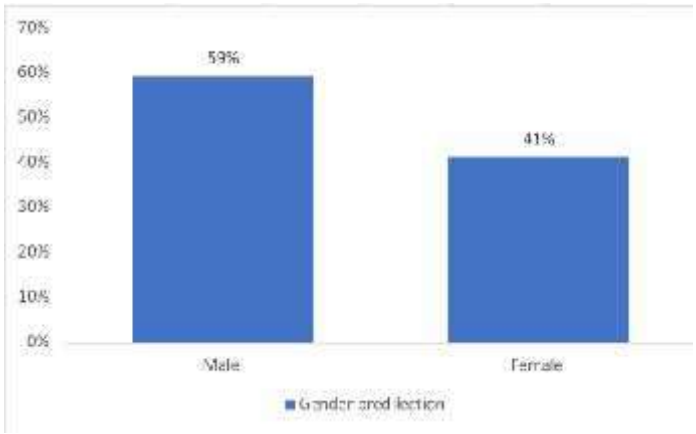
The participants were asked about the factors which cause potentially malignant disorders(PMDs) out of which, 23% of the individuals said that both adverse habits and sun exposure can be the main causative factors and 46% of the respondents admitted that they don't know about the cause (Tab.5).

Table 5: Factors causing PMDs



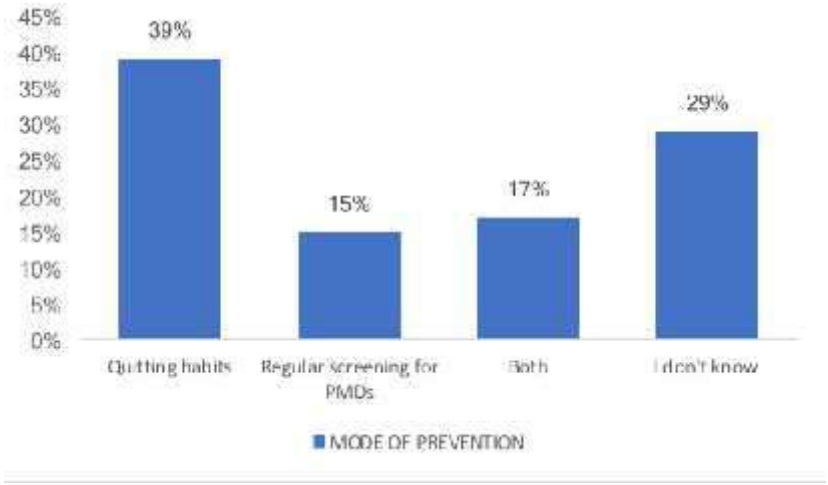
Out of 100 participants, 59% of the individuals said that PMDs are more common in male than female(41%).

Table 6: Graph showing Gender Predilection



The participants were asked about the mode of prevention, 17% of the individuals said that quitting habit and regular screening for PMDs can help to prevent the potentially malignant disorders(Tab.7).

Table 7: Graph showing Mode of prevention



Discussion

Precancerous Condition can be defined as a disease or patient habit which does not necessarily alter the clinical appearance of local tissue but is known to have a greater than normal risk of precancerous lesion or cancer development. A precancerous lesion is a morphologically altered tissue in which oral cancer is more likely to occur that its apparently normal counterpart whereas a precancerous condition is a generalized state associated with a significantly increased risk of cancer.Potentially Malignant Disorders is defined by WHO 2005 as the risk of malignancy being present in a lesion or condition either at time of initial diagnosis or at a future date(8).Oral cancer is a carcinoma that occurs in the oral cavity and belongs to the group of head and neck tumors. Malignant oral cavity tumors account for about 3-5% of all tumors. According to epidemiological data, oral cavity cancer is the sixth most frequent among all types of cancer, and one of the ten most common causes of death, with an incidence of 10 in 100,000 people(9).The most common type is squamous cell carcinoma that makes up 90% of all malignant tumors (10). Oral cancer accounts for about 4% of all malignant tumors in men, and 2 % in women, of an average age of about 60, although an increase in incidence has been recorded recently in people of younger age. The main etiologic factors for the development of oral cavities cancers are smoking, alcohol consumption, sun exposure, liver cirrhosis, dietary deficiencies, viruses, chronic tooth

injuries, etc (11). Oropharyngeal carcinoma belongs to the group of the most malignant tumors, given the fact that the survival rate is about 50%. Although the oral cavity is accessible for visual examination, oral cancers are generally diagnosed late. Lack of awareness in patients and the phase of negation and ignoring the symptoms are the reasons for not seeing a doctor right away, and is considered the “first loss of time”. The “second loss of time” occurs due to lack of awareness of medical and dental professionals and the lack of a timely diagnosis. The “third loss of time” is the period that passes from the diagnosis to the commencement of treatment (12). It is important to reduce the “first loss of time” by increasing awareness of the importance of self-examination, and to shorten the “second loss of time” through education of medical and dental staff.

Oral cancers mostly develop from potentially malignant lesions (precancerouses). Potentially malignant lesions (PML) are lesions of the oral mucosa which are at an increased risk for malignant transformation compared to healthy mucosa. At the congress of the World Health Organization in 2005, “potentially malignant diseases” was suggested as a term for malignant and precancerous conditions (13). The most common precancerous lesions are leukoplakia, erythroplakia, oral lichen planus, and actinic cheilitis. Leukoplakia is a white spot or plaque which cannot be clinically nor histologically characterized as any other lesion, and is not associated with any physical or chemical causal factor, aside from the use of tobacco. According to epidemiological data, general prevalence of leukoplakia ranges from 0.6% to 4.6%. It is most common in men of middle and older age. Cancer will develop from rom leukoplakia in 1-20% of cases. The main activators of cancerization are exogenous factors such as smoking, alcohol, human papilloma virus, or chewing betel (14). According to clinical studies, the most common site of malignant transformation of leukoplakia is the oral cavity floor (15)

Oral lichen planus is immune-mediated inflammatory mucocutaneous disease which occurs in adults, more frequently in females. It occurs in three basic forms (lichen ruber planus, erosivus, and bulosus), and it is classified by its appearance as either reticular, annular, nodular, atrophic, and sclerosus. Premalignant potential of this disease is from 0.4% to 3.3%, particularly for erosive lichen (16).

Actinic cheilitis is a potentially malignant lesion caused by exposure to sunlight. It affects males more often than females, and the most common localization is the lower lip. Off-white

or red changes may be ulcerated. It is estimated that the rate of malignant alteration ranges from 1.4% to 36% (17).

From the above, it was concluded that there may be potentially malignant lesions in the oral mucosa that can turn malignant, and that early diagnosis of PML is of great importance in cancer prevention and early stage cancer detection. If cancer is detected in the initial stage, the potential for remission is 80%.

In a study done by arpita et al, it has brought to light a disturbing aspect that though all the study subjects in this study had high risk behavior, only a handful recognized that their adverse habit could lead to OPMD. Tobacco use and alcohol consumption were appreciated as risk factors for oral cancer by 77% and 60% of the subjects, which was an encouraging finding, but only 14% and 8.6% were aware of these as high risk behaviors for OPMD. Among those who were aware of OPMD, not even half appreciated their lifestyle could lead to development of OPMD. The awareness of other risk factors like poor oral hygiene, actinic radiation, viral infections and micronutrient deficiency was very low. Formosa et al have reported that 92% of their study respondents agreed or strongly agreed that smoking is a strong risk factor for oral cancer, followed by tobacco chewing (84%), tobacco chewing with areca nut (68%), chewing areca nut alone (51%) and exposure to actinic radiation (71%) as risk factors. However, the results for alcohol intake, age, and HPV infection were found to be relatively poor with proportions 33%, 34%, and 23% respectively. In the study by Elango et al 77% of the subjects identified smoking, 64% alcohol and 79% pan chewing as a cause of oral cancer. Ariyawardana et al. reported that 80.7%, 47% and 17% were aware of links with tobacco smoking and alcohol consumption, respectively. Results similar to our study were reported by Amarasinghe et al. where majority of alcohol consumers and smokers and about half of the betel quid chewers surveyed were unaware of the risks conferred by their lifestyles. Pai et al. reported that smokers and past users of tobacco were found to be more aware of effects of tobacco on oral and general health as compared to smokeless and current users but awareness about OPMD remained low in their study as well. This situation calls for specific intervention. There is need for not only increasing awareness regarding OPMD and its presentation in the oral cavity, but the association between adverse life style and increased risk of developing OPMD needs to be stressed. In addition, information needs to be focused on making the tobacco and alcohol users aware of the synergistic potential of these high risk behaviors leading to development of oral cancer and OPMD. For the small number of

tobacco and alcohol users who were aware of OPMD as well as its causal relationship with their adverse lifestyle, it is understood that other factors must be reinforcing continued use. Further investigations have to be carried out to find out the reasons behind the continued practice of high-risk habits, despite knowledge. For this subset, specific counseling is necessary(18).

In a survey done among dental patients in Queensland, Australia, almost all of the participants were with secondary school education(44.7%). However, in this study, majority of the participants are undergraduate individual (38%). In the present study we did not observe any significant differences in the level of knowledge between genders and age groups. This suggests that the education level is the most important variable influencing level of knowledge. Therefore improving knowledge of people especially those with low education is particularly important.

In a study done byjayasinghe et al , identification of alcohol as a potential risk for oral carcinogenesis was low (47.8%). In contrast to the published literature, where the identification of alcohol as a risk factor is high among dental students in this study, only 43.4% of dental students in this study identified it correctly, whereas among medical students it was higher (49.3%). This shows the importance of emphasising the role of alcohol in causation of oral cancer in teaching curricula. Even though they had a good knowledge of some OPMDs, oral erythroplakia as an OPMD was identified correctly only by 18.3% of the respondents. Low awareness of erythroplakia as an OPMD has been reported by others as well. Only 11% knew the overall 5-year survival rate of oral cancer. Knowledge of OPMD and oral cancer among the participants was not satisfactory, and only 10.7% of the respondents had good knowledge of OPMD and oral cancer, and in 12.5%, it was very poor. Similar to the literature from other countries, dental students, especially students in the final year, had a significantly better knowledge ($p<0.05$). About 80% of the medical students had not examined an OPMD or a single oral cancer. A significant number of medical and dental students who have completed clinical training believe that they do not have sufficient knowledge on prevention and detection of OPMD and oral cancer. In a country where OPMD and oral cancers are common, this emphasizes the need for a change in the medical curriculum to incorporate this important area(19).

Ariyawardana and Vithanaarachchi reported in their hospital-based study that over 90% were aware of oral cancer and the figure for potentially malignant disorder was 45%. Higher prevalence of oral cancer in that population and the displayed oral cancer education material, such as posters and pamphlets in public hospitals, into which general public has free access, may be contributory for this high level of awareness(20).

Amarainghe et al reported comparable results for awareness of oral and pharyngeal cancer in their community-based study in rural Sri Lanka. However, this study reported significantly low level(23.0%) of public awareness of oral cancer in the U.K. The study reported in comparison to lung cancer of which the awareness was 97% whereas only 56% of the participants of the survey were aware of oral cancer. Relatively low prevalence of oral cancer may have contributed to the result.Knowledge of the risk factors among the general public is one of the most important parameters for efficacious prevention of OC in the community. In the study, it was found that 48.2% of the respondents were able to identify pan chewing as a strong risk factor while 46.8% identified cigarette smoking as a risk factor. Contrary to this, Park et al in their study based in Western Australia found that only 42 and 5% of patients were able to identify tobacco and alcohol respectively as risk factors for OC. The present study found level of education, the most significant sociodemographic factor affecting patients' risk factor knowledge; participants educated till undergraduate level or higher displayed an overall lesser level of awareness of OC and OPMDs(42%) through television, newspaper(37%), dentist(22%), and others(21%)(21).

According to the statistics, in 2012 the incidence of oral cancer in India is 53842 in males and 23161 in females. Considering the gender in all the age groups, men are more affected than women. In India, men are two to four times more affected than women due to the changes in the behaviour and lifestyle patterns and a study shows that the participants were aware that men(59%) are commonly affected by PMD than women(41%). Detection of oral potential malignant disorders at an early stage ,especially in high risk groups is of utmost importance to prevent further morbidity as they have shown a rate of progression and cancer transformation of up to 17% within a mean period of 7 years after diagnosis. The highest transformation rate is seen in heterogenous erythroplakia and erythroleukoplakia with dysplastic changes. In this study, the participants felt that quitting habits (39%) and regular

screening of oral cavity for PMDs (15%) can help us to prevent potentially malignant disorders

Conclusion

According to this survey, awareness of oral potential malignant disorders was low among patients. It was found that television, internet had played a significant role in patients who were aware of oral precancerous conditions.Potentially malignant disorders is asymptomatic at early stages, and hence, the affected individuals do not seek treatment. Therefore, knowledge of the clinical signs and symptoms of OC is of utmost importance.

Corresponding Author

E. Roja,

Reader, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600 077. Tamil Nadu, India

Email Id: itsrojaa@gmail.com

Contact Number: +91 99941 74480.

References

1. Sachin C. Sarode,Gargi S. Sarode, SwaradaKarmarkar, Jagdish V.Tupkari.,A new classification for potentially malignant disorders of the oral cavity. j.oraloncology.2011.06.005
2. Warnakulasuriya, S, Johnson, Newell W, Van Der Waal, I. Nomenclature and classification of potentially malignant disorders of the oral mucosa. Journal of Oral Pathology & Medicine 2007;36:575-80.
3. Misra V, Singh PA, Lal N, Agarwal P, Singh M. Changing Pattern of Oral Cavity Lesions and Personal Habits Over a Decade: Hospital Based Record Analysis from Allahabad. Indian Journal of Community Medicine: Official Publication of Indian Association of Preventive & Social Medicine 2009;34:321-5.
4. Pindborg JJ, Daftary DK, Mehta FS, A follow-up study of sixty-one oral dysplastic precancerous lesions in Indian villagers. Oral surg Oral med Oral patho 1977;43:383-90.

5. Reibel J. Prognosis of Oral Pre-Malignant Lesions: Significance of Clinical, Histopathological, and Molecular Biological Characteristics. Crit Rev Oral Biol Med 2003;14:47-62.
6. Neville BW, Damm DD, Allen CM, Bouquot JE. Epithelial Pathology. In: Neville BW, Damm DD, Allen CM, Bouquot JE (ed.'s). Oral & Maxillofacial Pathology. 3rd ed. Saunders, 2009. p315-88.
7. Rajendran R. Benign and Malignant Tumors of the Oral Cavity. In: Rajendran R, Sivapathasundaram B. Shafer's Textbook of Oral Pathology. 6th ed. Elsevier, 2009. p80-218.
8. Joseph Formosa, Awareness and Knowledge of Oral Cancer and Potentially Malignant Oral Disorders among Dental Patients in Far North Queensland, Australia,Asian Pac J Cancer Prev, 16 (10), 4429-4434.
9. Bulsara M. Knowledge and perceptions regarding oral and pharyngeal carcinoma among adult dental patients. Aust Dent J 2011 Sep;56(3):284-289.
10. Sree Vidya Krishna Rao1, Gloria Mejia1, Kaye Roberts-Thomson1, Richard Logan, "Epidemiology of Oral Cancer in Asia in the Past Decade- An Update (2000-2012)"
11. Shalini Gupta, Rajender Singh1, O. P. Gupta2, Anurag Tripathi, "Prevalence of oral cancer and pre-cancerous lesions and the association with numerous risk factors in North India: A hospital based study" Natl J Maxillofac Surg. 2014 Jul-Dec; 5(2): 142–148.
12. Vishma BK et al. Awareness of oral cancer and its risk factors in a rural community in Mandya, Karnataka, India Int J Community Med Public Health. 2016 Jan;3(1):347-352
13. Reichart PA, Philipsen HP. Oral erythroplakia: a review. Oral Oncol. 2005;41:551–561.
14. IndhulekhaVimalakshan, Dr. M.P. Santhosh Kumar,Knowledge and Awareness Regarding Oral Cancer among Dental Patients.Int. J. Pharm. Sci. Rev. Res., 43(2), March - April 2017; Article No. 21, Pages: 112-116
15. Sabana A. Nazar; Dr. T.N.Uma maheswari, Awareness Of Early Detection And Prevention Of Oral Cancer among Dentists - A Review. IOSR Journal of Dental and Medical Sciences (IOSR-JDMS).2279-0861. Volume 13, Issue 1 Ver. V. (Jan. 2014), PP 10-12.

16. SANTHOSH KUMAR MP, HARSHINI AK,KNOWLEDGE AND AWARENESS ABOUT ORAL CANCER AMONG UNDERGRADUATE DENTAL STUDENTS.Asian J Pharm Clin Res, Vol 9, Issue 4, 2016, 1-3
17. G. MuthuLaakshmi Awareness of Oral Cancer among a Hospital based Out-Patient Population- A Questionnaire Based Study .J. Pharm. Sci. & Res. Vol. 8(7), 2016, 687-691.
18. Agrawal M, Pandey S, Jain S, Maitin S. Oral cancer wareness of the general public in Gorakhpur city, India. Asian Pac J Cancer Prev 2012;13(10):5195-5199.
19. Kumar S,Debnath N, Ismail MB, kumar A, badiyani BK, Dubey PK, Sukhtankar LV. Prevalence and risk factors for oral potentially malignant disorders in Indian population. Adv Prev Med 2015;2015:208519,1-7.
20. Ariyawardana A, Vithanaarachchi N. Awareness of oral cancer and precancer among patients attending a Hospital in Sri Lanka. Asian Pac J Cancer Prev 2005 Jan-Mar;6(1):58-61.
21. Amarasinghe HK, Usgodaarachchi US, Johnson NW, lalloo R, Warnakulasuriys S. Public awareness of oral cancer, potentially malignant disorders and risk factors in some rural populations in Sri Lanka. Community Dent Oral Epidemiol 2010Dec;38(6):540-548.

Awareness Level about Early Identification and Detection of Oral Cancer in Dental Patients

M.A.Fazeelath Banu [1], Herald.J.Sherlin [2], Suresh.V [3]
Graduate Student [1], Reader [2],Department of Oral Pathology
Reader [3],Department of Prosthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction

Oral cancer is one of the most common cause of morbidity and mortality today. It is increased by tobacco and alcohol use. Oral cancer is a preventable disease due to its association with well-known risk factors and easy detectability. There is a significant deficiency in the awareness of oral cancer and its risk factors among the public. Raising public awareness could effectively contribute to achieving a significant reduction in the incidence of oral cancer.To study the awareness level about early identification and detection of oral cancer among dental patients.

Materials and Methods

A questionnaire based survey was done about the awareness and knowledge of risk factors, signs,symptoms, prevention and treatment including 50 dentalpatients. Sociodemographic information of the patientswas obtained.

Results

In total 96% of participants have heard of oral cancer. 80% are aware of the risk factors ,65% are aware of the signs and 88% are aware of the prevention and treatment.

Conclusion

There is lack in depth of knowledge of oral cancer. Mass media and health campaigns were the main sources of information. Regular oral cancer screenings should be implemented in chennai.

Key Words: Oral cancer, Symptoms, Signs, Prevention, Treatment

Introduction

The incidence of oral cancer is very high in many parts of the world[1,2], Oral and oropharyngeal cancer is the sixth most common cancer in men and ninth most common cancer in women[3]. It is more common than Hodgkin's disease, carcinoma of the brain, liver, thyroid gland, cancer of stomach[4]. India has one of the highest rates of oral cancer in the world; accounting for one third of the total cancers[5].The major risk factors for oral cancer include tobacco, alcohol, betal nut chewing[6-8].About 70-75% of oral cancer is due to prevalence of consumption of tobacco and alcohol[9,10].Besides these habits, other factors include human papilloma virus (HPV) infection[11],diet[12] and genetic susceptibility. Cancer arises by means of two prominent alterations namely genetic alterations and epigenetic alterations[14].Oral cancer is a preventable disease and cessation of these habits can reduce the risk of cancer within 5-10 years[15]. Although there is recent advances in detection and treatment, it is still diagnosed only in advanced stages[16]. Earlier detection of oral cancer provides early treatment, long term survival and improves the prognosis. In previous studies it has been reported that lack of public awareness is the most significant factor for delaying the treatment for oral cancer[18,19]. Oral cancer awareness is lesser when compared to other types of cancers[20-22].The potentially malignant disorderssuch as leukoplakia, erythroplakia, oral submucous fibrosis, non healing ulcers have been failed to recogniseby the patients which are the early signs and symptoms of oral cancer[23-25].Though the oral mucosa is visually accessible,it's not diagnosed early[26]. There is lack of scientific knowledge on risk factors, signs and symptoms among people which carries a low survival rate[27].Early diagnosis greatly increases the patient's chances of survival as the oral cavity is easily accessible for examination[20].Previous studies conducted in different places showed that awareness on signs, risk factors associated with oral malignancy is generally poor [19,24]. The purpose of this study is to evaluate the awarenessof oral cancer, its associated risk factors, signs and symptoms and to determine the knowledge about prevention and treatment among dental patients.

Materials and Methods

The study design population size was calculated based on the power of the previous study and using G power statistical software.This study included 50 out-patients (21 females, 29 males) aged 17 years and above from Saveetha dental college, Chennai. A questionnaire based survey was created to assess public awareness and knowledge about risk factors, signs

and symptoms, prevention and treatment associated with oral cancer. The survey contained 19questions divided into four parts: socio demographic information (3 questions), awareness and knowledge of oral cancer (3 questions), risk factors of the disease(4 questions), early signs and symptoms of the diasease (4 questions) and prevention and treatment (5 questions).

The response “definitely increases” was defined as correct for the risk factors questions. A “risk factor knowledge score” was given summing the number of correct responses to the four items and the results were evaluated into fourcategories: 0,1,2, 3 and above. The response “definitely yes and probably yes” was defined as correct for all the early signs questions. An “early signs knowledge score” was given by summing the number of correct responses to the four symptoms and the results were evaluated into four categories: 0, 1, 2, 3 and above. The response “yes” was defined as correct for the prevention and treatment. A “prevention and treatment knowledge score” was given summing the number of correct responses to the five items and the results were evaluated into four categories: 0,1,2,3 and above. Point of risk factors (PRF), point of early signs (PES) and point of prevention and treatment (PPT) were obtained for each patient[28].

Results

The study included 50 out patients among them 21(42%) were females and 29(58%) were males. The age of the participants was from 17 to 60 years the mean age being 38. There were 29 participants ageing from 17 to 39 (58%) and 21 participants ageing from 40 to 60 years (42%). The education level of the participants included 23 from high school education (46%), 26 from university education (52%) and 1 from elementary school (2%).

Out of 50 participants (Table 1), 48 participants (96%) have heard of oral cancer and 2 participants (4%) have not heard of oral cancer. Most of the patients have heard about oral cancer through TV, radio, newspaper 48 participants (96%). Very few have got information through internet 2 participants (4%). 41 participants (82%) have not undergone screening for oral cancer and 9 participants (18%) are not aware if they have undergone screening.

Table 2 shows distribution and analysis of PRF, PES, PPT in age groups, older patients have better knowledge on risk factors (85.7%) than younger age groups (75.8%). Prevention and treatment of oral cancer was answered maximum by the younger age groups (100%) and by 95.2% by the older age groups.

Considering the distribution and analysis fro gender (Table 3), there is no significant difference in response between males (87.3%) and females (88.8%). Males have better knowledge on risk factors (86.2%) than females (76.1%). Questions on signs and symptoms were answered better by females (95.2%) than males (82.7%). Table 4 shows groups classified based on education level. Participants with university qualification (89.7%) have answered slightly better than high school qualification (86.9%).

Among the risk factors, smoking was identified correctly by maximum number of participants (44 participants 88%) followed by paan chewing (23 participants 46%), long term trauma due to sharp tooth(4 participants 8%) and eating hot and spicy foods (1 participant 2%). In the sign and symptoms, a wound which does not heal for long time was answered by maximum number of participants (41 participants 82%), then sore and bleeding gums (40 participants 80%), followed by red patch (24 participants 48%) and white patch (22 participants 46%). When questioned about prevention and treatment, 44 participants (88%) answered that oral cancer is preventable,39 participants (78%) answered that cancer is treatable at an early stage , 30 participants (60%) answered that going to a dentist at an early stage can identify oral cancer and 27 participants (54%) answered that there is advanced modalities to detect oral cancer.

Table 1:Percentages of awareness and knowledge about oral cancer.

Items		N (Number)	Percent (%)
Have you ever heard of oral cancer?	YES	48	96
	NO	2	4
Where did you know about it?	Awareness program in TV, Radio & Newspaper	48	96
	Friends	0	0
	Internet	2	4
	Dentist	0	0
Have you undergone screening for oral cancer?	YES	0	0
	NO	41	82
	I DON’T KNOW	9	18

Table 2:Distribution and analysis of PRF, PES and PPT for age groups.

Variables	Points	Age groups		Total N
		16 – 39 yrs	40+ yrs	
PRF	PRF = 0	7 (70)	3(30)	10
	PRF = 1	10 (45.4)	12(54.5)	22
	PRF = 2	11 (64.7)	6(35.2)	17
	PRF = 3 & above	1 (100)	0(0)	1
PES	PES = 0	3 (50)	3(50)	6
	PES = 1	4 (44.4)	5(55.5)	9
	PES = 2	10 (62.5)	6(37.5)	16
	PES = 3 & above	12 (63.1)	7(36.8)	19
PPT	PPT=0	0 (0)	1(100)	1
	PPT=1	6(54.5)	5 (45.4)	11
	PPT=2	9(60)	6 (40)	15
	PPT=3 & above	14(60.8)	9 (39.1)	23

0 = Poor knowledge

1 = Low knowledge

2 = Medium knowledge

3 & above = High knowledge

Table 3:Distribution and analysis of PRF & PES for gender groups.

Variables	Points	Gender	Total

		Female	Male	
PRF	PRF = 0	5(55.5)	4(44.4)	9
	PRF = 1	7(30.4)	16(69.5)	23
	PRF = 2	9(52.9)	8(47)	17
	PRF = 3 & above	0(0)	1(100)	1
PES	PES=0	1(16.6)	5(83.3)	6
	PES=1	5(55.5)	4(44.4)	9
	PES=2	5(31.2)	11(68.7)	16
	PES=3& above	10(52.6)	9(47.3)	19
PPT	PPT=0	1(33.3)	2(66.6)	3
	PPT=1	4(66.6)	2(33.3)	6
	PPT=2	2(12.5)	14(87.5)	16
	PPT=3 & above	14(56)	11(44)	25

0 = Poor knowledge

1 = Low knowledge

2 = Medium knowledge

3 & above = High knowledge

Table 4:Distribution and analysis of PRF & PES for educational level groups.

Variables	Points	Elementary school	High school	University	Total
PRF	PRF = 0	1(10)	4 (40)	5 (50)	10
	PRF = 1	0(0)	10 (47.6)	11 (52.3)	21
	PRF = 2	0(0)	9 (50)	9 (50)	18
	PRF = 3 &above	0(0)	0 (0)	1 (100)	1

PES	PES=0	0(0)	4 (66.6)	2 (33.3)	6
	PES=1	1(10)	6 (60)	3 (30)	10
	PES=2	0(0)	6 (40)	9 (60)	15
	PES=3 & above	0(0)	7 (36.8)	12 (63.1)	19
PPT	PPT=0	1(33.3)	1 (33.3)	1 (33.3)	3
	PPT=1	0(0)	4 (80)	1 (20)	5
	PPT=2	0(0)	10 (55.6)	8 (44.4)	18
	PPT=3 & above	0(0)	8 (33.3)	16 (66.7)	24

0 = Poor knowledge

1 = Low knowledge

2 = Medium knowledge

3 & above = High knowledge

Discussion

Oral cancer is reported as having one of the highest mortality ratios amongst all malignancies[29]. Cohort studies show that oral cancer has risen in all age groups throughout the world in the last ten decades.The prevalence of oral cancer in indiais up to four times higher than other countries[30]. Oral cancer has a vast potential for prevention. It could be prevented by habits cessation and early detection by visual examination which could lead to reduction in mortality[31].

Studies on public awareness about detection and prevention of oral cancer have been conducted in several countries like Northern Germany[32], Florida[33], Sri Lanka[34]and in India it is was done in Gorakhpur city[35], Kerala[36].Although each country showed different results, the overall awareness level was insufficient[20,24,29].This study includes the knowledge and awareness of the patients about risk factors, signs and symptoms, prevention and treatment of oral cancer.

Most of the participants in this study were of younger age group and with high education level. More than 90% of the participants have heard of oral cancer which is better when compared with previous studies[20,37]. 80% of the patients have not undergone screening for oral cancer. This is a drawback in our health system which needs to be improved. Considering the risk factors, smoking was answered to a maximum level (84%) by the participants followed by paan chewing (46%). This may be due to general health campaigns which promote tobacco cessation. This is also because most of the oral cancer patients were described with habits of smoking[38]. Paan chewing was answered next to smoking as it is very common in south Indiaand it was related to social and cultural practices among rural people. Studies conducted in Newyork state and Newjerseyarea have shown that knowledge concerning risk factors was found as 76% for smoking and tobacco use. In Turkey, knowledge related to these risk factors was 57.6%[28]. In another study 80% of the population blamed smoking and chewing tobacco to be the major reason for the occurrence of oral cancer[39]. The results of the current study regarding the knowledge of risk factors was relatively high than the previous studies.In southasian population, most of the squamous cell carcinoma have preceding precancerous conditions arising due to smoking and betel nut chewing whereas in other European countries most of the carcinomas arise de novo[40].

Regarding signs and symptoms, non healing wounds was answered to maximum level of 82% by the participants followed by sore and bleedinggums which is 80%. In the previous studies, 57-66.5% have answered for sore and bleeding gums[24,33]. Painless white patch and red patch were answered to a relatively low level. The signs and symptoms were answered at a lesser level among the older age group. This could be due to fact that elderly personsperceived changes in oral mucosa as a part of ageing. In questions related to prevention and treatment oralcancer was answered as preventable by more than 80% of the participants. It was reported that higher education level and age increases the awareness and knowledge of oral cancer[33].

The sociodemographic level could affect the awareness level about oral cancer.Among the gender, female participants have better awareness about signs and symptoms than the male participants. There were similar results seen in previous studies[41].This is because women are generally more concerned about their health and well being and are aware about any physical changes occurring in the body. In age groups, the younger age group (16-39) have

answered correctly than the older age group (40-60). People with higher qualification and younger age had better knowledge and awareness[42]. The risk of oral cancer is inversely proportional to increasing level of education, occupation and income[43].

The younger age group have better knowledge about the risk factors, signs and symptoms. Mass media and health campaigns were the main sources of information for oral cancer[44].Among the educational level, people with university level of qualification have better knowledge about oral cancer. Education plays an important role in knowing about oral cancer.

The prevention of oral cancer depends on its early detection. Most of the oral cancers are preceded with a precancerous lesion. The American Dental Association has stated that Identifying white and red the spots that show dysplasia and removing them before they become cancer has proved to be one of the most effective methods for reducing the incidence and mortality of cancer[44].

Overall there is lack in depth of knowledge about oral cancer in this study. Though the general awareness of the people is good, focus should be done on conducting various oral health education programs for the recognition of risk factors, signs and symptoms, prevention and treatment. This could be done through oral cancer prevention campaigns based on media advertising such as radio, TV , newspaper articles[28].

Conclusion

The results of this study was better when compared to previous studies done in different populations.This study was first one of its kind to be conducted in Chennai population. Education level and age were significant factors associated with awareness of early signs, risk factors, prevention and treatment of oral cancer. More screening programs should be conducted for early identification of oral cancer.

Corresponding Author

Suresh.V

Reader,Department of Prosthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University,

162, Poonamalle High Road,Chennai-600077,Tamil Nadu, India.

Telephone Number: +91-9543192858

Email: sureshvbhat@gmail.com

Reference

1. Boyle P, Macfarlane GJ, Scully C. Oral cancer: necessity for prevention strategies. The Lancet 1993; 342:1129.
2. Johnson NW.Orofacial neoplasms: global epidemiology, risk factors and recommendations for research. Int Dental J 1991; 41:365-75.
3. Parkin DM, Pisani P, Ferlay J. Estimates of the world incidence of 25 major cancers in 1990. Int J Cancer 1999; 80(6):827-41.
4. Jemal A, Thomas A, Murray T, Thun M. Cancer statistics.CA Cancer J Clin. 2002; 52(1):23-47.
5. Ganesh R, Joseph John, Meignana Arumugham I.Is Oral cancer related to Socio demographic factors? - A Hospital based study of patients from urban areas of Tamil Nadu. Journal of Indian Association of Public Health Dentistry 2013; 11(1):39-43.
6. Balaram P, Sridhar H, RajkumarT, Vaccarella S, Herrera R.Oral Cancer in Southern India: The Influence of Smoking, Drinking, Paan-Chewing and Oral Hygiene. Int J Cancer 2002; 98:440-5.
7. De Stefani E, Bofetta P, Deneo-Pellegrini H, Ronco AL, Acosta G.The effect of smoking and drinking in oral and pharyngeal cancers: a case-control study in Uruguay. Cancer Letters 2007; 246:282-9.
8. Lin WJ, Jiang RS, Wu SH, Chen FJ, Liu SA. Smoking, alcohol, and betel quid and oral cancer: a prospective cohort study. J Oncol 2011; 525:976.
9. Johnson NW, NWJ Jatin P Shah, John G Batskis, Martin Dunitz. Aetiology and risk factors for oral cancer. In: Oral cancer 2003; 36.
10. Johnson NW, NWJ Jatin P Shah, John G Batskis, Martin Dunitz. Prevention of oral cancer. In: Oral Cancer 2003; 464.
11. Lohavanichbutr P, Houck J, Fan W, Yueh B, Mendez E. Genome wide gene expression profiles of HPV-positive and HPV-negative oropharyngeal cancer: potential implications for treatment choices. Arch Otolaryngol Head Neck Surg 2009; 135:180-8.

12. De Stefani E, Bofetta P, Ronco AL, Correa P, Oreggia F, Deneb-Pellegrini H. Dietary patterns and risk of cancer of the oral cavity and pharynx in Uruguay. Nutr Cancer 2005; 51:132-9.
13. Drummond SN, Gomez RS, Motta Noronha JC, Pordeus IA, Barbosa AA, De Marco L. Association between GSTT1 gene deletion and the susceptibility to oral squamous cell carcinoma in cigarette smoking subjects. Oral Oncol 2005; 41:515-9.
14. G.S.Asokan, S.Jeelani, N.Gnanasundaram. Promoter Hypermethylation Profile of Tumour Suppressor Genes in Oral Leukoplakia and Oral Squamous Cell Carcinoma. J Clin Diagn Res. 2014 Oct; 8(10): ZC09–ZC12.
15. Johnson NW, Bain CA. Tobacco and oral health. British Dental Journal 2000;189:200-6.
16. B. W. Stewart and P. Kleihues. World Cancer Report. International Agency for Research on Cancer 2003; Geneva, Switzerland.
17. Fritz. International Classification of Diseases For Oncology. World Health Organization 2000; Geneva, Switzerland, 3rd edition.
18. McLeod NMH, Saeed NR, Ali EA. Oral Cancer: Delays in Referral and Diagnosis Persist. Br Dent J. 2005; 198:681-684.
19. Hollows P, McAndrew PG, Perini MG. Delays in the Referral and Treatment of Oral Squamous Cell Carcinoma. Br Dent J. 2000; 188:262-265.
20. Warnakulasuriya KA, Harris CK, Scarrott DM, Watt R, Gelbier S, Peters TJ. An alarming lack of public awareness towards oral cancer. Br Dent J 1999; 187:319-22.
21. Lawoyin JO, Aderinokun GA, Kolude B, Adekoya SM, Ogundipe BF (2003). Oral cancer awareness and prevalence of risk behaviours among dental patients in south-western Nigeria. Afr J Med Sci 2003; 32:203-7.
22. Rogers SN, Hunter R, Lowe D. Awareness of oral cancer in the Mersey region. Br J Oral Maxillofac Surg 2011; 49:176-81.
23. Amarasinghe HK, Usgodaarachchi US, Johnson NW, Lalloo R, Warnakulasuriya S. Public awareness of oral cancer, of oral potentially malignant disorders and of their risk factors in some rural populations in Sri Lanka. Community Dent Oral Epidemiol2010; 38:540-8.
24. West R, Alkhatib MN, McNeill A, Bedi R. Awareness of mouth cancer in Great Britain. Br Dent J 2006; 200:167-9.

25. Devadiga A, Prasad KV. Knowledge about oral cancer in adults attending a dental hospital in India. Asian Pac J Cancer Prev 2010; 11:1609-13.
26. Ptton LL. The effectiveness of community based visual screening and utility of adjunctive diagnostic aids in the early detection of ora cancer. Oral Oncol2003; 39(7):708-23.
27. B.W.Stewartand, P.Kleihues. World Cancer Report. International Agency for Research on Cancer 2003; Geneva, Switzerland.
28. IlkayPeker, MeryemToramanAlkurt. Public Awareness Level of Oral Cancer in a group of Dental Patients. The Journal of Contemporary Dental Practice 2010; 11(2):049-56.
29. Parkin DM, Bray F, Ferlay J, Pisani P. Estimating the world cancer burden. Globocan 2000. Int J Cancer 2001; 94:153–6.
30. Moore SR, Johnson NW, Pierce AM, Wilson DF. The epidemiology of mouth cancer: a review of global incidence. Oral Dis 2000; 6:65-74.
31. Shodhganga.inflibnet.ac.in/bitstream/10603/2363/15/15_chapter 6
32. KatrinHertrampf, Hans-Jürgen Wenz, Michael Koller, JörgWiltfang. Public awareness about prevention and early detection of oral cancer: A population-based study in Northern Germany. Journal of Cranio-Maxillofacial Surgery 2012;40(3):82-6.
33. Tomar SL, Logan HL. Florida adult's oral cancer knowledge and examination experiences. J Public Health Dent. 2005; 65(4):221-30.
34. Hemantha K. Amarasinghe, Udaya S. Usgodaarachchi, Newell W. Johnson, RatilalLalloo, SamanWarnakulasuriya. Public awareness of oral cancer, of oral potentially malignant disorders and of their risk factors in some rural populations in Sri Lanka. Community Dentistry and Oral Epidemiology 2010; 38(6) Issue 6:540–548.
35. Agrawal Mamta,PandeySushma, Jain Shikha, MaitinShipra. Oral Cancer Awareness of the General Public in Gorakhpur City, India. Asian Paci c J Cancer Prev 2012; 13(10):5195-5199.
36. J KalavathyElango, Karimassery R Sundaram, PalethGangadharan, PramodSubhash, Sherry Peter, CivyPulayath, Moni Abraham Kuriakose. Factors Affecting Oral Cancer Awareness in a High-risk Population in India. Asian Pacific J Cancer Prev 2009; 10:627-630.
37. Patton LL, Agans R, Elter JR, Southerland JH, Strauss RP, Kalsbeek WD. Oral cancer knowledge and examination experiences among North Carolina adults. J Public Health Dent 2004; 64(3):173-80.

38. Abbe R. Cancer of the mouth. NY Med. J 1915; 102:1-2.
39. Chandini Ravikumar, Abilasha Ramasubramaniam. A survey on oral hygiene and oral cancer awareness. WJPPS 2016; 5(12):1057-1062.
40. Johnson NW,Warnakulasuriya KAAS.Epidemiology and aetiology of oral cancer in the United Kingdom. Community Oral Health 1993; 10:13-24.
41. Prayman E, Yang YH, Warnakulasuriya S. Oral cancer awareness of patients attending health centres in Trinidad. Int J Clin Dent 2009;2:1-11.
42. Scott L. Tomar, DrPH, Henrietta L. Logan. Florida Adult's Oral Cancer Knowledge and Examination Experiences. Journal of Public Health Dentistry 2005; 65(4):221-230.
43. Ganesh R, John J, Saravanan S. Socio demographic profile of oral cancer patients residing in Tamil Nadu- A hospital based study. Indian J Cancer 2013; 50:9-13.
44. Ghani WM, Doss JG, Jamaluddin M, Kamaruzaman D, Zain RB. Oral cancer awareness and its determinants among a selected Malaysian population.Asian Pac J Cancer Prev 2013;14(3):1957-63.

Awareness of Biomedical Waste Management among Class-IV Employees at Saveetha Dental College and Hospital- A Cross-Sectional Questionnaire Study

Abiraamasri BL[1], Siva Kumar[2], DhanalakshmiRavikumar[3],
Graduate Student[1], Senior Lecturer[2],Department of Oral and Maxillofacial Surgery,Senior Lecturer[3],Department of Pedodontics,
Saveetha Dental College,Saveetha Institute of Medical and TechnicalSciences, Saveetha University, Chennai, India

Abstract

Introduction: The biological waste is any waste that is generated during diagnosis, treatment or immunization of human being/animals or during research activities. Utmost importance of biomedical waste disposal should be known by the individual handling it, as it may be bio-hazardous leading to the spread of infectious diseases.The aim of the study is to determine the awareness regarding biomedical waste management policy and practices, attitude towards biomedical waste management among class IV employees in Hospitals.

Materials and Methods: A cross sectional study was conducted using a questionnaire, distributed to 50 individual at Saveetha Dental College and Hospitals. The questionnaire was used to assess the knowledge of biomedical waste disposal and the result was obtained by grading each correct and incorrect answer given by the individuals.

Results: The study shows that the class IV employees have less knowledge about color-coding and storage of biomedical waste.

Conclusion: The present study concludes that there is an average level of knowledge and awareness about biomedical waste generation hazards, legislation and management among class IV employees.

Key Words: *Biomedical waste, disposal management, class IV employee,cross-sectional study, waste disposal.*

Introduction

Over the years, there have been tremendous advances in the health care system so it is ironic that health-care settings, which restore and maintain community health, also threaten patients' well- being. One major threat arises from poor waste management practices, which pose a huge risk to the health of the public, patients, and professionals and contribute to environmental degradation. Biomedical waste is defined as any waste that is generated during diagnosis, treatment and immunization of human/animal or during research. Dental waste includes cotton, sharp objects, latex, glass, extracted teeth etc. These waste have high potential to cause infectious disease when exposed to healthy human being [1]. In health care centers, wastes produced are categorized into hazardous and non-hazardous waste. 85% non-hazardous waste and 15% hazardous among which 10% is infectious and 5% is non-infectious waste. If the infectious waste gets mixed with non-infectious, the entire mass becomes potentially infectious[2,7]. A total of 80% of the waste generated in the hospitals is composed of general waste while the remaining 20% comprises of infectious, toxic or radioactive waste [3]. Improper waste management, lack of awareness about the health hazards from BMWs, insufficient financial and human resources, and poor control of waste disposal are the most critical problems faced with healthcare waste[4,10] The harmful effect of medical waste on us and environment is enhanced if the appropriate handling of these wastes is not adopted. Improper disposal methods of these wastes may lead to the spread of serious and harmful diseases such as AIDS, hepatitis B and C, and tuberculosis (TB) among the healthcare personnel, waste handlers, patients and their visitors, and community where the waste is indiscriminately

deposited [5,11] .The success of biomedical waste management programme rests on the knowledge and practice of the Health Care Worker (HCW) especially, Class IV workers, as they will be handling BMW most commonly [12]. This study was done at a teaching hospital in Chennai with over 1000 to 1500 outpatients' everyday on an average. About 0.5 to 1tonnes of biomedical waste is generated everyday in the hospital and proper segregation of the waste at source is a challenge. It has a multi-disciplinary biomedical waste management committee to oversee the programme [13].

Materials and Method

A cross sectional study was conducted using a questionnaire study, distributed to 50 individual (46 females and 4 males) class IV employees at Saveetha Dental College and Hospitals. The questionnaire is used to assess the knowledge of the class IV employees about biomedical waste management and disposal. A set of 10 questions comprising of simple questions about color-coding system, waste management, storage and needle stick injury was given to all the class IV employees. [Figure 1] Each correct and wrong answer given by the sample was given a score and the result was calculated accordingly.

QUESTIONS:
1)-Do-you-have-knowledge-about-color-coding-system-in-waste management?
a)-Yes———b)-no

2)-Do-you-wear-gloves-while-you-handle-biomedical-waste?
a)-Yes———b)-no

3)-Do-you-follow-color-coding-in-your-clinic?
a)-Yes———b)-no

4)-Color-coding-for-infectious-waste,-bandage,-gauze,-cotton?
a)-Yellow—b)-red—c)-blue—d)-black

5)-Color-coding-for-plastic-waste?
a)-Yellow—b)-red—c)-blue—d)-black

6)-Color-coding-for-glass-bottles-and-out-dated-medicine?
a)-Yellow—b)-red—c)-blue—d)-black

7)-Color-coding-for-needles,-sharp-objects?
a)-Yellow—b)-red—c)-blue—d)-black

8)-Do-you-know-that-improper-biomedical-waste-management-can-lead-to hazardous-infection?
a)-Yes———b)-no

9)-How long-can-biomedical-waste-be-stored?
a)-24hrs——b)-48hrs

10)-Are-you-aware-of-needle-stick-injury?
a)-Yes———b)-no

Figure 1: Questionnaire used to evaluate the knowledge of Class IV employees

Results

Figure 2 depicts the knowledge of class IV employees in the teaching Hospital towards biomedical waste management. The figure infers that the class IV employees have less knowledge for 4,5,6,7 and 9 questions as per the given questionnaire. The study showed that the class IV employees have less knowledge about color-coding and storage of biomedical waste.Among the given set of questions, 68% of the employees gave wrong answer to “how long the bio-medical waste can be stored”, which showed the highest percentage of wrong answer.

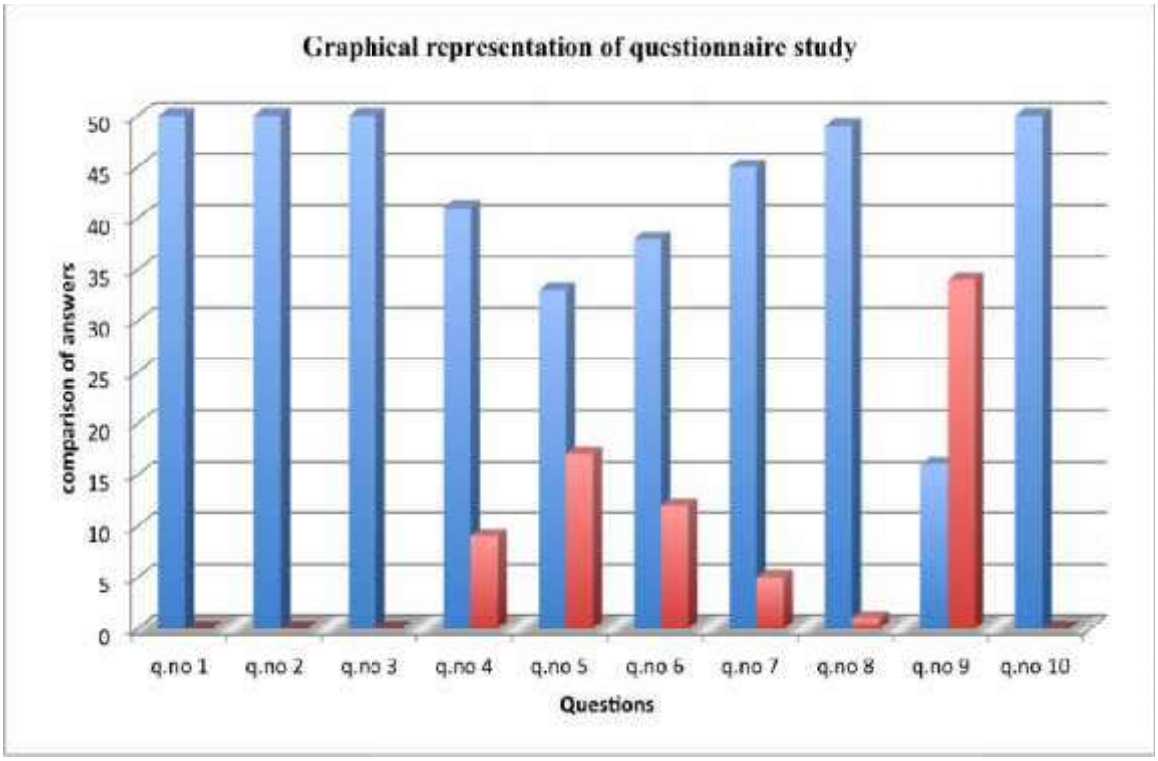


Figure 2: Graphical representation of percentage of correct and incorrect answers provided by the class IV employees

Discussion

The center where the study was at a private teaching hospital, Chennai India. It showed that there was anpoorlevel of knowledge and awareness about biomedical waste generation hazards, legislation and management among class IV employees. Fortunately the level of knowledge and awareness about needle-stick injury was highly adequate. The results of the study was similar to the study done by Alok Sharma [1] .BMW generated by health care establishments can be broadly categorised as general waste, infectious waste, and non-infectious but hazardous waste [25]. Almost 75-85% of waste generated in health-care establishments is non-risk or general waste, which constitutes paper, cardboard boxes, plastic packaging, and kitchen waste . Infectious waste, which includes human anatomical wastes, infectious disposable plastic items and sharps (e.g., needles, razors, scalpels), accounts for only a small fraction, comprising about 10-15% of the total

volume of waste generated in a hospital. However, this small fraction is of the greatest con- cern because it poses a direct threat to the health and hygiene of human beings by transmitting viral, bacterial, fungal or parasitic diseases [25].Low socio-economic status and illiterate people is the most common people working as scavengers, hence it is mandatory to impart knowledge regarding biomedical waste management, including the risks involved in scavenging discarded needles and other sharp items[14] .Hospital wastes are broadly classified as pathological waste, infectious waste, sharp waste, pharmaceutical waste, radioactive waste and chemical waste[15]. Categorizing these biomedical waste by sorting them into color coded plastic bags or container is the effective way to manage biomedical waste[2,6] .In the present study knowledge of sorting biomedical waste based on color coding plastic bags by class IV employees were found to be satisfactory, which was comparable to previous study[7,16].Health hazard associated with poor management in handling biomedical waste include contracting disease like tetanus, rabies, poliomyelitis, meningitis, HIV, Hepatitis Betc. Hence a sound knowledge on heath hazard associated with improper management of biomedical waste will avoid contractingthese highly infectious diseases[2,17] .In our study all the class IV employees were pretty well aware of health hazard associated with poor management in handling biomedical waste, which was found satisfying in comparing to previous study where the awareness of class IV employees were not up to limit[18] .Any type of biomedical waste storage shouldn't be stored beyond 48 hours[2]. Unfortunately most of the participants of this study were not aware of the above-mentioned criteria. Hence a special emphasis has to done in future during training program on biomedical waste[19] .Needle stick injury plays an important role in transmitting deadly infectious diseases like HIV, Hepatitis B. Hence a thorough knowledge on needle stick injury associated issue is a must for any health care professionals. In our study all the participants were well aware about the serious consequences of needle stick injury, which was comparable to previous study [8,20] .A study conducted by Imti,in a tertiary care hospital, Uttar Pradesh, suggested that correct knowledge regarding the same was adequately sufficient across all 4 groups of healthcare personnel which was similar to another study by MathurinLucknow [23].Chudasamin a tertiary care hospital in Rajkot and Madhavi*et al*.

in Khammam,who found 95.4% and 94.8% of the participants were aware of BMW, respectively. Nearly 30.8% of the subjects were aware about BMW rules which were 51.4% in the study by Chudasama.About 1.5% of the total study population had received formal training on BMW management, which is 44.7% and 16.3% in studies of RajkotandKothamangalam,respectively. In the Rajkot study,approximately 96.5% of subjects knew that different colored bags were used for segregation of BMWs whereas almost all the participants (100%) from our study opined the same. Only 6.6% knew correctly about all the colors used for coding. Results from a study conducted in a tertiary care hospital of West Bengalamong the junior doctors show that nearly 76.4% of the subjects knew about various types of color- coded bags for collection of BMW. In a similar study in Rajkot,84.4% of participants identified all colored bags used for BMW collection [24]. Rajkot depicted that about 40.4% of the subjects knew about BMW management categories, which is much higher in comparison with the current study, where it is found to be only 11.6%. In contrary to the present study, about 52.2% and 55.9% of the study population in two studies conducted by Madhavi*et al*. in KhammamandBasuamong interns in West Bengal have a correct knowledge of the same. In another study conducted by Madhukumar and Ramesh in Bangalore,it was only 38.5% [25].The findings of the present study suggest that there is an urgent need to train and educate the dentists and auxiliaries in the dental hospital/clinics of Amritsar through extensive training and re-training programmes. Furthermore, it is also suggested that a proper waste management educational programme should be included in the curriculum for dental education so as to give due importance to this vital issue. Because the study was confined to one major region of North India, further regional studies are required on a larger population to generalize the results, in the formulation and implementation of BMW guidelines. The topic is very relevant to all countries and it is suggested that all public and private healthcare providers should audit the BMW knowledge and practices of their staff at regular intervals. The results of this study will help the hospital authorities to develop a strategy for improving BMW management. BMW management programmes cannot be successfully implemented without the effective knowledge, willingness, motivation, and cooperation from all sections of employees of any health care setting. A

"cradle to grave" approach should be followed in regard to the collection, transportation, treatment and disposal of BMW. There needs to be a sensitization of employees to this issue coupled with effective implementation of rules (such as surprise visits from monitoring authorities and performance based incentives that will facilitate successful implementation of the programme), which is vital for better outcome in future [26]. Nor Masitah Mohamed et al, 2017, concluded that most of the respondents are completely aware of biomedical waste management, which is of paramount importance to reduce the risk of possibly infections material and waste that may be faced the individuals handling these wastes. Among the respondents, 92.5% of them knew the availability of any guidelines laid down by government for biomedical waste management. Union Ministry of Environment and Forests had brought the rules of biomedical waste management into focus under the provision of Environment (protection) act, 1986 [4]. *Sunil Kumar et al., 2012*, about 87.8% of the respondents were aware of various color coding bags that are used to dispose waste. However, a small percentage, approximately 10 respondents were not sure about that. It is statistically significant that 93.3% of the total respondents were following the color coding system to segregate waste in clinics. 94.2% members of the study segregate amalgam in their clinic whereas others were lacking this practice. This study present that dental practitioners had fairly higher good level of attitude and practice. It is observed that more than 95% of the respondents use rubber dam routinely during placing or removing amalgam restorations, and the rest 3.3% of them were not using rubber dam during amalgam restorations. Using rubber dam during operative procedures is important for patient safety. Plus, one study stated that the use of rubber dam during removal of an amalgam restoration had significantly reduced the peak mercury level rise in the patient's plasma [27]. **Gilbert, 2010,** 80% of dentists disagreed that black plastic bags are used to discard human and animal waste. Remaining 5% and 15% of them agreed and were not sure about it respectively. 82.5% of the respondents were disposing infected needles in puncture proof bags, meanwhile 5% had opinion that they should be thrown in red bags and 3.3% chose yellow bags. More than eighty-five percent were using yellow bags for disposal microbiological and biotechnological waste. Next, it was found that 84.3% of the participants were using yellow bags to throw

extracted teeth and only 4.2 percent of them preferred red and black bags to discard extracted teeth .

Conclusion

The present study concludes that there is an average level of knowledge and awareness about biomedical waste generation hazards, legislation and management among class IV employees in South India. Numerous literature review pertinent to biomedical waste management and awareness suggests that this is a perineal problem in many other health care institutions in developing countries. Hence safe disposal of biomedical waste make our whole eco-system safe.Periodical scrutinizing and training are required at all level to make hospital and as well as living environment an health hazard free zone.

Corresponding Author

Dhanalakshmi Ravikumar,

Senior Lecturer,Department of Pedodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077, Tamil Nadu, India.

Email id: dhana9677@gmail.com

Phone Number: +91 9597011442.

References

1. Sharma A, Awareness of Biomedical Waste Management Among Health Care Personnel in Jaipur, India, Oral Health Dent Manag 2013; 12(1): 78-82.
2. Pulavarthi S, Biomedical waste management. Int J AJ Inst Med Sci 2012; 1(1): 67-74.
3. SanthoshMP, Knowledge, awareness and practices regarding biomedical waste management among undergraduate dental students, ajpcr 2017; 10(8)
4. Noor masitah Mohamed shukri and Dr. Jayalakshmi, Dentist knowledge, attitude and practices towards biomedical waste management, IJCR 2017; 9(2):156

5. AshikariswanaNl, Knowledge and attitude in regards to dental care waste management amongst dental students- Questionnaire study, pharm. Sci & Res; 2016;8(9), 1070-1072.
6. Gupta S, Boojh R. Biomedical waste management practices in Balrampur Hospital, Lucknow, India. Waste Manag Res 2006; 24:584-91.
7. BhawaniShanker Sharma, Biomedical waste management, Int J MulDisciplin Consort 2014; 1(1): 29-31.
8. KaruneshSaxena, Bio Medical Waste Awareness and Implementation Gap-Bridging the Gap "Cleanliness is next to Godliness". PacifBusi Rev Int 2015; 7(10):56-59.
9. Varsha K. Pavithan, Knowledge, attitude and practice of needle stick and sharps injuries among dental professionals of Bangalore, India, J Int SocPrev Community Dent.2015 Sep-Oct; 5(5): 406-412.
10. Dutta R, knowledge, attitude and practice of bio-medical waste management among private practitioners in Poonamalleetaluk, Chennai, Int J Community Med Public Health, 2017: 4(8);2930-2933.
11. Ismail MI, Kulkarni AG, Kamble SV et al. knowledge, attitude and practice about bio-medical waste management among personnel of a tertiary health care institute in Dakshina Kannada, Karnataaka. Al Ameen J Med Sci 2013; 6(4):376-80.
12. Imtiaz D, Gupta SB, Singh JP, Assessment of knowledge of hospital staff regarding biomedical waste management in a tertiary care hospital in Uttar Pradesh. Scholars Journal of Applied Medical Sciences 2014; 2(3C): 1070-74.
13. Balaban B, Matthews DJ, Clayton GH, Carry T. Corticosteroid treatment and functional improvement in Duchenne muscular dystrophy: long-term effect. Am J Phys Med Rehabil. 2005; 84:843–50.
14. Beenakker EA, Fock JM, Van Tol MJ, Maurits NM, Koopman HM, Brouwer OF, et al. Intermittent prednisone therapy in Duchenne muscular dystrophy: a randomized controlled trial. Arch Neurol. 2005;62:128–32.

15. Biggar WD, Harris VA, Eliasoph L, Alman B. Long-term benefits of deflazacort treatment for boys with Duchenne muscular dystrophy in their second decade. NeuromusculDisord. 2006; 16:249–55.
16. Henricson EK, Abresch RT, Cnaan A, Hu F, Duong T, Arrieta A, The cooperative international neuromuscular research group Duchenne natural history study: glucocorticoid treatment preserves clinically meaningful functional milestones and reduces rate of disease progression as measured by manual muscle testing and other commonly used clinical trial outcome measures. Muscle Nerve. 2013; 48:55–67.
17. Angelini C, Pegoraro E, Turella E, Intino MT, Pini A, Costa C. Deflazacort in Duchenne dystrophy: study of long-term effect. Muscle Nerve. 1994;17:386–91.
18. Fenichel GM, Florence JM, Pestronk A, Mendell JR, Moxley RT, III, Griggs RC, Long-term benefit from prednisone therapy in Duchenne muscular dystrophy. Neurology. 1991; 41:1874–7.
19. Mendell JR, Moxley RT, Griggs RC, Brooke MH, Fenichel GM, Miller JP, Randomized, double-blind six-month trial of prednisone in Duchenne's muscular dystrophy. N Engl J Med. 1989;320:1592–7.
20. Rahman MM, Hannan MA, Mondol BA, Bhoumick NB, Haque A. Prednisolone in Duchenne muscular dystrophy. Bangladesh Med Res Counc Bull. 2001;27:38–42.
21. Sudhakar V, Chandrashekar J. Dental health care waste disposal among private dental practices in Bangalore City, India. Int Dent J., 2008;58:51-4.
22. Imtiaz D, Gupta SB, Singh JP, Shrotriya VP, Malik S. Assessment of knowledge of hospital staff regarding biomedical waste management in a tertiary care hospital in Uttar Pradesh. Sch J Appl Med Sci 2014;2:1070- 4.
23. Mathur V, Dwivedi S, Hassan M, Misra R. Knowledge, Attitude, and practices about biomedical waste management among healthcare personnel: A cross- sectional study. Indian J Community Med 2011;36:143- 5.

24. Madhukumar S, Ramesh G. Study about awareness and practices about health care wastes management among hospital staff in a medical college hospital, Bangalore. Int J Basic Med Sci 2012;3:7- 11.
25. Basu M, Das P, Pal R. Assessment of future physicians on biomedical waste management in a tertiary care hospital of West Bengal. J Nat Sci Biol Med 2012;3:38- 42.
26. Sunil Kumar VC, Manjunatha M, Badami V, Pradeep PR. Bio- medical Waste Management: A Review. J Oral Health Comm Dent. 2012;6:141-4.
27. Gilbert, Gregg H., “Rubber Dam Use During Routine Operative Dentistry Procedures: Findings FromThe Dental PBRN.” Operative dentistry, 35.5 (2010): 491-499."

Awareness of Complications of Diabetes among Rural and Urban Diabetic Population in Chennai

Ashwini Shenai[1], Halleys Kumar[2], Nabeel Nazar M[3]

Graduate Student[1], Assistant Professor[2], Department of General Medicine,Reader[3],Department of Oral and Maxillofacial Surgery

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Diabetes is chronic, multisystem disorder with life threatening complications. According to a study by the Lancet, India is ranked among the top three countries having high diabetic populations. Easily managed by glycemic control and simple alteration in lifestyle, it is most often allowed to escalate to the level of complications due to lack of adequate knowledge of the disease and its management. Moreover, regional and socioeconomic differences among rural and urban population of the city could result in skewed perceptions of the disease, in terms of treatment and control. Awareness of these patients is crucial to preventing acute complications and reducing the risk of long term complications, in order to have a positive impact on the morbidity and mortality levels associated with the disease. Representative data on their levels of awareness would help plan large scale diabetes awareness programs to reach and masses. Thus, the study aims to assess and compare rural and urban diabetic population of Chennai city, on their knowledge of Diabetes and associated complications.

Materials and Methods: A cross-sectional community-based study was carried out on 100 diagnosed diabetics, of which 50 belonged to rural areas, and 50 belonged to urban. A predesigned questionnaire pertaining to knowledge, attitude and practices relating to the disease was administered to them. Data obtained from the two groups was compiled and statistically analyzed on Microsoft Excel 2016.

Results: Out of the 100 self- reported diabetic subjects, with mean age of 45-65 years, it was evident that urban residents had consistently higher awareness rates about diabetes mellitus, its

management and complications, compared to rural populations. The results emphasize the interrelation between demography and awareness

Conclusion: Urban diabetic patients are more aware than rural diabetic patients about diabetes mellitus, its management and complications. The study emphasizes interrelation between demography and awareness levels. The results underscore the need to design and implement awareness programs directed at increasing awareness among rural population.

KeyWords: *Diabetes mellitus**,** Awareness, Complications, Rural, Urban*

Introduction

Diabetes is a chronic, multifactorial, multisystemic condition, characterized by hyperglycemia, and associated with disorders of protein, fat and lipid metabolism [1]. The disease is associated with high levels of morbidity and mortality levels, as a result of its micro and macro vascular complications.

India has come to be known as the Diabetic Capital of the world; with studies estimating that every fifth diabetic in the world is an Indian. Statistics reveal that by 2030, up to 79.4 million individuals, in India alone, will be afflicted by the disease [2,3]. This could have grave implications on the social and economic sector of the developing country. With most affected individuals expected to be in the 45 to 64 age group, the disease poses a threat to the productivity of working individuals, the consequences of which will fall on the sustainability of health care financing of the individual as well as the government. Moreover, conventional approaches to management of the disease, and prevention of its complications, are not only expensive, but also largely inaccessible to certain communities [4,5].

No longer considered a disease of the rich, its prevalence its rapidly increasing among the poor in urban slums, the middle areas, and those dwelling in rural areas. With poorer access to education and healthcare among people belonging to lower socioeconomic groups, it is the need of the hour to increase awareness about the nature and complications of the disease, with emphasis on prevention. Evidence based studies support the notion that patients' knowledge on the various aspects of the disease, importance of glycemic control and selfcare practices is key to prevention of complications, as they show more compliance to treatment [6,7].

A correlation between demography and awareness could provide an insight into level of awareness and its adequacy. In this context, the current study was planned with the objective of assessing the comparative level of awareness of diabetic patients residing in urban and rural areas of the city, regarding the complications of Diabetes. The results could help shed light on the areas where emphasis needs to placed in terms of planning public health programs.

Materials and Methods

Study design, setting, and participants:

This is a cross-sectional community- based study, carried out in November 2016. 100 adult patients diagnosed with Diabetes Mellitus were included. Out of 100, 50 were from urban area(Adyar, Nungambakkam) and 50 from rural(Kattupakkam, Karyanchavadi) in and around Chennai city, based on convenience sampling.

Study Tool

A predesigned questionnaire consisting of items on demographic profile including name, age, sex, level of education, employment statusand specific questions to assess their knowledge of complications, practices for preventing and management of complications, as well as their health seeking behavior. The questions used to obtain data were as follows:

1. Are you aware diabetes has complications? Yes/No
2. If yes, what were your sources of information? Physician/Newspaper/Television/Internet/Schools
3. Uncontrolled DM can cause rapid breathing with fruity odour. Yes/No
4. Uncontrolled DM can cause foot ulcers. Yes/No
5. Uncontrolled DM can cause decreased sensation in feet. Yes/No
6. Uncontrolled DM can cause eye related complications. Yes/No
7. Uncontrolled DM can cause kidney related complications. Yes/No
8. Uncontrolled DM can cause increased BP. Yes/No
9. Uncontrolled DM can cause heart disease. Yes/No
10. Uncontrolled DM can lead to stroke. Yes/No
11. Uncontrolled DM can cause coma. Yes/No
12. Which of the following practices do you follow to prevent complications?

a. Wearing closed shoes
b. Inspecting feet for cuts and bruises
c. Regular monitoring of blood sugar levels
d. Losing excess weight
e. Consumption of high fiber diet
f. Regular eye checkups

Methodology

Patients were explained purpose of the study, confidentiality assured and verbal consent was obtained before collection of data. The questionnaire was translated to local language and administered by interviewer.

Inclusion and Exclusion Criteria

All adult patients(aged 18 and above) diagnosed with Diabetes. No patient refused to participate.

Statistical Analysis

Data was compiled and analyzed on Excel 2016 version.

Results

The total number of surveyed participants were 100 (58 Male, 42 Female), as seen in figure 1, of which 50 belonged to rural areas of Kattupakkam and Karyanchavadi, and 50 belonged to urban areas of Adyar and Nungambakkam. The main feature of the study lies in the contrast between the two. The demographic characteristics of the study population is as tabulated in Table 1. The mean participants interviewed were above age of 45 years(73%), followed by those in the 35-45 year age bracket(23%), and only 4% of the interviewed population were below 35 years(Figure 2).

Data was collected and tabulated based on levels of literacy; participants were classified as illiterate, those who had received only primary level of education, those who had received up to secondary level of education, and those who were holders of a degree (Figure 3).

It was determined that majority of rural cohort had received only primary education (50%), while the urban population made up majority of the percentage who had received secondary education and degrees. On tabulating the participants' employment status, it was evident that it was higher among urban than the rural population (Figure 4).

When questioned on their sources of information (Figure 5), for both, the rural and urban population, majority reported their local physician to be their major source of information on the disease

Table 1: Demographic characteristics of study participants (n=100)

CHARACTERISTIC	FREQUENCY(n=100)
Gender	
Male	58
Female	42
Age	
<35 years	4
35-45 years	23
>45 years	73
Level of education	
Illiterate	3
Only primary	31
Upton secondary	34
Degree	32
Employment status	
Employed	64
Unemployed	36

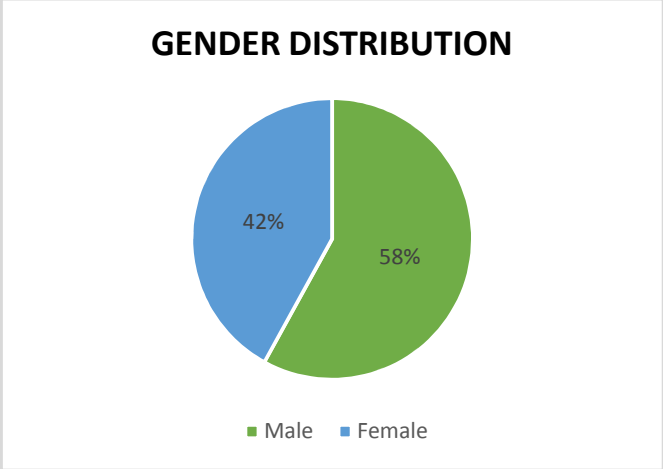


Figure 1: Gender distribution among study participants

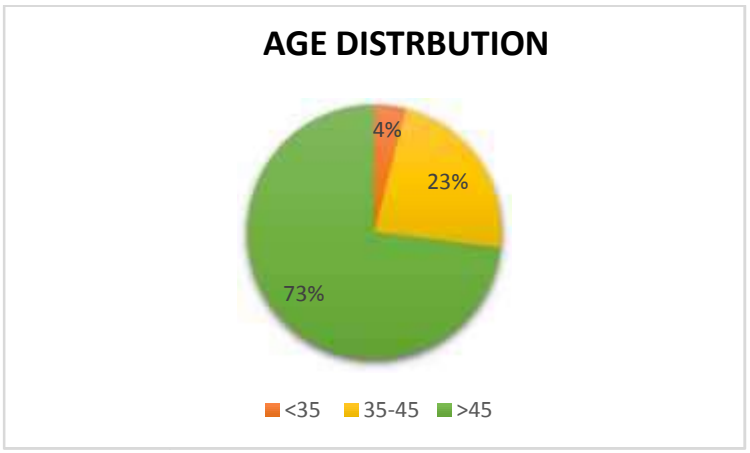


Figure 2: Age distribution among study participants (n=100)

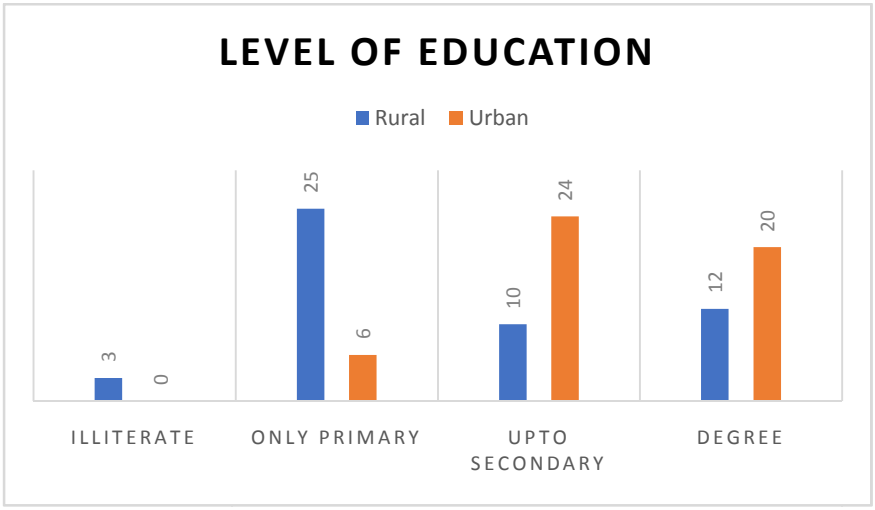


Figure 3: Educational qualification among rural(n=50) and urban population (n=50)

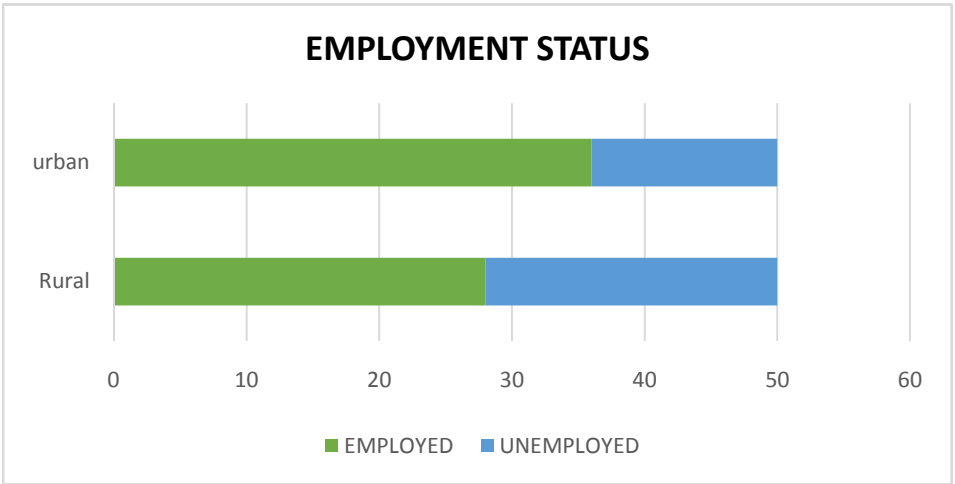


Figure 4: Employment status among rural and urban population

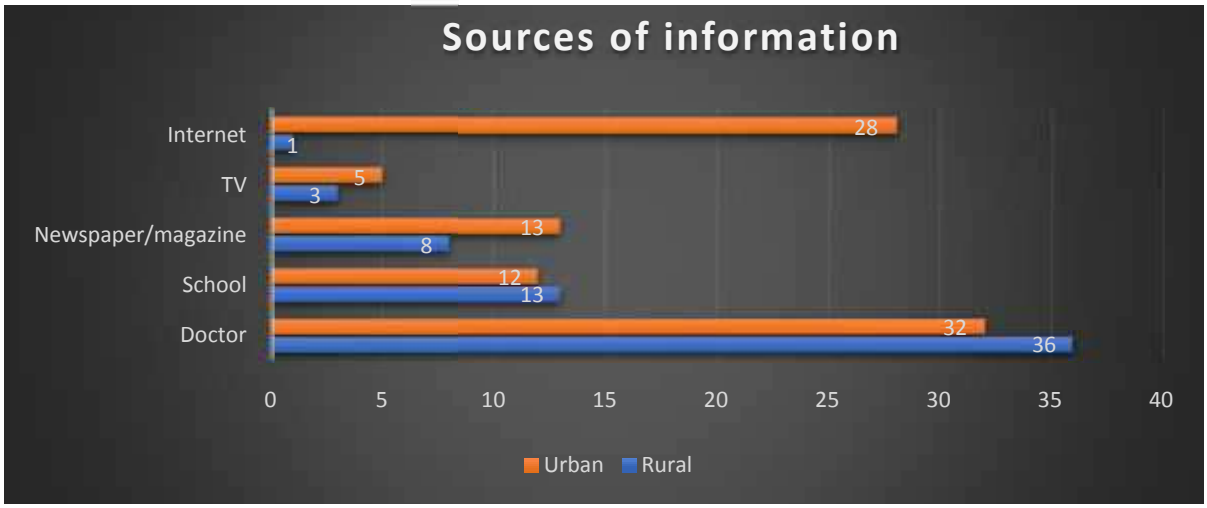


Figure 5: Sources of information for rural and urban population

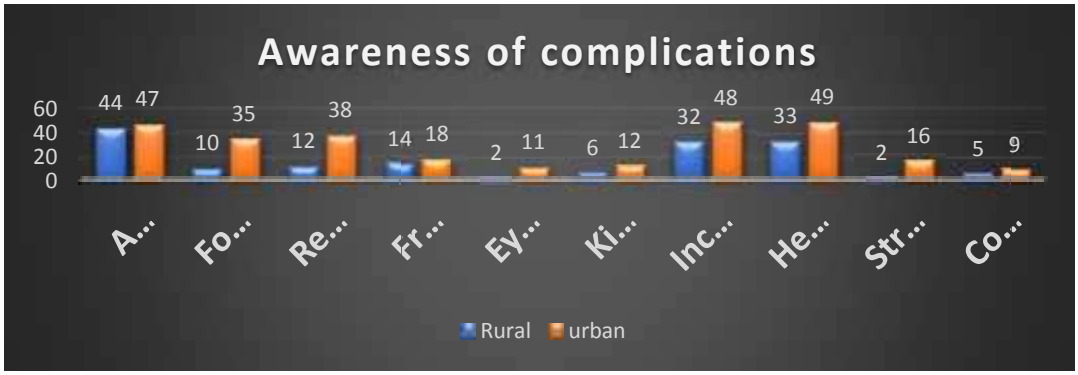


Figure 6: Awareness among rural and urban population on complication

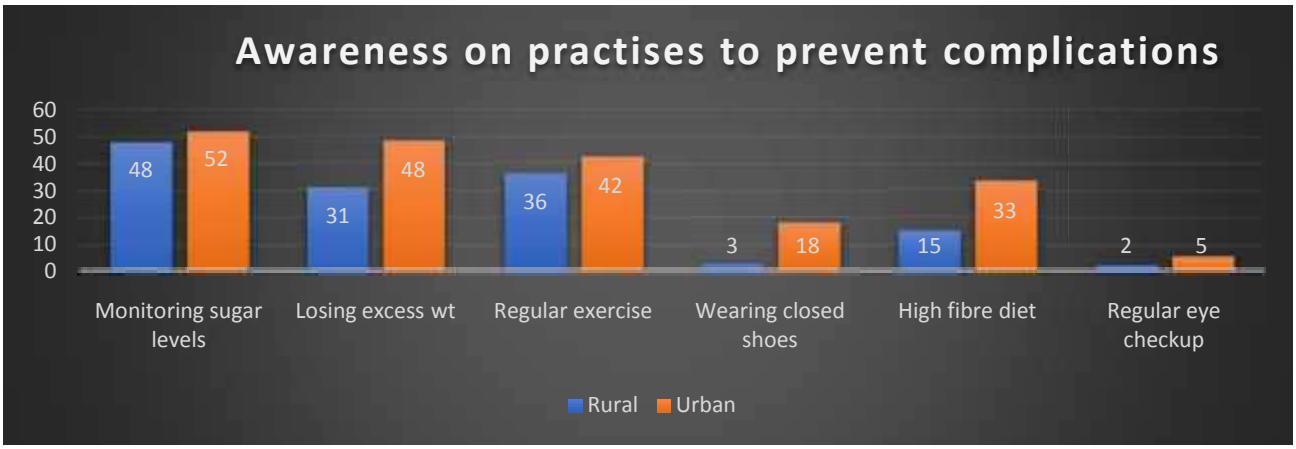


Figure 7: Awareness among rural and urban population on practices to prevent complications

Discussion

In this study, we sought to identify, investigate and evaluate by means of exploratory and evaluatory research, the awareness of urban and rural diabetic patients about the disease. The strategy was to administer a questionnaire that would test their knowledge attitudes and practices regarding the disease, its complications and management. The major finding is the general lack of awareness among Chennai residents, with urban diabetic residents much more knowledgeable compared to rural diabetic residents.

The results emphasize a need for imparting community based diabetic education to the general population and to the diabetic population in particular. Chennai, metropolitan city in Southern India, where this study was carried out, is considered a model city in terms of diabetes education activities. It is known for having the first ever diabetic clinic in India, established at the Stanley Medical Hospital in 1948[8]. It is also home to the highest number of government run diabetic clinics in the country. A survey conducted between 2004-07 revealed that over 770 diabetes awareness and free screening camps were conducted as part of its Prevention, Awareness, Counselling and Evaluation (PACE) Diabetes Project [9]. With such impressive credentials, it is a matter of concern when 18% of our interviewed participants were not aware that diabetes posed any serious threat to health. It is also worrisome to think where other cities might stand in terms of awareness when Chennai, after so many measures, shows unsatisfactory results.

The epidemic of non-communicable lifestyle diseases poses a major public health concern, in developing countries such as India. While India is considered the diabetic capital of the world, not enough is done to tackle the problem[10]. With more than 32 million diabetic subjects in India currently, the numbers are expected to double by 2013. Moreover, WHO reports the highest increase of affected individuals to be between the ages of 45-65 years; those falling into this age bracket are usually considered to contribute the most to the economic levels of the country[11,12]. Apart from being a huge economic burden to the country, a price cannot be placed on intangible costs that come with compromised mental health and poor productivity as a result of the disease. Coincidentally, in our study, the mean age of the participants was between the ages of 45-55 years.

Increasing amount of evidence indicates diabetes is a lifestyle disease, easily preventable by simple lifestyle alterations such as dietary modifications and improving exercise behaviors[13,14]. This has been evidenced in studies such as the Diabetes Prevention Programme, the Finnish Diabetes Prevention Study, and the Da Quingstudy[15]. A study by Tan et al showed that early educational intervention improved patients' knowledge and long-term control of the disease. Previous studies have shown a clear link between patient education and prevention of complication of diabetes[16,17].

The fact that self-reported diabetics in the study were generally unaware about the disease and its complications reveals an urgent need to implement programs to impart diabetes education to patients. The regional differences in awareness levels could be attributed to varying levels of education, with those from rural areas having limited access to resources to educate themselves. Those with no formal education had high unawareness rates compared to the educated group, which is in agreement with previous studies[18]. The urban population scored higher on their awareness about complications compared to the rural populations.

Patients were asked to cite their sources of information on the disease. The popular option among both the urban and rural population was the primary care physician. This goes to show that nationwide programs aimed at providing evidence -based guidance to physicians, general practitioners and diabetes educators, ensures improved education and treatment outcome for the patient. Two such efforts by the Indian government, namely Certificate Course in Evidence Based Diabetes Management [CCEBDM] and National Diabetes Educator's Program [NDEP] have made a difference to diabetes care and education[19]. It was also disappointing to note that neither group ranked schools very high on the list. It is crucial that the high-risk group, which is the next generation, is targeted at the level of primary education in school[20]. Early intervention makes a huge difference in disease control and prevention of complications. Maximum individuals from the urban cohort chose internet as their preferred source, which was in contrast to the rural cohort (1%). It was found that those having access to technology had higher health literacy compared to individuals with no access. Accessible technology is crucial to understanding health information and can act as an enabler in diabetes self -care management[21].

A positive correlation has been found between number of complications per patient and patients' healthcare expenditure[22]. The presence of complications weighed heavily on consultation, hospitalization and generally overall costs. A study carried out in Chennai comparing diabetes care costs for patients with and without complications has revealed that the total costs for patients from 2008-09 revealed that total costs incurred by patients without complications of the disease was INR 4493, compared to INR 14691.75 for patients with complications[23]. On investigating different types of complications, it was found that foot complications incurred the highest costs, followed closely by renal, cardiovascular and retinal complications[24].

The results of our study confirm the interrelation between awareness and socioeconomic status of the population. Our study observed that those belonging to a lower socioeconomic status were significantly less aware than those belonging to a higher class. A study by Shobana et al revealed low income individuals bore the highest burden of diabetes[25]. However, it was found in a study done in India on type II DM, from 1998 to 2005 that the urban population showed higher expenditure numbers in terms of absolute terms and as proportion of income [26]. This could be attributed to higher charges for consultation, laboratory tests and drugs, and more expensive treatments in urban areas. These individuals, being economically better off, were found to seek healthcare in private hospitals with better healthcare facilities, and hence, faced better prognosis. On the other hand, those from lower socioeconomic strata could only afford public hospitals funded by the state; these were found to be crowded, with overworked staff and insufficient treatment options. The rural population were found to spend significantly lesser, more due to issues of inaccessibility and affordability, rather than because of a lower need. A study by Rayappa found that unemployed, uneducated people from rural areas were more likely to be diagnosed later as they could not afford to consult a doctor and thus, developed complications [27]. Thus, early detection would provide as a means of cost reduction.

The participating individuals were questioned on their practices of prevention. Misconceptions due to inadequate education on the matter drives misguided perceptions on eating, food habits and exercise behavior[28,29] Changing societal perceptions of health would drive the community towards making healthier life choices. With rapid epidemiological transition to urban areas, compounded by industrialization and rapid pace of growth, developing countries are most affected. A consequence of that being that communicable diseases are replaced by non-

communicable disease[30]. Both cohorts were well aware of the importance of regularly monitoring blood sugar levels. However, urban population were more aware of the practice of losing excess weight. In a developing country such as India, which struggles with malnutrition and poverty, obesity is seen as a sign of good health and prosperity[31]. However, with demographical transitions, obesity poses as a major public health concern, with underweight individuals being replaced by obese. Thus, rural individuals must be made aware of the dangers of excess weight and the benefits of exercise.

The study emphasizes on a need for comprehensive diabetes education programs and community level awareness programs. Mobile health units can be implemented to access remote areas of the country. Mass media campaigns, public lectures and door to door campaigns can also make a difference. There is a need for policy makers to review existing health coverage schemes and concentrate on reducing economic burden of the disease.

The limitation of the study lies in its small sample size. While these values may provide a slice into the scenario in Chennai, extrapolating these values on a larger scale to generalize them to the entire Indian subcontinent may result in erroneous perceptions of the impact of the disease. Thus, the authors recommend more nationwide cross-sectional studies covering larger populations.

Conclusion

The present study provides direct evidence that urban diabetic patients are more aware than rural diabetic patients about diabetic mellitus and its complications. It is imperative that diabetes awareness campaigns are executed to provide education to the masses.

Corresponding Author

Nabeel Nazar M

Reader, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email ID: nabeelnazarm@gmail.com

References

1. Nayak B S,Roberts L. Relationship between inflammatory markers, metabolic and anthroprometric variables in the Carribean type 2 diabetic patients with and without microvascular complications. J of Infl. 2006 ; 3:17-17
2. Arun Kumar, Manish K Goel, Ram Bilas Jain, Pardeep Khanna and Vikas Choudhary. India towards Diabetic control: Key issues. Australas Med J. 2013; 6(10): 524-31
3. Seema Abhijeet Kaveeshwar, Jon Cornwall. The current state of diabetes mellitus in India. Australas Med J. 2014 ;7(1):45-48.
4. World Health Organization. Diabetes mellitus. Fact sheet N°1382014 [16.10.2014]; Available from: http://www.who.int/mediacentre/factsheets/fs138/en/.
5. Ashwini S, Anitha R. Antihyperglycemic activity of Caralluma fimbriata: An In vitro Approach. Phcog Mag.2017; 13(S3):499-504
6. Visser A, Snoek F. Perspectives on education and councelling for diabetic patients. Patient Educ Couns. 2004;53:251-5.
7. Rani PK, Raman R, Subramani S, Perumal G, Kumaramanickavel G, Sharma T. Knowledge of diabetes and diabetic retinopathy among rural populations in india, and the influence of knowledge of diabetic retinopathy on attitude and practise. Rural remote health. 2008; 8:838.
8. Deepa, Mohan & Raj, Deepa & S Shanthirani, C & Datta, Manjula & C Unwin, N &Kapur, Anil & Mohan, Viswanathan. Awareness and knowledge of diabetes in Chennai – The Chennai Urban Rural Epidemiology Study [CURES-9].JAssoc Physicians India. 2005; 53:283-7.
9. Somannavar S, Lanthorn H, Deepa M, Pradeepa R, Rema M, Mohan V. Increased awareness about diabetes and its complications in a whole city: effectiveness of the "prevention, awareness, counselling and evaluation" [PACE] Diabetes Project [PACE-6]. J Assoc Physicians India. 2008 Jul; 56(3):497-502.

10. Pradeepa R, Mohan V. The changing scenario of the diabetes epidemic: implications for India. Indian J Med Res. 2002; 116: 121-32
11. Smyth S, Heron A. Diabetes and obesity: The twin epidemics. Nat Med.2006;12:75-80
12. Gayathri Devi Kumaresan**,** M Subha. Awareness on oral disorders in diabetes mellitus among general population in Chennai. Int J Orofac Res 2017;2:28-3.
13. Ramachandran A, Snehalatha C, Baskar AD, Mary S, Kumar CK, Selvam S, Catherine S, Vijay V.Temporal changes in prevalence of diabetes and impaired glucose tolerance associated with lifestyle transition occurring in the rural population in India.Diabetologia. 2004 May; 47(5):860-5
14. ADeshpande AD, Harris-Hayes M, Schootman M. Epidemiology of diabetes and diabetes-related complications. Phys Ther. 2008 Nov; 88(11):1254-64.
15. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, Nathan DM. Diabetes Prevention Program Research Group. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med 2002; 346:393-403.
16. Bjork S, Kapur A, King H, Nair J, Ramachandran A. Global policy: aspects of diabetes in India.Health Policy. 2003 Oct; 66(1):61-72.
17. KY Tham, JJY Ong, DKL Tan, KY How. How much do diabetic patients know about diabetes mellitus and its complications? Annals Academy of Medicine 2004; 33(4): 503-509
18. Caliskan D, Ozdemir O, Ocaktan E, IdilA.Evaluation of awareness of diabetes mellitus and associated factors in four health center areas.Patient Educ Couns. 2006 Jul; 62(1):142-7.
19. Joshi S, Joshi SR, Mohan V. Methodology and feasibility of a structured education program for diabetes education in India: The National Diabetes Educator Program.Indian J Endocrinol Metab. 2013 May; 17(3):396-401
20. Li G, Hu Y, Yang W, Jiang Y, Wang J, Xiao J, Hu Z, Pan X, Howard BV, Bennett PH et al. Effects of insulin resistance and insulin secretion on the efficacy of interventions to retard development of type 2 diabetes mellitus: the DA Qing IGT and Diabetes Study. Diabetes Res Clin Pract2002; 58:193-200.
21. Divya Ramasamy, Awnish Kumar Singh, Surapaneni Krishna Mohan, Meenakshi N. Association of sociodemographics, technology use and health literacy among type 2

diabetic individuals living in an Indian setting: an exploratory cross-sectional study.Int J Diabetes Dev Ctries (2016) 36: 303-12. https://doi.org/10.1007/s13410-015-0444-7

22. Ramachandran A, Ramachandran S, Snehalatha C, Augustine C, Murugesan N, Viswanathan V, Kapur A, Williams R. Increasing expenditure on health care incurred by diabetic subjects in a developing country: a study from India. Diabetes Care. 2007;30(2):252–256. doi: 10.2337/dc06-0144
23. Kumpatla S, Kothandan H, Tharkar S. The costs of treating long term diabetic complications in a developing country: a study from India. JAPI. 2013;61:17
24. Goutham Kesavamoorthy,Awnish K Singh, Shruti Sharma, Jyoti BalaKasav, Surapaneni Krishna Mohan and Ashish Joshi. Burden of Diabetes Related Complications Among Hypertensive and Non Hypertensive Diabetics: A Comparative Study.J Clin Diagn Res. 2015 Sep; 9(9): LC10–LC14.
25. Shobhana R, Rao PR, Lavanya A, Vijay V, Ramachandran A. Cost burden to diabetic patients with foot complications–a study from southern India. J Assoc Physicians India. 2000;48(12):1147
26. Shobhana R, Rama Rao P, Lavanya A, Williams R, Vijay V, Ramachandran A. Expenditure on health care incurred by diabetic subjects in a developing country–a study from southern India. Diabetes Res Clin Pract. 2000;48(1):37–42. doi: 10.1016/S0168-8227(99)00130-8
27. Rayappa: The economic cost of diabetes care the bangalore urban district diabetes study**.**Int J Diab Dev Ctries. 1999, 19**.**
28. A Joshini Shanmugam, AnithaRoy.Knowledge and Awareness of Insulin Usage among Diabetic Patients in Chennai.Int. J Sci Res. May 2017. 6(5): 889-91
29. Kiren J,VishnuPriya,GayathriR.Awareness of Balanced Diet among Diabetic Patients: A Survey.J Pharm. Sci. & Res. 2017; 9(2): 245-7
30. Popkin BM, Adair LS, Ng SW.Global nutrition transition and the pandemic of obesity in developing countries.Nutr Rev. 2012 Jan; 70(1):3-21
31. Soo KL, Wan AM, Abdul MH, Lee YY.Dietary practices among overweight and obese Chinese children in Kota Bharu, Kelantan.Malays J Nutr. 2011 Apr; 17(1):87-95

Awareness of Oral and Maxillofacial Surgery among General Population

AshikAhamedA [1], Dinesh Prabu M 2, Pradeep D [3],

Graduate Student [1], Senior Lecturer [2],Department of Oral and Maxillofacial Surgery,

Senior Lecturer [3], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction : Oral and Maxillofacial Surgery (OMFS), a dentistry specialty recognized by the Federal Dentistry Board in the mid-1960s, is responsible for the diagnosis, and clinical and surgical treatment of traumatic, congenital, developmental and iatrogenic lesions in the maxillofacial complex. Even today, difficulties are experienced owing to the lack of knowledge of the general public and health professionals concerning the scope of OMFS.Oral and Maxillofacial Surgery had a very strong development in past areas,such as facial trauma,dentofacial deformities,jaw pathologies,temporary mandibular joint disorders and oral cancers. The aim of this study is to assess the awareness among Medical professionals about oral and maxillofacial surgery.

Materials and Methods: The questionnaire was intended to determine the percentage of respondents in each group that made the correct referral of patients presenting with different clinical situations.

Results: The results of our present study shows there is a lack of awareness regarding oral and maxillofacial surgery among general population.

Conclusion: This study is to create awareness and to assess the awareness in Oral and Maxillofacial Surgery among Medical professionals.

Key Words:*Awareness, General population, professionals, Surgery*

Introduction

Oral and maxillofacial surgery is one of the emerging speciality in dentistry. OMFS specialises in treating many diseases, injuries , defects in the head, neck, face,jaws, and the hard and soft tissues of the oral and Maxillofacial region. It is an internationally recognised surgical speciality . In some countries around the world, including the United States , Canada and Australia ,it is a recognised speciality of dentistry. But in others such as the UK and the most of Europe, it is recognised as both a speciality of medicine and dentistry and you need to get both the degree in medicine and dentistry to practice.

The scope of the speciality has also increased vastly as compared to earlier years where it was restricted to mainly dento-alveolar surgery and basic maxillofacial trauma. Currently dento-alveolar surgery, maxillofacial trauma, pathology of head and neck, cleft lip and palate, bone grafting ,facial deformity correction, aesthetic facial surgery, TMJ surgery, implant surgery etc.,are just some of the sub specialities being treated by maxillofacial surgeons all around India.

In spite of all these advances in maxillofacial surgery, it is still vaguely understood by both the dental and medical fraternities . The awareness is even worse among the general population. Previous studies conducted in the USA and the UK have reported that the medical and dental fraternity and the general public are largely unaware of the scope of oral and maxillofacial surgery[1][2]. Ameerally et al [1],conducted a study in England and concluded that up to 79% of general population had not heard of OMFS, and around 74% did not understand its role and scope. Ifeacoetal[3]concluded that most of the medical professionals had heard of this particular speciality, but they were not clear about the clinical expertise this branch could offer. In a study by Hunter[4], it was found that medical professionals, who did not know about this branch, refer their patients for treatment of conditions that overlapped different specialties mostly did not favour the choice of an oral and maxillofacial surgeon.

If the awareness even in developed countries is lacking, the situation in a developing country like India is bound to be worse. We therefore need to study to check the awareness of the oral and maxillofacial surgery among general population in India.

Materials and Methods

Sampling and Survey Instruments and Statistical Analysis

The study was done in Saveetha dental College and Hospital, Chennai, TamilNadu, India to assess the current awareness levels about the oral and maxillofacial surgery inthe Indian society. The randomised cross-sectional study was conducted among the generalpopulation of about 105 participants. A questionnaire was prepared in English, that listed 10general questions which focused on the knowledge about the oral and Maxillofacial surgery.

The questionnaire consisting of 10 questions were given to the participants.The sample of 105 was a randomly selected Group of individuals who would beconsidered to represent the whole population. They were selected after applying a multi-stagerandom sampling method for selection. They were previously asked questions before the actualquestions in the questionnaire in order to avoid bias in our study. The individuals who had anyrelations or friends in the dentistry department were omitted .The data have been collected through pre designed self administeredquestionnaire.The survey had utilised the Surveyplanet website app. Through the app the survey had beentaken, the collected data were organised and analysed . The questionnaire consisting of 10questions were given to 105 participants. The 105 participants were then asked to answer allthe questions. The questionnaires were then reviewed, analysed and evaluated. The results were collected andanalysed using Apple numbers software app.

Objective

The questionnaire was intended to determine the percentage of respondents in each age group that have the correct knowledge about the OMFS.The aim of this survey was to investigate the current level of awareness about OMFS in different age groups.

Results

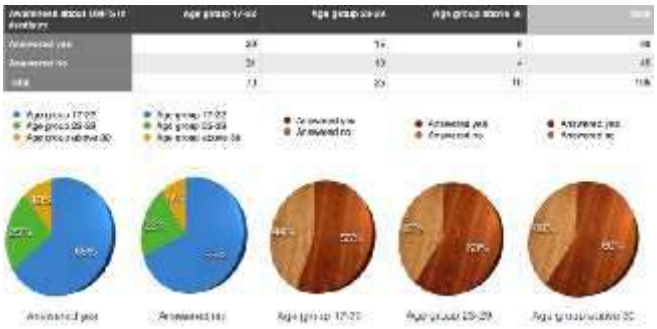
Only the completely filled questionnaires were selected for the study. The incomplete questionnaires were rejected to avoid confusion in derivingthe results .The number of participants were different in all the age groups but the proportion of male and female participants were equal in the age groups. This probably would avoid the inequality in the distribution of the sample size in each agegroup.The demographic data of the participants were analysed. The age group division was to find out the awareness among the general population. Results for the three groups of participants were given in the tables below. In this study also, male participants were more predominant comparing the female participants. In all the age groups, about 60% of male are participants. Of the total 105, 70 belongs to age

group 17 to 22, 25 belongs to age group 23 to 29, 10 belongs to age group above 30.The gender distribution, male and female, was found to be almostequalin all age groups, whereas the predominant gender was male in each age group.(Table1)

About 57% of participants knew about OMFS in dentistry. Among the participants who knew OMFS, 65%are from age group 17 to 22, 25% are from age group 23 to 29,10% from the above 30 age group.Overall , almost 60% in each age group answered yes which shows almost 60% of people in every age group knew about the OMFS in dentistry which may be due to the name factor.(Table 2)

Table 1–Participant's distribution among different age groups.

Table 2–Knowledge about OMFS in Dentistry depending on the age group.



About 63%(66/105) of sample population were aware that a dentist can treat oral cancer. In the category who had answered yes, about 57% of age 17 to 22 were aware that a dentist can treat oral cancer and about 29% of age group 23 to 29 were aware that a dentist can treat cancer . In age group of 17 to 22,about 54% were aware that the dentist can treat oral cancer.(Table 3)

About 64%(67/105) of participants knew that a dentist can do maxillofacial surgery. About 67% Who knew that the dentist can do maxillofacial surgery were from age-group 17 to 22. About 64% of age group 17 to 22 knew that a dentist can do maxillofacial surgery. About 25% of age group 23 to 29 were aware that a dentist can do maxillofacial surgery.(Table 3)

About 62% of participants were aware that the dental related surgical procedures were done under anaesthesia. In the sample who knew that dental related surgical procedures were done under anaesthesia , about 69% were from age group 17 to 22 and 23% were from age group 23 to 29 and 8% were from age group above 30. About 64% in age group 17 to 22 knew this.About 60% in age group 23 to 29 were aware of this.(Table 3)

Table 3–Participant's knowledge aboutprocedures and treatment that could be provided by the dentists.

Are you aware that your dentist can treat oral cancer	Age group 17-22	Age group 23-29	Age group above 30	Total
Answered yes	(57%)38	(20%)19	(14%)9	66
Answered no	32	6	1	39
Total	70	25	10	105
Percentage of answered yes in the specific group	54%	76%	90%	
Are you aware that a dentist can do maxillofacial surgery	Age group 17-22	Age group 23-29	Age group above 30	Total
Answered yes	(67%)45	(25%)17	(8%)5	67
Answered no	25	8	5	38
Total	70	25	10	105
Percentage of answered yes in the specific group	64%	68%	50%	
Are you aware that dental related surgical procedures done under anaesthesia	Age group 17-22	Age group 23-29	Age group above 30	Total
Answered yes	(69%)45	(23%)15	(8%)5	65
Answered no	25	10	5	40
Total	70	25	10	105
Percentage of answered yes in the specific group	64%	60%	50%	

About 16% of 105 participants had undergone maxillofacial surgery in their lifetime . Of the people who have undergone ,59% are from age group 17 to 22.This higher value might be due to the sample size bias favouring the first group . Only 14% in the age group of 17 to 22 had undergone maxillofacial surgery. About 16% in the age group of 23 to 29 had undergone the maxillofacial surgery, hence they be aware of this OMFS. About 30% in the age group of above 30 had undergone maxillofacial surgery.

The number has increased when it comes to the family members and friends. About 20% of 105 participants had family members or friends who had undergone maxillofacial surgery. Of the people who have such family or friend, about 57% are from age group 17 to 22 which would also fall under thebias.About 17% in age group 17-22 had such family or friend. About 28% in age group of 23 to 29 had family members of such category. But in the age group of above 30, the number has decreased to 20%, this may be due to the limited number of sample in the group.(Table 4).

Table 4– Percentage ofParticipants and their family members who have undergone Maxillofacial surgery in study population.

Have you ever undergone any maxillofacial surgery	Age group 17-22	Age group 23-29	Age group above 30	Total
Answered yes	(59%)10	(24%)4	(17%)3	17
Answered no	60	21	7	88
Total	70	25	10	105
Percentage of answered yes in the specific group	14%	16%	30%	
Has anyone in your family undergone maxillofacial surgery	**Age group 17-22**	**Age group 23-29**	**Age group above 30**	**Total**
Answered yes	(57%)12	(33%)7	(10%)2	21
Answered no	58	18	8	84
Total	70	25	10	105
Percentage of answered yes in the specific group	17%	28%	20%	

About 57% of total sample have known that the jaw related fractures are treated by maxillofacial surgeon. About 63% of total sample have known that cleft lip could be treated by dentist. About 66% of total sample have known that the maxillofacial surgeon could treat dental trauma. About 55% of total sample have known that the OMF surgeon could do Aesthetic jaw bone correction.(Table 5)

Table 5 – Percentage distribution of study population regarding the knowledge about the specific treatments and procedures in OMFS done by dentists and OMF surgeons.

Knowledge about OMFS	Age group 17-22 Answered yes	Age group 23-29 Answered yes	Age group above 30 Answered yes	Total Answered yes	Total answered no
Jaw related fractures are treated by maxillofacial surgeon	40 (30 answered no) (69% belonged to this group who answered yes)(57%of this group answered yes)	15 (10 answered no) (25% belonged to this group who answered yes)(60%of this group answered yes)	5 (5 answered no)(8% belonged to this group who answered yes) (50%of this group answered yes)	60(57%of total)	45
A Dentist can treat cleft lip	40 (24 answered no) (70% belonged to this group who answered yes)(66%of this group answered yes)	13 (12 answered no) (20% belonged to this group who answered yes)(52%of this group answered yes)	7 (3 answered no) (10% belonged to this group who answered yes) (70%of this group answered yes)	66(63%of total)	39
A maxillofacial surgeon can treat dental trauma	46 (22 answered no) (69% belonged to this group who answered yes)(59%of this group answered yes)	16 (9 answered no) (23% belonged to this group who answered yes)(64%of this group answered yes)	8 (4 answered no)(8% belonged to this group who answered yes) (60%of this group answered yes)	70(66%of total)	35
OMF surgeon can do aesthetic Jaw bone correction	40 (30 answered no) (69% belonged to this group who answered yes)(57%of this group answered yes)	15 (10 answered no) (26% belonged to this group who answered yes)(60%of this group answered yes)	3 (7 answered no)(5% belonged to this group who answered yes) (30%of this group answered yes)	58(55%of total)	47

Even though the knowledge percentage of general population sample in our study had shown increased results, the numbers are not denoting the actual knowledge of sample knowing the actual difference between the maxillofacial surgeon and the dentist. The sample might have understood that a dentist would do care dental problems, and maxillofacial surgeon to facial problems. According to

thenominalsuggestions,the sample might have answered the questions. The results were given in the table.

Discussion

Among the groups, even though the age group of 17 to 22 was greater in number than the others, the distribution was somewhat similar in each age group. About mostly 60% in all the age groups, were aware of the speciality. Most of the findings in this article would not surprise older and practising maxillofacial surgeons. It does bring home reality that OMFS speciality is not easily recognised by the public[5].In study done by Reddy et al[6], found that all study groups were not clear about the scope of the specialty and its capabilities. This may be true as the general population may know about the speciality but they are not aware of the scope of the speciality and what the speciality could do to them.There's not much a difference between each age groups in our study, and also there's not muchdifference between the results in each age groups .

Two limitationswithregardtoourquestionnairewere :itwasinEnglishwhich made it difficult for some of the participants , and also, the questionnaire required time andconcentration to complete,andit was difficult for the respondents due to their inability to know about the survey but we took sure amount of struggle in order to make the participants knew about the survey and the Surveyplanet website app had helped to reach therequired participants. As the strength of sample size was more in the age group of 17 to 22, the results are slightly biased towards this group than the other group, favouring the group 17 to 22. This may be slightly true as today's resources are readily available to the teens as most of the social media are the major source of information. The older generation might have heard this speciality from their friends or family members who have gone to the OMFS doctors or by personal experience being referred to the OMFS specialist by the generalphysicians[23].For which the general physicians should know more about the OMFS specialty. Apparently, some students and professionals are not aware of the wide surgical scope of the specialty. A majority of health-care professionals recognise OMFS, but some students and professionals are not aware of the wide surgical field of the specialty [20][21][22] .

The nominal suggestions of the speciality played a major part in distortion of results towards the higher knowledge among general population which is good in some manner but is not worth without the actual knowledge about the scope of the speciality .Although this does not mean that no effort should be made to inform the public about what OMF surgeons do, it does indicate that no name alone can ever be completely descriptive [18][19].

Oral and maxillofacial surgery initially started as a branch of surgical dentistry by dentists who had a special interest in surgery. They were then known as oral surgeons. In World War II, the specialty increased its remit to include maxillofacial trauma by a few dedicated units in the UK and the USA. Then over a period of time, they increased their scope to include facial deformity correction, pathology and so on to reach its current position. By this time the specialty was known as oral and maxillofacial surgery. In certain countries it is now mandatory to get both a medical and dental qualification before becoming a maxillofacial surgeon. Whether this dual qualification is needed to practice maxillofacial surgery or whether it is used as a "political" weapon to remove the tag of a "dentist" is still under debate. The dental and medical association hears this topic on a regular basis in nearly every conference.

In India, lately there has been a vast increase in the number of trainees in Oral and maxillofacial surgery and this has improved the visibility of the specialty. This is mainly due to the increase in the number of college seats available at a postgraduate level lately. This study shows that it is very important to promote the specialty among the general population and also in the media. Having a simpler name could be the first step in making the specialty well known. Otolaryngologists have now changed their name to ENT surgeons, making it easier to pronounce and remember[13][3][16][17].

Although OMFS is a branch of dentistry, it is one of the specialties that bridges to the medical community so closely. Current trend shows that a full medical degree is not that necessary for OMFS specialists to practice with competency. OMFS specialists are qualified to treat not only dento-alveolar surgeries, but also a full scope of jaws and facial surgeries including facial fractures, congenital facial deformities, temporo-mandibular joint (TMJ) disorders, pathologies related to the jaws, salivary gland diseases, facial reconstructions with extra-oral bone or soft tissue harvesting. With adequate training or, in some situations, collaborating with other specialties, the scope could be expanded to cranial-facial reconstructions, oncology and reconstructions using local or distant microvascular free flaps. Although the specialty involves in the treatment of a lot of important conditions or diseases, it is disappointed to see the lack of awareness from the general public. It was reported that there were only 11.5 % who had ever heard of and knew what OMFS was according to an older study.Although this is the case, in general, there is an increasing trend towards the recognition of Oral and Maxillofacial Surgery as a specialised branch for the treatment of many conditions as compared to the past[13][14][15][16] .

According to the older studies, OMFS was introduced to a majority of people (65%) by a friend or a family member. This indicates that most of the knowledge was by personal experiences of people around. There was almost no public image about this specialty. The government or tertiary institute could

publicise this field by media like TV, radios or newspapers through interviews or in terms of case study. The local association should also take a leading role to arouse the public awareness regarding our specialty, either by organising public talks or distributing leaflets / booklets / magazines regularly. In our study, the awareness has increased as the information about the speciality are readily available through Internet.There are 58% of interviewers answered yes to the question that OMFS belongs to the field dentistry. It is not surprising as the name OMFS itself indicated that is something about the oral cavity, thus dentistry.

Parnes [7] stated that the governing bodies of the American Association of Oral and Maxillofacial Surgery formed a task force to discuss a possible name change for the specialty. Any change from the current name was rejected at that time. One of the concerns over changing the name was that another specialty of dentistry or medicine might adopt the abandoned name.

Laskin et al. [8] evaluated the knowledge of 12 different specialties to determine whether such unfamiliarity is true only for OMFS, or whether it occurs with other specialties also. The result of their study shows that every effort should be made to inform the public about what OMF surgeons do.

Clinical entities like facial asymmetries are a great challenge to any operating surgeon. Orthognathic surgical procedures may be needed in correcting such dentofacial deformities. This requires a multidisciplinary approach that includes orthodontists, periodontists, and prosthodontists. An OMFS would live up to the realistic expectations of the patient by giving a desired aesthetic look, simultaneously securing the occlusion. Of great concern is the undeniable fact that most of the general public (35%) are unaware of this fact. In this regard, 25% of the general public and 33% of the medical professionals preferred plastic surgeons. Similar findings were observed by Ifeacho et al [3][12].

A cross-sectional study done by Rastogi et al. [9] in Manipal Teaching Hospital, showed that medical professionals would like to consult OMFS for fracture of mandible, maxilla and zygoma. Also, OMFS scored an absolute majority in clinical situations like dental implant and removal of wisdom tooth and around 76 % for mandibular reconstruction.

The present study, nevertheless, has shed light on what the perception is of the domain of the OMFSamongthe general public. Procedural awareness mentioned within the questionnaire was significantly still weak. The Association of Oral and Maxillofacial Surgeons should take a leading role to arouse public awareness concerning the specialty, by audit, publications, or organising public talks or camps regularly. Educational institutions are the leading lights to promote awareness. This would have an

effect on the dynamics of the entire system, not only to the patients or the public, but also to the doctors who provide the treatment.The Association of Oral and Maxillofacial Surgeons of India (AOMSI) observe February 13th as the day of OMFS to commemorate its first meeting held on this date in 1969. The objective of this initiative is to bring about increased awareness in the general public about the advances the specialty has made. This we feel needs to be followed and expanded.

Greater progress needs to be made in the education of medical and dental students as well as the general public if the specialty of OMFS is to be practiced to its full potential [10, 11].

Conclusion

In summary, there is a very low awareness in OMFS to the general public in terms of its scope of service, the provision of service and training by the government and institutes. It is very discouraging to learn that most of the people do not really knowOMFSas they may miss the chance to be treated or to be trained. There is no doubt that the specialty deserves a better public awareness. The stress should be put on three areas, namely the public image of who OMF surgeons are, awareness of the treatment OMF surgeons could provide, the awareness of the details of training pathways and opportunities. The objectives are to let more people know who they should seek help from and provide open opportunities for people to be trained.

The surprising fact in this matter was our colleaguesin Otorhinolaryngology (ENT) and plastic surgery are well known than OMFS. The worrying aspect is that the plastic surgeons would not be found easily in the city but they are well-known. On the other hand, OMFS specialists could be found in the city hospitals but they are not known to exist to the general populations. This survey was performed only in the Northern region of Chennai areas, and therefore the results may not be applicable elsewhere. Regional variations exist, and surgeons are responsible for educating their own community and referral circles about the scope of their practice, which will depend on the training, experience and areas of interest. It is clear that greater progress needs to be made in the education of medical and dental students, as well as the general public, if the specialty of OMFS is to be practiced to its full potential[10][11].

Corresponding Author

Dinesh Prabu M,Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University,162, Poonamallee High Road, Chennai 600077,

Tamil Nadu, India.

Ph: +919790798719

Email: rogerprabu@gmail.com

References

1. Ameerally P, Fordyce AM, Martin IC. So you think they know what we do? The public and professional perception of oral and maxillofacial surgery. Br J Oral Maxillofac Surg. 1994;32:142–145.
2. Michael JH, Rubeiz T, Rose L. Recognition of the scope of oral and maxillofacial surgery by the public and health care professionals. J Oral Maxillofac Surg. 1996;54:1227–1232.
3. IfeachoSN,MalhiGK,JamesG.Perceptionbythepublicandmedicalprofession of oralandmaxillofacial surgery hasitchangedafter10years?BrJOralMaxillofacSurg2005;43:289–93.
4. HunterMJ,RubeizT,RoseL.Recognitionofthescopeof oral andmaxillofacial surgery bythepublicandhealthcareprofessionals.JOralMaxillofacSurg1996; 54:1227–32.
5. Rangarajan S, Kaltman S, Rangarajan T, Lopez E. The general public's recognition and perception of oral and maxillofacial surgery. Oral Surg Oral Med Oral Pathol Radiol Endod 2008;106:15.
6. Reddy K, Adalarasan S, Mohan S, Sreenivasan P, Thangavelu A. Are people aware of oral and maxillofacial surgery in India? J Maxillofac Oral Surg. 2011;10(3):185–189.
7. Parnes EI. Recognition of the scope of oral and maxillofacial surgery by the public and health care professionals—discussion. J Oral Maxillofac Surg. 1996;54:1233.
8. Laskin DM, Ellis JA, Jr, Best AM. Public recognition of specialty designations. J Oral Maxillofac Surg. 2002;60:1182–1185.
9. Rastogi S, Dhawan V, Modi M. Awareness of oral and maxillofacial surgery among health care professionals—a cross sectional study. J Clin Diag Res. 2008;2:1191–1195.
10. Brennan DS, Spencer AJ, Singh KA, Teusner DN, Goss AN. Practice activity trends among oral maxillofacial surgeons in Australia. BMC Health Services Res. 2004;4:37.

11. Dodson TB, Guralnick WC, Donoff RB, Kaban LB. Massachusetts general hospital/Harvard Medical School MD oral maxillofacial surgery program: a 30-year review. J Oral Maxillofac Surg. 2004;62:62–65.
12. Vadepally AK, Sinha R, Uppada UK, Reddy BV RK, Agarwal A public. J Cranio Max Dis 2015;4:21-7.
13. Sherick DG, Buchman SR, Patel PP. Pediatric facial fractures: Analysis of differences in subspeciality care. PlastReconstr Surg 1998; 102: 28.
14. Strother EA: Maxillofacial surgery in World War I: The role of the dentists and surgeons. J Oral Maxillofac Surg 2003; 61:943.
15. Simpson DA, David DJ: World War I: The genesis of craniomaxillofacial surgery? ANZ J Surg 2004; 74:71.
16. Le BT, Holmgren EP, Holmes JD, et al: Referral patterns for the treatment of facial trauma in teaching hospitals in the United States. J Oral Maxillofac Surg 2003; 61:557.
17. Bowers D. Statistics from scratch——an introduction for health care professionals. Oxford, Wiley; 2000.
18. Spina AM, Smith TA, Marciani RD, Marshall EO. A Survey of resident selection procedures in Oral Maxillofacial Surgery. J Oral Maxillofac Surg 2000; 58: 660–66.
19. Szuster FSP, Nastri AL, Goss AN, Spencer AJ. Survey of Australian and New Zealand Oral Maxillofacial Surgery trainees and recent specialists—education and experience. Int J Oral Maxillofac Surg 2000; 29: 305–08.
20. Lesny RJ. A Survey of resident selection procedures in Oral Maxillofacial Surgery. J Oral Maxillofac Surg 2000; 58: 666–67.
21. Langdon JD: Training for oral and maxillofacial surgery, academic oral surgery, and surgical dentistry in the United Kingdom. J Oral Maxillofac Surg 2006; 64:1803.
22. Henderson D, Liversedge RL, James PL. A study of dentally qualified medical graduates. Br J Oral Surg 1977; 15:173–84.
23. Willis DHR, Tong DC, Thomson WM, Love RM: Maxillofacial trauma and the GDP – spe- cialty recognition and patterns of referral. New Zealand Dental Journal 2010; 106(3): 97-102.

Awareness of Root Coverage Techniques and Their Implementation in Clinical Practice among Periodontists in Chennai – A Questionnaire

Rebecca Rachel George[1], Radhika Arjun Kumar[2], Ravindra Kumar Jain[3]
Graduate student[1,] Reader[2], Department of Periodontics, Reader[3], Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: Gingival recession, either localized or generalized, may be associated with one or more surfaces, resulting in attachment loss and root exposure, which can in turn result to clinical issues such as root surface hypersensitivity, root caries, cervical root abrasions, difficult plaque control, reduced cosmetic appeal and aesthetic concern. The aim of this study is to assess knowledge of various root coverage techniques and their implementation among periodontists in Chennai.

Materials and Methods: We conducted an online survey amongst Periodontists working in Chennai, Tamil Nadu to which 65 Periodontists responded. 29 were male participants and 36 were female. The questionnaire consisted of 12 questions, 11 of them giving the possibility of multiple choices of answers and one question being open ended for the participant to fill in an appropriate response. The demographic profile, interests, frequency of performing and satisfaction in root coverage techniques were assessed.

Results: Periodontists well versed in literature and advanced root coverage techniques were found to perform root coverage procedures more frequently than those who were not.

Conclusion: This study concludes that less experienced Periodontists perform root coverage techniques (both conventional and advanced) more frequently and that most Periodontists agree that connective tissue graft proves more effective amongst other techniques listed.

Keywords: *Recession, root coverage techniques, connective tissue graft*

Introduction

Over the years, a large number of surgical techniques have been introduced to correct labial, gingival recession defects. Aesthetic concerns are the most common reason to perform these procedures. Clinical studies have evaluated many of the techniques.In subjects who exercise oral home care and have regular dental check-ups, gingival recession was the predominant periodontal finding before 40 years [1]. And sites with recession seem to be susceptible for additional apical displacement of the gingival margin [2]. Several procedures have been proposed for the treatment of buccal gingival recessions and three systematic reviews have assessed their efficacy by evaluating some quantitative aspects of treatment outcomes. The first review [3] included the coronally advanced flap, the lateral positioned flap, the free gingival graft, the connective tissue graft, guided tissue regeneration (GTR) with resorbable and non-resorbable membranes. Combining the results of six clinical trials [4-9], a significant advantage of connective tissue grafts over GTR was demonstrated by meta-analysis in terms of reducing recession [3]. Overall it was concluded that all procedures were effective in reducing gingival recessions, with a slight advantage of connective tissue graft over- GTR. The second review [10], published after the completion of the present survey, compared connective tissue graft with GTR and came to the same conclusions. An additional review [11]also evaluated data from currently available studies on root coverage procedures to repair gingival recession. GTR-based root coverage was found to successfully repair gingival recession defects, but conventional mucogingival surgery resulted in statistically better root coverage and width of keratinized gingival.

Patient’s desire to improve esthetics is often mentioned as a major motive for intervention. Yet, in reviewing the pertinent literature, it can be noted that esthetic aspects have not been analyzed in comparative studies and could therefore not be taken into consideration for the above-mentioned conclusions. Other justifications include dental hypersensitivity, problems in controlling plaque formation due to an unfavorable contour of the gingival margin, and the expectation that this will prevent further progression. While studies have documented favorable clinical results and their relative stability over time, treatments have not been compared to no therapy or a sham intervention, and thus the evidence for an advantage of surgical interventions in controlling these factors are limited [12].

Little is known about the knowledge transfer from research to clinical practice. It is essential to learn about possible gaps between research and practice in order to adapt to continuing education and to ensure that researchers include questions that are relevant to practicing dentists. The purpose of this study was therefore to evaluate the views, knowledge and preferences of a large sample of practicing dentists in Periodontology, focusing specifically on the treatment of gingival recessions, and to compare the findings with the current evidence available in the dental literature.

Materials and Methodology

We conducted an online survey amongst 80 Periodontists [13,14] working in educational institutions as well as in private clinics in Chennai, Tamil Nadu to which 65 Periodontists responded. 29 were male participants and 36 were female. The questionnaire consisted of 12 questions, 11 of them giving the possibility of multiple choices of answers and one question being open ended for the participant to fill in an appropriate response. The demographic profile, interests, frequency of performing and satisfaction in root coverage techniques were assessed.

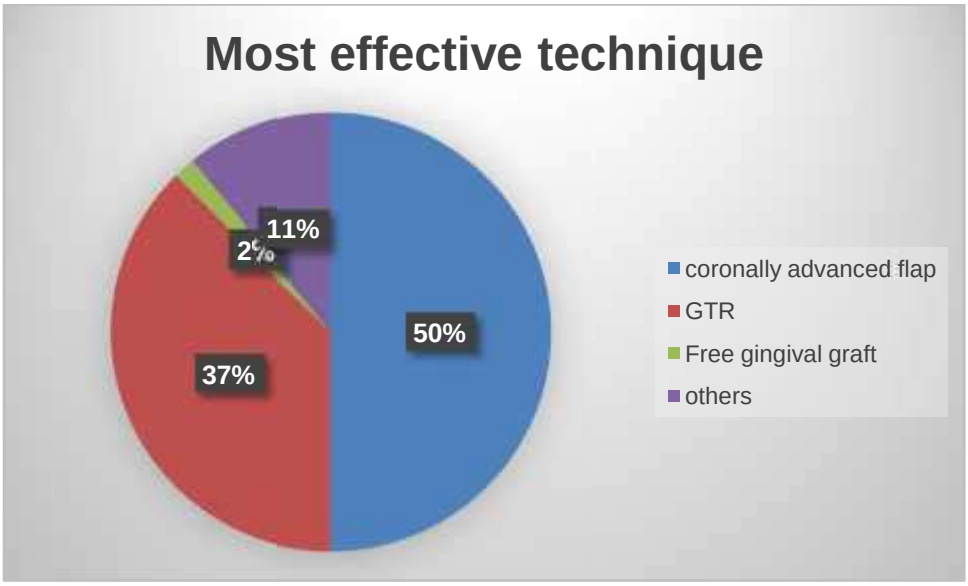
Results

Most of the participants predominantly belonged to the age group of 30-50 years (34 in no.). In terms of years of practice, newer private practitioners with 0-5 years of experience made up 52% (34 in no.) of the sample population of periodontists and 35% (23 in no.) with 6-10 years of experience.

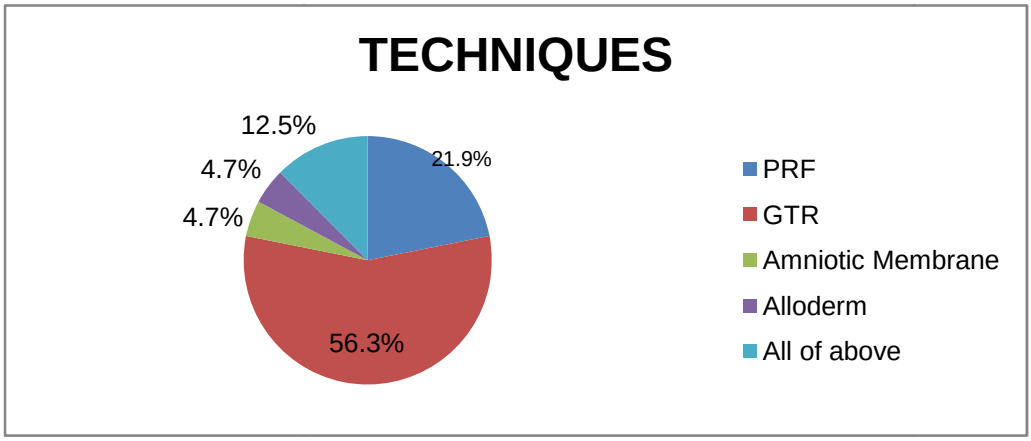
When asked about the clinical situations where they would perform root coverage techniques, dentinal hypersensitivity (33%) (21 in no.) and aesthetics (33%) (21 in no.) were the most common responses.

Newer root coverage techniques such Vestibular Incision Subperiosteal Tunnel Access (VISTA) and root coverage using autologous fibroblasts cell culture were some of the responses to the other advanced techniques the participants have performed so far (Graph2).

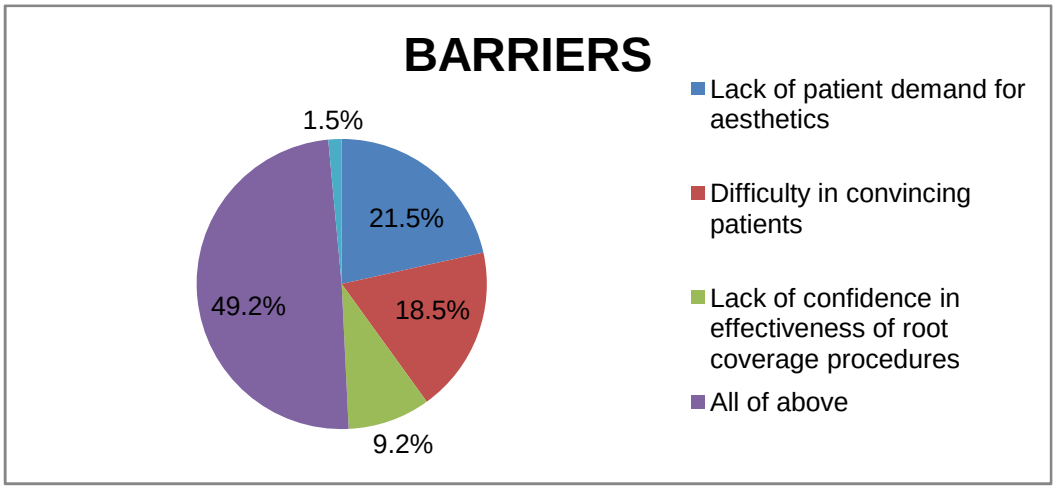
Graph1: Most effective root coverage procedure



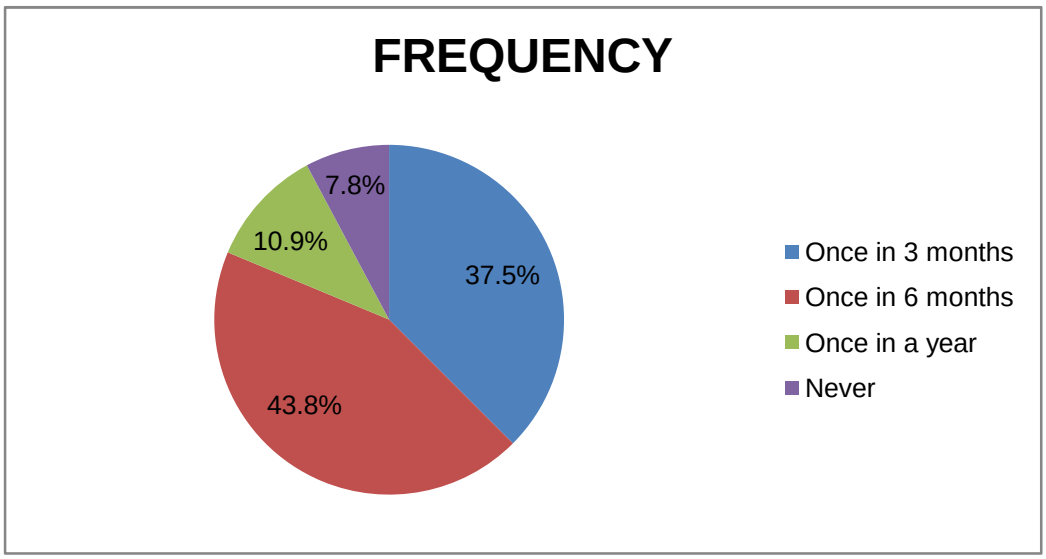
Graph 2: Newer advanced root coverage technique implemented in private practice.



Graph 3: Barriers faced in performing root coverage techniques



Graph 4: Frequency of performing root coverage techniques



Discussion

In our survey Periodontists of the age group 30-40 years participated predominantly and they comprised of44percentileof the total while periodontists of the age group 50 years and above were only 3 percentile of the 65 participants.

From the results of the survey, we can infer that periodontists who are 0-5 years into clinical practice perform root coverage procedures more frequently and are well aware of the advanced procedures for recession. This may be attributed to their recent completion of post graduation and performing root coverage techniques in their post graduate set up. The survey showed majority of the periodontists opted for connective tissue graft for root coverage over other mentioned techniques [Graph 1]. This is in concordance with the studies by Jepson and Rosetti.E who concluded that connective tissue graft was a more effective method of root coverage techniques. [4, 8]

Among the barriers faced in performing root coverage procedures like non conscious about esthetics, inability in convincing patients, lack of confidence in effectiveness of root coverage procedures most of the periodontists opted for the all of the above option which suggests that the correlation between frequency of performing root coverage techniques and barriers faced is inversely proportional [graph 3]. About 43.8% participants did root coverage procedure once in 6 months, and 37.5% did once in 3 months [graph 4].

In a study by Zaher C A [12] aesthetic concerns were the predominant indications for root coverage procedures. They suggested further research should therefore include aesthetic aspects as primary clinical outcome variables for root coverage procedures [15]. One of the most common esthetic concerns associated with the periodontal tissue is gingival recession [16]. Esthetics was suggested as a major indication of root coverage procedures by general dentists, but hypersensitivity was the major concern for periodontists for performing root coverage procedures. Patients have, recently, become more conscious of dental esthetics and accordingly have been demanding precision treatment of their exposed root surfaces [17].

The major limitations of this study include the smaller sample size, and since this is a questionnaire study it is highly subjective and depends on Individual responses.

Conclusion

In this questionnaire study we conclude thatConnective tissue graft along with GTR technique was the most commonly preferred root coverage technique [18]. Approximately half of the participants performed root coverage procedures once in 6 months and followed by many of them who performed at least once in 3 months. Esthetic unawareness, difficulty in convincing patients and Lack of confidence were the proposed reasons for not performing root coverage techniques by the participants.

Corresponding Author
Ravindra Kumar Jain
Reader,Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India
Telephone Number: +91 9884729660
E-mail: orthoravi2@gmail.com

References

1. Heitz-Mayfield, L. J., Schatzle, M., Loe, H., Burgin, W., Anerud, A., Boysen, H. & Lang, N. P. (2003) Clinical course of chronic periodontitis. II. Incidence, characteristics and time of occurrence of the initial periodontal lesion. J ClinPeriodontol. 2003 Oct; 30(10):902-8.

2. Serino, G., Wennstrom, J. L., Lindhe, J. &Eneroth, L. (1994) the prevalence and distribution of gingival recession in subjects with a high standard of oral hygiene. J ClinPeriodontol. 1994 Jan; 21(1):57-63.

3. Roccuzzo, M., Bunino, M., Needleman, I. &Sanz, M. (2002) periodontal plastic surgery for treatment of localized gingival recessions: a systematic review. J ClinPeriodontol. 2002; 29Suppl 3:178-94; discussion 195-6.

4. Jepsen, K., Heinz, B., Halben, J. H. & Jepsen, S. (1998) Treatment of gingival recession with titanium reinforced barrier membranes versus connective tissue grafts. J Periodontol. 1998 Feb; 69, 383–391.

5. Trombelli, L., Scabbia, A., Tatakis, D. N. &Calura, G. (1998) Subpedicle connective tissue graft versus guided tissue regeneration with bioabsorbable membrane in the treatment of human gingival recession defects. J Periodontol. 1998 Feb; 69, 1271–1277.

6. Zucchelli, G., Clauser, C., De Sanctis, M. &Calandriello, M. (1998) Mucogingival versus guided tissue regeneration procedures in the treatment of deep recession type defects. J Periodontol. 1998 Feb; 69(2):138-45.

7. Borghetti, A., Glise, J. M., Monnet-Corti, V. &Dejou, J. (1999) Comparative clinical study of a bioabsorbable membrane and subepithelial connective tissue graft in the treatment of human gingival recession. J Periodontol. 1999 Feb; 70(2):123-30.

8. Rosetti, E. P., Marcantonio, R. A., Rossa, C., Jr., Chaves, E. S., Goissis, G. &Marcantonio, E., Jr. (2000) Treatment of gingival recession: comparative study between subepithelial connective tissue graft and guided tissue regeneration. J Periodontol. 2000 Sep; 71(9):1441-7.

9. Tatakis, D. N. &Trombelli, L. (2000) gingival recession treatment: guided tissue regeneration with bioabsorbable membrane versus connective tissue graft. J Periodontol. 2000 Feb; 71(2):299-307.

10. Oates, T. W., Robinson, M. &Gunsolley, J. C. (2003) surgical therapies for the treatment of gingival recession. A systematic review. Annals of Periodontology 8, 303–320.

11. Al-Hamdan, K., Eber, R., Sarment, D., Kowalski, C. & Wang, H. L. (2003) Guided tissue regeneration-based root coverage: meta-analysis. J Periodontol. 2003 Oct; 74(10):1520-33.

12. Zaher C-A, Hachem J, Puhan MA, Mombelli A. Interest in Periodontology and preferences for treatment of localized gingival recessions. A survey among Swiss dentists. J ClinPeridontol 2005; 32: 375–382.

13. Harris RJ. Clinical evaluation of 3 techniques to augment keratinized tissue without root coverage. Journal of periodontology. 2001 Jul 1;72(7):932-8.

14. Langer B, Langer L. Subepithelial connective tissue graft technique for root coverage. Journal of periodontology. 1985 Dec;56(12):715-20.

15. Grover V, Kapoor A, Malhotra R, Sachdeva S. Interest and satisfaction of dentists in practicing periodontics: A survey based on treatment of gingival recession. Dental Research Journal. 2012; 9(4):404-413.

16. Thamaraiselvan M, Elavarasu S, Thangakumaran S, Gadagi JS, Arthie T. Comparative clinical evaluation of coronally advanced flap with or without platelet rich fibrin membrane in treatment of isolated gingival recession. J Indian SocPeriodontol 2015; 19:66-71.

17. Parthasarathy GB, Sankari M, Jayakumar ND, VargheseSheeja S. Connective Tissue Graft with Platelet Rich Fibrin – A Novel Root Coverage Approach. J Clin Dent Sci 2017; 2(1):30- 37.

18. Harris RJ. A comparison of 2 root coverage techniques: Guided tissue regeneration with a bioabsorbable matrix style membrane versus a connective tissue graft combined with a coronally positioned pedicle graft without vertical incisions. Results of a series of consecutive cases. Journal of periodontology. 1998 Dec;69(12):1426-34.

Awareness of Varicose Veins among Dental Students – Pilot Study

Nathasha A/P Sivakumar[1], Brundha.M.P[2], Dhanalakshmi Ravikumar[3]

Graduate Student[1], Senior Lecturer[2], Department of General Pathology,

Senior Lecturer[3], Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: In the modern living and working conditions, dentists are working in an environment with more effort and holding of fixed positions for longer duration of time. So the present study was undertaken to evaluate their knowledge about varicose veins to give knowledge to undergraduate dental students.

Materials and Methods: 50 dental students were selected on this study. The questionnaires were distributed among the dental students aging 20-26 years old. Informations were collected via a structured proforma, which includes demographic variables. The students were asked to choose only one answer for each question whuch they think would be the most appropriate answer. All data collected was compiled to do statistival analysis.

Results: This survey shows that 70% of the students said they were aware of varicose veins from other sources while 16% of them were alerted by their friends and 8% of them by work colleagues.

Conclusion: With the light of available evidence this study concludes that there is a need to create awareness among undergraduate sental students regarding the complication of varicose veins. This may help the clinician to avoid complications in their general practice.

Key Words: *Varicose veins, Non return valves, deoxygenated blood, pigmentation, lower limb veins.*

Introduction

Chronic venous disease is a common condition presented to the general physicians worldwide. Varicose veins , is a commonly reported venous disease. Varicose veins are tortuous, widened veins in the subcutaneous tissues of the legs and are often easily visible. Their valves are usually incompetent so they might have reflux of blood occurs, and this resulting venous hypertension can cause symptoms[1]. Varicose veins are widely seen as medically unimportant and deserving low priority for treatment. They are common, affecting nearly a third of adults in Western societies, and few people with varicose veins are ever harmed by them. However, this can cause concern and distress on a large scale, most of which can be dealt with by good explanation and reassurance, or by a variety of treatments which are evolving rapidly at present time. Patients can now be referred for more precise assessment and a greater range of therapeutic options than ever before [2].

Varicose Veins is common disease which affects one third of the population of which prevalence is observed in the Western Europe and the United States. A study revealed that, from the affected population, there is around 1-73% of females (especially during pregnancy) exposed to this disease and on an average 2-56% of males. Thus, we may conclude that women are more likely to be affected than men[3]. Varicose Veins or Venous Insufficiency is a disease which involves enlargement and gnarling of the Veins usually of legs. In this disorder, there is reflux flow of blood through the valves of legs, hence instability in circulation of blood.

The risk factors of includes age, hereditary, pregnancy, obesity, occupation which involves prolonged hours of standing, Diet, Type of physical activity, Excess use of hormones, etc[4]. These factors are not clearly known yet. Symptoms related to Varicose veins may not be observed in case of some affected population. If seen the symptoms of this disease at initial stages include severe pain, swelling, itching, heavy legs, and lipodermatosclerosis (skin thickening). If left untreated, the further complications lead to bleeding veins, eczema, skin pigmentation or discoloration. Later it can cause venous ulcers, and hence complete vein incompetence. Diagnosis of the varicose veins disease is done using the duplex scan method of investigation

Varicose vein is a dilated, elongated, tortuous and it is often palpable superficial venous system of the body and especially in the lower extremities. Other commoner areas are the umbilicus (Capus medusa), scrotum/ vulva (varicocele) and rectum (haemorrhoids) [5]. They can be found in the upper extremities along the distributions of the cephalic and basilica veins, though rarely.

Visible varicose veins in the lower extremities are estimated to affect at least a third of the population and 28.6% of people with varicose veins progressed to develop serious venous diseases. Also 3-6% of people with varicose veins in their life time progress to develop venous ulcers [6].

In the lower extremities, there are two main superficial veins, namely the great saphenous vein (GSV) and the small saphenous vein (SSV).The proportion of limbs whose GSV and SSV affected with varicosity ranges from 25-35%. Isolated SSV varicosity occurs in about 14% of limbs [7].

The incidence of chronic venous insufficiency and related varicose veins is variable and fluctuates according to factors such as age, sex, and geographical locations. Extrinsic environmental factors and intrinsic pathological conditions contribute to the disease, including family history, obesity, older age, pregnancy and female gender [8]. The clinical features and Doppler ultrasonography are currently used in making preoperative diagnosis Current treatment procedures are divided into conservative and surgical. The conservative methods are leg elevation, exercise, bandaging and massage, generally referred to as Bisgaard regime. The surgical methods are high ligation, multiple ligation and stripping, endovenous laser ablation (EVA), sclerotherapy and ambulatory phlebectomy [9]. In Nigeria, a developing country, the newer techniques are currently not available. Assessing the outcome of treatment requires well defined measures and this is particularly difficult in varicose vein surgery

Varicose veins do occur when there are incompetent perforators, incompetent valves of the superficial veins, deep vein thrombosis, atriovenous fistula (AVF) and pelvic mass(es) like pregnancy, tumours obstructing blood flow towards the heart. Other causes include family history and idiopathic[10].

A study on large population of UK has shown age adjusted prevalences of 40% in men and 32% in women, although women more often present for treatment. The age of onset varies; some people develop varicose veins in their teenage years, but prevalence rises with age. Varicose veins often appear in first pregnancy, and further pregnancies can make them worse. A family history is common but people should be reassured that having close relatives with severe symptoms from varicose veins does not confer any great likelihood that they will have similar problems[12].

Varicose veins can cause a variety of symptoms of discomfort in the legs, but it is important to try to differentiate these from the many other reasons for leg pains. The Edinburgh vein study

found that the symptoms significantly associated with varicose vein were itching, heaviness, and aching, but the relation of these with varicose veins was inconsistent, particularly in men [13]. Traditional pointers to symptoms being caused by varicose veins include worsening of symptoms after prolonged standing or walking and towards the end of the day, relieving symptoms by elevating the legs or wearing support hosiery, and tenderness over the veins [14].

There are several factors which can be related to the development of varicose veins. That includes, age, gender, family history, obesity and most importantly the standing occupation[15].The reverse flow of blood in veins is prevented by one-way valves. The veins starts leaking, leading to the stagnation of th deoxygenated blood in the leg veins [16].

Extensive evaluation of the venous disease and the ligation of the site deep to the superficial reflux are one of the way of preventing the recurrence. On recurrence, Doppler sound is accurate in deep venous assessment and in demonstrating the leakage of the veins [17]. Few studies have revealed that, up to 15% of men and 25% of women have visible varicose veins. One should know early symptoms of this disease so as to avoid complications which can be dangerous as visible dilated veins over the thighs and legs [18].

It is also a very common disorder usually neglected by patients due to lack of pain in the initial stages of the disease. Varicose veins said to be responsible for a variety of symptoms such as swelling, aching, cramps and tingles [19].

The early recurrence is found in a few cases due to incorrect diagnosis. Late recurrence, in the majority of cases, is due to incorrect surgery or the overlooking of gastrocnemius vein incompetence. In some cases deep reflux, soleal arch compression or left iliac vein compression was found to be the possible causes of recurrence [20].

The condition of varicose veins is significantly common in the population of Tamil Nadu, especially in older individuals and women. Individuals whose occupations involve standing for long hours are prone to varicose veins. A large portion of affected population failed to undergo treatment for varicose veins. The awareness about the treatments available for varicose veins may encourage the affected population to undergo the required treatment [21].

In the modern living and working conditions, dentists are working in an environment with more effort and holding of fixed positions for longer duration of time. So the present study was undertaken to evaluate their knowledge about varicose veins to give knowledge to undergraduate dental students.

Material and Methods

- **Ethical Approval**

The present cross-sectional study was carried out after obtaining Ethical approval from the Institutional review board of Saveetha Dental College.

- **Study Design**

The survey was conducted in a single dental school in Chennai. This was one of the first study to assess the knowledge of undergraduate about varicose veins. Since it was easy to recruit the study population from a single dental school, purposive sampling technique was chosen. A sample of 50 undergraduate students in a Saveetha Dental School, Chennai, India was included in the study.

The study was undertaken in two stages stage 1 and Stage 2. Stage 1 comprised formulating, designing, and validating the questionnaire, whereas Stage 2 tested the validated questionnaire among 50 undergraduate dental students.

- **Stage 1 (Designing and Validation of Questionnaire)**

A standardized self-constructed questionnaire was formulated by two investigators (NS, DR,). Both the investigators (NS, DR,) independently formulated the questionnaire, and after a consensus, they arrived at a final list of 15 questions. The questionnaire was based on the source of knowledge, Etiology, signs and symptoms and its preventive measures. Initially, content validation of the questionnaire was performed by circulating the questionnaire to 10 qualified General dentists. A panel discussion was conducted among 10 qualified General dentists, and they had rated the questionnaire using content validity ratio [14-16]. There was a good agreement between the investigators, with a rating of >0.7. Finally, the questionnaire was distributed to 10 random General dentists for face validation, and it was evaluated using 5-point Likert scale.

- **Stage 2 (Testing of Validated Questionnaire)**

After the content and face validation, the questionnaire was distributed to 50 undergraduate students to complete the questionnaire. Distribution and collection of the questionnaire were done by one Graduate dentists (NS).

- **Statistical Analysis**

Data were collected and entered into SPSS software version (SPSS Inc., Chicago, IL, USA) 20.0 for percentages.

Results

This survey shows that 70% of the dental students said they were aware of varicose veins from other sources while 16% of them were alerted by their friends. 8% of the dental students were aware of it by work colleagues. (Table 1). Among the 50 students, 50% of them were saying that standing for a long period of time is the most common cause of varicose veins. Meanwhile, 25% of the dental students were told that the disease might occur because of obesity. 12% of them were told that aging might be the most common cause of this disease.(Figure 2). Awareness regarding the signs and symptoms of varicose veins 52% of the dental students were aware that visible bluish-purple cluster in the legs is the most common clinical feature seen in varicose veins. Followed by 16% of them stated that varicose veins might be seen as swelling of ankle and feet. 10% of the dental students were telling that aching and itching in the legs is the signs. (Table 3). In awareness about the preventive measures, out of the 50 students, 56% of the students say that it can prevented by avoiding standing for an extended period of time. 26% of them said that it can be prevented by exercising to improve the strength of the leg, while 14% of them said that losing weight or maintaining a healthy wait could be the best preventive measure.(Table 4). Among the 50 students, 58% of them were not aware of the treatment for varicose veins. While 18% of them were saying it would be Sclerotherapy and 14% says that it can be corrected by surgery.(Figure 5). Table 6 and Figure 7 depicts the various treatment and preventive measures available for varicose veins and their awareness among students.

Table 1: Knowledge about varicose veins among dental students

STATEMENT	YES	NO
NUM OF STUDENTS	48	2
PERCENTAGE (%)	96	4

- Among 50 dental students, 96% of them were aware of varicose veins.

Table 2: Source of knowledge

STATEMENT	NUM OF STUDENTS	PERCENTAGE(%)
Friend	8	16
Family member	1	2
Work collegue	4	8
Patient	2	4
Others	35	70

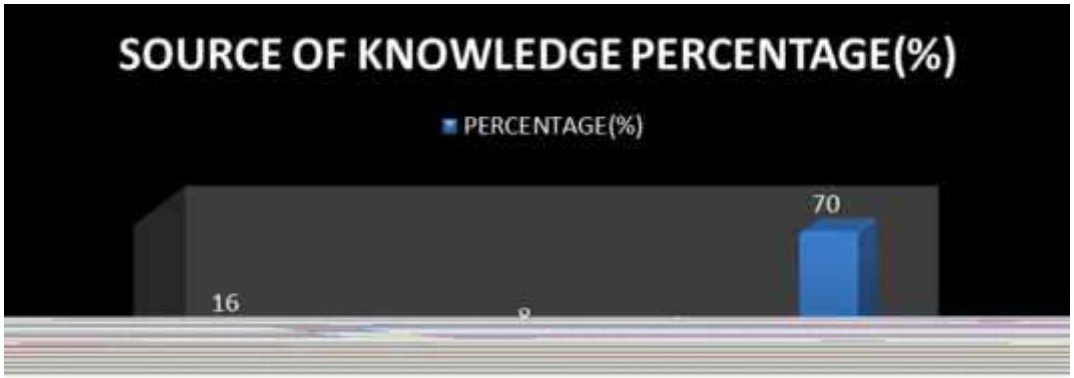


Figure 2: Source of knowledge

- The source of knowledge about varicose veins was 70% from others, according to the dental students

Table 3: Awareness about the causes

STATEMENT	NUM OF STUDENTS	PERCENTAGE (%)
Hereditary	2	4
Aging	6	12
Standing for long period of time	25	50
Obesity	13	25
Leg injury	1	2
Don’t know	3	6

- Among 50 dental students, 50% of them were aware that standing for a long period of time was the common cause for varicose veins

Table 4: Most common signs and symptoms of varicose veins

STATEMEMT	NUM OF STUDENTS	PERCENTAGE (%)
Aching and itching in legs	5	10
Discomfort	3	6
Heaviness	2	4
Swelling of ankle and feet	8	16
Brown skin discoloration in the area around the varicose veins	1	2
Visible bluish-purple clusters in the legs	26	52
Ulcers in the area around the varicose vein problem	3	6
Don't know	2	4

- 52% of the dental students, told that visible bluish cluster in the legs are the most common cause of varicose veins.

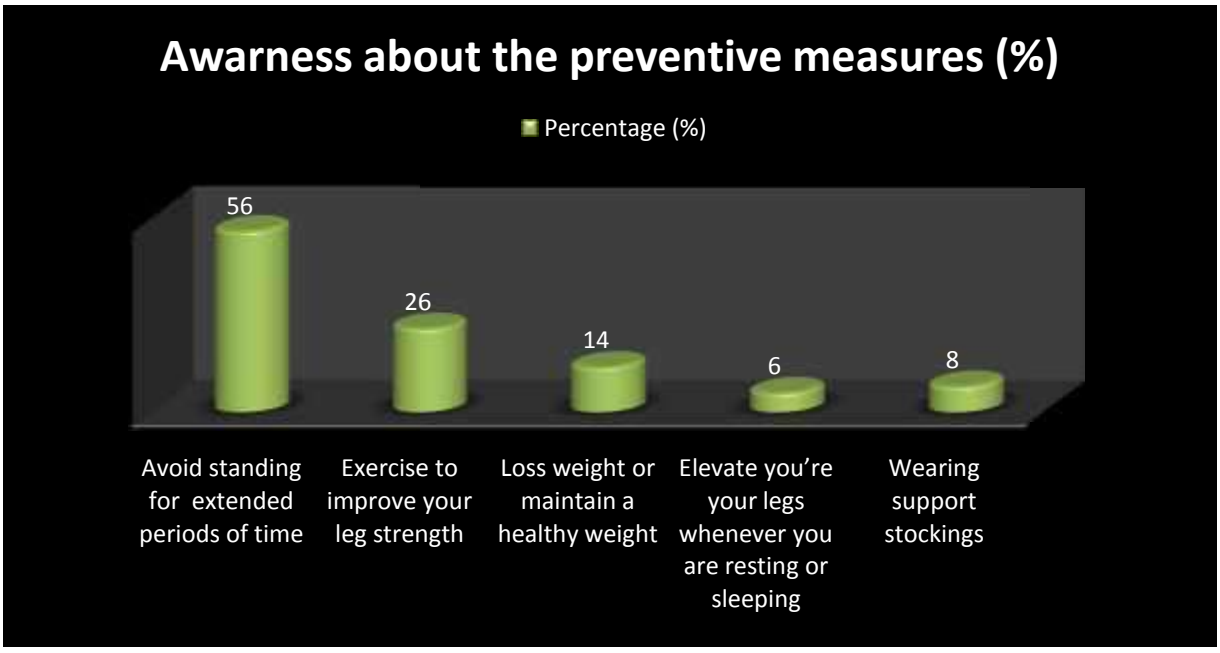


Figure 5: Awareness about the preventive measures

- Among 50 of the dental students, 56% of them are saying that avoid standing for a extended periods of time would be the best preventive measure for varicose veins.

Table 6: Awareness about the Treatment measures

Statement	Num of students	Percentage (%)
Surgery	7	14
Endovenous therapy	5	10
Sclerotherapy	9	18
Don't know	29	58

- 58% out of the 50 dental students don't know about the common treatment of varicose veins

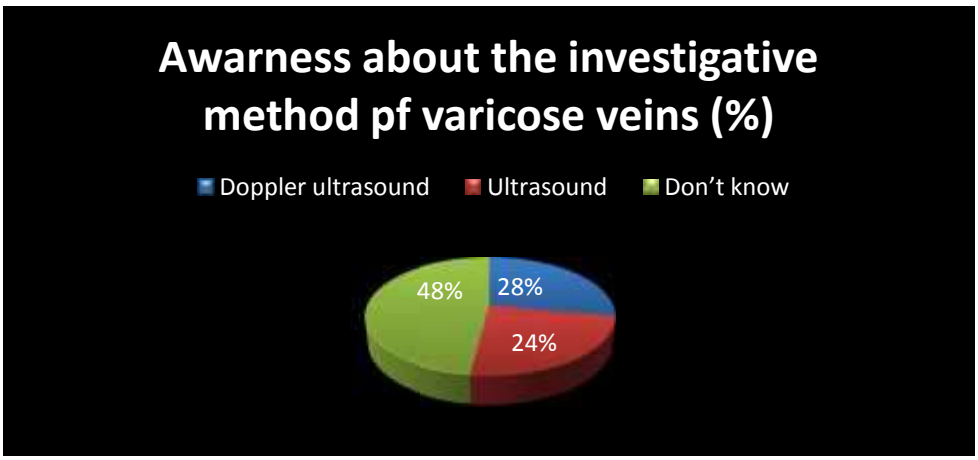


Figure 7: Awareness about the preventive measures

- Among 50 of the dental students, 48% of them were telling that Doppler ultrasound was the common method to identify varicose veins nowadays.

Discussion

This is one of the first study to exclusively evaluate the knowledge on varicose veins in undergraduate dental students. In the modern living and working conditions, dentist"s are working in an environment with more effort and holding of fixed positions for longer duration of time. So the present study was undertaken to evaluate their knowledge about varicose veins to incultate knowledge on them.

The questionnaire used in this research had been validated by content and face validation at the beginning of the study, based on the validation technique explained previously. The aim of this validation process was to gain the experts opinion and to modify the questionnaire in ease of understanding.

Of all the 50 students, 94% of them were aware of the causes of varicose veins, while 6% of them were not. A similar kind of study was done among staff nurse in Mangalore by Venisha et al and the author had concluded 61% of staff nurse had adequate knowledge about causes of varicose veins. A study done by A.W Willie et al has proved that the dentist are more likely to develop neurocirculated diseases such as varicose veins and this is considered as one of the occupational hazards of dentistry.

Among 50 students, 48 students were aware of the signs and symptoms of varicose veins while 2 students were ignorant about the signs and symptoms. Questions regarding the preventive measures 56% of students stated that avoiding of standing for a long period of time can be the best preventive measure. Questions regarding the treatment options, most of the undergraduate (58%) were not aware of the common treatment modality for varicose veins. Finally questions regarding the investigative methods for varicose veins 48%of undergraduates were aware about the investigative measures such as Doppler ultrasound.

Based on the results of the present study. It is clearly evident, that, knowledge and the information regarding the etiology, preventive measures, investigations, and treatment options of varicose veins are not sufficient among undergraduate dental students

Conclusion

With the light of available evidence this study concludes that there is a need to create awareness among undergraduate dental students regarding the complication of varicose veins. This may help the clinician to avoid complications in their general practise. Because even in this modern era, varicose veins has been the major problem seen in the dentists. So it is beneficial and important for every dental students to have knowledge on varicose veins.

Corresponding Author

Dhanalakshmi Ravikumar,

Senior Lecturer, Department of Pedodontics and Preventive Dentistry,

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences,

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

E-Mail : dhana9677@gmail.com,

Telephone number - 9597011442

Reference

1. Jennifer , The epidermiology of Chronic Venous Insufficiecy and Varicose Veins. J.annepidem. 2004; 15(3):78-382
2. John T hobbs ,Surgery and sclerotherapy in treatment of Varicose veins. Arch Surg. 1974; 109(6):793-796
3. JH Abramsom, The epidermiology of varicose veins. A survey in western Jerusalem. Jech. 1973; 35(3):213-217
4. Arenander E. Hemodynamic effect of varicose veins and results of radicular surgery. Acta ChirScand Suppli 1960:260.26
5. Carter Jf. Comparision of the stripping operation with other methods of treatment . Lanset 1954; 1:743-5
6. AW.Willie. How to avoid the occupational hazards of dentistry. Australian Dental Journal :1967; 8(8):26-29
7. Venisha, A Descreptive study to assess the knowledge regarding risk factorsand preventive measures of varicose veins among staff nurses of selected hospital at Mangaluru with a view develop an information booklet. Recent scientific research2001; 6(10):245-7
8. Assess the prevalence of varicose veins among home weavers, Sharanya, IJPBS,2017(4) SP04; 116-120
9. Campbell WB, Niblett PG, Peters AS, MacIntyre JB, Sherriff S, Palfreyman S. The clinical effectiveness of hand held Doppler examination for diagnosis of reflux in patients with varicose veins. Eur J Vasc Endovasc Surg 2005; 30: 664-9.
10. Goodwin H. Litigation and surgical practice in the UK. Br J Surg 2000; 87: 977
11. Michaels JA, Brazier JE, Campbell WB, MacIntyre JB, Palfreyman SJ, Ratcliffe J. Randomised controlled trial comparing surgery with conservative treatment for uncomplicated varicose veins. Br J Surg2006; 93: 175-81

12. Ratcliffe J, Brazier JE, Campbell WB, Palfreyman S, MacIntyre JB, Michaels JA. Cost effectiveness analysis of surgery versus conservative treatment for uncomplicated varicose veins in a randomised controlled trial. Br J Surg 2006; 93:182-6.

13. Assess the knowledge on varicose veins among police constables at Arakonam, IJPBS2017(4)SP04; 105-109

14. Mundy L, Merlin TL, Fitridge RA, Hillier JE. Systematic review of endovenous laser treatment for varicose veins. Br J Surg 2005; 92:1189-94.

15. Hobbs JT. Surgery and sclerotherapy in the treatment of varicose veins. Arch Surg 1974; 109:793-6

16. Bountouroglou DG, Azzam M, Kakkos SK, Pathmaraj M, Young P, Geroulakos G. Ultrasound-guided foam sclerotherapy combined with sapheno-femoral ligation compared to surgical treatment of varicose veins: early results of a randomised controlled clinical trial. Eur J Vasc Endovasc Surg 2006; 31:93-100

17. Cabrera J, Cabrera J Jr, Garcia-Olmedo MA. Treatment of varicose long saphenous veins with sclerosant in microfoam form: long-term outcomes. Phlebology 2000; 15:19-23

18. Shrija, Study to assess knowledge regarding varicose veins among nursing students of Saveetha Medical College, IJPBS2017(4) SP04; 96-100

19. Campbell WB, Kumar AV, Collin TW, Allington KL, Michaels JA. The outcome of varicose vein surgery at 10 years: clinical findings, symptoms and patient satisfaction. Ann R Coll Surg Engl 2003; 85:52-7

20. Swathy. S, Thenmozhi, A study on prevalence of varicose veins in 30-80 years old individuals, RJPT. 2015; 8(8):14-17.

Awareness on Contraception and STDs among General Population

Prema Sivakumar[1], M.P Brundha[2], Subash Sharma[3]
Graduate student[1],Head[2], Department of General Pathology,Reader[3],Department of Conservative Dentistry and Endodontics.
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: In the present day world, awareness and practice on contraception is very important. In a developing country like ours, it's of paramount importance due to the exponential population increase. In the Indian society, even amongst the educated population, contraception and sexually transmitted diseases are never discussed openly and are considered taboo. Birth control, also known as contraception and fertility control, are methods or devices used to prevent pregnancy. Planning, provision and use of birth control is called family planning. Birth control methods have been used since ancient times, but effective and safe methods only became available in the 20th century. Apart from taboos, people also believe in various traditional practices which may not be effective at all. The aim of this study is to assess the awareness and knowledge of contraceptives and sexually transmitted diseases amongst the general population.

Materials and Method: The sample size of the study is 55, with a random mix of males and females (30-M, 25- F) in a varied range of ages (25-65)

Conclusion: From the study it can be concluded that the overall awareness about contraception and STDs is 57%.

Result: The overall percentage of people who believe in contraception was 78% and the percentage of overall correct answers, which is an insight into the awareness level, was 57%. The awareness levels of males (57%) and females (58%) were almost the same.

Key Words*: Contraception, Awareness, Sexually transmitted disease, IUDs, oral contraception*

Introduction

With the second most populous country in the world, contraceptive knowledge, practice and proper implementation are essential for the improvement of health of existing population and a balance of the available resources and ultimately population stabilization. Family planning is estimated to have brought down maternal deaths by 25% and child deaths by 20%. [1] In this modern age, though people may possess half-baked knowledge about contraceptives, proper implementation and awareness is lacking. The choice of contraceptives has increased manifold over years, with the methods varying in their degree of effectiveness or convenience for the user. Many women have not yet identified the type of contraception that's most effective for their lifestyle and meets their personal requirements. [2] Studies indicate that the total fertility rate of a nation is inversely related to the prevalence rate of contraceptive use. [3]So this study was aimed at gauging the awareness of the general public and also kindling in them the need to be aware of the various methods.

Materials and Method

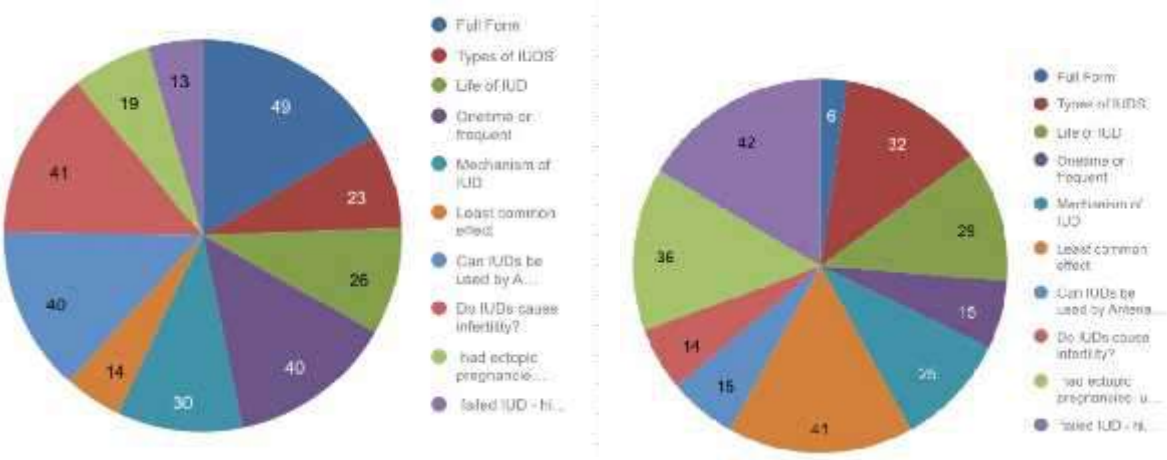
This study was a questionnaire based study conducted among 55 individuals. It had a group of basic questions (Age, sex, education) , and groups of questions each about the various types of contraception (IUDs, hormonal, barrier, emergency contraception, permanent contraception) followed by a final set of questions about sexually transmitted diseases. While designing the questionnaire, some very common myths and beliefs related to contraception were included. The questions were mostly of a multiple choice type (with some questions having a possibility of more than one right option) and a few open ended questions where the person would fill in based on whatever they're aware about from their pre-existing knowledge. The questions were intentionally made so as to test only the awareness of the methods and STDs and did not delve into any personal details, due to the high cloud of stigma surrounding this topic. Usually surveys like these are circulated only amongst women, but in this study the questionnaire was circulated online via email, to both males and females of varying ages (25-65) because contraception isn't for females alone, and awareness of the same is a pre-requisite for a responsible society. Also, the questionnaire was circulated only amongst educated people since the questions were solely awareness based. After collection of the data, it was tabulated and analyzed statistically.

Results

The questionnaire was split into different sections and analyzed, with graphs made wherever deemed necessary. The overall percentage of people who believe in contraception was 78% and the percentage of overall correct answers, which is an insight into the awareness level, was 57%. The awareness levels of males (57%) and females (58%) were almost the same.

A. IUDs

The average awareness percentage was found to be 54 %(Refer Fig. 1)



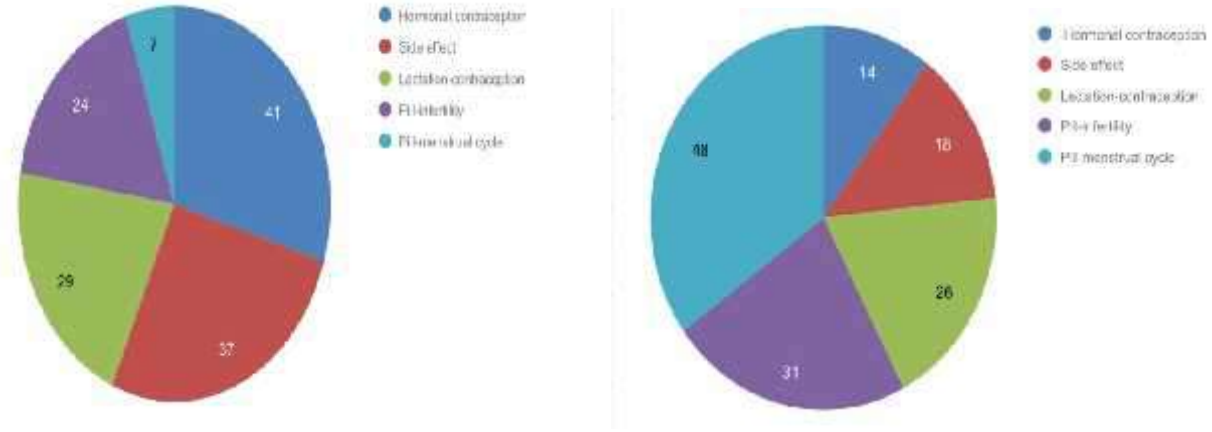
Aware Unaware

Figure 1: Awareness on IUDs:

1. Full form 2.types of IUDs with options 3. Length of using an IUD 4. How often it has to be changed 5. Mechanism of working of IUDs are all except (with option) 6. Least common side effect of IUDs (with options) 7. Can IUDs be used by ante-natal women 8. Do they cause infertility 9. Can it be used by those who have had ectopic pregnancies 10. Does a failed IUD increase the risk of ectopic pregnancies.

B. Oral Contraception

The Average awareness percentage was found to be 50.2% (Refer fig 2)



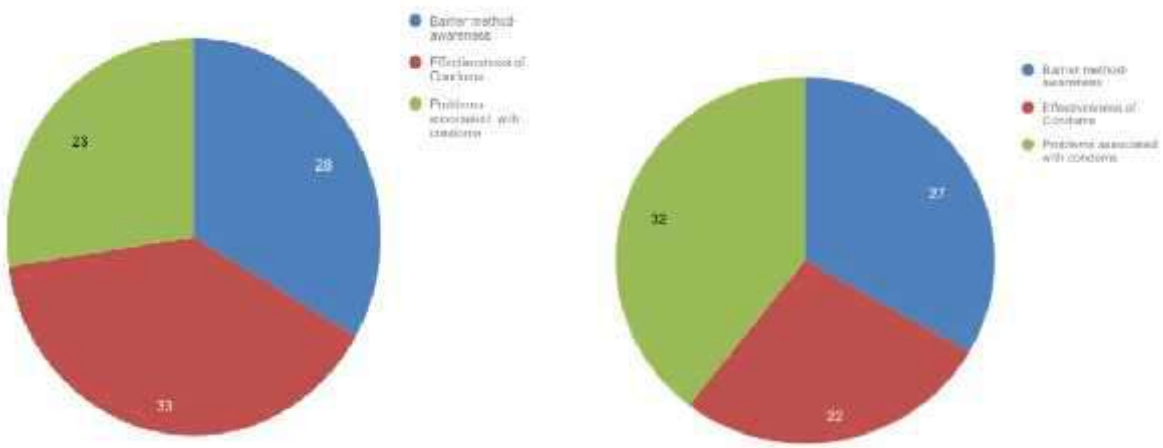
Aware Unaware

Figure 2: Awareness on methods of Oral contraception:

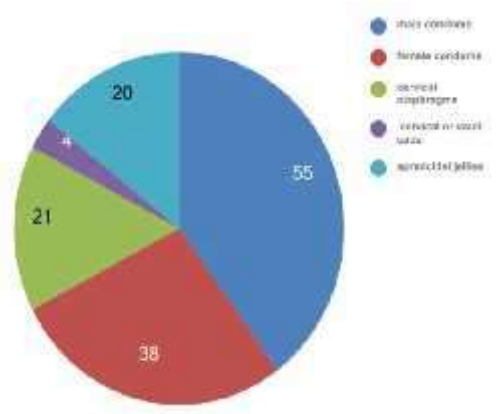
1. Type of contraception (hormonal) 2. Not a commonsideeffect- cancer 3. Necessity of contraceptives during lactation 4.Does Use of OCPs for a long time causesinfertility.

C. Barrier Contraception.

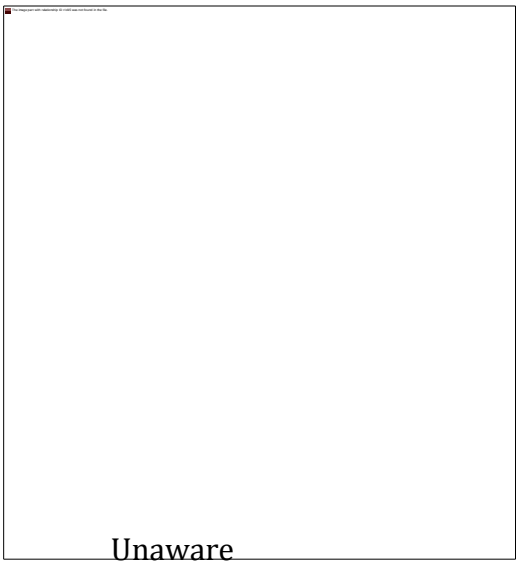
The average awareness percentage for this method was found to be 51 % (Refer Fig. 3).41% of the samples were aware of the fact that the problems associated with failed condoms are pregnancy and STDs due to breakage of condom or leakage through condom which may result from its poor quality. Refer to Fig. 4 for levels of awareness about types of barrier contraception.



Aware Unaware



Aware



Unaware

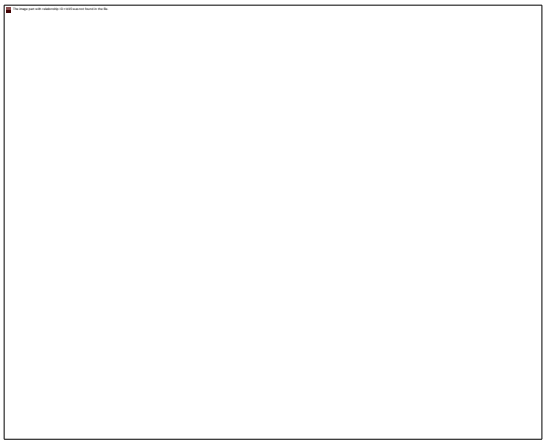
Figure3:1. Awareness on Barrier method of contraception 2. Are condoms 100% effective 3.Problems associated with condoms.

Figure 4:Awareness on types of Barrier contraception:

1. Male condoms 2.female condoms 3.cervical diaphragms 4. Cervical or vault caps, spermicidal jellies

D. Sexually Transmitted Diseases (STDs):

85% of the sample was aware of the full form of STDs, 82% were aware that condoms are the method of contraception that helps in prevention of STDs. Six sexually transmitted diseases were enlisted and the people were asked to tick the diseases they were aware about. (Ref Fig 5)



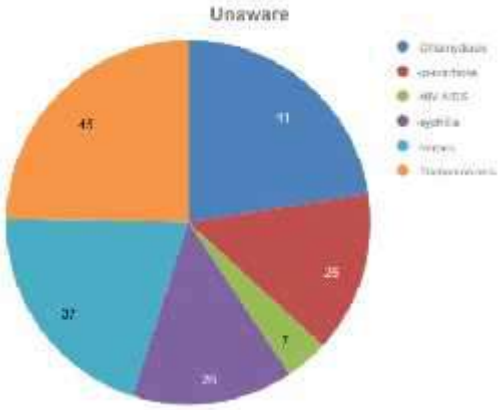


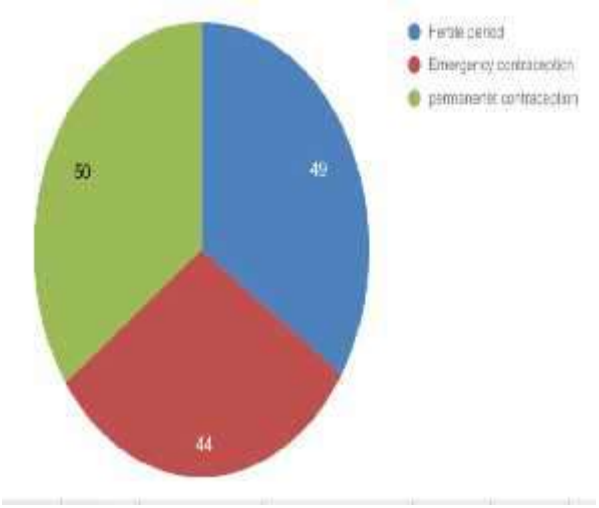
Figure 5: Awareness on STDs:

1. Chlamydiasis 2.gonorrhea, 3.HIV AIDS, 4.Syphillis, 5.Herpes,6. Trichomoniasis

E. Other Miscellaneous Questions (Refer Fig. 6 and 7)

Regarding the methods of emergency contraception, 73% were aware about the progestin only pill, whereas only 13% were aware about the Copper IUDs.

Aware Unaware



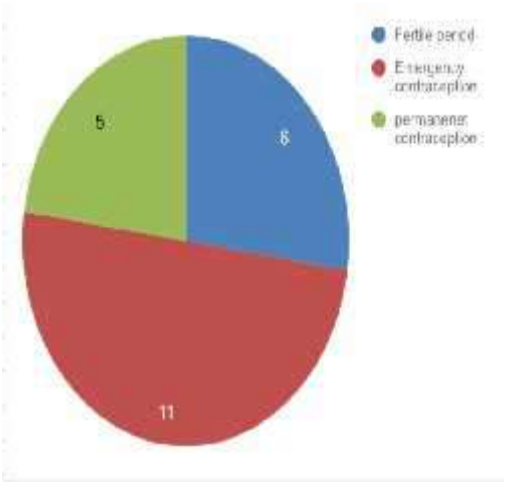


Figure 6: Miscellaneous questions 1.Is it necessary to be aware offertile period

2. Awareness of emergency contraception 3. Awareness of permanent contraception

Aware Unaware

Figure 7: Awareness on permanent contraception:

1. Is it for males/females/both? 2. It is also called – sterilisation 3.For males: Vasectomy 4. For females: Tubectomy.

Based on the overall sample size it was found that the awareness on contraception and STDs was 57%.

The awareness of females on Contraception STDs was 58% and males were 57%.

The questionnaires were sent online to 101 people, but the response was given only by 55 people. 45.5% of the recipients of the questionnaire did not respond, even after continuous follow up.

Discussion

Considering the difficulty of questions put forth to the sample population, the awareness levels were quite good in regard to contraceptives and their use, and sexually transmitted diseases (57%).

Most people did not identify the fact that a failed IUD could potentially cause an ectopic pregnancy. One surprising trend observed among the sample population in oral contraceptives was many people marking it as barrier contraception instead of hormonal contraception. A majority of the study subjects believe that using contraception to alter the menstrual cycle is unhealthy in the long run. (87%)

People (56%) also believe that being on the pills for a long time can cause trouble by completely nullifying the chances at becoming pregnant later.
Male condoms was the method of barrier contraception that everyone was unanimously aware about (100%) and cervical/ vault caps were the least known method of contraception among the study population. (7%)

The spread of sexually transmitted diseases is also a major concern owing to the modern life styles of people. 85% of the population is aware about sexually transmitted diseases, of which a majority (87%) were aware of AIDS followed by syphilis and gonorrhea (53% each).
Only 13% of the entire sample size was aware that copper IUDs are also a type of emergency contraception. People displayed a generally good awareness to emergency (80%) and permanent contraceptive methods (91%).

There is a lot of stigma associated with the discussion of contraceptives, thathave always been kept under wraps and have been a very sensitive topic. Multiple studies have been conducted to check the awareness amongst women alone [4-6]. A lot of difficulty was observed even during the collection of samples. Though particular care had been taken to avoid personal questions, many did not respond. Overall, 45.5% of the people haven't responded to the questionnaires sent across despite frequent follow up.

As mentioned above, a lot of questions were based on popular myths, and it was hardly surprising to see that most people still believe in those myths without even knowing why.
For example, some people even gave some very strange answers for the methods of contraception they were aware of, like pushing a stone into the vagina. Though most of the people believe that permanent contraception is for both males and females, some people still think that it's only for females. Contraception awareness studies must also be conducted amongst males, as there are fewer studies cornering this spectrum. [7-9]

The present study was conducted to assess the awareness and knowledge about various methods of contraception, which would ultimately reduce the rates of abortion. But many prospective trials have to be conducted in order to obtain more extensive data. [10-12].

Hormonal contraception which is highly common among women and not men. Many researches and studies are being carried out to develop allopathic anti-fertility drugs for men.[13,14] It would be valuable if studies were also conducted amongst teenagers , who usually present late for abortion as the awareness levels may be low. [15-17]. Counseling about contraception awareness would also help to a great extent as it will reduce the stigma around the topic and create more aware individuals.[18-20]

This study may not be very accurate because of the limited sample size, and as all the people in the sample were educated.

Conclusion

The overall awareness level, according to the study conducted was 57% with regard to use of contraception and sexually transmitted diseases in general Indian population which was from an urban population, with almost no difference in the levels of awareness of males and females. Though the awareness on contraceptives has definitely increased over the ages, the fact that the people may still not be implementing the various methods effectively remains. Awareness on contraception and about sexually transmitted diseases is of paramount importance for the development of our society and stabilization of the population here in India, more so because of the uncontrolled population inflation and the gross imbalance and discrepancy between the present population levels and the availability of resources, employment opportunities which will ultimately lead to a poor standard of living. These are the factors that will hinder or act as potential roadblocks in the progression of India from a developing to a developed country. Contraception, when viewed from this juncture, is not only a scientific method, but also a moral responsibility of the citizens, for a balance in the population and for better access of the limited resources available in our country.

Corresponding Author

Subash Sharma

Reader,Department of Conservative Dentistry and Endodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Telephone Number- +919884533118
Email- drsubashsharma@gmail.com

References

1. Johnson S, Pion C, Jennings V.Current methods and attitudes of women towards contraception in Europe and America. Journal of Reproductive Health. 2013;10(2):7-14
2. Alaknanda Bhattacharya. Contraceptive awareness-a survey among antenatal women scholars. Scholars Journal of Applied Medical Sciences (SJAMS) 2015; 3(5):1505-1508
3. Mauldin WP, Sinding SW. Review of existing family planning policies a programs: lessons learned. New York population council, 1993; 3(5):49-55
4. Lavonia KS, ParanaNSL. A study on Contraceptive knowledge, attitude and practice among reproductive age group of women in a tertiary institute. International Journal of research and health Sciences, 2014; 2(2):577-580
5. Ramesh BM. A study of social-psychological factors affecting fertility and family planning acceptance. IIPS News1,1987:28(4) :19-25
6. Mustafa R, Afreen U, Hashmi HA. Contraceptive Knowledge, Attitude and Practice among Rural women. Journal of the College of Physician and surgeon,2008;18(9):542-545
7. Gupta S. Weight gain on the combined pill – is it real. Hum Reprod Update. 2000; 6(5): 427-431.
8. Machado RB, de Melo NR, Prota FE, Lopes GP, Megale A. Women's knowledge of health effects of oral contraception in five Brazilian cities. Contraception journal. 2012; 86(6): 698-703.
9. Skouby SO: Contraceptive use and behavior in the 21st century: a comprehensive study across five European countries. Eur J Contracept Reprod Health Care. 2004; 9(3): 57-68.
10. Mosher WD, Jones J. Use of contraception in the United States: 1982–2008. Vital Health Stat. 2010; 23(8): 1-44.
11. Joffe H, Cohen LS, Harlow BL. Impact of oral contraceptive pill use on premenstrual mood: predictors of improvement and deterioration. Am J Obstet Gynecol. 2003; 189(6): 523-530.

12. Kavya R. Studies on the anti-fertility efficacy of Abrime and Embrelin, the compounds of plant origin on mouse testis and uterus. Research Journal of Pharmacology and Technology. 2015; 8(9): 369-375
13. Rapkin AJ, Morgan M, Sogliano C, Biggio G, Concas A. Decreased neuroactive steroids induced by combined oral contraceptive pills are not associated with mood changes. Fertile Steril. 2006; 85(5): 1371-1378.
14. J, Gemzell-Danielsson K, Roumen F, Marintcheva-Petrova M, van Bakel B, Oddens BJ. The CHOICE study: Effect of counselling on the selection of combined hormonal contraceptive methods in 11 countries. Eur J Contracept Reprod Health Care. 2012, 17(1): 65-78.
15. Hoque, M. E. & Ghuman, S. Sexual Behaviour and Knowledge Regarding Sexually Transmitted Infections among Undergraduate Students in Durban, South Africa. Gender and Behaviorjournal. 2011; 9(1):3710–3728.
16. Oddens BJ.Determinants of contraceptive use among women of reproductive age in Great Britain and Germany. II. Psychological factors. J Biosoc Sci. 1997; 29(5):437-70.
17. Pearson VA, Owen MR, Phillips DR, Gray DJ, Marshall MN. Pregnant teenagers' knowledge and use of emergency contraception. BMJ. 1995;310:1644.
18. Haddad LB, Nour NM. Unsafe abortion: Unnecessary maternal mortality. Rev Obstet Gynecol. 2009;2:122–6.
19. Norris Turner A, Ellertson C. How safe is emergency contraception? Drug Saf. 2002;25:695–706
20. Emuveyan EE. Advances in contraception. In: Kwawukume E, Emuveyan EE, editors. Comprehensive Gynaecology in the Tropics. Accra: Graphic Packaging Ltd; 2005; 1:233-235

Awareness on the Correlation between Periodontal Diseases and Cardiovascular Diseases – A Questionnaire Based Study among Cardiologists in Chennai

Maria Kurian[1], Radhika ArjunKumar[2], SreeDevi Dharman[3]

Graduate student[1] ,Reader[2], Department of Periodontics,

Senior Lecturer[3], Department of Oral medicine and Radiology

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: Periodontal disease is common among adults and is a potential source of chronic inflammation. Although a direct causal link between periodontal diseases and vascular diseases has not been shown to date, there are numerous mechanisms through which periodontal infection may affect the onset or progression of atherosclerosis and Coronary heart disease (CHD). For those at risk of developing or those with preexisting cardiovascular disease, critical factors to consider include the presence of other medical conditions, such as diabetes, that may affect the cardiac disease; and also the presence of risk factors that may lead to periodontal diseases. The aim of this study is to assess the awareness among cardiologists on the correlation between periodontal diseases and cardiovascular diseases.

Materials and Method: An online questionnaire based study was conducted to assess the awareness on the correlation between periodontal disease and cardiovascular diseases among cardiologists in Chennai. Data was collected using a structured, self-administered questionnaire which was designed after reviewing recent articles, developments and also similar questionnaires that are based on the objectives of the study.The questionnaire was distributed online to cardiologists in and around Chennai of which 33 had responded. All questionnaires were anonymously collected and the data was kept confidential and not used for anything apart from the study purpose.

Result: The study results reveal that the knowledge and the awareness on the correlation between periodontal disease and cardiovascular diseases among cardiologists in Chennai is good.97% of the doctors were able to state that there is a proven correlation between periodontal

disease and cardiovascular diseases. 94% of them stated correctly that the etiology of periodontal disease is bacterial plaque.

Conclusion: Further seminars and workshops can be conducted in hospitals in order to educate the doctors on the advances on this correlation.Cardiologists may be able to detect pre-cardiac conditions by taking into account the periodontal health as well as prevent the progression from pre-cardiac to cardiac disease.

Keywords: *Atherosclerosis, Cardiologists, Periodontal disease, Conditions*

Introduction

Periodontitis is an inflammatory disease which is of microbial etiology. It causes destruction of the periodontium which consists of the gingival tissues, periodontal ligament fibers and also the surrounding alveolar bone. Due to this destruction, teeth become mobile and fall off. [1] National Health and Nutrition Examination Survey (NHANES III) carried out between 1988 and 1994 stated that 34.5% of dentate U.S. citizens 30 years and above had periodontitis with a relationship seen between the increasing age and periodontal disease [2]. And in developed countries, cardiovascular disease accounts for 50% of deaths [3]. Cardiovascular disease (CVD) is the leading cause of mortality in the United States[4-6].Several diseases such as atherosclerotic CVD,hemorrhagic stroke, congestive heartfailure, hypertension, Rheumatic Heart disease (RHD), and congenital heart defects comes under the wide list of Cardiovascular diseases.[7]Accumulating evidence shows that inflammation played a central role in the pathogenesis of CVD [8,9].Several infectious diseases have been implicated as possibly causing myocardial infarction (MI). A link between oral health and cardiovascular disease has been proposed for more than a century. There is concern about the possible links between periodontal disease (PD) and atherosclerotic vascular disease (ASVD). The 2 disorders share several common risk factors, including cigarette smoking, age, and diabetes mellitus. Patientsareoften presented with claims that Periodontal disease treatment strategies offer ASVD protection.In related research, periodontal disease has also been related to coronary heart disease (CHD). Several observational studies have indicated that periodontitis, a chronic inflammatory periodontal disease that results in the breakdown of bone that surrounds teeth, may be associated with an increased risk for MI.A periodontal disease causes a sort of chronic inflammation [10] which allows the bacteria to enter the bloodstreamby the micro ulceration of the epithelium of the periodontal pocket [11-13]. The contact between germs and macrophages causes a direct release of pro-inflammatorymediators (Interleukin IL-8, Tumor necrotizing factor – alpha TNFα), that facilitates the attraction of neutrophils from vessels (innate immunity). Increased hepatic synthesis of LDL cholesterol is favored by TNFα.It is seen that IL-6 released later helps in the productionof C-reactive protein (CRP) in the liver. This fact accelerates the atherosclerosis plaque progression.[14]C-reactive protein (CRP) is a classic marker for inflammation.[15] Periodontitis isshown to raise inflammatory markers such as CRP in the blood [16-18] which is an acute phase reactant protein considered to be a riskindicator for both periodontitis as well as

cardiovascular disease.[19-22] Hence, if cardiologists are aware of the correlation between the two diseases, early detection and treatment of the periodontal disease can be done thereby, reducing the risk for cardiovascular diseases. This study was conducted to find out the awareness already possessed by the cardiologists and to determine how the awareness could be improved if necessary.

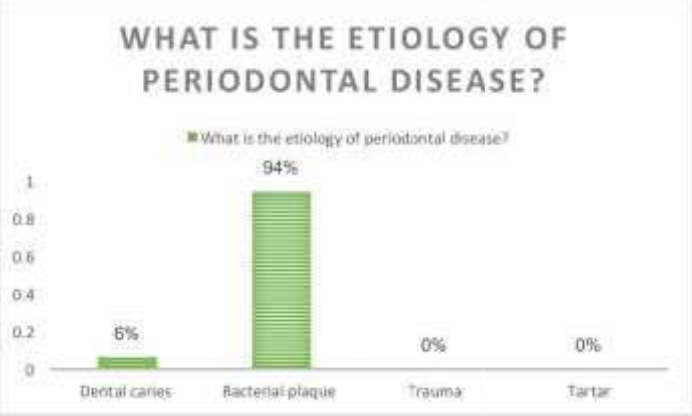
Materials and Methods

An online questionnaire based study was conducted to assess the awareness on the correlation between periodontal disease and cardiovascular diseases among cardiologists in Chennai. Data was collected using a structured, self-administered questionnaire which was designed after reviewing recent articles, developmentsand also similar questionnaires that are based on the objectives of the study. The questionnaire was divided into three parts. The first part included questions based on demographic data, the second part consisted of questions related to their knowledge on the correlation between periodontal disease and cardiovascular diseases and the final part consisted on ways a cardiologist may help to prevent periodontal disease. The questionnaire was distributed online to cardiologists in and around Chennai of which 33 had responded. All questionnaires were anonymously collected and the data was kept confidential and not used for anything apart from the study purpose.

Result

The 33 cardiologists who participated in this survey were between the ages of 31 to 60 years. They had varied years of experience such as between 1 to 30 years of experience in their field. Of the 33 cardiologists, 28 were males and 5 were females. 97% of the doctors were able to state that periodontal disease is a type of bacterial disease and that there is a proven correlation between periodontal disease and cardiovascular diseases. 94% of them stated correctly that the etiology of periodontal disease is bacterial plaque whereas the remaining 6% believed dental caries as the etiology (Fig.1). 79% of the cardiologists believed the inflammation of the gingiva as the initial sign of periodontal disease and 21% suggested it to begingival bleeding (Fig.2). When asked on the management of the periodontal disease, 88% suggested cleaning the teeth,

9% suggested mouthwash and 3% suggested antibiotics (Fig.3). They all agreed that complete management of the disease is required to reduce the risk of cardiovascular diseases. 97% of the cardiologists had received some information on the correlation between periodontal disease and cardiovascular disease from medical college. All of them enquire their patients for a history of periodontal disease (Fig.4). All of them agree that all doctors must educate their patients on the importance of oral health. 91% of them examine their patients for periodontal disease (Fig.5) and all of them refer their patients to a dentist for an oral examination. 88% are able to identify a case of periodontal disease on their own (Fig.6). All of them agree that there is an increase in CRP levels during inflammation and can be used to assess the functioning of the cardiovascular system. 79% of the cardiologists state that during periodontal disease, monocyte infiltration, endothelial damage and gram negative bacteraemiaoccur(Fig.7). All the doctors agree that dental checkups must be conducted for all patients (Fig.8).



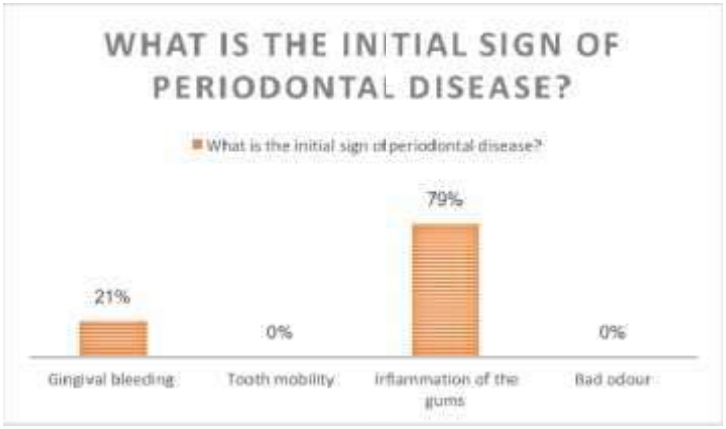


Figure. 1 – The cardiologists response when asked about the etiology of periodontal disease.

Figure.2 – Their response when asked about the first sign of periodontal disease.

Figure.3 – Their response when asked about thes management of periodontal disease.

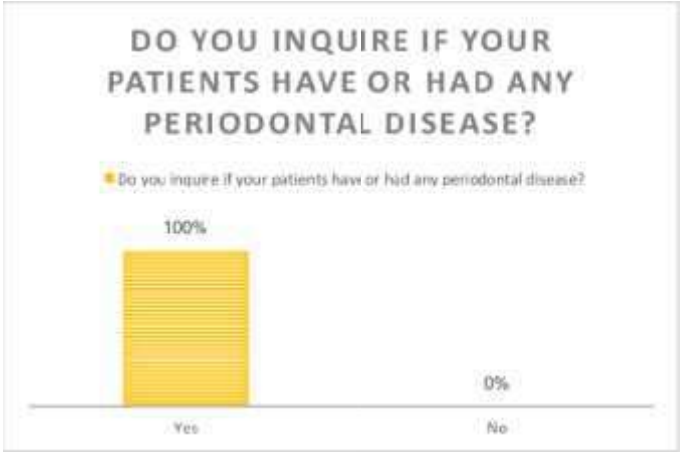


Figure.4 – Their response when asked if they enquire patients about their periodontal health.

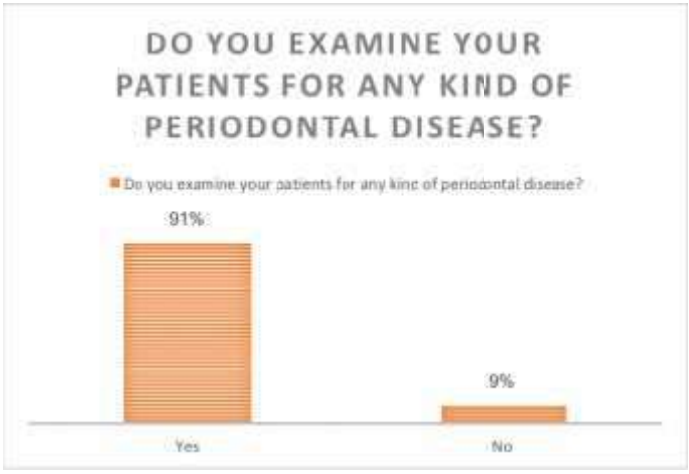


Figure.5-Their response when asked if they examine for any periodontal disease

Figure.6-Their response when asked if they could identify periodontal disease

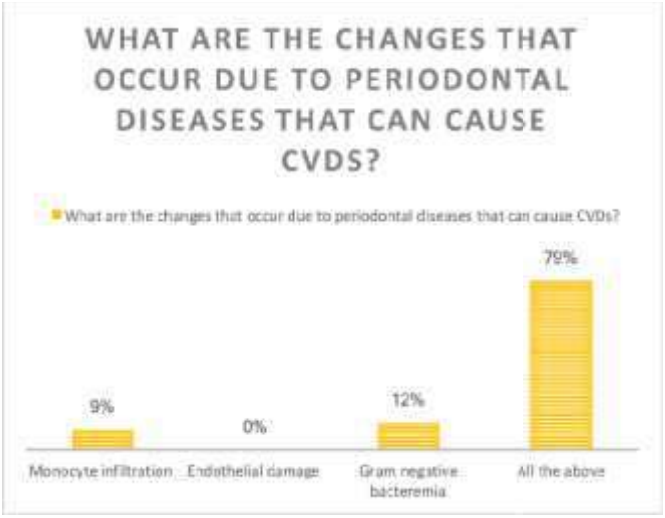


Figure.7-Their response when asked about the changes that take place during disease

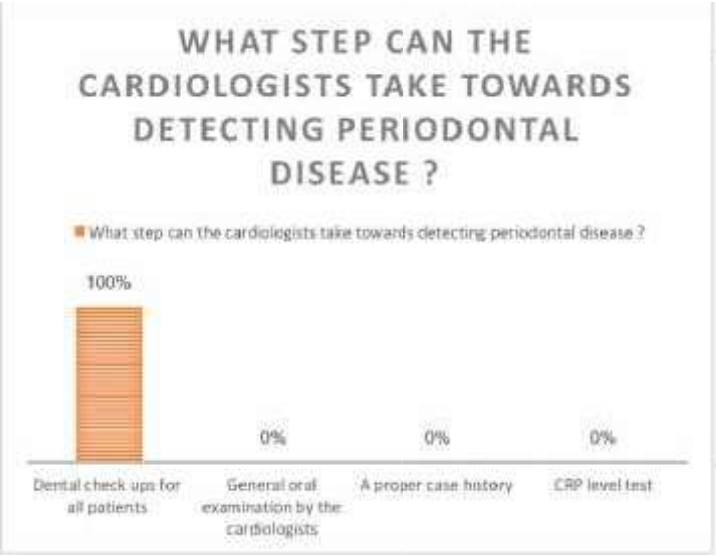


Figure.8-Their response when asked about the steps they can take to detect periodontal disease

Discussion

Periodontitis is a major public health concern because of its high prevalence in the population and its negative impact on oral health. In addition, as a progressive, chronic inflammatory disease, untreated periodontitis has the potential to lead to CHD [23]. Periodontal disease is a risk factor for the development of various systemic conditions like diabetes, cardiovascular disease, osteoporosis and adverse pregnancy outcome. [24] Risk factors associated with the development of PD include local, systemic, and genetic factors. Although several bacterial species are currently recognized as causally associated with periodontitis, merecolonization of the subgingival niche by these species is not sufficient for disease to occur. Instead, PD is thought to evolve from the stage of gingivitis, that is, a local inflammatory process without concomitant loss of periodontal tissue support that likely represents a stable, largely protective host response to periodontitis, an environment characterized by loss of connective tissue

attachment and alveolar bone, influenced by detrimental environmental exposures and specific genetic predispositions that are likely important determinants of susceptibility.Risk factors of periodontal disease include poor oral hygiene; cigarette smoking and also systemic conditions such as diabetes mellitus, rheumatoid arthritis etc. In addition, a number of genetic polymorphisms have been variably associated with propensity for periodontitis and ASVD.Risk factors of ASVD are multifactorial and involving genetic, environmental, and lifestyle factors. The associated risk factors include ethnicity, age, and family history of ASVD, which cannot be changed and those factorsthat can be modified or even controlled, includinghypertension, smoking, obesity, physical inactivity and diabetes mellitus. There is a clear role of the interactions between risk factors with cellular and non-cellular mechanisms in the slow process of atheromatous plaque, with unreliable evidence that by eliminating the risk factors, it is possible to prevent the atherosclerotic process and its clinical manifestations. In addition factors such as chronic inflammation which is associated with long-term progression of atherosclerosis, there are also “triggering factors” which are acute and include increased inflammation and also a cascade of hemostasis and thrombosis. Atherosclerotic plaqueruptures with thrombosis, leading to occlusion of the vessel progressing to clinical conditions such as MI or stroke.Many risk factors are shared between ASVD and PD and which could form the relationship between them. Various factors such as old age, smoking, alcohol, race, socioeconomic status, male gender, diabetes mellitus, and obesity are associated with both ASVD and periodontal disease.There is proof that periodontal bacteria and its byproducts of the bacteria have an effect on different systems in the body[25]. Most of the cardiologists had some knowledge on the etiology of periodontal disease of the bacteria and its effect on the body. For instance, a recent study of physicians knew that bacteria was the etiology of periodontal disease (86%)[26]. The current study had shown similar results, with 94% of the cardiologists reporting bacteria as the etiology of periodontal disease. Periodontal microorganisms which are found in dental plaque appears in the bloodstream and is directly proportional to the severity of the periodontal infection and canaffect distant sites. They have been thought to lead to systemic inflammation,impairment of insulin action, vast increase in glycogenesis in the liver, and can also alter the gut microflora which leads to systemic inflammation and metabolic changes. Porphyromonasgingivalis counts is found to be higher in sub-gingival dental plaque found in periodontal disease, where it leads to

inflammation and periodontal destruction.The organisms enter the bloodstream following tooth brushing,and has been found in the intima of distant blood vessels including in coronary arteries, where it proliferates, initiating an inflammatory cascade leading to apoptosis and consequent endothelial dysfunction, which is associated with hypertension. It also stimulates foam cell production in the intima leading to atherosclerosis. P. gingivalis has been shown to increase low-density lipoprotein (LDL) and total cholesterol levels by up-regulation of a protein which impacts circulating levels of LDL cholesterol in mice.There are two plausible underlying causal structures that can explain these findings: first, that periodontal disease (PD) causes CVD, and second, that PD and CVD have somecommon cause such as smoking or genetic polymorphisms that can lead individuals to develop both PD and CVD.From a study conducted in Iran, it was seen that 82% of the cardiologists agreed that inflammation is the key link between cardiovascular disease and periodontal disease [27] which is almost similar (79%) to the results from this current study. There is said to be an unknown genetic factors that may influence both the inflammatory diseases which is periodontal disease and cardiovascular disease and may therefore explain the association found in clinical studies [28].Apre-determined genetic phenotype is used to determine the risk of developing both atherosclerosis and infection. This is a hypothesis in which the presence of periodontal pathogens causes a susceptible person to developperiodontal disease. In the same person, atherosclerosis may develop, but, in this model the periodontal disease does not lead to the atherosclerosis.Only 54% of the cardiologists at the Iran study had some sort of knowledge on the correlation between the two diseases, whereas in this study, 97% of the cardiologists were said to have been oriented about the correlation. Only 80% of the doctors from the Iran study believed that doctors should educate the patient on the importance of oral health. But from this study it is seen that all the doctors agreed that educating their patients on oral health is very important. Physicians were asked if they perform oral examinations on their patients, 21% responded that they never perform oral examinations on their patients because they were not sure on how to diagnose it [29]. The most possible explanation of how periodontitis can lead to the development of CHD is that periodontal microorganisms and their products located in the periodontal pockets may enter into the circulation and lead to an inflammatory reaction. Mediators produced as part of this host response leads to the development, maturation and instability of fatty lesions (atheroma) in the

arteries, thereby increasing the risk of a CHD adverse event. Research shows that the microorganisms which enter the bloodstream during chewing, brushing, flossing or scaling depends upon the patient's dental flora which gives a picture of the periodontal status [30,31]. Bacteraemia is common, and is caused by a wider range of microorganisms in patients with periodontitis than those who are in an healthy state. Another possible mechanism by which periodontitis might contribute to CHD risk is that the antibodies which are produced as an inflammatory response to bacterial plaque may promote the development of atheroma by reacting with the endothelial cells and with blood lipids [30, 31]. Studies have proved that treating the periodontal disease reduces the inflammation within the circulation. Periodontal therapy is seen to have positive effects on two risk factors which are important markersof CHD: levels of C-reactive protein (CRP); and measures ofthe functioning of endothelial cell [30-33]. C - reactive protein (CRP) is a liver-produced, acute-phase reactant that serves as a systemic marker of inflammation. Levels of CRP can be used to monitor patients with overwhelming infections, and elevated CRP levels have been demonstrated in persons with ischemia and myocardial infarction.When monitoring a patient's acute-phase response, a serum CRP concentration exceeding 10 mg/L is generally regarded as the threshold indicative of significant inflammatory disease.Thus adequate awareness on the correlation between periodontal disease and cardiovascular disease is necessary among cardiologists to help promote better health and treatment to their patients.Studies have suggested that periodontitis is associated with coronary heart disease and cerebrovascular disease.Periodontitis is a chronic infection by Gram-negative bacteria that affects the supporting structures of the teeth.A mechanism proposed that periodontitis creates bacterial pathogens, antigens, endotoxins, and inflammatory cytokines that lead to athero-genesis and also thromboembolic events. In response to infection and inflammation, certain persons may exhibit greater expression of local and systemic mediators and may thereby be at increased risk for atherosclerosis. The atherosclerosis process may result in decreased arterial patency and/or decreased compliance of the vessel. Ultimately, atherosclerotic lesions may fissure and/or rupture, resulting in occlusion of the vessel lumen, precipitating a myocardial infarction or stroke.

Conclusion

ItwasseenthattheoverallawarenessamongCardiologistsinandaroundMogappaironthecorrelationbetweenperiodontaldiseaseandcardiovasculardiseaseswasgood.Allofthem agreedthatduringperiodontaldisease, thereisinflammationofthegingivaandthatcompletemanagementofperiodontaldiseaseisrequired in order to facilitate better cardiac health. All the doctors inquire the patients on their oral health and also send their patients for a dental check up. They agreed that there is a rise in the CRP levels during periodontal disease which is a marker to assess the cardiac health. Finally, all of them agreed that Cardiologists should advice their patients on the importance of oral health. Various programs like seminars, symposiums or any other awareness programs on the correlation of periodontal disease and cardiovascular disease can be arranged for doctors at hospitals so that it can benefit their patients in the long run.

Corresponding Author

Sreedevi Dharman

Senior lecturer,Department of Oral Medicine Department

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

E-mail:sanjamrut@gmail.com

Telephone Number: +919841009003

References

1. Jangid K, Doraiswamy JN, Varghese S, Malaiappan S. Periodontitis as a Risk Factor for Cardiovascular Diseases-Hype or a Fact?. Journal of Cardiovascular Disease Research Vol. 2015 Jan;6(1):25.

2. WHO report data of the year 1995
3. Lloyd-Jones D, Adams R, Carnethon M, De Simone G, Ferguson TB, Flegal K, Ford E, Furie K, Go A, Greenlund K, Haase N. Heart disease and stroke statistics—2009 update: a report from the American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation. 2009 Jan 27;119(3):e21-181.
4. Data from Centers for Disease Control and Prevention on Heart Diseases Facts and Statistics.
5. Joshipura K, Zevallos JC, Ritchie CS. Strength of evidence relating periodontal disease and atherosclerotic disease. Compendium of continuing education in dentistry (Jamesburg, NJ: 1995). 2009 Sep;30(7):430.
6. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. Circulation. 2002 Mar 5;105(9):1135-43.
7. Libby P. Inflammation and cardiovascular disease mechanisms–. The American journal of clinical nutrition. 2006 Feb 1;83(2):456S-60S.
8. Spahr A, Klein E, Khuseyinova N, Boeckh C, Muche R, Kunze M, Rothenbacher D, Pezeshki G, Hoffmeister A, Koenig W. Periodontal infections and coronary heart disease: role of periodontal bacteria and importance of total pathogen burden in the Coronary Event and Periodontal Disease (CORODONT) study. Archives of internal medicine. 2006 Mar 13;166(5):554-9.
9. Pussinen PJ, Alfthan G, Rissanen H, Reunanen A, Asikainen S, Knekt P. Antibodies to periodontal pathogens and stroke risk. Stroke. 2004 Sep 1;35(9):2020-3.
10. Suzuki JI, Aoyama N, Ogawa M, Hirata Y, Izumi Y, Nagai R, Isobe M. Periodontitis and cardiovascular diseases. Expert opinion on therapeutic targets. 2010 Oct 1;14(10):1023-7.
11. Miyamoto T, Yumoto H, Takahashi Y, Davey M, Gibson FC, Genco CA. Pathogen-accelerated atherosclerosis occurs early after exposure and can be prevented via immunization. Infection and immunity. 2006 Feb 1;74(2):1376-80.
12. Alonso González R, Pérez-Hernández A, Silvestre Rangil J, Silvestre Donat FJ. Cardiovascular disease versus periodontal disease: Chronic systemic infection as a link.
13. SumitMalhotra, HariParkash. Coronary Artery Disease and Periodontitis: A Prospective Study.JIMSA April - June 2013 26(2), 109-115

14. Czerniuk MR, Górska R, Filipiak KJ, Opolski G. Inflammatory response to acute coronary syndrome in patients with coexistent periodontal disease. Journal of periodontology. 2004 Jul 1;75(7):1020-6.
15. Howell TH, Ridker PM, Ajani UA, Christen WG, Hennekens CH. Periodontal disease and risk of subsequent cardiovascular disease in US male physicians. Journal of the American College of Cardiology. 2001 Feb 1;37(2):445-50.
16. Loos BG, Craandijk J, Hoek FJ, Dillen PM, Velden UV. Elevation of systemic markers related to cardiovascular diseases in the peripheral blood of periodontitis patients. Journal of periodontology. 2000 Oct 1;71(10):1528-34.
17. Hujoel PP, Drangsholt M, Spiekerman C, DeRouen TA. Periodontitis–systemic disease associations in the presence of smoking–causal or coincidental?. Periodontology 2000. 2002 Oct 1;30(1):51-60.
18. Hujoel PP. Does chronic periodontitis cause coronary heart disease?: A review of the literature. The Journal of the American Dental Association. 2002 Jun 1;133:31S-6S.
19. Tuominen R, Reunanen A, Paunio M, Paunio I, Aromaa A. Oral health indicators poorly predict coronary heart disease deaths. Journal of dental research. 2003 Sep;82(9):713-8.
20. Lösche W, Marshal GJ, Krause S, Kocher T, Kinane DF. Lipoprotein- associated phospholipase A2 and plasma lipids in patients with destructive periodontal disease. Journal of clinical periodontology. 2005 Jun 1;32(6):640-4.
21. Slavkin HC. Does the mouth put the heart at risk?. The Journal of the American Dental Association. 1999 Jan 1;130(1):109-13.
22. MeenakshiPriyanka V., Jaiganesh Ramamurthy; Periodontal disease- Systemic disease inter relationship questionnaire study ; Int J Pharm Bio Sci 2015 July.; 6(3): (B) 16 - 21
23. Friedewald VE, Kornman KS, Beck JD, Genco R, Goldfine A, Libby P, Offenbacher S, Ridker PM, Van Dyke TE, Roberts WC. The American Journal of Cardiology and Journal of Periodontology editors' consensus: periodontitis and atherosclerotic cardiovascular disease. American Journal of Cardiology. 2009 Jul 1;104(1):59-68.
24. Owens JB, Wilder RS, Southerland JH, Buse JB, Malone RM. North Carolina internists' and endocrinologists' knowledge, opinions, and behaviors regarding periodontal disease

and diabetes: need and opportunity for interprofessional education. Journal of dental education. 2011 Mar 1;75(3):329-38.

25. Kashefimehr A, Faramarzi M, Shirmohammadi A, Zarandi A, Ilkhani S. Awareness regarding the effects of periodontal diseases on coronary heart system among cardiologists in Tabriz, Iran. Journal of Periodontology & Implant Dentistry. 2014;6(1):23.
26. Rohde LE, Hennekens CH, Ridker PM. Survey of C-reactive protein and cardiovascular risk factors in apparently healthy men. American Journal of Cardiology. 1999 Nov 1;84(9):1018-22.
27. Mosley M, Offenbacher S, Phillips C, Granger C, Wilder RS. North Carolina cardiologists' knowledge, opinions and practice behaviors regarding the relationship between periodontal disease and cardiovascular disease. American Dental Hygienists Association. 2015 Jun 1;89(suppl 2):38-48.
28. Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes. New England journal of medicine. 1992 Jan 23;326(4):242-50.
29. Hegele RA. The pathogenesis of atherosclerosis. Clinica Chimica Acta. 1996 Mar 15;246(1-2):21-38.
30. Kinane DF, Lindhe J. Pathogenesis of periodontitis. Clinical Periodontology and Implant Dentistry, 3rd ed. 1997, 189-225.
31. Cochran D. Periodontology in the age of inflammation: a changing landscape. The Journal of the American College of Dentists. 2009;76(1):23-6.

Choice of Rotary Instrument Usage among Endodontists – A Questionnaire Study

Kiruthika Patturaja[1],Jayalakshmi. V[2], L. Leelavathi[3]

Graduate Student[1],Senior Lecturer[2],Department of Conservative Dentistry and Endodontics, Senior Lecturer[3],Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: A rotary instrument is used to remove or reduce tooth matter and to shape teeth during endodontic procedure. Rotary instrument includes burs and endodontic files. Various brands of rotary instruments are currently available in the market which includes dentsplyprotaper, Mtwo, endostar, k3 XF files, Heroshaperetc. The choice of rotary instrument by a dentist is governed by various factors like their efficiency, ductility, sharpness, cross section and flexibility.The aim of the study is to find the rotary instrument usage and preferred brand of rotary instrument among endodontists working in dental colleges, Chennai.

Materials and Methods: An online questionnaire based study was conducted among 97 endodontistsworking in dental colleges in Chennai.A total of 10 questions consisting of both open ended and close ended questions related to usage, preferred brand and properties were included in the questionnaire. Data werecollected from the filled questionnaire and analysed. Descriptive statistics was used.

Results: From the study it was reported that 88% of endodontists use rotary instruments for endodontic procedures in their clinical practice.The most preferred brand of rotary instrument used by endodontists is Mtwo(40%) because of its good cutting ability and shaping followed by 20% k3, 18% protaper, 6% heroshaper and 36% combination of different rotary instruments.

Conclusion: Majority of the endodontists preferred using rotary instruments and nearly half of them opt to use mtwo file in their clinical practice.

Key Words:*rotary instruments, endodontists,NiTi, flexible.*

Introduction

The root canal treatment consists of removal of bacterial debris from the canal by biomechanical means like cleaning and shaping to prevent reinfection (1).Root canal anatomy is quite complex and to clean and shape these canals successfully requires appropriately designed instruments (2). The cleaning and shaping of root canal systems by various techniques includes usage of hand instruments and rotary instruments.The aim of root canal treatment is to prepare tapered funnel shape with the smallest diameter at the apex and the widest diameter at the canal orifice.

Stainless steel instruments have been used for a long time in endodontics; these instruments are ineffectivein the maintenance of the original canal shape because of their low flexibility and its time consuming (3). Lack of flexible nature of stainless steel instruments has led to procedural errors and decreased success rate (4,5). Hand instruments have the tendency to straighten themselves after using in a curved canal and, if used improperly, they could straighten curved canals (6).

The engine driven Nickel-Titanium (NiTi) rotary instrumentshave been engaged in faster and easier root canal system (7).Rotary instruments are preferred by practitioners for their super-elasticity, excellent flexibility, improved cutting efficiencyand better centering ability(8).Rotary instruments have been found to removefilled restorative material efficiently and safely (9,10) . Rotary instruments has shown increased fatigue resistance to sterilisation preventing torsional stress (11).However there are certain disadvantages concerned with rotary instruments such as cost and instrument fracture (12).

Various brands of rotary instruments are available in market and currently used by endodontists which includes k3,Mtwo ,heroshaper, protaper , endostar, HyFlex, etc.The aim of this study isto find the rotary instrument usage and the most preferred brand of rotary instrument used among endodontistsworking in private dental colleges in Chennai.

Materials and Methods

An online questionnaire based study was conducted among endodontists working in dental colleges.The Questionnaire was circulated among120 endodontists working in private dental colleges in Chennai, India through mail. This study was conducted from 15thDecember - 31stDecember 2016.Ethical clearance was obtained from the Institution Review Board for conducting the survey.A sample size of 94 was calculatedbased on the prevalence of rotary instrumentsusage in the previous study (17). Convenient sampling methodology was followed to select the study samples. Mail was sent to the endodontists explaining about the purpose of the study. Out of 120 endodontists,97accepted to participatein the study.A pretested questionnaire was used and the questionnaire consists of 10 questions which were both open ended and close ended questions. There was neither time limitation nor true/false based questions. The questions were related to the usage, brand, frequency, advantages and limitations of the rotary instrument used. Questions in the questionnaire used were selected from national and international surveys. Participation was voluntary and anonymous and no personal data were collected.

Questionnaire was mailed to them and completely filled questionnaire was considered for analysis. The returned questionnaire containing unanswered questions were excluded. Data was entered in the excel sheet and descriptive statistics were obtained from the data collected.

Results

A cross sectional study was conducted among 97 endodontists working in dental colleges in Chennai and it presented an overall view about the choice of rotary instrument used and preferred among endodontists.

The results of the study showed that 31% of endodontists have used rotary instruments in endodontic practice and 13 % used only hand instruments for cleaning and shaping , while 56 % used combination of both rotary and hand instruments,asthey claimed rotary instruments are not used in the anterior tooth region due to lack of cuspal and dentinal walls (figure 1).It was found from the study that around 90 endodontists ie.88% preferred to use rotary instruments for cleaning and shaping of the root canal and about 12 % endodontists preferred to use hand instruments (fig 2).

. Based on the physical properties of rotary instruments about 47 % of endodontists has chosenitflexible which is an important property for working in straight and curved root canals. About 32% of endodontists has reported corrosion resistance as an ideal property and 21% has claimed rotary instruments to be fatigue resistant (figure 3).

Depending upon the frequency of rotary instruments usage in a week for endodontic procedure, 31 (32%) endodontists has used it only once in a week.The highest frequency of rotary instruments usage was found to be three times in a week by 53 endodontists (54.7%) . while about11 (11.3%) endodontists use rotary instruments five times in a week; rotary usage formore than 5 times in a week was the least reported 2 %. This frequency is based on experience with rotary instruments(table 1). The most preferred brand of rotary instrumentisMtwoby40% ofendodontists, followed by 20%k3, 18 %protaper,6% heroshaper and6%ofendodontists use other different brands of rotary instruments(fig 4).About36% of endodontists use combination of one or more brands of rotary instruments for effective cleaning and shaping.Most commonly used combination of rotary instruments among endodontists in the study were heroshaper and protaper for effective cleaning and shaping (fig 5). Question about advantages of rotary instruments revealed the following response,35% of endodontists preferred rotary instruments for its good cutting ability while22%preferred rotary for its perfect shaping of canals.18 % of endodontists preferred rotary instruments for its time saving property and 14% for efficiency of the rotary instrument. Very few endodontistschoose rotary instruments to be economical 6% and for its long term usage 5% (figure 6). The most commonly associated limitation of rotary instrument was file fracture by 39(40%) of endodontists . Theleastcommonly reported limitation of rotary instrument is ledge formation12 (13%).Other common disadvantages reported by endodontists are canal perforation 20 (21%) and excess dentin removal 26 (26%)during Canal preparation by rotary instrument (table 2).

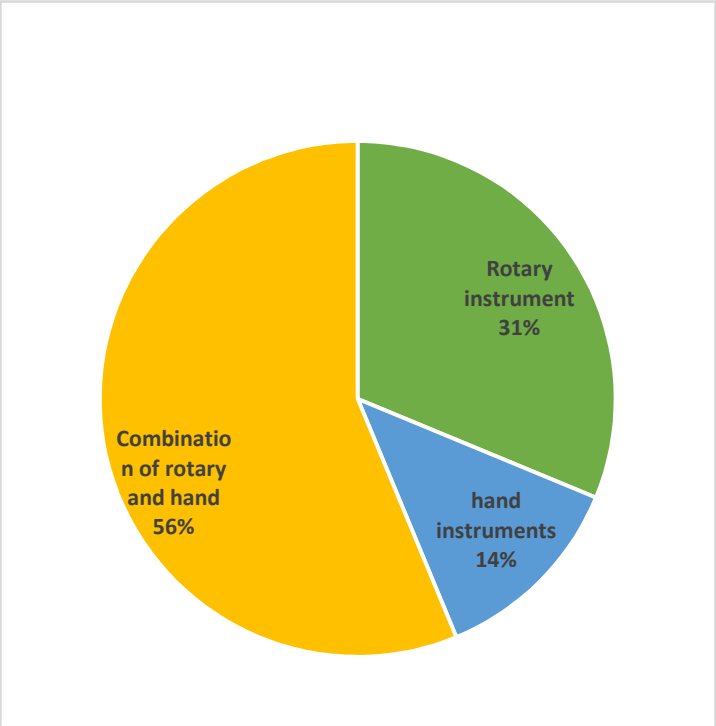


Figure 1: Distribution of study population based on instrument used for endodontic practice

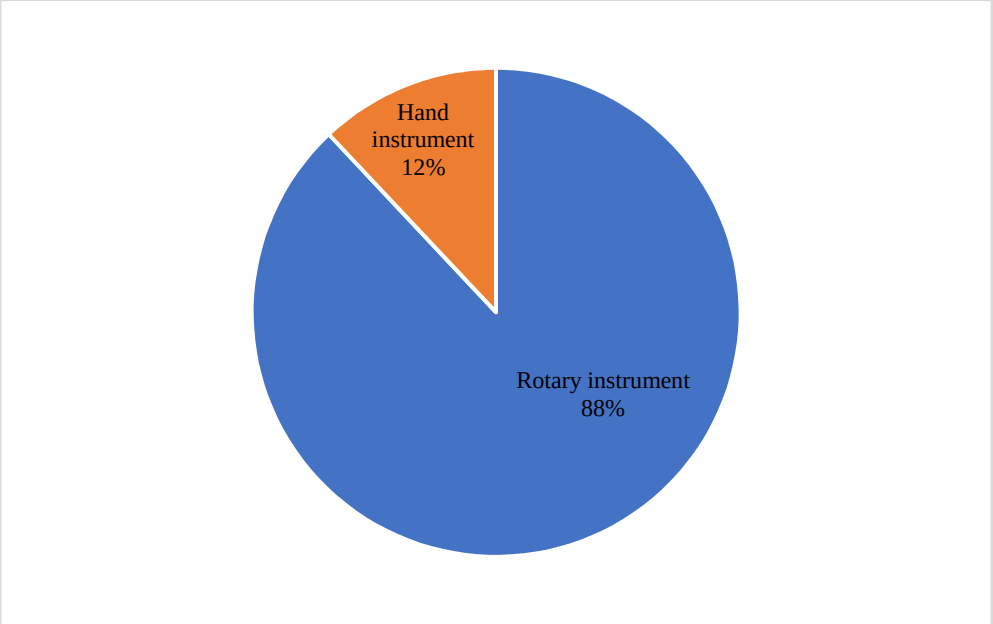


Figure 2: Response of study participants based on preferred instrument used for

endodontic practice.

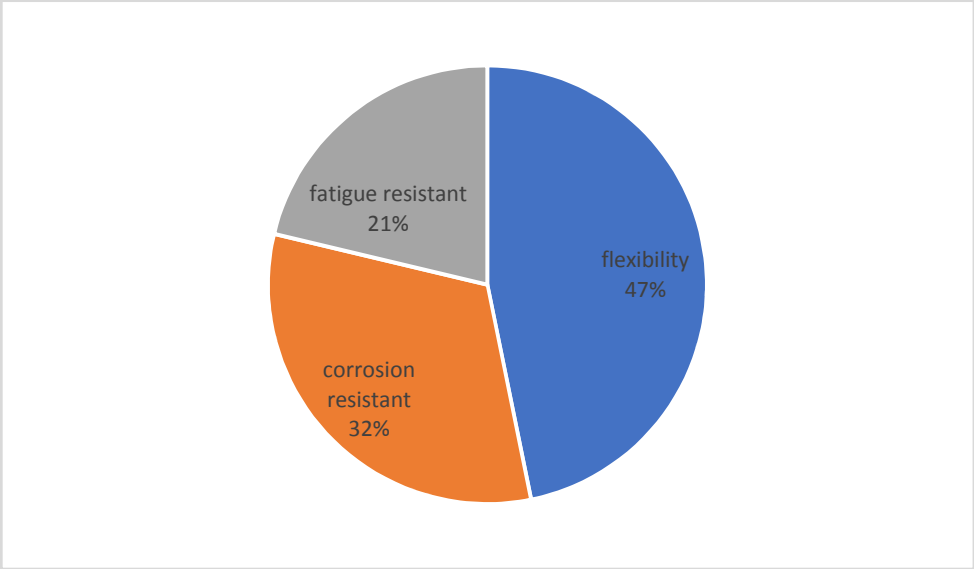


Figure 3: Response of study participants based on properties of rotary instrument used

Table 1: Responses of study participants related to frequency of rotary instrument usage

Frequency of rotary instrument usage (weekly)	N (%)
Once	31 (32%)
3 times	53 (54.7%)
5 times	11 (11.3%)
>5 times	2(2%)

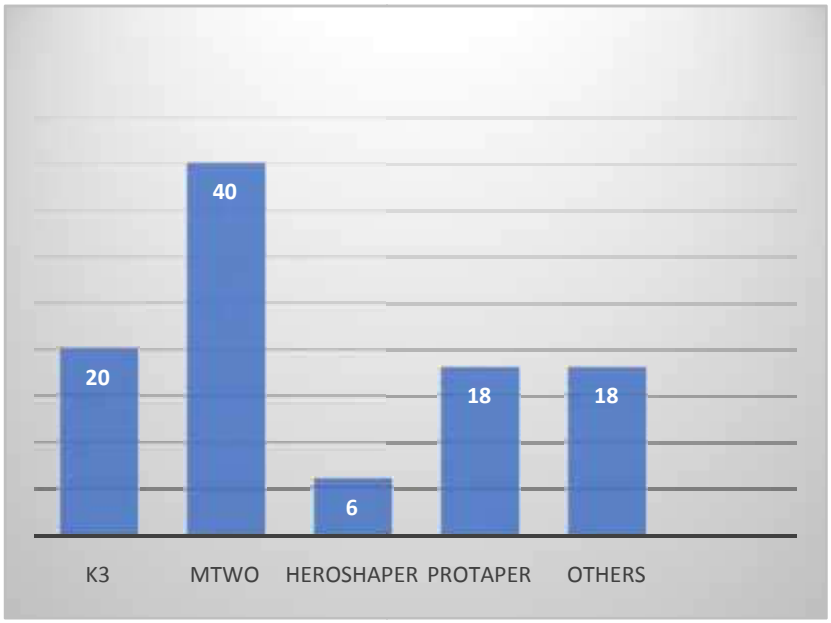


Figure 4: Distribution of study participants based on preferred brand of rotary instrument

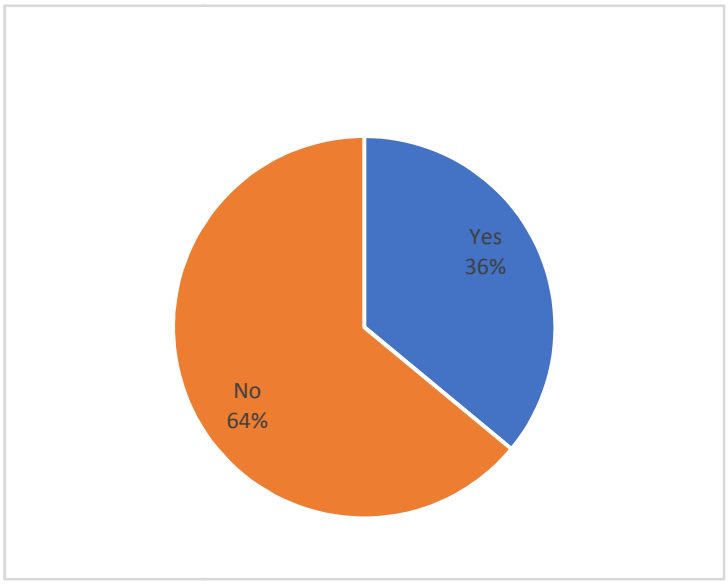


Figure 5: Response based on combination of rotary instruments used

Table 2: Responses of study participants regarding limitations of rotary instruments

Limitations of rotary instruments	N (%)
File fracture	**39** (40 %)
Excess dentin removal	**26**(26 %)
Canal perforation	**20** (21%)
Ledge formation	**12** (13%)

Figure 6: Responses of study participants regarding advantages of rotary instrument

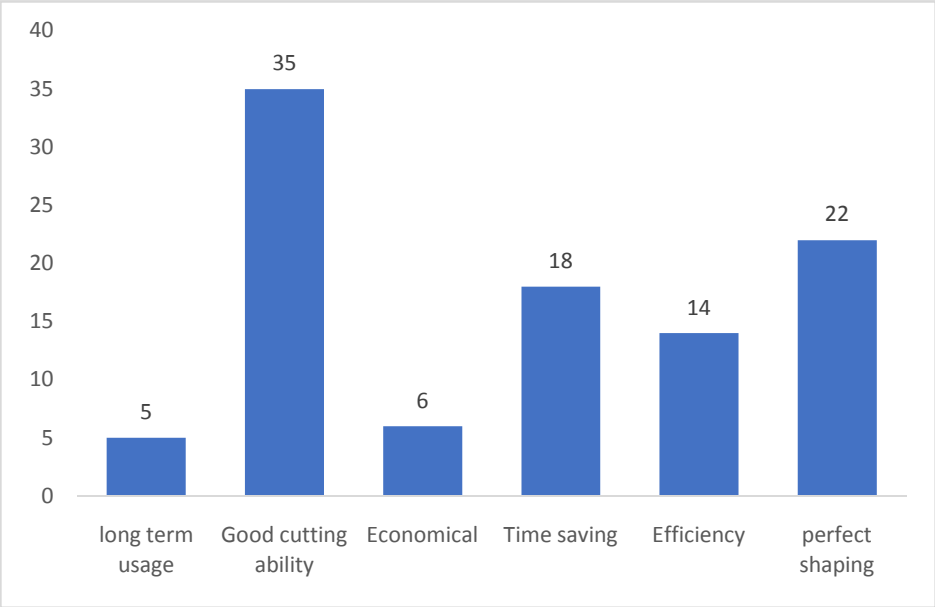


Figure 6: Responses of study participants regarding advantages of rotary instrument

Discussion

The cleaning and shaping of root canal is one of the important step in endodontic therapy. Cleaning and shaping remove all the tissue debris and inner layers of root canal dentin regardless of the instrumentation technique **(**3**) .**Rotary instruments has a greater ability to negotiate curved canals and reduce iatrogenic errors by maintaining the original path and allows larger apical preparation of canals (13).Hence a study was done to find the rotary instrument usage among endodontists working in dental colleges in Chennai. The investigations from our study show that most of the endodontists about 88% prefer using rotary instruments for their endodontic practice. In a study conducted by Jyothi.Set al among practitioners in chennai, 2016it was found that non- rotary manual endodontic files were commonly used by the practitioners (14)

There are various advantages of rotary instruments which include flexibility ,good cutting ability ,time saving and perfect shaping of the canals. The super-elastic property of NiTi, allows safe and effective instrumentation of curved and narrow root canals using hand piece driven instruments operated at low speed (15).It has been reported in our study that flexibility (40%) is an ideal property for usage of rotary instruments.In an invitro study by Peter et al, 2012 it was reported that high fatigue resistance and flexibility was enhanced by HyFlex rotary instruments (16) . Machado et al,2010compared the disinfectant property of two rotary instruments, ProTaper and Mtwo reduced the amount of bacteria in the mechanical disinfection of the root canal system (17).Few disadvantages are also concerned with the use of rotary instruments which include cost, instrument fracture, difficulty to use and lack of knowledge (4). The major limitation reported was file fracture by 40% of endodontists . Flexural fatigue of rotary instruments is the main reason for file fracture (18).Schirrmeister, J. F. et al 2006, compared hand file and rotary instruments in removing the guttapercha from curved root canals and reported thatRaCe system is an efficient and safe device for gutta-percha removal in curved root canal compared to flex master, protaper and hand instruments (19).

Recently introduced rotary NiTi files are able to produce a uniformly tapered canal configuration without canal transportation. Electropolishing may have beneficial effects in prolonging the fatigue life of rotary NiTi instrumentsbyreducing themsurface irregularities that serve as points for stress concentration and crack initiation(20).However, unpredictable

instrument separation remains a deterrent to their popularity. Moreover, cost of rotary files also restrained their use. Majority of practitioners only replaced their instruments when signs of distortion and bluntness were obvious, which is likely to result in a higher risk of instrument separation in the canal (4)

Mohammad Ali et al ,2009study on the usage of rotary instruments among general dentists and endodontist, general dentists and dental students require more training and more comprehensive education regarding rotary instruments and techniques (21).It was reported by 17% of endodontists that rotary instruments helps in faster canal preparation and is time saving. Abu-Tahun et al,2015 investigated among fifth year undergraduate dental students in Australia thatrotaryfiles were able to prepare root canals faster with more preparation accuracy compared to hand instruments for same tooth(22).Endodontists were significantly more satisfied than those treated by general dentists mainly because of the shorter treatment time by using rotary instruments (23).

The most preferred brand of rotary instrument reported was Mtwo (40%) followed by combination of different rotary instruments like Heroshaper ,protaper.Ina study done by AlaviehVahidEt al,2009 in Australia four rotary instruments were compared and assessed for preserving canal curvature, preparation time and change of working length it was reported that Mtwo rotary instruments required significantly less instrumentation time, and the ProTaper significantly changed the canal curvature(5). In our present study, Canalperforationwas reported by 26% of the endodontists . Bier et al , 2009 estimated the ability to induce dentinal damage during canal preparation using NiTiandfoundthatProTaper, ProFile, and GT preparations resulted in dentinal defects in 16%, 8%, and 4% of teeth, respectively during canal preparation (24).In the present study it was reported that excess dentin removal 26% is one of the limitation of rotary instruments.Ramanathan et al 2017, in an invitro study concluded that ProTaper Universal and ProTaper Next should be used judiciously, as it causes higher thinning of root dentin of the root when compared with Mtwo (25).In the present study, the assessed frequency of rotary instrument used by endodontist was three times (54%) in a week.Parashos et al, 2014 conducted a survey among Australian dentist and reported about 70% of dentists used the instruments two to five times,19% used six to ten times and 12% used instrument only once this usage paws basedonfile size and

canal shape(4) .Rotary instrument usage increased with increasing work experience suggesting that dentist with less experience are less likely to use (21).

Therefore it is acknowledged that interpretation of any survey data must consider the possibility of incorrect answers because of factors related to questionnaire design, question wording and respondent factors.Generalisability of this study may be limited, as it represents only views of practitioners working in dental colleges in Chennai.

Conclusion

It can be concluded from the study thatmajorityof endodontists use rotary instruments for endodontic procedures in their clinical practice. Nearly half of the study participants reported that the most preferred brand of rotary instrument wasMtwo because of its good cutting ability and shaping.

Corresponding Author

L. Leelavathi,

Senior Lecturer, Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India.

Mobile Number: +91 9443702882,

Email Id: karleela81@gmail.com

References

1. 1.Bystrom A, Sundqvist G. Bacteriologic evaluation of the efficacy of mechanical root canal instrumentation in endodontic therapy. Scand J Dent Res 1981;89:321– 8.
2. Mangalam, S., C. V. Rao, and L. Lakshminarayanan. "Evaluation of apically extruded debris and irrigant using three instrumentation techniques." *Endodontology* 14 (2002): 19-23

3. Ersev, Handan. "A comparison of the shaping effects of 5 nickel-titanium rotary instruments in simulated S-shaped canals." Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology 109.5 (2010): e86-e93.
4. Parashos, P., and H. H. Messer. "Questionnaire survey on the use of rotary nickel–titanium endodontic instruments by Australian dentists." International endodontic journal 37.4 (2004): 249-259.
5. Vahid A, Roohi N, Zayeri F. A comparative study of four rotary NiTi instruments in preserving canal curvature, preparation time and change of working length. Australian Endodontic Journal. 2009 Aug 1;35(2):93-7.
6. Cheung, Gary SP, and Christopher SY Liu. "A retrospective study of endodontic treatment outcome between nickel-titanium rotary and stainless steel hand filing techniques." Journal of endodontics 35.7 (2009): 938-943.
7. Gambill JM, Alder M, Carlos E. Comparison of nickel-titanium and stainless steel hand-file instrumentation using computed tomography. Journal of Endodontics. 1996 Jul 1;22(7):369-75.
8. Daugherty, Darrell W., Tom G. Gound, and Toby L. Comer. "Comparison of fracture rate, deformation rate, and efficiency between rotary endodontic instruments driven at 150 rpm and 350 rpm." Journal of endodontics 27.2 (2001): 93-95.
9. Karova E, Dogandzhiyska V. Undergraduate Teaching for Using Rotary Nickel-titanium Endodontic Instruments. International Journal of Dental Sciences and Research. 2016 Nov 9;4(6):95-9.
10. Hülsmann, M., and V. Bluhm. "Efficacy, cleaning ability and safety of different rotary NiTi instruments in root canal retreatment." International Endodontic Journal 37.7 (2004): 468-476.
11. Viana AC, Gonzalez BM, Buono VT, Bahia MG. Influence of sterilization on mechanical properties and fatigue resistance of nickel–titanium rotary endodontic instruments. International Endodontic Journal. 2006 Sep 1;39(9):709-15.
12. Alapati SB, Brantley WA, Svec TA, Powers JM, Mitchell JC. Scanning electron microscope observations of new and used nickel-titanium rotary files. Journal of Endodontics. 2003 Oct 31;29(10):667-9.

13. Walia H, Brantley WA, Gerstein H. An initial investigation of the bending and torsional properties of Nitinol root canal files. Journal of endodontics. 1988 Jan 1;14(7):346-51.
14. Jyothi.S, Pradeep.S. Practice of endodontic therapy among practitioners: a questionnaire survey. International Journal of Pharmacy & Technology, 2016; 8(3): 16546-16558
15. Serene TP, Adams JD, Saxena A. Nickel-titanium instruments: applications in endodontics. IshiyakuEuroAmerica; 1995.
16. Peters OA, Gluskin AK, Weiss RA, Han JT. An in vitro assessment of the physical properties of novel Hyflex nickel–titanium rotary instruments. International endodontic journal. 2012 Nov 1;45(11):1027-34.
17. M.E. de Lima Machado, L.A.B. Sapia, S. Cai, G.H.R. Martins, C.K.Nabeshima .Comparison of two rotary systems in root canal preparation regarding disinfection. J Endod, 36 (2010), pp. 1238-1240
18. Sattapan B, Nervo GJ, Palamara JE, Messer HH. Defects in rotary nickel-titanium files after clinical use. Journal of Endodontics. 2000 Mar 31;26(3):161-5.
19. Schirrmeister JF, Wrbas KT, Schneider FH, Altenburger MJ, Hellwig E. Effectiveness of a hand file and three nickel-titanium rotary instruments for removing gutta-percha in curved root canals during retreatment. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 2006 Apr 30;101(4):542-7.
20. 20.Anderson ME, Price JW, Parashos P. Fracture resistance of electropolished rotary nickel–titanium endodontic instruments. Journal of Endodontics. 2007 Oct 31;33(10):1212-6.
21. Mohammad Ali Mozayeni, Amin Golshah, and NafisehNikKerda. A Survey on NiTi Rotary Instruments Usage by Endodontists and General Dentist in Tehran, Iran. Endod J. 2011 Autumn; 6(4): 168–175.
22. Abu- Tahun I, Al- Rabab'ah MA, Hammad M, Khraisat A. Technical quality of root canal treatment of posterior teeth after rotary or hand preparation by fifth year undergraduate students, The University of Jordan. Australian Endodontic Journal. 2014 Dec 1;40(3):123-30.

23. Dugas NN, Lawrence HP, Teplitsky P, Friedman S. Quality of life and satisfaction outcomes of endodontic treatment. Journal of endodontics. 2002 Dec 31;28(12):819-27.
24. Bier CA, Shemesh H, Tanomaru-Filho M, Wesselink PR, Wu MK. The ability of different nickel-titanium rotary instruments to induce dentinal damage during canal preparation. Journal of Endodontics. 2009 Feb 28;35(2):236-8.
25. 25.Ramanathan S, Solete P. Cone beam computed tomography evaluation of root canal preparation using various rotary instruments: an invitro study. The journal of contemporary dental practice, 2017; 16(11): 869-872

Annexure 1: Questionnaire

1. what type of instruments do you use for endodontic procedure?

a. Rotary instruments

b.hand instruments

c.Combination of a and b.why?

2. Which type of instrument do you think is more convinient ?

a.rotary instruments

b.hand instrument

3.Which brand rotary instruments do you prefer?

a. K3

b. Mtwo

c. Heroshaper

d. Protaper

e. Others

4.Do you use combination of rotary instruments?

a.yes

b.no

5.If yes , mention the combination of rotary used

6.How frequently(in a week) do you use rotary instruments?

a.once

b.three times

c. Five times

D. More than 5 times

7.Why do you prefer using rotary instruments?

a.Time saving

b. Efficiency

c.perfect shaping

8.what are the advantages of the rotary instrument you use?

a.long term usage

b.cutting ability

c.Cross section

d. Economical

9.What property do you think is important for rotary instruments?

a.flexibility

b.corrosion resistant

c.fatigue resistant

10.what do you think is the limitation of endodontic instrument

a.file fracture

b. Excessive dentin removal

c.canal perforation

d.ledge formation

Cognisance of Endodontic Mishaps Among UndergraduateStudents

M.Indhumathi[1], Nivethithan.T[2], Hemavathy.O.R[3],

Graduate Student[1], Tutor[2], Department of Publicj Health Dentistry

Reader[3], Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The main aim of Root canal treatment is to restore the diseased pulp. Endodontic mishaps are some accidental happenings that occurs during the procedure it may be seen during access opening, instrumentation, obturation and post space preparation. So it is important to know about the errors occurring during the root canal treatment. The aim of the study is to evaluate the awareness of endodontic mishaps among undergraduate students.

Materials and Methods:A cross-sectional questionnaire based study on awareness of endodontic mishaps among undergraduate students. Data were collected from 200 undergraduate students of Saveetha dental college, Chennai. Correct responses to the questions were allocated 1 mark while wrong response received no mark. Statistical analysis was performed to analyse the study.

Conclusion: This study is useful to gain knowledge about the procedural errors during root canal treatment.

Key Words: *cognisance, knowledge, mishaps*

Introduction

Endodontics generally denotes to the procedures relating to the treatment of diseased dental pulp occurring due to effects of long standing dental caries.[1]The mainpurpose of the endodontic treatment is to relieve pain and maintain the structure and integrity of the tooth in the oral cavity.[2,3]Root canal treatment forms the core of endodontic procedures and its primary goal is to debride and sterilize the necrosed pulp tissue and to obturateall root canals to form a fluid-tight seal on the apical foramen of the tooth, so that any possibility of a secondary infection occurrence due to the mouth cavity or periradicular tissue leakage into the root canal system can be avoided.[4]Endodontic mishaps or procedural accidents are those unfortunate accidents that happen during treatment, some owing to inattention to detail, others being totally unpredictable.[5,6,7]. These endodontic mishaps play a vital role in determining the prognosis of the treatment done to the diseased teeth.[14,15]

Materials and Methods

A cross sectional questionnairetodetermine the cognisance of endodontic mishaps among undergraduate students was designed .Data were collected from 200 undergraduate dental students of Saveetha dental college, Chennai.The undergraduate dental training in Saveetha dental college is divided into the pre-clinical levels first and second year of study and the clinical levels [3rd and 4th] of study. This study was conducted for third year, fourth year and internship students. There are twenty directquestions regarding the endodontic mishaps, their identification and its prevention.[12,13] Each question had four options and each correct responses to the questions were allocated 1 mark and the wrong responses revealed no marks. The questions are given below.In this study the percentage of awareness has to be noted based on their knowledge and the statistical analysis was performed to analyse the study.

Results

Endodontic treatment can be considered a stressful procedure for undergraduate students. It usually requires competent technical skills and experience, as well as an understanding of pulp anatomy and its variations.[15] Knowledge of root canal morphology and tooth pulp chamber will allow the student to avoid any mishaps. In many cases, insufficient knowledge makes endodontics more difficult than necessary by not creating a proper access that allows

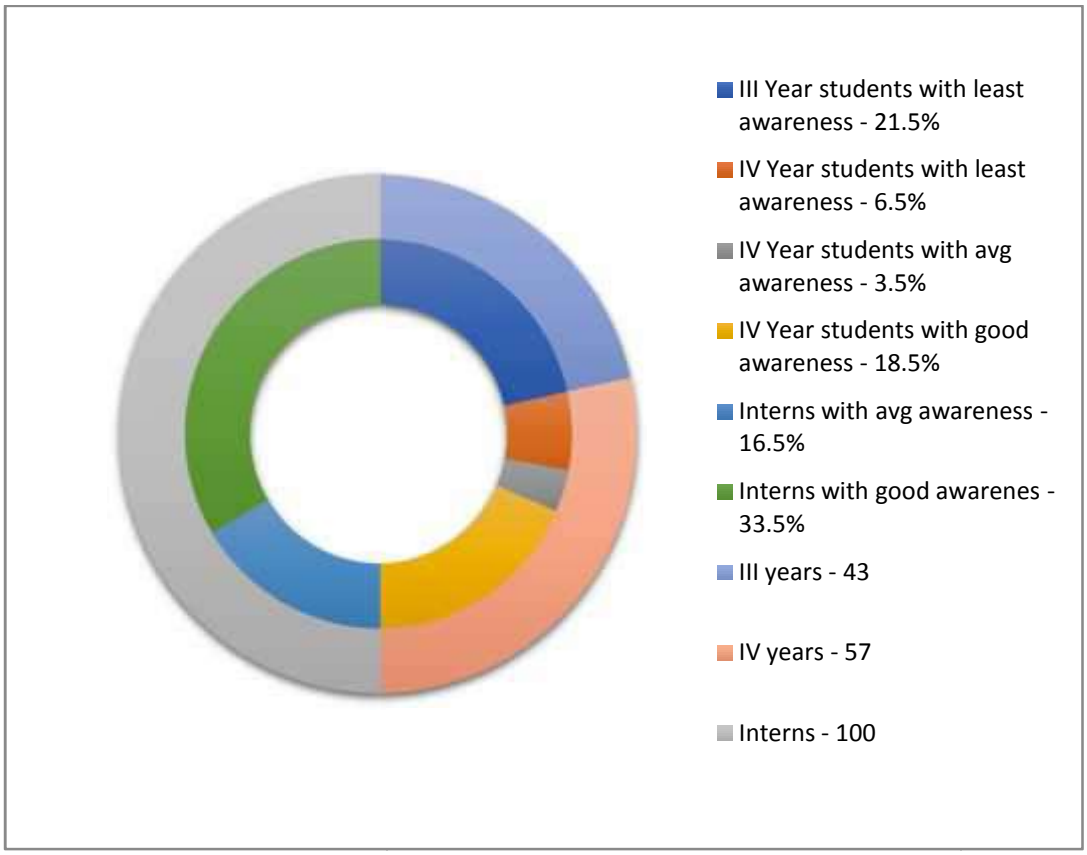
straight line approach to the canals. This is especially true for mesiobuccal canals in both molar teeth. Perforation of pulp chamber can occur during a fruitless search for the canal orifices, drilling a tooth with a completely calcified chamber, orienting the bur to the wrong direction, or looking in the wrong direction for the other canal. Students reported gouging and perforation during access cavity preparation. This is considered normal for undergraduate student.[16]

In order to reduce the incidence of iatrogenic defects, step-down technique is recommended for undergraduate students. This technique depends on the use of larger instruments at the canal orifice during preparation, then working down the root canal with progressively smaller files.[16,17] This offers several advantages, including straighter access to the apical region and enhanced tactile control, as well as improved irrigant penetration and suspension of debris. Using this approach, most of the problems that occur during root canal instrumentation can be avoided.[18,19]

The study records based on the questionnaire, table 3 showed that all 200 students contacted regarding the study responded positively agreed to participate. These 200 students included 72 males and 128 females; they all belonged to undergraduate clinical training in Saveetha Dental College which includes third year, fourth year and internship students. [8,9]Among the 200 students, third year students accounted to 43 [21.5%], final year students accounted to 57[28.5%] and interns accounted to 100[50%] of the total participants. The questionnaire circulated had 20 questions and the least number of correct responses to any of those 20 questions accounted to 110 [55%]. By analysing the data obtained from the questionnaires , showed that almost all third year students [21.5%] who participated had the least awareness, among 57[28.5%] final year students who participated; 37 students [18.5%] had good awareness, 7 students[3.5%] had average awareness and 13 students [6.5%] had least awareness; and among all interns who participated 100 students[50%], 67 students [33.5%] had good awareness and remaining 33 students [16.5%] had average awareness. So based on the data collected the table 4, shows the results indicate that; 44[77%]of 57final year students and all 100 interns who participatedhad above average awareness;37 [65%] of 57 total final year students and 67 [67%] of 100interns had good awareness among the total 200 participants. In total comparing all students, the interns had good awareness to endodontic mishaps, which can be clearly attributed to their clinical experiences and knowledge acquired during their undergraduate training.[10,11]

Table 1: Data obtained from questionnaire based on the criteria of cognisance of endodontic mishaps among undergraduate students.

Year of study	**Correct responses [Out of 20 questions]**	**No of students who answered correctly.**	**Awareness among the students.**
Third year	5-10	43 [21.5%]	Poor
Final year	15-20	37 [18.5%]	Good
	10-15	07 [3.5%]	Average
	5-10	13 [6.5%]	Poor
Internship	15-20	67 [33.5%]	Good
	10-15	33 [16.5%]	Average



Graph 1: Doughnut chart showing data obtained.

Table 2: Showing the aggregate data representing the above average and good awareness among undergraduate students.

Year of study	**Number and percentage Of students from each year among 200 UG students**	**Number and percentage Of students among their respective batches**	**Awareness among the students.**
Third year	43 [21.5%] of 200	43 [21.5%]	Least
Final year	53 [28.5%] of 200	37 [65%] of 57	Good
		44 [77%] of 57	Good + Average
Internship	100 [50%] of 200	67 [67%] of 100	Good
		100 %	Good + Average

Discussion

The study was conducted based on a self prepared questionnaire circulated among 200 undergraduate students of Saveetha dental college, Chennai. 72 males and 128 females answered the questionnaire based on this study.They belong to 18-24 yrs of age group.This survey was done among undergraduate students studying third year, fourth year and doing internship of Bachelor degree of dental surgery in Saveetha dental college.[20] The data obtained was categorised individually for all 20 questions and the resulting correct responses were considered to determine the awareness among students.

Table 3: Data of the questionnaire

Questions	**Option A**	**Option B**	**Option C**	**Option D**
1.Which age group do you belong to?	18-24	25-30	31-40	41-60
2.Mention your educational qualification	Secondary education[school]	Bachelor Degree	Master Degree	Diploma
3.Endodontic mishaps are most commonly associated with?	Access related	Instrument related	Obturation related	None of the above
4.Which is the least common access related error you will face during	Treating wrong tooth	Missed canals	Access cavity perforations	Crown fracture

RCT?				
5.Which is the most common Instrument related error you will face during RCT?	Ledge formation	Canal perforation	Instrument separation	None of the above
6.What are the errors which occurs during obturation in RCT?	Over or under extended root canal fillings	Nerve parasthesia	Vertical root fractures	All of the above
7.What are the other important errors that occurs after RCT?	Post space perforation	Tissue emphysema	Both a&b	None of the above
8.Under filling of root canal is most commonly seen in ?	Over instrumentation	While applying very less pressure on condensing the coronal portion	Both a&b	None of the above
9.What are the causes for Missed canals during RCT?	Grossly decayed tooth	Faulty access cavity design	Improper knowledge of canal morphology	All of the above
10.Furcation	Poor access	While doing	Working length	None of the

gets perforated while doing RCT is most commonly due to?	preparation	cleaning and shaping	determination	above
11.Broken instrument is due to?	Use of deformed files	Excessive pressure to the files	Reuse of files for more patients	All of the above
12.Over filling during obturation is most commonly due to?	May occur with soft materials or debris	Hard materials like gutta percha or silver cones	Both a&b	None of the above
13.Common errors that occur during post space preparation ?	Deviation from normal morphology	Removing excess Guttapercha	Both a&b	None of the above
14.How to prevent missing canals during access preparation?	Check for the canal anatomy	Good preoperative radiographs	Both a&b	None of the above
15.How to prevent instrument separation during RCT?	Avoid reusing the files	Use of NiTi files	Both a&b	None of the above

16.How to prevent perforation of crown or root	Use standard burs in optimal angulation	Proper access cavity design	Both a&b	None of the above
17.How to avoid ledge formation during bio mechanical preparation	Use files in sequence	Inaccurate working length	Both a&b	None of the above
18.How to prevent canal blockage	Use step back technique	Use adequate irrigation	Both a&b	None of the above
19.How to prevent instrument aspiration	Use rubber dam isolation	Use cotton roll isolation	Both a&b	None of the above
20.How to prevent nerve paresthesia	Avoid over instrumentation	Ensure confinement of filling material inside the canal	Both a&b	None of the above

Table 4: Data of correct and wrong responses by both males and females for each question.

No of questions	**Correct responses out of 200 students.**	**Wrong responses out of 200 students.**

	[Both males and females]	**[Both males and females]**
1.Which age group do you belong to?	200	0
2.Mention your educational qualification	200	0
3.Endodontic mishaps are most commonly associated with?	113	87
4.Which is the least common access related error you will face during RCT?	167	33
5.Which is the most common Instrument related error you will face during RCT?	178	22
6.What are the errors which occurs during obturation in RCT?	123	77
7.What are the other important errors that occurs after RCT?	165	35
8.Under filling of root canal is most commonly seen in ?	154	46
9.What are the causes for Missed canals during RCT?	134	66

10.Furcation gets perforated while doing RCT is most commonly due to?	123	77
11.Broken instrument is due to?	112	88
12.Over filling during obturation is most commonly due to?	110	90
13.Common errors that occur during post space preparation ? 14.How to prevent missing canals during access preparation?	125	75
15.How to prevent instrument separation during RCT?	156	44
16.How to prevent perforation of crown or root	178	22
17.How to avoid ledge formation during bio mechanical preparation	111	89
18.How to prevent canal blockage	104	96

19.How to prevent instrument aspiration	173	27
20.How to prevent nerve paresthesia	145	55

Table 5: Criteria for evaluating cognisance of endodontic mishaps among undergraduate students.

Correct responses [Out of 20 questions]	**Awareness level**
15-20	Good
10-15	Average
5-10	Poor

Conclusion

Based on this questionnaire, it can be concluded that students in their internship periodhad greater cognisance about mishaps occurring during endodontic procedures among all undergraduate students.

Corresponding Author

Hemavathy.O.R,

Reader, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha University,Saveetha Institute of Medical and Technical Sciences.

162 Poonamallee High Road,Chennai -600077.Tamil Nadu, India.

Email: hemadocmd@gmail.com

Mobile number: 9626033600

References

1. Neda Haji Hassani, Mahin Bahkshi, Samanch Sahabi. Frequency ofIatrogenic errors through root canal treatment procedure in 1335 Charts of dental patients.Journal of International Oral Health 2015; 7[4]:1-4.
2. Chaurasiya S , Yadav G , Tripathi AM , Dhinsa K. Endodontic Failures and its Management: A Review . Int J Oral Health Med Res 2016;2[5]: 144148 .
3. Alsulaimani RS, Almanei KK, Abbtain RA, Binrabba RS, Ashri NY [2016] The Correlation between Endodontic Mishaps and Single-Visit Treatment in King Saud University. Int J Dent Oral Health 2[9]: doi.
4. Pragati Mirikar, Arvind Shenoy, Goud K Mallikarjun.Nonsurgical management of endodontic mishaps in a case of radix entomolaris.J Conserv Dent | Oct-Dec 2009 | Vol 12 | Issue 4.
5. Mozayeni M, Asnaashari M, Modaresi S. Clinical and radiographic evaluation ofprocedural accidents and errors during root canal therapy. Iran Endod J 2006;1:97 100.
6. Karabucak B, Setzer F. Criteria for the ideal treatment option for failed endodontics:
   Surgical or nonsurgical. Compend Contin Educ Dent 2007;28:304 10.
7. L. I. Grossman, “Endodontic case reports,” Dental Clinics of North America, vol. 18, no. 2, pp. 509–527, 1974.
8. U. Sjögren, D. Figdor, S. Persson, and G. Sundqvist, “Influence of infection at the timeofroot filling on the outcome of endodontic treatment of teeth with apical periodontitis,” International Endodontic Journal, vol. 30, no. 5, pp. 297–306, 1997.
9. N. Imura, E. T. Pinheiro, B. P. F. A. Gomes, A. A. Zaia, C. C. R. Ferraz, and F. J. Souza-Filho, “The outcome of endodontic treatment: a retrospective study of 2000 cases performed by a specialist,” Journal of Endodontics, vol. 33, no. 11, pp. 1278–1282, 2007.
10. M. Lazarski, W. Walker, C. Flores, W. Schindler, and K. Hargreaves, “Epidemiologicalevaluation of the outcomes of non-surgical root canal treatment in a large cohort of insured dental patients,” Journal of Endodontics, vol. 27, no. 12, pp. 791–796, 2001.
11. N. M. Chugal, J. M. Clive, and L. S. Spångberg, “Endodontic infection: some biologic and treatment factors associated with outcome,” Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology, vol. 96, no. 1, pp. 81–90, 2003.

12. L. Z. Strindberg, “The dependence of the results of pulp therapy on certain factors: an analytic study based on radiographic and clinical follow-up examination,” Acta Odontologica Scandinavica, vol. 14, supplement 21, article 1175, 1956.
13. K. Kerekes and L. Tronstad, “Long-term results of endodontic treatment performed with a standardized technique,” Journal of Endodontics, vol. 5, no. 3, pp. 83–90, 1979.
14. U. Sjögren, B. Hägglund, G. Sundqvist, and K. Wing, “Factors affecting the long-termresults of endodontic treatment,” Journal of Endodontics, vol. 16, no. 10, pp. 498–504, 1990.
15. G. Bergenholtz, U. Lekholm, R. Milthon, G. Heden, B. Odesjo, and B. Engstrom, “Retreatment of endodontic fillings,” European Journal of Oral Sciences, vol. 87, no. 3, pp. 217–224, 1979.
16. Brikic A, Gurkan B, Olgac V. Surgical approach to itraogenic complications of endodontic therapy: A report of two cases. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;107:52–3.
17. Hauman CH, Chandler NP, Tond DC. Endodontic implications of maxillary sinus: Areview. Int Endod J. 2002;35:127–4.
18. Nimigeen VR, Nimigen V, Maru N, Andressakis D, Balatsouras DG, Danielidis V, et al. The maxillary sinus and its endodontic implication: A clinical study and review. B-ENT. 2006;2:167–75.
19. Mahalakshmi Nandakumar, Iffat Nasim, Management of perforation: A review. Department of Conservative dentistry, SIMATS, Chennai, J Adv Pharm Edu Res 2017;7[3]:208-211.
20. Prasanna Neelakandan, Nithya Jaganathan, Nabeel Nazar, Ethanopharmalogical approach in Endodontic treatment: A focused review. Department of Conservative dentistry and Endodontics, SIMATS, Chennai, International Journal of Drug Development & Research | October-December 2011 | Vol. 3 | Issue 4 | ISSN 0975-9344.

Dental Caries and its Relation to Diabetes among Out Patient Visiting Private Dental College and Hospitals in Chennai – A Cross Sectional Survey

Savitha P [1], Meignana Arumugham.I [2] , Pradeep.S [3]

Graduate Student [1],Professor [2],Department of Public Health Dentistry

Senior Lecturer [3],Department of Conservative Dentistry and Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Dental caries are common chronic disease conditions that cause pain and disability across all age group. If left untreated, dental caries lead to pain, infection, abscess, tooth loss, and eventually eduntulism. Oral health habits are measures people learn and practice regularly in order to maintain good oral health or prevent oral disease.

Aim: The aim of the study was to assess the dental caries and its relation to diabetes.

Materials and Methods: The samples were composed of 120 subjects of diabetic patients. The data were gathered by pre – tested questionnaire, clinical examination for caries experience using DMFT index. The pre-tested questions were designed to determine the level of dental caries incidence being after diagnosed with diabetes and their frequent dental checkups.

Results: The mean DMFT value for male was 2.2 for age group 35 – 44 and 2.8 for 45 – 54 and 3.3 for 55 – 64 and 3.9 for 65 –74. The mean DMFT value for female was 2.7 for age group 35 – 44 and 3 for 45 – 54 and 3.2 for 55 – 64 and 3 for 65 – 74. The mean DMFT score for male and female increases according to their age group.

Conclusion: The present study confirmed that the presence of dental caries was not significantly elevated in diabetic patients, but it increased with age. And also found that female diabetic patients are more prone to dental caries with increase in age. Furthermore the diabetic patients reqires improvement in their general and oral health behaviour.

Key Words*: DMFT, Diabetic, Caries, Age, Incidence*

Introduction

Oral health conditions are known to affect various aspects of quality of life [1].Diabetes is a common chronic metabolic disorder which affects millions of people. Diabetes is characterised by sustained hyperglycaemia affects multiple organ system together with oral cavity [2]. There are two basic types of diabetes: types I diabetes caused by autoimmune damage to the pancreatic beta cells resulting in failure of insulin deficiency. While type II diabetes occurs as a result of insulin resistance with relative insulin deficiency [3]. The oral manifestations include xerostomia, gingivitis, periodontitis, dental caries and opportunistic infections of tongue and oral mucosa [4].Oral health habits are measures people learn and practice regularly in order to maintain good oral health or prevent oral disease. [5]

Dental caries are common chronic disease conditions that cause pain and disability across all age group. If left untreated, dental caries lead to pain, infection, abscess, tooth loss, and eventually eduntulism[6]. It is important to know that patients with DM are susceptible to other oral conditions, such as periodontal and salivary disorders (dry mouth), which could increase their risk of developing new dental caries.

Increased counts of *Mutans streptococci* and*lactobacilli* are some of the factors implicated to be responsible to predispose diabetics to higher incidence of dental caries [7].The increased risk of dental caries would be related to certain factors such as poor oral hygiene or a lack of blood glucose control [8]. The association between diabetes and dental caries, particularly among adults has gained less attention.

Some studies have demonstrated increased caries incidence in diabetes, while other have shown similar or decreased caries rate [9]. The present study examines whether there is greater incidence of dental caries among diabetic patients.

Materials and Method

The pre- tested interviewer administrated questionnaire based cross sectional survey was conducted among the diabetic patients who attended private dental hospitals. The sample of one hundred and twenty diabetic patients was recruited into study. The diabetic patients included were both type I and type II diabetic patients. The pre tested questions were used to determine the level of dental caries incidence being after diagnosed with diabetes and also their frequent dental checkups. The question also focuses about their oral hygiene activity

The clinical examination included an evaluation of dental caries, by using dental mirror and dental probe under light source. The dental caries was assessed using the decayed missing filled teeth (DMFT) Index according to the criteria and recommendations of the Klein, Palmer, Knutson 1938.

Result

The following datas were obtained and analysed from 64 males and 56 females,

Table 1: shows the demographic distribution of diabetic patients according to age and Gender.

AGE GROUP (YEARS)	MALE		FEMALE		TOTAL	PERCENTAGE
	n	%	n	%	N	%
35 – 44	8	6.7	3	2.5	11	9.2
45 – 54	12	10	17	14.1	29	24.1
55 – 64	23	19.1	21	17.5	44	36.7
65 – 74	21	17.5	15	12.5	36	30
TOTAL	64	53.3	56	46.6	120	100

Table 2: shows the mean value of DMF(T) score among male diabetic patients

Age group	No. of subjects	Mean DMF(T) value < 2.9	Mean DMF(T) value >2.9
35 – 44	8	2.2	-
45 – 54	12	2.8	-
55 – 64	23	-	3.3
65 – 74	21	-	3.9

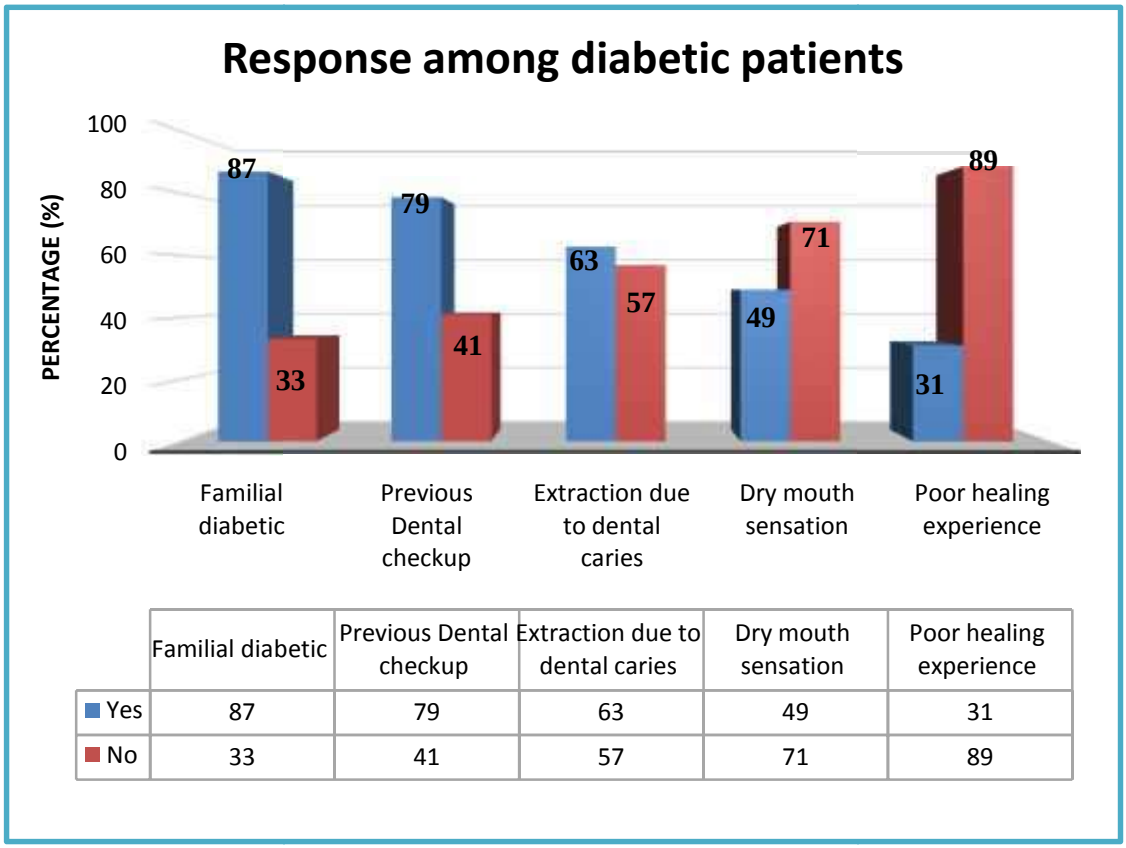
Table: 3 shows the mean value of DMF(T) among female diabetic patients.

Age group	No. Of subjects	Mean DMF(T) value < 2.9	Mean DMF(T) value >2.9
35 – 44	3	2.8	-
45 – 54	17	-	3
55 – 64	21	-	3.2
65 – 74	15	-	3.6

Table :4 shows the oral health behaviour and medical characteristic for diabetic patients.

Question	No. of responses	Percentage (%)
Tooth brushing frequency		
Once in a day	87	72.5
Twice a day	24	20
More than twice	9	7.5
Oral cleaning method		
Tooth brush and paste	87	72.5
Tooth brush and powder	21	17.5
Other (finger, charcoal powder)	12	10
Disease duration		
< 5 years	18	15
5 – 10 years	72	60
> 10 years	30	25
Regular dental visit		
6 month once	47	39.1
1 year once	54	45
More frequently	19	15

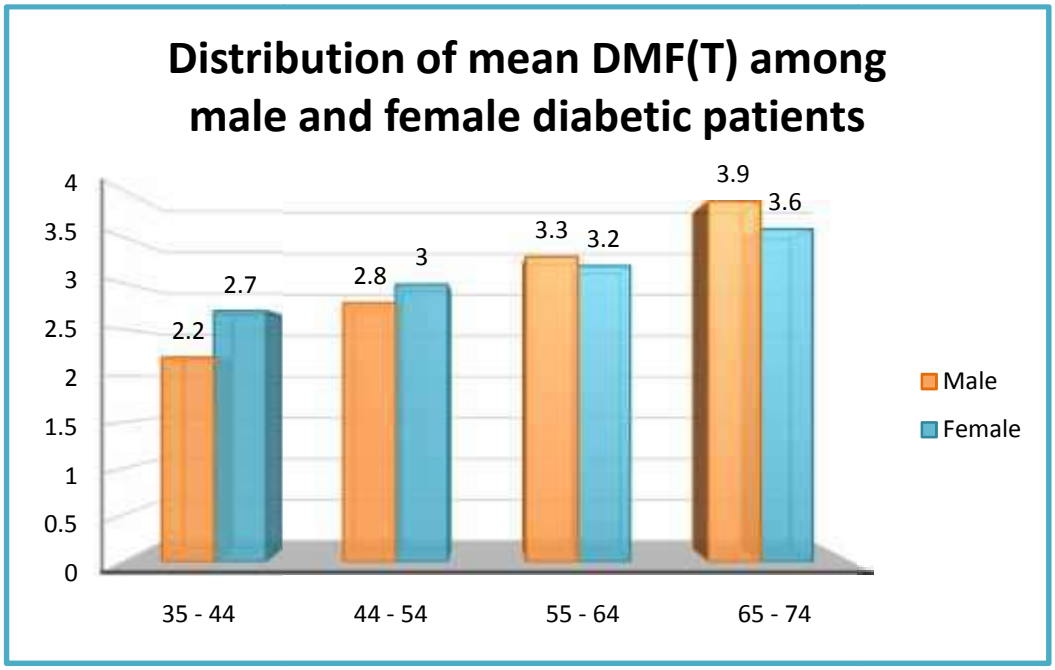
Graph 1: Responses among diabetic patients



	Familial diabetic	Previous Dental checkup	Extraction due to dental caries	Dry mouth sensation	Poor healing experience
Yes	87	79	63	49	31
No	33	41	57	71	89

Graph 1 shows the responses among diabetic patients about their previous dental check-up, known familial diabetic, extraction due to dental caries, experience in poor healing of tissues and dry mouth sensation.63 subject experienced extraction due to dental caries after being diagnosed with diabetes and 31 subject experienced poor healing of oral tissues. 49 subject experienced dry mouth sensations. 79 subjects have gone for regular dental check up after being with diabetes. 87 subjects have been known familial diabete

Graph 2: shows the distribution of mean DMFT among male and female diabetic patients



Graph 2 shows the distribution of mean DMFT among the male and female diabetic patients.The mean DMFT value for male was 2.2 for age group 35 – 44 and 2.8 for 45 – 54 and 3.3 for 55 – 64 and 3.9 for 65 –74. The mean DMFT value for female was 2.7 for age group 35 – 44 and 3 for 45– 54 and 3.2 for 55 – 64 and 3 for 65 – 74. The mean DMFT score for male and female increases according to their age group.

Discussion

Dental caries is caused by demineralization that is triggered by the accumulation of microbial plaque flora, decrease in the flow rate of saliva causing reduction in the cleansing, and buffer activity and diminished levels of calcium that is essential for the repair of decayed tooth [10]. Dental caries is the most prevalent infectious disease in humans, that commonly affects people of all ages throughout their lifetime. Diabetes is a chronic disease that may impact on personal behaviour. Increasing prevalence of diabetes in the population has made it an important public health issue. The prevalence of dental caries and its burden on the general population is of significant public health interest since dental caries is the most common infectious disease known to man [11].Low fluoride exposure, increase frequency of taking sugary food and soft drinks and observed xerostomia were the prominent reasons being

reported behind this increased risk for caries [12]. According to World Health Organization (WHO), DM has been increasing worldwide so abruptly that it is declared as an epidemic [13].

The present study found that the demographic distribution of study subjects in which female 56 study subjects were more prone to dental caries when comparing to 64 male study subjects due to their oral health activity.

The study found that the mean DMFT values were found to be greater among female study subjects when comparing to male study subjects which is greater than 2.9 was found in 45 – 54, 55 – 64, 65 – 74 age groupand most of the study subjects were known diabetic for 5 – 10 years.

The result of the oral health behaviour questionnaire showed better oral health habits regarding brushing frequency among the diabetic patients and frequency of the tooth brushing, this result agreed with other study that was showed in Almas K et al in (2001),[14] while the other result showed the diabetics were somewhat less likely to visit their dentists for routine examination Pohjamo L et al (1995),[15].

The result of the distribution of the dental caries for both groups according to the age showed the mean values of DMFT Index were increased when the age increased with strong correlation this result agreed with Moore PA et al. 2001[16], reported high caries prevalence among older diabetics patients. And also found that female diabetic patients are more prone to dental caries with increase in age.

People with diabetes appear to have contributing factors that results in increased risk for dental caries [17]. In addition, people with diabetes has a high chances of developing abscess from dental caries. People are less informed the risk for dental diseases in comparison with their knowledge of their increased risk for systemic diseases in DM. Thus, it is necessary for dental professionals and related government medical agencies to promote awareness of the relationship between DM and oral heath to prevent harmful complications[18]. Studies have found that patients with diabetes are more susceptible to increase their risk for developing new and recurrent dental caries [19].

Conclusion

The present study confirmed that the presence of dental caries was not significantly elevated in diabetic patients, but it increased with age. And also found that female diabetic patients are more prone to dental caries with increase in age.Furthermore the diabetic patients reqires improvement in their general and oral health behaviour.

Corresponding Author

S. Pradeep

Senior Lecturer,Department of Conservative Dentistry and Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, PoonamalleHigh Road,Chennai 600077,Tamil Nadu, India.

Telephone Number: +91 9710404482

Email: pandu.pradeep@gmail.com

References

1. Ganesh R, John J. A correlation between dental caries and dental impact on daily living: A cross sectional study. Indian Journal of Oral Sciences. 2013;4(2):70-74.
2. Kumar V, Abbas AK, Fausto N, Aster JC. Robbins and Cotran Pathologic Basis of Disease, Professional Edition E-Book. Elsevier Health Sciences; 2014.
3. Garranza FA. Influence of systemic disease on the periodontium. Glickmans clinical periodontology, 8th edition, 1996 .
4. Reddy CV, Maurya M. A comparative study to assess the oral health status and treatment needs of diabetics and non-diabetic population attending some of the hospitals in Mysore City. Journal of Indian Association of Public Health Dentistry. 2008;6(12):1-14.

5. Singh A, Thomas S, Dagli RJ, Kaţ R, Solanki J, Bhateja GA. To access the effects of salivary factors on dental caries among diabetic patients and non diabetic patients in Jodhpur City. J Adv Oral Res. 2014;5:10–4.
6. R.Pradeep Kumar,Shristi Nadar. Oral Health Knowledge, Attitude, and Practice of Patients Visiting a Private Hospital in Chennai. IOSR Journal Of Dental and Medical Sciences 2015;14(6):12-15.
7. Greenberg M.S. and Glick M. Oral Medicine: Diagnosis and Treatment, 10th ed. Harcourt Private Limited: New Delhi, 563 (2003)
8. Khovidhunkit S.P., Suwantuntula T., Thaweboon S., Mitrirattanakul S., Chomkhakhai U., Khovidhunkit W., Xerostomia, hyposalivation, and oral microbiota in type 2 diabetic patients: a preliminary study, J Med Assoc Thai 2009; 92(9):1220-1228.
9. Newman M.G., Takei H., Carranza F.A. and Klokkevold P.R., Clinical Periodontology, 10th ed. Philadelphia: Saunders Elsevier, 113-131 (2003).
10. Jones R.B., McCallum R.M., Kay E.J., Kirkin V. and McDonald F., Oral health and oral health behaviour in a population of diabetic clinic attenders, community Dent Oral Epidemiol.1992, (20), 204-207.
11. Caufield PW, Griffen AL. Dental caries. An infectious and transmissible disease. Pediatrclin north Am. 2000 Oct; 47(5): 1001-19,v.
12. Bots CP, Brand HS, Veerman EC, Amerongen BV, Amerongen AV. Preferences and saliva stimulation of eight different chewing gums. International dental journal. 2004 Jun 1;54(3):143-8.
13. Smyth S, Heron A. Diabetes and obesity: the twin epidemics. Nature medicine. 2006 Jan 1;12(1):75-80.
14. Almas K., Al-Qahtani M. The relationship between periodontal disease and blood glucose level among type II diabetic patients. The Journal of Contemporary Dental Practice 2001; 2(4): 1-6.
15. Pohjamo L, Tervonen T, Knuuttila M, Nukkala H. Adult diabetic and non-diabetic subjects as users of dental services: A longitudinal study. ActaOdontolScand 1995; 53:112-114.
16. Moore PA, Weyant RJ, Etzel KR, Guggenheimer J, Mongelluzzo MB, Myers DE, Rossie K, Hubar H, Block HM, Orchard T. Type 1 diabetes mellitus and oral health: Assessment of coronal and root caries. Community Dent Oral Epidemio 2001; 29:183-194.

17. Eldarrat AH. Awareness and attitude of diabetic patients about their increased risk for oral diseases. Oral health & preventive dentistry. 2011 Jun 1;9.
18. Kumaresan GD, Subha M. Awareness on oral diseases in diabetes mellitus among general population in chennai. Int J Orofac Res 2017;2:28-31
19. Selwitz RH, Ismail AI, Pitts NB. Dental caries. The Lancet. 2007 Jan 12;369(9555):51-9.

Dental Caries Related Quality of Life and Socioeconomic Status of Adult Population In Perambur, Chennai

S. Rohini[1], Nabeel Nazar[2]
Graduate Student[1], Reader[2], Department of Oral and Maxillofacial Surgery
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Oral health has a major role in the general well-being of a person. Oral health problems can have wider social, economic and psychological concerns thereby affecting the quality of life. Oral Health Related Quality of Life indicates an individual's perception of how their well being and quality of life is influenced by oral health. The aim of the study is to o determine the oral health related quality of life and the association between dental caries socioeconomic status of adult population in perambur, Chennai.

Material and Methods: The study sample consists of 200 adults and a questionnaire comprised of 10 questions related to oral habits and Kuppuswamy scale (modified for year 2015) to evaluate oral health related quality of life & socioeconomic status of adults in Chennai.

Result: The oral health of an individual and their socioeconomic status was analyzed and found that upper middle class females were commonly affected with dental caries than other class females. The most commonly affected tooth with dental caries was upper right 1stmolar followed by lower left and right 1st molar.

Conclusion: This study showed that there is a strong relationship between the socioeconomic status and oral health. The main purpose of this study is to highlight the importance of assessing oral health related quality of life in the clinical practice so that the clinicians can easily identify the actual difficulties faced by the patients due to oral problems and formulate appropriate treatment plan and goals

Key Words: *Dental caries, Kuppuswamy scale, Oral health, Quality of life,socioeconomic status.*

Introduction

Dental caries is a multifactorial infectious diseasethoroughout the world leading to pain, chewing difficulties, speech problems, general health disorders, psychological problems, and lower quality of life. The prevalence and incidence of dental caries in a population is influenced by a number of risk factor such as sex, age, socioeconomic status, dietary patterns, oral hygiene habits, salivary immunoglobulins, bacterial load and fluoride intake. Dietplays an important role in the development of dental caries. Many previous studies showed that the consumption of foods with high carbohydrate content constitutes an important risk factor for the development of dental caries. Frequency of sugar intake increases the risk for caries development.[1]

Oral health is a state in which the oral tissues contribute positively to an individual's wellbeing in all dimensions of health allowing them to eat, speak and socialise with others without any discomfort. Thus oral health has an important role in the general wellbeing of a person.[2]Oral health problems have been increasingly recognised as important factors causing a negative impact on daily performance & quality of life.

Oral health problems have wider social, economic& psychological concerns thereby affecting the quality of life. Oral health related quality of life denotes a person's perception/concern of how oral health influences life quality and overall wellbeing. It comprises of individual's satisfaction with oral health, self-esteem and the ability to eat, speak & engage in social interactions.

Socioeconomic status (SES) refers to the placement of persons, families, households and census tracts or other aggregates with respect to the capacity to create or consume goods that are valued in our society.[3]Indicators of socioeconomic status (SES), such as household income or education level, are important factors that have an impact on oral health. Several reports have shown that low socioeconomic status groups exhibited worse oral health and a higher prevalenceof caries compared with high socioeconomic status groups. To reduce these

socioeconomic inequalities, many countries have endeavoured to improve the oral health of their citizens through health promotion programs, such as public health campaigns promoting brushing with fluoride toothpaste, school programs of rinsing with fluoride solution, and water fluoridation.

Low socioeconomic status, low monthly household income and low educational level are associated with less access to dental services and oral hygiene products, poorer knowledge regarding oral health and oral hygiene and, consequently, a greater frequency and severity of dental caries

Problems like tooth decay, pulpitis and periodontitis causes pain. Loss of tooth structure can interfere with proper chewing, speaking & aesthetic appearance. Craniofacial disorders, malocclusion, appearance of teeth or denture significantly affect self-esteem, social relationships and communication capability. [4]Hence these problems cause considerable distress to individuals and affecting their quality of life. The association between social & economic status and dental caries prevalence has been evaluated in this study using indices among adult population in perambur alone.

Materials and Methods

The study sample consists of 200 adults and the DMFT (decayed, missing due to caries, filled teeth) index was adopted for assessment of dental caries in accordance with the standards recommended by the world health organisation (WHO).

A questionnaire comprised of 10 questions related to oral habits and Kuppuswamy scale (modified for year 2015) to evaluate oral health related quality of life & socioeconomic status of adults in Chennai. The data was analysed and results were tabulated.

Results

The study was conducted among 200 adults, in which females were dominant than males and in the age group of 34-44 years. Majority of them were graduate and post graduate. From the study it was observed that majority of the adults cleaned their teeth with tooth brush and paste once daily and visited dental office in 6 months for regular dental check up. Most of them also had previous experience of tooth ache due to dental caries and difficulty in consuming hot and cold food items. The oral health of an individual and their socioeconomic status was

analyzed and found that upper middle class females were commonly affected with dental caries than other class females. The most commonly affected tooth with dental caries was upper right 1stmolar followed by lower left and right 1 st molar.

The relationship between the prevalence of dental caries and occupation of the adults were assessed. The unemployed adults were commonly affected with dental caries than any other profession. Thus occupation and socioeconomic status of the subjects had a greater impact on prevalence of dental caries.

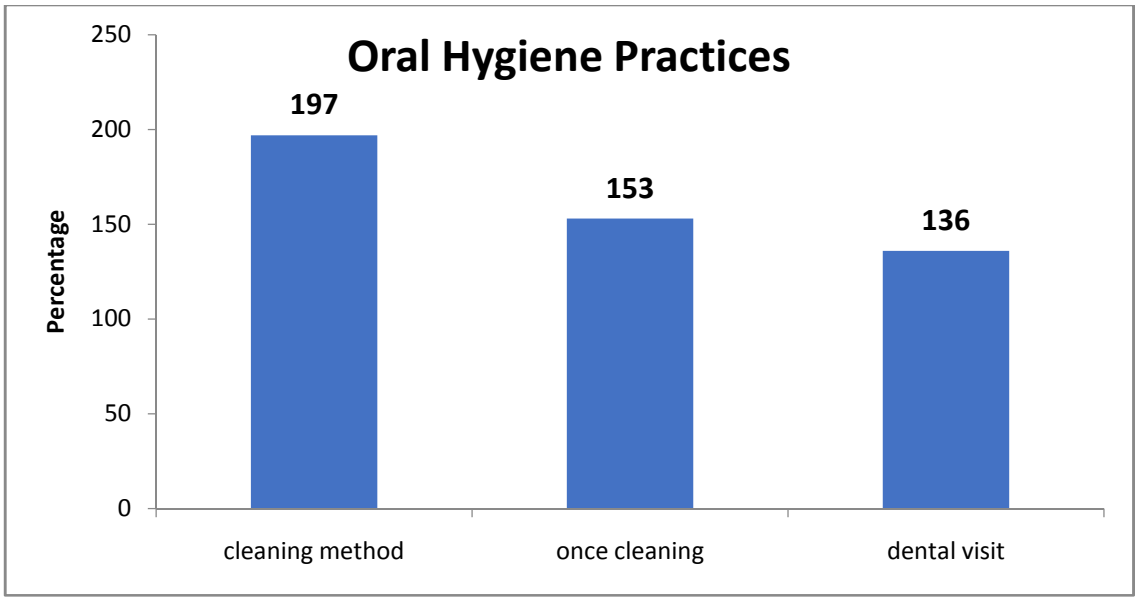


Figure: 1 It shows that majority of the adults cleans their teeth with tooth brush and paste once daily and visits dental office once in 6 months.

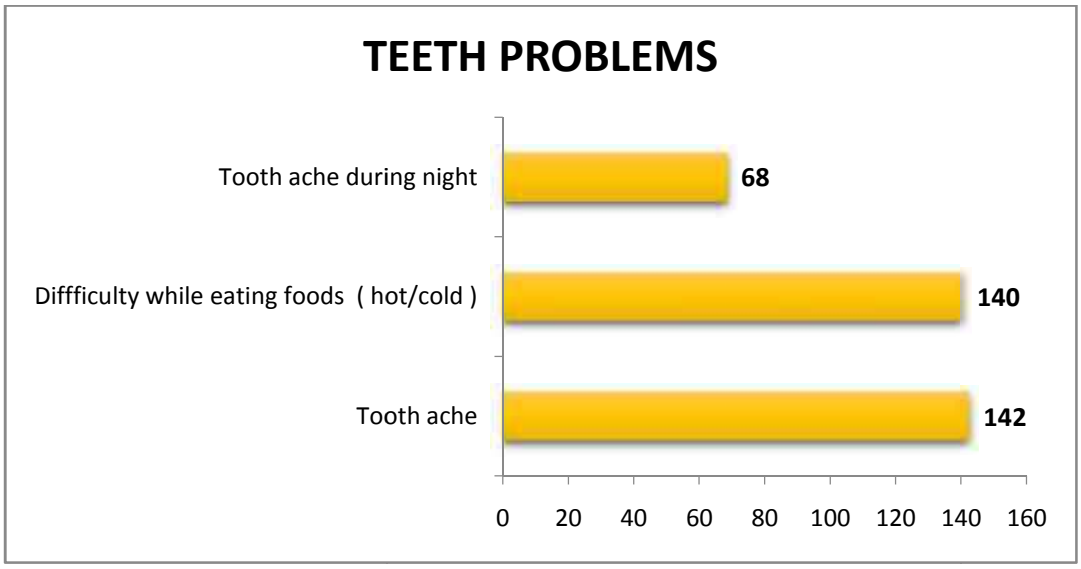


Figure: 2 It shows that majority of adults had previously experienced tooth ache due to dental caries and had difficulty while eating hot or cold foods and tooth ache during night.

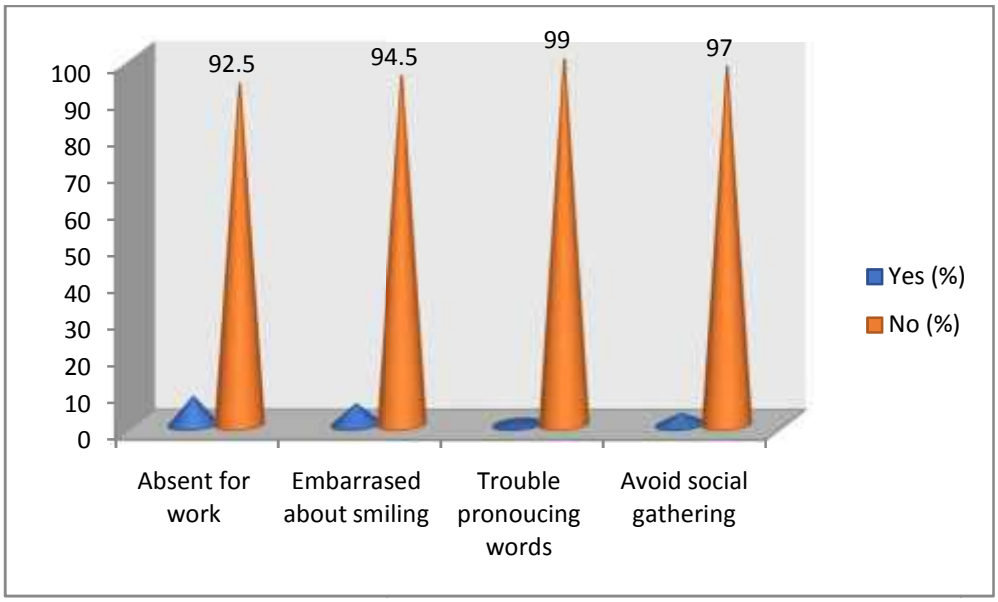


Figure: 3 Oral health related quality of life

Fig: 3 It shows that oral health of the adults and their socioeconomic status didn't have any significant effect on their quality of life like trouble in pronouncing words (99%), avoidance of social gathering (97%), Embarrassed about smiling (94.5%), and absent for work (92.5%).

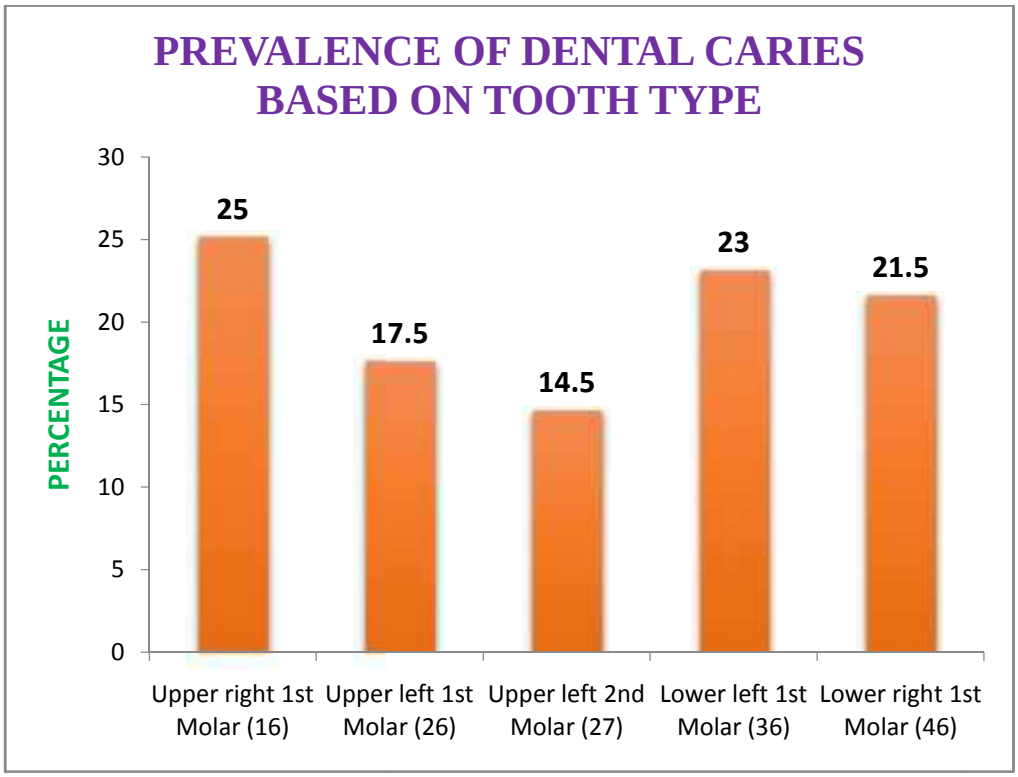


Figure: 4 It shows that upper right 1st molar (25%) was most commonly affected by dental caries, followed by lower left 1st molar (23%) and lower right 1st molar (21.5%).

Table: 1 Demographic Distribution Of Subjects

AGE GROUP (yrs)	FEMALE		MALE		TOTAL	
	n	%	n	%	n	%
<25	17	8.5	13	6.5	30	15
25-34	30	15	17	8.5	47	23.5
35-44	27	13.5	27	13.5	54	27
45-54	17	8.5	22	11	39	19.5
55-64	8	4	9	4.5	17	8.5
65-74	3	1.5	8	4	11	5.5
75-84	1	0.5	1	0.5	2	1
TOTAL	103	51.5	97	48.6	200	100

Table: 1 It shows the demographic status of the adults and majority of adults were between the age group of 35-44 years. Females were dominant than males in this study.

Table: 2 Education Statuses Of Subjects

EDUCATION	MALE		FE3MALE		TOTAL	
	n	%	n	%	n	%
Graduate/ post graduate	38	19	60	30	98	49
High school	22	11	20	10	42	21
Illiterate	4	2	5	2.5	9	4.5
Middle school	11	5.5	7	3.5	18	9.0
Post high school	15	7.5	5	2.5	20	10
Primary school	1	0.5	1	0.5	2	1
profession	6	3	5	2.5	11	5.5
TOTAL	97	48.5	103	51.5	200	100

Table: 2 It shows education status of the subjects and majority of them were graduate/ post graduate (49%), followed by high school (21%)

Table: 3 Mean Value Of Dmft Score And Occupation Of Subjects

OCCUPATION	<2.9		>2.9		TOTAL	
	n	%	n	%	n	%
Clerical/shop owner	17	8.5	6	3	23	11.5
Profession	12	6	25	12.5	37	18.5
Semi profession	13	6.5	12	6	25	12.5
Semi-skilled worker	18	9	11	5.5	29	14.5
Skilled worker	17	8.5	21	10.5	38	19
Unemployed	22	1	22	11	44	22

Unskilled worker	4	2	0	0	4	2
TOTAL	103	51.5	97	48	200	100

Table: 3 It shows the relationship between the prevalence of dental caries and occupation of subjects. Unemployed adults (22%) were commonly affected by dental caries than any other profession.

Table: 4 Socioeconomic Statuses And Mean Value Of Dmft Score

SOCIOECONOMIC STATUS	<2.9		>2.9		TOTAL	
	n	%	n	%	n	%
Upper (I)	5	2.5	2	1	7	3.5
Upper middle (II)	37	18.5	41	20.5	78	39
Lower middle (III)	27	13.5	22	11	49	24.5
Upper lower (IV)	28	14	29	14.5	57	28.5
Lower (V)	6	3	3	1.5	9	4.5
TOTAL	103	51.5	97	48.5	200	100

Table: 4 It shows the relationship between the socioeconomic status and prevalence of dental caries. It is shown that upper middle (II) class (39%) adults were more commonly affected by dental caries than other classes.

Table: 5 Mean Value Of Dmft Score Among Gender

GENDER	<2.9		>2.9		TOTAL	
	n	%	n	%	n	%
Male	51	25.5	52	26	103	51.5
Female	52	26	45	22.5	97	48.5
TOTAL	103	51.5	97	48.5	200	100

This table shows that females (51.5%) were more commonly affected by dental caries than males (48.5%).

Discussion

Oral health and the lifestyle of an individual influences the oral health related quality of life. To evaluate the prevalence and severity of dental caries according to WHO recommendations, this study used DMFT score. The study included 200 adults from perambur region in Chennai, among which 103 were females and 97 were males between the age group of 18 to 85 years. In the present study, it was observed that females (51.5%) were more commonly affected by dental caries than males (48.5%) (Table: 5) This result was similar to the studies done by Lawrence et al [5]& Navin ingle.[6]

Occupation of an individual also has an influence on the prevalence of dental caries. In this study unemployment (22%) was associated with higher incidence of dental caries than other occupations like clerical/shop owner, profession, semi profession, etc. (table: 3) The second most common occupation associated with incidence of dental caries was skilled workers (19%).[7]

The association between oral health & socioeconomic status of adults has been well established in this study using Kuppuswamy scale. The upper middle class (II) was found to be more prone to dental caries (39%) than other classes. (Table: 4) This high rate of dental caries is due to their rich lifestyle. This result was different from the study done by Jain R et al[8]& Deva Priya Appukuttan et al in 2016,[8,9]which showed more prevalence of dental caries in lower socioeconomic status. The prevalence of dental caries was high in the low socioeconomic status because of their poor oral hygiene practice, lack of awareness, improper food intake and family status.[10]According to study done Evans et al.,[11] low family income may affect food selection and nutrient intake by mothers and also infants during the tooth development period. It may also affect the degree of education, health values, life-style and access to health care information. As a consequence, family income can be an indirect factor for tooth susceptibility to caries.[12]It is also possible that mothers completing higher levels of education are more responsible regarding health,more likely to maintain good dietary and hygiene behaviours, and are likely to have more positive health attitudes. Moreover, the widespread belief that primary teeth are temporary and not as important as permanent teeth is likely to be higher among mothers with low levels of education.[13]The

recognition that maternal education is a strong determinant of childhood caries confirms that oral health cannot be achieved without educational policies in developing countries. Such programmes may have an impact on the health of infants and preschoolers, including dental caries.

The prevalence of dental caries based on tooth type was also evaluated in this present study. From the study, it was observed that the upper right 1st molar (16) was more commonly affected by dental caries (25%) than other teeth. (Fig: 4) [14]In general terms, the canines and incisors demonstrated fewer dental caries. From the anatomical point of view, such occurrences have been attributed to the anatomical oral position of the teeth, making them accessible to fluoride exposure and hygienic habits. Caries is much more frequent in sites of food retention and bacteria accumulation. Forty to fifty percent of the cavities are normally found on the grooves and cracks of occlusal molar surfaces.[15]

In a study by Li et al.[16]showed that the permanent teeth, particularly the first and second molars are more susceptible to cracks formation, making the adhesion and colonization of cariogenic microorganisms possible. As a result, the dental caries on the occlusal surface are higher than on other surfaces.[17]Naturally, any alteration in oral habits such as oral hygiene, topical fluoride exposure and changes in dietary habits will influence the cariogenic indices in any of the socioeconomic population categories.[18,19]

Oral hygiene practices, teeth problems & oral health related quality of life were also evaluated in this study and found that majority of adults cleaned their teeth with tooth brush (98.5%), once daily (76.5%) and visited dental office once in 6 months (68%). (Fig: 1)[20,21] Among teeth problems, tooth ache (71%) was most commonly experienced by all adults but tooth ache during night (34%) was least experienced by them. It was also found that oral health had least effect on oral health related quality of life.(Fig: 2)[22]Individuals didn't face any trouble in pronouncing words, embarrass about smiling nor avoid any social & peer gathering because of their oral health problems. (Fig: 3) [23]Adults who visit dentist regularly have better oral health status and better oral health related quality of life, requiring less emergency treatment.[24]

Conclusion

This study showed that there is a strong relationship between the socioeconomic status and oral health. The main purpose of this study is to highlight the importance of assessing oral health related quality of life in the clinical practice so that the clinicians can easily identify

the actual difficulties faced by the patients due to oral problems and formulate appropriate treatment plan and goals. [25]

Corresponding Author

Nabeel Nazar

Reader, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email: nabeel_nazar@yahoo.com

Telephone Number: 9025961929

References

1. Petersen P.E. The world oral health report 2003: Continuous improvement of oral health in the 21st century – the approach of the WHO global oral health programme. Comm Dent Oral Epidemiol. 2003; 31:3-24.
2. Meignana Arumugham, Joseph John. Socio-economic status, construct and its relation to health – Indian scenario, J Oral Heal Res; 2011; 3:2.
3. Locker D. Oral health and quality of life. Oral Heal Prev Dent. 2004; 2:247-53.
4. Gift HC, Atchison KA. Oral health and health-related quality of life. Med Care. 1995; 1:NS57-77.
5. Lawrence HP, Thomson WM, Broadbent JM, Poulton R. Oral health-related quality of life in a birth cohort of 32-year olds. Comm Dent Oral Epidemiol. 2008; 36: 305–316.
6. Ingle NA, Chaly PE, Zohara CK.Oral Health related quality of life in adult population attending the outpatient department of a hospital in Chennai, India. Jour IntOral Health. 2010; 2: 45-52.
7. Wamala S, Merlo J, G. Inequity in access to dental care services explains current socioeconomic disparities in oral health: The Swedish National Surveys of Public Health 2004–2005. J Epidemiol Comm Health. 2006, 60:1027–33.
8. Sabbah W, Tsakos G, Chandola T, Sheiham A, Watt RG. Social gradients in oral and general health. J Dent Res. 2007; 86; 992–96.

9. R.A Devishree et al. The role of social economic status on dental caries and its prevention among outpatients visiting private Dental college hospital. Int J. pharm. Sci & Res. Vol 10(2), 2018, 369-371.
10. ChanchalKatariya, Ganesh Jeevanandan. Awareness of dental health among children with different socioeconomic status – A questionnaire. Int. J. Pharma Bio Sci 2018 April; 9(2): (p) 166-172.
11. Evans RW, Lo ECM, Darvell BW. Determinants of variation in dental caries experience in primary teeth of Hong Kong children 6-8 years. Commun Dent Oral Epidemiol. 1993; 21:1-3.
12. Weinstein P. Public health issues in early childhood caries. Community Dent Oral Epidemiol 1998; 26 (Suppl.1): 84 – 90.
13. Ismail AI. Determinants of health in children and the problem of early childhood caries. Pediatr Dent 2003; 25: 328 – 333.
14. Riedy CA, Weinstein P, Milgrom P, Bruss M. An ethnographic study for understanding children's oral health in a multicultural community. Int Dent J 2001; 51: 305 – 312.
15. Ismail AI. Prevention of early childhood caries. Com- munity Dent Oral Epidemiol 1998; 26 (Suppl. 1): 49–61.
16. Li Y, Navia JM, Bian JY. Caries experience in deciduous dentition of rural chinese children 3-5 years old in relation to the presenceor absence of enamel hypoplasia. Caries Res. 1996; 30:8-15.
17. Jain R, Dupare R, Chitguppi R, Basavaraj P. Assessment of validity and reliability of hindi version of geriatric oral health assessment index (GOHAI) in Indian population. Ind J Public Health. 2015;59:272.
18. D, Sri Sakthi et al. Dental caries experience and treatment needs among construction workers of Chennai city, India. Journal of Oral Health Research, [S.l.], july 2010.
19. R. Pradeep Kumar, Joseph John, S. Saravanan, and I. MeignanaArumugham,Oral health knowledge, attitudes and practices of patients and their attendants visiting college of Dental surgery, Saveetha university, Chennai,The Jour of Ind Med Asso, (13) 2009; 43-53.
20. SusanT.Reisine, Socioeconomic Status and Selected Behavioural Determinants as Risk Factors for Dental Caries., Jour of Dent Edu(65), 2001; 10: 34.

21. Shristi nadar, R. Pradeep Kumar, Oral Health Knowledge, Attitude, and Practice of Patients Visiting a Private Hospital in Chennai.Jour of Dent and MedSciences 14, (6) 2015; PP 12-15.
22. Paulander J, Axelsson P, Lindhe J. Association between level of education and oral health status in 35, 50, 65 and 75 year olds. Jour Clin Periodontol. 2003; 30:697704.
23. Acharya S. Oral health related quality of life and itsassociated factors in an Indian adult population. Oral Heal & Prev Dent 2008; 6: 175-184.
24. Ravi Kumar BP, Shankar Reddy D, Rao AR. Kuppuswamy's Socio-Economic Status Scale. A Revision of Economic Parameter for 2012. Int Jour of Res and Dev of Heal 2013; 1: 2-4. 19.
25. Klein H, Palmer CE, Knutson JW. Studies on dental caries index, dental status and dental needs of elementary school children. Pub Heal Rep. 1988; 53: 751-765.

Dental Experience, Anxiety, and Oral Health in Low-Income Chennai Children

S.Thaminee[1],Ganesh[2],V.Ashok[3],
Graduate Student[1],Reader[2], Department of Pedodontics, Reader[3],Department of Prosthodontics,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: Dental problems are more common in children now a days due to the change in food habits.Various treatment modalities are available including fillings,root canal treatment and crown.The ambience in the dental clinic and the noise created by the compressor,suction apparatus makes the child apprehensive,low income children have more exposure to noises and their fear threshold are more compared with high income children.This study was to evaluate the prevalence of dental anxiety and the factors influencing dental anxietyand poor oral health among the low-income children in Chennai.

Materials and Methods: One hundred and fifty children were examined who reported to Pedodontic Department in Saveetha Dental College,chennai. Oral healthstatus of Male and female patientwere assessed based on the decayed, filled teeth index, simplified oral hygiene index, and Streptococcus mutansscore.The assessment excluded patient had Facial deformities,andmentally retardant,suffering from congenital diseases.Dental anxiety was assessed using the facialimage scale and Frankl scale. Early dentalexperience wasclassifiedas: no previous dental visits; preventive control; restorative treatment; and emergency visit.

Result: Dental caries is the key factor for the child to visit the dental clinic.The dental setup and its factors are one of the triggering agent to child's anxious and the previous dental experiences. Patients with systemic problems have high rate of dental caries.

Conclusion: In low income children, Prior dental experiences of six-year-old children were directly related to their dental caries experience. Children who had preventive visits and those

who had never seen a dentist before had low rates of dental caries.Patients have low percentage of dental visit for their preventive treatment due to unawareness of their parents. Simplified oral hygiene index and dental anxiety levels showed no statistically significant differences among the types of previous dental experiences.

Key Words:*Dental caries,Anxiety,Oral health,Dental experience,Fear,Preventive treatment,Low-Income*

Introduction

Dental caries is the most common chronic disease of childhood and is, therefore, of great importance to public health. Dental caries is the key factor for the child to visit the dental clinic.The dental setup and its factors are one of the triggering agent to child's anxious and the previous dental experiences(17,18).

Patients with systemic problems have high rate of dental caries.Anxiety and fear towards dental treatment are common problems frequently experienced by patients worldwide, hence for better understanding, management and development of treatment strategies for dentally anxious patients,and their dental experience and oral health the present study was undertaken(1).

The study aimed to evaluate the prevalence of dental anxiety and the factors influencing dental anxiety,poor oral health among the Low-income children in Chennaiand is about the anxious and experience of the child in dental clinic and how their anxious makes the treatment complicated and the background of their anxious is dental caries(19).The severity of dental caries measured by the decayed, missing,filling teeth and OHI-(S) index(2).

Materials and Methods

One hundred and fifty children were examined under Indusian criteria both female and Male.A sample of 150 questionnairewere circulated containing 15 questions. Oral healthstatus were assessed based on the decayed, extracted, or filled teeth index, simplified oral hygiene index, and Streptococcus mutans score. Dental anxiety was assessed using the facial imagescale andFrankl scale. Earlydentalexperience was classifiedas: no previous dental visits; preventive control; restorative treatment; and emergency visit(20).

Questionnaire

Dental Experience, Anxiety, and Oral Health In Low-Income Chennai Children

Name: **Sex:Male/Female**

Age:

Socioeconomic Status: A)<25,000/Year ,B)25,000-50,000/Year,

C)50,000-1,00,000/Year

Questions:

1)Does your child had any previous dental visit?

A)Yes

B)No

2)How was the child's experience for the first dental visit?

A)Pleasant

B)Fair

C)Poor

D)Unpleasant

3)Does the child is anxious to any of the dental set up?

A)Yes

B)No

If YES –To what:

4)Does your child had any treatment on his/her first dental visit?

A)Yes

B)No

C)Emergency

5)Does your child has any forward looking to the dentist after their first dental visit?

A)Yes

B)No-Why?

6)How was your child's oral health before visiting a dentist?

A)Excellent

B)Good

C)Fair

D)Poor

7)What's your child's daily frequency of brushing?

A)Once

B)Twice

8)Does your child brushes their teeth on their own?

A)Yes

B)No

9)What kind of snacks Does your child take often?

A)Chocolate/sweets

B)Beverages

C)Ice cream

10)Have your child had any preventive treatment for dental caries?

A)Yes

B)No

11)How was the child's behaviour during the dental procedure?

A)Uncooperative

B)Cooperative

12)Are you aware of the preventive treatment for your child's dental caries?

A)Yes-How?

B)No

13)Does your child has any systemic problems?

A)Malnutrition

B)Communicable disease

C)Infectious disease

D)Congenital disease

E)Learning /Speech difficulties

F)Growth retardation

14)What's the rate of caries in children undergoing restorative and emergency treatment?

A)<2

B)2-5

C)>5

15)Oral health assessment:

A)OHI-S:

B)DMFT/S: Decay- Missing- Filling-

Results

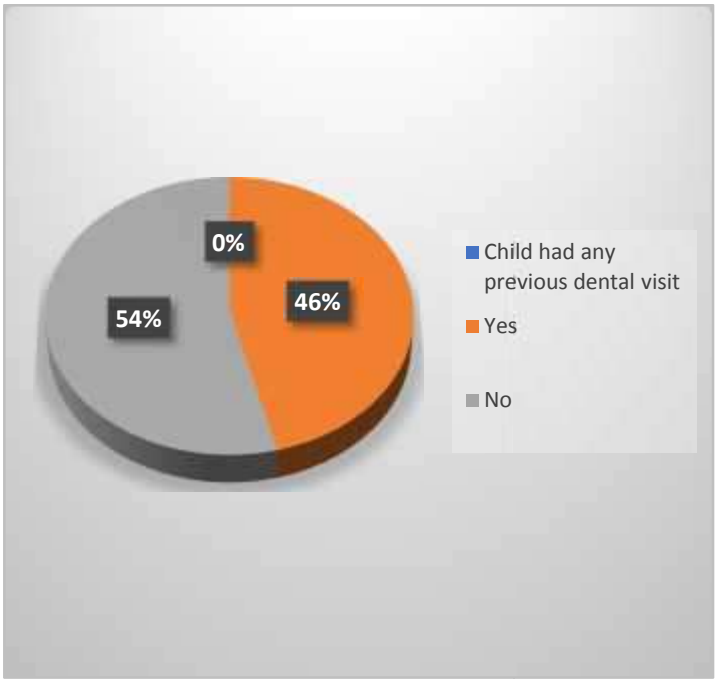


Figure 1: Previous dental visit

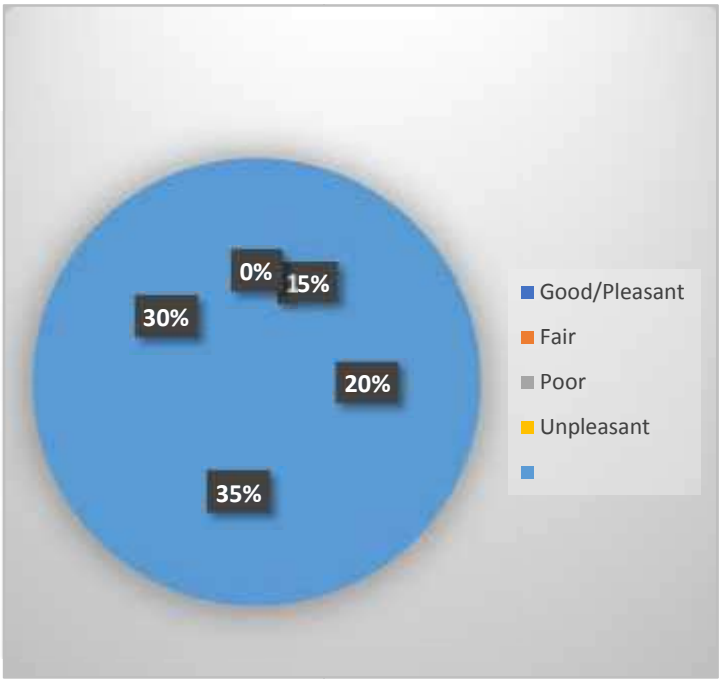


Figure 2: Dental experience of child

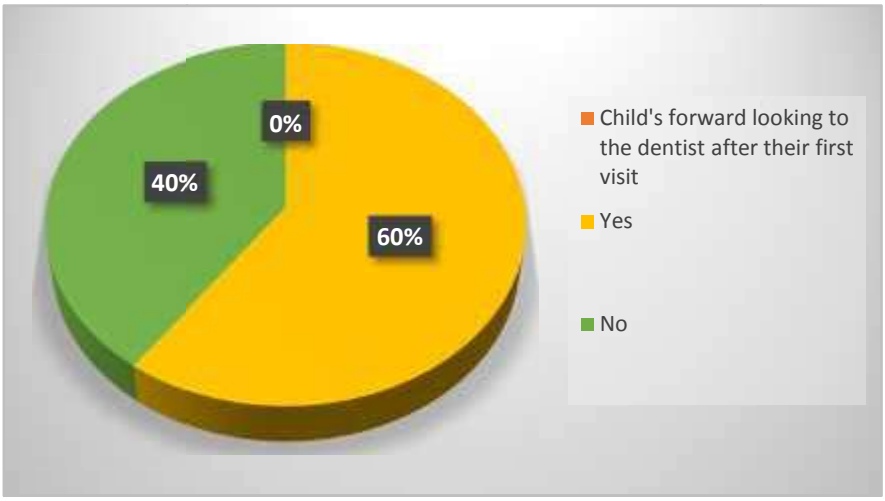


Figure 3: Child's forward looking to the dentist after their visit

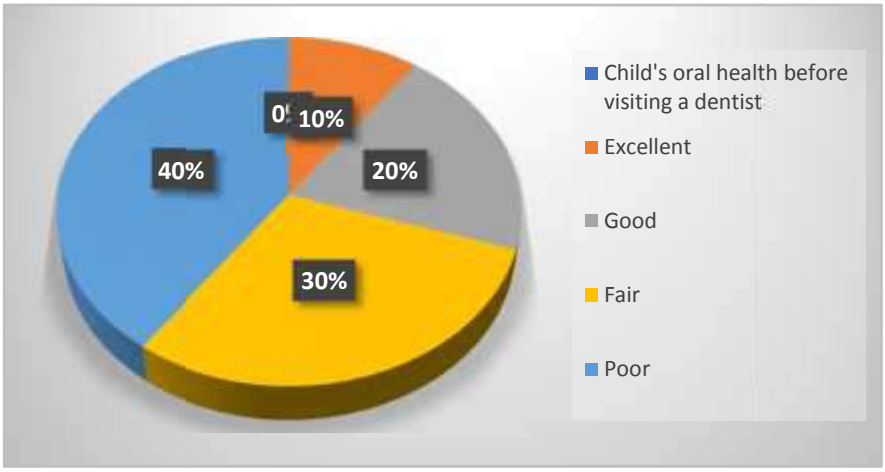


Figure 4: Child's oral health before visiting a dentist

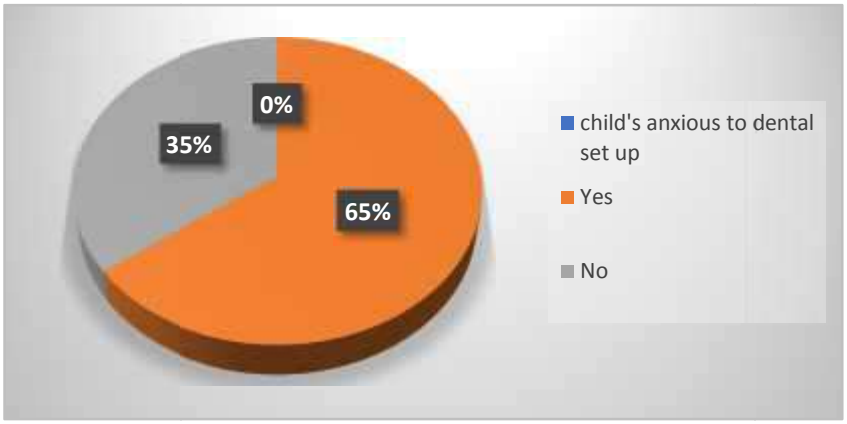


Figure 5: Child's anxious to dental set up

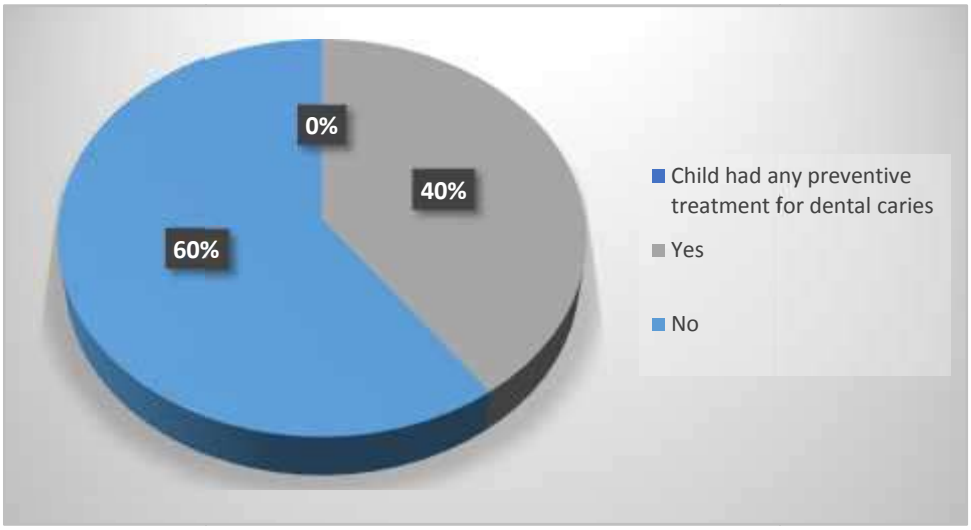


Figure 6: Child had any preventive treatment treatment for dental caries

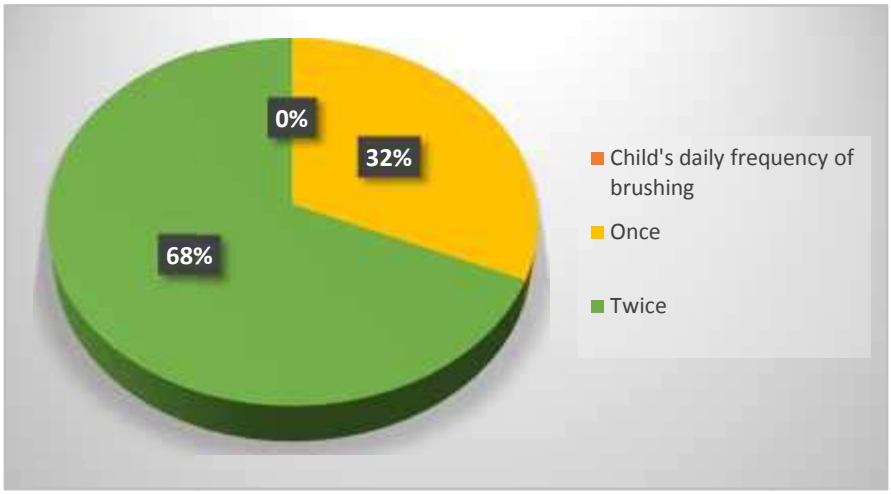


Figure 7: Child's daily frequency of Brushing

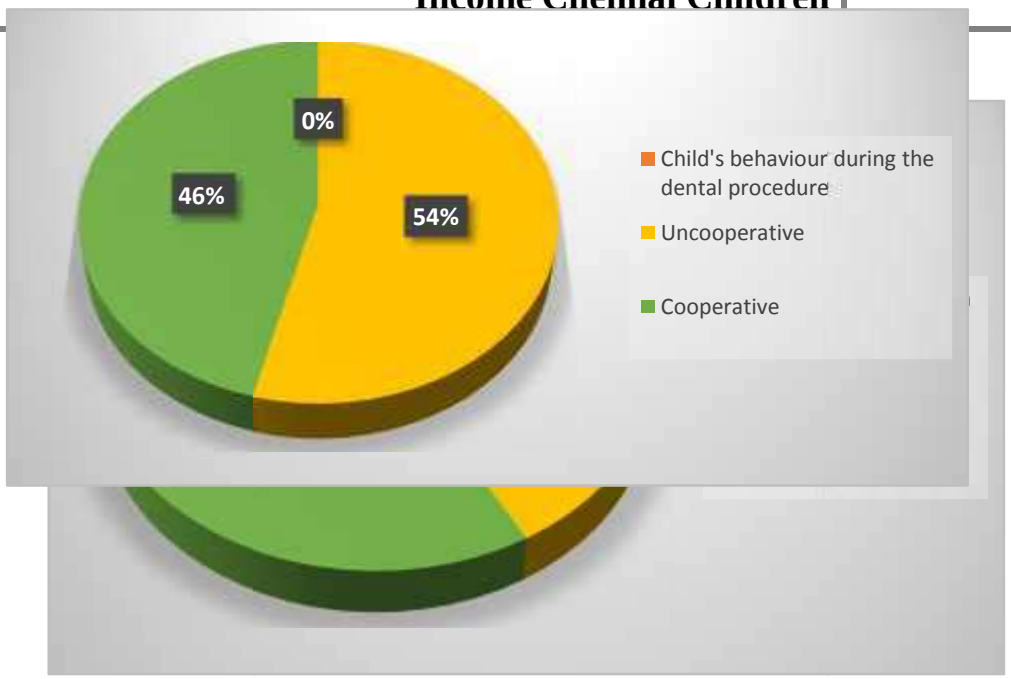


Figure 8: Child brushing habit

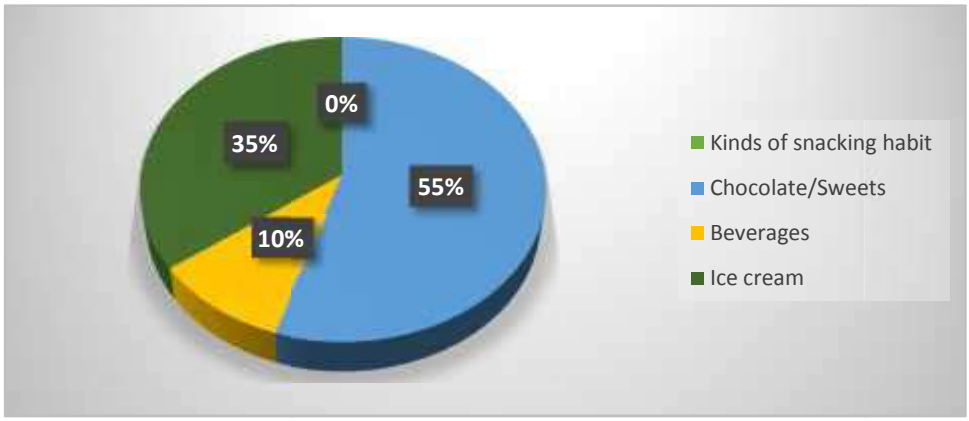


Figure 9: Child's behaviour during the dental procedure

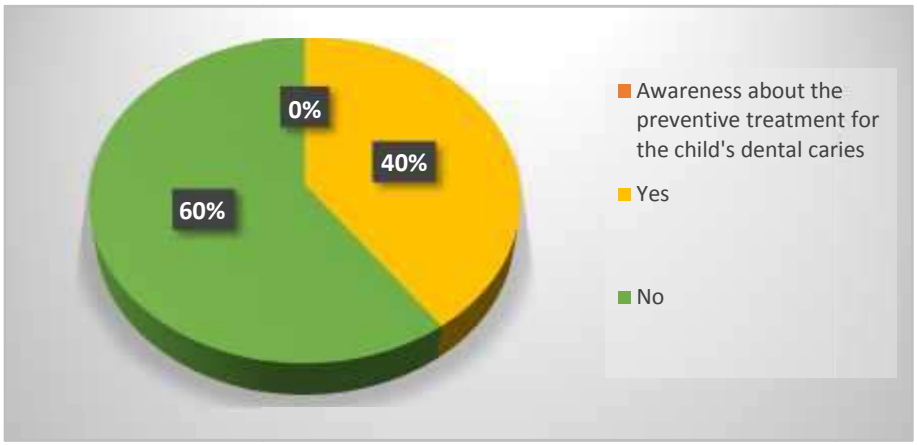


Figure 10: Snacking habit

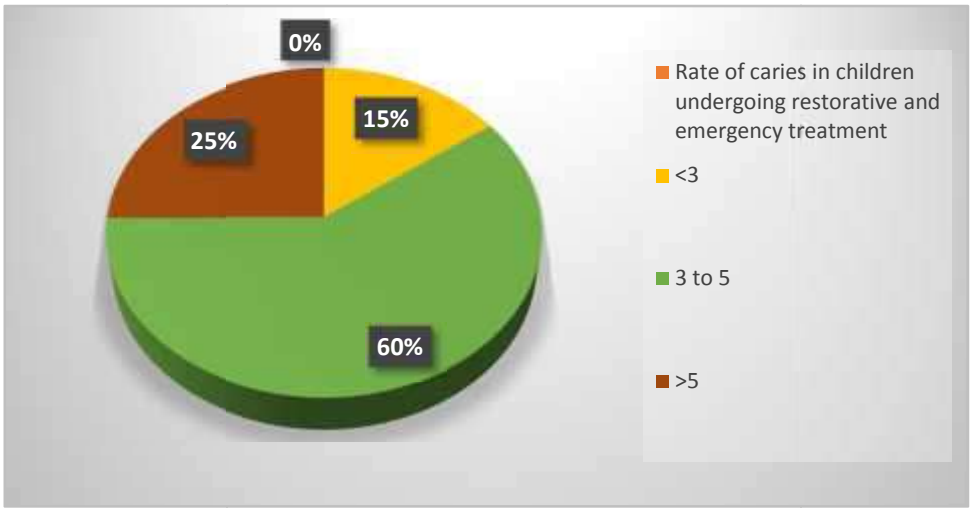


Figure 11: Rate of caries in children undergoing restorative andemergency treatment

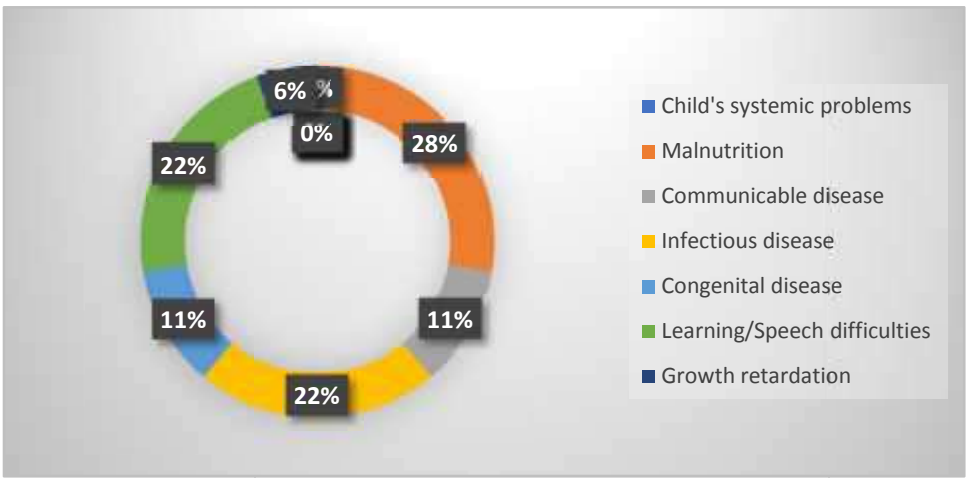


Figure 12: Child's systemic problems

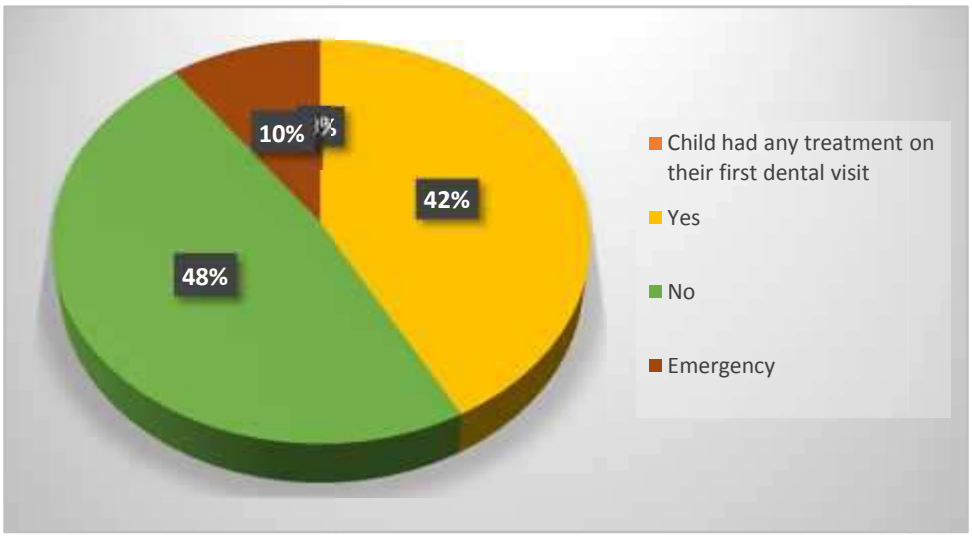


Figure13: Child's treatment on their first dental visit

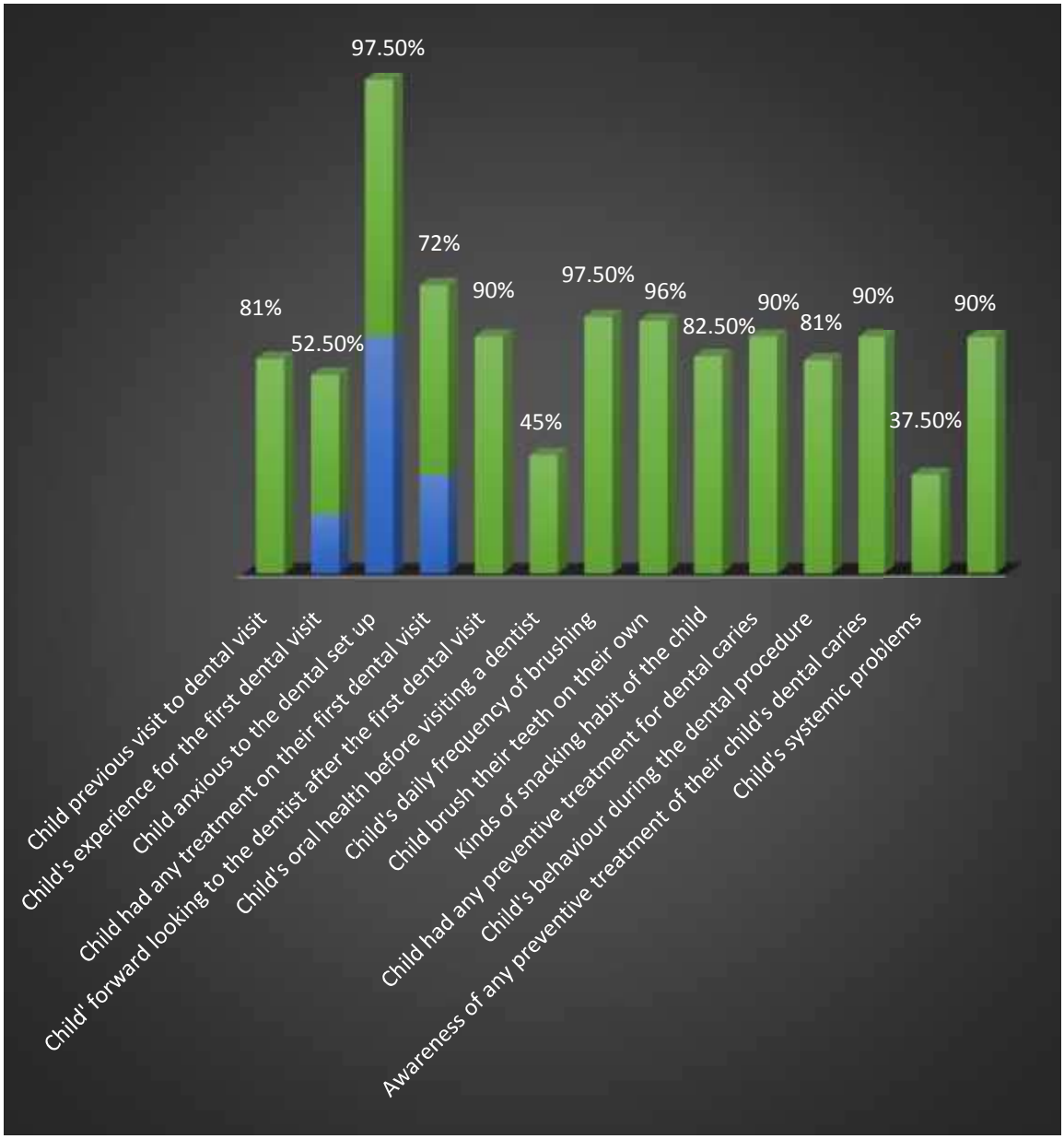


Figure14: Oral health in low-income children

Discussion

This study aimed to assess the relationship between early dental experience and clinical/microbiological indicators of oral health status and dental anxiety at the time of admission to a dental clinic for oral health assessmentof the children. Few children who visited to dental clinic due to emergency visit had a dmft/S index of zero. The emergency in these cases was most likely not related to dental caries but to other causes, such as dental trauma. The preventive controlhad a significantly lower prevalence of caries and comprised

the highest percentage of children without caries. The oral health assessment showed that the child underwent restorative treatmentand emergency visit to dental clinichad a higher caries risk than child had preventive control treatmentand no prior dental visit groups.Our results are consistent with those of Wennhall et al.,who showed that an early start in oral health programs has significant benefit in the prevention of caries and that the more children attend dental exams, the lower the incidence of caries. In this study, children attending the dentist for the first time at six years of age did not exhibit significant differences in dmft/S scores compared to children who had previous preventive treatment. However, there was a significantdifferencein the caries component, with higher values for the Non-Visitors to dental clinic than children whom already had preventive treatment group. Therefore, while the values for the dmft/S index were similar, children who never had a dental visit had poorer oral health than children who attended Preventive treatment highlighting its importance. AnniLuoto et al.,(14) in which associations between Child Perceptions Questionnaire and dental fear were not statistically significant. This may be due to the fact that non-school going children are mainly street children, and among them there is a false impression that dentistry is mainly involved with extraction and is a painful procedure that may be further complicated by wide range of health problems, including malnutrition, communicable and infectious disease, poor oral health, cognitive disorders and learning difficulty(15). The study showed that subjects who rated their oral health as poor had higher levels of dental anxiety than those subjects who rated their oral health as good or average, this was in accordance with the findings by Locker and Liddell(4), Doerr et al.(7). Contrary to the findings of Erten et al.(8), Skaret et al.(9), Hagglin et al(10)the results from the present study showed no significantdifference in dental attendance based on anxiety level and this was similar to the reports from Indian studies by Acharya(3), EktaMalvaniaandAjithrishnan(11) but contrary to the findings of Pavi et al. (12), Stole et al. (13). The relationship between dental anxiety and socio economic status has not been clearly determined. In this study uneducated patients and patients with less or no income had no knowledge about their child's dental caries preventive treatment; this is in accordance with other studies [5,6].

Conclusion

Based on the results of this study, the following conclusion can be made

1. Prior dental experiences of six-year-old children were directly related to their dental caries experience.

2. Children who had preventive visits and those who had never seen a dentist before had low rates of dental caries.

3. Patients have low percentage of dental visit for their preventive treatment due to unawareness of their parents.

4. Simplified oral hygiene index and dental anxiety levels showed no statistically significant differences among the types of previous dental experiences.

Dental anxiety refers to the fear going to the dentists. It exists in a considerable number of children and it is a major plight and concern in pediatric dental practice(16).

Corresponding Author

V.Ashok,

Reader, Department of Prosthodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu,India.

Email Id:ashokprostho@gmail.com

Mobile Number: 9962422299

References

1. Sheilam,A.Oral Health, General Health And Quality Of Life- *Bull World Health Org*2005;83(9): 644-5.
2. Trangmar,K.National Oral Health Diagnosis Of Six-Year-Old Children. Division Of Disease Prevention And Control.Oral Health Department;*British Dental Journal*2007;108(3):22-6.
3. Acharya, S.Factors Affecting Dental Anxiety And Beliefs In An Indian Population. *Journal OfOral Rehabilitation*2008; 196(35): 259-267.
4. Locker D, Liddell AM. Correlates Of Dental Anxiety Among Older Adults. *Journal Of Dental Research*1991;70(3): 198-203.

5. Moore R, Birn H, Kirkegaard E, Brødsgaard I, Scheutz F.Prevalence And Characteristics Of Dental Anxiety In Danish Adults. Community Dentistry AndOral Epidemiology. *Journal OfDental Research*1993; 21: 292-296.
6. Arm EldJM, Spencer AJ, Stewart JF.Dental Fear In Australia: Who's Afraid Of The Dentist?*Australian Dental Journal*2006; 51: 78-85.
7. Doerr PA, Lang WP, Nyquist LV, Ronis DL. Factors Associated With Dental Anxiety. *Journal OfAmerican Dental Association*1998; 129: 1111-1119.
8. Erten H, Akarslan ZZ, Bodrumlu E. Dental Fear And Anxiety Levels Of Patients Attending A Dental Clinic. *Quintessence International Journal*2006; 37: 304-310.
9. Skaret E, Raadal M, Berg E, Kvale G. Dental Anxiety And Dental Avoidance Among 12 To 18 Year Olds In Norway. *European Journal OfOral Sciences*1999; 107: 422-428.
10. HäGglinC, Hakeberg M, Ahlqwist M, Sullivan M, Berggren U.Factors Associated With Dental Anxiety And Attendance In Middle-Aged And Elderly Women. Community Dentistry AndOral Epidemiology.*Journal OfDental Research*2000; 28: 451-460.
11. Malvania EA, Ajithrishnan CG.Prevalence And Socio Demographic Correlates Of Dental Anxiety Among Group Of Adult Patients Attending A Dental Institution In Vadodara City, Gujarat, India. *Indian Journal OfDental Research*2011; 22: 195-199.
12. Pavi E, Kay EJ, Stephen KW. The Effect Of Social And Personal Factors On The Utilisation Of Dental Services.Glasgow, Scotland. Community Dental Health.*Journal OfDental Research*1995; 12: 208-215.
13. Stole AC, Holst D, Schuller AA.Decreasing Numbers Of Young Adults Seeking Dental Care On Yearly Basis. A Reason For Concern? Nortannlegeforentid. *Journal OfDental Research*1999; 109: 392-395.
14. Luoto A, Lahti S, Nevanperä T, Tolvanen M, Locker D.Oral-Health-Related Quality Of Life Among Children With And Without Dental Fear. *Int J Paediatr Dent*2009;19:115–20.
15. J. Victoria, BC.The Crisis Of Chronic Disease Among Aboriginal Peoples: A Challenge For Public Health, Population Health And Social Policy, University Of Victoria. Centre For Aboriginal Health Research; University Of Victoria, Centre For Aboriginal Health Research;2009;108(3)3:22-6.

16. F. BareeraRezvi, Dr. Marian Anand Bennis. Comparison Of Anxiety Levels Of Children Facing Dentists Wearing Normal Masks Versus Child FriedlyMasks;Saveetha Dental College And Hospital;SaveethaUniversity;*IJPT*2017; ISSN: 0975-766X.
17. SutharshanaVelingiri,DeepaGurunathan.AssessmentOf Parent's Preference To General Or Local Anesthesia For Children Undergoing Dental Treatment;Saveetha Dental College;DepartmentOf Pedodontics;*Jp-Journals* 2017;108(3):10015-1333.
18. JembulingaSabarathinam,Dr.Geo Mani And Dr.DeepaGurunathan.AwarnessAnd Attitude About Dental Treatments Among Middle School Students;Saveetha Dental College;DepartmentOf Periodontics;*JPSR2016*; 7(4): (B) 637 – 641;2016.
19. Gayathri Devi Kumaresan;SaravanaKumar.AwarnessAmong School Going Children's In Chennai About Dental Health Care;Saveetha Dental College;DepartmentOf Pedodontics.*JPSR*2016;54-(3):45-47.
20. Deepa Gurunathan;Shanmugavek.A.K;Dental Neglect Among Children In Chennai;Saveetha Dental College;DepartmentOf Pedodontics.*JPSR*2016;53-(1):4.

Difficulties Encountered by the Dental Students during Transition from Preclinical to Clinical Endodontics – A Questionnaire Study

Bhavani Ganesan[1], Sandhya Raghu[2], Jaiganesh Ramamurthy[3]

Graduate Student[1], Reader[2], Department Of Conservative And Endodontics, Reader[3], Department Of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Feedback from the students is beneficial to review the curriculum and improve the standard of training.Endodontic treatment can be one of the most difficult dental procedures a student encounters during preclinical and clinical practice. Research on the difficulties of endodontic procedure performed in preclinical and clinical years are limited. Hence this study had been planned to analyse the difficulties.The aim of the study was to analyse the difficulties encountered by the dental students during transition from preclinical to clinical endodontics.

Materials and Methods: The questionnaires were distributed to 100 students including third year, final years and house surgeons pursuing under graduate program in dental surgery. The questions were focused on experiences during each step of root canal procedure including rubber dam placement, access related difficulties, difficulties during working length determination, shaping and cleaning and obturation. The data were analyzed to achieve the results.

Results: Most of the students reported that they had difficulty in rubber dam application in preclinical than clinical endodontics. Most of the students reported that reaching the pulp chamber or locating the orifices was much easier in preclinical procedure than clinical procedure. Students also reported that difficulties encountered during cleaning and shaping was more in clinical procedure. Time taken to adapt with the usage of magnifying loupes and rubber dam placement were the major concern of the students. Regarding incidents of instrument distortion in the form of bending, instrument separation, at pre-clinical stage: 57% said they never has such incidents, 24% said sometimes they encountered instrument

distortion, 19% said they had frequent incidents of instrument distortion. Whereas at clinical stage, 64% said they never had such incidents, 27% said sometimes they had instrument distortion, 9% said they had frequent incidents of instrument distortion.

Conclusion: This study suggested that students have better practice in endodontic procedure.By identifying the problems encountered by the students at the right time could help them overcome the clinical difficulties during endodontic procedures. This in turn would reduce procedural time and increases the success of the treatment.

Key Words: *Dental Students, Endodontics, Pre –Clinical, Obturation, Dental Procedures.*

Introduction

Theoretical and practical knowledge is taught to dental students in their dental course. The quality of guidance can be analysed by taking the students perceptions into consideration. Feedback from the students is beneficial to review the curriculum and improve the standard of training. [1]Endodontic treatment can be one of the most difficult dental procedures a student encounters during preclinical and clinical practice. Research on the difficulties of endodontic procedure performed in preclinical and clinical years are limited. [2]

Due to the increase life expectancy in the population and the desire of individuals to preserve their natural teeth, there is an increasing demand for endodontic treatment and this will presumably increase in the years ahead. This reality necessitates dental students to be satisfactorily equipped with knowledge as well as experience in endodontic procedures prior to working independently.The aim of the study was to evaluate the difficulties by the dental students during transition from preclinical to clinical endodontics.

Materials and Methods

The questionnaires were distributed to 100 students including third year, final years and house surgeons pursuing under graduate program in dental surgery. Thequestions were focusedon experiences during each step of root canal procedure including rubber dam placement, access related difficulties, difficulties during working length determination, shaping and cleaning and obturation. The data were analyzed to achieve the results.

Table 1:Questionnaire

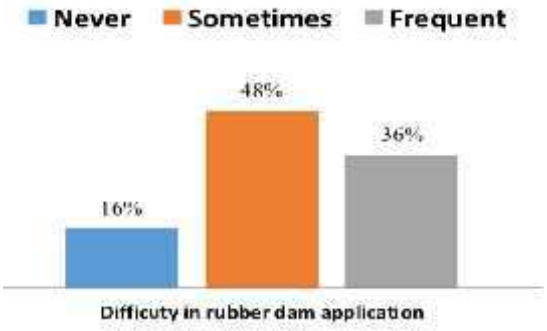
Year of Studying: BDS - Third year/ Final year/ Intern

PRECLINICAL	**CLINICAL**
1. Did you take time to adapt with the usage on magnifying loupes for pre-clinical endodontic procedure ? a) Yes b) No	Did you take time to adapt with the usage on magnifying loupes for clinical endodontic procedure ? a) Same as the preclinical b) Initial difficulty c) Still having difficulty
2.Did you have difficulty in rubber dam application ? a) Never b) Sometimes c) Frequent	Do you have difficulty in rubber dam application? a) Never b) Sometimes c) Frequent
3.Time taken for the placement of rubber dam? a) Less time b) More time	Time taken for the placement of rubber dam? a) Same as preclinical b) Less than preclinical c) More time than preclinical
4.Did you take a time for clamp selection ? a) Less time b) More time	Do you take a time for clamp selection ? a) Same as preclinical b) Less than preclinical c) More than preclinical
5.Did you have difficulty in reaching the pulp chamber ? a) Never b) Sometimes	Do you have difficulty in reaching the pulp chamber ? a) Never b) Sometimes

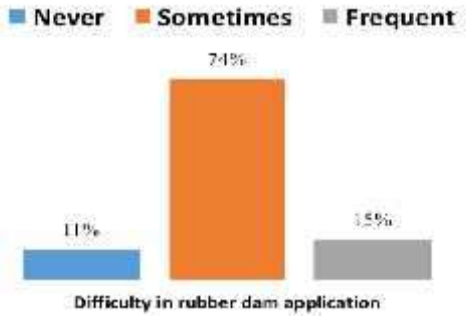
c) Frequent	c) Frequent
6.Did you have difficulty in locating the orifices in mandibular molars ? a) Never b) Sometimes c) Frequent	Do you have difficulty in locating the orifices in mandibular molars ? a) Never b) Sometimes c) Frequent
7. Did you have difficulty in the angulation (upper anteriors) ? a) Never b) Sometimes c) Frequent	Do you have difficulty in the angulation (upper anteriors) ? a) Never b) Sometimes c) Frequent
8.Did you have difficulty in removal of pulp tissue ? a) Never b) Sometimes c) Frequent	Do you have difficulty in removal of pulp tissue ? a) Never b) Sometimes c) Frequent
9. At the end of cleaning and shaping, do you loose your actual working length (master cone short of the apex)? a) Never b) Sometimes c) Frequent	At the end of cleaning and shaping, do you loose your actual working length (master cone short of the apex)? a) Never b) Sometimes c) Frequent
10.Did you have instrument distortion (blending, instrument separation, breakage etc) a) Never b) Sometimes C) Frequent	Did you have instrument distortion (blending, instrument separation, breakage etc) a) Never b) Sometimes c) Frequent

11.Have you done a ledge formation ? a) Never b) Sometimes c) Frequent	Have you done a ledge formation ? a) Never b) Sometimes c) Frequent
12. Have you done a apical perforation ? a) Never b) Sometimes c) Frequent	Have you done a apical perforation ? a) Never b) Sometimes c) Frequent
13. Did you have difficulty in placement of sealer ? a) Never b) Sometimes c) Frequent	Do you have difficulty in placement of sealer ? a) Never b) Sometimes c) Frequent
14.Did you have difficulty in placement of accessory cone in the canal ? a) Never b) Sometimes c) Frequent	Did you have difficulty in placement of accessory cone in the canal ? a) Never b) Sometimes c) Frequent
15. Did you have difficulty in using a spreader during obturation? a) Never b) Sometimes c) Frequent	Did you have difficulty in using a spreader during obturation ? a) Never b) Sometimes c) Frequent .

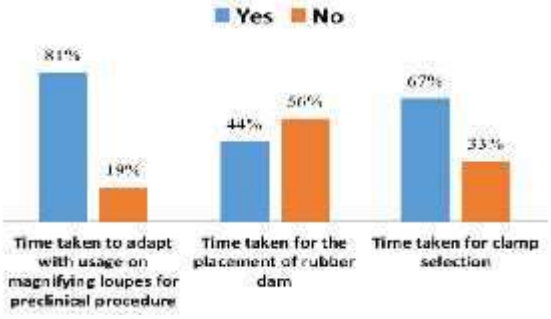
Results



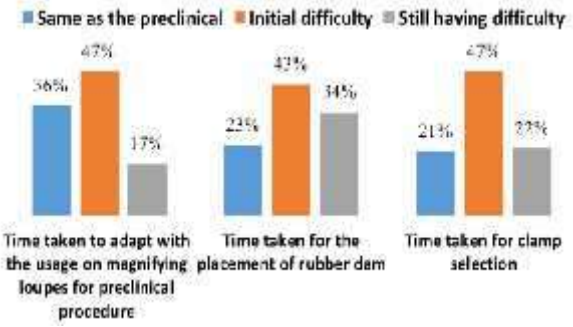
Graph 1: Pre-Clinical



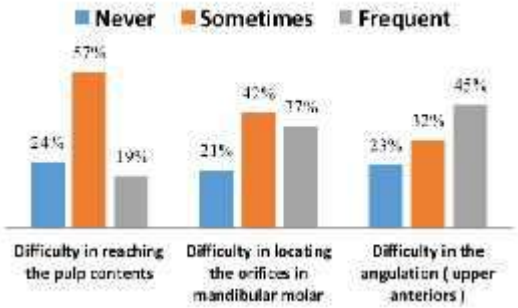
Graph 2: Clinical



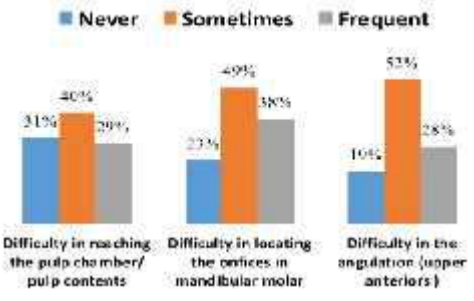
Graph 3: Pre-Clinical



Graph 4: Clinical

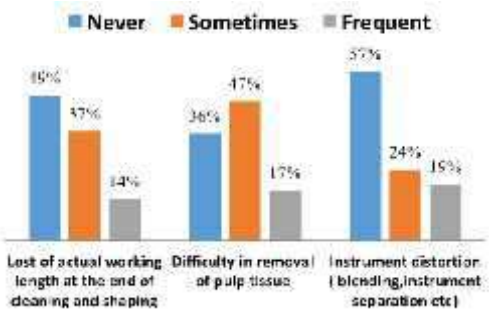


Graph 5: Pre-clinical

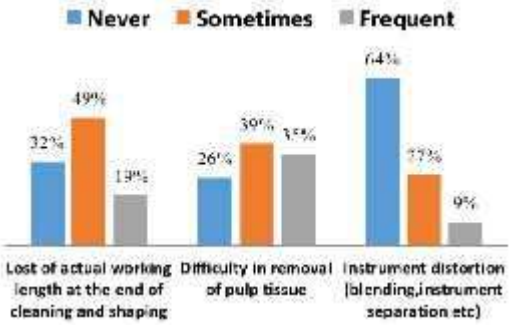


Graph 6: Clinical

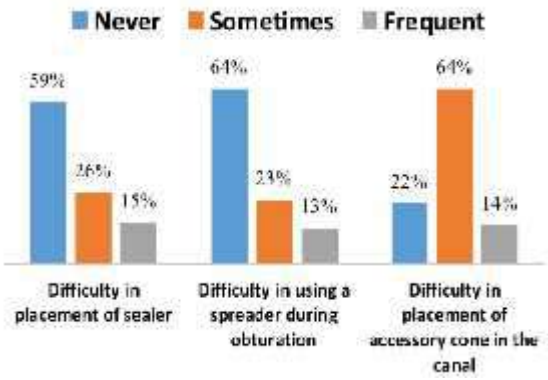
Graph 7: Pre-Clinical



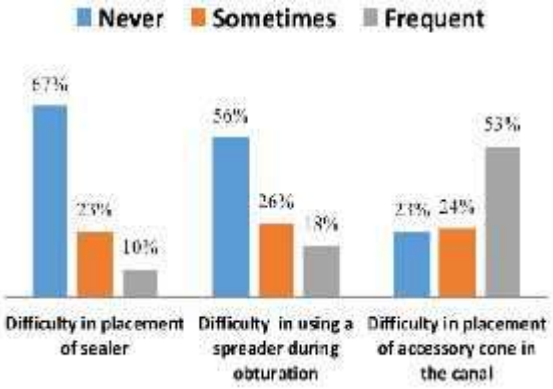
Graph 8: Clinical



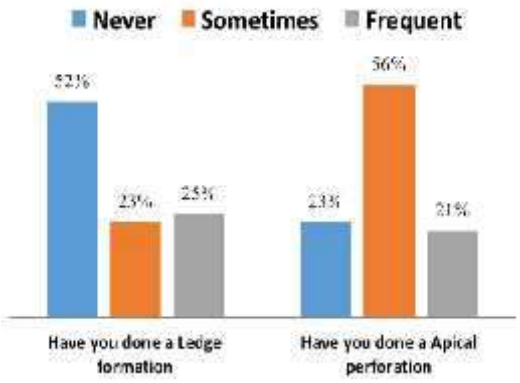
Graph 9: Pre-Clinical



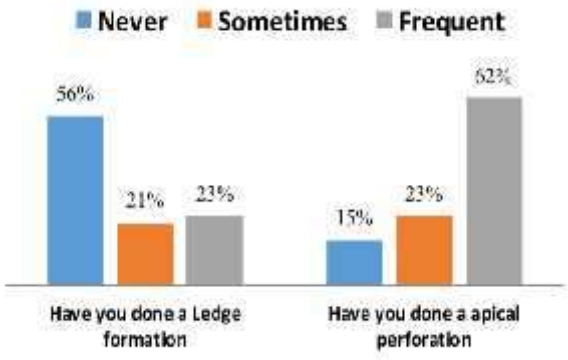
Graph 10: Clinical



Graph 11: Pre-Clinical



Graph 12: Clinical



With respect to difficulty in rubber dam application, during preclinical period 74% of students said sometimes they found it difficult to place the rubber dam, whereas 15% of students said they frequently had the difficulty and 11% said they never faced the difficulty.
In clinics, 48% of the students said sometimes it was difficult placing the rubber dam and 36% said frequently they had problems in placement and 16% said they never had the difficulty.

Regarding time taken to adapt with the usage on magnifying loupes for the procedure, 81% of students said they took long time to adapt and 19% said they were able to adapt quickly for using loupes in pre-clinical procedures. In clinical procedures, 36% of the students said time taken to adapt was similar to pre-clinical procedures and 48% said they had difficulty only in the initial stage and 17% said they are having difficulty even in the current stage and not getting changed. With respect to time taken for rubber dam clamp selection, 67% of the students said they took long time in pre-clinical level and 33% said they were able to select in less time and for students doing clinical procedures, 21% said its same as pre-clinical and 47% had initial difficulty in clamp selection and 22% said they have difficulty in clamp selection even in the present stage.

With respect to difficulty in reaching the pulp chamber and pulp contents, 57% of the students said sometimes they had difficulty in reaching the pulp chamber and 19% said they had frequent problems in reaching the pulp chamber and 24% said they never had problems in reaching the pulp chamber and pulp contents in pre-clinical level. In clinical level, 40% of the students had sometimes difficulty in reaching the pulp chamber and 31% said they never had that difficulty and 29% said they had frequent problems in reaching pulp chamber and pulp contents.

Same way when difficulty in locating the orifices in mandibular molar was analysed, 42% of the students said sometimes they had difficulty in locating the orifices and 37% had difficulty in accessing the orifices frequently and 21% never had difficulty in pre-clinical level and in clinical stage 49% had sometimes difficulty in locating the orifices of molar and 38% said its frequent and 23% said they never had the difficulty.
When asked about the difficulty in angulation for upper anteriors, 45% of the students said they had frequent difficulty in orienting themselves for angulation of the tooth and 32% said

sometimes they faced difficulty and 23% said they never had problems with the angulation in preclinical level. In clinical situations, 28% said they had frequent problems with angulation, 52% said sometimes they had problems and 19% said they never faced any difficulty in orienting themselves for the angulation of the anterior teeth.

When asked about the loss of working length at the end of cleaning and shaping, at the pre-clinical stage, 37% said sometimes they faced the issue and 49% said they never faced it and 14% said frequently they had problems with loss of working length at the end of cleaning and shaping. Whereas at clinical stage, 32% said they never had such an issue and 49% said sometimes they had the issue and 19% said frequently they had problems in loss of working length. When asked about the difficulty in removing pulp tissue, 47% said sometimes, 17% said frequent difficulty in removing the pulp tissue and 36% said they never had any difficulty in preclinical level. In clinical cases, 39% said sometimes they faced the difficulty, 26% said they never had difficulty and 35% said frequently they faced difficulties in removing the pulp tissue.

Regarding incidents of instrument distortion in the form of bending, instrument separation, at pre-clinical stage: 57% said they never has such incidents, 24% said sometimes they encountered instrument distortion, 19% said they had frequent incidents of instrument distortion. Whereas at clinical stage, 64% said they never had such incidents, 27% said sometimes they had instrument distortion, 9% said they had frequent incidents of instrument distortion.

When asked about the difficulty in placement of sealer, at pre-clinical level, 67% said they never faced any difficulty, 23% said sometimes and 10% said frequent difficulty in placement of sealer. In clinical situations, 59% said they never faced difficulty in placing sealer, 26% said sometimes only and 15% said frequent difficulty in placing sealer. When asked about the difficulty in using a spreader during obturation, 56% said they never had the difficulty, 26% said sometimes only and 18% said frequent encounters of difficulty. In clinical situations, 64% said they never had the difficulty, 23% said sometimes only and 13% said frequent episodes of difficulty in placing the spreader.

When asked about the difficulty in placement of accessory cone in the canal, in pre-clinical stage 23% said they never had the difficulty, 24% said sometimes only and 53% said they

had frequently encountered the problem. In clinical situations, 22% never had such instances, 64% said sometimes only and 14% said they had frequent episodes of difficulty in placement of accessory cone.

When asked about the ledge formation during RCT, in pre-clinical stage, 52% said they never had such an issue, 23% said sometimes only and 25% said frequently. With regards to apical perforation, 23% said they never done that, 56% sometimes only and 21% said frequently done apical perforation.

Discussion

Regular evaluation and feedback from students is necessary to improve and to overcome the difficulties. [3-5]Most of the students reported that they had difficulty in rubber dam application in preclinical than clinical endodontics.

Graph shows that time taken to adapt with the usage of magnifying loupes and rubber dam placement were the major concern of the students.Reason for difficulty in usage of magnifying loupes are, they have limitations such as convergent vision, deficient magnification, image distortion, colour alteration, small depth of focus, reduced working field, and fatigue caused by extended use. [6-8]To overcome this problem, choice of correct loupes and appropriate usage is indicated.Patient cooperation is a concern during rubber dam placement which could have been the reason for difficulty in a clinical scenario. [9]

Most of the students reported that reaching the pulp chamber or locating the orifices was much easier in preclinical procedure than clinical procedure.Students also reported that difficulties encountered during cleaning and shaping was more in clinical procedure.The problems with procedural errors can be reduced with the use of appropriate technique. [10-11]To retain the actual working length, recapitulation and proper irrigation is indicated. Instrument distortion can be reduced by avoiding inadvertent use of hand instruments. [12-15]Different methods have been used for locating the position of canal terminus and measuring the working length of root canals. These include radiographic methods, electronic methods, tactile method and other adjunctive methods. [16-20]

Considering obturation related difficulties, placement of accessory cone in the root canal was more difficult in preclinical than clinical endodontics, which can be managed with right

choice of master cone, spreader size and accessory cone. Most of the students reported that they had no difficulty in placement of sealers.

Conclusion

From the results of this study following conclusions can be made:

1.Placement of rubber dam during pre-clinical exercises reduced the difficulty of placement when they do their clinical cases.

2.Usage of magnifying loopes in pre-clinical exercises helped them to adapt themselves for using it in clinical situations.

3.Pre-Clinical exercises reduced the difficulty of reaching the pulp chamber, pulp contents, and teeth angulation when they performed in clinical situations.

4.Better instrument handling with less incidents of instrument distortion like bending, instrument separation occurred when they practiced well in pre- clinical exercises.

5.Efficiency of obturation was increased when the students practices pre-clinical obturation exercises before their clinical cases.

6.Frequency of formation of ledge and perforation significantly reduced when they practiced root canal procedures in pre-clinical stage.

This study suggested that students have better practice in endodontic procedure when they practiced proper pre-clinical training.By identifying the problems encountered by the students at the pre-clinical level itself could help them overcome the clinical difficulties during endodontic procedures.This in turn would reduce procedural time and increases the success of the treatment.

Corresponding Author

Jaiganesh Ramamurthy,

Reader, Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High road, Chennai 600 077, Tamil Nadu, India.

Phone Number: +91 98404 43463,

Email id: dr.r.jaiganesh@gmail.com

References

1. Henzi D, Davis E, Jasinevicius R, Hendricson W, Cintron L, Isaacs M. Appraisal of the dental school learning environment: The students' view. J Dent Educ 2005;69(10):1137-47.
2. AnumRehman, Harsh Rajvanshi, Reem T. Youzbaki; Common errors in access preparation by preclinical dentistry Students – A cross sectional study;IOSR Journal of Dental and Medical Sciences (IOSR-JDMS)2016;15(9):69-74.
3. Cardall WR, Rowan RC, Bay C. Dental education from the students' perspective: Curriculum and climate. J Dent Educ2008;72(5):600-9.
4. Henzi D, Davis E, Jasinevicius R, Hendricson W. North American dental students' perspectives about their clinical education. J Dent Educ2006;70(4):361-77.
5. Bush H, Bissell V. The evaluation of an approach to reactivelearning in the undergraduate dental curriculum. Eur J Dent Educ 2008;12(2):103-10.
6. Pradeep S, Vinoddhine.R .The Role of magnificationin Endodontics.Annalsand Essences of Dentistry2014; 6(2):38-43.
7. G. Midhun Mohan, V. Susila Anand.Accuracy of Different Methods of Working Length Determination in Endodontics.IOSR Journal of Dental and Medical Sciences2013; 12(4):25-38.
8. Berk NW, Close JM, Weyant RJ. Do student perceptions of their dental curriculum change over time? J Dent Educ 1998;62(11):934–7.
9. Levy G, d'Ivernois JF, Brun D. A French dental school programme appraisal of 5–9 years standing. Eur J Dent Educ 1997;1:70–7
10. Casada JP, Willis DO, Butters JM. An investigation of dental student values. J Am Coll Dent 1998;65(3):36–41.
11. Bertolami C. Rationalizing the dental curriculum in light of current disease prevalence and patient demand for treatment: form vs. content. J Dent Educ 2001;65(8):725–35.
12. Davis EL, Tedesco LA, Meier ST. Dental student stress, burnout, and memory. J Dent Educ 1989;59(3):587–97.
13. McCunniff MD, Holmes LG. Evaluation of attitudes—dental class of 1991: a nine year longitudinal study. J Am Coll Dent 1999;66(3):20–8.
14. Marciani RD, Smith TA, Kohn MW. Senior residents' opinions about oral surgery programs. J Oral Surg 1978; 36(7):508–12.

15. Pender N. Recent graduate opinion on orthodontic training. Br J Orthod 1982;9(2):73–6.
16. Morris S. What kind of people want to become dentists? Br Dent J 1992; 173:143–4.
17. Zadik D, Gilad R, Peretz B. Choice of dentistry as a career and perception of the profession. J Dent Educ 1997; 61(10):813–6.
18. Lee JD. Which kids can “become” scientists? Effects of gender, self-concepts, and perceptions of scientists. Soc Psychol Q 1998; 61(3):199–219.
19. Lee JD. More than ability: gender and personal relationships influence science and technology involvement. Soc of Educ 2002; 75:349–73.
20. Chmar JE, Weaver RG, Valochovic RW. Dental school vacant budgeted faculty positions: academic year 2004–05. J Dent Educ 2006; 70(2):188–98.

Etiologyfor Tooth Extraction among Individuals of Different Age Groups in Chennai

N. Vidulasri[1], Nithya Jagannathan[2], Caroline Jacob[3],
Graduate Student[1], Reader[2], Department of General Medicine**,**Senior Lecturer[3], Department of Periodontics,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction:Dental extractions are often performed when teeth have become challenging to restore as a result of tooth decay, periodontal disease or dental trauma. However, despite advances in dental therapy, individuals hesitate to save teeth thereby prolonging periods of edentulism which if unreplaced may pose difficulties in restoring esthetics and function.
The aim of this study is toextrapolate reasons for tooth extraction among individuals of different age groups in a population in Chennai.

Materials and Methods: A questionnaire surveywasdesigned to evaluate the reasons of extraction which was carried out among 221 patients with 500 tooth extractions having been performed for these individuals forvarying reasons such as periodontitis, dental caries, and trauma, orthodontic, prosthetic and endodontic failures.

Results: 500 extractions performed in 221 patients revealed that 113 (22.6%) teeth extracted was due to dental caries, 186 (37.2%) was extracted due to periodontitis, 94(18.8%) due to orthodontic problems, 75(15%) due to impaction and 32(6.4%) due to other causes.

Conclusion: In this study out of 500 teeth extractions, 186 (37.2%) teeth were extracted due to periodontitis which was also noted to be the major cause for tooth extraction in males over 31 years of age. The next most common cause of tooth extractions were due to teeth affected by dental caries and pulpitis(22.6%).In females,the latter was the major for tooth extraction, followed by periodontitis with the number of extractions increasing in individuals between the age groups 21 to 40 years. Among all age groups the most commonly extracted teeth were the lower posterior teeth.

Key Words:*caries, exfoliated teeth, exodontia, periodontal disease, tooth loss*

Introduction

Loss of teeth has become a major oral health problem worldwide and is attributed to caries, periodontal disease, trauma, impaction and orthodontic problems.The removal of teeth is one of the most commonly performed dento-alveolar procedures in oral and maxillofacial surgery.(1) Studies on tooth mortality can provide valuable information regarding the patterns of oral disease in individual populations.Preventive strategies have to be implemented to minimize tooth loss due to oral diseases.(2)Studies concerning the epidemiology of oral disease has shown that dental caries and periodontal diseases are the most prevalent conditions that affect the oral cavity.(3)Most of these surveys were designed to investigate the etiology, severity and distribution of tooth loss based on the patient demographics and tooth type.(3,4)Dental caries and periodontal disease are the foremost reasons for extraction of permanent teeth(5,6)while extraction due to orthodontic treatmentreplaced caries as the most common reason for extracting teeth in individuals younger than 20 years of age.(6) In cases of trauma, extensive tooth injury may require extraction. Assessment of tooth mortality data in different parts of the world is essential to evaluating the adequacy of dental care and preventive oral health programs.Studies indicatethat populations with poorer socioeconomic conditions have higher prevalence of the condition and extent of teeth mortality, which increases with the age of the individual.(7-9)Prior to the introduction of antibiotic use, chronic tooth infections were frequently associated with a variety of health-related conditions resulting in the removal of such diseased teeth as a common treatment option for such conditions.(10) Understanding the reasons fortooth extracted may improve oral health outcomes.Tooth loss has various harmful effects on an individual which may include the impairment of masticatory function, unpleasant aesthetics, poor phonetics, temporomandibular dysfunctions, psychological issues, social withdrawal and decrease in confidence.(11) The impact of tooth loss also includes decreased function of speech and mastication especially in the elderly.(12,13) Several complications may also occur during extraction of teeth, including damage to adjacent teeth, retained roots, fractured jaws in individuals with osteoporosis and nerve injury during the extraction of mandibulararthird molars. Therefore, the aim of this study was to elucidate the

reasons for the extractions of tooth among different age groups in males and females while analyzing teeth that were most commonly extracted.

Materials and Methods

The present study was undertaken to extrapolate the reasons for tooth extraction in different age groups among aconvenient sample size of 221 patients in a dental hospital in Chennai, India.The reasons for extractions were categorized into caries and pulpitis, periodontal disease, orthodontic problems, impacted teeth and other causes (cysts or tumour of the tooth, prosthetic removal, fractured tooth, supernumerary tooth).Demographic data and information regarding the etiology for tooth extraction was collected and entered in the proforma . The subjects were divided into seven groups from between 0-80years of age which included those belonging to the ages of 0-10 years, 11-20 years,21-30 years,31-40 years,41-50 years,51-60 years, 61-70 years and 71-80years.

The type of the tooth extracted was based on its location which was designated as maxillary anterior, maxillary posterior, mandibular anterior or mandibular posterior.The data was analysed and tabulated for male and female subjects based on the different reasons for tooth extraction. Comparisons between different age groups and commonly extracted tooth among different age groups were also analyzed.

Results

Table1 describes the predominant causes of extraction of the 500 teeth extracted. 113 (22.6%) teeth extracted was due to dental caries, 186 (37.2%) was extracted due to periodontitis, 94(18.8%) due to orthodontic problems, 75(15%) due to impaction and 32(6.4%) due to other causes.

Table1:Etiology for the tooth extraction

Etiology	Total no (n)	%
Caries and pulpitis	113	22.6
Periodontitis	186	37.2
Orthodontic	94	18.8

Impacted	75	15
Others	32	6.4

Table 2: Tooth extraction causes listed by gender and age groups

Etiology	**Females**							
	0-10	11-20	21-30	31-40	41-50	51-60	61-70	71-80
Caries and pulpitis	3	3	16	15	11	9	3	0
Periodontitis	0	0	6	5	16	29	17	3
Orthodontic	0	5	37	3	0	0	0	0
Impacted	0	2	30	5	0	0	0	0
Others	1	2	5	3	3	1	0	0
	Males							
Caries and pulpitis	3	3	16	7	7	6	9	2
Periodontitis	0	0	3	9	28	20	42	8
Orthodontic	0	3	42	4	0	0	0	0
Impacted	0	2	34	2	0	0	0	0
Others	1	1	6	5	4	0	0	0

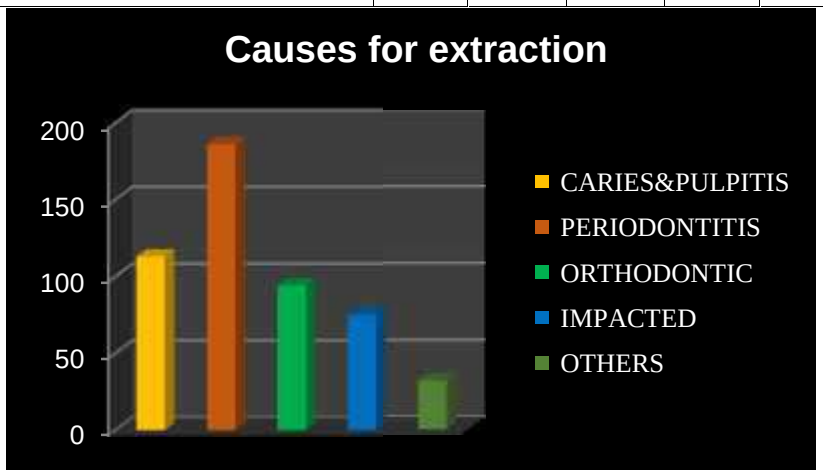


Figure 1: Graph depicting the etiology for tooth extraction

Dental caries and pulpitis were found to be a significant cause for extraction among female patients over 20 years and it was also the most significant cause of extraction for

thecombined age groups of 21-40 years. In addition, periodontitis was the major cause of tooth extraction in males which increased with age and among combined age groups of 31-80 years. In patients ranging between 21-40years,orthodontic problems was found to be a cause for tooth extraction. Impactions were a major cause of tooth loss in patients ranging between the age groups of 21-40 years. Other causes for extraction were equally distributed among all the agegroups.

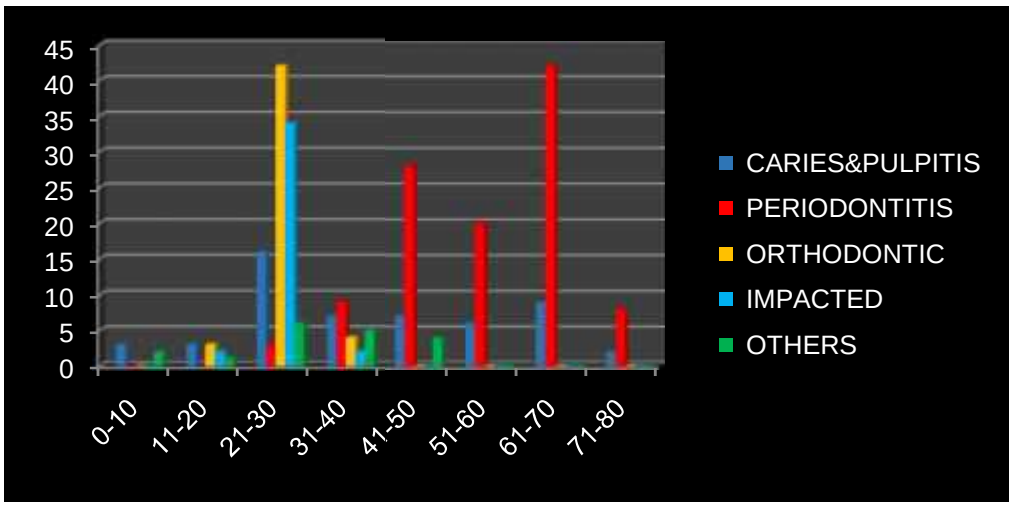


Figure 2: Etiology for tooth extraction in various age groups among males

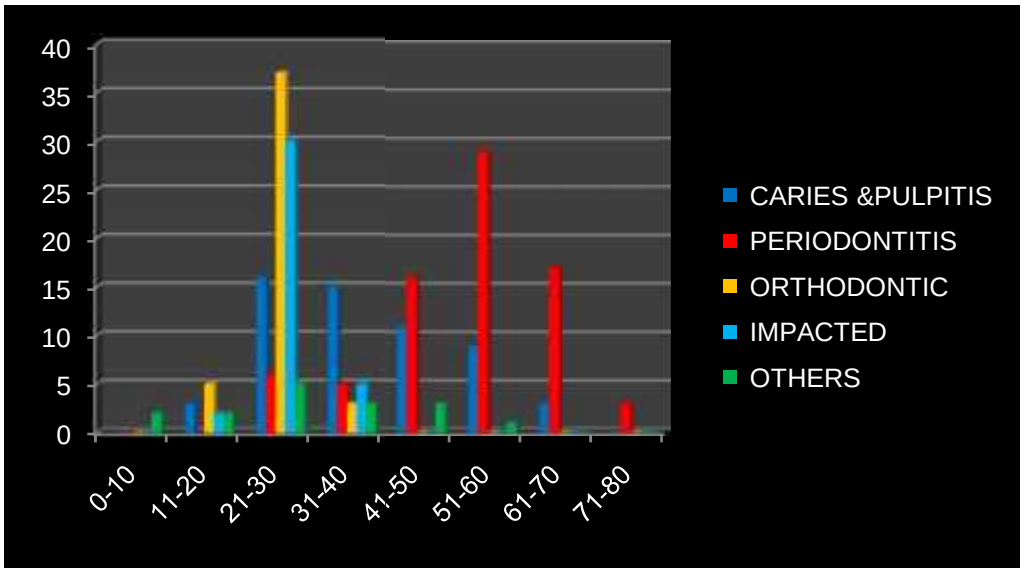


Figure 3: Etiology for tooth extraction among various age groups in females

Table 3:Description of extracted teethcategorized by age and tooth groups

AGE GROUP (Years)	Max.Ant	Max.post	Man.Ant	Man.post
0-10	1	2	1	3
11-20	1	5	0	8
21-30	3	27	0	48
31-40	6	30	4	19
41-50	8	44	19	33
51-60	13	50	12	21
61-70	15	35	16	35
71-80	12	8	6	15

(Max.Ant- Maxillary anteriors, Max.Post- Maxillary posteriors, Man.Ant- Mandibular anteriors, Man.Post- Mandibular posteriors)

Table 3 describes the most commonly extracted tooth type in different age groups. Maxillary posterior teeth were commonly extracted in patients ranging between 31-60 years which was closely followed by mandibular posterior teeth. Between the age group of 61-70 years, both mandibular and maxillary posteriors had the same incidence of extraction. However, among the oldest age group of 71-80 years, mandibular posterior teeth were most frequently extracted followed by maxillary posterior teeth. Maxillary anterior teeth were commonly extracted in the age groups of 51-60 years and 71-80 years while mandibular anterior teeth were most often extracted among patients aged between 41-50 years but was barely appreciable in individuals younger than 30 years of age

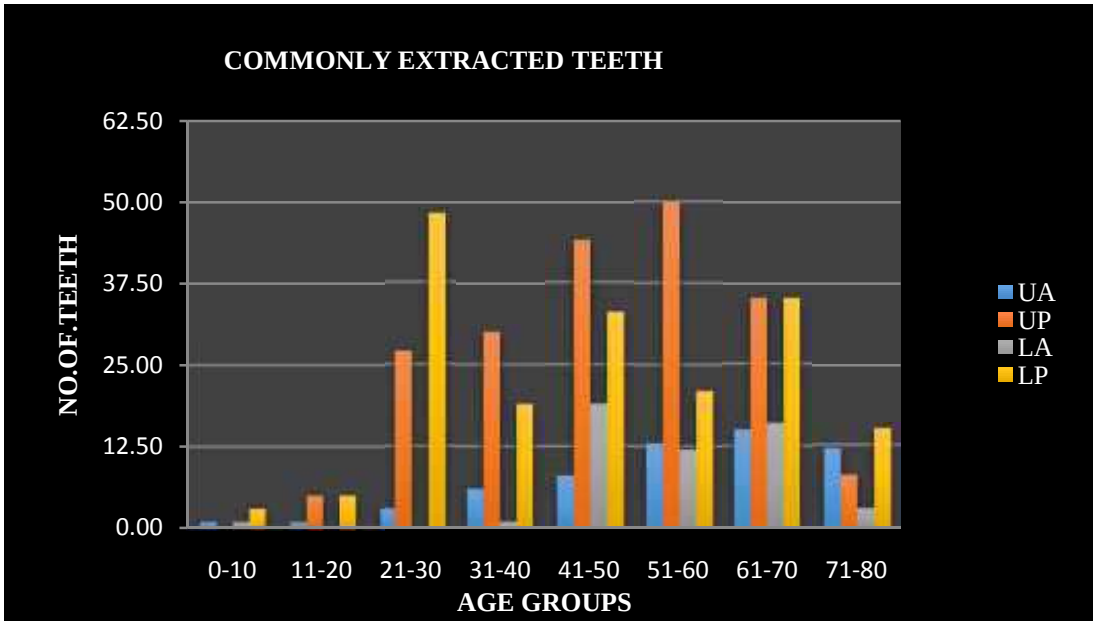


Figure 4: Graph depicting extracted teeth groups among various age groups (UA: maxillary anteriors, UP: maxillary posteriors, LA: mandibular anteriors, LP: mandibular posteriors)

Discussion

Tooth loss is the dental equivalent of mortality.The end result of oral disease can reflect both the patient's own attitude to dental hygiene and the availability and accessibility of oral health care.(4)Great variation exists in the frequency and causes of tooth extraction in different countries.(5)Research suggest that the incidence of dental caries and periodontal diseases has progressively increased with the advent and availability of refined sugar in the average diet.(6)In Singapore, a survey of the reasons for tooth extraction showed that the percentage of teeth extracted for periodontal reasons and caries was about the same. In Brazil, 70% of tooth extraction was found to be due to caries, while extraction due to periodontal disease accounted for only 15% of tooth loss.(6,7) Dental practices in southern Wales indicated that caries was the main reason for tooth extraction (59%) in patients attending routine dental treatment while periodontal disease was the second most common reason (29%).(8) A survey performed in Tehran proposed that dental caries and its complications were the leading reason for extraction followed by periodontal.(9) Furthermore, extractionsperformed in male patients were more due to periodontitis have been associated with the deleterious effects oftobacco andarecanut.(10)The current study also suggests that male patients lose more teeth than female patients as a result ofperiodontitis

while female patients lose teeth to both caries and periodontitis. This has been previously discussedas the main causes of tooth loss as the result of such extraction.(11, 12) However, other studies indicate thatperiodontitis is the main reason for extraction of teeth as compared to dental Caries (13). Other results suggest that 62% of extractions of permanent and primary teeth were due to caries, with periodontal disease being the main reason for extraction in patients over 40 years of age(14-16), which was similar to the results in this study. These differences may be attributed to global variances in diet, socio-economic factors, level of dental awareness as well as water fluoridation. This study also showed that posterior teeth were more frequently extracted compared to anteriors which in accordance to previous studies.(17-19)In a study by Daameh et al, incisors were most frequently extracted in older individuals due to periodontitis(20), with mandibularanterior teethbeing most periodontal involvement.(21) In this study incisors were the second most common teeth to be extracted after molars which was due to the effect of periodontitis on those teeth. A more likely reason for the high periodontal extraction in anterior teeth is due to the fact that they are less susceptible to caries, retained for a longer duration in the mouth, and then may be subjected to the risk of periodontal disease(21,22). Maxillary premolars and molars in males were most commonly extracted tooth as a result of periodontal disease, but as was the case for caries, the presence of caries was the main reason for loss of all maxillary teeth in females.(23)The majority of extractions of third molars has been shown to occur in the age group 21–30 years (48.2%), while most of the extracted first premolars (58%) occurred in the age group 10–20 years (24), which was in accordance with this study. This may have been due toimpacted third molars and teeth that had to be extracted for orthodontic treatment especially in patients between the age groups of 21-40 years. Irregular tooth brushing and irregular visits to dentists significantly increased the number of third molar extractions due to caries and periodontal disease compared with those who reported regular tooth brushing and making regular dental visits.(25) Therefore, regular dental visits would be the best way of reducing need for more invasive and uncomfortable proceduressuch as extractions.(26) Studies have also reported that subjects with higher education levels had comparatively fewer tooth extractions than those with incomplete or lower education levels. This relationship is in agreement with other studies in which tooth loss was associated with a low level of education.(27)Retention of a complete dentition throughout lifetime should be the primary goal of the dental professional. Dental hygienists, including therapists and dental nutritionists, would help in the development of a sound oral hygiene and dietary changes, which in effect would reduce the

occurrence of dental caries and periodontal diseases. In addition, community water fluoridation is one among the most cost-effective methods for preventing tooth decay. Globally, studies denote reduction of dental caries about 40-50% in deciduous dentition and 50-65% in permanent teeth.(28) Despite the fluoride-related preventive efforts in primary schools as a national protocol since 1997 (29, 30),dental caries remains as the major problem in a proportion of the population. Regular dental checkups are still not a routine pattern of behaviour for allindividuals as evidenced by the levels of periodontitis observed which has been linked to various systemic diseases.

Conclusion

Dental extractions are a cause of anxiety for patients both in terms of pain management and the edentulous space created post-extraction.Possible complications also include alveolar osteitis, infection, haemorrhage, paresthesia and less common iatrogenic effects. Avoiding extractions therefore suggests that prevention of tooth loss is a herculean challenge due to the various causes. The results of this present study indicate that periodontitis is the major cause for tooth extraction in males with caries and periodontitis among female patients.Strategies for efficient methods to screen and impart dental education for both etiologies with prolonged periods of follow-up is the need of the hour and must be seriously considered to improve the quality of life for such patients.

Corresponding Author
Caroline Jacob,
Senior Lecturer,Department of Periodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India
162,Poonamallee High Road, Chennai 600077,Tamil Nadu, India
Mobile Number: +91 9840035702
E-mail Id:caroline_jacob@ymail.com

References

1. Nazar MN, Puthiriraj V. Analgesics Following Mandibular Third Molar Surgery. Int J

Pharm Clin Res. 2014;6 (1): 13-19.

2. Morita M, Kimura T. Reasons for extraction of permanent teeth in Japan. Community Dent Oral Epidemiol. 1994;22(5):303-06.

3. Hull S, Worthington HV, Clerehugh V, Tsirba R, Davies RM, Clarkson JE. The reasons for tooth extractions in adults and their validation. J Dent. 1997;25(3):233-37.

4. Agerholm D. Reasons for extraction by dental practitioners in England and Wales: a comparison with 1986 and variations between regions. J Dent. 2001;29(4):237-41.

5. Chauncey HH, Glass RL, Alman JE. Dental caries. Principal cause of tooth extraction in a sample of US male adults. Caries Res. 1989;23(3):200-05.

6. George B, John J, Saravanan S, Arumugham IM. Prevalence of permanent tooth loss among children and adults in a suburban area of Chennai. Archives of Oral Sciences and Research 2011;1(2):72-8.

7. Mishellany-Dutour A, Renaud J, Peyron MA, Rimek F, Woda A. Is the goal of mastication reached in young dentates, aged dentates, and aged denture wearers? Br J Nutr 2008;99(1):121-8

8. Susin C, Oppermann RV, Haugejorden O.Tooth loss and associated risk indicators in an adult urban population from south Brazil. Acta Odontological Scandinavica 2009; 63(2), 85–93.

9. Jafarian M , Etebarian A.Reasons for Extraction of Permanent Teeth in General Dental Practices in Tehran, Iran. Med PrincPract 2013;22(3):239–244

10. Chestnutt IG, Binnie VI, Taylor MM. Reasons for tooth extraction in Scotland. J Dent. 2000;28(2):295–7.

11. Susin C, Haas AN, Opermann RV. Tooth loss in a young population from south Brazil. J Public Health Dent 2006;66 (2):110-115.

12. Razak IA, Jaafer N, Matnor G. The causes of tooth mortality of permanent teeth in a Malaysian population. J Ir Dent Assoc. 1989;35:39–41.
13. Barbato PR, Nagano HC, Zanchet FN. Tooth loss and associated socioeconomic, demographic, and dental-care factors in Brazilian adults. Cad SaudePublica. 2007; 23(8):1803-14.
14. Musacchio E, Perissinotto E, Binotto P. Tooth loss in the elderly and its association with nutritional status, socio- economic and lifestyle factors. Acta Odontol Scand 2007;65(2):78–86.
15. Al Shammari KF, Al Ansari JM, Al Melh MA, Al Khabbaz AK. Reasons for tooth extraction in Kuwait. Med PrincPract. 2006;15(6):417–422.
16. Thomas S, Al-Maqdassy SE: Causes and pat- tern of tooth mortality among adult patients in a teaching dental hospital. J Med BS 2010;2(1):160–167.
17. Farsi J. Common causes of extraction of teeth in Saudi Arabia. Saudi Dental J. 1992;4(3):101–105
18. Fardal O, Johanessen AC, Linden GJ. Tooth loss during maintenance following periodontal treatment in a periodontal practice in Norway. J Clin Periodontol. 2004;31(7):550-555.
19. Mosha HJ, Lema PA. Reasons for tooth extraction among Tanzanians. East Afr Med J. 1991;68(1):10–14.
20. Reich E, Hiller KA. Reasons for tooth extraction in the western states of Germany. Community Dent Oral Epidemiol. 1993;21(6):379–383.
21. Hamasha AA, Al Qudah MA, Bataineh AB, Safadi RA. Reasons for third molar teeth extraction in Jordanian adults. J Contemp Dent Pract. 2006;7(5):88–95.
22. Kay EJ, Blinkhorn AS: The reasons underly- ing the extraction of teeth in Scotland. Br Dent J 1986;160(8):287–290.
23. Shigli K, Hebbal M, Angadi GS. Relative contribution of caries and periodontal disease in adult tooth loss among patients reporting to institute of dental sciences

Belgaum, India. Gerodontology. 2009;26(3):214-218.

24. Daameh D. Reasons for permanent teeth extraction in north of Afghanistan. J Dent. 2006;34(1):48-51.

25. Burt BA, Ismail AI, Morrison EC, Risk factors for tooth loss over a 28 year period. J Dent Res. 1990;69(5):1126-1130

26. Alesia K and Khalil HS. Reasons for and patterns relating to the extraction of permanent teeth in a subset of the Saudi population. Clin CosmetInvestig Dent. 2013;5:51-56.

27. Grossi S, Genco R, Machtei E, Ho A: Assessment of risk for periodontal disease and Risk indicators for alveolar bone loss. J Periodontol 1995;66(1):23–29.

28. Ong G. Periodontal reasons for tooth loss in an Asian population. J Clin Periodontol. 1996;23(4):307-309.

29. Hessari H, Vehkalahti MM, Eghbal MJ, Sa- madzadeh H, Murtomaa HT: Oral health and treatment needs among 18-year-old Iranians. Med PrincPract 2008;17(4):302–307.

30. Pakshir HR: Dental education and dentistry system in Iran. Med PrincPract 2003; 12(1):56–60.

31. Santhosh Kumar M.P, Evaluation of Various Factors for Extraction of Permanent Teeth in a Dental College.J. Pharm. Sci. & Res. Vol. 8(1), 2016, 40-46

32. Hari Priya.M,Santhosh Kumar MP, Dental Extraction – Post Operative wellness Survey. Int.J. PharmtechRes. 2015,8(4), 666-669.

33. Balasubramaniyam.P, Nabeel.N, Complications following mandibular third molar surgery. International Journal of Clinical Dentistry 2015;7(4):337-346.

Evaluate the Commonly used Behaviour Management Techniques among Undergraduate Students- Survey

M.Indhumathi[1], Kanthaswamy.A.C[2], Hemavathy.O.R[3],
Graduate Student[1], Senior Lecturer[2], Department of Pedodontics, Reader[3],
Department of Oral and Maxillofacial Surgery,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: Good behavioural management techniques are essential for treating children. The establishment of good rapport between dentist and patient makes the success of treatment, whether in terms of the child's co-operation in undergoing treatment or in following prevention and advice. Various methods were used in pediatric dentistry to manage the child.The aim of the study was to analyze and assess the behaviour of children during their dental visit and to determine the effect of behavior management techniques.

Materials and Methods: The study was conducted in the Department of Pedodontics, to assess the behaviour of children during their dental visits and to determine the effect of behavior management techniques on children. The study was done by analyzing the records of children between the age group of 3 years who had undergone dental treatment.

Results: In this study, there were about 45% of the studentsfeltcomfortable with the parental accompaniment for their pediatric patients under the age of 3, 54% of the students felt pressured by parental expectations and 85% of the students felt comfortable with the tell show do technique.

Conclusion: Among all behaviour management techniques, the undergraduate studentsfeltcomfortable with the Tell show do techniquein dental practice.

Key Words:*Child's behaviour, techniques, management, undergraduate students, attitude.*

Introduction

One of the most challenging problems faced by dental practitioners and dental students is behaviour management.(1,9)Psychological variables (anxiety and/or stress), socio-cultural (individual characteristics, children's maturity, previous dental experience) and legal requirements (parent's consent) are involved in dental treatmentinterfering with professional performance.Behaviour management techniques (BMTs) that facilitate the delivery of treatment in a safe, efficient and compassionate manner are an integral component in the practice of pediatric dentistry.(2,8,10) This helps in good rapport between the dentist and the child in order to achieve the goal of the dentist.(3,4,5)There are different behaviour management techniques that are used in dentistry for good cooperation of the child.They are Tell show do, nitrous oxide, papoose board, voice control, hand over the mouth, oral pre medication, active restraint, general anaesthesia, dentist spending time in waiting room prior to treatment, dental assistant spending time in waiting room prior to treatment, live modelling, video modelling.These techniques are helpful in the success of dental treatment.(6,7)

Materials and Methods

This is across-sectional questionnaire based study on commonly used behaviour management techniques among undergraduate students. Data were collected from 100 undergraduate students of Saveetha dental college, Chennai. The questionnaires were based on three categories that included parental accompaniment scale, parental expectation scale and proportion of dentists feeling with each behaviour management techniques.Therewere eight open questions under parental accompaniment scale, four questions under parental expectation scale and twelve questions under proportion of dentists feeling with each behaviour management techniques. The questionnaires were used to assess the knowledge of the dental practitionersbasedonthe behaviour of children during their dental visits and to determine the effect of behavior management techniques on children.(15)

Results

This study was performed among 100 undergraduate students of Saveetha dental college, Chennai. 68 females and 32 males answered the questionnaire.They were under the age group of 18-23 yrsstudying 3 rdyr, 4 thyr and Internship of Bachelor degree of dental

surgery.Therewere five options for each questionnaire.Theywere strongly agree, agree, neutral, disagree and strongly disagree.(21,22)

Table 1: Shows the parental accompaniment scale based on no of questions and no of undergraduate students who answered the questions.

No of questions	**Strongly agree (in %)**	**Agree (in %)**	**Neutral (in %)**	**Disagree (in %)**	**Strongly Disagree (in %)**
1.In General, children under the age of 3 behave better when one of their parent is present during the course of the treatment.	45%	15%	10%	5%	25%
2.In general, children over the age of 3 behave better when one of their parent is present during the course of treatment.	37%	14%	29%	20%	0
3.In general, separating children from their parents when dental treatment commences	25%	45%	30%	0	0

simply increases the child's anxiety.					
4.In most cases, parental accompaniment of the child assists the dentist because it increases the child's anxiety.	34%	26%	22%	12%	6%
5.In general, I support parent accompaniment of the child during the course of the treatment.	23%	57%	20%	0	0
6.I think parental accompaniment of the child during treatment is counter productive.	15%	34%	40%	5%	6%
7.In general, I feel comfortable managing a child when the parent is present during treatment.	28%	32%	20%	7%	13%
8.I prefer	12%	67%	12%	9%	0

parents to stay in the reception room whilst I am treating the child.					

Table 2: Shows the parental expectation scale based on no of questions and no of undergraduate students who answered the questions.

No of questions	**Strongly agree (in %)**	**Agree (in %)**	**Neutral (in %)**	**Disagree (in %)**	**Strongly disagree (in %)**
1.Many parents have unrealistically high expectations of their child's treatment.	20%	50%	10%	7%	13%
2.I often feel pressured by parental expectations of their child's treatment.	37%	48%	15%	0	0
3.I feel increasingly pressured by parental expectations.	54%	23%	12%	6%	5%

4.I sometimes feel that parents do not trust me in treating their child.	42%	28%	10%	13%	7%

Table 3: Shows proportion of dentists feeling 'totally comfortable or uncomfortable'with each behavioural management technique.

Behaviour management techniques	**Totally comfortable**	**Uncomfortable**
Tell show do	85%	15%
Nitrous oxide	20%	80%
Papoose board	35%	65%
Voice control	68%	32%
Hand over mouth	47%	53%
Oral pre- medication	12%	88%
Active restraint	32%	68%
General anaesthesia	45%	55%
Dentist spending time in waiting room prior to treatment	56%	44%
Dental assistant spending time in waiting room prior to treatment	68%	32%
Live modelling	82%	18%
Video modelling	75%	25%

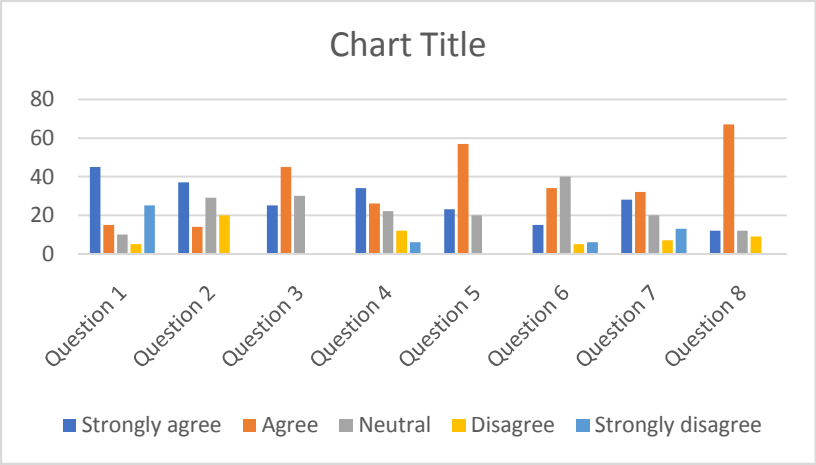
Discussion

The study was based on three categories. They were based on parental accompaniment scale, parental expectation scale and proportion of dentists feeling for each behaviour management techniques.The questionnaire contains eight direct questions.100 undergraduate students

answered these questions. Five similar options were given to all the questions. The option were strongly agree, agree, neutral, disagree and strongly disagree. The students answered the questionbased on their way of treating the child and the success of the treatment.

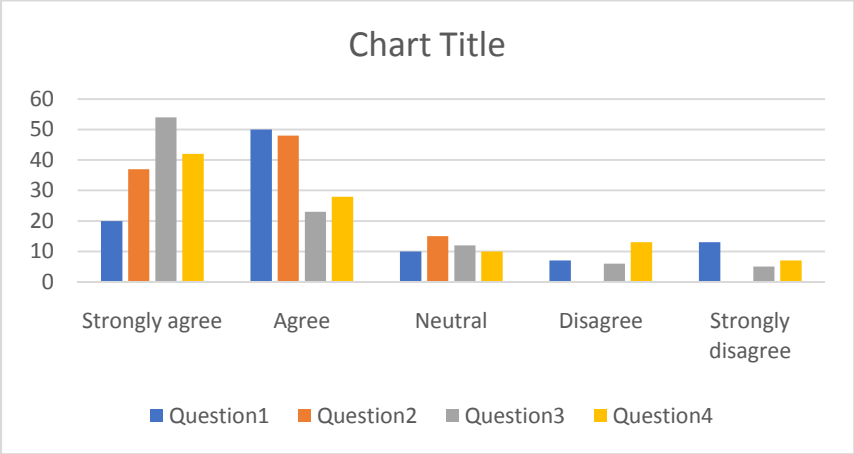
In majority of the cases, The parental accompaniment was strongly agreed only under the age of 3 to make the behaviour of the child better during the course of the treatment. It was strongly disagreed, asit may increase the child's anxiety during the treatment.(5)Generally, the behaviour of the child was different, if they were above the age of 3and it is difficult to manage the childwith the parent during the treatment. Based on the parental accompaniment the dentist feeling was recorded in chart 1,

Chart 1: Shows the parental accompaniment scale based on no of questions and no of undergraduate students who answered the questions.



The expectation level of the parents would be higher, due toincreased use of the internet for information and the trend of medical television shows may have increased the awareness of oral cavity.In majority of the cases, the parents does not trust their dentist and they felt pressured in most of the situations during the treatment.(5)In parental view, the basic trust on the dentist was more importantto overcome the treatment. The expectation level of the parent was recorded in chart 2,

Chart 2: Shows the parental expectation scale based on no of questions and no of undergraduate students who answered the questions.



In the last several decades, the acceptability of various behaviour management techniques have changed with pharmacological techniques like sedation and general anaesthesia getting better acceptance in recent studies.(7)

Examining the results, oral premedication and general anaesthesia were ranked the lowest acceptable techniques in 1984 and 1991.(21) However, acceptability for both pharmacologic methods increased in subsequent studies from 2005 till 2017.(22) Now it is quite interesting to speculate the reasons of this paradigm shift towards pharmacological methods. Changing attitudes toward acceptability of behavior management techniques may be attributed to changes in parenting styles over the past years. A recent study reported parents are more overprotective and less likely to set limits on children's behaviour. As a result, there may be a shift towards more pharmacologic behavior management techniques.(10)

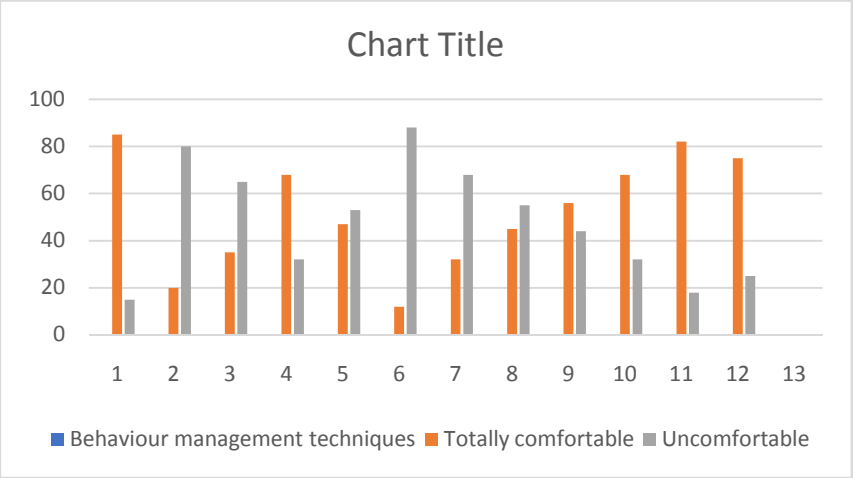
Changes in medications, with increased safety profiles and efficacy, used for oral premedication (sedation) over the years may also contribute to the rising acceptability. Overall, parents may perceive oral premedication (sedation) and general anesthesia to be less risky, more cost-effective, more comfortable for their children than in the past .(11,13)

Most parents in the present study preferred Tell – Show – Do followed by Live modelling to be employed on their children. These findings were consistent with a previous study, in which the most preferred techniques were Tell-Show-Do, Live modelling and Video modelling.(14) Findings were also consistent with previous study which found that most parents preferred an explanation as proper approach for treating their children.

Most of thedentistsin the present study moderately preferredHand over Mouth followed by physical restraint and voice control. These findings were consistent with another study which found physical restraint technique unacceptable by parents, however, the parents accepted GA in emergency dental situations.The acceptance of general anesthesia and sedation has

shown an increase in the present study when compared other studies. The most significant outcome of the study was the increased acceptance of pharmacologic behaviour management techniques.

Chart 3: Shows proportion of dentists feeling 'totally comfortable or uncomfortable'with each behavioural management technique.



Conclusion

This study, states that the parents wished to accommodate their child during treatment so that they feel comfortablewith the treatment, but the dental practitioner doesn't want parents to accompany their child in all situations.(20) In some situations parents were asked to leave the child because of some unwanted behaviour of the child .Behaviour assessment helps us to plan appointments and provide quality oral health care to children. Proper use of management techniques improves behaviour on subsequent visits, making things easy for the child and the pediatric dentist. Assessment and evaluation helps us to reinforce our beliefs in our own techniques.This study shows the commonly used behaviour management techniques. They were evaluated based on the parental accompaniment scale, based on parental expectation scale, based on proportion of dentists whether they were feeling comfortable or uncomfortable with each behaviour management techniques.It is a questionnaire based study among undergraduate students. The questionnaire contains direct questions with similar options. The evaluation of this is study based on the way of treating the child and the success of the treatment. The result of the study shows the dentists feelscomfortable with the parent accompaniment during the treatment, but not in all situationsand also the dentists feel increasingly pressured by theparentalexpectation. A wide variety of behaviour management

techniques are available in paediatric dentistry but Tell show do techniqueis the commonly used behaviour management techniquefor treating the children among undergraduate students. This is a method of introducing child patients to a procedure in a step wise manner. It has its own hierarchy tell, then, show then do to allow the child to assimilate the procedure in a graduated manner. Thus the proper use of the behaviour management techniques makes the child feel comfortable and minimise the anxiety during dental procedures.

Corresponding Author

Hemavathy.O.R,

Reader, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai -600077, Tamil Nadu, India.

Mobile Number: +91 9842564465,

Email: hemadocmd@gmail.com

References

1. American Academy of Pediatric Dentistry. Clinical guidelines on behaviour guidance for the pediatric dental patient. At: www.aapd.org/media/policies.asp. Accessed: May 9, 2006.
2. Ng MW. Behaviour management conference panel IV report: educational issues. Pediatr Dent 2004;26(2):180-3. Pediatr Dent 2004;26(2):180-3.
3. York KM, Mlinac ME, Deibler MW, Creed TA, Ganem I. Pediatric behavior management techniques: a survey of predoctoral students. J Dent Educ 2007;71(4):532–9.
4. Crossley ML, Joshi G. An investigation of paediatric dentists' attitudes towards parental accompaniment and behavioural management techniques in the UK. Br Dent J 2002;192(9):517–21.
5. Adair SM. Behavior management conference panel I report: rationale for behavior management techniques in pediatric dentistry. Pediatr Dent. 2004;26:167–70.
6. American Academy of Pediatric Dentistry. Proceedings of the consensus conference: Behavior management for the pediatric dental patient. American Academy of Pedi- atric Dentistry. Chicago, Ill; 1989.

7. Roberts JF, Curzon ME, Koch G, Martens LC. Review: Behaviour management techniques in paediatric dentistry. Eur Arch Paediatr Dent 2010;11:166-74.
8. Abushal MS, Adenubi JO. The use of behavior management techniques by dentists in Saudi Arabia: A survey. Saudi Dent J 2000;12:129-34.
9. Chambers, D. W.: "Behavior Management Techniques for Pe-diatric Dentists: An Embarrassment of Riches," J Dent Child,44:30-34, 1977.
10. Oredugba FA, Sanu OO. Behavior Management Techniques Employed by Nigerian Dentists for their Child Patients. PesquiBras OdontopediatriaClinIntegr 2009; 9: 271-6.
11. Howitt JW, Stricker G. Child patient's response to various dental procedures. J Am Dent Assoc 1965;7:70-4.
12. Venham L, Bengston D, Cipes M. Children's response to sequential dental visits. J Dent Res 1977;56:454-9.
13. Venham L, Quatrocelli S. The young child's response to repeated dental procedures. J Dent Res 1977;56:734-8.
14. Venham L. The effect of mother's presence on child's response to dental treatment. J Dent Child 1979;46:219-25.
15. Folayan MO, Idehen EE, Ufomata D. The effect of sociodemographic factors on dental anxiety in children seen in a suburban Nigerian Hospital. Int J Pediatr Dent 2003;13:20-6.
16. Allen KD, Stanley RT, McPherson K. Evaluation of behavior management technology dissemination in pediatric dentistry. Pediatr Dent 1990;12:79-82.
17. Carr KR, Wilson S, Nimer S, Thornton JB Jr. Behavior management techniques among pediatric dentists practicing in the southeastern United States. Pediatr Dent 1999;21:347-53.
18. Peretz B, Glaicher H, Ram D. Child-management techniques their differences in the way female and male pediatric dentists in Israel practice? Braz Dent J 2003;14:82-6.
19. Adair SM, Waller JL, Schaffer TE, Rockman RA. A survey of members of the American Academy of Pediatric Dentistry on their use of behavior management techniques. Pediatr Dent 2004;26:159-66.
20. Koenigsberg SR, Johnson R. Child behavior during three dental visits. J Dent Child 1975;38:33-6.

21. Ravikumar D., Gurunathan D., Karthikeyan S., Subbramanian E.M.G., Victor Samuel A.Age and environment determined children's preference towards dentist attire - A cross - Sectional study.. Journal of Clinical and Diagnostic Research2016;10:16-19.
22. Ravikumar D., Mahesh R., Subramanian E.M.G., Vishnu Priya V., Gayathri R., Geetha R.V.Comparative evaluation of oral microbiota of children with and without early childhood caries born to caries free mothers..Asian Journal of Pharmaceutical and Clinical Research. 2016;3-9.

Evaluating the Knowledge of Dental Students about the Widely Accepted Landmarks for Occlusal Plane Orientation- A Questionnaire Study

Benazir HussainM[1], Sangeetha R[2], Radhika Arjunkumar[3]

Graduate Student[1], Senior Lecturer[2], Department of Prosthodontics, Reader[3], Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

Abstract

Introduction: Occlusal plane orientation is an important factor in the construction of a complete denture. Occlusal plane could be oriented using landmarks in the mandibular arch as well as in the maxillary arch. In the mandibular arch there are few landmarks which could be used to orient the occlusal plane like the retromolar pad, corner of the lips (lower lip length) whereas the maxillary arch has a number of landmarks, of which the ala-tragal line is the most commonly used. The present study was designed to assess the knowledge of dental students on landmarks used in occlusal plane orientation. The aim of this study is to assess the knowledge of the dental students about the widely used landmarks for occlusal plane orientation in construction of prosthetic dentures andwhether they implicate during the treatment of patients.

Materials and Method: A cross- sectional questionnaire survey was conducted among 100 students of saveetha dental college and hospital divided into two groups (inclusion criteria- final years[50] and interns[50]). Data were collected using a self- administered questionnaire adapted from several studies addressing various aspects of knowledge and attitude of students on landmarks used in occlusal plane orientation for the construction of denture both in static and dynamic relation.

Statistical Analysis: The data was first transferred into Microsoft spread sheets. All of these results were analysed by using SPSS statistical software in terms of percentages. (statistical package for social sciences, SPSS Inc., Chicago, IL, USA). Associations between

discreet variables were tested by chi square test. p value≤ 0.05 was considered to be statistically not significant.

Result: In present study, the results showed that there was lack of knowledge on the landmarks used in occlusal plane orientation among the students. Interns had a good knowledge on occlusal plane orientation and implicated it during the treatment of patients when compared to final years since interns got more of clinical exposure than the final years.

Conclusion: In conclusion many students had a good knowledge on landmarks used in complete denture construction but the same did not reflect in their attitude and practice. It is worthwhile to attempt more of teaching should be given on various landmarks used in occlusal plane orientation among the students so that they get to more about the landmarks and implicate in their clinical practice.

Key Words*: occlusal plane, camper line, landmark, esthetics, denture*

Introduction

Occlusion in prosthodontics is extremely important .Occlusal plane orientation is been playing an important role in construction of complete dentures. Complete denture prosthodontics is a challenge for the restoring dentist for the reason that rehabilitation of edentulous patients with conventional complete dentures, be it tissue supported or implant supported, has to be done by considering various biological and mechanical factors to restore function and health of the stomatognathic system [1]. Developing an occlusion that is compatible with functional movements of the stomatognathic system is one of the key factors in determining the prognosis of the completely edentulous patients [2]It is said that for the success of complete denture prosthesis the teeth must be arranged in the correct plane of occlusion. The orientation of occlusal plane forms the basis for teeth arrangement conducive to the satisfactory esthetics and proper function. Occlusal plane is said to be the " plane established by incisal and occlusal surface of the teeth. Occlusal plane orientation can be established by both maxillary and mandibular arch[3]. Occlusal plane helps in phonetics and aesthetics anteriorly whereas in posteriors it forms a milling surface. Any changes in the plane of occlusion will lead to the modification of physical and functional relationship of oral musculature leading to an alteration in function, comfort and aesthetic value. The correct

orientation of the occlusal plane plays a vital role in optimal esthetic achievement. In the natural smile, the incisal tip follow the curve of the lower lip[4]. This effect is an expression of a correctly oriented occlusal plane; if the occlusal plane hangs posteriorly, the lip-line viewed from the front will appear straight and contribute more than any other factor to the so-called 'denture look'. With the occlusal plane correctly oriented, however, the natural anterior curve will be achieved almost automatically and contribute a proper sense of perspective to the dental composition [4].The plane of occlusion, forms an essential part of the concept of mechanically balanced articulation [5]. The position of occlusal plane in denture wearers should be as close as possible to the plane, which was previously occupied by the natural teeth [6]. Such position of the occlusal plane provides normal function of the tongue and cheek muscle, thus enhancing the denture stability [7].

It is believed that teeth oriented on an occlusal plane in harmony with the individuals physiognomy is responsible, in part, all conditions being equal, for stable, retentive full dentures [8]. Faulty orientation of the occlusal plane will affect the interaction between tongue and buccinators muscles [9]. Where the occlusal plane is too high, the tongue cannot rest on the lingual cusps of the lower denture and prevent its displacement. Also it forces the tongue into a new position that is higher than its normal position [10]. This higher position of the tongue causes the floor of the mouth to raise and create undue pressure on the border of the lingual flange and results in partial loss of border seal [11]. There is also tendency for accumulation of food in the buccal and lingual sulci. An occlusal plane that is too low could lead to tongue and cheek biting [12] Hence our goal in treating the patients is to restore the occlusion with free of occlusal interference. In mandibular arch there are few landmarks which could used to orient the occlusal plane like retromolar pad,corner of the lips meanwhile the maxillary arch has a number of landmarks in which the ala tragus line is most commonly used. Considering the importance of the accurate establishment of the location and inclination of occlusal plane on function, esthetics and speech, a method to conform it to the occlusal plane that existed in the natural teeth seems necessary. The question which arises in clinical practice is how to discover which position was occupied by the "natural occlusal plane" after the loss of natural teeth and exactly, how can the optimal position of the occlusal plane be found in every edentulous patient [13].

The occlusal plane in anterior and posterior regions may vary and therefore, these should be evaluated separately. It is generally agreed that in the anterior region the vertical height of the

occlusal plane is governed by esthetic requirements [14] and less frequently by functional demands. The anterior maxillary occlusal plane may be determined by lip relationships at rest and when smiling. Speech also provides for positional accuracy [15]. When viewed from the front, the occlusal plane should be parallel to the interpupillary line [16].With regard to the orientation of the occlusal plane in the posterior region, however, there are contrasting views. Various authors have postulated various landmarks for determining the occlusal plane. The objective of this study is to evaluate the knowledge and attitude of dental students about the landmarks used in occlusal plane orientation and whether they implicate it in their daily practise.

Materials and Method

Study Design

A cross-sectional study was conducted among 100 students of saveetha dental college, divided into two groups (50 final years and 50 interns) Chennai,Tamilnadu. The study was conducted during the month of April 2015 after taking prior informed consent from the students who were willing to participate. Inclusion criteria were the students who have done atleast one prosthetic denture for the patients. In addition, students who where studying final year and internship were only included in the study. Final years and students who were doing internships were only included in the study since they seem to have more exposure on clinical practice when compared to third years as the third years have just entered into clinical practice. Henceforth first year, second year and third year have been excluded in the study.

Methodology

The self-administered questionnaire written in English was adapted from several studies The students were asked to fill in the questionnaire which had 2 set of questionnaires.It had 14 questions, 7questions in the knowledge of landmarks in mandibular arch and 7 questions on landmarks of maxillary arch,and took about 10–15 min to complete. The scoring for knowledge and attitude on landmarks of occlusal plane orientation included yes/NO. This

pretested questionnaire addressed the knowledge regarding the occlusal plane orientation both in static and dyamic relation.

Result

Mandible

Graph 1: Aware that retro molar pad is used in occlusal plane orientation?

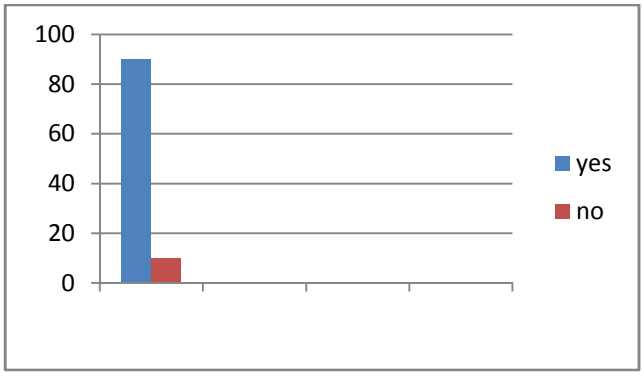


Table 1: depicts that , 91% of students were aware of use retro molar pad landmark in occlusal plane orientation whereas only 9% unware of it. The statistic result showed it is significant at p<.05

Year	Yes	No
Final year	41(45)[0.2]	9(5)[1.8]
Intern	48(45)[0.2]	2(5)[1.8]
Total	91	9

Chi square statistic is 4. P value is 0.0455, result is significant at p< .05

Graph 2: Do you implicate the use of retro molar pad in occlusal orientation in your dental practice?

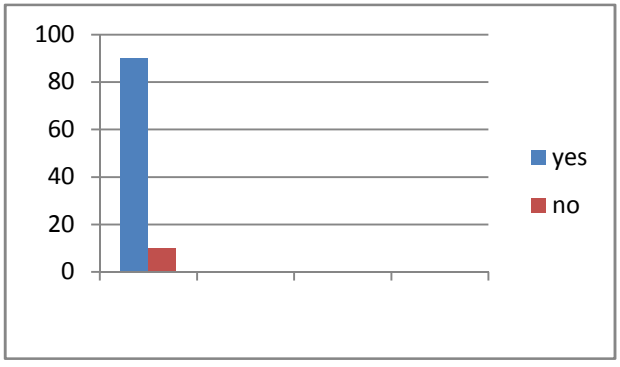


Table 2:Demonstrated, about 90% of students implicated the use of retro molar pad in the dental practice. Interns were more aware and used retro molar pad as landmark for occlusal plane orientation when compared to final years

Year	Yes	No
Final year	39(45)[0.2]	11(5)[1.8]
Intern	48(45)[0.2]	2(50)[1.8]
total	90	10

Chi square statistic is 4. P value is .0455, result is significant at p<.05

Graph 3:Do you think occlusal plane coincide with the middle third of retro molar pad?

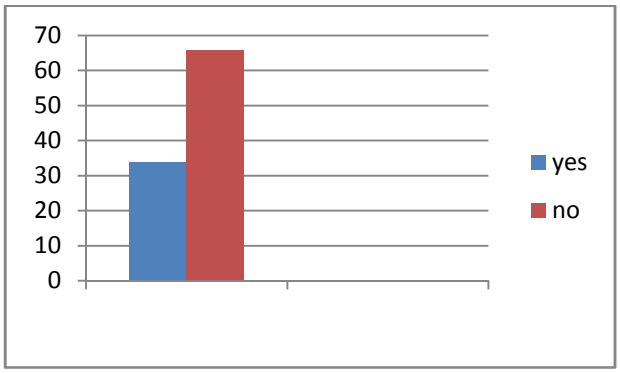


Table 3: Depicts, when asked about occlusal plane coincidence with the middle third of retro molar pad, 90% gave a positive affirmation on it.

Year	Yes	No
Final year	42(45) [0.2]	8 (5) [1.8]
Intern	48(450 [0.2]	2 (5) [1.8]
Total	90	10

Chi square test statistic is 4, p value is .0455. it is significant at <.050

Graph 4:Do you think the lateral border of the tongue reliable for occlusal plane orientation?

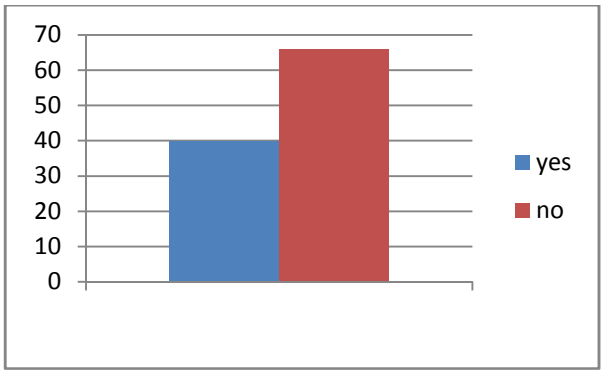


Table 4: Depicted, 66% of the students think lateral border of the tongue is not more reliable in occlusal plane orientation and 34% agreed lateral border of tongue should be used as significant landmark in occlusal plane orientation.

Year	Yes	No
Final year	21(17) [0.94]	29 (33) [0.48]
Intern	13(17) [0.94]	37(33) [0.48]
Total	34	66

Chi square test statistic is 2.852, p value is .091258, result is not significant at $p<.05$.

Graph 5: Do you use commissure of lips for the occlusal plane orientation?

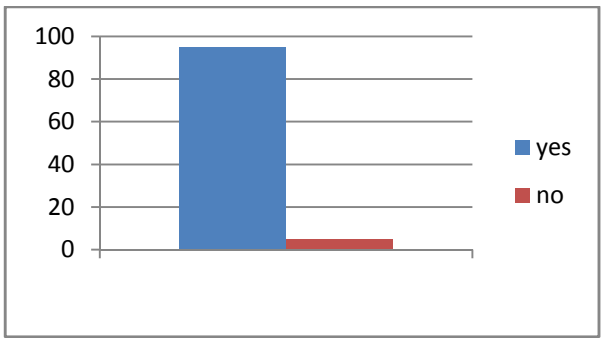


Table 5:Showed positive results towards the use of commissure of lips for occlusal plane orientation, students agreed that commissure of lip is seem to be the main landmark for orientation in mandible

Year	Yes	No
Final year	47 (47.5) [0.01]	3 (2.5) [0.1]
Intern	48 (47.5) [0.01]	2 (2.5) [0.1]
Total	95	5

Chi square test statistic 0.2105.p value is .646355. not significant at p<.05

Graph 6: Is the commisure of lips more reliable for occlusal plane orientation?

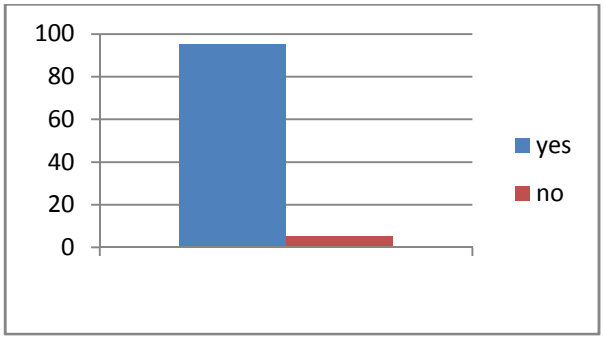


Table 6: Depicted, students agreed that commissure of lip is seem to be the main landmark for orientation in mandible and also most reliable.

Year	Yes	No
Final year	47(47.5) [0.01]	3(2.5) [0.1]
Intern	48(47.5) [0.01]	2(2.5) [0.1]
Total	95	5

Chi square test statistic 0.2105.p value is .646355. not significant at p<.05

Graph 7: Do you use buccinator groove as a landmark in occlusal plane orientation?

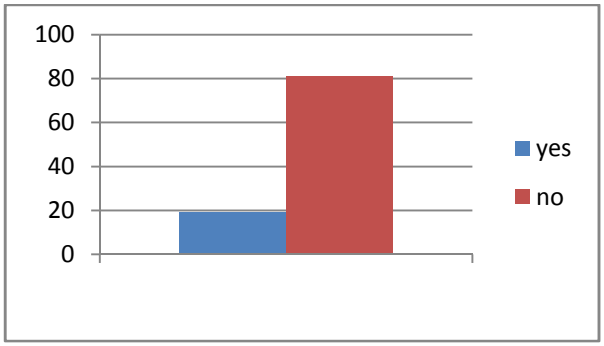


Table 7:Depicts, about 81% of students were unware of the buccinator groove being used as a landmark in occlusal plane orientation.

Year	Yes	No
Final year	8 (9.5) [0.24]	42(40.5) [0.06]
Intern	11(9.5) [0.24]	39(40.5) [0.06]
Total	19	81

Chi square test statistic 0.5848.p value is .4449, not significant at p<.05

Questionnaire 2

Maxilla

Graph 8: Are you aware that parotid papilla is used in orientation of occlusal plane?

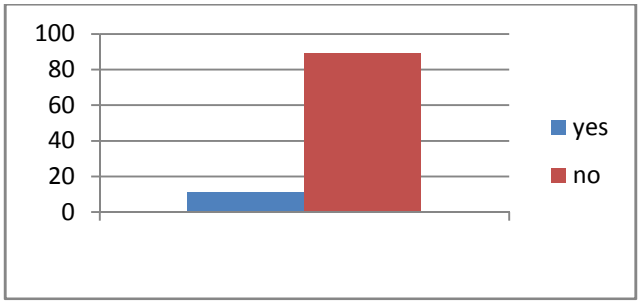


Table 8: Demonstrated, 89% of students believed that use of parotid papilla was not necessary in occlusal plane orientation.

Year	Yes	No
Final year	3(5.5) [1.14]	47 (44.5) [0.14]
Intern	8(5.5) [1.4]	42 (44.5) [0.14]
Total	11	89

Chi square test statistic 2.5538.p value is .1100, not significant at p<.05

Graph 9: Do u think hamular notch is reliable in occlusal plane orientation?

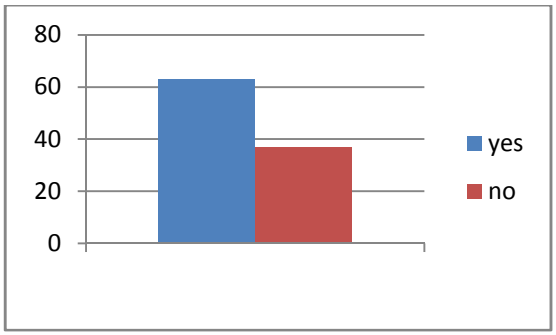


Table 9:Showed, 63% of participant thought hamular notch is reliable in occlusal plane orientation whereas 37% disagreed with it.

Year	Yes	No
Final year	27(31.5) [0.64]	23(18.5) [1.09]
Intern	36(31.5) [0.64]	14(18.5) [1.09]
Total	63	37

Chi square test statistic 3.4749, p value is .0623, not significant at p<.05

Graph 10: Is the parallelism of occlusal plane to ala Tragus line necessary

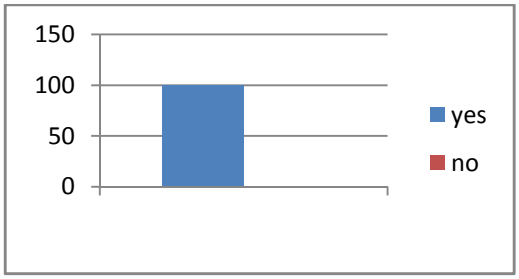


Table 10:Depicts, all the students believed that the ala tragus line is the must when doing complete denture procedure and it should be parallel to the occlusal plane of rim.

Year	Yes	No
Final year	50(50)[0]	0
Intern	50(50)[0]	0
Total	100	0

Chi square test statistic is 0, p value is 1, not significant at <.05

Graph 11: Do you think the occlusal plane orientation is necessary for aesthetics and functioning of denture?

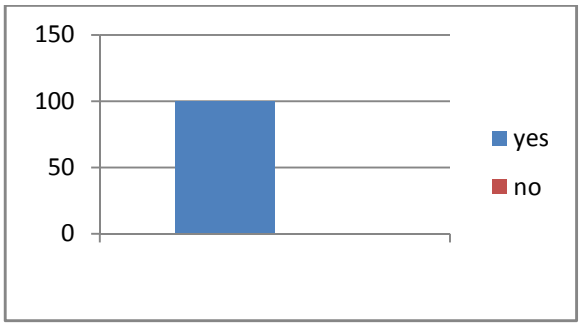


Table 11: Depicts, the main aim for doing occlusal plane orientation is for aesthetics and functioning of denture. All the participants accepted that occlusal plane orientation is a must for aesthetic and functional movement.

Year	Yes	No
Final year	50(50)[0]	0
Intern	50(50)[0]	0
Total	100	0

Chi square test statistic is 0, p value is 1, not significant at <.05

Graph 12: Are you aware that interpupillary line is used in occlusal plane orientation

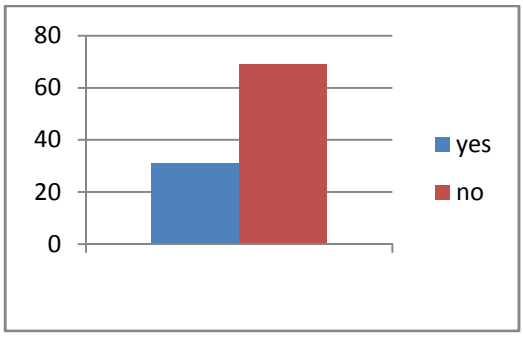


Table 12: Showed mixed response from the participates where few were aware of interpupillary line used in occlusal plane orientation whereas some participants were not aware of it.

Year	Yes	No
Final year	9(15.5) [2.73]	41(34.5) [1.22]
Intern	22(15.5) [2.73]	28 (34.5) [1.22]
Total	31	69

Chi square test statistic is 7.9009, p value is .004941, it is significant to <.05

Graph 13: Are you aware that visibility of maxillary rim should be 2 mm

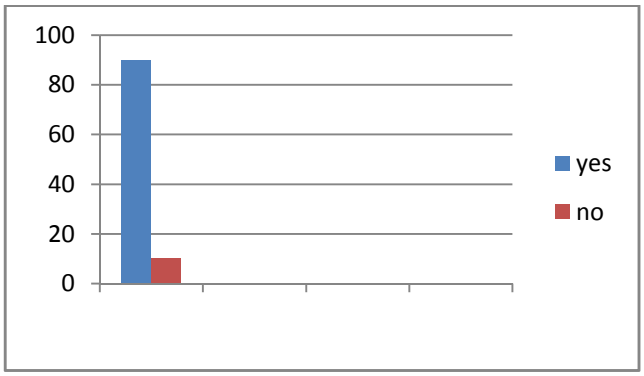


Table 13:Depicted except few, 90% of the participants were aware that maxillary rim should be 2 mm visible when the mouth is in the rest position and it is considered to be significant landmark in occlusal plane orientation

Year	Yes	No
Final year	42(45) [0.2]	8 (5) [1.8]
Intern	48(450 [0.2]	2 (5) [1.8]
Total	90	10

Chi square test statistic is 4, p value is .0455. it is significant at p <.50

Graph 14: Do you implicate in the clinical practise?

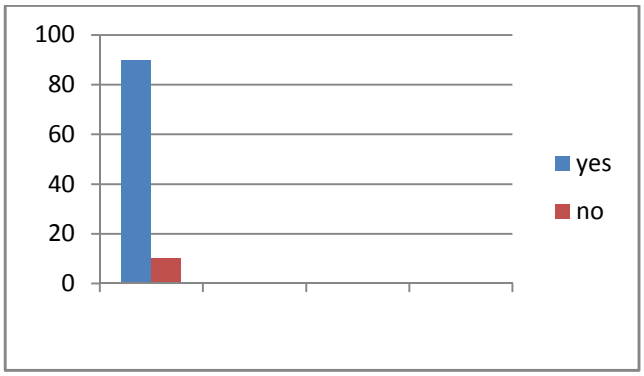


Table 14: Depicted**,** visibility of maxillary occlusal rim about 2mm at rest is must and most students have implicated it in their construction of complete denture.

Year	Yes	No
Final year	42(45) [0.2]	8 (5) [1.8]
Intern	48(450 [0.2]	2 (5) [1.8]
Total	90	10

Chi square test statistic is 4, p value is .0455. it is significant at p <.50

Discussion

The artificial replacement of lost teeth is generally desired for two prime reasons: esthetic and restoration of function. Prosthodontists are often confronted by edentulous patients complaining about ever increasing facial wrinkles and protruded chin with difficulties in function of mastication and speech. They have to limit the routine activities within the confines of anatomical, physiological and functional harmony, compensating for his losses and providing with optimum esthetic and function.[17] Such rehabilitation requires ideal positioning of teeth so that they appear natural and create a pleasing profile and yet, are conductive to function, phonetics and mastication. This in turn is possible only if the occlusal plane which forms an indispensable part of the prosthesis is correctly oriented vertically in the neutral zone[18].The Occlusal Plane orientation for the best esthetics and function has been of great concern to Prosthodontists because of its role in preservation and prevention of residual ridges. The stability of complete denture largely depends on the Occlusal Plane which is one of the most important factors that determines the success of removable prosthesis[19]. Considering the importance of the accurate establishment of its location and effect of its inclination on function, esthetics and speech, a method to guarantee its conformity with the Occlusal Plane of the missing teeth seems necessary. The orientation of Occlusal Plane has led to innumerable controversies.

Graph.2 demonstrated , about 90% of students implicated the use of retro molar pad in the dental practice and believed that occlusal plane coincided with the middle third of retro molar pad. Interns were more aware and used retro molar pad as landmark for occlusal plane orientation when compared to final years. In contrast to this a study done by Gupta et al used a metallic scale which passed along the cusp tip of cuspid to mandibular second or third molar and extended posteriorly to the retromolar pad. They concluded that in maximum

number of cases the occlusal plane coincided with the middle third of the retromolar pad. it is a soft tissue area and at times its anterior and posterior borders cannot be demarcated accurately[20]. Therefore relying only on this one landmark cannot guarantee complete validity to the occlusal plane orientation. However, when used as one of the guides for the occlusal plane orientation, the area in the lower third of the retromolar could be used.

Graph.4 depicted, 66% of the students think lateral border of the tongue is not more reliable in occlusal plane orientation and 34% agreed lateral border of tongue should be used as significant landmark in occlusal plane orientation. A study done by Ghosn et al suggested that since the tongue is a highly movable organ in the oral cavity and thus its reliability in the occlusal plane orientation is questionable. Also, after extraction of all teeth the tongue becomes hypertrophied, resulting in the change of the anatomy of the lateral border of the tongue[21].

Graph.6 depicted, students agreed that commissure of lip is seem to be the main landmark for orientation in mandible and also most reliable. A study done by Shigli et al suggested that Deciding on the commissure of lips as a guide for occlusal plane orientation is most unreliable, as with age there is drooping or turning down of the corners of the mouth. Therefore, commissures cannot be similar to as observed in dentulous patients[22].

A number of guides for its orientation have been implicated. These are Anthropometric, anatomic, biometric landmarks like Camper's plane and retromolar pad. Most of the Prosthodontist encountered difficulty in scheming an Occlusal Plane which is mechanically as well as esthetically acceptable.Ala-Tragus Line is one of the most popular methods is to orient the occlusal plane parallel to a line drawn from the lowest point of the ala of the nose to the external auditory meatus or tragus. Definitions of the ala-tragus line by different authors are a cause of confusion due to disagreement on the exact point of reference, on the ala and the tragus, for this line. Clapp in 1910 was the first to relate Camper’s line/plane to occlusal plane The Camper's Occlusal Plane was the first effort in modern dentistry to establish occlusal plane. It was based on scientific research and was made parallel to ala-tragus plane posteriorly. [23]

Kumar et al. found ala-tragus line is the most controversial landmark in occlusal plane orientation. However, this extra-oral landmark is reliable as both its ends do not change with age. Majority of the studies show that the line joining the inferior portion of the tragus with

the ala of the nose is more often than not parallel to the occlusal plane. Nonetheless, the superior and middle part of the tragus cannot be completely overruled as the lines formed with these landmarks and the ala of the nose have also proved to show parallelism with the occlusal plane. Therefore, a combination of more than one landmark should be used along with the ala-tragus line for orienting the occlusal plane[24].Although clinical judgment and experience are invaluable, method of making a systemic analysis of the Occlusal Plane to the craniofacial structures using cephalometrics would be a definitive aid. This would be especially true for completely edentulous patients since many of the existing landmarks have been lost. So as the location of Occlusal plane in complete denture fabrication is very subjective and it is widely variable depending upon the uncertainty of reference landmarks and the individual judgment. Therefore, the reliability of various reference planes as a clinical guideline for establishment of Occlusal plane needs to be tested.

Limitation of the Study

The design of the study was cross sectional in nature. The students participated in this study were final years and interns so the application of the results of this study to the general dental practitioner and post graduate could not be appropriate. The small sample size is also a limitation of the study.

Conclusion

This study throws a light that students must know various landmarks are available for the orientation of occlusal plane in edentulous patients. More emphasis must be given in teaching various landmarks used in occlusal plane orientation among the students.However, based on the various studies done on dentulous patients, no single method seems entirely accurate to locate the occlusal plane. Therefore, using one method alone will not suffice the determination of occlusal plane in edentulous patients. A combination of various landmarks along with a judicious clinical judgment should be taken into account for the location of the occlusal plane in edentulous patients**.**

Corresponding Author

Radhika Arjunkumar

Reader, Department of Periodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai-600077, Tamil Nadu, India

Email id: radhikaarjunkumar@gmail.com

Telephone Number:+919884281681

References

1. Palmer CA. Gerodontic nutrition and dietary counseling for prosthodontic patients. Dent Clin North Am. 2003;47:355–71.

2. Winkler S. Essentials of Complete Denture Prosthodontics. 2nd ed. Delhi: AITBs Publishers; 2000;41;200-52

3. Boucher CO. Prosthodontic Treatment for Edentulous Patients. 9th ed. Delhi: CBS Publishers; 1990;39;319-470

4. Lang BR. Complete denture occlusion. Dent Clin North Am. 2004;48:641–65. vi.

5. Beck HO. Occlusion as related to complete removable prosthodontics. J Prosthet Dent. 1972;27:246–62.

6. Levin B. A re evaluation of Hanau's Laws of Articulation and the Hanau Quint. J Prosthet Dent. 1978;39:25

7. Zarb GA, Bolender CL. Prosthodontic Treatment for Edentulous Patients. 12th ed. New Delhi: Mosby; 2004;12;102-209

8. Sheppard IM, Sheppard SM. Denture occlusion. J Prosthet Dent. 1968;20:307–18

9. Brewer AA, Hudson DC. Application of miniaturised electronic devices to study of tooth contact in complete dentures, a progress report. J Prosthet Dent. 1961;11:62–72.

10. Rahn AO, Heartwell CM. Textbook of Complete Dentures. 5th ed. Philadelphia: Lea and Febiger; 1993;14;289-521

11. Swenson MG. Complete Dentures. 1st ed. St. Louis: C. V. Mosby Co; 1940;15:382-708

12. The glossary of prosthodontic terms. J Prosthet Dent. 2005;94:10–92.

13. Sharry JJ. Complete Denture Prosthodontics. 3rd ed. St. Louis: Blaktison Publication; 1974:45:78-105

14. Gibbs CH, Mahan PE, Lundeen HC, Brehnan K, Walsh EK, Holbrook WB. Occlusal forces during chewing and swallowing as measured by sound transmission. J Prosthet Dent. 1981;46:443–2

15. NadhirahFaiz, Marian Anand Bennis and Dhanraj.Knowledge and attitude of dental students' preparedness in performing clinical procedures after a fixed Prosthodontics course.Ijcr2016;33;495-503

16.Sloane RB, Cook J. A guide to the orientation of the plane of occlusion.J Prosthet Dent 1953;3:53-65.

17. Monteith BD. A cephalometrically programmed adjustable plane: New concept in occlusal plane orientation in complete dentures. J Prosthet Dent 1985;54:388-94.

18. Monteith BD. Evaluation of cephalometric method of occlusal plane orientation for complete denture. J Prosthet Dent 1985;54:36642

19. NurulSyamimibintiMohdAzlan Sunil and PreethamNittla. Review on various complete denture technique.Rjpt. 2016;13:89-165

20. Gupta RH, Singh SP. Relationship of anatomical landmarks with occlusal plane. J Indian Prosthodont Soc. 2009;9(3):142–147.

21. Ghosn CA, Zogheib C, Makzoume JE. Relationship between the occlusal plane corresponding to the lateral borders of the tongue and the ala-tragus line in edentulous patients.J Contemp Dent Pract. 2012;13(5):590–594.

22. Shigli K, Chetal B, Jabade J. Validity of soft tissue landmarks in determining the occlusal plane. J Indian Prosthodont Soc. 2005;5:139–145

23. Clapp GW. Mechanical side of anatomical articulation. New York: The Dental Digest; 1910:28:279-521

24. Kumar S, Garg S, Gupta S. A determination of occlusal plane comparing different levels of the tragus to form ala-tragal line or Camper's line: a photographic study. J Adv Prosthodont. 2013;5(1):9–15

Evaluation of Awareness among Dental Students and General Dental Practitioners towards Prevention and Management of Dry Socket- A Questionnaire Study

Benazir Hussain M[1], Mimansa Bhoj[2], Sivakumar M[3]

Graduate Student[1],Senior Lecturer[2], Department of Oral and Maxillofacial Surgery, Senior Lecturer[3], Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

Abstract

Introduction: Alveolar osteitis is a post-operative complication that interferes the healing process after tooth extraction. This occurs when a blood clot dissolves and alveolar bone exposure happens. These complications are usually associated with the impacted 3rd teeth. Since it is a most common complication that occurs after extraction, it is very essential for the dental students and dental practitioners to know the importance of dry socket**.**The aim of this study was to evaluate knowledge and awareness on prevention and management of dry socket among dental students and general dental practitioner.

Materials and Method:A cross- sectional questionnaire survey was conducted among 100 dental students (inclusion criteria- undergraduates -final years and interns) and 100 general dental practitioners in chennai. A short pretested questionnaire of 15 questions was given to them .The questionnaires were prepared and data collected using a self- administered questionnaire addressing various aspects of knowledge on prevention and management of dry socket among dental students and dental practitioners.

Result: This study revealed that knowledge about the prevention and management of dry socket among most of the general dental practitioners was adequate when compared to undergraduate dental students.

Conclusion: More emphasis must be given on the prevention and management of complications in exodontia such as dry socket.

Key Words*: dry socket, eugenol, chlorhexidine, mandible, curretag*

Introduction

The most common procedures which is been carried out in dental clinics and the most frequently done task at oral and maxillofacial surgery clinics is the extraction of teeth. This procedure is frequently followed by complications in the mandible incase of wisdom teeth[1]. Extraction of wisdom teeth is most commonly followed by complications in the mandible, which includes both iatrogenic(caused by the clinician) (e.g., nerve injury, bone fractures, etc.) and inflammatory ones, such as dry socket, postoperative pain, delayed wound healing, postoperative infection, hematoma, swelling and trismus etc[2]. Although the overall complication rate might be generally low, and most complications are minor, this surgery is so frequent that the population's morbidity of complications may be noticeable; thus, identifying methods to control or reduce them is a major concern. Besides, not all complications are rare. There are frequent and debilitating complications as well, including postoperative pain[3].

Pain is also one of the most common postoperative complications of extraction and might be caused by the release of pain mediators from the injured tissues. Pain is an important factor in clinical practice and could even discourage patients from seeking dental treatment. It begins after the anesthesia subsides and reaches its peak levels during the first postoperative day. If dry socket or infection occur, the onset of inflammation will complicate alleviation of postoperative pain[4].

Dry socket is known to be the most common complication of exodontia . It is also referred as alveolar osteitis ,postoperativealveolitis, alveolalgia, alveolitissicca dolorosa, septic socket, necrotic socket,andfibrinolyticalveolitis.This occurs when a blood clot dissolves andthe exposure of alveolar bone happens. Dry socket has following symptoms such as severe and progressive pain, halitosis, regional lymphadenitis which usually occurs 3 to 5 days after extraction.[5]

The incidence of dry socket is most common in mandible when compared to maxilla. Dry socket occurs mostly due to dislodgement of clot, bacterial infection, trauma, smoking, curretage of alveolus.[6] It shows increased fibrinolytic activity and activation of plasminogen to plasmin in the presence ofactivators in dry sockets. This fibrinolytic activity is affects the integrity of the post extraction blood clot.[7]However, prevention is very effective in dry socket. Identification of risk factors and their elimination using antibiotic prophylaxis had resulted indecrease in the incidence of dry socket.[8]

The occurrence of dry socket in an day to day oral surgery either dental practice is unavoidable. The most common risk factors of dry socket are smoking, surgical trauma, extraction of wisdom teeth,single extractions, age, sex, medical history, systemic disorder, extraction site, amount of anaesthesia, operator experience, antibiotics which we use prior to surgery, rate of difficulty of the surgery and the previous surgical site infection in addition to oral Contraceptives, menstrual cycle and immediate irrigation of post extraction site with normal saline.[9] The proper options of treatment are directed toward palliative care, such as the irrigation of the surgical site, avoiding curetting the extraction socket[10]. Packing with a zinc oxide– eugenol paste on iodoform gauze on the extraction site can be considered to relieve acute pain episodes, there is also new agents in the market which helps to accelerate the healing of the socket such as PRGF and GECB[11].

The prevention methods for dry socket include avoiding smoking before and after tooth extraction and a traumatic surgery, the use of antibiotics, such as, azithromycin, can be considered, the other preventive measures such as chlorhecidine rinse or gel and zinc oxide eugenol pack with iodoform gauze can be effective in the reduction of dry socket incidence[12].

The aim of our study is to assess the knowledge regarding prevention and management of dry socket among undergraduate dental students and general dental practitioners in chennai.

Materials and Method

Study Design

A cross-sectional study was conducted in Saveetha dental college Chennai,Tamilnadu among final years, interns and general dental practitioner in Chennai. The study was conducted during the month of April 2016 to October 2017 after taking prior informed consent from the students and practitioners who were willing to participate. Inclusion criteria were the students studying final year and doing internship in Saveetha dental college and dental practitioners who work in private sector in Chennai.

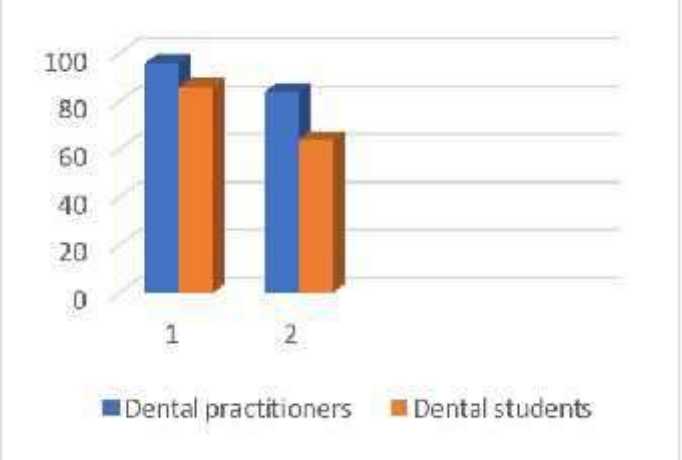
Methodology

This study was conducted to assess the knowledge of prevention and management of dry socket among the undergraduate dental students who were attending the Final year,

internship and general dental practitioners in chennai. 100 dental students and 100 dental practitioners were randomly enrolled in the study, who were present at the day of study. A validated short re-tested questionnaire of 14 questions was distributed among all the students and dental practitioners of the study. The questionnaire was designed at the aim of collecting the data about the prevention and management ofdry socket. Data extracted were statistically analyzed and results obtained.

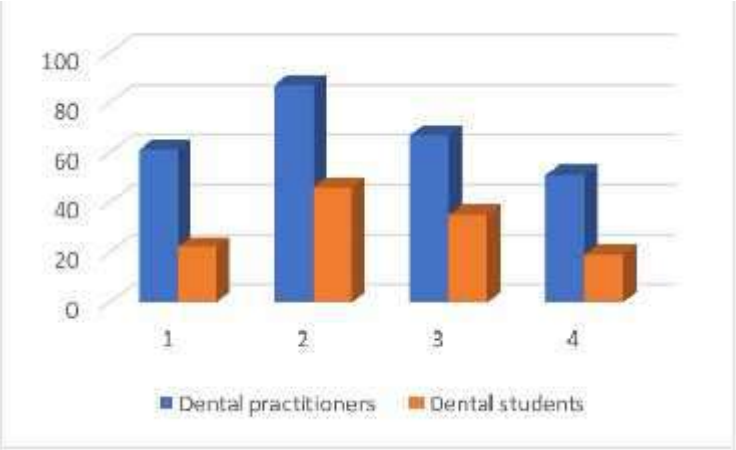
Result

Graph 1: Awareness about dry socket among dental students and dental practitioners



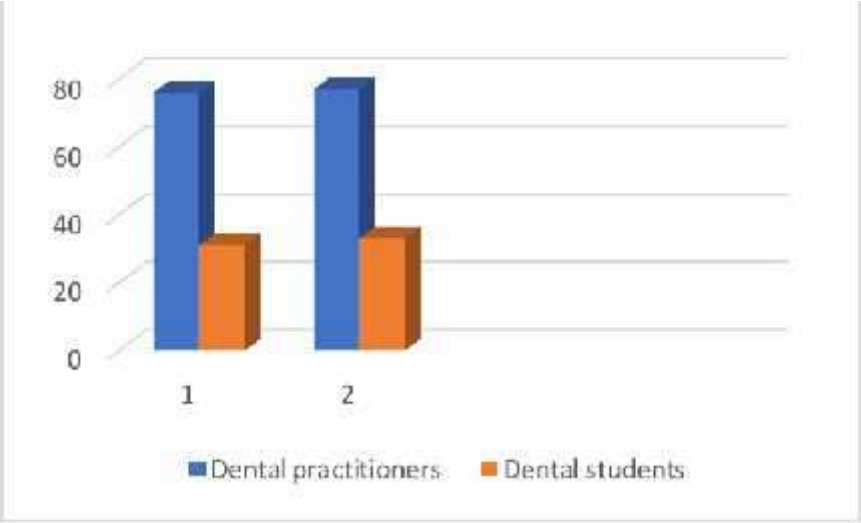
In this study, 90% of general dental practitioners knew that dry socket is a post operative complication of extraction and about 80% of the practitioners were aware that main cause of dry socket is due to dislodgement of clot (graph .1).

Graph 2: Awareness among dental students and practitioners towards management of dry socket



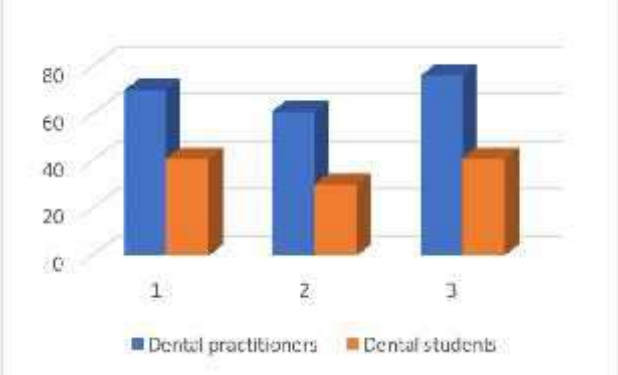
This study revealed that ,57% of dental practitioners used tranexa and chlorexhidine for the management of dry socket whereas only 33% of dental students had adequate knowledge on management of dry socket using tranexa and chlorexhidine. According to the survey 82% of practitioners believed that use of sutures and local haemostatic doesn't not help in management of dry socket. 62% dental practitioners had knowledge on effective management of dry socket using alvogyl when compared to dental students.(graph.2)

Graph3: Awareness towards advancement in management of dry socket



Anti-microbial photodynamic therapy and the low level laser therapy are known to be advancement in management of dry socket. Only 30% of dental practitioners were aware of advancement in management of dry socket and about 70% of dental students had inadequate knowledge on advancement therapy.(graph.3)

Graph 4:Awareness towards prevention of dry socket



70% of practitioners were aware that TTC and 0.2% of chlorexidinewas used for the prevention of dry socket. 85% of dental practitioners believed that induction of fresh bleeding is a proven modality for prevention of dry socket. Dental students had lack of knowledge on prevention of dry socket when compared to dental students.(graph.4)

This study revealed that knowledge about the prevention and management of dry socket among most of the general dental practitioners was adequate when compared to undergraduate dental students. This concludes more emphasis must be given on the prevention and management of complications in exodontia such as dry socket.

Discussion

Difficulties and complication of extraction are unpredictable. Taking medical history prior to surgery will make the dentist to deal better with the complications[14]. Dentist must be certain always to follow proper surgical techniques and should know limitations before beginning any extraction [15].The most common complication of extraction is alveolar ostetitis. According birnstudy ,there is increased fibrinolytic activity in dry sockets and the activation of plasminogen to plasmin with the presence of tissue activatorsreveals that increase in fibrinolysis decreased the chance of dissolution of the blood clot before the 2nd day after surgery ,because the blood clot contains antiplasmin , which is to be neutralized before clot dissolution occur. His study revealed that main cause of alveolar ostitis is due to dislodgement of blood clot[16].

According to our survey it revealed 80% of the practitioners were aware that main cause of dry socket is due to dislodgement of clot and awareness among dental students were inadequate when compared to dental practitioners.Chlorhexidine is a bisguanide antiseptic which is used as a preoperative irrigant and mouthwash tend to reduce the quantity of oral microbiota. Field et al(1987)[17] proposed that there was a decreased incidence of dry socket after irrigation of the gingival crevice with 0.2% CHX digluconate when compared with no irrigation or irrigation with saline. It revealed that incidence of dry socket was less when it was irrigated with chlorexidine. Our study proposed that ,57% of dental practitioners used tranexa and chlorexhidine for the management of dry socket whereas about 80% of dental students had inadequate knowledge on management of dry socket using tranexa and chlorexhidine.

Alvogyl is a medicated dressing which has the combination of (eugenol, iodoform and butamen), is used has a topical anesthetic and antibacterial for the management of dry

socket.62% dental practitioners had knowledge on effective management of dry socket using alvogyl when compared to dental students.

According to kaya G. et al[18], study, management of dry socket was significantlybetter in patients who are treated by curettage and irrigation followed by continuous- mode diode laser irradiation . Low level laser therapy also increased the speed of wound healing and reduced inflammation when compared to Alvogyl .It was used after the irrigation of socket as a continuous mode diode irradiation.

According to our study group, 70% of dental students were unaware of low level laser therapy and only 30% dental practitioners had knowledge on advancement therapy. This concludes more emphasization must be given on complication of exodontia such as dry socket.

Ortoluzzi MC et al[19]. observed the incidence of dry socket and they reported that there were higher pain levels and pain persisting longer than two days were observed with more traumatic surgeries, or associated with postoperative complications. Smoking was found to be statistically associated with the development of postoperative complications.

Mohammed H Abu Younis and Ra'ed O Abu Hantash[20] stated that the occurrence of dry socket are considered to be because of smoking, surgical trauma and single extractions are and factors like: age, sex, medical history, extraction site, amount of anesthesia, and operator experience have no effect on the observation of dry socket. The overall frequency of dry socket was 3.2%. Non-surgical extractions showed about 1.7% in incidence of dry socket while it was 15% following surgical extractions. The smokers showed high incidence of dry socket (12%) than in non-smokers (4%). However, this study showed a strong association between the amount of smoking and the dry socket incidence. Single extraction cases showed higher incidence of dry socket(15%),wisdom teeth than in the multiple extraction cases (6%). Age, sex, medical history, extraction site, amount of local anesthesia used in extraction and operators has no role in the occurrence of dry socket.

Eshghpour M et al.,andHasanMomeni[21], did two studies to identify the risk factor & the risk group of dry socket. Eshghpour M, et al[21],reported that the incidence of dry socket was about was 19.14%. Antibiotics, systemic disorder,age,gender which is been used prior to

surgery has no associations with alveolar osteitis significantly but the dry Socket incidence was significantly associated to smoking, difficulty index of the surgery according to pre-surgery radiograph evaluation and surgeons perception on post-surgery, length of surgery, and number of carpules which has been used to reach anesthesia, HasanMomeni, et al,stated that the incidence of dry socket was found to be more common in female than male which shows more predilection among females towards dry socket. 2.5 to 1 was ratio between mandible to maxilla and most common teeth involved is mandibular third molars than other teeth. Trauma, poor oral hygiene and smoking facilitate to increase the incidence of dry socket.Haraji et al[22], reported that the Alveolar Osteitis is decreased by the modified triangular flap more than the buccal envelope flap. In this study the patients where examined who were candidates for extraction of a bilaterally impacted mandibular third molar with the same difficulty index. In these patients a modified triangular flap was placed on one side and a buccal envelope flap (control) was placed on the other side,wound healing and dry socket were assessed at three and seven days after surgery.

Halabí D et al.[23], stated an increased risk of alveolar osteitis is most commonly associated with that the previous surgical site infection, traumatic extraction, and tobacco smoking

Al-Sukhun J et al[24].compared the use of the selective cyclooxygenase-2 (COX-2) inhibitor celecoxib among the patients, pre-emptively, and the patients who use the ibuprofen.The reported that the ibuprufen group had incidence of dry socket higher than the celecoxib group and the placebo group.

Rodrigues MT et al[25]. studied the effect of experimentally induced infection (the inoculation material contain *Capnocytophaga ochracea, Fusobacterium nucleatum, Prevotellamelaninogenica, Streptococcus anginosus, Treponemasocranskii* and *Streptococcus sanguis*) in the rat sockets, they reported that, it produced higher levels of serum C- reactive protein and showing the potential of disseminated infection and disturb in the alveolar repair process in an interesting experimental model for alveolitis studies. Krakowiak PA reported that; in certain patients, the normal process of healing can be delayed in some cases, because the sites was previously affected by osteomyelitis.

Another similar study emphasised that at the end of extraction on the development of dry socket that is alveolar osteitis after removal of impacted mandibular third molars use of

socket irrigation with a normal saline solution which is been routinely used showed significant differene in dry socket incidence [26](77.8% on the irrigated versus 22.2% on non-irrigated side) which revealed between the extraction protocol versus modified approach without any end-of-surgery irrigation. The study indicated that the post extraction socket bleeding is very important for the proper uncomplicated socket healing. If it's not washed away with irrigation solution at the end of extraction, the normal blood clot has a higher likelihood to form, and therefore, can potentially lead to an uncomplicated socket healing without development of alveolar osteitis.

Treatment option:Vessal G et al., and BezerraTP[27],studied that the dry socket were managed by antibiotics, and they reported that the amoxicillin was the most commonly used antibiotic. This study also reported that the multiple suture groups showed longer operation time, there was less pain, swelling and trismus significantly in the suture-less group, and significant difference between the two treatment groups in terms of pain, swelling and trismus was lesser. They also showed that the local hemostatic, primary closure, sutures and tranexamic acid showed lesser risk of postoperative bleeding after tooth removal in patients on continued warfarin medication.

In other study Kaya G. et al[18], compare effects of alvogyl, the SaliCept patch, and low-level laser therapy in the management of dry socket and the result showed no significant differences in the management of alveolar osteitis among the patients that treated by curettage and irrigation followed by alvogyl applied directly to the socket and the patients that treated by curettage and irrigation followed by a SaliCept patch applied directly to the socket. Patients treated by curettage and irrigation followed by continuous-mode diode laser irradiation showed better management of dry socket more than the patients who treated by curettage and irrigation alone- curettage and irrigation followed by alvogyl applied directly to the socket- curettage and irrigation followed by a SaliCept patch applied directly to the socket.

Burgoyne CC et al[28], assessed use of eugenol on a gauze strip versus a thermosetting gel containing 2.5% prilocaine and 2.5% lidocaine towards the efficacy of pain control for post extraction alveolar osteitis and he reported that the efficacy of the two preparations was not significantly different.

Conclusion

This study revealed that knowledge about dry socket prevention and management among most of the undergraduate dental students was inadequate when compared to general dental practitioners. Patients can attain good quality of treatment if our future dentist have more knowledge on post extraction complications and the methods of preventing and managing the complication.

Corresponding Author

Sivakumar M

Senior Lecturer, Department of Oral and Maxillofacial Surgery

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Telephone Number- +919443602627

Email Id: drsivaomfs@gmail.com

References

1. Hugh W. MacMillan**.** Diagnosis and Treatment of Post-Extraction Complications, The Journal of the American Dental Association 1930;70-8.
2. NivedhitaDalmia.Dry socket - a post extraction complication. 2015;12:34-104
3. Kar IB, Mishra N, Sethi AK, Mahavoi BR. Unusual case of post extraction bleeding. National Journal of Maxillofacial Surgery. 2011;2(2):204-6.
4. Guralnick W, Berg L. GELFOAM in oral surgery. Oral Surg. 1948;1:629-32.
5. Jenkins HP, Janda R, Clarke J. Clinical and experimental observations on the use of gelatin sponge or foam. Surgery. 1946;20:124-132.
6. 6.Correll JT, Prentice HR, Wise EC. Biologic investigations of a new absorbable sponge. Surg Gynecol Obstet. 1945;181:585-9.
7. Council on Pharmacy and Chemistry. Absorbable gelatin sponge: new and nonofficial remedies. JAMA. 1947;135:921.

8. Jenkins HP, Janda R**.** Studies on the use of gelatin sponge or foam as a hemostatic agent in experimental liver resections and injuries to large veins. Ann Surg. 1946;124:952-961.
9. Anand K. Sajnani ,Nigel M. King. Complications associated with the treatment of impacted maxillary canines Singapore dental journal December 2014vol 35:53-7.
10. Butler DP, Sweet JB. Effect of lavage on the incidence of localized osteitis in mandibular third molar extraction sites.Oral Surg Oral
11. Catellini JE. Maxiallary molar extraction. Med Oral Pathol Oral Radiol Endod 1977;44:14-2[11].Review of factors contributing to dry socket through enhanced fibrinolysis.J Oral Surg. 1979;37:42-46
12. Birn H**.** Bacteria and fibrinolytic activity in dry socket Acta Odontol Sand 1970;28:773
13. Houston JP , McCollum J , Pietz D , Schneck D. Alveola osteitis :a review of its etiology , prevention and treatment modalities . Gen Dent 2002;50:457-63
14. Birn H. Fibrinolytic activity of normal alveolar bone. Acta Odontol Scand 1971;29:141
15. Stein**.** Fibrinolytic activity of normal alveolar bone. Acta Odontol Scand 1972;23-32
16. Daly B , Sharif MO , Newton T , Jones K , Worthington HV. Local interventions for the management of alveolar osteitis .2003;23:41-190
17. Field. Post extraction management. J Pharm. 1987;12:34-10
18. kaya G. Diagnosis and Treatment of Post-Extraction Complications, The Journal of the American Dental Association 1998;23;70-102
19. OrtoluzziMC .Dry socket - a post extraction complication. J Pharm 2015;23:42-104
20. Mohammed H Abu Younis ,Ra'ed O Abu Hantash.Unusual case of postextraction bleeding. National Journal of Maxillofacial Surgery.2011;2(2):204-6.
21. EshghpourM ,HasanMomeni.Clinical and experimental observations on the use of gelatin sponge or foam. Journal of Surgery. 1946;20:124-132.
22. Krithika P, Dhanraj M. Effectiveness of local hemostatic agents following dental extraction: A systematic review. Sper. 2016;181:585-9
23. Halabí.Absorbable gelatin sponge: new and nonofficial remedies. JAMA. 1947;135:921.

24. Al-Sukhun J.Studies on the use of gelatin sponge or foam as a hemostatic agent in experimental liver resections and injuries to large veins. Ann Surg. 1946;124:952-961
25. Rodrigues M. JAMA Control of bleeding after dental extraction. 1961;175(11):962-6
26. Santhosh Kumar MP, Lavanya. Knowledge about Post Extraction complication. J Pharm 2016; 8(6): 47
27. Svensson R, Hallmer F.Gelfoam in oral surgery; a report of 250 cases. Oral Surg Oral Med Oral Pathol 1948; 1: 629-32.
28. Burgoyne CC.Clinical and experimental observations on the use ofgelatin sponge or foam. Surgery 1946; 20:124-32.

Evaluation of Internship Programme of Undergraduate Students in A Private Dental College

Sahana Kritivasan[1], Pradeep Kumar[2], Shabeenataj[3]

Graduate Student[1], Reader[2], Department of Public Health Dentistry, Tutor[3], Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

Abstract

Introduction: Dental education is at a critical state due to advances in health care delivery system. In dentistry one can gain extensive amount of knowledge only when they are students.The aim of this study is to assess the Internship programme of undergraduate students of a private dental college in Chennai.

MaterialsandMethod: A cross-sectional study was conducted among undergraduates. A pretested questionnaire was used to assess the internship experience like critical thinking knowledge,problem solving skills .senseof responsibility,creativity,professionalattitude, appearances, quality of work and ethics. Descriptive were analysed.

Results: It shows that about 72.3% of interns were satisfied about their internship achievements.

Conclusions: The study concludes that the internship programme have built in the clinical competency of the interns.

Key Words*: internship, self evaluation, learning, communication, curriculum*

Introduction

Dental curriculum changes dynamically hence it needs to be monitored and reconstructed according to the needs of the student, community and profession, otherwise it becomes stagnant.According to Shetty V.B, a good curriculum should always restructures itself and should receive feedback from all stakeholders. [1] The dental education is at a critical stake due to the advances and changes in the health care delivery system. There is a wide belief that the dental students are not satisfied with their experience in the dental colleges which may be due to the stressful environment. [2]Psychological stress or distress has long been regarded as an important influence on the learning and performance of the students. It serves as a motivation to some portion of the student community and for the rest it has adverse negative effect on their studies, health and personal lives. [3]

Being a consumer of the dental programme, the students view must be considered by the curriculum evaluation committee while updating the dental curriculum [4]. A self assessment can be conceived as an ingrained habit or trait to reflect an individual. Students of dental profession are bright; goal oriented and is reflective, sightful and consistent in academic and in assessing their skills and ability. [5].Hence the study was designed to analyse the perception of the interns about the present scenario of dental education, clinicaltrials, teacher-student relationship, professional outlook and patient care.

Materials and Method

A pre-tested structured questionnaire were given to 65 students of Saveetha Dental College and Hospitals, Chennai who were undergoing the internship programme. The feedback of the interns regarding the questionnaire were gathered and the data collected were entered for further evaluation. Evaluation of the data was done and the output was obtained.

Result

The results showed an overall satisfaction in achieving the internship roles and responsibilities along with performance(table 1), organisation and working environment(table 2) and finally the academic portfolio and extra curricularsactivites(table3).

Table 1: Internship Roles and Responsibilities with Performance

Items	Strongly disagree %	Somewhat disagree %	Neither agree nor disagree %	Somewhat agree %	Strongly agree %
Achievement of internship objectives	4.6	6.2	16.9	53.8	18.5
Demonstration of communication skill	3.1	13.8	6.2	49.2	27.7
Application of classroom knowledge	4.6	7.7	20.0	40.0	27.7
Critical thinking and problem solving skill demonstration	7.7	7.7	15.4	49.2	20.0
Professional appearance	4.6	4.6	9.2	53.8	27.7
Response to supervision and criticism	6.2	9.2	20.0	44.6	20.0
Self sufficient, independent as well as a team worker capacity	1.5	1.5	15.4	40.0	41.5
Exhibition of responsibility and dependability	3.1	7.7	7.7	50.8	30.8
Exhibition of positive attitude towards work and co workers	3.1	4.6	7.7	36.9	47.7
Exhibition of professional attitude to behave ethically	6.2	13.8	6.2	49.2	24.6
Sensitive to diversity	6.2	12.3	23.1	40.0	18.5
Adaptation to changing	3.1	3.1	16.9	47.7	29.2

circumstances					
Sufficient quantity and quality of work production	6.2	6.2	18.5	40.0	29.2
Demonstrated awareness of strength/ weakness	1.5	9.2	7.7	52.3	29.2
Was comfortable handling all assigned responsibilities	6.2	0.0	20.0	49.2	24.6

Table 2: Organisation and working environment

Title	Strongly disagree %	Somewhat disagree %	Neither agree nor disagree %	Somewhat agree %	Strongly agree %
Providence of sufficient orientation to its mission, purpose, culture,policies and practices	10.8	1.5	13.8	56.9	16.9
The work environment was stimulating	7.7	12.3	13.8	46.2	20.0
The organisation invited me to provide feedback and input on the work in which I was engaged and to discuss organisational matter	13.8	10.8	16.9	43.1	15.4
The work was challenging	7.7	3.1	12.3	44.6	32.3
The internship tasks and experiences helped to meet the learning goals and objectives	4.6	1.5	16.9	49.2	27.7

Providence with access and insight into a variety of activities to contribute to the learning	4.6	3.1	15.4	50.8	26.2
Was conscious of my needs as an intern and made clear the task responsibilities	7.7	1.5	18.5	50.8	21.5
Assigned an appropriate amount of work	7.7	10.8	18.5	41.5	21.5
Discussed ways by which learning objectives could be achieved	7.7	4.6	27.7	43.1	16.9
Providence of regular assistance and willingness to answer the queries regarding work setting and specific tasks	4.6	7.7	20.0	50.8	16.9

Table 3: Academic Portfolio and extra curricular activities

Title	Strongly disagree %	Somewhat disagree %	Neither agree nor disagree	Somewhat agree %	Strongly agree %
Academic coursework provided preparations of professional knowledge and skills	6.2	3.1	16.9	46.2	27.7
Comparison helped in modulating the internship programme	7.7	10.8	18.5	44.6	18.5
Usage of CAD CAM, RVG and advances record	7.7	10.8	10.8	41.5	29.2

management system was useful					
Outreach program helped to explore more unknown problems	4.6	7.7	18.5	47.7	21.5
Participation in conferences, seminars and presenting scientific papers and posters provided insight	3.1	3.1	18.5	46.2	29.2
Research work helped in career building	9.2	7.7	24.6	35.4	23.1
Library provided adequate resources for various academic and research purposes	9.2	13.8	20.0	40.0	16.9
Usage of wifi and ipad was beneficial	9.2	10.8	10.8	41.5	27.7
No. Of clinical procedures provided adequate training and professional skills	10.8	9.2	13.8	43.1	23.1
UTSAV provided platform to exhibit hidden talents	6.2	6.2	6.2	24.6	56.9

Discussion

It is very important to consider the perspective of graduating dental students regarding their curriculu, motivation and support. Also it is necessary o assess their internship experience like critical thinking knowledge,problem solving skills.sense of responsibility,creativity,professional attitude, appearances, quality of work and ethics.

The study show the various perceptions of the interns of a private dental institution should that there was overall satisfaction in acheievong the internship roles and responsibilities along with

performance. It also gives us an insight of the strengths and weakness of the current curriculum of that dental college which is monitored regularly.

Table 1 shows that about 49.2% of studnts have necessary communicaton skills. This is in accordance with the study conducted by Lanning S.K et al who quoted that, communication skills learned in the clinical setting, helps in providing a formal approach towards the patient and it is more efficient in enhancing the students confidence[3]. Also 49.2%of the interns have the ability of critical thinking and problem solving skills. About 50.8% of interns exhibit responsibility and defendabilty. About 49.2% were comfortable in handling all the assigned responsilities.

More than 40 percent of the students expressed that, they were able to apply their theoretical knowledge in their clinical practice which is higher than a similar study conducted by Ashri N in King Saud University,Riyadh.[4].

The internship tasks and responsibilities were handled comfortably by about 52.3 percent of the students which reflects that the students have gained adequate confidence to meet their goals and to understand their strength and weaknesses.

A study conducted by Kumar S in Rajasthan highlighted that students had stress due to the fear of their clinical in charge and the environment provided by the faculty to them during their clinical training [8].

The study by Chaudhary S mentioned that the way the practitioner carries himself influences the patients image of him or her[9]. Nearly 53.8 percent of the interns believed in maintaining a good professional appearance.

Nearly 40 percent of the students were elated about being a self sufficient worker as an individual as well as a team worker. A study in UK by Evans J showed the need for the development of a positive attitude towards inter professional education which will help in

maintaining a good team work with the technicians and their colleagues which will lead to a better collaborative practice.[10]

This was furtherillustrated in a study done by Leisnert L in Sweden where the students responded that patient needs to be approached holistically which should be a permanent part of education.[11] About 47.7 percent of the students appeased positive attitude towards their co workers. This helps them to respect each other's role in providing care for the patient.

The Association of American Medical colleges considers ethical responsibility to self and others, reliability, adaptability and dependability as the crucial for effective professional behaviour.[12] Being an intern student who is going to be exposed to private practice soon needs to be aware and exhibit the same. About 50.8 percent of the students exhibited responsibility and dependability during the course work.

Nearly 50 percent of the students exhibited professional attitude to behave ethically and 40 percent of the students were sensitive to diversity at the working environment. Diversity during the clinical practice includes the difference in gender, socioeconomic status, religion of the co workers and patients.

About 47.7 percent of the students were able to adapt to the changing circumstances and 46.2 percent of the interns believed that the working environment was stimulating for them to perform from better to best.

Table 2 shows that about 56.9% agree that the working environment provides sufficient orientation to its mission , purpose , culture ,polices and practices . About 50.8% of the interns were stiulated by regular assistance and willingness of the faculty to clear their queries regarding specific tasks. Attitude of the faculty towards the intern helped in modulating their perspective of achieving their objectives.

Nearly 43.1 percent of the interns were satisfied in providing feedback and discussing organisational matters. Asimilar study was done by Curtis D.A in University of California which the organisation invited its students to a provide a self assessment feedback of preclinical examinations.[13]

The number of clinical procedures done by the interns provided them with adequate training and professional skills. This was strongly agreed by 66.1 percent of the students while in a similar approach done in Riyadh reported only 59.8 percent of its students were satisfied with the quantity and quality of work produced by them in different branches of dentistry that helped them in becoming a self sufficient clinician at the undergraduate level.[4]

Adding on to the work production, selection of proper techniques and giving proper care provides a good orientation to their mission as a clinician. This was also sighted in a study by Marchan S.[6]

A major portion of the study population agreed that the work was challenging and will help them in improving their performance and observational skills during their clinical practice.

Table 3 shows that about 41.4% of the iterns were highl elated with the usage of facilites like like CAD-CAM, RVG, TOUCH ON CLOUD, DENTAL COLLEGE RECORD MANAGEMENT SYSTEM USING IPAD.[15,16,17,18,19]Also it has newer methods of learning and opens out vast areas of resources. These revolutionary technologies enhance the dental practise and education.[18'19]The ciruculam also provides oppurtunites to add credentials to their profile by participating in conferences and seminars. This enourages the to tackle different obstacles that arise while engaged in research work.

A library is an important aspect of every institution but the usage and the importance of it is lacking in almost all the students.Only 40 percentage of the students realised the benefits of the adequate resources found in the library for various academic and research oriented purposes

where as in a parallel study done by Shetty V highlighted its grief of only 35 percent of its students were satisfied with the library resources.[1]

Apart from the academic mission and orientation, the institution also provides an equal opportunity for its students to showcase their talents. By providing a platform for these talents to its students the institution will be able to explore ways to reduce stress among them and increase the efficiency of work produced by them.

Conclusion

This study has investigated the internship program of a private dental college in Chennai. Feedack of the interns highlights the success of the ciriculum. The enormous work produced by the interns helps to adapt to changing environment and overcome the stress. Patients satisfaction, time management and potent assessment becomes fruitful with efficient communication skills. The strength of the current program were a provision to critical thinking , problem solving skills , exhibiting responsibility , providing good working environment and revolutionary computer technologies.

Corresponding Author

Shabeenataj,

Tutor, Department of Public Health Dentistry

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email: - shabeenataj2592@gmail.com,

Telephone Number: +91 9843390930

References

1. Shetty V.B,Shirahatti R.V,PawarP,BDS.Student's Perceptions of Their Education on Graduation from a Dental School in India.Journal of Dental Education.2012;76(11).

2. Ganesh R,Sajida Sultana.N,Naz T.Crossroads of Dental Education:Perception among the Interns of the Dental Schools in Tamil Nadu,India.e-Journal of Dentistry.Jul-Sep 2013;3(3).
3. Lanning S.K,D.D.S;RansonS.L,WilletR.M.Communication Skills Instruction Utilizing Interdisciplinary Peer Teachers:Program Development and Student Perceptions.Journal of Dental Education.2007;72(2).
4. Ashri NA, AlhamadAR, Alnasser LU. Students and recent graduates perspective of dental curriculum at King Saud University, Riyadh-Saudi Arabia. Pakistan Oral & Dent. Jr. 2006;26(2):175-82.
5. Gordon M.J.A Review of the Validity and Accuracy of Self-assessments in Health Professions Training.AcademicMedicine.December 199; 6(12)66-70.
6. MarchanS.M,SmithW.A.J.Self Perceived Clinical Competencies and Proficiencies of Internationally Trianed Dentists at a Non-US Site Completing an Advanced Education in General Dentistry Residency.Open Access Scientific Reports.2012;1(6).13-14.
7. SpegmanA.M,GeisingerCenter of Health Research;Herrin S,DC,Western States Chiropractic College.Chiropractic Interns' Perceptions of Stress and Confidence.The Journal of Chiropractic Education.2007;2(2)21-24.
8. Kumar S,DagliR.J,MathurA,JainM,PrabuD,Kulkarni S.Perceived sources of stress amongst Indian dental students.European Journal of Dental Education 2009;13(4)39-45.
9. ChaudharyS,GowdaT.M,Kumar.T.A.B;Menta D.S .Journal of Dental Education.2013;7(10)77-84.
10. Evans J,Henderson A.The Future of Education and Training in Dental Technology:Designing a Dental Curriculum that Facilitates Teamwork across the Oral Health Professions. European Journal of Dental Education. 2012;2(6)25-29
11. Leisnert L, Karlsson M, Franklin I, Lindh L, Wretlind K. Improving Teamwork between Students from Two Professional Programmes in Dental Education.European Journal of Dental Education. 2012;3(1)17-26.
12. Middlemas S; Haftel H, MD, MHPE; Ross P.T, MA; Lypson M.L,. Assessing Interpersonal Communication Skills. The Use of Standardized Patients. 2009;5(8)54-58.

13. Curtis D.A, Lind S.L, Setia G, Finzen F.C, Dental Students Self Assessment of Preclinical Examinations. Journal of Dental Education. March 2008;7(3)72-75.
14. Barrak A. Al, Yami R Al. Assessment of Skills and Attitude of Dental Students and Interns towards e-Learning in KSU. International Journal of Excellence in Education. September 2013;6(1)65-67.
15. Murthykumar K, Veeraiyan DN, Prasad P. Impact of Video Based Learning on the Perfomance of Post Graduate Students in Biostatistics: A Retrospective Study. Journal of Clinical and Diagnostic Research : JCDR. 2015;9(12):ZC51-ZC53.
16. Renuka S, Gurunathan D. Usage of internet for academic purposes by undergraduate students. Journal of Advanced Pharmacy Education & Research| Jan-Mar. 2017;7(1).
17. Aruna S, Kavitha MS, Thenmozhi MP. EFFECTIVENESS OF SMALL GROUP LEARNING IN PROMOTING STUDENT LEARNING. Research Journal of Pharmacy and Technology. 2015;8:1076-78
18. KadambariSriram and Brundha M.P ' Effectiveness Of Papers 53 App In Teaching Methodology', International Journal of Current Advanced Research, 2017;06(03): 2886-2889.
19. P Thejeswar, E & Thenmozhi, MS. Educational Research-iPad System vs Textbook System. Research Journal of Pharmacy and Technology. 2015;8:1158- 59

Evaluation of Knowledge, Experience and Perception about Medical Emergencies among Dental Graduates (Interns)

M.Indhumathi[1], Shantha Rajan[2], Hemavathy.O.R[3],
Graduate Student[1],Senior Lecturer[2], Department of Pedodontics,Reader[3], Department of Oral and Maxillofacial Surgery,
*Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, I*ndia

Abstract

Introduction: The study is based on effective management of an emergency situation in the dental office . The lack of training and inability to cope with medical emergencies can lead to tragic consequences and sometimes legal action. For this reason, all health professionals including dentists must be well prepared to attend to medical emergencies. Providing basic life support [BLS] is dentist's most important contribution until definitive treatment for a medical emergency can be given. The aim of the study is to evaluate the knowledge, experience and perception about medical emergencies amongst dental graduates (interns)

Materials and Methods: Data were collected from 100 Interns of Saveetha dental college of chennai, India by using a structured questionnaire consisting of 7 item questionnaire (6 closed and 1 open).
Statistical analysis was performed to analyse the study.

Result: Among 100 dental students, who participated in the following study, only 70% of them were aware of the medical emergency drugs and their uses , the confidence level of the students were abruptly good in those students and 30% of the students should improve the level of confidence in using and handling of the drugs.

Conclusion: The study is useful to gain knowledge for the dental students in case of medical emergency situations.

Key Words*: Medical emergencies, dental graduates, basic life support.*

Introduction

Medical emergencies can be alarming to any clinician but these situations are less alarming if proper preparations are made. Medical emergencies occur in dental practice more frequently .Fortunately, serious medical emergencies in dental practice are not common but they are all the more alarming when they occur . A thorough patient history can draw the practitioner's attention to potential medical emergencies that could occur.(1) Changing demographics in the population, leading to increased longevity have led the people having medical conditions which predispose to a medical emergency or taking medication may influence their dental management and persons aged above 65 years or over are considered to be taking medication with a potential effect on dental care . An increasing proportion of the population is medically at risk. According to the European resuscitation council, sudden cardiac arrest is a leading cause of death in Europe, affecting about 7, 00,000 individuals a year .Thus, an effective management of an emergency situation in the dental office is ultimately the dentist's responsibility.(2) Although a number of studies have been carried out which sought to ascertain the emergency drugs and equipments, the lack of training and inability to cope with medical emergencies can lead to tragic consequences and sometimes legation action . For this reason, as health professionals, dentists should be well prepared to collaborate with the medical emergencies. (3) Providing basic life support (BLS) is the dentists' most important contribution until definitive treatment for a medical emergency can be provided .Few studies described how the dentists consider themselves in managing medical emergency situation and very few studies to our knowledge have reported studies involving fresh dental graduates . The aim of this study is to learn the experience of handling medical emergencies, their skills and competency and how well they felt are prepared to manage such events with appropriate use of drugs and equipments in a dental setting.(4)

Material and Methods

A cross sectional questionnaire approach was chosen to probe dental graduates' (Interns) knowledge, experience and perceptions of medical emergency in the dental office. This research was conducted at Saveetha dental college , Chennai, during the academic year 2016. A total of hundred and five (100) Bachelor of Dental Surgery graduates from the respective Saveetha dental college,who were undergoing the

Internshipand who could look back over their experiences regarding the dental-medical emergency were volunteered to participate, with an invitation issued in the college. Respondents were told the study was completely confidential and encouraged to answer a 7 item pretested questionnaire (6 closed ended and 1 open ended) which was in a tick box format.Data collection procedures followed the standards of the Saveetha dental college students and all the graduates (Interns) signed a consent form.(22) The purpose of this study was to evaluate the dental graduates, knowledge, experience, and perceptions regarding medical emergencies in the dental practice. The questionnaire sought information on the frequency and type of medical emergencies encountered by the interns in the past 4 years.The knowledge, confidence of administering the essential drugs and equipments required to be in the emergency drug box (8). The amount of medical emergencies training undertaken by participants on the past 4 years and what percent of this was basic life support training (9). If participants felt that more training of general medical emergencies is required. Data were collected from various aspects.

Results

Hundred dental graduates answered the questionnaire. Sixty-one (61) of hundred and five (58.1%) had experienced an emergency situation during their graduation. Syncope / faint was seen by 40.9% of the respondents, 37.1% with hypoglycemic attacks, allergic reactions by 17.1%, epileptic attacks by 7.6%, asthmatic attacks by 4.5% and angina 0.9%. The frequencies of the emergencies encountered were once or even more. (Table 1 & 2) shows the percentage of dental graduates about the knowledge of identifying the recommended drugs, essential pieces of equipments in the emergency kit and the confidence of using them. (21) Forty- five out of hundred (42.9%) had received no medical emergency training, and the remaining sixty (57.1%) had received a medical emergency and basic life support (BLS) training for less than 5 hours. Hundred and two graduates (97.1%) felt the need for more medical emergencies training. (Table 3) shows the competency in the areas of drug administration/other procedures. From an open ended question, we observed that the knowledge of the dental graduates (Interns) was at an acceptable level as majority of them expressed to terminate the treatment and activate the emergency services (EMS) when any emergency situation in the dental office occurs. (20)

Table 1: Essential drugs recommended being present in the drug kit and the level of confidence in using them

(In percentage)

Drug	**Yes**	**No**	**Unsure**	**Confident**	**Not confident**
Adrenaline	96.4	40.3	-	90.5	30.2
Aspirin	56.2	79.5	36.3	62.5	40.3
Glucagon	45.3	50.3	40.5	70.7	45.3
Glyceryltrinitrate	62.7	48.6	20.6	2.7	38.4
Prednisolone	57.3	59.7	39.5	3.5	60.3
Chlorpheniramine	66.2	49.3	45.6	2.8	50.3
Salbutamol	90.6	21.6	12.9	13.3	23.5
Glucose	80.8	12.4	13.7	89.6	4.6
Hydrocortisone	56.3	11.5	15.9	5.7	89.7
Midazolam	78.4	20.4	34.6	54.7	45.6
Dextrose	98.4	40.3	4.5	80.5	67.3
Oxygen	99.2	3.6	4.8	50.3	4.8
Atropine	90.6	23.5	45.6	3.6	59.6

Discussion

The results of this study confirmed that the dental graduates (Interns) are not capable of competently managing a medical emergency and perceived a need for more intensive education in medical emergencies, and they strongly desire to obtain this knowledge (5,10).Several studies have assessed qualified dental surgeons on management of medical emergencies, (4,11,12,13) but to our knowledge there is no reported study involving fresh Dental graduates (Interns). So this cohort was targeted to probe and quantify the perceptions of medical emergency in the dental office. Although there have been relatively few studies carried out regarding medical emergencies in dental hospitals, our study results show that the proportion of specific medical emergency events occurring were similar with those studies (9,11,14). The most commonly encountered emergencies seen by the Interns in both the colleges were syncope/faints, followed by asthmatic and hypoglycemic attacks.(18)This would indicate that the training should be focused on dealing with these emergencies. A higher frequency sixty one (58.1%) respondents had faced a life threatening situation in the

study contrary to the studies (3,8,11). The reason for differences in the frequency/ total number of emergency could be explained by having several persons reporting the same event occurring in the dental school more than once.(17) There has not been any published data regarding emergency drugs and equipments recommendations against which we can compare our results. The guidelines differ in recommended drug and equipments, but when a direct comparison is made between commonly recommended emergency drugs conclusions can be drawn. We found that respondents in our study had a good knowledge in identifying the four common drugs like adrenaline,glucose, oxygen and glycerlytrinitrate. The knowledge was not at an acceptable level, particularly when discus-sing drugs like Midazolam, prednisolone and chlorpheniramine maleate and very few respondents recognized these as being essential drugs (8,9) (Table 1).Similar results were found when investigating level of knowledge regarding emergency equipments. Single use syringes, oxygen face mask and blood glucose measurement device were recognized to some extent and lowered knowledge was seen regarding equipments like pocket masks, portable suction and self inflating child and adult bag valve mask (9,15) (Table 2).The confidence in the use of drugs and equipments mentioned were at a very lower level than the knowledge for all the drugs and equipments mentioned.(19) This suggests that although training is received in the theoretical aspect of emergencies, participants are not particularly confident to treat emergencies and may require further practical training (Table 1, 2 &3).From the responses regarding the number of hours of medical emergencies training undertaken in the under-graduate curriculum, it is evident that there are more definitive guidelines regarding the number of hours of training is recommended. Only 57% of respondents had undergone medical emergency and basic life support (BLS) training for less than 5 hours which was very low. The result may be due to the lack of definitive guidelines about the training with medical emergencies in the dental curriculum (9).Overall in the study, a large number of graduates stated that they did not know how to proceed in those situations even though they received training in the management of medical emergencies at some time, they expressed the need for further medical emergencies training (3,5,8,9,11,16).

Table 2: Essential equipments recommended being present in the kit and the level of confidence in using them.
(In percentage)

Drug	**Yes**	**No**	**Unsure**	**Confident**	**Not very confident**
Oxygen face mask	95.3	2.1	-	90.3	-
Single use syringe	56.3	46.2	2.5	32.3	30.2
Oropharyngeal airway	76.3	60.3	35.4	40.5	2.3
Pocket mask	65.3	50.2	5.7	21.4	4.5
Self inflating child and adult bag valve mask (Ambu bag)	79.3	32.5	11.3	34.2	12.4
Portable suction	68.5	40.2	2.3	45.3	2.3
Blood glucose measurement device	30.2	80.5	6.2	67.4	25.7
Automated external defibrillator	79.2	30.2	3.2	80.3	18.4

Table 3: Do you feel competent in the following areas.?
(In percentage)

Area	**Yes**	**No**

Administration of intravenous drug	30.5	32.5
Intramuscular injections	50.3	32.3
Maintaining an airway	78.3	23.6
Using an Ambu bag / bag valve mask	45.6	27.3
Using a glucose meter	35.6	78.5
Using a defibrillator	50.3	12.3
Administering oxygen	89.7	20.7

Conclusion

The study showed that syncope is the commonest medical emergency event and the others are hypoglycemic attacks, allergic reactions, epileptic attacks and asthma-tic attacks. All in all, dental graduates had a superficial knowledge of medical emergencies, drugs and equipments and they expect this topic should be an integral part of their curriculum.[10]A majority of them perceived the need for further training by means of hands on courses. The study has allowed to find deficiencies in the way the dentists were trained dealing with medical emergencies and identify a need for improvement, be by increasingthe volume and quality of training which undergraduates perceive in order to enhance their capability to recognize and manage a medical emergency and to become well qualified practitioners.[7]

Corresponding Author

Hemavathy.O.R,

Reader, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai -600077, Tamil Nadu, India.

Email: hemadocmd@gmail.com,

Mobile Number: 9626033600

References

1.Greenwood M. Medical emergencies in the dental practice. *Perio-1.dontol 2000.2008;*

46:27-41.

2. Coulthard P, Bridgman CM, Larkin A, Worthington HV. Appro-2.priateness of a Resuscitation Council (UK) advanced life support course for primary care dentists. *Br Dent J.2000; 188: 507-12.*

3. Atherton GJ, McCaul JA, Williams SA. Medical emergencies in 3.general dental practicein Great Britain. Part 1: their prevalence over a 10 year period. *Br Dent J.1999; 186: 72-9.*

4.Laurent F, Augustin P. Managing a cardiac arrest: evaluation of 4. final year predoctoral dental students. *J Dent Educ. 2009; 73: 211-8.*

5. Carvalho RM, Costa LR, Marcelo VC.Brazilian Dental students' 5.perceptions about medical emergencies: a qualitative exploratory study. *J Dent Educ.2008; 72: 1343-6.*

6. Gonzaga HF, Buso L, Jorge MA, Gonzaga LH, Chaves MD, Al-6. media OP. Evaluationof knowledge and experience of dentistsoffice São Paulo State, Brazil about cardiopulmonary resuscitation. *Braz Dent J. 2003; 14: 220-2.*

7. Atherton GJ, McCaul JA, Williams SA.Medical emergencies in 7.general dental Practicein Great Britain. Part 3: Perceptions of training and competence of GDPs in their management. *Br Dent J.1999; 186: 234-7.*

8.Champman PJ. A questionnaire survey of dentists regarding 12.knowledge and Perceivedcompetence in resuscitation and occu-rrence of resuscitation emergencies.*Aus Dent J.1995;40:98-103.*

9. McCarthy FM. Emergency drugs and devices – Less is more. J 10.*Calif Dent Assoc. 1993;21: 19-25.*

10. James DW. General anesthesia, sedation and resuscitation in dentistry. *Br Dent J. 1991; 171: 345-7.*

11. Atherton GJ, McCaul JA, Williams SA. Medical emergencies in general dental practice in Great Britain.Part 2: drugs and equipments possessed by GDPs and used in the management of emergencies. *Br Dent J. 1999 ; 186: 125-30.*

12. Broadbent JM, Thomson WM. The readiness of New Zealand general dental practitioners for medical emergencies. *N Z Dent J.2001; 97: 82-6.*

13. Atherton GJ, Pemberton MN, Thornhill MH.Medical emergen-11.cies: the experienceofstaff of a UK dental teaching hospital. *Br Dent J. 2000; 188: 320-4.*

14.Haas DA. Management of medical emergencies in the dental offi-8.ce: conditions in Eachcountry, the extent of treatment by the den-tist.*Anesth Prog. 2006; 53:20–4.*

15.Adewole RA, Sote EO, Oke DA, Agbelusi AG.An assessment of 9.the competence and experience of dentists with the management of medical emergencies in a Nigerian teaching hospital. *Nig Q J Hosp Med. 2009; 19:190-4.*

16.Chate RA. Evaluation of a dental practice cardio-pulmonary resuscitation training scheme. *Br Dent J.1996; 181:416-20.*

17. Haas DA. Management of medical emergencies in the dental office: Conditions in each country, the extent of treatment by the dentist. *Anesth Prog 2006;53:20-4.*

18. Carvalho RM, Costa LR, Marcelo VC. Brazilian dental students' perceptions about medical emergencies: A qualitative explorative study. *J Dent Educ 2008;72:1343-9.*

19. Chapman PJ. Medical emergencies in dental practice and choice of emergency drugs andequipment: A survey of Australian dentists. Aust Dent J 1997;42:103-8.

20. Gupta T, Aradhya MR, Nagaraj A. Preparedness for management of medical emergenciesamong dentists in Udupi and Mangalore, *India. J Contemp Dent Pract 2008;9:92-9.*

21.LeelavathyL,Chandrasekhara Reddy, ChalyPreetha Elizabeth, Indra Priyadarshni.Experience, awareness, and perceptions about medical emergencies among dental interns of Chennai city, India- A questionnaire based study. Department of Public Health Dentistry, SIMATS. *Journal of Indian Association of Public Health Dentistry Vol. 14, Issue 4, | October-December 2016.*

22. AadilAhamed, Santhosh Kumar M P. Knowledge, Attitude and Perceived Confidence in Handling Medical Emergencies among Dental Students. Department of Oral and Maxillofacial surgery, SIMATS.*J. Pharm. Sci. & Res. Vol. 8(7), 2016, 645-649.*

Evaluation of Knowledge, Experience and Perceptions about Medical Emergencies among Dentist in Chennai,India.

Vijayaalakshmi.L.G[1],Shantha[2], V.Jayanth Kumar[3]
Graduate Student[1], Lecturer[2], Department of Pedodontics,Reader[3], Department of Oral Medicine and Radiology,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Dentist encounters medical emergencies in their clinics which can be life-threatening.These emergencies should be treated immediately and cannot be avoided or referred,because they put the life and health of patients at risk.Therefore, the aim of this study was to evaluate how well dentist understand medical emergency during dental treatment.
The aim of this research is to evaluate the knowledge, experience and perceptions about medical emergencies amongst dentists in Chennai, India.

Materials and Method: Data was collected from 50 dentists of Chennai at self convenience using a structured questionnaire consisting of 10 item questionnaire.The sample was selected based on certain inclusion and exclusion criteria.Inclusion criteria includes postgraduate, South Chennai dentists.Exclusion criteria includes students,dental assistants.

Result: The study reveals that 60% the dentists in Chennai were confidently able to deal the life threatening emergency situations whereas 100% of dentists have knowledge how to manage the life threatening emergency situations.

Conclusion: Based on the above study it can be concluded that the level of training and level of knowledge on medical emergencies of the dentist in Chennai are good but not upto the desirable standard.

Key Words: *Medical emergencies, dental graduates, basic life support, emergency drugs.*

Introduction:

Every dental health professional should have the essential knowledge to identify, assess and manage a potentially life-threatening situation.Dentist encounter medical emergencies in their clinics which can be life threatening.These emergencies should be treated immediately and cannot be avoided or referred,because they put the life and health of patients at risk.(1) Professional bodies usually have its own code of conduct for their members.(2)The code of ethics for dentist, toward patients and public was standardized by the Dental Council of India, "Treat the welfare of the patients as paramount to all other considerations and shall conserve it to the utmost of his ability."(3)In 1971–1972, health professions education worldwide was assessed by the World Health Organization commission and the commission concluded that education and health service system are inextricably interwoven with each other and questions about the training of health-care providers follow soon after when questions arise about the delivery of service.(4)Dental professionals are expected to provide safe and painless dental procedures when they complete their education from dental colleges. Undergraduate dental education's aim has been described as "to produce a caring, knowledgeable, competent, and skillfull dentist who is able, on graduation to accept professional responsibility for the effective and safe care of patients who appreciates the need for continuing professional development, who is able to utilize advances in relevant knowledge and techniques and who understands the role of the patient in decision-making."(5)A medical condition demanding immediate treatment is an emergency.

Medical emergencies have many forms including syncope, asthmatic attacks, epileptic attacks, cardiac arrest, etc(6).In dental offices, medical emergencies do occur. A survey was conducted among 4000 dentists by Fast and others, and the results of the survey revealed that for 10 years 7.5 emergencies per dentist was reported.(7)A total of 13,836 emergencies were reported within a 10-year period in a survey conducted among 2704 dentists throughout North America.(8)It is mandatory that every dental health professional should have basic knowledge to manage a potentially life-threatening situation. A study was conducted in Japan between 1980 and 1984 by the Japanese Dental Society and it was reported in the study that anywhere from 19% to 44% of dentists had a patient with a medical emergency in any 1 year(9)and about 70.2% of general dental practitioners in the United Kingdom have managed medical emergency even.(10)One study conducted in Australia reported that approximately 15% of dental surgeons had done cardiopulmonary resuscitation (CPR) for the patients in their dental offices in 1 year.(11)

Although a number of studies have been carried out which sought to ascertain the emergency drugs and equipments, the lack of training and inability to cope with medical emergencies can lead to tragic consequences and sometimes legation action.Changing socioeconomics in the populace, prompting expanded life span have driven the general population having therapeutic conditions which incline to a medicinal crisis or taking solution may impact their dental administration and people matured over 65 years or over are thought to produce drug

with a potential results on dental care.An expanding extent of the populace is therapeutically in danger. According to the European resuscitation council, sudden cardiac arrest is a leading cause of death in Europe, affecting about 7, 00,000 individuals a year.(13)For this reason, as all the health professionals, dentists must be well prepared to attend to and collaborate with the medical emergencies .Providing basic life support (BLS) is the dentists' most important contribution until definitive treatment for a medical emergency can be provided(14). Few studies have assessed how competent dentists consider themselves in managing medical emergencies, and very few studies to our knowledge have reported studies involving fresh dental graduates.(15)In a study conducted by Gupta *et al*it was reported that less than half (42.1%) of the dentists received training during their undergraduate and postgraduate education.(12)The lack of training and inability to cope with medical emergencies can cause tragic consequences and sometimes legation action.(10).Hence, as all the health care professionals, dental care professionals must be well prepared to manage medical emergencies.

Therefore, the aim of this study is to learn the experience of handling medical emergencies, their skills and competency and how well they felt are prepared to manage such events with appropriate use of drugs and equipments in a dental setting.

Materials and Method

Data were collected from 50 dentist in Chennai using a structured questionnaire consisting of 7 item questionnaire (6 closed and 1 open ended).
The sample was selected based on certain inclusion and exclusion criteria.
Inclusion criteria:
1. Postgraduate
3.South chennai dentists.

Exclusion criteria:
1.Students
2.Dental assistants.

Results

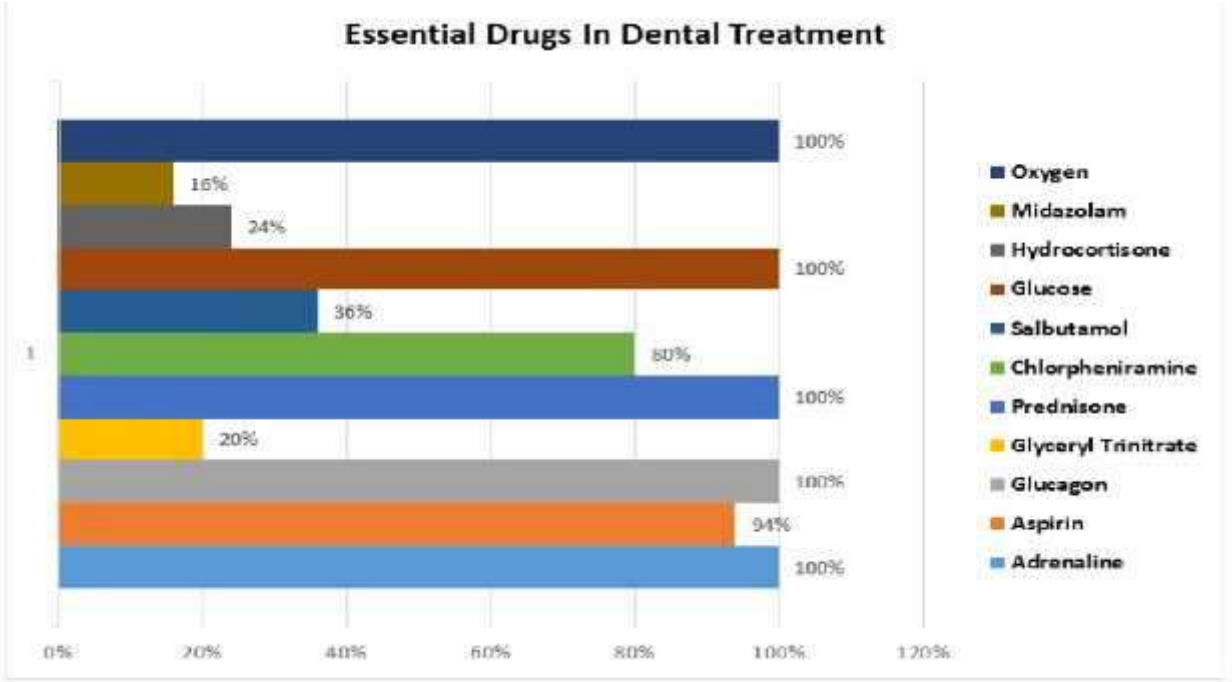


Figure 1: Shows the percentage of dentists about the knowledge of identifying the recommended drugs in their emergency kit.

Figure 2: Shows the percentage of dentists confidence of using the recommended drugs in their emergency kit.

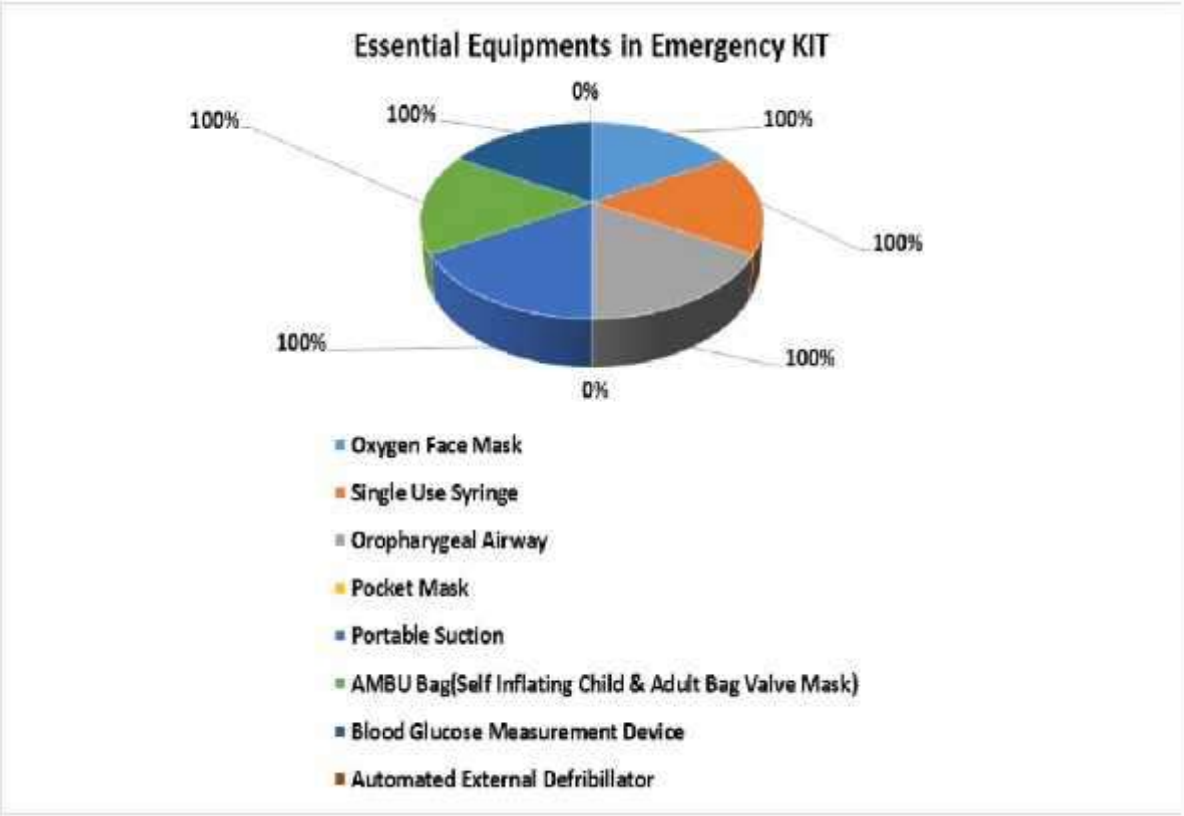


Figure 3:Shows the percentage of dentists about the knowledge of essential pieces of equipments in the emergency kit.

Figure 4: Shows the percentage of dentist's confidence of using essential pieces of equipments in the emergency kit.

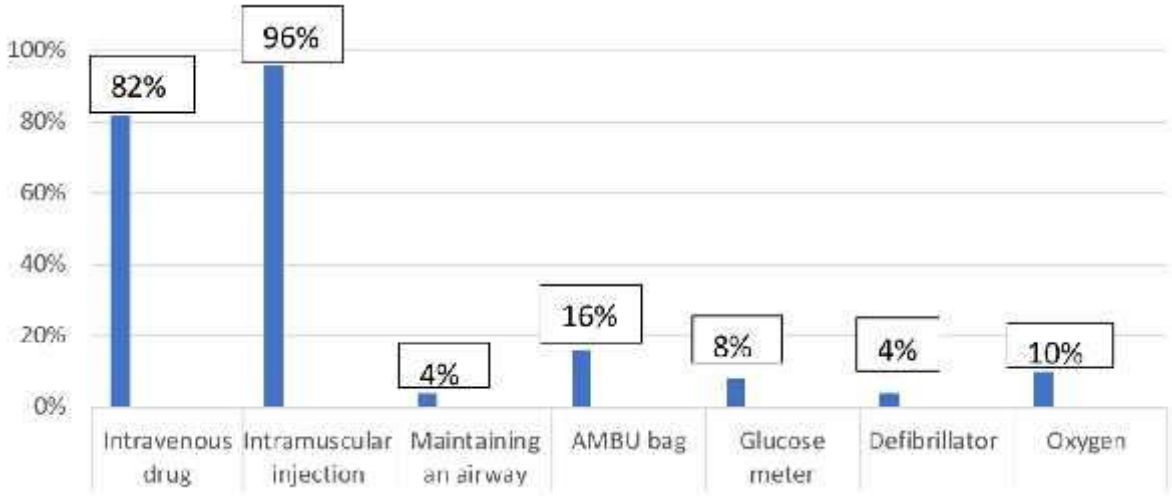


Figure 5: Shows the competency in the areas of drug administration/other procedures.

Discussion

Medical emergencies have been reported to occur frequently in dental practice.(16) Fortunately, serious medical emergencies in dental practice are not common but they are all the more alarming when they occur.(17) A thorough patient history can draw the practitioner's attention to potential medical emergencies that could occur.(16) Dental professionals most important contribution is to provide BLS to the patients until absolute treatment for medical emergency situations. Data obtained from dentists in independent studies by Fast et al and Malamed et al report that the dental office environment is not immune to the occurrence of potentially life threatening.In a 10 year period, more than 30,000 emergencies were reported by more than 4,000 dentists, Junaid Ahmed et al surveyed the nature of the emergencies varied significantly, from syncope to cardiac arrest and anaphylaxis. It is believed that the overwhelming majority of emergencies encountered were precipated by the increased stress that is so often present in the patient in the dental environment. Increased stress can result from fear and anxiety or inadequate pain control (18-19). Stress is associated with an increased occurrences of emergency situations was also reported by Matsuura et al who reported that 77.8 percent of life threatening systemic complications in the dental office developed either during local anaesthetic administration or during dental treatment. (20)

The above data dictates the need for the dental surgeon to be trained to promptlyrecognise and efficiently manage emergency situations.Our study showed knowledge about medical emergencies among the dental graduates. They hand the best knowledge was about handling syncope, diabetic emergencies and asthma and the knowledge about cardiac arrest was very minimal. Birang et al in their study showed that the knowledge score of Esfahan dentists was

5.42/10(21). Gupta et al stated that the knowledge of dental surgeons from Udupi and Mangalore, Karnataka was from moderate to poor(22).Regina M et al, in their study stated that dental students have little understanding about medical emergencies (23). The knowledge of Mashhad dentists was also poor as stated in their study conducted in the province of Iran (24). These results indicate a need for a revision in the curriculum of dental graduate studies. The dental graduates in our study showed a good level of confidence in checking carotid pulse, performing Heimlich maneuver, cardio, pulmonary resuscitation and artificial respiration. We believe that though the knowledge level is adequate what the graduate ack is in the skills. In the present study a large number of graduates stated that they did not know how to react in those situations even though they received training in the management of medical emergencies at some time in their curriculum.

From the responses regarding the number of hours of medical emergencies training earmarked in the undergraduate curriculum, it is evident that there is a definite need to increase the number of hours spent of emergency management. Only 54.7% of respondents had undergone training in medical emergency and basic life support (BLS) training. Providing basic life support (BLS) is dental surgeons most important contribution until definitive treatment for a medical emergency can be given. The purpose of BLS is to prevent inadequate circulation or respiration thorough prompt recognition of the problem and intervention to support a victims circulation and respiration through CPR (25). However a number of studies have found that about half the dentists from all over the world are notable to perform CPR properly (26-28). The result may be due to the lack of definitive guidelines from the regulatory bodies about the training with medical emergencies in the dental curriculum. In the dental curriculum, management of medical emergencies in dental practice comes under the syllabi of general medicine and oral surgery (29). The exact duration and the methodology has not been specified, which needs to be updated to enable students to develop a confident approach towards handling medical emergencies.

The dental graduates expressed the need for further medical emergencies training and were prepared to be life long learners about the topic at hand, and expressed their willingness to attend continuing professional development programs by which they can update their knowledge about medical emergencies. Our result was in accordance with other studies done in the same field (30-35). The confidence in the use of drugs and equipment mentioned were at a very lower level than the knowledge for all. The drugs and equipment mentioned. This suggests that although training is received in the theoretical aspect of emergencies, participants are not particularly confident to treat emergencies and may require further practical training. Athereton JS's study suggested that up to 44% of dentists may have a patient with a medical emergency in a year. The result of this study also reflect that medical emergencies occur quite frequently because more that n half of the dentists claimed to have previously encountered medical emergency during their training(36). The result of this study suggests that a deficiency in dental academy's curriculum on medical emergencies. A

curriculum refers to the overall content of what is to be taught about a subject matter.Guidelines are in place for the management of medical emergencies in a dental setting and the purpose of these guidelines is to set out agreed philosophy and responsibilities of all dentists in relation to resuscitation and management of medical emergencies in the dental surgery (37). Other important points revealed by this study are that only the theoretical training in medial emergencies seems to be effective.

This study also suggest that the dentists may not be prepared to handling medical emergencies effectively because major claimed not to have been taught how to perform many of the potentially lifesaving procedures. Dentists are members of the medical profession and should be confident in dealing with emergencies which may arise during their work. This result is similar to what was reported among a group of medical students where 85.3% of them had never take any basic life support course (38). Prevention and preparation are often the best antidotes for an emergency. Beginning by obtaining a good health history at the patient's past and present health status. It should also include questions indicating problems the patient may not be aware of , but which may alter treatment. A list of medication names and dosages that are currently prescribed to the patient should be ascertained(39).The result of this study amplifies the need to ensure that this recommendation is taken very seriously.

Conclusion

Based on the above study it can be concluded that the level of training and level of knowledge on medical emergencies of the dentist in Chennai are good but not upto the desirable standard.The study has allowed to find deficiencies in the way the dentists were trained dealing with medical emergencies and identify a need for improvement, be by increasing the volume and quality of training which they perceive in order to enhance their capability to recognise and manage a medical emergency and to become well qualified practitioners.Since they are dealing with the life threatening situations, it is therefore necessary to put proper strategies in place, such as the development of the practical model, to improve the areas of weakness this study identified.for the patients in their clinic and they should be well knowledgeable and trained to deal with the emergency situations in the dental office.

Correponding Author

V. Jayanth Kumar

Reader, Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600 077, Tamil Nadu, India

Email Id: doctorjayanth@gmail.com

Contact Number: +91 8056135807.

References

1. Greenwood M. Medical emergencies in the dental practice. Periodontol 2000. 2008;46:27-41.
2. .Sabarinath B, Sivapathasundharam B. Ethics in dentistry. J Educ Ethics Dent 2011;1:24-7.
3. .Dentists (Code of Ethics) Regulations, 1976 Extract from the Gazette of India, Part II, Sec. 3, Sub Sec (1) Appearing on Page No. 2223-7.
4. .Hendricson WD, Cohen PA. Oral health care in the 21st century: Implications for dental and medical education. Acad Med 2001;76:1181-206.
5. .Rafeek RN, Marchan SM, Naidu RS, Carrotte PV. Perceived competency at graduation among dental alumni of the University of the West Indies. J Dent Educ 2004;68:81-8.
6. .CNAHealth Pro Medical Emergencies.Availablefrom:http:www.dentalrisk.com/Portals/20/articles/MedicalEmergencies.pdf.
7. .Morrison AD, Goodday RH. Preparing for medical emergencies in the dental office. J Can Dent Assoc 1999;65:284-6.
8. .MalamedSF.Knowing your patients. J Am Dent Assoc 2010;141 Suppl 1:3S-7S.
9. .Haas DA. Management of medical emergencies in the dental office: Conditions in each country, the extent of treatment by the dentist. Anesth Prog 2006;53:20-4.
10. .Carvalho RM, Costa LR, Marcelo VC. Brazilian dental students' perceptions about medical emergencies: A qualitative explorative study. J Dent Educ 2008;72:1343-9.
11. .Chapman PJ. Medical emergencies in dental practice and choice of emergency drugs and equipment: A survey of Australian dentists. Aust Dent J 1997;42:103-8.
12. .Gupta T, Aradhya MR, Nagaraj A. Preparedness for management of medical emergencies among dentists in Udupi and Mangalore, India. J Contemp Dent Pract 2008;9:92-9.

13. .Coulthard P, Bridgman CM, Larkin A, Worthington HV. Appropriateness of a Resuscitation Council (UK) advanced life support course for primary care dentists. Br Dent J. 2000;188:507–12.
14. .Atherton GJ, McCaul JA, Williams SA. Medical emergencies in general dental practice in Great Britain. Part 1: their prevalence over a 10 year period. Br Dent J. 1999;186:72–9.
15. .Laurent F, Augustin P. Managing a cardiac arrest: evaluation of final year predoctoral dental students. J Dent Educ. 2009;73:211–8.
16. .Greenwood M. Medical emergencies in the dental practice. Periodontol 2000. 2008;46:27–41.
17. .Coulthard P, Bridgman CM, Larkin A, Worthington HV. Appropriateness of a Resuscitation Council (UK) advanced life support course for primary care dentists. Br Dent J. 2000;188:507–12.
18. .Fast TB, MartinMDandEllisTM,Emergency preparedness:a survey of dental practioners.JAmerDent Assoc1986; 112:499-501.
19. .Malamed SF, Managing medical emergencies. J Amer Dent Assoc1993:124:40-53.
20. .Matsuura H, Analysis of systemic complications and deaths during dental treatment in Japan. Anesth Prog1990;36:219-28.
21. .Birang R, Kaviani N, Behnia M, MirghaderiM.Knowledge and equipment preparedness of Isfah and dentist about medical emergencies in 2006.Iran Education Med Sci J.2006;5:47-54.
22. .Tanupriya Gupta, Shankararadhya,Anup.Preparedness for management of medical emergencies among dentists in Udupi and Mangalore,India.TheJournal of Contemporary Dental Practice2008;9(5) 1-10.
23. .Regina M.Carvalho, LucianeR.Costa, VaniaC.Marcelo Brazilian Dental Students Perception about Medical Emergencies : A Qualitative Exploratory Study.Journal of Dental Education 2008;72(11) 1343-9.
24. .Amirchanghmaghi , J Sarabadani , Z Delavarian , P MosannenMozafary , A Shahri, Z Dalitsani. The Knowledge and Preparedness of General Dentists about Medical Emergencies in Iran ; Iran Red Crescent Med J2011;13(3):217-8.
25. .PeskinRM,SiegelmanLI.Emergency cardiac care:moral,legal and ethical considerations.DentClinNorthAm 1995;39(3):677-88.

26. .Chapman PJ.Medical emergencies in dental practice and choice of emergency drugs and equipment:a survey of Australian dentists.Aust Dent J 1997;42(2):103-8.
27. .Chapman PJ.A questionnaire survey of dentists regarding knowledge and perceived competence in resuscitation and occurrence of resuscitation emergencies.Aust Dent J 1995;40(2):98-03.
28. .GirdlerNM,Smith DG. Prevalence of emergency events in British dental practice and emergency management skills of British dentist.Resuscitation 1999;41(2):159-67.
29. .The Dental Council Of India.Available from http://www.dciindia.org/BDSsyl.pdf
30. .Atherton GJ,McCaulJA,WilliamsSA.Medical emergencies in general dental practice in Great Britain.Br Dent J.1999;186:72-9.
31. .CarvalhoRM,CostaLR,MarceloVC.Brazilian Dental students perceptions about medical emergencies: a qualitative exploratory study.J Dent Edu.2008;72:1342-9.
32. .Haas DA.Management of medical emergencies in the dental office: conditions in each country, the extent of treatment by the dentist.2006;53:20-4.
33. .AdewoleRA,SoteEO,OkeDA,AgbelusiAG.An assessment of the competence and experience of the dentists with the management of medical emergencies in a Nigerian teaching hospital.2009;19:190-4.
34. 34.AthertonGJ,PembertonMN,ThornhillMH.Medicalemergencies: the experience of staff of a UK dental teaching hospital.Br Dent J.2000:320-4.
35. 35.Broadbent JM, Thomson WM. The readiness of New Zealand general dental practitioners for medical emergencies. N ZDent J. 2001;97: 82-6.
36. 36. Atherton JS. Teaching and learning; curriculum.2011.
37. 37.Bilbao PP, Lucido PI, Iringan TC, Javier RB. Quezon City: Lorimar Publishing, Inc;2008. Curriculum Development.
38. 38.Harden RM. AMEE Guide No.21:Curriculum mapping: A tool for transparent and authentic teaching and learning. Med Teach. 2001;23:123–37.
39. 39.AadilAhamed et al(Saveetha Dental College and Hospitas).Knowledge, Attitude and Perceived Confidence in Handling Medical Emergencies among Dental Students. /J. Pharm. Sci. & Res. Vol. 8(7), 2016, 645-649.

Evaluation of Patient's Experience and Overall Satisfaction with Implant Retained Over Denture and Conventional Complete Denture - A Questionnaire Based Survey

Shubha Poorani.E [1], Ashok.V [2], Sreedevi Dharman [3]
Graduate student [1],Reader [2],Department of Prosthodontics
Senior Lecturer [3], Department of Oral Medicine
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Rehabilitation of patients with complete Edentulousnessare more complex in prosthetic dentistry because of its psychosocial, aesthetic and functional implications. Teeth serves as a part of the masticatory system and it mainly contributes to phonetics, functions and aesthetics. To evaluate the patient's experience and overall satisfaction with surgical and prosthetic procedures with implant over denture and conventional complete denture.

Materials and Methods:A questionnaire based survey was conducted. Questionnaire was prepared based on the aesthetic, function and comfort level of the patient. 30 patients were randomly selected who undergone treatment for completely edentulous condition.

Result: The purpose is to know the different opinions of the patients regarding the prosthodontics treatment so that efforts can be taken to produce better results and satisfaction. Patient’s feel economically complete denture (70%) is better than implant supported over denture (30%) because the implants are cost effective. Based on function, implant supported over denture was 70% higher than complete denture which was 30%. Hence the overall satisfaction in this study was better in implant supported over denture (60%) than the complete denture (40%) because they play important role with retention.

Conclusion : The comfort level with implant retained over denture is superior to the conventional complete denture. Aesthetics, functions are comparatively good in both conventional complete denture and implant retained over denture.

Key Words*: experience, satisfaction, conventional complete denture and implantover denture*

Introduction

Great improvements in medical field have greatly increased average human life expectancy, indicating that patients now have greater chances of turning into completely edentulous. For edentulous patients who receive dentures, providing enough stability and support for the dentures is not sufficient, instead,acceptance of dentures by the patients to endure a process of adjustment. The patient encounters more difficulties when they wear complete dentures for a longer period of time because the ridges are atrophied [1]. This kind of atrophy becomes worse when thepatients are wearing a mandibular complete denture because of the tongue mobility issues and decreased contact surface. Many other problems when combined such as related to neuromuscular coordination and the denture's ability to form a tight seal with the surrounding soft tissues, patients will show much more dissatisfaction with the complete dentures especially mandibular dentures [2].

Since then Brånemark's success towards titanium root-form dental implants, when dental implant therapy has given new hope for edentulous patients. Dental implant treatment provides patients with better stability and increased biting .Rehabilitation of patients with complete Edentulousness are more complex in prosthetic dentistry because of its role as comfort, aesthetic and functional implications. Teeth serves as an important part of the masticatory system and it mainly concerned with phonetics, functions and aesthetics [3,4].

The psychological variations in patients due to tooth loss leads to little concern, when most of them adapt to replace the missing in the form of bridges, crowns and dentures[5]. Implants play an very important role to have greater retention and stability of the complete dentures, thereby initiating efficient functioning, psychological and fixed partial reconstructions to avoid the need to prepare intact adjacent teeth [6,7]. Additional factors involved may be preventing continuous alveolar bone resorption, by preserving ridge height and width, and improving aesthetics and comfort in anterior regions [8, 9].

Many of the studies have focused on the osseointegration [10, 11, and 12] through the clinical aspect without considering patients' attitudes towards implant treatment and their suggestions about aesthetics, function, comfort and satisfaction. The present study evaluates patients' satisfaction and experiences of surgical and prosthetic procedures as well as their opinions regarding function, aesthetics and comfort.

Conventional complete denture prostheses have been commonly used for the missing dentition replacement. Complete denture fabrication and techniques have advancements and has been improved over the years; however some attention has been paid towards the psychological impact of the patient before and after receiving the prostheses. Psychological factors play an important role which provides valuable information for the satisfactory outcome of complete denture treatment. Various other factors like mastication and speech also contribute to the success of this treatment.Force with the prostheses [3, 4]? However, for some edentulous patients with certain unfavourable oral conditions or financial concerns, support for fixed partial dentures is not feasible. Therefore, over dentures retained or supported by implants have been adopted [13, 14, 15].

In general, implant-retained over dentures can be applied over placing as few as one or up to many implants and then using attachments to provide retention to the denture [14, 15, and 17].

The critical concerns for frequently denture-wearing patients are related to appearance, speech, occlusion, and Masticatory functions [16, 17, 19]. It is common for clinicians to use intraoral conditions as gingival and periodontal health, and prosthesis condition to examine the treatment outcome [18, 19].However; these conditions do not completely reflect the definite needs of patients [17].

Dentists focus on each patient’s needs regarding the usage of dentures, including their need regarding comfort levels, appearance, feeling, function, speech, and the patient’s confidence, in order to estimate patient satisfaction [20, 21] The final goal was to achieve a standard, such that edentulous patients are completely satisfied with the treatment and have a better understanding of the impact of edentulouson the their quality of life. A good dentist has to consider the given patient’s specific requirements. In the view of most dentists, the survival rate of dental implants, the life span of dentures, and treatment complications are the most important considerations. However, what patients are concerned about the most are how many benefits the treatment will provide and whether the costs are worthwhile. Moreover, considerations of how the treatment could possibly affect the patient’s social life and mental well-being are paramount. Therefore, dentists should evaluate a given treatment with

function, aesthetics, and the patient's feelings and expectations in mind [24].

Materials and Method

30 edentulous patients who undergone treatment for replacement were randomly selected from the database in Department of prosthodontics, Saveetha Dental College, Chennai. 12 edentulous patients have implant retained over denture. 18 patients have conventional complete denture. Questionnaire was prepared based on the aim and objective of the study. After obtaining their written consent to participate in the study, the questionnaire was filled in consultation with the patient. The detail regarding the newly constructed prostheses was recorded as it was reported by the patient. The patient was encouraged to give frank opinion about his/her new denture and was assured that the identity would be kept confidential. The questions were asked in his/her own mother tongue. Once the questionnaire was completed for 30 patients, they were grouped based on the scale of fair,satisfied,good,excellent for different parameters like i) Functioning, ii) aesthetics, iii) comfort iv)) overall satisfaction for maxillary and mandibular dentures.

Questionnaire:

1) When did you lose your teeth?

   A) 1 MONTH AGO B)6 MONTHS AGO C)MORE THAN ONE YEAR

2) How did you lose your teeth?

   A) TRAUMA B) CARIES C)MOBILITY

3) Did you visit the dentist immediately after losing your teeth?

   A) YES B) NO

4) Do have any previous experience with dentures. If yes, what prosthesis?
   A) YES B) NO

5) Have you any idea about fixed prosthesis?
   A) YES B)NO C)NO IDEA

6) Have you any idea about over dentures?

A) YES B) NO C)NO IDEA

7) Which prosthesis would you prefer?

A) CONVENTIONAL COMPLETE DENTURE
B)IMPLANT OVER DENTURE
C)NO IDEA

8) Why do you prefer your decision?

A) ECONOMIC B)AESTHETIC C)COMFORT

9) How efficient do you think your denture is?

A) FAIR B) SATISFIED C)EXECELLENT

10) Are you satisfied with the aesthetics?

A) FAIR B) SATISFIED C)EXECELLENT

11) How is the denture functioning?

A) FAIR B) SATISFIED C)EXECELLENT

12) How is the comfort level of the denture?

A) FAIR B) SATISFIED C)EXECELLENT

Results

Fig-1 Shows satisfaction and experience of complete denture.50% of the population are concerned about the cost effective factor. 20% shows satisfaction towards aesthetics and function. 10% of the complete denture wearers are satisfied with the comfort level.

Fig-2 showssatisfaction and experience of implant overdenture.30% of the population shows satisfaction towards aesthetics, function and comfort. 10% of the patients wearing implant supported over denture are satisfied with the cost.When comparing the overall satisfaction among implant supported over denture and conventional complete denture, the comfort level with implant supported over denture shows higher satisfactory level (bar chart). When comparing satisfaction of the aesthetic factor and function, the implant supported over denture are significantly better than conventional complete denture. Treatment option for complete edentulous patients ranges from removable prosthesis and fixed prosthesis. Due to cost factor fixed prosthesis retained with implants are less popular in patients. Moreover the fixed prosthesis with implants is technique sensitive and needs multidisciplinary approach. This causes prolongation of treatment time. The conventional complete dentures are chosen to reduce the treatment cost as well as the treatment time.

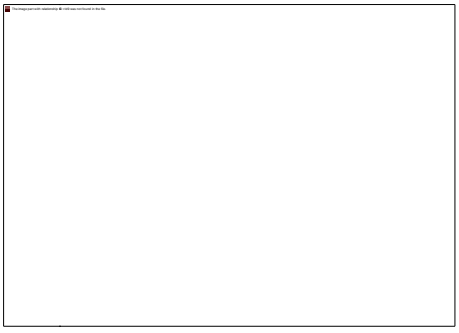


Figure 1: Satisfaction and experience of complete denture

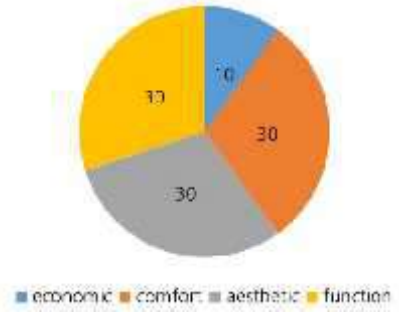


Figure 2: Satisfaction and experience of implant over denture

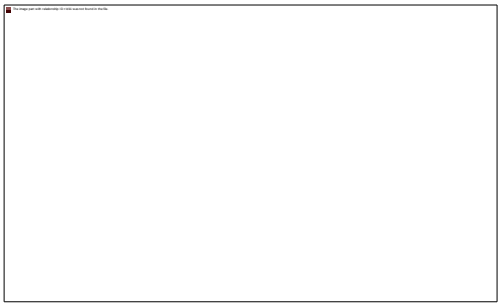


Figure 3: Comparison of conventional complete denture and implant supported over denture based on aesthetics, Comfort and function

Table 1: Patients overall satisfaction over conventional complete denture and implant supported over denture

	Complete denture	Implant retained overdenture
Economic	70%	30%
Comfort	20%	80%
Aesthetic	35%	65%
Function	30%	70%
Satisfaction	40%	60%

Discussion

The survey shows overall satisfactions with implant retained over denture are better. The comfort level between the two shows very significant variation. The treatment option of Implant supported over denture cannot be suggested for medically compromised patients. In residual ridge resorption patients, especially in mandible the retention and stability of complete denture are compromised. In this situation implant retained over dentures are suggested. Patient's feel economically complete denture (70%) is better than implant supported over denture (30%) because the implants are cost effective. Based on comfort, implant supported over denture was 80% higher than complete denture which was 20%. Based on aesthetics, implant supported over denture was 65% higher than complete denture which was 35%. Based on function, implant supported over denture was 70% higher than complete denture which was 30%. Hence the overall satisfaction in this study was better in

implant supported over denture (60%) than the complete denture (40%).

No gender-based difference was observed in our study. However, some previous studies have indicated that male patients are more satisfied than female patients. One possible reason for this difference is that most females are much more aware of pain and operations. Therefore, non-surgical treatments will be the first choice for elderly female patients [22].
Most previous studies on patient satisfaction have only reported the average age of the participants, but differences in satisfaction levels among different age groups have not been

In terms of masticating ability for different food types, soft foods had the best score while hard and sticky foods had the lowest score. Similar results have been found in most other studies. Awad studied 102 patients (48 wearing complete dentures and 54 wearing mandibular implant-retained over dentures) and applied VAS to evaluate the masticating ability for different food types.

In 2002, Raghoeber showed that mandibular implant-retained over dentures were comparatively better than complete dentures in the first and fifth years after treatment, both in performance of the clinic and satisfaction of the patient. For placing the mandibular implant-retained overdentures,showed a better score than the complete dentures in the tenth year, although the difference was less significant [23].

In 2004, Timmerman published a randomized con- trolled trial study comparing two mandibular implant-retained over dentures with a ball or bar attachment to a four mandibular implant-supported over denture with three bar-and-clip attachments.(26)Although there was no significant difference among these three treatments, patient satisfaction in terms of retention and stability with the mandibular four implant-supported over denture was much better than for the other treatments after 8 years. This result also demonstrated that this design was more stable [24].

A similar result was found by Heydecke in an analysis of 60 seniors (30 with complete dentures and 30 with implant-re- tained over dentures). According to an analysis of mental and social ability using the OHIP-20 (oral health impact profile), patients wearing mandibular implant-retained over dentures had better performance.

Complete edentulous is related with decreased masticatory function, as well as unfavourableaesthetics’to loss of support for the musculature of face, reduced vertical dimension, and impairment of speech. The conventional method for treating edentulous patients is to provide them with conventional complete dentures. And also denture must be adjusted overtime for progressive tissue changes related with denture wearing. Patients who wear conventional dentures complainabout the instability of the prosthesis, particularly the mandibular denture due to retention. Denture instability leads to a feeling of insecurity, inefficient mastication, and overall dissatisfaction with the denture. Many recent advances in implant dentistry have allowed a change from conventional complete denture to implant-supported overdenture for oral rehabilitation of edentulouspatients [25].The McGill consensus statement in 2002 and some other studies states that the mandibular implant over dentures should be the first treatment of choice for edentulous patients. Implant-retained over dentures have many advantages over conventional complete dentures as chewing efficiency, masticatory bite force.

Chewing replaces the body's nutrients, thereby facilitating the maintenance of the body composition. Mastication is the first step and consists of a rhythmic separation and apposition of the jaws and involves biophysical and biochemical processes. Although data on the importance of chewing on various stages of digestion are very much limited, it has been concluded that the chewing efficiency as low as 25% adequate for complete digestion of food. Patients do not, however, have alternative smaller number of teeth by more prolonged o number of chewing strokes; they hardly swallow large food particles. Hence, loss of teeth can lead to a reduced chewing efficiency, and there is known factor of restricted dietary choice with systemic effects. Clinical experience suggests a relationship between the quality of prosthetic service and denture wearers chewing performance. Dentures move during mastication because of dislodging forces of the surrounding the muscles[27].These movements manifest themselves as displacing, lifting, sliding, tilting, or rotating of the prosthesis. Mandibular implant overdenture helps in better stability, support, and retention which reduces the denture movements and furthermore helps in achieving better chewing efficiency and masticatory bite force. The masticatory performance is reduced to one-fourth to one seventh of the performance of dentate subjects, depending on age and type of food. Thus, denture wearers need seven times more masticatory cycles to reduce food to half of its

original size [29,30]. Reported masticatory bite forces using complete denture is much smaller than those produced by natural dentition which is of the order of 200 N. Although maximum bite forces of 60–80 *N* have been reported for complete dentures and 150–170 *N* for implant-supported overdenture [28]. Edentulism is affiliated with anatomical, functional, and psychologic changes in patients. In complete dentures, the absence of stability, retention, and reduced chewing ability are common oral findings accompanied by poor quality of life. The stated benefits of an implant overdenture include bone preservation, improvement in masticatory ability, masticatory force, increased stability and retention, and significantly higher patient satisfaction. There is a great variety of methodologies describing chewing efficiency.

Complete dentures (CD) are the traditional form of treatment for the edentulous patient, and despite a number of limitations, they are still commonly used today.However, due to a lack of predictable outcome and variation in the patient's ability to adapt to CDs [5] especially in the mandible, alternate forms of treatment have been developed, With the advent of osseointegrated implants in the early 1980s, novel forms of treatment that possess greater retention and stability have been introduced; these include implant-retained mandibular over dentures (OD) and complete fixed dentures [13].

While comparing complete denture, implant retained over denture provides edentulous patients with a better quality life due to their greater retention and stability, increased level of function and aesthetics, and reduced alveolar ridge resorption.These features improve the patient's inability to chew and speak, which are common complaints associated with CDs28].More recently, the use of OD for the treatment of the edentulous patient has been considered as the first choice in treatment for the edentulous mandible [31].

Most of the recent studies have focused on the clinical aspects of osseointegration [29, 30,31] without considering patients' attitudes toward implant treatment and their opinions about aesthetics, function, comfort and satisfaction.The importance of providing patients with high quality dentures should be self-evident, if for no other reason than to avoid harmful effects on the oral tissues [32].

Restoration of functions, esthethic and maintenance of patient's health are the basic objectives for an ideal complete denture therapy [33].During a patient's management, an

operator may be satisfied with the quality of work done by them, but the success of the treatment is determined by satisfaction[34].

Conclusion

The comfort level with implant retained overdenture is superior to the conventional complete denture. Aesthetics, functions are comparatively good in both conventional complete denture and implant retained overdenture.The comfort level with implant supported over denture is significantly better than conventional complete denture. In severe resorbed ridge situations implant supported over dentures are suggested.

Corresponding Author

Sreedevi Dharman

Senior Lecturer, Department of Oral Medicine

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India

Email id: sanjamrut@gmail.com

Telephone Number: 9841009003

References

1. BoerrigterEM,GeertmanME,VanOortRP,BourmaJ, RaghoebarGM, van Waas MA. Patient satisfaction with implant-retained mandibular over dentures. A comparison with new complete dentures not retained by implants: A multicentre randomized clinical trial. Br J Oral Max Surg 1995; 33:282-8.

2.Tallgren A. The continuing reduction of the residual alveolar ridges in complete denture wearers: A mixed-longitudinal study covering 25 years. 1972. J Prosthet Dent 2003; 89:427-35.

3. Allen PF, McMillan AS, Walshaw D. A patient-based assessment of implant-stabilized and conventional complete dentures. J Prosthet Dent 2001;85:141-7.

4. Misch CE. Contemporary implant dentistry St. Louis Mosby: Elsevier; 2008.

5. Grogono AL, Lancaster DM, Finger IM. Dental implants: a survey of patients’

attitudes.The Journal of Prosthetic Dentistry. 1989;62(5):573–576.

6. Palmer RM, Smith BJ, Palmer PJ, Floyd PD. A prospective study of Astra single tooth implants. Clinical Oral Implants Research. 1997;8(3):173–9.
7. Lekholm U, GrÃndahl K, Jemt T. Outcome of oral implant treatment in partially edentulous jaws followed 20 years in clinical function. Clin Implant Dent Relat Res. 2006;8(4):178–86.
8. Zitmann NU, Marinello CP. Treatment outcomes of fixed or removable implant-supported prostheses in the edentulous maxilla. Part I: patients' assessments. The Journal of Prosthetic Dentistry. 2000;83(4):424–433.
9. Chang M, Odman PA, Wennström JL, Andersons B. Esthetic outcome of implant-supported single-tooth replacements assessed by the patient and by prosthodontists. The International Journal of Prosthodontics. 1999;12(4):335–341.
10. Adell R, Eriksson B, Lekholm U, Branemark PI, Jemt T. Long term follow up study of osseointergrated implants in the treatment of totally edentulous jaws. International Journal of Oral and Maxillofacial Implants. 1990;5(4):347–359.
11. Bergendal T, Engquist B. Implant-supported over dentures a longitudinal prospective study. International Journal of Oral and Maxillofacial Implants. 1998;13(2):253–62.
12. BlumbergS, Lindquist LW. Psychological reactions to Edentulousness and treatment with jawbone-anchored bridges. Acta Psychiatric a Scandinavica. 1983;68(4):251–262.
13. Doundoulakis JH, Eckert SE, Lindquist CC, Jeffcoat MK. The implant-supported

Overdenture as an alternative to the complete mandibular denture. J Am Dent Assoc 2003;134:1455-8.

14. Naert I, De Clercq M, Theuniers G, Schepers E. Overdentures supported by osseointegrated xtures for the edentulous mandible: A 2.5-year report. Int J Oral Maxillofac Surg 1998;3:91-6.
15. Naert I, Gizani S, Vuylsteke M, Van Steenberghe D. A 5-year prospective randomizedclinical trial on the in uence of splinted and unsplinted oral implants retaining a mandibular: Prosthetic aspects and patient satisfaction. J Oral Rehabil 1999;26:195-202.
16. Preiskel HW. Overdenture made easy. Quintessence 1996.
17. Wright PS. Two implants for all edentulous mandibles. Br Dent J 2006;200:469.
19. Lindquist LW, Carlson GE, Hedegård B. Changes in bite force and chewing efciency after denture treatment in edentulous patients with denture adaptation dif culties. [erratum

appears in J Oral Rehabil May 1986;13:293

20. Heydecke G, Thomason JM, Awad MA, Lund JP, Feine JS. Do mandibular implant over dentures and conventional complete dentures meet the expectations of edentulous patients? Quintessence Int 2008;39:803-9.
21. Zitzmann NU, Marinello CP. Treatment outcomes of fixed or removable implant-supported prostheses in the edentulous maxilla. Part I: Patients‘ assessments. J ProsthetDent 2000;83:424-33.
22. Cune MS, de Putter C, Hooqstraten J. Treatment outcome with implant-retained over dentures: Part II--Patient satisfaction and predictability of subjective treatment outcome. J Prosthet Dent 1994;72:152-8.
23. Pan S, Awad M, Thomason JM, Dufresne E, Kobayashi T, Kimoto S. Sex differences in denture satisfaction. J Dent 2008;36:301-8.
24. Meijer HJ, RaghoebarGM GM, van’t Hof MA, Geertman ME. Implant-retained over dentures compared with complete dentures with or without preprosthetic surgery. Satisfaction and subjective chewing capabilities. Ned TijdschrTandheelkd 2002;109:475-80.
25. Timmerman R, Stoker GT, Wismeijer D, Oosterveld P, Vermeeren JI, van Waas MA. An eight-year follow-up to a randomized clinical trial of participant satisfaction with three types of mandibular implant-retained Overdentures. J Dent Res 2004;83:630-3.
26. Boven GC, RaghoebarGM GM, Vissink A, Meijer HJ. Improving masticatory performance, bite force, nutritional state and patient's satisfaction with implant Overdentures: A systematic review of the literature. J Oral Rehabil 2015; 42:220-33
27. Thomason JM, Kelly SA, Bendkowski A, Ellis JS. Two implant retained Overdentures – A review of the literature supporting the McGill and York consensus statements. J Dent 2012; 40:22-34.
28. Albrektsson T, Blomberg S, Bran mark A, Carlson GE. Edentulousness—an oral handicap. Patient reactions to treatment with jawbone-anchored prostheses. Journal of Oral Rehabilitation. 1987; 14(6):503–511.
29. Strassburger C, Heydecke G, Kerschbaum T. Influence of prosthetic and implant therapy on satisfaction and quality of life: a systematic literature review. Part 1: characteristics of the studies. Int J Prosthodont 2004; 17 (1):83–93.

30. Strassburger C, Kerschbaum T, Heydecke G. Influence of implant and conventional prostheses on satisfaction and quality of life: a literature review. Part 2: qualitative analysis and evaluation of the studies. Int J Prosthodont 2006; 19(4):339–48.

31. Feine JS, Carlsson GE, Awad MA, Chehade A, Duncan WJ, Gizani S. The McGill consensus statement on Overdentures: mandibular two-implant over dentures as first choice standard of care for edentulous patients. Gerodontology 2002; 19(1):3–4.

32. Sanchana, V. B. and Dr. Suresh.Oral health related quality of life for patients treated with complete dentures, removable and fixed partial dentures.International Journal of Recent Advances in Multidisciplinary Research.Vol. 02, Issue 07, pp. 0526-0529, July, 2015

33. Fahmida binti Abd Rahman, Dr. Ashish Jane /J. Knowledge,Attitude and Practice of Various Impression Techniques available for Flabby Ridge in Edentulous Patients among Dental Interns.Pharm. Sci. & Res. Vol. 9(2), 2017, 208-211.

34. Kaviena Baskaran. Comparative Study of Patient Satisfaction on Receiving Bio functional Prosthetic System (BPS) and Conventional Complete Denture.IJSR.2015; 2319-7064

Factors Contributing To Not Seeking Orthodontic Treatment In Male Adolescents - A Questionnaire Study

Rebecca Rachel George[1], AV Arun[2], Ravindra Kumar Jain[3]
Graduate student[1,] Professor[2], Department of Orthodontics,
Reader[3], Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction : Motivation is an impetus for patients to seek orthodontic treatment; it also affects adherence, treatment outcomes, and satisfaction towards orthodontic treatment. The aim of this study was to assess the lack of motivation of adult male patients or the other reasons responsible for not seeking orthodontic treatment, and classify the patients according to their reasons. Prejudiced notions about treatment and various fears have created a resistance or hesitation towards orthodontic treatment in men. The aim of this study is to assess the mind set and overall attitude of male adolescent patients toward not seeking orthodontic correction for malocclusion.

Materials and Methods :A questionnaire based study was conducted at the department of Orthodontics, Saveetha Dental College, a total of 50 male subjects with malocclusions requiring orthodontic interventions were selected for the study. Data was collected using a structured, self-administered questionnaire which was designed after reviewing recent articles, developments and also similar questionnaires that are based on the objectives of the study.

Results :69% of subjects were aware that they had a malocclusion whereas the remaining 31% were not aware. On asking as to why they were not correcting the malocclusion, 18% stated that it was embarrassing to wear braces, 4% stated that the treatment was too expensive, 19% stated that the treatment was that of a long duration, 7% felt they were too old for seeking orthodontic treatment, 6% had no interest to do the treatment, 9% stated it would painful, another 16% had concerns that food would get stuck to the braces, 21% were scared to undergo treatment.

Conclusion : The unwillingness of male adolescent patients for seeking orthodontic treatment is studied and the reasons are discussed. This will help in educating them on the long term

effects of malocclusion and to convince them for opting to go ahead with orthodontic treatment.

Keywords*: malocclusion, orthodontic intervention, braces*

Introduction

Orthodontic treatment is the process of aligning the teeth or correcting other irregularities, which thus improves health, function, appearance, and social well-being [1, 2]. Males are generally more concerned about the comfort rendered during treatment of their teeth. Understanding the factors that decide the requirement for orthodontic treatment will enable a more systematic planning and also better assessment of treatment needs and patient's priorities [3].

There is a lack of research on the motivation of adults seeking orthodontic treatmentespecially in men [4]. Both positive and negative motivations are correlated with adherence to treatment [5]. In orthodontic clinical practice, as well as in other dental specialties, treatment success does not depend solely on factors such as proper diagnosis, biocompatibility, and skill of the dentist but also depends on the patient's cooperation [6]. If the patient is not cooperative and does not follow the instructions given by the dentist it may lead to unsatisfactory outcomes of the treatment adolescence is a period when important physical, social, and emotional changes appear [7, 8].

The cooperation of the adolescent patients can be altered based on extrinsic factors (social stereotypes, gender, educational level, family) and the intrinsic factors (personality, temperament). The unwillingness of male adolescent patients with malocclusions for orthodontic treatment is studied so as to be able to educate them on the long term effects of malocclusion. Hence we can convince them for opting to go ahead with orthodontic treatment.Self-perception of orthodontic treatment plays a major role in bringing the patient to the orthodontist for treatment and the perception varies according to psychological status, basic knowledge of malocclusions and also perception changes according to the comments an individual receives from the surrounding people [9].

Social psychologists have described various theories on patient compliance. Many components of 'Health Belief Model' are applicable to orthodontic treatment situation. Variables like treatment-seeking behavior, personality type and barriers to action comply well with the compliance of the patient [10,11]. A compliant orthodontic patient may be described as a patient who practices good oral hygiene, follows appropriate diet, maintains

appliance without breaking, keeps regular appointments, and follows instructions of the doctor. The purpose of the present study is to evaluate the current perspective of Indian orthodontic patients towards not seeking orthodontic treatment.

Materials and Methods

A questionnaire based study was conducted at the department of Orthodontics, Saveetha Dental College, a total of 50 male subjectswith malocclusions requiring orthodontic interventions were selected for the study. Data was collected using a structured, self-administered questionnaire which was designed after reviewing recent articles, developments and also similar questionnaires that are based on the objectives of the study.

The questionnaire was divided into three parts. The first part included questions based on awareness of malocclusion, the second part consisted of questions related to their knowledge on sequelae of malocclusions and the final part consisted of an open question asking them to state out their reason for not undergoing orthodontic treatment. All questionnaires were anonymously collected and the data was kept confidential and not used for anything apart from the study purpose.

Questionnaire

This questionnaire was modified from the questionnaire of Ingervall *et al*. and Ng'ang'a *et al.* [12, 13]The questionnaire contained the following questions:

1. Do you find that your teeth are irregular (not straight)?

2. If your teeth are irregular, does this affect your::

3. If you think you need orthodontic treatment, why haven’t you done so yet?

Results

Figure 1 indicates, that69% of them were aware that they had a malocclusion whereas the remaining 31% were not aware.When asked about the sequelae of malocclusion, 48% of adolescent males pointed out the difficulty in mastication during orthodontic treatment while 24% said that food accumulation is a problem, 9% stated that compromised aesthetics was their concern and 19% were concerned of dental caries that could occur during treatment. On asking as to why they were not correcting the malocclusion, 18% stated that it was embarrassing to wear braces, 4% stated that the treatment was too expensive, 19% stated that the treatment was that of a long duration, 7% felt they were too old for seeking orthodontic treatment, 6% had no interest to do the treatment, 9% stated it would painful, another 16% had concerns that food would get stuck to the braces, 21% were scared to undergo treatment.

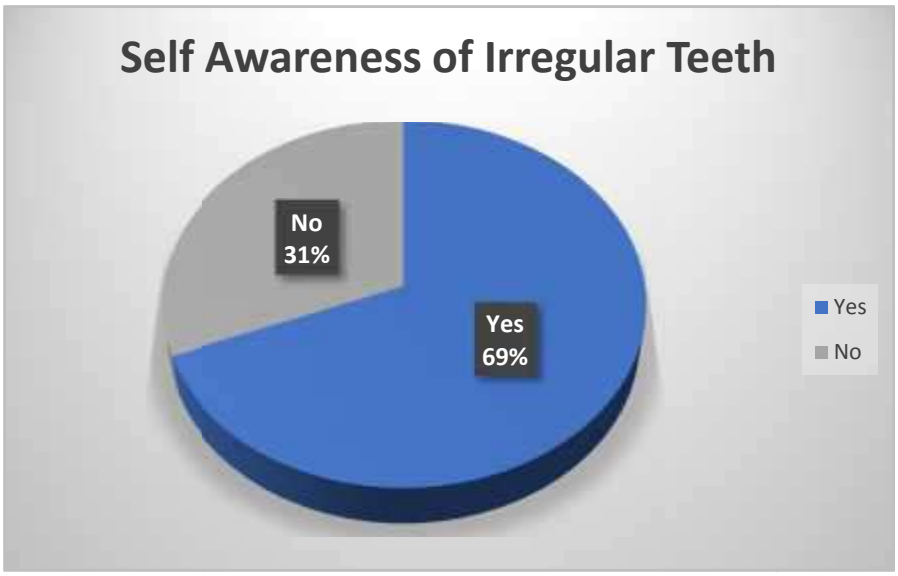


Figure1- Pie chart depicting self awareness of irregular teeth.

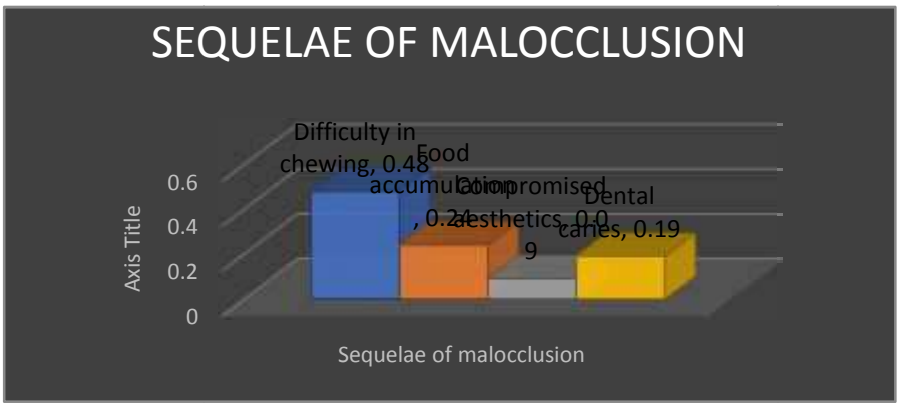


Figure 2- Bar chart of sequelae of malocclusion.

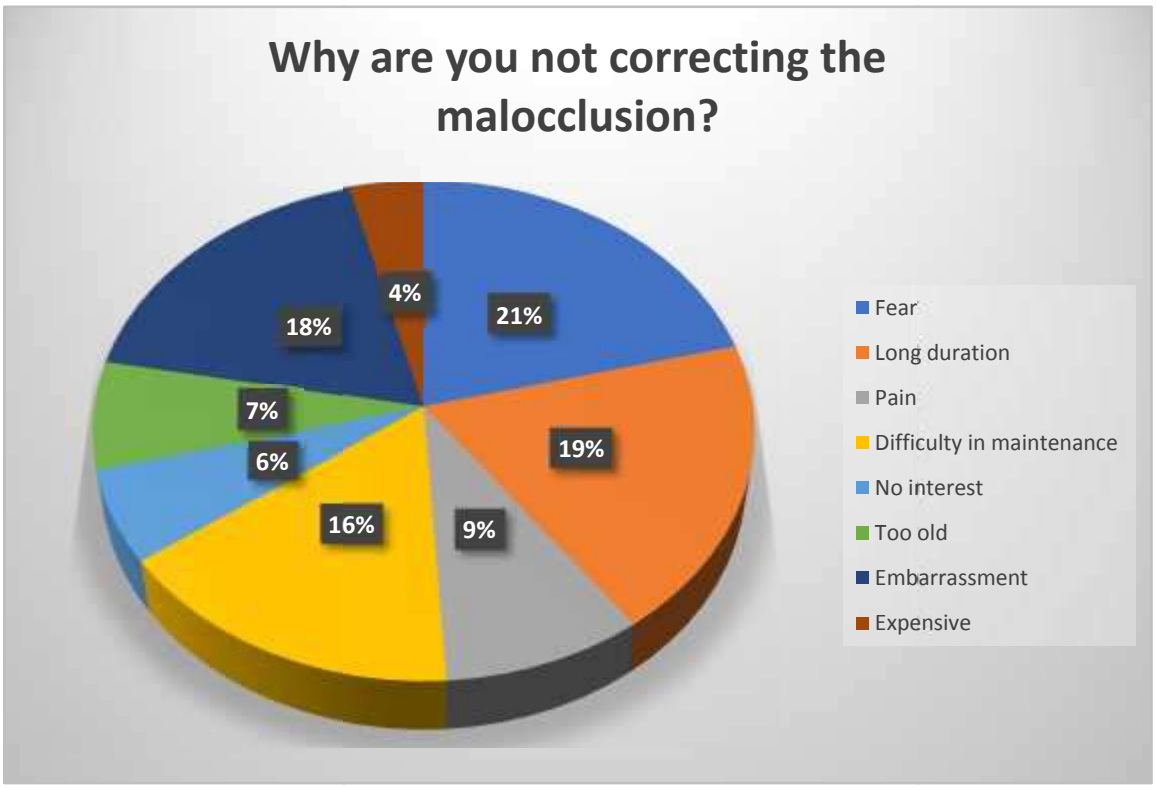


Figure 3- Pie chart of reasons for not correcting irregular teeth.

Discussion

Most adults who seek orthodontic treatment do so because they feel their smile is the most important feature of their facial appearance, and they wish to improve their smile to feel happier about their looks and enhance their self-esteem. Some have problems with their teeth

and gums, or they are just unhappy with their smile and the irregularity of their teeth. Other adults do so to improve their bite, chewing, or to make it easier to maintain good oral hygiene.

This survey conveyed that patients had satisfactory knowledge of whether they possess malocclusions or not as well as the sequelae of the same. When looking at the factors contributing to the negative attitude of patients in not seeking orthodontic treatment, fear, no interest and time constraints were the main concerns.Closely following were the concerns of difficulty in maintenance and embarrassment. The movement of teeth by orthodontic appliancestypically causes some discomfort to the patient. It has been reported that fear of pain is a key factor discouraging a patient from seeking orthodontic treatment [14].

In our study sample we conclude that for male patients, esthetics was not a major reason for seeking orthodontic treatment hence they lacked the motivation to seek orthodontic treatment and that can be a main reason for lack of interest towards orthodontic treatment.

In the study done byAl Zubair NM, the most common reason for not seeking orthodontic treatment in children was

1) Lack of awareness about orthodontic treatment;

2) Fear of pain and of tooth removal [15].The predominance of fear of pain is similar to that of Batayine [16] Al-Huwaizi [17] andGatchel [18].

Various studies identified female orthodontic patients as more cooperative than males [4-7]. The present study showed similar findings that maleadolescents were concerned more about pain and time duration of orthodontic therapy.

Egolf et al[5] studied history factorsassociated with compliance. These factors were considered as a combination of personality type, negative motives and positive motives. This study assessed the reason for patients not going forward with orthodontictreatment.The assessment on attitude of the patient was related to perception on treatment service, cost and also personal factors such as embarrassment.

The factors contributing to the above mentioned study vary from the present study due to variations in sample size and geographical variations.

These information are pertinent for better clinical management for service providers. It can help improve their service and enhance rapport with the patients. Actually, orthodontist's interpersonal behaviour and role in patient motivation is another aspect of successful orthodontic treatment. The orthodontist should give positive feedback and communicate with the patient on the issues of patient cooperation and their inconveniences. Orthodontists should explain about the appliance including retainers, and advice on oral hygiene, dietary control and appliance maintenance methods. Patients who are dissatisfied with the treatment and interpersonal aspects tend to avoid care and jeopardise the name, and fame of the orthodontic practice and the practitioner.

Debates about the "ideal" timing of orthodontic treatment have focused on issues of biologic development and readiness. In this article we examine psychologic issues that should be considered in the decision to initiate orthodontics in the younger child or to wait until adolescence or later. Psychologic development during the preadolescent and adolescent stages may influence the child's motive for, understanding of, and adherence to treatment regimens.

Conclusion

The oral-facial region is usually an area of significant concern for the individual because it draws the most attention from other people in interpersonal interactions and is the primary source of vocal, physical, and emotional communication. As a result, patients who seek orthodontic treatment are concerned with improving their appearance and social acceptance, often more than they are with improving their oral function or health. Enhancing these aspects of quality of life is an important motive for undergoing orthodontic treatment. Regardless of age, patients' and their parents' or caregivers' expectations about improvements in oral function, esthetics, social acceptance, and body image are important for both general dentists and orthodontists to consider when advising patients about these procedures and during the treatment process. This review of research on the impact of conventional and surgical orthodontics on quality of life examines the association between oral health-related quality of life and severity and type of malocclusion, as well as the impact of treatment and patient characteristics on quality of life. It is good to emphasise the importance of clinicians' having a clear understanding, before initiating treatment, of their

patients' quality of life and their expectations about improvements in specific domains of quality of life [19].

The unwillingness of male adolescent patients for seeking orthodontic treatment is studied and the reasons are discussed. This will help in educating them on the long term effects of malocclusion and to convince them for opting to go ahead with orthodontic treatment. Despite having good knowledge on orthodontic treatment, patient's attitude and practice towards orthodontic treatment is mostly moderate. It is important for practicing orthodontists to inform patients about the retainers, appliance maintenance and build inter-personal rapport with the patients. The practitioners shall improve the patient attitude by shortening the waiting time of patients at waiting room, adequately spend time on procedure and charge the treatment cost more reasonably [20].

Corresponding Author
Ravindra Kumar Jain
Reader,Department of Orthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India.
Telephone Number: +91 9884729660
E-mail: orthoravi2@gmail.com

References:

1. Liu Z, McgrathC, Hägg U. The Impact of Malocclusion/Orthodontic Treatment Need On The Quality Of Life. A Systematic Review.Angle Orthod. 2009; 79(3):585–591.
2. Zhang M, McgrathC, Hägg U. The Impact Of Malocclusion And Its Treatment On Quality Of Life: A Literature Review. Int J Paediatr Dent. 2006; 16(6):381–387.
3. Jayachandar D, Saravana Dinesh S P. Factors Affecting Patient's Desire For Seeking Orthodontic Treatment. Int J OrthodRehabil2016; 7(3):89-91
4. Pabari S, Moles DR, Cunningham SJ. Assessment of Motivation and Psychological Characteristics Of Adult Orthodontic Patients. Am J Orthod Dentofacial Orthop.2011; 140(6):E263–E272.

5. Egolf RJ, Begole EA, Upshaw HS. Factors Associated With Orthodontic Patient Compliance With Intraoral Elastic And Headgear Wear. Am J OrthodDentofacialOrthop. 1990; 97(4):336–348.

6. Arnrup K, Broberg AG, Berggren U, Bodin L. Treatment Outcomein Subgroups of Uncooperative Child Dental Patients: An Exploratory Study. Int J Paediatr Dent. 2003; 13(5):304–309.

7. Cucalon A, Smith R. Relationship Between By Adolescent Orthodontics Patients and Performance On Psychological Test. Angle Orthod. 1990; 60(2):253–289.

8. Slakter MJ, Albino JE, Fox RN, Lewis EA. Reliability and Stability Of The Orthodontic Patient Cooperation Scale. AmjOrthod. 1980; 78(5):559–563.

9. Shilpa R. Self Perceived Orthodontic Treatment Need In Dental Students. Int. J. Pharm. Sci. Rev. Res., 2017; 46(2):39-44.

10. Haynes RB, Taylor DW, Sackett DL. Compliance in Health Care Baltimore Johns Hopkins University Press. As seen in DiMatteo (1993) Health Psychology. 1979; 12:93-102.

11. Tulloch JFC, Shaw WC, Underhill BDS, Smith A, Jones G, Jones M. A Comparison Of Attitudes Toward Orthodontic Treatment In British And American Communities. Am J Orthod1984; 85(3):253-9.

12. Ingervall B, Moulin B, Thilander B. Prevalence and Awareness of Malocclusion InSwedish Men. Community Dent Oral Epidemiol1978; 6(6):308-14.

13. Ng′Ang′APM, Stenvik A, Ohito F, Ogaard B. The Need and Demand for Orthodontic Treatment in 13- To 15-Year-Olds inNairobi, Kenya. Acta OdontolScand1997; 55(5):325-8.

14. Wilson S, Ngan P, Kess B. Time course of the discomfort in young patients undergoing orthodontic treatment. Pediatr Dent. 1989 Jun;11(2):107-10.

15. Al-Zubair NM. Perception of Occlusion and Reasons for Not Seeking Orthodontic Treatment amongYemeni Children. J Orthod Res 2014; 2(2):68-73.

16. Batayine FA. OcclusalFeaturesand Perception of Occlusion ofJordanian Adolescents: A Comparative Study withanIraqi Sample. Master Thesis. Iraq: College OfDentistry, University Of Baghdad; 1997.

17. Al-Huwaizi AF. OcclusalFeatures, Perception of Occlusion, Orthodontic Treatment Need and Demand among 13-Year-Old Iraqi School Children. Doctoratethesis.Iraq: College OfDentistry, University Of Baghdad; 2002

18.Gatchel RJ. The Prevalence of Dental Fear and Avoidance: Expanded Adult and Recent Adolescent Surveys. J Am Dent Assoc1989; 118(5):591-3.

19. Tung AW, Kiyak HA. Psychological influences on the timing of orthodontic treatment. American Journal of Orthodontics and Dentofacial Orthopedics. 1998 Jan 1;113(1):29-39.

20.Kiyak HA. Does orthodontic treatment affect patients' quality of life?. Journal of dental education. 2008 Aug 1;72(8):886-94.

Frequency Of Denture Cleansing – A Survey

Sruthi Srinivasan[1], Pon Kirubha Robin[2], Vinoth Kumar S[3]
Graduate student[1,] Senior Lecturer[2], Department of Prosthodontics,
Senior Lecturer[3], Department of Prosthodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: Most of the patients clean their dentures only once a day. But according to ideal practices the complete dentures should be cleaned regularly at least twice a day to prevent ulcers, periodontal diseases and various lesions in the oral cavity. Like natural teeth dentures also should be cleaned with a brush or with denture cleaners. This must be done regularly to prevent food accumulation and plaque formation. The most common outcome of improper cleaning of the dentures may lead to a condition called denture stomatitis which is the inflammation of the mouth due to poor oral hygiene. Hence, it is necessary to clean the dentures properly to avoid the above mentioned problems.

Materials and methods: The study was conducted with 50 patients of age groups ranging from 40 to 65 years. A questionnaire was prepared and the information was collected based on details such as age, gender, duration of denture wearing, frequency of denture cleansing, the materials used to clean the dentures and nocturnal denture wearing habits. The purpose of the study was explained to the patients and their consent along with their signature and was recorded on the questionnaire.The data was later tabulated and statistical analysis was done using SPSS software and the results were given.

Result : The present study reveals that most of the patients were aware about importance of denture cleansing and materials used to clean the dentures. They were also aware about the technique to clean dentures and also about the nocturnal use of dentures and adverse effects of not maintaining their dentures properly.

Conclusion : Based on the study, the denture hygiene of patients has significantly improved with patients brushing their dentures twice a day or sometimes thrice a day with cleansing liquid and tablets. Still there are a few patients who slept with their dentures at night which should be avoided. Thus dentists should take the responsibility of providing post placement denture hygiene instructions and also to motivate the patients.

Key Words *Dentures, Frequency, Cleansers, Elderly*

Introduction

Replacement of missing teeth is successful only when patients are motivated and aware of correct prosthesis use and hygiene. The quality of the denture, occlusion, denture age, and hygiene are important factors to be considered 1.. The most common lesions include denture stomatitis, angular chelitis, traumatic ulcers, denture irritation hyperplasia or epulis fissaratum, flabby ridges and can even result in oral carcinomas 2.. These lesions are acute or chronic reactions to plaque or a reaction to constituents of the acrylic base, or a mechanical denture injury.Denture stomatitis is an inflammation of the oral mucous membrane involving the palate mucosa when it is completely or partially covered by denture 3. .Food particles may get lodged between the denture and gingiva or the palate and allows the growth of Candida species which may cause denture stomatitis 4.. Denture stomatitis may also be caused due to wearing dentures at night, smoking, age of the denture and poor oral hygiene 5, 6.. Hence once an edentulous patient is fitted with a complete denture, the most important phase of the treatment will be proper denture maintenance. Tooth loss in any adult population is common as the age advances because the factors that leads to the loss of teeth – dental caries, loss of periodontal support, a history of dentoalveolar trauma, a history of dental care – are additive over time. For this reason, the rates of complete tooth loss are customarily the highest in the oldest age groups mostly after the 5^{th} decade of life 7..

Loss of teeth in elderly people is very common and it may be due to caries, periodontal infections, trauma or bone resorption. Hence in such cases, the only possible treatment will be the fabrication of a complete denture. This treatment will be successful only if the patients are fully aware of denture maintenance and hygiene 8, 4.. Proper care of the dentures and the surrounding oral mucosa is mandatory to avoid certain complications. Poor denture hygiene can lead to various complications like denture stomatitis, angular chelitis, traumatic ulcers and oral carcinomas 9, 15..

For elderly people, proper care of mucosal tissues and care of the dentures are mandatory to maintain the overall health. In addition, there may be greater social consequences of mouth malodour due to unclean oral prosthesis for someone whose dietary intake is strongly linked to socialisation, such as an older person who attends a senior activities

centre for meals. Dentures which are not cleansed adequately cause or contribute to oral mucosal lesions, impairment in eating, and thus will have a more profound effect on a fragile elder than on a younger, healthier person 10, 11..

Various methods of denture cleansing are available such as mechanical and chemical methods. The most common and widely used is the mechanical method of denture cleansing which involves using a toothbrush. There are brushes specifically designed and sold commercially for this purpose15.. Chemical denture cleansers are immersion type cleansers which are sold in powder and tablet forms. They can be broadly divided into alkaline peroxides, alkaline hypochlorite, acids (hydrochloric acid and phosphoric acid), and disinfectants (chlorhexidine gluconate and salicylate) 15.. Combination of mechanical method and chemical method is the most recommended as well as effective and safe method to clean dentures, for persistent accumulations and stains.

Patients should be instructed to rinse their dentures and their mouths after meals whenever possible. The mucosal surfaces of the residual ridges and the dorsal surface of tongue also should be brushed daily with a soft brush; denture cleansers may also be used. However, it has been observed that the majority of denture wearers do not pay necessary attention to the cleanliness. This may be due to decreasing manual abilities due to advanced age 12, 13, and 14.. Thus it is the primary duty of the dentist to create awareness about denture hygiene and also about the products available to clean the dentures. So, this cross-sectional study aimed to investigate the denture cleansing habits among denture wearers.

Materials and Methods

The study was conducted with 50 patients of age groups ranging from 40 to 65 years. A questionnaire was prepared and the information was collected based on details such as age, gender, duration of denture wearing, frequency of denture cleansing, the materials used to clean the dentures and nocturnal denture wearing habits. The purpose of the study was explained to the patients and their consent along with their signature and was recorded on the questionnaire.The data was later tabulated and statistical analysis was done using SPSS software and the results were given.

Statistical Analysis

The tabulated data were analysed using SPSS version 17.0. Descriptive statistics were obtained and the frequency distribution along with the cumulative percentage was also calculated. The analysed data was interpreted.

Results

Out of 50 patients who were interviewed, 50% (25) were males and 50% (25) were females and 46% of patients have been wearing their dentures for 1 to 3 years, 40% of patient'shave been wearing their dentures for 5 to 10 years and 14% have been wearing dentures for more than 10 years. This data is evident in table -1.

Table-1 showing Duration of denture wearing

Duration of denture wearing	Frequency	Percent	Valid Percent	Cumulative Percent
1-3	23	46.0	46.0	46.0
5-10	20	40.0	40.0	86.0
>10	7	14.0	14.0	100.0
Total	50	100.0	100.0	

Out of the 50 patients38 patients (76%) were comfortable with their dentures while 12 patients (24%) were not comfortable with the dentures and experienced some problems.
The data is shown in table -2.

Table-2 showing Comfort level of patients

Comfort level	Frequency	Percent	Valid Percent	Cumulative Percent
Yes	38	76.0	76.0	76.0
No	12	24.0	24.0	100.0
Total	50	100.0	100.0	

The next topic which was asked was about the method the patients used to clean their dentures. It was concluded that 46% of patients used tooth brush to clean their dentures,

while 40% of patients used their hand to clean the dentures and the remaining 7% used materials like cotton or cloth to wipe their dentures. The data is shown in table-3.

Table -3 showing the cleansing methods used

Methods used for cleaning	Frequency	Percent	Valid Percent	Cumulative Percent
Tooth brush	23	46.0	46.0	46.0
Hand	20	40.0	40.0	86.0
Others	7	14.0	14.0	100.0
Total	50	100.0	100.0	

When the patients were asked about the material they used to clean (table-4) their dentures, it was seen that 46% of patients used their tooth pastes to clean; 28% used cleansing powder and 26% used cleansing liquid to clean their dentures.

Table-4 showing material used to clean

Materials used to clean	Frequency	Percent	Valid Percent	Cumulative Percent
Tooth paste	23	46.0	46.0	46.0
Cleansing powder	14	28.0	28.0	74.0
Cleansing tablet	13	26.0	26.0	100.0
Total	50	100.0	100.0	

The patients were also asked about their awareness of the cleansing products available in the market. To this 68% of patients gave a positive reply and 32% gave a negative answer.

Table-5 showing the awareness towards cleansing products

Awareness	Frequency	Percent	Valid Percent	Cumulative Percent
Yes	34	68.0	68.0	68.0
No	16	32.0	32.0	100.0
Total	50	100.0	100.0	

The most important aspect of this survey was to know the frequency of cleansing. When the patients were questioned regarding this, it was observed that 22% of patients cleaned their dentures only once a day, while 56% of patients cleaned their dentures twice a day.

Cleaning their dentures was associated with the advice of the dentist. Patients who followed their regular dental check up kept their dentures cleaner compared to the others.

Table-6 showing frequency of cleansing

Frequency of cleansing	Frequency	Percent	Valid Percent	Cumulative Percent
Once	11	22.0	22.0	22.0
Twice	28	56.0	56.0	78.0
Others	11	22.0	22.0	100.0
Total	50	100.0	100.0	

The next question that was asked was about the nocturnal denture wearing habits. It was seen that 54% of patients slept with their dentures and 46% of patients removed their dentures during night.

No significant difference was observed between gender and denture hygieneand alsoage and denture hygiene.

Table-7 showing the nocturnal use of dentures

Nocturnal use of dentures	Frequency	Percent	Valid Percent	Cumulative Percent
Yes	27	54.0	54.0	54.0
No	23	46.0	46.0	100.0
Total	50	100.0	100.0	

Discussion

Poor denture hygiene has a negative impact on elderly people and can cause many systemic manifestations and infections. When a denture is placed in the mouth, a coating of glycoprotein develops.This thin pellicle becomes contaminated with food debris and microorganisms. Hence, denture stained by tobacco and medicines results in unpleasant taste and malodour. This favourscandidalgrowth and proliferation in the denture leading to mucosal irritation. Denture care is, therefore, an imperative step to maintain denture quality, aesthetics and longevity to ensure good oral health status.Thus it is essential to maintain proper denture hygiene regularly 15..

In this study it is seen that 46% of patients have been wearing dentures for the past 1 to 3 years only whereas 40% of them have been using dentures for 5-10 years and only a small portion of patients are long term denture wearers that is for more than 10 years. It is also observed that 76% of patients were comfortable with their dentures whereas 26% experienced some problem and were not comfortable in using their dentures.

It is also seen that 46% of patients used tooth brush to clean their dentures and 40% used only their hand to clean their dentures and 7% of patients used substances like cotton or cloth to just wipe their dentures without washing them. It is thus seen that these patients have either not received the appropriate information from their dentist or have not been following them as per the instructions given.

When the patients were assessed for their awareness about various denture cleansers available in the market it was observed that 68% of patients were aware about the availability of denture cleansers in the market whereas 32% of patients were totally not aware about such cleaning agents.

The most important aspect of this study was to assess the frequency of denture cleansing and it was seen that 22% of patients cleaned their dentures once a day whereas 56% of patients cleaned their dentures twice a day. Thus it is observed that majority of patients are cleaning and maintaining their dentures properly.

It is also observed that majority of the patients remove their dentures during night. Patients were also additionally aware that nocturnal use of dentures may result in denture stomatitis and other oral mucosal lesions.

It was also observed that gender and denture hygiene were not significantly related. None of the patients were aware of denture brushes and they used ordinary tooth brushes to brush their dentures. Thus it is essential that dentists give all the possible instruction to their patients on how to clean the dentures and also the oral mucosa to maintain a good oral hygiene.

Peracini et al. reported 58.49% of the patients using immersion for cleaning and among the substances used for immersion of the dentures, water was the most frequently used (38.71%) followed by sodium hypochlorite (33.87%). In the study by Baran and Nalça, 42.9% of the patients immersed their dentures in water and only 1.6% immersed them in hypochlorite solution. Hoad-Reddick et al. found that a combination of methods (brushing and soaking) was used more frequently 1..

The primary objective of maintaining the complete denture is to prevent the occurrence of lesions like commissural chelitis, burning mouth syndrome, mouth ulcer, denture irritation hyperplasia, gagging leading to caries and periodontal disease in the oral cavity. In elderly patients, care of the mucosal tissues and the dentures of the edentulous mouth are very important for overall health. The main aim of cleaning the denture is to remove the plaque adhering to the denture which in turn will eliminate the cause of denture stomatitis.Denture cleansers can be used for this purpose. An ideal denture cleanser should be simple to use, should be efficient in removing organic and inorganic matter or plaque from denture surface, have antimicrobial properties and be compatible with all denture base materials 16.. Chemical agents for denture cleansing have advantage of being simple to use, and several investigations have shown their efficacy in reducing biofilm formation in vitro 17-19. and in vivo 20, 21.. Thus it is the duty of every dentist to educate the patient about the importance of denture hygiene and frequency of cleaning the dentures 22..

Conclusion

Based on the study, the denture hygiene of patients has significantly improved with patients brushing their dentures twice a day or sometimes thrice a day with cleansing liquid and tablets. Still there are a few patients who slept with their dentures at night which should be avoided. Thus dentists should take the responsibility of providing post placement denture hygiene instructions and also to motivate the patients.

Corresponding author

VinothKumar. S

Senior Lecturer,Department of Prosthodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Email: doctor.vinoth.kumar@gmail.com

Telephone number: +919865659599

References

1. Budtz-Jørgensen E. Oral mucosal lesions associated with the wearing of removable dentures. J Oral Pathol. 1981; 10(2):65–81.
2. Barbeau J, Seguin J, Goulet, JB, de Koninck L, Avon SL, Lalonde B. Reassessing the presence of Candida albicans in denture-related stomatitis. Oral Surg Oral Med Oral Pathol. 2003; 95(1):51-9.
3. Manderson RD, Ettinger RL. Dental status of the institutionalized elderly population of Edinburgh. Community dent oral Epidemiol 1975;3(3):100-107.
4. Gendreau L, Loewy ZG. Epidemiology and aetiology of denture stomatitis. J Prosthodont 2011; 20(4): 251-60.
5. Parizi MT, Taheri S, Amini P, Afshar MK, Afshar MK. Evaluation of denture hygiene among removable denture wearers referred to clinics of Kerman, Iran. Journal of Oral Health and Oral Epidemiology. 2013;2(1):44-8.
6. Patel IB, Madan G, Patel B, Solanki K, Chavda R. Behaviours and hygiene habits of a sample population of complete denture wearers in Ahmadabad. J Int Oral Health. 2012; 4(2):29–38.
7. Budtz-Jørgensen E. Oral mucosal lesions associated with the wearing of removable dentures. J Oral Pathol. 1981; 10(2):65–83.

8. Douglass CW, Gammon MD, Atwood DA. Need and effective demand for prosthodontic treatment. Journal of Prosthetic Dentistry. 1988;59(1):94-104.
9. Shay K. Denture hygiene: A review and update. J Contemp Dent Pract. 2000; 1(2):28–41.
10. Abelson DC. Denture plaque and denture cleansers. Journal of Prosthetic Dentistry. 1981;45(4):376-9.
11. Manderson RD, Ettinger RL. Dental status of the institutionalized elderly population of Edinburgh. Community Dent Oral Epidemiol. 1975; 3(3):100–7.
12. Kanli A, Demirel F, Sezgin Y. Oral candidiasis, denture cleanliness and hygiene habits in an elderly population. Aging Clin Exp Res. 2005; 17(6):502–7.
13. Polyzois GL. Denture cleansing habits. A survey. Aust Dent J. 1983; 28(3):171–3.
14. Namrata M. A questionnaire survey of dental students' attitude toward denture cleansing. Int J Orofac Res 2017; 2(1):8-10.
15. Ingram DM, Bosse GM, Baldwin R. Ingestion of a denture cleanser: Did it cause gastric perforation?. Journal of Medical Toxicology. 2008 Mar 1;4(1):21-4.
16. Jagger DC, Harrison A. Denture cleansing--the best approach. British dental journal. 1995 Jun 10;178(11):413.
17. Nikawa H, Yamamoto T, Hamada T, Sadamori S, Agrawal S. Cleansing efficacy of commercial denture cleansers: ability to reduce Candida albicans biofilm activity. International Journal of Prosthodontics. 1995 Nov 1;8(6).
18. Webb BC, Thomas CJ, Harty DW, Willcox MD. Effectiveness of two methods of denture sterilization. Journal of oral rehabilitation. 1998 Jun;25(6):416-23.
19. Jagger DC, Al-Akhazami L, Harrison A, Rees JS. The effectiveness of seven denture cleansers on tea stain removal from PMMA acrylic resin. International Journal of Prosthodontics. 2002 Nov 1;15(6).
20. Gornitsky M, Paradis I, Landaverde G, Malo AM, Velly AM. A clinical and microbiological evaluation of denture cleansers for geriatric patients in long-term care institutions. Journal-Canadian Dental Association. 2002 Jan;68(1):39-45.
21. Roshene R, Robin P, Raj JD. A Survey of Denture Hygiene in Older Patients. Journal of Pharmaceutical Sciences and Research. 2015 Oct 1;7(10):897.

General Dental Practitioners Knowledge of Dental Radiography among Dentist in Chennai,India.

Vijayaalakshmi.L.G[1],Shantha[2], V. Jayanth Kumar[3]
Graduate Student[1], Lecturer[2], Department of Pedodontics,Reader[3], Department of Oral Medicine and Radiology,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Dental radiographs are one of the principal diagnostic methods used in dentistry.The safe and effective use of x-ray equipment is important to the protection of the patient and members of the dental team.The aim of this research is to evaluate the general dental practitioner'sknowledge of dental radiography.

Materials and Methods: The data was collected from 50 general dental practitioners at self convenience using a structured questionnaire consisting of 9 item questionnaire.The sample was selected based on inclusion criteria and exclusion criteria.Inclusion criteria includes undergraduate,postgraduate and chennai dental practitioners. The exclusion criteria includes the students and dental radiograph assistants.In the questionnaire, details were asked about the age of the intraoral radiographic unit, kVp and mA of the equipment, the type of timer on the machine and about the geometry of the machine and the geometric technique used for intraoral radiography.Furthermore, the questionnaire contained questions about the type of intraoral detectors that were used and whether the dentists were also using panoramic machines and, if so, which type of detector they had. Among the possible answers, one option was ''no idea''.The data thus obtained was subjected to statistical evaluation.

Result: 80%(40 dentist)of the equipment used in South chennai dentist was reported to operate between 60 and 70 kVp. ,whereas 20%(10 dentist) claimed to be working at a tube voltage of 50 kVp.

Conclusion: The results of this study showed that the knowledge of dentist in South Chennai regarding dentomaxillofacial radiology is excellent.The level of awareness of practitioners regarding radiation hazards and safety was found to be acceptable.

Key Words: *general practitioners,knowledge, radiology, radiation protection,exposure.*

Introduction

As a dental professional, the utilization of radiograph for finding and further treatment is significantly more regular than different fields of medicine.It is accounted for that around 45% of dental patients require radiographs for determination. (1)Dental radiographs is one of the essential demonstrative techniques utilized as a part of dentistry.Intra oral and extra oral radiographs are two sorts of radiographs available.The sheltered and powerful utilization of x-beam hardware is imperative to the assurance of the patient and individuals from the dental team.Its assumes a critical part in the identification of oral disease.(2)Exposure to such radiation is related with an expanded hazard in the long haul of threatening sickness in those persons.Furthermore, it is accepted that the likelihood of event of these antagonistic impacts is straightforwardly corresponding to the level of presentation, with no measurements threshold.The dangers related with the vital introduction to ionizing radiation might be generous, and must be limited through careful adherence to great practice.(3)

Specialized advances in radiological equipment significantly affect lessening of radiation dosages to patients during intra oral and extra oral radiographs. During extra oral radiography, the utilization of uncommon earth escalating screens has diminished the radiation presentation significantly.(1,4,5)Although the quantity of radiographs taken every day in essential dental care is over the top, the most serious hazard emerges when there is proof of poor picture quality or non-diagnostic images inferable from poor specialized information and the insufficient handling of films (6-10). It has been evaluated that the disposal of non-beneficial examinations could prompt a 30% lessening in the aggregate populace measurements, got from medical radiology.(11) Dental hygienists assume an imperative part in handling and they should accept the accountability for poor preparing rehearses, the majority of which concern not supplanting preparing arrangements as oftentimes as required, overdeveloping and overfixing. Accordingly, numerous dental

practitioners tend to build the presentation time to make up for the disgraceful preparing, therefore expanding the radiation measurements to patients. (12)

For dispensing with unnecessary X-beam examinations, each expert who works X-beams ought to be appropriately prepared as per the Ionizing Radiation Medical Exposure Regulations 2000.(13) We live in an ocean of radiations. We are constantly exposed to naturally occurring ionizing radiation i.e.,background radiation and furthermore presented to ionizing radiation from manmade sources, for the most part through medical procedures.On an average, doses from a diagnostic X-ray are much lesser, in dose effective terms, than the natural background radiation.Radiation has turned into a part of present day living, achieving each section of our general public. The essential hazard from dental radiography is radiation actuated malignancy. The writing on conceivable destructive impacts of expert analytic presentation for dental specialists isn't steady. The hazard required with dental radiography is surely little in correlation with numerous different dangers that are a typical piece of regular daily existence. In any case, no premise exists to accept that it is zero. Likewise, the biologic impacts of ionizing radiation ingested during dental radiography are questionable. The radiographic examination utilized as a part of all fields of medicinal administrations and adds to the advancement of the well being, both separately and broadly. Radiographic examination has a basic impact of dental practice. Certain measure of radiation is unavoidably conveyed to patients, it ought to be as low as reasonably achievable (ALARA). International Commission for Radiation Protection (ICRP) is the administrative body which sets down standards for radiation security at the universal level. In India, it is the Atomic Energy Regulatory Board (AERB) which gives the standards to radiation assurance.

AERB prescribes standards for reasonable dosages of radiation from X-beam tubes, the protecting required for the dividers of a X-beam tube room, the lead equal protecting attire to be worn by radiation specialists and sets down safe measurements limits for radiation laborers and for the overall population.Before undertaking any radiological examination, it is important that the provider comprehends the potential dangers and the advantages of radiation. The dangers can be stochastic (of which likelihood increments with dosage) and deterministic (of which severe increments with measurement).Cancer induction and hereditary effects are stochastic impacts and blood dyscrasias,cataracts and imparired fertility are cases of deterministic effects.Biological effects of ionizing radiation are gathered into stochastic and deterministic effects. Over a specific decided dosage of radiation organic harm

starts to show up, this is called as deterministic impact. Stochastic impact is that in which there is no specific dosage level above which natural change happens in the body.

Ionizing radiation causes both the impacts relying upon radiation doses and body's reaction to these radiations. Dental practitioners and in addition patients are more inclined to the danger of stochastic effects because of its lack of a dose threshold limit. The positive parts of diagnosis of disease and detection of disease should be considered while assessing the risk of impacts of radiation. It is essential for dental practitioner to refresh their insight about new patterns in analytic strategies, protective measures, etc.This can be accomplished by methods for proceeding with training, continuing education, journals,workshops, and other media.In India, diagnostic radiation facilities are administered by Atomic Energy Regulatory Board (AERB).The role of the AERB is to guarantee that utilization of ionizing radiation and atomic vitality in India does not make undue hazard the well being of individuals and the environment.It is required to enlist all diagnostic radiation facilities in e-Licensing of Radiation Application (eLORA) system of AERB. From December first, 2013, it is mandatory for dental specialists and dental institutions to enroll in eLORA and get a license to operate dental X-ray units, panaromic machines, and cone bean computed tomography. It is additionally essential for manufacturers of X-ray machines to get a license available to be purchased in India by AERB.

The current radiation protection standards are based on 3 principles : Justification of a practice, optimization and dose limitation.In spite of the fact that the radiation dosage levels in dental practice are generally low, one ought to think about the combined impact of repeated exposures. There ought to be a making progress toward radiation assurance measures in the private dental offices.(14)There is an association between knowledge and the use of low-dose techniques and attitudes towards risks.(15)A dental professional should always be prepared to improve his or her knowledge through continuous education, it is a well-known fact that this is more likely to occur if the continued education is mandatory for all dental professionals, for whatever local or federal reasons.(16)

The reason for performing this study was to alert the dental professional societies that more attention should be paid to radiation protection in dentistry in our country. The aim of this research is to evaluate the general dental practitioners knowledge of dental radiography .

Materials and Method

Data was collected from 50 general dental practitioners using a structured questionnaire consisting of 9 item questionnaire.The sample was selected based on certain inclusion and exclusion criteria.

Inclusion Criteria

1. Undergraduate
2. Postgraduate
3. Chennai dental practitioners

Exclusion Criteria

1.Students

2.Dental radiograph assistants

In the questionnaire, details were asked about the age of the intraoral radiographic unit, kVp and mAofthe equipment, the type of timer on the machine and, about the geometry of the machine and the geometric technique used for intraoral radiography.Furthermore, the questionnaire contained questions about the type of intraoral detectors that were used and whether the dentists were also using panoramic machines and, if so, which type of detector they had. Among the possible answers, one option was ‘‘no idea’’.The data thus obtained was subjected to statistical evaluation.

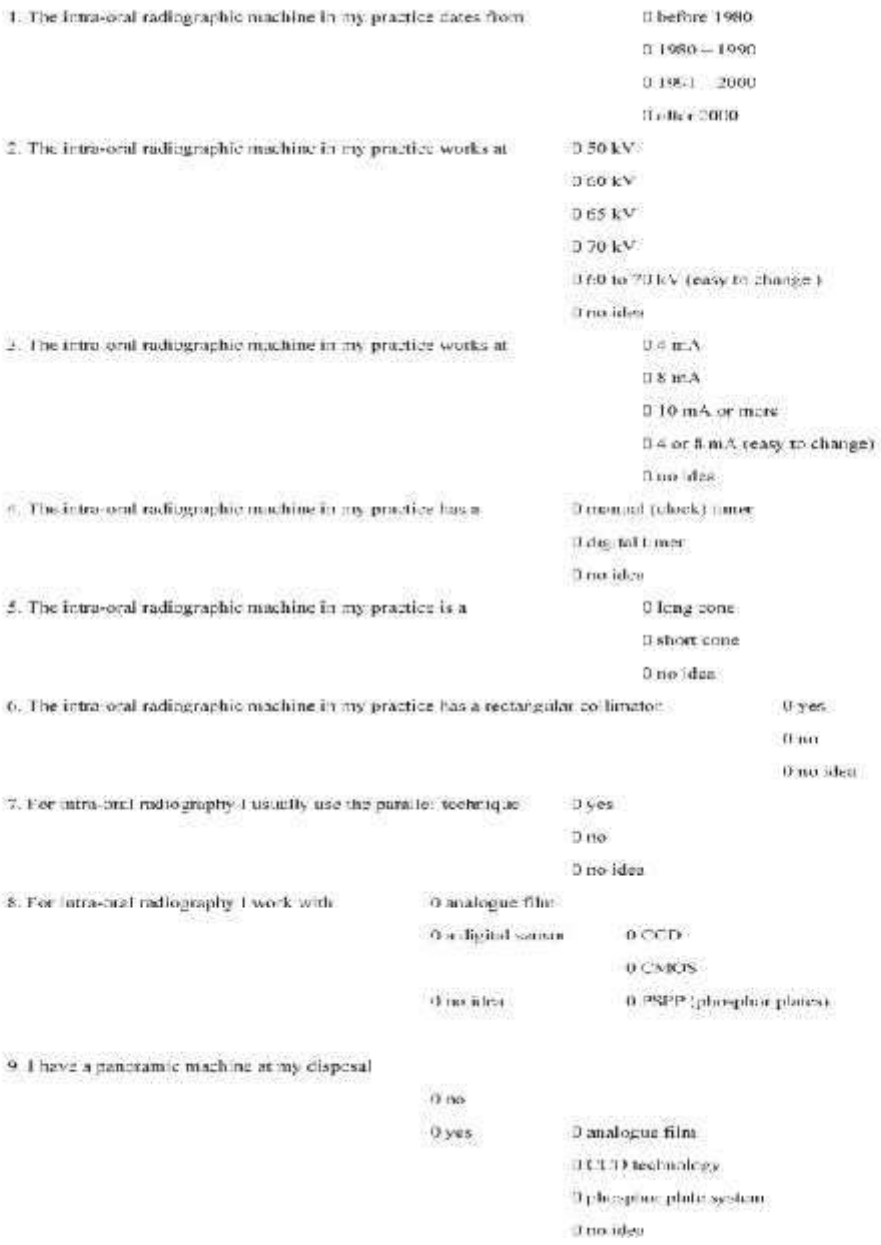


Figure 1: Questionnaire

Result

Table1: Responses from the study subjects to the questionnaire in percentage(%)

Intra - Oral Radiographic Machine	Condition	Percentage
Practice dates from	After 2000	100%
Works at (Kv)	70Kv	20%
Works at (Kv)	60Kv to 70Kv	80%
Works at (mA)	4 or 8mA	92%
Works at (mA)	No Idea	8%
Timer	Digital Timer	100%
Shape	Long Cone	6%
Shape	Short Cone	94%
Rectangular Collimator	Yes	20%
Rectangular Collimator	No	80%
Parallel Technique	Yes	18%
Parallel Technique	No	82%
Works with	Digital Censor - CCD	24%
Works with	Digital Censor - PSPP	76%
Panaromic Machine Disposal	No	10%
Panaromic Machine Disposal	Yes - Analogue Film	80%
Panaromic Machine Disposal	Yes - CCD Technology	10%

Discussion

As mentioned above the data was collected from 50 general dental practitioners using a structured questionnaire consisting of 9 item questionnaire.The sample was selected based on certain inclusion criteria and exclusion criteria.Inclusion criteria which includes the Chennai dental practitioners,undergraduates,postgraduates and Exclusion criteria includes Students,dental radiograph assistants.Regarding the age of the intraoral radiographic equipment used by the questioned population of chennai dentists, it was found that almost 100%(50 dentist) were using radiographic machines for intraoral radiography equipment that was after 2000.

As the answers about the tube voltage of the intraoral equipment was mild difference the answers were condensed into two groups. Therefore, tube voltage ranging from 60 to 70 kV was considered as one category. 80%(40 dentist)of the equipment used in South chennai dentist was reported to operate between 60 and 70 kVp. ,whereas 20%(10 dentist) claimed to be working at a tube voltage of 50 kVp.

The majority of the South Chennai dentists were using 4 or 8mA(easy to change) equipment for intraoral radiographs ; 8%(4 dentist) claimed they not knew what they were working .

100% (50 dentist)claimed to be working with a digital exposure timer, whereas none said they were still working with a manual (clock-like) exposure timer.

It was found that 94% (47 dentist)said that they were working with a short cone geometry, whereas 6% (3 dentist)claimed to be working with a long cone geometry.

20% (10 dentist)said they were using a rectangular collimator.80%(40 dentist) responded that they were using circular collimatior.

18%(9 dentist)claimed to be working with the parallel technique, whereas 82%(41dentist) said they were working with the bisecting angle technique.

76%(38 dentist)of the dentists in South Chennai were still using PSPP sensor films for intraoral radiography. 24%(12 dentist) had digitized to intraoral sensors as charge coupled device (CCD).

About 10% (5 dentist)of the dentists has no intention idea what sensor they using.10%(5 dentist) of South Chennai dentist don't not posses a dental Panaromic machine. Others 80% use analogue films and 10% (5 dentist)use CCD technology.

A dental professional should know his or her equipment and it is expected to know the main guidelines on radiation protection.The overall conclusion of this study,is that a great deal of work done to ameliorate the quality of radiographs and the knowledge and attitude of general dental practitioners regarding dentomaxillofacial radiology.(2) This is a task for dentomaxillofacial radiology specialists, who will have to concentrate in the first place on undergraduate teaching and then on postgraduate teaching. The latter is not less important than the first as the working dentist population needs to change their attitude regarding radiation protection and dentomaxillofacial radiology general knowledge. (3)This has also been stressed in other surveys. From the results ,it is evident that continued education in dentomaxillofacial radiology is essential, especially when considering a change from analogue to digital radiology.(4)That 92%(46 dentist)of the dentists in the present survey claimed to be working with a short cone radiographic machine is probably biased by the fact that they are not aware of the difference between a short cone and a short spacer cone, which is also called a beam indicating device or position indicating device.(17)It can be assumed that dentists owning a machine with a short spacer cone, because the manufacturer positioned the focus near the rear end of the machine, gave the wrong answer. Therefore this result should be interpreted with care. The opposite for long cone could also be the case, of course.

(18)The results are, therefore , excellent.This could mean, for instance,increased in the number of hours of under and /or postgraduate education. If this can be decided by the local authorities and enforced at university level, it had a chance of succeeding. However, at

present, every university has its own curriculum.And as known from other studies, it is not always easy to change curricula and convince faculty staff colleagues of this issue.(19)

Regarding the knowledge of Chennai's general dental practitioners, there are various reasons for the results of this questionnaire being excellent . Perhaps the quality and quantity of the undergraduate education is good.However, it is the personal impression that, for many years, dentomaxillofacial radiology has been taught at every university in the country by medical radiologists or general dentists with specific dentomaxillofacial radiology training.The major problem with the undergraduate education is the number of hours that should be dedicated to dental radiology.The age of the dentists was not assessed and also the university where they had studied. Age was not considered an important question in this study, as the age of a dentist does not always reflect that person's knowledge of dental radiology.Furthermore, it is known that not every young dentist starts with new equipment and, vice versa, that not every "older" dentist is working with old equipment. Therefore, the age of the intraoral radiographic equipment was considered more important and more relevant as a question.(20)The level of knowledge and the attitude of dentists regarding radiation safety will have a direct impact on patient exposure to radiation (Shahab et al. 2012). Many studies have been done to determine the knowledge and attitude of dentists regarding radiation safety.

Shahab et al reported a study pertaining to radiation safety to assess the knowledge of dentists with regard to basic information in relation to radiation protection and methods of reducing the radiation dose to the patient. The majority of dentists did not employ appropriate procedures to decrease exposure to unwanted radiation.(21)

Mutyabule and Whaites conducted a study to assess radiation protection measures in dental practices in Uganda. It was found that operators lacked sufficient knowledge regarding radiation safety protection measures.(22)

Aps assessed dentists' knowledge regarding radiation safety. The dentists were asked about various methods of dose reduction to the patients. The results of the study highlighted the need to increase knowledge of dental practitioners regarding radiation safety and methods of dose reduction.(23)

Lee and Ludlow assessed the attitude of dentists regarding radiation safety. The dentists were asked about primary knowledge of radiation safety and the method of reducing the dose to the patient such as speed of the film, collimation of the X-ray tube and the regular use of

shielding. Results of the study confirmed that there is a demand to reinforce the dentist's working knowledge about the issue of radiation safety.

Math et al assessed the understanding of dentists regarding radiation safety standards with regards to the X-ray machine, collimation of the tube, regular use of a film holder, shielding and the position of the operator during radiation exposure. The result of this study also highlights the need to increase the practitioner's awareness and attitude regarding radiation hazard and use of appropriate methods to reduce the radiation dose.(24)

Jacobs et alconducted a study regarding the perceptions of the dentists of radiation protection. The results of this study concluded that there was a need to apply strict guidelines toward radiation safety.(25)

Majority of the practitioners still followed the position and distance rule, few were found to be using lead barriers while mere used lead aprons and some using a combination of various safety techniques. Distance in radiation protection refers to distance from the source and the individual. As the distance increases, radiation exposure reduces. Moreover, shieldingincludes both protective barriers such as lead shield and personnel protective measures such as lead apron. Ninety-eight percent reduction in scattered radiation and attenuate dose to 0.04 µR can be achieved using lead aprons.Patient should wear thyroid collar during radiation exposure as it reduces attenuation of scattered radiation to 92%(46 dentist).Proper shielding from radiation and by increasing the distance from source protect radiographer as well as patient for unnecessary exposure to radiation. According to position distance rule, radiographer position should be at least 6 feet from the source at an angle of 90 to 135° to the central ray of X-ray beam.

Conclusion

The results of this study showed that the knowledge of dentist in South Chennai regarding dentomaxillofacial radiology is excellent.This emphasizes that there is better under and postgraduate education in dentomaxillofacial radiology in Chennai. The present report is therefore an important statement for the dental professional society.The level of awareness of practitioners regarding radiation hazards and safety was found to be acceptable. However, implementation of their knowledge with respect to patient and personnel safety was found wanting. Insisting that they follow the protocols and take necessary safety measures by means of continuing medical education programs, pamphlets, articles, and workshops is strongly recommended.From the time of its discovery, X-rays have played a vital role in the field of

medical and dental science. Ranging from diagnostic to therapeutic applications, the use of X-rays is manifold. Probably, the most widespread application is in the field of dentistry from the simple diagnosis of incipient caries, multiple fractures to aiding in more complex procedures such as precision implant planning. The modalities at the disposal of dentists range from intraoral radiography to cone beam computed tomography. During the course of their training, all health-care personnel are trained regarding radiation hazards and requisite safety measures. However, the sincerity with which the matter is considered needs to be assessed from time to time. In the present study, we attempt to evaluate the awarenessand validate the knowledge of radiograpy among general dental practitioners in Chennai,India.Protection of one's self and patient from all kinds of health hazards is the hallmark of concerned doctors. The AERB recommendations should reach out through the dentists' platform (e.g., IDA) to the dental practitioner. Better "safe than sorry" remains no more a virtue but a fundamental necessity.

Corresponding Author

V. Jayanth Kumar

Reader, Department of Oral Medicine and Radiology,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamalle High Road, Chennai 600 077, Tamil Nadu, India

Email Id: doctorjayanth@gmail.com

Contact Number: +91 8056135807.

References

1.TamizhPaavi.Tha,JayanthKumar.V(Saveetha Dental College and Hospitals).To study awareness about radiation protection among dental students of Chennai-A questionnaire based study.Int J Pharm Bio Sci 2017 Jan ; 8(1): B 542-551.

2.Gijbels F, Debaveye D, Vanderstappen M, Jacobs R. Digital radiographic equipment in the Belgian dental office. RadiatProtDosimetry 2005; 117: 309–312.

3. Ilguy D,Ilguy M, Dincer S, Bayirli G. Survey ofdental radiological practice in Turkey. Dentomaxillofac Radiol 2005; 34: 222–227.

4.Tugnait A, Clerehugh V, HirschmannPN.Radiographic equipment and techniques used in general dental practice. A survey of general dental practitioners in England and Wales. JDent2003; 31: 197–203.

5. Salti L, WhaitesEJ.Survey of dental radiographic services in private dental clinics in Damascus, Syria.Dentomaxillofac Radiol 2002; 31: 100–105.

6.Rushton VE,Horner K. A laboratory evaluation of four quality control devices for radiographicprocessing.JDent1994;22:213–222.

7.Platin E, Ludlow JB. Knowledge and adoption of radiographic quality assurance guidelines by general dentists in North Carolina.Oral Surg Oral Med Oral Pathol Oral Radiol Endod 1995; 79: 122–126.

8.Rushton VE, Horner K, Worthington HM. Aspects of the use of panoramic radiography in general dental practice. Br Dent J 1999; 186: 342–344.

9.Tugnait A, Clerehugh V, Hirschmann PN. Radiographic equipment and techniques used in general dental practice: a survey of general dental practitioners in England and Wales. J Dent 2003; 31: 197–203.

10.Alcaraz M, Martinez Y, Perez L, Jodar S, Velasco E, CanterasM.Theestatus of Spain′s dental practices following the European Union directive concerning radiological installations. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2004; 98: 476–482

11.Brown FR, Shaver JW, Lamel DA. The Selection of Patients for X-ray Examination.U.S.Department of Health, Education and Welfare. HEW Publication (FDA). Rockville, MD, Bureau of Radiological Health1980; 80–8104

12.Yakoumakis EN, Tierris CE, Stefanou EP, Phanourakis IG, Proukakis CC. Image quality assessment and radiation doses in intraoral radiography. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2001; 91: 362-368

13.Tan RT, Burke FJT. Response rates to questionnaires mailed to dentists. A review of 77 publications. Int Dent J 1997;47:349–354

14.Tugnait A, Clerehugh V, Hirschmann PN. Radiographic equipment and techniques used in general dental practice: a survey of general dental practitioners in England and Wales. J Dent 2003; 31: 197–203.

15.Svenson B, Soderfeldt B, GrondahlHG.Knowledge or oral radiology among Swedish dentists.Dentomaxillofac Radiol 1997; 26: 219–224.

16.Swan ES, Lewis DW. Ontario dentists: Radiologic practices and opinions. J Can Dent Assoc 1993;59:62–67

17. Mutyabule TK, Whaites EJ. Survey of radiography and radiation protection in general dental practice in Uganda.Dentomaxillofac Radiol 2002; 31: 164–169.

18.Svenson B, Soderfeldt B, GrondahlHG.Attitudes of Swedish dentists to the choice of dental X-ray film and collimator for oral radiology.Dentomaxillofac Radiol 1996; 25: 157–161.

19.Davies C, Grange S, Trevor MM. Radiation protection practices and related continuing professional education in dental radiography: a survey of practitioners in the North-east of England. Radiography 2005; 11: 255–261.

20. Smith NJ. Continuing education in radiation protection: assessment of a one-day course. Br Dent J 1991; 170: 186-188.

21.Shahab.S,Kavosi.A,Nazarinia.H,Mehralizadeh.SMohammadpour.M,Emami.M.
Compliance of Iranian dentists with safety standards of oral radiology. Dentomaxillofacial Radiology 2012;41:159-164.

22.Mutyabule.T,Whaites.E.Survey of the radiography and radiation protection in general dental practice in Uganda. Dentomaxillofacial Radiology 2002;31:164-169.

23.Aps,JKM.Flemish general dental practitioner knowledge of dental radiology. Dentomaxillofacial Radiology2010;39: 113-118.

24.Math S,MurugeshappaD,Annigeri R,Kalra,DCompliance of Indian dentists with oral radiology safety measures. Journal of Oral and Maxillofacial Radiology 2014;1(3): 104-110.

25.Jacobs.R,Vanderstappen.M,Bogaerts.R,Gijbels.F.Attitudeof the Belgian dentist population towards radiation protection. Dentomaxillofacial Radiology2004 ;33: 334-339.

Knowledge and Attitude about ICDAS Caries Detection among Conservative and Public Health Dentistin Chennai

Vidhya.T.K[1],Sandhya.R[2], Saravana Pandian[3],

Graduate Student[1],Reader[2],Department of Conservative Dentistry and Endodontics,Senior Lecturer[3], Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: The aim of this study is to evaluate knowledge and attitude about ICDAS caries detection among conservative and public health dentistry staff among different colleges in Chennai.To determine the knowledge about ICDAS caries detection among staff of conservative and public health dentistry.

Material and Method: A questionnaire survey was conducted among 50 conservative and public health dentist. The results were evaluated and presented. The international caries detection assessment system (ICDAS) present a new technique for the measurement of dental caries developed from the systematic reviews of literature on the clinical caries detection system and other sources.

Result: To know the knowledge and attitude of ICDAS caries among conservative and public health dentistry staffs.

Conclusion: The ICDAS of future depends on the acceptance of the concepts of integration and utility within a caries detection and assessment system. ICDAS is a valuable method of teaching caries detection and its learning program significantly improves their caries diagnostic skills.

Key Words*: ICDAS, Caries, Enamel, Dentin, Fluorosis.*

Introduction

The International Caries Detection and Assessment System (ICDAS) presents a new technique for the measurement of dental caries developed from the systematic reviews of literature on the clinical caries detection system and other sources [1,2]. All these reviews found that the new caries detection criteria measured different stages of the caries process.The new emphasis on caries measurement and management may indicate that the dental community worldwide has started to recognize that we need new approaches in caries detection, assessment, and management [3]. The development of new technologies and applications has the potential to supplement clinical caries detection, but these assessments will have to be clinically meaningful by providing measurements over and above the rattle of the arrested initial and subclinical lesions [4]. The aim of this study is to evaluate the knowledge and attitude about ICDAS caries detection among conservative and public health dentist.Dental caries is a complex multifactorial disease of the calcified tissues of the teeth, caused by the following factors such as host, agent, substrate and time as demonstrated by the Keyes circle. Detecting carious lesion at the earliest possible stage of its development is definitely helpful in appropriate treatment planning for the same. The ICDAS was developed to bring forward the current understanding of the process of initiation, progression of dental caries to the field of epidemiological and clinical research. This system allows us to record the severity and incidence of the caries in its continuum. In 2002 ICDAS I was developed and was later modified to ICDAS II in 2005. The ICDAS I and II criteria incorporate concepts from the research conducted by Ekstrand et al (1995, 1997) and other caries detection systems described in the systematic review conducted by Ismail et al (2004).

ICDAS

The ICDAS activities have been carried out under the supervision of and on behalf of an unfunded,informal,and an adhoc and voluntary committee, which was assembled in an attempt to advance some of the key recommendations in the area of caries detection and assessment criteria. The principles of the ICDAS committee are: integration, scientific validation, and utility of the criteria in different research and practice settings.Dental caries is a dynamic process with cycles of demineralization followed by remineralization. It is hard to categorize a complex disease like dental caries into a scale because the process is continuous

and can be measured.The ICDAS measures surface changes and potential histological depth of the carious lesions by relying on surface characteristics. The primary requirement for applying the ICDAS system is the examination of clean and dry teeth. The ICDAS examination was visually aided by ball-ended explorer that is used to remove any remaining plaque and debris and to check for surface contour, minor cavitation or sealants. It is highly advisable that the teeth are cleaned with a toothbrush or a prophylaxis head/cup before the clinical examination. The use of sharp explorer is not necessary because it does not add to the accuracy of the detection and it may damage the enamel surface covering the early carious lesions[5]. To improve oral health of the populations, World Health Organization has set the promotion of self care of as one of the goals for the year 2020. Recommended oral self care (ROSC) includes tooth brushing more than once a day, lesser consumption of sugar containing snacks once daily or rarely and regular use of fluoride containing tooth paste. Studies have shown there is an association between increased knowledge regarding oral hygiene and better oral health. Those who have assimilated the knowledge and feel a sense of personal control over their oral health are more likely to adopt self-care practices. The study evaluated the oral health-related knowledge, attitudes and practices of law students belonging to Saveetha university. Health is a universal human need. It has been established that optimal health cannot be attained independent of oral health.(6)Developing such knowledge plays a key role in improving the oral health. The young college students play a vital role in health promotion and preventive information dissemination among the family and their society. It is therefore important that their oral health knowledge is good.(7,8)

ICDAS Codes

The ICDAS detection codes for coronal caries range from 0 to 6 depending on the severity of the lesion.

Code 0: Sound Tooth Surface with Restoration or Sealant

A sound tooth surface adjacent to a restoration/sealant margin, there should be no evidence of caries. Surfaces with marginal defects less than 0.5 mm in width, developmental defects, and extrinsic or intrinsic stains will be recorded as sound. Stained margins consistent with

noncarious and which do not exhibit signs consistent with demineralization should be scored as sound.

Code 1: First Visual Change in Enamel

When seen wet there is no evidence of any change in color attributable to carious activity, but after prolonged air drying an opacity or discoloration consistent with demineralization is visible that is not consistent with the clinical appearance of sound enamel.

Code 2: Distinct Visual Change in Enamel/Dentin Adjacent to a Restoration/Sealant Margin

If the restoration margin is placed on enamel, the tooth must be viewed wet. When wet, there is an opacity consistent with demineralization or discoloration that is not consistent with the clinical appearance of sound enamel and if the restoration margin is placed on dentin, discoloration that is not consistent with the clinical appearance of sound dentin or Cementum is code 2.

Code 3: Carious Defects of < 0.5 mm with the Signs of code 2

Cavitation at the margin of the restoration/sealant less than 0.5 mm, in addition to either an opacity or discoloration consistent with demineralization that is not consistent with the clinical appearance of sound enamel or with a shadow of discolored dentin.

Code 4: Marginal Caries in Enamel/Dentin/Cementum Adjacent to Restoration/Sealant with Underlying Dark Shadow from Dentin

The tooth surface may have characteristics of code 2 and has a shadow of discolored dentin which is visible through an apparently intact enamel surface or with localized breakdown in enamel but no visible dentin. This appearance is often seen more easily when the tooth is wet and is a darkening and intrinsic shadow which may be grey, blue, orange or brown in color.

Code 5: Distinct Cavity Adjacent to Restoration/Sealant

Distinct cavity adjacent to restoration/sealant with visible dentin in the interfacial space with signs of caries as described in code 4, in addition to a gap > 0.5 mm in width or in those instances where margins are not visible, there is evidence of discontinuity at the margin of the restoration/ sealant and tooth substance of the dentin as detected by 0.5 mm ball-ended probe run along the restoration/sealant margin.

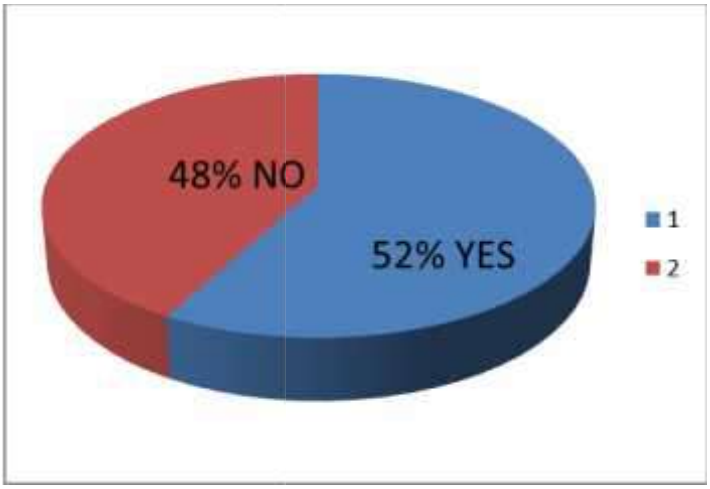
Code 6: Extensive Distinct Cavity with Visible Dentin

Obvious loss of tooth structure, the extensive cavity may be deep or wide and dentin is clearly visible on both the walls and at the base. It is essential to evaluate the clinical application of ICDAS in detection of caries, hence the aim of this study was to analyse the knowledge and attitude about ICDAS caries detection among conservative and public health dentist.

Methodology and Results

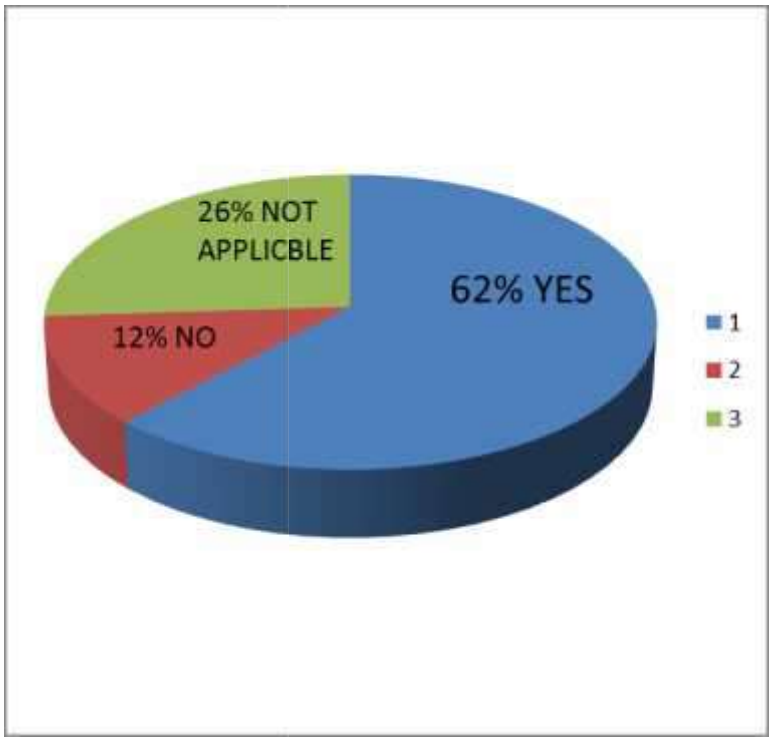
A questionnaire survey was conducted among 50 conservative and public health dentist. The results were evaluated and presented as follows,

1) Have you read articles where ICDAS is used for evaluation of caries?
   29 (58%) people have read articles where ICDAS is used for evaluation, 21(48%) people have never read.



Graph 1: Have you articles of ICDAS.

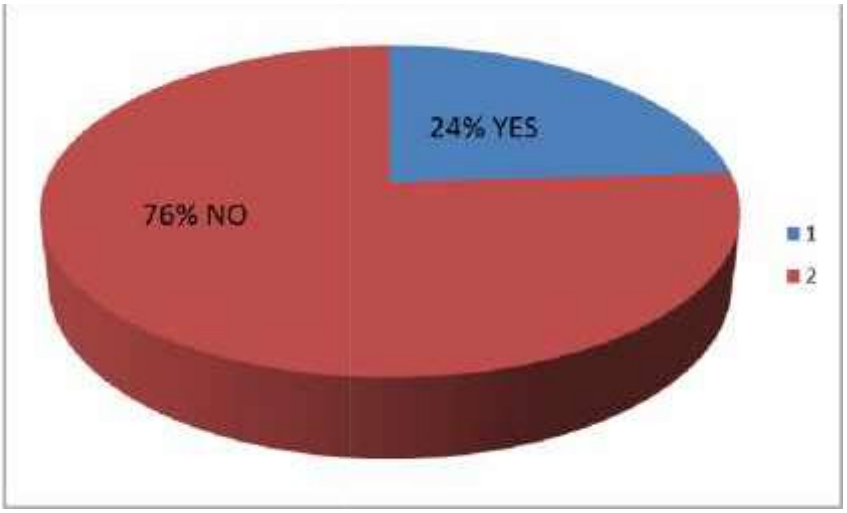
2) If yes, are the reports favourable towards ICDAS in detecting caries over DMFTS?
   31 (62%) people says the reports are favourable, and 6 (12%) says its unfavourable, 13(26%) says its not applicable.



Graph 2: Are the reports favourable towards ICDAS in detecting caries over DMFTS

3) Do you follow ICDAS for caries detection?

12(24%) of dentist says they follow ICDAS for detection of caries, 38(76%) says they don't follow ICDAS for detection of caries.



Graph 3: Do you follow ICDAS for caries detection

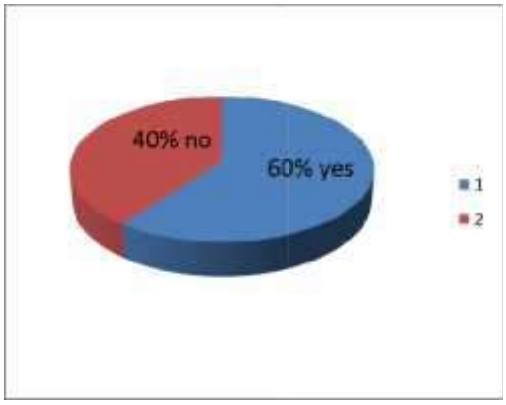
4) Reason, why?

Dentist who don't follow says its complicated, they are unaware of it.

Dentist who follow ICDAS says its very accurate.

5) Is ICDAS method easy to detect the stage of caries?

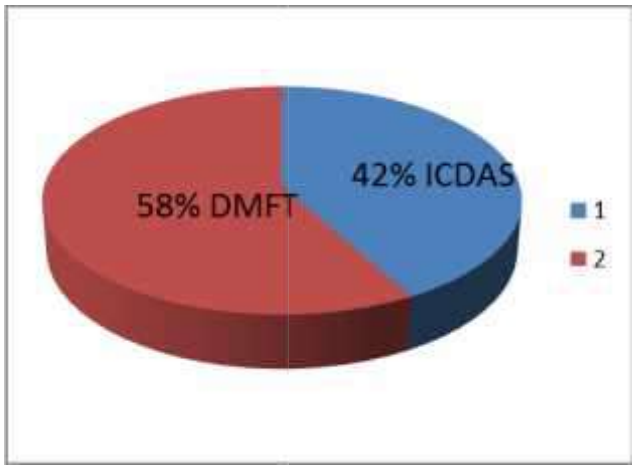
   30 (60%) says yes and 20(40%) says no.



Graph 4:Is ICDAS method easy to detect the stage of caries

6) Which system is very useful in detection of early carious lesion?

   21 (42%) says its DMFT and 29(58%) says its ICDAS



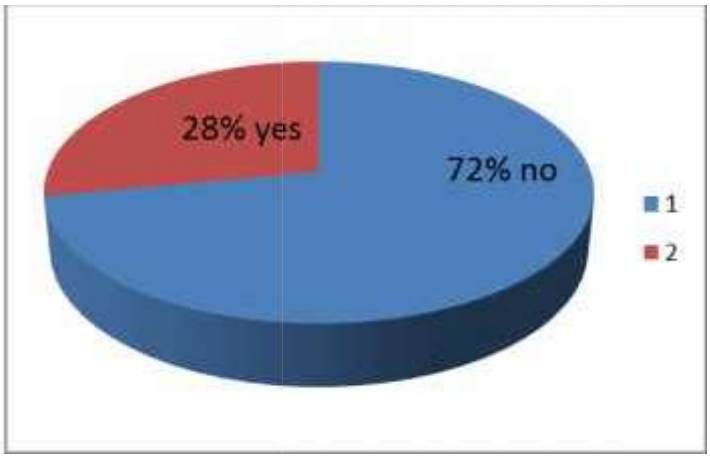
Graph 5:Which system is very useful

7) Which probe should be used to detect caries?

   WHO probe(87%) is used to detect caries.

8) Does DMFT show any details about dental status of caries?

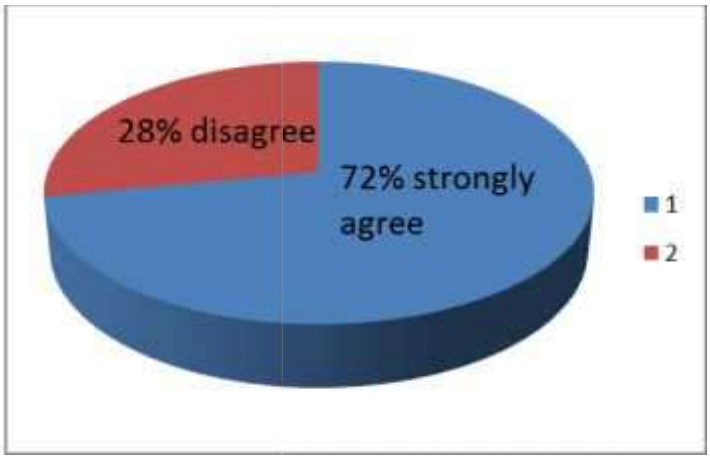
   36(72%) says no , 14(28%) yes



Graph 6: Does DMFT show any details about dental status of caries

9) Identifying the stage of caries influences the treatment plan?

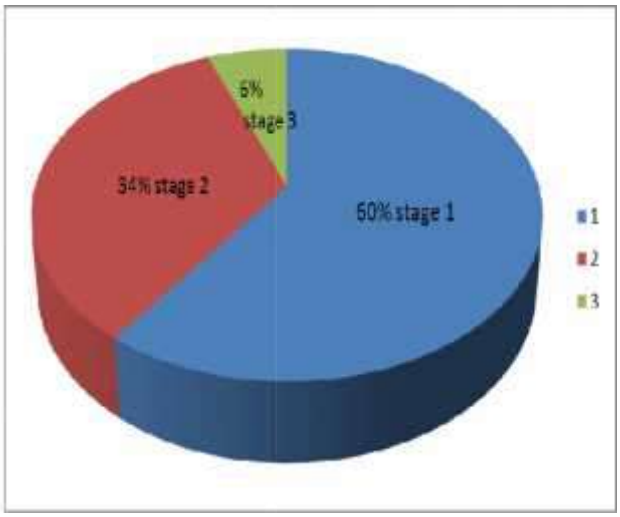
36(72%) dentist strong agree and 14(28%) disagree



Graph 7: Identifying the stage of caries influences the treatment plan

10) First visual change in enamel is an dental term of which stage?

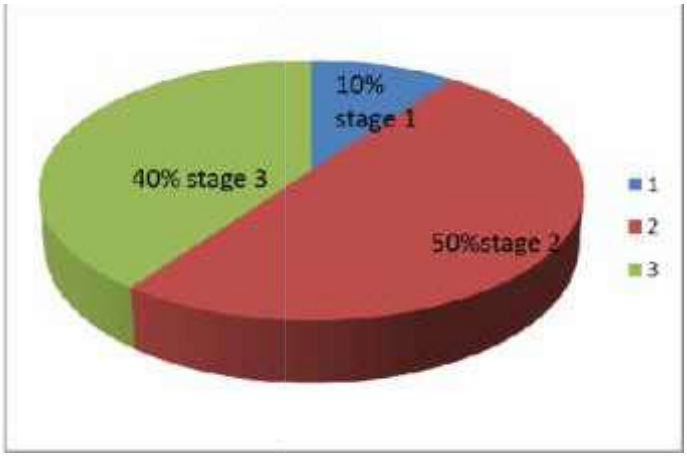
30(60%) of dentist says its stage 1,17(34%) says its stage 2, 3(6%) says its stage 3



Graph 8: First visual change in enamel is an dental term of which stage

11) Distant visual change in enamel is an dental term of which stage?

5(10%) dentist says stage 1, 25(50) says stage2, 20(40%) says stage 3



Graph 9: Distant visual change in enamel is an dental term of which stage

12) Which of the following guidelines are you aware of (tick one or both)

Table 1: Are you aware of following guidelines

S.NO		I AM ONLY AWARE	I HAVE READ
1	Radiographic diagnostic guidelines(ADA)		
2	Systemic fluoride protocol(CDC)		
3	Topical fluoride protocol(ADA)		

4	Guidelines for pit and fissure sealants(ADA)		

34(68%) of dentist are aware of the following guidelines, and 16(32%)have read the articles.

13) Which of the following are necessary to assess caries risk?

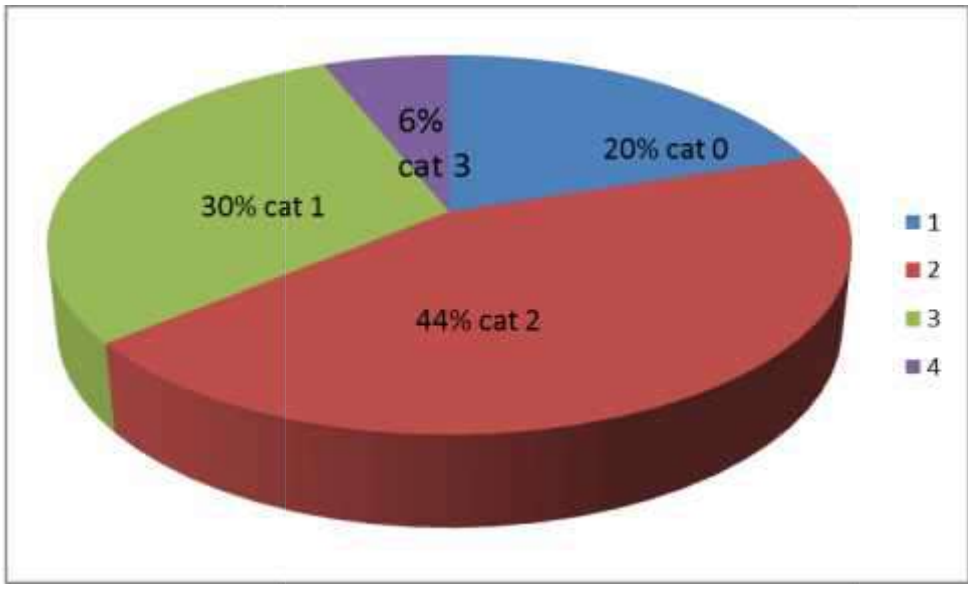
Table 2: Are they following necessary to assess caries risk

S.NO		VERY IMPORTANT	IMPORTANT	LESS IMPORTANT	NO CORRELATION
1	Diet				
2	PH salivary assessment				
3	Saliva buffering capacity				
4	Flow rate of saliva				
5	Socioeconomic status				

Every dentist says diet, PH salivary assessment, saliva buffering capacity, flow rate of saliva, socioeconomic status is very important to assess caries risk.

14) Under which categories fluorosis, enamel hypoplasia and tooth wear come?

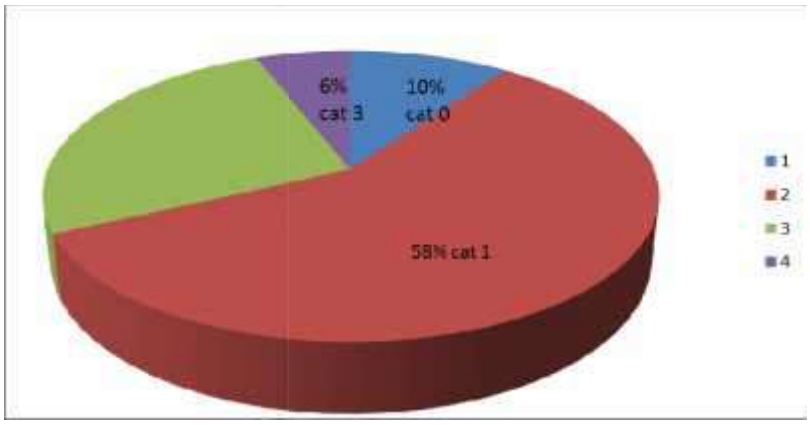
10 (20%) dentist says it comes under category 0,22(44%) says its 1, 15(30%) says its 2,3(6) says its 3.



Graph 10: Under which categories fluorosis, enamel hypoplasia and tooth wear come

15) Pits and fissures comes under which categories of ICDAS?

5(10%) dentist says its comes under category 0, 29(58%) says its 1, 13(26%) says its 2, 3(6%) says its 3.



Graph 11: Pits and fissures comes under which categories of ICDAS

Discussion

According to present study, 29 (58%) people have read articles where ICDAS is used for evaluation, 21(48%) people have never read. 31 (62%) people says the reports are favourable, and 6 (12%) says its unfavourable, 13(26%) says its not applicable. 12(24%) of dentist says they follow ICDAS for detection of caries, 38(76%) says

they don't follow ICDAS for detection of caries. Dentist who don't follow says its complicated, they are unaware of it. Dentist who follow ICDAS says its very accurate. 30 (60%) says yes ICDAS method easy to detect the stage of caries and 20(40%) says no. 21 (42%) says its DMFT is very useful in detection of early carious lesion and 29(58%) says its ICDAS. WHO probe(87%) is used to detect caries. 36(72%) says no DMFT does not show any details about dental status of caries , 14(28%) yes its shows.36(72%) dentist strong agree Identifying the stage of caries influences the treatment plan and 14(28%) disagree. 34(68%) of dentist are aware of the following guidelines(Radiographic diagnostic guidelines(ADA), Systemic fluoride protocol(CDC), Topical fluoride protocol(ADA),Guidelines for pit and fissure sealants(ADA), and 16(32%)have read the articles. Every dentist says diet, PH salivary assessment, saliva buffering capacity, flow rate of saliva, socioeconomic status is very important to assess caries risk.

Dental caries is a dynamic process with cycles of demineralization followed by remineralization. It is hard to categorize a complex disease like dental caries into a scale because the process is continuous and can be measured. The ICDAS is a good tool to identify the nature of caries, the icdas has the sub category (9,10)

Code 0- Pits and fissures; smooth surface (mesial or distal); free smooth surfaces and caries associated with restorations and sealants (CARS) comes under category 0, There should be no evidence of caries.(11) Surfaces with developmental defects such as enamel hyperplasia, fluorosis, tooth wear (attrition, abrasion, and erosion), and extrinsic or intrinsic stains will be recorded as sound First visual change in enamel(code 1): When seen wet there is no evidence of any change in color attributable to carious activity, but after prolonged air drying, a carious opacity or discoloration (white or brown lesion) is visible, which is not consistent with the clinical appearance of sound enamel, or when there is a change of color due to caries it is not consistent with the clinical appearance of sound enamel. Distinct visual change in enamel(12,13) (code 2) : The tooth must be viewed wet. When wet there is a carious opacity (white spot lesion) and/or brown carious discoloration that is wider than the natural fissure/fossa, which is not consistent with the clinical appearance of sound enamel.

Iranzo-Cortezet al in 2017, assess the diagnostic efficacy of the International Caries Detection and Assessment System (ICDAS II) criteria and the DIAGNOdent laser

fluorescence (LF) pen in occlusal caries lesions, using histological sections as the gold standard. It was concluded that both methods are efficacious individually but combining the two is recommended to improve the diagnosis (14). Nogueria et al in 2017, did a study to evaluate, prospectively, the influence of examiner's experience in interpreting and applying the caries detection systems ICDAS (IC) and Nyvad (NY).ICDAS criteria seem to be instinctively understood by students without clinical experience. Nyvad's concepts performed better after two years where the students deepened their theoretical knowledge and experienced clinical practice, collaborating with the identification of activity signs (15).Melger et al in 2016, did a study to describe and compare findings regarding the prevalence and severity of dental caries when using ICDAS and DMFT/dmft in an epidemiological study with children and their mothers. it was observed that DMFT/dmft index would underestimate 60% of non-cavitated lesions in children and 16.6% in adults. The DMFT/dmft underestimated the presence of disease to disregard non-cavitated lesions for the pediatric population evaluated. Whereas ICDAS gives information about the stage or progression of caries(16).Wan baker in 2016, did a study to investigate the accuracy of the Canary System (CS) to detect proximal caries lesions in vitro, and compared it with conventional methods: International Caries Detection and Assessment System (ICDAS) II and bitewing radiography (BW).The CS demonstrated greater accuracy in detecting proximal lesions than ICDAS-II and BW, although without significantly higher specificity (17).Henry et al in 2016, did a study to assess the prevalence of ECC in 0- to 3-year-old children from rural areas of South India, using the ICDAS.The results demonstrate the high prevalence of ECC in this population and the need to consider early diagnosis and specific preventive interventions. For that early diagnosis ICDAS is used (18).Arangannal et al in 2016, did a study to assess the prevalence of dental caries in school children aged between 6-14 years using the International Caries Detection and Assessment System (ICDAS II).The surfaces with early caries lesion (ICDAS code 2) are more common in the surveyed population. Careful monitoring and the preventive program could decrease the risk for dental caries and minimize the need for invasive and painful treatment procedures in the latter part of their life (19).Brun et al in 2016, did a study to evaluate The International Caries Detection and Assessment System (ICDAS) of dental caries. The aim of the present study was to compare the ICDAS scores and radiologically

evaluated caries depths to the histologically evaluated carious lesions in permanent teeth.The present study indicates an acceptable validity of the ICDAS II criteria when applied to permanent teeth. Especially, dentin lesions can be reliably detected. Thus, ICDAS assessment provides the possibility of reducing X-ray exposure for caries detection.

Conclusion

The future of ICDAS depends on the acceptance of the concepts of integration and utility within a caries detection and assessment system. ICDAS is a valuable method of teaching caries detection and its learning program significantly improves their caries diagnostic skills. ICDAS would certainly promote preventative therapies worldwide that encourage the remineralization of noncavitated lesions resulting in inactive lesions and the preservation of tooth structure, function and esthetics and a much decreased DMF all-over. An active collective effort of the dental health care team can make awareness program more effective and can also make people more aware about the importance of dental caries prevention.(20)

Corresponding Author

Saravana Pandian,

Senior Lecturer, Department of Orthodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id- sporthoresearch@gmail.com

Telephone Number: +91 9003101627

Reference

1.Chesters RK, Pitts NB, Matuliene G, Kvedariene A, Huntington E, Bendinskaite R, Balciuniene I, Matheson JR, Nicholson JA, Gendvilyte A, Sabalaite R. An

abbreviated caries clinical trial design validated over 24 months. Journal of dental research. 2002 Sep;81(9):637-40.

2.Ekstrand KR, Kuzmina I, Bjørndal L, Thylstrup A. Relationship between external and histologic features of progressive stages of caries in the occlusal fossa. Caries research. 1995;29(4):243-50.

3.Banting D, Eggertsson H, Ekstrand KR, Ferreira-Zandoná A, Ismail AI, Longbottom C, Pitts NB, Reich E, Ricketts D, Selwitz R, Sohn W. Rationale and evidence for the international caries detection and assessment system (ICDAS II). Ann Arbor. 2005 Sep;1001:48109-1078.

4. Pitts NB, Stamm JW. International Consensus Workshop on Caries Clinical Trials (ICW-CCT)—final consensus statements: agreeing where the evidence leads. Journal of dental research. 2004 Jul;83(1_suppl):125-8.

5. Ekstrand K, Qvist V, Thylstrup A. Light microscope study of the effect of probing in occlusal surfaces. Caries research. 1987;21(4):368-74.

6.M.P.Santhosh Kumar,Knowledge Attitude and Practices towards Oral Health among Law Students in Chennai, Saveetha Dental College, /J. Pharm. Sci. & Res. 2016;8(7):650-653.

7. Donnelly M, McGilloway S, Mays N, Perry S, Lavery C. A three- to six- year follow- up of former long- stay residents of mental handicap hospitals in Northern Ireland.British journal of clinical psychology. 1997 Nov 1;36(4):585-600.

8. Petersen PE. Continuous improvement of oral health in the 21st century: the approach of the WHO Global Oral Health Programme. Zhonghuakouqiangyi xue zazhi= Zhonghuakouqiangyixuezazhi= Chinese journal of stomatology. 2004 Nov;39(6):441.

9. Mehta A, Kaur G. Oral health-related knowledge, attitude, and practices among 12-year-old schoolchildren studying in rural areas of Panchkula, India. Indian Journal of Dental Research. 2012 Mar 1;23(2):293.

10. Hobdell M, Petersen PE, Clarkson J, Johnson N. Global goals for oral health 2020. International dental journal. 2003 Oct 1;53(5):285-8.

11. Woolgrove J, Cumberbatch G, Gelbier S. Understanding dental attendance behaviour. Community dental health. 1987 Sep;4(3):215-21.

12. Hamilton ME, Coulby WM. Oral health knowledge and habits of senior elementary school students. Journal of Public Health Dentistry. 1991 Dec 1;51(4):212-9.

13. Humagain M. Evaluation of knowledge, attitude and practice (KAP) about oral health among secondary level students of rural Nepal-a questionnaire study.

14. Iranzo-Cortés JE, Terzic S, Almerich-Silla JM. Diagnostic validity of ICDAS and DIAGNOdent combined: an in vitro study in pre-cavitated lesions. Lasers in medical science. 2017 Apr 1;32(3):543-8.

15.Melgar RA, Pereira JT, Luz PB, Hugo FN, Araujo FB. Differential impacts of caries classification in children and adults: a comparison of ICDAS and DMF-T. Brazilian dental journal. 2016 Dec;27(6):761-6.

16.Jan J, Bakar W, Zaripah W, Mathews SM, Okoye LO, Ehler BR, Louden C, Amaechi BT. Proximal caries lesion detection using the Canary Caries Detection System: an in vitro study. Journal of investigative and clinical dentistry. 2016 Nov 1;7(4):383-90.

17.Henry JA, Muthu MS, Saikia A, Asaithambi B, Swaminathan K. Prevalence and pattern of early childhood caries in a rural South Indian population evaluated by ICDAS with suggestions for enhancement of ICDAS software tool. International journal of paediatric dentistry. 2017 May 1;27(3):191-200.

18.PonnuduraiArangannal SK, Jayaprakash J. Prevalence of dental caries among school children in Chennai, based on ICDAS II. Journal of clinical and diagnostic research: JCDR. 2016 Apr;10(4):ZC09.

19. Braun A, Guiraud LM, Frankenberger R. Histological validation of ICDAS II and radiological assessment of occlusal carious lesions in permanent teeth. Odontology. 2017 Jan 1;105(1):46-53.

20. Nivedha.V and ShabeenaTaj, Knowledge and Awareness on the Role of Diet in the Incidence of Dental Caries among General Population, Journel of dental and oral health 2017;9:435-444.

.

Knowledge and Attitude of Parents Regarding Oral Health Care Maintenance of Their Children

Benazir Hussain M[1], Mahesh Ramakrishnan[2], Deepa Gurunathan[3]

Graduate Student[1], Reader[2], Department of Pedodontics, Professor and Head[3], Department of Pedodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

Abstract

Introduction: The oral health of children depends on awareness of their parents regarding importance of primary teeth and importance of maintenance of oral heath. Parents have an important role in making decisions about their child's oral health as good oral health habits in childhood are shaped by parents. The aim of this study is to evaluate the awareness and knowledge of parents towards professional pediatric dental treatment and oral health care maintenance.

Materials and Method: A cross sectional questionnaire survey was conducted among 100 parents of 5 to 15 years school going, who visited Saveetha Dental College. A self administered questionnaire addressing various aspects of knowledge and attitude of parents toward pediatric dental treatment and oral health care was answered by participants. The data was first transferred into Microsoft excel spread sheets. The results were analysed by using SPSS statistical software and descriptive statistics was done. (statistical package for social sciences, SPSS Inc., Chicago, IL, USA).

Result: In the present study, Tooth brush and paste was the most common method of oral hygiene (94%), meanwhile only 5% use manual hand brushing. Most of the children (51%) brushed the teeth once a day, while 45% brushed twice a day. Approximately a half (45%) of parents supervised and advised for children during tooth brushing procedure. About 71% of the parents was unaware that pulp capping will be done for primary teeth. It also revealed that about 55% believed that there is spread of caries of from primary teeth to the permanent dentition. In

our study, most were aware that decay is preventable and only 29% were aware that fluoride prevents decay. When asked about the acceptance of preventive treatment if advised by the dentist, 28% were unsure or would not undertake preventive procedures for their children. This study revealed that most parents were not aware of the importance of dental visits at an early age.

Conclusion: Most parents had good knowledge regarding importance of primary teeth and oral hygiene maintainance, but the same did not reflect in their attitude and practice.

Key Words*: oral cavity, caries, pedodontist, oral hygiene, primary teeth*

Introduction

Health is understood to be "a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity"(1) Health education is any combination of learning experiences designed to facilitate voluntary actions conducive to health. These actions or behaviours may be on the part of individuals, families, institutions or communities. Thus the scope of health education may include educational interventions for children, parents, policy makers, or health care providers. It has been well-documented in dentistry and other health areas that correct health information or knowledge alone does not necessarily lead to desirable health behaviours(2) .The oral cavity is the major part of the body and it is very important for the maintenance of good health. Oral health is having both healthy teeth affecting people physically and psychologically, its influence on their growth, function, speech, aesthetics, and socialization. Oral health is important for good health and well-being of individuals which covers a range of health promotion and disease prevention concerns(3). Oral health is an essential component of general health and influences a person's quality of life. Oral health behaviours play a central role in the prevention of many oral diseases. Oral health related habits are established early in life.

In early years of a child's life, the milk teeth plays an important role in speech, chewing, maintaining space and it also helps us in guiding the eruption of permanent teeth. Children should have a high standard of health, including oral health, but as children, they are dependent on adults for their health(4). Dental caries is the most common chronic disease of childhood and

may affect a child's quality of life due to various factors including pain, infection, sleep loss, absence from school with compromised education, poor mastication, compromised appearance, inadequate nutrition, growth and development. In industrialized countries, dental caries remains a significant public health problem.(5) Dental caries is a disease that generally is preventable by primary oral care measures, whereas dental treatment of carious primary teeth in children is expensive with further dental treatment being subsequently required in many young children who have had comprehensive dental treatment provided. Dental caries or its treatment in the primary dentition is a strong predictor for future development of dental caries in the primary, mixed and permanent dentitions(6). Paediatric dentistry is related to the child's psychology, to gain the child's confidence, and co-operation to perform the desired treatment in a kind manner. Paediatric dentist plays an important role in promoting child's future dental health by stimulating the attitude and behaviour regarding dental care (5).Most parents are unaware of the role a pedodontist and the importance of dental visits at an early age .The most parents have underestimated that the milk teeth are going to exfoliate and it not worth paying much attention without knowing the complication. Untreated dental can have serious impact on individuals, such as pain, discomfort, social and functional limitations, which ultimately can impair oral health-related quality of life (6).

Many dental health education programs have been conducted in schools and offices for the promotion of oral health care; however, but these efforts will not be succeeded until people are not aware of the importance of oral health and positive attitude towards the paediatric treatment modalities (7). Oral health education always begins with awareness of importance of oral health. The goal of oral health education is to improve knowledge, which may lead to adoption of favourable oral health behaviours that contribute to better oral health(8)The programs for the prevention of oral diseases concern teaching about oral hygiene and healthy eating, fluoride prophylaxis, periodic check-ups, sessions of professional oral hygiene, and secondary prevention programs. It is important issue which needs continuing supervision from the health care professional (9). Prevention of disease, disability and suffering should be a primary goal of any society that hopes to provide a decent quality of life for its people. Prevention on the community or population based level is the most cost effective approach and has the greatest impact on a

community or population, whether it is a school, neighbourhood, or nation. An effective community prevention program is a planned procedure that prevents the onset of a disease among a group of individuals (10). Many different approaches to preventing dental diseases exist and the most cost-effective method is health education. However knowledge gained may serve as a tool to empower population groups with accurate information about health and health care technologies, enabling them to take action to protect their health(11). A basic oral health care program introduced by World Health Organization for less industrialized countries includes oral health education and emphasizes on the integration of health education with other oral health activities such as provision of preventive, restorative and emergency dental care (12).The knowledge, attitude and behaviour of parents towards dental treatment tend to influence their child to accomplish a great dental attitude(13). So many oral health care programs must be implemented to achieve a wide spread of knowledge among the parents and their children(14). This study is to evaluate the awareness and knowledge of parents towards professional pediatric dental treatment and oral health care maintainence.

Materials and Method

Study Design

A cross-sectional study was conducted among 100 parents of school going childrens in the outpatient Department of saveetha dental college, Chennai,Tamilnadu. The study was conducted during the month of April 2016 after taking prior informed consent from the parents who were willing to participate. Inclusion criteria were parents getting their children treated for dental caries or had a past dental history and who were willing to participate. In addition, parents with children with no medical conditions or no medications prescribed to them were included in the study. Childrens age between 5 years to 15 years were included in the study. Age of 12 to 15 years seem to be accepted as a monitoring age for caries since all permanent teeth except third molars would have been erupted by this age. They seem to be more prone to dental caries at this age . Parents who could not read and write were excluded from the study. Parents who were not

the primary caretakers of their children or who had children with medical problems were excluded from the study.

Methodology

The self-administered questionnaire written in English was adapted from several studies. It had 22 questions, 8 each in the knowledge regarding oral hygiene instruction and 14 in the attitude of parents towards pediatric dental procedure, and took about 10–15 min to complete. The scoring for practice and attitude towards pediatric dental procedure included yes/NO/do not know responses and knowledge regarding oral hygiene were given options. All aspects of oral health promoting factors in children including oral hygiene, diet, and fluoride, and awareness regarding infant oral health and practices were addressed. This pretested questionnaire addressed the knowledge, attitude, and practices of oral health care for prevention of early childhood caries.

Result

Demographic data of participants are represented in (**Table.1)**.The results of answers are presented in (**Table.2)**. Tooth brush and paste was the most common method of oral hygiene (94%), meanwhile only 1% of participants answered that use mouthwash and 5% use manual hand brushing .Most of the children (51%) brushed the teeth once a day, while 45% brushed twice a day and 4% answered that brush less them once a day. Approximately a half (45%) of parents supervised and advised for children during tooth brushing procedure. Meanwhile 27% of parents were brushing children teeth, 16% of parents gave only advices and 12% did not participate in children tooth brushing. 84% of the parents believed that sweets can affect child oral health. 12% thought that sweets couldn't affect child oral health and 4 % didn't have opinion about this item.67% of the parents believed that oral health affect general health, while 26% of participants were thinking that oral health cannot affect on general health and 7% did not know. 69% of the parents changed their childrens brush 3 months once while 27% changed 6 months once and 4% changed yearly once. 39% of parents answered tongue cleaner was the most

common auxillary aid used where 62% had no practice of using auxillary aids. It also depicted that only 49% of the participants made their children to clean their tongue regularly.

Mostly 57% fathers filled the questionnaire whereas only 43% mothers filled the questionnaire **(Table.1)**

The result showed that **(Table.3)** 87% of the parents were aware that there are two sets of teeth(milk teeth and permanent teeth) while 13% responded that they were not aware of it. 55% have brought their childrens to dentist for check up whereas 45% of the parents gave a negative response. Most of the parents (71%) did not know pulp capping and root canal treatment will be done in childrens while 29% were aware about it. 73% of the parents responded they will go to pedodontist if they were referred, while 17% showed negative response and 10% had no opinion on it. 55% believed decay in the primary teeth will affect the underlying permanent teeth, whereas 25% said no and 20% had no idea about it. 39% of parents responded they will give preventive measure treatment such as fluoride and pit and fissure sealant if the dentist recommends but 33% said they will not take up any treatment for their childrens. Only 14% of the parents believed that application of fluoride to childrens once in 6 months will be resistant to decay and 86% showed affirmative response. 97% were not aware that pedodontist are well trained in behavioural management and also showed 84% of the parents were ready to receive more information about pedodontic treatments and oral hygiene maintainence for their children whereas 16% were not interested about it.

Table1: Demographic characteristic of participants

Demographic data	Number(%)
Female	43(43%)
Male	57(57%)
Total	100

Table 2: Distribution of study population about knowledge on oral hygiene instructions among parents

	Number	Percentage
Method of oral hygiene		
Tooth brush and paste	94	94%
Mouth wash	1	1%
Manual hand brushing	5	5%
Frequency of tooth brushing		
Once a day	51	51%
Twice a day	45	45%
Less than once	4	4%
Parents role in child tooth brushing		
Parents brush children teeth	27	27%
Parents supervise and advice	45	45%
Parents give advice	16	16%
Do not participate	12	12%
Sweets affects oral health		
Yes	84	84%
No	12	12%
Don’t know	4	4%

Does oral health affect general health		
Yes	67	67%
No	26	26%
Don't know	7	7%
Frequency of changing brush		
Once in 3 months	69	69%
Once in 6 months	27	27%
Yearly once	4	4%
Most common auxillary aids used		
Tongue cleaner	31	31%
Interdental brush	1	1%
Floss	6	6%
None	62	62%
Make your children clean their tongue		
Yes	49	49%
No	51	51%

Table 3: Distribution of study population on attitude of parents towards pediatric dental procedure

	Yes(%)	No(%)	Don't know(%)
Do you know we have two sets of teeth- milk teeth and permanent teeth	87%	13%	-
Has your child ever been to the dentist before	55%	45%	-
Are you aware of pediatric dentistry	57%	43%	-
Are you aware that pulp capping is done in milk teeth as well?	29%	71%	-
If you were referred to go to pediatric dentistry, will you go?	73%	17%	10%
Do you think infection from milk teeth can spread to permanent teeth lying under it and damage it?	55%	25%	20%
Do you think space between milk teeth is normal?	45%	24%	31%
Are are you aware Paediatric dentistry treatment can help to correct crooked teeth at a later stage?	51%	24%	25%

Do you think teeth decay can be prevented?	74%	9%	17%
Do you know that application of fluoride every 6 months can make your child's teeth more resistant to decay?	29%	14%	57%
If a dentist advises you for preventive treatment like sealants and fluoride application to prevent decay in child's teeth, would you do?	39%	33%	28%
Medicine syrups plays a big role in tooth decay, do you agree?	38%	32%	30%
Do you know pedodontist are well trained in behavioural management	3%	97%	
Would you like to receive more information about your child teeth protection and oral hygiene maintainable?	84%	16%	

Discussion

Parents are role models for their children. Children learn habits by imitating their role models and the best way to teach them is to practice these oral hygiene practices themselves.(15). Dental caries is a preventable disease and if it is noticed at an early stage.(16) Hence, prevention at the root level that is primordial prevention and oral health education of parents is essential as the preschool age group (2–4 years of age) is dependent on them for their oral health care needs. Later, from 2 years onward, oral health promotion strategies such as fluoridated toothpaste, etc., can be begun in cooperation with the parents. To attain these goals, one needs to assess the existing levels of knowledge, attitude, and practices to bring about the necessary changes. (17) In addition, children with primary dentition affected by dental caries are prone to the development of dental caries in permanent dentition. Hence, children reinforced at an early age by their parents are motivated and trained for a lifetime.

The role of bacteria in the causation of dental caries was not acknowledged by the majority of the parents which was reflected by the prevalent practice of sharing utensils and biting food into small pieces before feeding in this study. This is in accordance with studies by Mani *et al*.(18) The focus should be on parents/carers to encourage to limit their children practice of sharing utensils and biting food invited to carry out oral hygiene practices in a proper way carers and children should be advised that foods and drinks containing sugar substitutes are available, but should be consumed in moderation. Sugar-free medicines should be used when available. Parents and care takers should be encouraged to brush their child as soon as the first tooth appears, using a soft toothbrush and water only.(19) Meanwhile the results of this survey showed, Tooth brush and paste was the most common method of oral hygiene (94%), where the study conducted by Suvarna (20)showed 84% of the participants used tooth brush and paste, whereas 45% parents supervised and gave advises during children tooth brushing procedure in the present study which is more than the study carried out by Suvarna(20). More children brushed teeth twice a day (39%) when compared to previous study(20).There is generally failure in use of interdental auxillary aid. In a study conducted in Saudi Arabia in 2001 showed 64% of the subject did not use dental floss, interdental brush and tongue cleaner as an preventive auxillary aid which is similar to our results (21). Tongue cleaning was done by 49% of children in the present study which is in contrast with the study done by Jain et al(22) in which only 20% of the studied

population cleaned their teeth. Other studies by Al-Zahrani *et al*(23) also revealed that parents are aware of milk teeth and permanent teeth and also affect the general health of the child. In this study, it showed that 87% of the parents were aware of importance milk teeth and permanent teeth. Only an average of 57% of children visited dentist and were aware of pedodontist which also showed similar result in the study carried out by jain et al(22).

The study revealed, 84% of the parents believed that sweets can affect oral health of children. 12% thought that sweets couldn't affect child oral health and 4 % didn't have opinion about this item. 67% of the parents believed that oral health affect general health, while 26% of participants were thinking that oral health cannot affect on general health and 7% did not know about it. Al-Zahrani et al(23) also showed similar results that average of 64.5% of the parents believed that oral health has an impact on general health and sweets can affect oral health.

In the present study, most of the parents (71%) did not know pulp capping will be done in children while 29% were aware about it. The study conducted by MCknight et al,(24) showed 63% were not aware of pulp capping treatment which is comparatively lesser than our present study.73% of the parents responded they will go to pedodontist if they were referred. 39% of parents responded they will give preventive measure treatment such as fluoride and pit and fissure sealant if it was recommended by the dentist. Meanwhile 33% said they will not take up any treatment for their childrens. This results were similar to the study conducted by Zahrani et al et al(23) where 38.5% of parents agreed to take up the preventive treatment if the dentist suggests. Only 55% of the parents were aware that infection from a primary teeth can spread to the permanent tooth underlying underneath it and damage it. This findings is comparable to the studies conducted by Al Zahrani et al (23)who reported 71% were aware of the same. In our study most were aware that decay is preventable and only 29% were aware that fluoride prevents decay. This was contrary to the study conducted by suresh et al,(25) where the parents knowledge about fluoride was inadequate. In the present study 62% of the parents were unaware that medicated syrups may lead to dental decay. This finding is similar to the other studies conducted by H Landt et al(26). 97% were not aware that pedodontist are well trained in behavioural management and also showed 84% of the parents were ready to receive more

information about pedodontic treatment and oral hygiene maintainence for their children. This was similar to the study conducted by H Landt et al(26).

From the present study, regarding the importance of maintenance and management of their children's dental health which signifies a genuine lack of awareness than an attitude of indifference towards importance of primary teeth. This indicates that improvement of basic knowledge and dental health education is mandatory in our population.

In conclusion, many parents had good knowledge, but the same did not reflect in their attitude and practice. It is worthwhile to attempt regular oral health promotion education programs, with stress on prevention and treatment modalities for their children.

Corresponding Author

Deepa Gurunathan

Professor and Head, Department of Pedodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai-600077, Tamil Nadu, India.

Email: drgdeepa@yahoo.co.in

Telephone Number: +919994619386

References

1. Subashri. A, T. N. Uma Maheshwari. Knowledge and attitude of oral hygiene among dental students, department of behavioural science, IJDR, 2016:54:1840-1842

2. Petersen PE, Peng B, Tai B, Bian Z, Fan M. Effect of a school-based oral health education programme in Wuhan City, Peoples Republic of China. Int Dent J. 2004; 54:33–41.

3. Park K. 21st ed. Jabalpur. Preventive and Social Medicine.2011; 32:32-102

4. Frenkel H, Harvey I, Needs K. Oral health care education and its effect on caregivers' knowledge and attitudes: A randomised controlled trial. Community Dent Oral Epidemiology. 2002; 30:91–100

5. Tai B, Du M, Peng B, Fan M, Bian Z. Experiences from a school-based oral health promotion programme in Wuhan City, PR China. Int J Paediatr Dent. 2001; 11:286–91.

6. Redmond CA, Blinkhorn FA, Kay EJ, Davies RM, Worthington HV, Blinkhorn AS. A cluster randomized controlled trial testing the effectiveness of a school-based dental health education program for adolescents. J Public Health Dent. 1999; 59:12–7.

7. Friel S, Hope A, Kelleher C, Comer S, Sadlier D. Impact evaluation of an oral health intervention amongst primary school children in Ireland. Health Promot Int. 2002;17:119–26.

8. Chapman A, Copestake SJ, Duncan K. An oral health education programme based on the National Curriculum. Int J Paediatr Dent. 2006;16:40–4.

9. Mariño R, Calache H, Wright C, Schofield M, Minichiello V. Oral health promotion programme for older migrant adults. Gerodontology. 2004;21:216–25.

10. Nicol R, Petrina Sweeney M, McHugh S, Bagg J. Effectiveness of health care worker training on the oral health of elderly residents of nursing homes. Community Dent Oral Epidemiol. 2005;33:115–24.

11. Peng B, Petersen PE, Tai BJ, Yuan BY, Fan MW. Changes in oral health knowledge and behaviour 1987-95 among inhabitants of Wuhan City, PR China. Int Dent J. 1997;47:142–7.

12. Freitas-Fernandes LB, Novaes AB, Jr, Feitosa AC, Novaes AB. Effectiveness of an oral hygiene program for Brazilian orphans. Braz Dent J. 2002;13:44–8

13. Buischi YA, Axelsson P, Oliveira LB, Mayer MP, Gjermo P. Effect of two preventive programs on oral health knowledge and habits among Brazilian schoolchildren. Community Dent Oral Epidemiol. 1994;22:41–6.

14. Rong WS, Bian JY, Wang WJ, Wang JD. Effectiveness of an oral health education and caries prevention program in kindergartens in China. Community Dent Oral Epidemiol. 2003;31:412–6

15. Vachirarojpisan T, Shinada K, Kawaguchi Y. The process and outcome of a programme for preventing early childhood caries in Thailand. Community Dent Health. 2005;22:253–9.

16. Laiho M, Honkala E, Nyyssönen V, Milen A. Three methods of oral health education in secondary schools. Scand J Dent Res. 1993;101:422–7.

17. Harris N, Primary Preventive Dentistry. Rjpt, 1990;43:56-102

18. Mani. Effectiveness on oral health of a long-term health education programme for mothers with young children. Br Dent J. 2000;188:201–5.

19. V.Gopinath, Oral hygiene practices and habits among dental professionals in Chennai, Department of Periodontics, IJODR. 2010;21:3-137

20. Suvarna. Development and testing of an audio-visual aid for improving infant oral health through primary caregiver education. J Can Dent Assoc. 2005;71:241, 241a–241.

21. Jamjoom HM. Preventive oral health knowledge and practice in Jeddah, Saudi Arabia. J KAU med sci 2001;9:17-25

22. Jain. Oral health habits amongst children in the United Kingdom in 2003. Br Dent J. 2006; 200(9):487–91.

23. Al-Zahrani. Dietary habits and oral hygiene as predictors of caries in 3-year-old children. Community Dent Oral Epidemiol 1983; 11: 308-11.

24. Mcknight . Oral health relayed quality of life. Park oral Dent J 2009;29:397-402.

25. Suresh. Testing a preventive symptomatic theory of dental health behaviour. Am J public health nation health 1969;59:98-105.

26. H Landt. Effectiveness of an oral health education and caries prevention program in kindergartens in China. Community Dent Oral Epidemiology. 2000;31:412–6

Knowledge and Attitude towards Digital Radiography among Dentists

Rene Jochebed. S[1],Deepa Gurunathan[2], Revathy Gounder[3]
Graduate Student[1],Professor[2], Department of Pedodontics,Reader[3], Department of Prosthodontics,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Digital imaging modalities which are recently developed offer better options by significantly reducing the dosage of radiation exposed and at the same time offer better quality with user- friendly options. Such things must be made known to the dentists who will in turn benefit the patient as well. The aim of this study was to determine the knowledge and attitude towards digital radiography among dentists.

Materials and Methods: A systematic random survey of practicing dentists in Chennai using a questionnaire with questions pertaining to individual details, knowledge level and approach toward digital radiography. A total of 50 dentists were included in the survey.

Results: In this study, it was found that majority of the dental practitioners prefer digital imaging modalities in their practice. However, detailed knowledge regarding the digital imaging remains unknown to some.

Conclusion: Proper precautions need to be taken by the dentists during radiation exposure, which has to be made known to them, and also the various advances in the digital imaging modalities, which provide better options for the dentist as well as the patient.

Key Words:*Advances, Digital imaging, Dentists, Knowledge, Radiation*

Introduction

The selection of an ideal diagnostic technique is an essential step in the treatment of a disease. An ideal diagnostic tool will provide the essential information, while minimizing the cost and adverse effects to the patient. Radiographs are valuable tools in diagnosis and treatment prognosis in various fields of dentistry. The discovery of Xrays was done by Wilhelm Conrad Roentgen in the year 1895. The first original roentogram was taken by Dr. Otto Walkoff in January 1896 from a portion of a glass imaging plate for an exposure time of 25 minutes in his own mouth. Ever since, imaging in dentistry has seen a major progress and is applied in various fields of dentistry. [1] As a result of rapid developments in the field of dental imaging and technology, the use of digital imaging has found to be increased over the past years.

Both intra oral radiographs and extra oral radiographs are being used to help in diagnosis and treatment is various fields in dentistry. Two dimensional and three dimensional radiographs are used in diagnosis and treatment panning. 2 Dimensional radiographs provide excellent insight into the internal structure of the teeth and the supporting structures and hence can be used in the diagnosis of dental caries, periodontal and periapical lesions and osseous defects as well.Intra oral radiographs give a picture of the tooth and the internal components, the periodontal structures and the surrounding alveolar bones. Extra oral radiographs which include the lateral cephalometric radiograph show the structural relationship between the bones, the maxillary and the mandibular jaw bones, and the airways spaces.

Conventional imaging modalities which require a detailed processing have been developed in such a way to minimize the processing time and to fasten the image reception process.Attempts have been made in dental imaging to develop techniques that provide optimal information, while minimizing harm to the exposed patient. [2] Most practitioners favor the use of digital radiographic techniques which have many advantages, which include time effectiveness, use of low radiation dosage, elimination of requirement of chemicals and development processes, and have storage capacity and easier ways to communicate with other dentists also. [3] There are also disadvantages with the use of digital imaging, such as high cost for installment and maintenance, and in some cases, even the radiation dose.

Imaging modalities such as Computed Tomography(CT) and Cone Beam Computed Tomography(CBCT) have been developed with the aim of reducing radiation exposure of the

patient and limit the exposure to the head and neck region. CBCT works on the principle that by focusing a cone shaped X-ray beam on a two- dimensional (2D) detector that rotates 360^0 or less around the patient's head to produce a series of 2D images which is then studied. CBCT was introduced with the benefit of having a lesser cost factor, require lesser installation space and have a rapid scan time and limited area of exposure only to the head and neck. Drawbacks of CBCT include beam hardening and scatter from dental materials and poor soft tissue contrast.[4]Although it could be assumed that the radiation dose levels in dental practice are relatively low and not harmful to health, the cumulative effect of repeated exposures must be considered. It is necessary to determine the level of knowledge of dental practitioners, quality care, radiation protection and whether they are being used efficiently in oral and maxillofacial radiology. The aim if this study is to evaluate the knowledge and attitude of dentists towards radiation hazards and protection, the various digital radiography methods and the recent advances in the field of digital imaging.

Materials and Methods

The present survey was undertaken among practicing dentists in various areas in Chennai. The institutional ethical clearance was obtained prior to the start of the study. A questionnaire with 15 questions was administered to a total of 50 dentists and collected personally. The questionnaire comprised of questions regarding the use and preference of digital imaging modality, knowledge regarding radiation dosage, precautions taken and their attitude towards developments in dental imaging techniques. A comprehensive questionnaire was prepared based on a study done in Mangalore, India. [5]. All the dentists who undertook this survey were explained in detail about the purpose of this study and were assured about the anonymous processing of the questionnaire. The questionnaire comprised of the following sections of questions such as Demographic details which includes age, sex and work experience; Duration and reason for usingdigital radiography; Radiation protection which includes the use of protective barriers and knowledge regarding radiation safety and knowledge about recent advances in digital imaging

The results were obtained and the statistical analysis was done and tables were drawn with the analyzed data.

Results

The mean age of the respondents was found to be 35. In this study, it was found that 69.76% of the respondents started using digital radiography in routine practice less than the past 5 years, 27.9% for the past 5 years and 4.6% more than past 10 years. Intra oral radiographic examination us the backbone of imaging for a dental professional. However, despite the increased usage over the past few years, the knowledge regarding the maximum permissible dosage for digital imaging was not well known.

Less radiation dose	30.43%

The safe distance from the radiation exposure was known by around 64.4% of the dentists who undertook the survey. 55% stated that they use CBCT for implant placement, 25% in orthodontics and 6% in the evaluation of cysts and tumors. Periodic checks of the radiography equipment were said to be done by 51% of the respondents. The advantage of CBCT over CT was stated to be because of the low radiation dose (58.8%), continuous image can be obtained (17.6%) and also because of its high accuracy and high resolution. In this study, the reasons for using digital radiography was found to be less radiation dose (30%), short time required for image processing (30%), no development required (8%), measurements and adjustments can be made (4%) and others (2%). (Figure 1).The commonly used digital radioprahic methods as found in this study were IOPA (84%), Bitewing radiograph (8%), OPG (2%) and CBCT (2%). (Figure 2).The most commonly employed method of radiation protection was the use of lead aprons (30%) and a protective barrier wall (30%). (Figure 3).The easiest to manage in a dental office was said to be IOPA- 75%, followed by OPG- 13% and CBCT- 4%. (Figure 4). Also,57.7% said that they were not aware of the recent developments in the field of dental imaging using digital radiographic techniques, and would like to update themselves through the internet (37.14%), journals (28. 2%), conferences (25.7%), and others (8.5%). (Figure 5)

Table 1: Reason for using digital radiography

Short time	30.43%
Easy to store	26.08%
No development required	8.69%
Adjustments and measurements can be made	4.34%
Others	2.17%

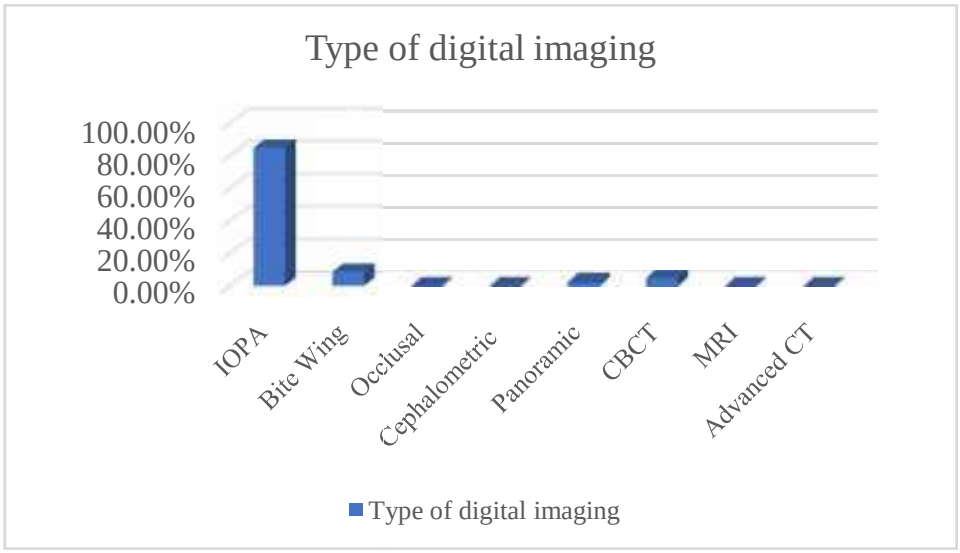


Figure 2: Distribution of various types of digital imaging modalities being used

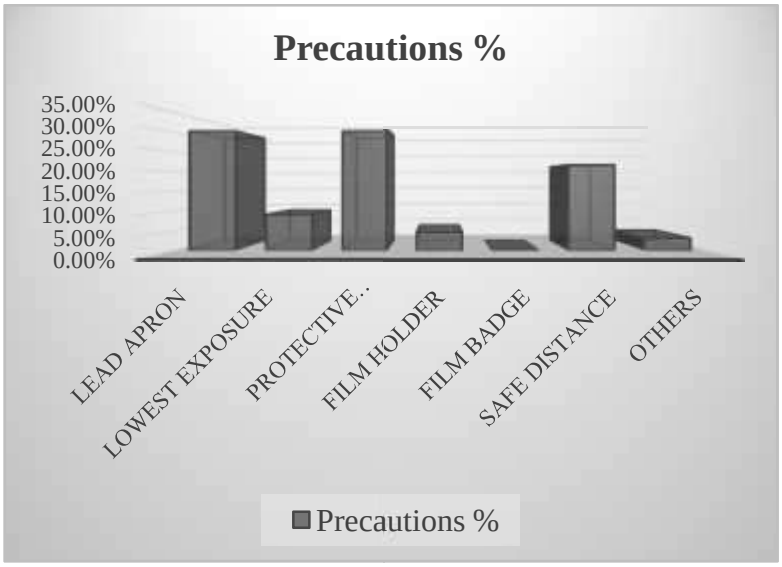


Figure 3: Radiation protection methods being practiced by the dentist

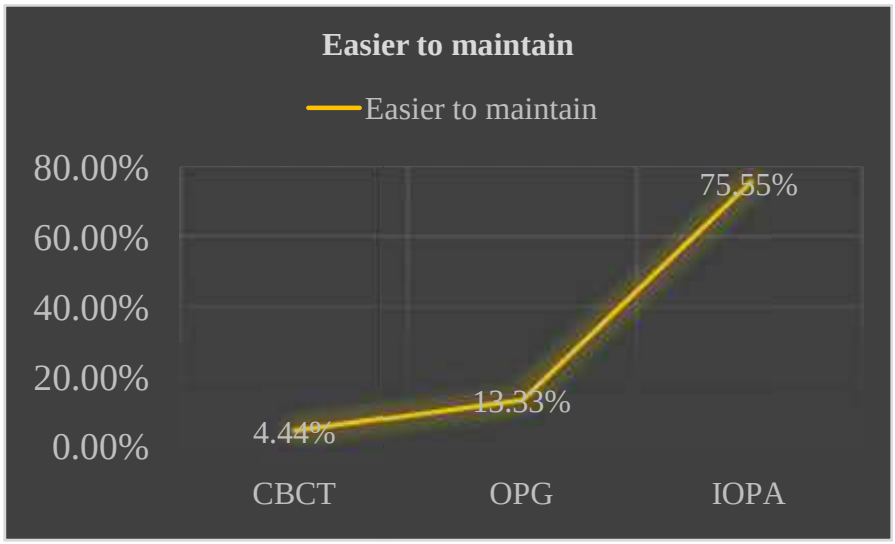


Figure 4: Mode of digital imaging modality is easier to maintain in practice

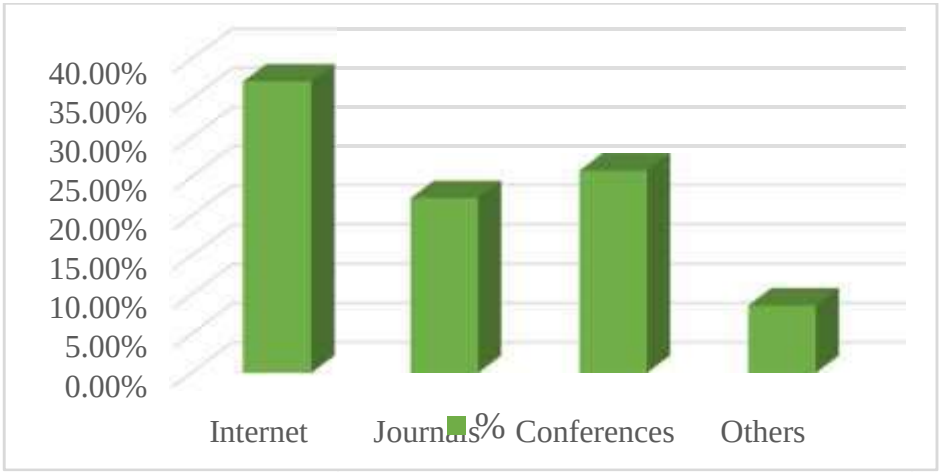


Figure 5: Distribution of dental practitioners willing to enhance knowledge regarding advances in digital imaging techniques through various modalities.

Discussion

The data shows that the preference of digital radiography is increasing among dentists over the past years. Among the participants of this study, 69% have started using digital radiography over the past 5 years alone. Wenzel and Moystad in 2001 reported the use of

digital radiography in 2001 as 14% of their sample, and compared with the 67% in a 2011 study by Dolekoglu et al. [6]This shows that the use of digital imaging modalities has probably increased over the years with the advancement in technology.Also, dental practitioners who have recently graduated were found to be more familiar with digital radiographic examination, considering the recent development of digital technology. A similar result was obtained by Karla Rovaris et. al who conducted a survey in Brazil to investigate the use and acceptance of digital radiographic examinations by dental practitioners in Brazil, which also is a developing country like India. [7]

The various types of intra oral radiographic methods used are: periapical, occlusal and bitewing radiographs. The periapical radiograph provides diagnostic information involving the tooth and periapical regions. The occlusal radiograph helps to visualize a larger pathology like a cyst which cannot be visualized by any other radiographic method. The bitewing radiographs help to evaluate the interproximal regions of adjacent and opposing teeth simultaneously. The extra oral radiographic methods used in dentistry include panoramic radiographs, lateral skull view, Posterior- Anterior View and Lateral Cephalogram. CBCT is another imaging technique which is a recent development and has proven to have better accuracy in determining the extent and location of anatomical structures and defects in the teeth and surrounding structures.

Also, digital imaging has profound advantages than conventional imaging modalities. It has been stated that the amount of dose reduction for intra oral digital imaging is about 50%- 60% when an E- speed film is used. Other advantages include short processing time, elimination of a dark room, chemical handling and errors associated with developing the film image.A study was done by Mohammed Mahdi et. al to compare the accuracy of conventional and digital radiography in root canal working length determination and the results of the study state that there was no difference observed when using CCD, PSP and conventional imaging techniques.[8]The safe distance from the radiation exposure was known by around 64.4% of the dentists who undertook the survey. In a similarstudy done by Katarzyna et. al to assess the knowledge regarding digital radiography and CBCT, it was found that the radiation awareness among dentists in inadequate. [9]

The most commonly preferred imaging modality for the 3D imaging of the head and neck region was stated to be CBCT (47.7%). CBCT processes two dimensional cone- beam projections and provides a 3 Dimensional full volumetric reconstruction so the target area

can be assessed in all planes. [10]CBCT has profound uses in dentistry, which includes the localization of supernumerary teeth, detection of cysts and tumors of the jaw, assessment of root canal configurations, detection of root fractures, treatment planning for placement of dental implants, peri- implant bone defects and orthodontic diagnosis.[11]Another study done by Keerthana et. al has stated that about 82% were aware of CBCT being used in maxillofacial radiology. [12] The Periapical lesions become visible on radiographs only when 30-60% of mineral bone loss has happened. When such peri apical lesions are covered by a thick cortex or cancellous bone, they could be radiographically undetectable. Two dimensional images restrict the information regarding the size and extent of the periapical lesion. In such cases, a three dimensional image is essential which is provided by the Cone Beam Computed Tomography which is not considered as the gold standard in endodontics. [13] CBCT is said to give all the information as given by a CT, at just $1/8^{th}$ of the radiation dose. [14]However, maintenance and installment of CBCT is not very easy. In this study, 40% had stated that they did not know the maximum permissible dose of radiation for a dentist. This has to be addressed with prime concern because knowledge regarding the permissible dosage is essential to limit and prevent damage to the body cells which are caused by radiation exposure.Some epidemiological studies show that there is no increased risk of cancer among the dentist population, whereas other studies show a higher prevalence of thyroid and breast cancer among female dentists and of melanomas in male dentists. [15] This brings us to a decision that knowledge regarding the safety dosage limit and precautions has to be well known by dentists in order to prevent any systemic complications. In this study, 51% of dentists have stated that they use radiation protection equipments in their daily practice. However, a majority of dentists at large are not making use of protective equipments. This has to be treated with concern and knowledge regarding radiation protection must be emphasized.

Conclusion

Recent developments offer better quality imaging which has to be made known to the dental practitioners. Dentists should prescribe special imaging only when they expect that diagnostic yield will benefit the patient care and improve clinical outcomes significantly. More awareness of the radiation protection and knowledge about the safe dosage and

distance has to be made known to the dentists to ensure maximum benefit as well as as low as harm possible.

Corresponding Author

Deepa Gurunathan

Professor, Department of Pedodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id: drgdeepa@yahoo.co.in

Phone Number: 9994619368

References

1. Naseem Shah, Nikhil Bansal, Ajay Logani. Recent advances in imaging technologies in dentistry. World J Radiol 2014; 28(6): 794- 807.

2. R Jcobs, M Vanderstappen, R Bogerts and F Gijbels. Attitude of the Belgian dentist population towards radiation protection. Dentomaxillofacial Radiol 2004; 33: 334- 339.

3. Bhawana A Bhagat, Arun P Nagrik, Sushil B Yemle. Dental Practitioner's Awareness, Knowledge and Attitude towards Cone Beam Computed Tomography. IOSR- JDMS 2016;15: 33- 37.

4. K Kamburoglu, S Kursun and ZZ Aksarslan. Dental students knowledge and attitudes towards cone beam computed tomography in Turkey. Dentomaxillofacial Radiol 2011; 40: 439- 443.

5. Shishir Ram Shetty, Renita Lorina Castelino, Subhas G Babu, Prasanna, Anusha Rangare Laxmana, Roopashri. Knowledge and attitude of dentists towards cone beam computed tomography in Mangalore- a questionnaire survey. Austin J Radiol 2015; 2(2): 1016- 1021)

6. Mojdeh Mehdizadeh, Sahar Goli Booshehri, Farimah Kazemzadeh, Parisa Soltani, Mahmood Reza Kalantar Motamedi. Level of knowledge of dental practitioners in Isfahan, Iran about cone- beam computed tomography and digital radiography. Imaging Sci Dent 2015; 45: 133- 135.

7. Karla Rovaris. Brazilian young dental practitioners use and acceptance of digital radiographic examinations. Imaging Sci Dent 2016; 46: 239- 244.

8. Mohammad Mahdi Yaghooti Khorasani and Hamed Ebrahimnejad. Comparision of the accuracy of conventional and digital radiography in root canal working length detetmination: An in vitro study. JODD 2017; 11(3): 161- 165.

9. Mine Gecgelen Cesur,Alev Yilmaz, Torun Ozer. Knowledge and attitude towards digital radiography and CBCT among orthodontists. Biomed Res 2016; 27(3): 959- 964)

10. Adriana Gabriela Creanga, Hassem Geha, Vidya Sankar, Fabricio B. Teixeira, Clyde Alex McMahan, and Marcel Noujeim. Accuracy of digital periapical radiography and cone-beam computed tomography in detecting external root resorption. Imag Sci Dent 2015; 45: 153- 158.

11. Nicolau Silveria- Neto, Mateus Ericson Flores, João Paulo De Carli, Max Dória Costa, Felipe de Souza Matos,Luiz Renato Paranhos,' and Maria Salete Sandini Linden. Peri-implant assessment via cone beam computed tomography and digital periapical radiography: an ex- vivo study. Clinics 2-17; 72(11): 708- 713.

12. Keerthana Balabaskaran, Dr. Arathy Srinivasan. Awareness and attitude among dental professional towards CBCT. IOSR- JDMS 2013; 10(5): 55- 59.

13. Tadas Venskutonis et. al. Accuracy of Digital Radiography and cone beam computed tomography on periapical radiolucency detection in endodontically treated teeth. J Oral Maxillofac Res 2014 2014; 5(2): 1-4.

14. The usage of digital radiography and cone beam computed tomography among Turkish Dentists. Dolekoglu et al, Dentomaxillofacial Radiology 2007; 40: 379- 384.

15. Various Imaging Modalities for the investigation of Dental Implant: A Cross- Sectional Study. Mounika S, Dr. Revathy Goundar, Research J. of Pharm and Tech 2016; 9 (11): 1930-1932.

16. Naseem Shah, Nikhil Bansal, Ajay Logani. Recent advances in imaging technologies in dentistry. World J Radiol 2014; 28(6): 794- 807.

17. R Jcobs, M Vanderstappen, R Bogerts and F Gijbels. Attitude of the Belgian dentist population towards radiation protection.Dentomaxillofacial Radiol 2004; 33: 334- 339.

18. Bhawana A Bhagat, Arun P Nagrik, Sushil B Yemle. Dental Practitioner's Awareness, Knowledge and Attitude towards Cone Beam Computed Tomography. IOSR- JDMS 2016;15: 33- 37.

19. K Kamburoglu, S Kursun and ZZ Aksarslan. Dental students knowledge and attitudes towards cone beam computed tomography in Turkey. Dentomaxillofacial Radiol 2011; 40: 439- 443.

20. Shishir Ram Shetty, RenitaLorinaCastelino, Subhas G Babu, Prasanna, AnushaRangareLaxmana, Roopashri. Knowledge and attitude of dentists towards cone beam computed tomography in Mangalore- a questionnaire survey. Austin J Radiol 2015; 2(2): 1016- 1021)

Knowledge and Practice of Endodontic Treatment Carried Out by General Practitioners in Chennai- A Questionnaire Survey

RathnaSubhashini M.H[1], Shabeenataj[2], Raghu Sandhya[3]

Graduate Student[1],Tutor[2], Department of Public Health Dentistry, Reader[3],Department of Conservative Dentistry and Endodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Root canal treatment (RCT) is taken into account as a necessary component within the dental services provided to the population in developed furthermore as developing countries.It is the responsibility of the academics and dental schools to prepare their students to adopt the guidelines and recommended standards in root canal debridement, shaping and obturation..The aim of this questionnaire survey was to evaluate the knowledge and practice of endodontic treatment carried out by general practitioners in Chennai.

Material and Method: A questionnaire was developed with 15 questions based on knowledge and practice ofendodontic treatment and was sent to 50 general dental practitioners (n = 50). The collected data were entered into a personal computer and analyzed.

Results: In the present study all the participants, i.e. 100% reported that they perform root canal procedures in posterior teeth. Isolation was mainly done using rubber dam in 32%of dentists. The step back technique was the most popular root canal preparation technique (76%).42% of dentists in Chennai tended to use hand instruments and 40% of the themprefer K-files for cleaning and shaping the root canals. Every dentists in Chennai take radiographs in every steps of RCT. 58% of dentists used normal saline for irrigation.Every dentists reported that, they use gutta- percha as obturating material with 52% of them used lateral condensation technique.Every dentists used Zinc- oxide eugenol as sealer. The coronal access cavity was sealed by temporary restorative material immediately after the treatment and the permanent sealing was done later on by themselves.

Conclusion: Based on this survey, it was demonstrated that dentists performed procedures which often deviated from well-acknowledged endodontic quality guidelines. General practitioners did not seem to keep up with recently introduced techniques, but use more conventional methods. Most of the conventional techniques used by dentists were reported to have increased success rate.

Key Words: *Endodontic treatment, isolation, irrigants, instruments, obturation, filling material.*

Introduction

Endodontic treatment is an essential part of comprehensive quality dental care. Root canal treatment (RCT) is defined as the combination of mechanical instrumentation of root canal system, its chemical debridement and filling with an inert material, designed to maintain or restore the health of the peri-radicular tissues. The manner of execution of treatment procedures is so diverse even within prescribed protocols that it is difficult to define it any more precisely and it is accepted that this treatment intervention is not by its nature standardizable. [1,2]

The goal of endodontic and restorative therapy is to restore the normal function and occlusion of the tooth and to maintain the stability of the dental arch. The strength of the endodontically treated teeth is directly connected to the bulk of the remaining dentin. Root filled teeth with intact coronal structure have a good long term prognosis [2,3]. Several studies have revealed that the majority of dentists do not comply with the formulated guidelines on the quality of RCT[4-7]. It is the responsibility of the academics and dental schools to prepare their students to adopt the guidelines and recommended standards in root canal debridement, shaping and obturation [1,3] .The rationale of this study was to evaluate the practice and knowledge about root canal treatment of molar teeth by private general dental practitioners in Chennai. The purpose of the questionnaire was not only to collect baseline data, but also to determine the endodontic knowledge and practice of dentists. Furthermore, it was hoped to gain an insight into potential problems regarding endodontic treatment procedures that could explain the present standard of RCT carried out by general dental practitioners.The aim of this cross-sectional survey was to evaluate the knowledge and practice of endodontic treatment carried out bygeneral practitioners in Chennai.

Material and Method

A survey on general dental practitioners in Chennai was carried out to investigate common materials and methods employed in RCT. A questionnaire was developed with 15 questions based on knowledge and practice about endodontic treatment and was sent to 50 general dental practitioners (n = 50). The collected data were entered into a personal computer and analyzed. The questionnaire consisted ofquestions concerning different aspects of endodontic

treatment including the provision of root canal therapy stages, materials, the choice of instruments, isolation methods, radiographs taken throughout the treatment, the use of canal irrigants and intracanal medicaments, the choice of obturation technique, temporary and permanent coronal restoration, and case monitoring and follow-up. The collected data were entered into a personal computer and analyzed.

Result

Based on the questionnaire survey, it is found that , all 50 practitioners performed RCTby themselves including molar root canal treatment(100%). The practitioners did not use rubber dam for all the treatment. Considering the frequency of usage 90% dentists reported occasionalusage of rubber dam(90%). Almost everyone preferred to take radiograph in every step of the treatment. Gutta-percha (100%)was the only material used by every dentist in Chennai to obturate with zinc-oxide eugenol (100%)as sealer. The coronal access cavity was sealed by temporary restorative material immediately after the treatment and the permanent sealing was done later on by themselves.

Table 1: Choice of isolation method

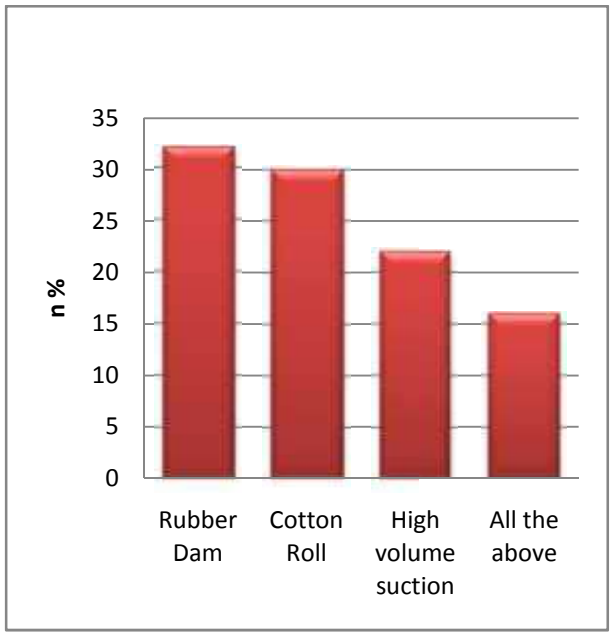


Table 2: Choice of cleaning and shaping technique

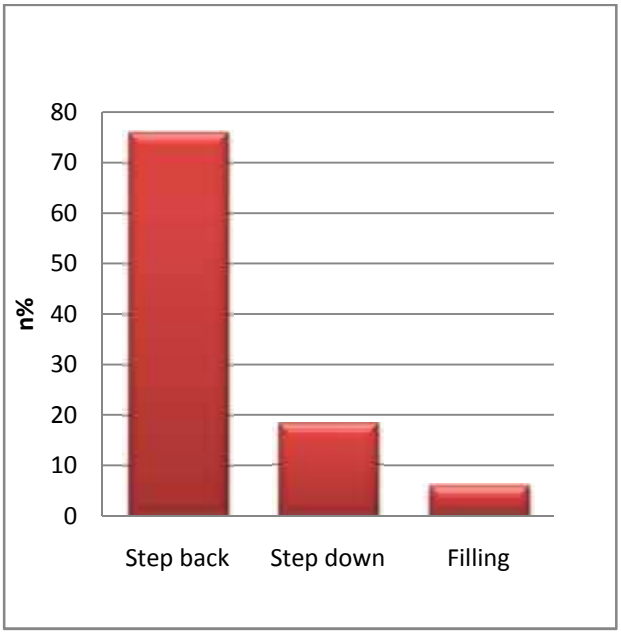


Table 3: Type of instrument

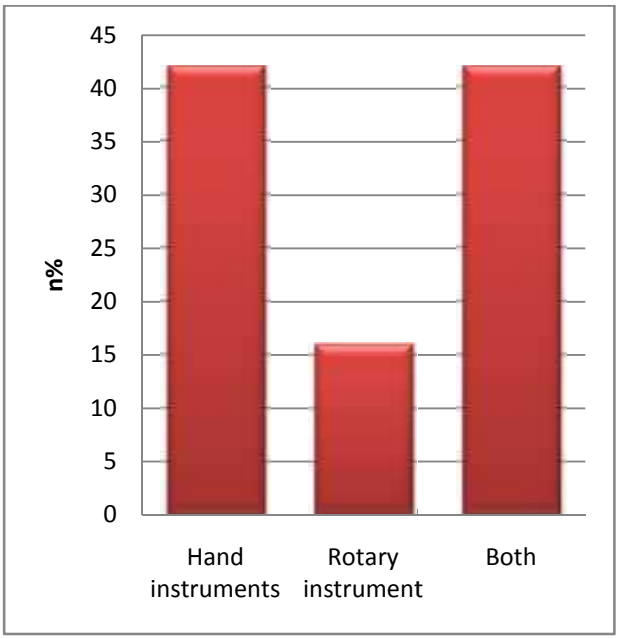


Table 4: Preference of hand instruments

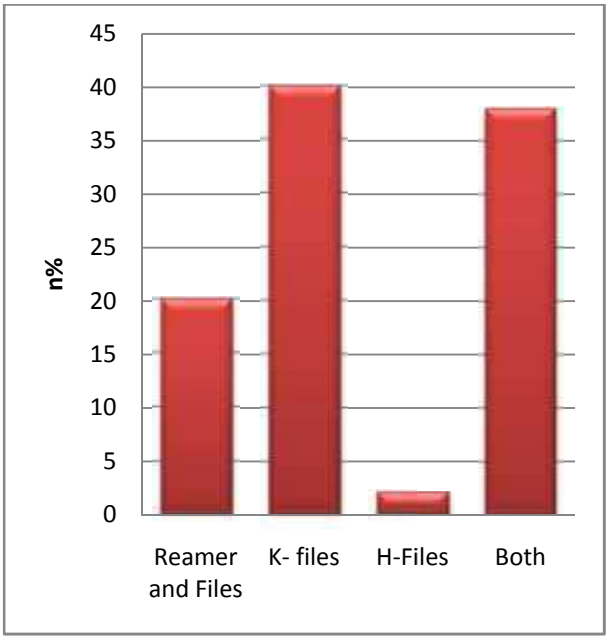


Table 5: Choice of irrigants

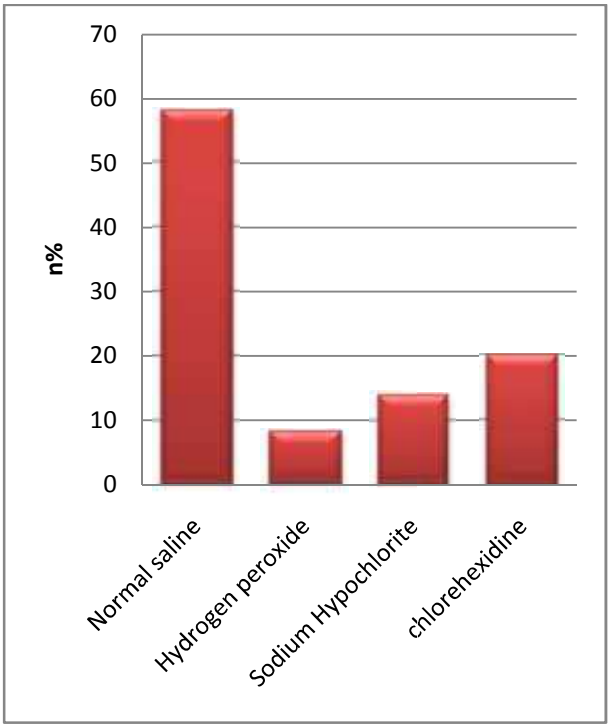


Table 6: Preference of obturation technique

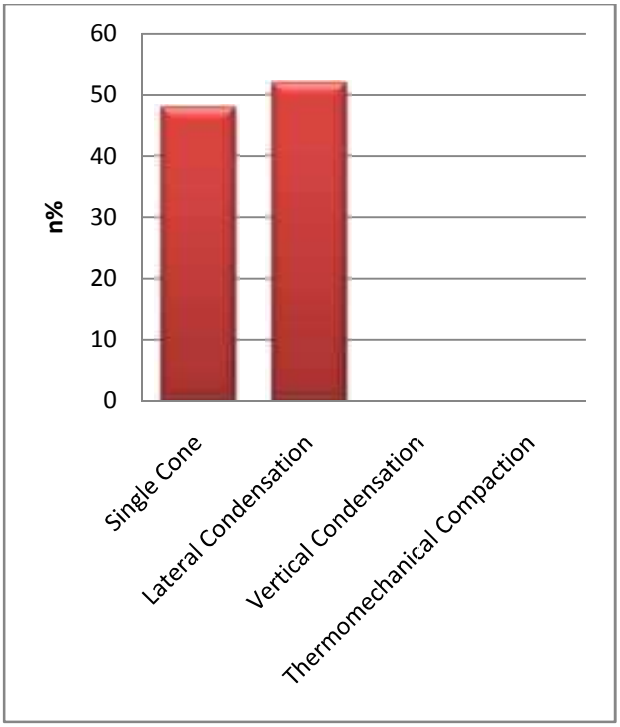
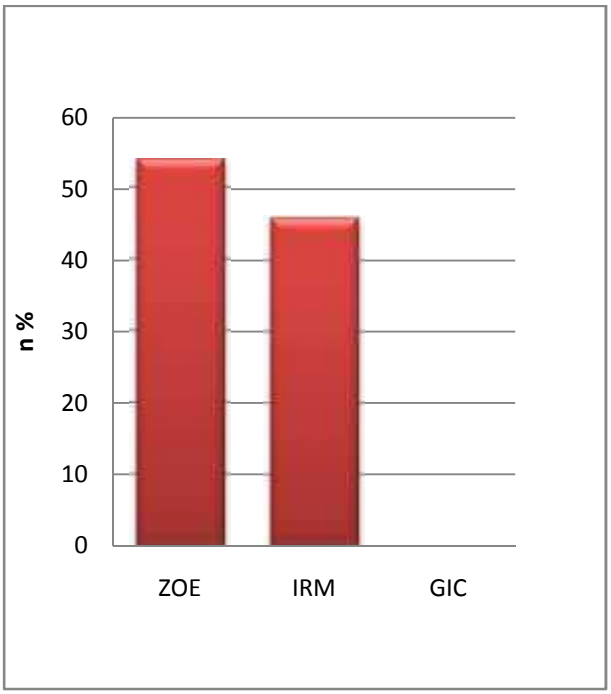


Table 7: Preference of temporary restorative material



Discussion

The reason for this study was to gather both qualitative and quantitative information on endodontic treatment by dentists working in Chennai and to analyze the advantages and disadvantages of each. In the present study all the participants, i.e. 100% reported that they perform root canal procedures in posterior teeth. The use of rubber dam is always recommended during root canal treatment to provide isolation, protection and improve visibility, 32% of dentists reported using rubber dam occasionally while, 30% of dentist use cotton rolls for isolation. However, 59% of American dentists [9] , 60% of dentists in UK and 57% dental practitioners in New Zealand [10,11] reported using rubber dam during every endodontic procedures.The reasons for not using rubber dam could be the extra cost, additional time, lack of adequate skills or training, absence of patient's acceptability or inadequate education in the undergraduate teaching curriculum [12]

The step back technique was the most popular canal preparation technique among general dental practitioners for about 76%. The filing (push-pull) technique, on the other hand, was used by only 6 % of the dentists. Whereas, 18% of dentists use step down technique. Generally, dentists in Chennai tended to use hand instruments 42% and 16% of dentists use engine driven techniques for shaping the root canal system, while 42% of dentists reported using both. Most of the dentists prefer K-files are used for shaping the canals(40%), 20% of dentists use Reamers and K-files, and very few doctors use H- files(2%). 38% percentage of dentists use all three instruments for better quality of treatment. Every dentists in Chennai take radiographs in every steps of RCT. In the current survey, most general dental practitioners used normal saline(58%) as root canal irrigant, 20% of dentists in Chennai usedchlorhexidine as intracanalirrigant, while 14% used sodium hypochlorite. The material of choice for irrigating the root canal system wassodium hypochlorite because of its effective antimicrobial and tissue solving action. However, using sodium hypochlorite or hydrogen peroxide without isolating the field of operation with a rubber dam presents a hazardous practice. A similar trend ofusing sodium

hypochlorite as a root canal irrigant without using rubber dam for isolation, was noticed amongst Flemish dentists [6]. A very few practitioners use hydrogen peroxide as irrigant in endodontic treatment.

Absolutely every dentists in Chennai use gutta-percha points as obturation material in endodontic treatment. 52% of dentists used lateral condensation technique for obturation which had increased success rate, where 48% used single cone technique which is easier and less time consuming. Cold lateral condensation of gutta-percha in conjunction with a root canal sealer is the most widely accepted technique for obturating root canals[13]. It is a relatively simple and versatile technique that does not require expensive equipment. It is therefore not surprising that it is the technique used by the majority of responding practitioners (especially the younger ones) in their general practice. Single-cone/point techniques cannot reliably fill all the root canal space in three dimensions and are not recommended, although this technique was practiced once upon a time. Similarly, paste only root fillings are difficult to control with the obvious risk of under- or over-filling of the canal. This is particularly problematic with paraformaldehyde-based sealers, as they can cause extensive damage to the peri-radicular tissues [14-16]. The trend is toward the use of warm gutta-percha techniques to improve the filling of accessory anatomy such as fins and apical deltas[17]. However, 31.3% of the dentists in Jordan used a single cone technique, in common with 68% of Swiss dentists[18]. The temporary filling material after endodontic treatment used in most common is zinc oxide eugenol by 54% of doctors, and 46% use intermediate restorative material as temporary filling material.

Based on this study, the number of practitioners using inappropriate techniques/materials was quite small and there are possibilities of changing behavior with education to bring successful results [19-23]. This study sheds light on current preferences for various endodontic materials and techniques. It also notes the potential possibility for educational interventions.

Conclusion

Within the limitations of this study, itwas demonstrated that dentists performed procedures which occasionally deviated from well-acknowledged endodontic quality guidelines. General practitioners seem to use more conventional methods. It is recommended and vital to impart higher level of continuing dental education for general practitioners to update their knowledge in the field of endodontics.

Corresponding Author

Raghu Sandhya,

Reader,Department of Conservative Dentistry and Endodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Science,Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Mobile Number: 9884610410,

Email: drsandhyaendo@gmail.com

Reference

1. Y.-L. Ng1 , V. Mann , S. Rahbaran , J. Lewsey , K. Gulabivala. Outcome of primary root canal treatment: systematic review of the literature – Part 2. Influence of clinical factors. International Endodontic Journal 2008;41(1): 6-31

2.Khalid S, Al- Fouzan, A survey of root canal treatment of molar teeth by general dental practitioners in private practice in Saudi Arabia. The Saudi Dental Journal2010 ; 22(3): 113-117.

3.Iftikhar Akbar.Knowledge, Attitudes and Practice of Restoring Endodontically Treated Teeth by Dentists in North of Saudi Arabia . International Journal of Health Sciences 2015 ; 9(1): 41–49**.**

4.Gatewood RS, Himel VT, Dorn SO: Treatment of the endodontic emergency: a decade later. Journal ofEndodontics 1990; 16(6): 284-291.

5.Al-Omari W M, Survey of attitudes, materials and methods employed in endodontic treatment by general dental practitioners in North Jordan . Biomed Central 2004;4:1.

6.Weiger R, Hitzler S, Hermle G, Lost C: Periapical status, quality of root canal fillings and estimated endodontic treatment needs in an urban German population. Endodontic Dental Traumatology 2006; 13(2): 69-74.

7. Jenkins SM, Hayes SJ, Dummer P. A study of endodontic treatment carried out in dental practice within the UK. International Endodontic Journal 2001, 34(1): 16-22.

8.Slaus G, Bottengerg P: A survey of endodontic practice amongst Flemish dentists. International Endodontic Journal 2002, 35(9): 759-767.

9. Hommez G. M. G ,Braem M , De Moor R. J. G. Root canal treatment performed by Flemish dentists. Part 1.cleaning and shaping. International Endodontic Journal 2003;36:166-173.

10. Whitten BH, Gardiner DL, Jeansonne BG, Lemon RR: Current trends in endodontic treatment: report of a national survey. Journal of American Dental Association 1996, 127(9): 133-341.

11. Koshy S, Chandler NP: Use of rubber dam and its association with other endodontic procedures in New Zealand.. NewZealand Dental Journal. 2002; 98(431): 12-6.

12. Ahmed HM, Cohen S, Lévy G, Steier L, Bukiet F. Rubber dam application in endodontic practice: an update on critical educational and ethical dilemmas. Australian dental journal. 2014 Dec 1;59(4):457-63.

13.R.V. Geetha, Vishnu Priya Veeraraghavan. Evaluation of Antibacterial Activity of Five Root Canal Sealants against Enterococcus faecalis – An In vitro Study. International Journal of Pharmaceutical Sciences Review and Research 2016;40(2): 221-223

14.Kaufman AY, Rosenberg L. Paresthesia caused byEndomethasone. Journal of Endodontics 1980; 6(4): 592–3

15.K Marshall, J page. The use of rubber dam in UK- A survey. British Dental Journal 1990;169:286-291.

16. Erison RT, Yucel T, Kucukay S. Endomethasone root canal filling material in the mandibular canal: a case report. Oral Surgery, Oral Medicine and Oral Pathology 1989:68(3), 343–5.

17.Priyanka.S.R , Dr.Veronica.A Literature Review of Root-End Filling Materials. Journal of Dental and Medical Sciences 2013;9(4):20-25

18. Barbakow F: The status of root canal therapy in Switzerland in 1993. Journalof the DentalAssociation South Africa 1996, 51(12): 819-822.

19. Lawson NC, Gilbert GH, Funkhouser E, Eleazer PD, Benjamin PL, Worley DC, National Dental PBRN Collaborative Group General dentists' use of isolation techniques during endodontic procedures: from the National Dental Practice-Based Research Network. Journal Endodontics 2015; 41(8):1219-25.

20. American Dental Association. Bureau of Economic Research, American Dental Association.Survey Center.Survey of dental practice. American Dental Association, Bureau of Economic Research and Statistics.; 1965.

21. Koch M, Eriksson HG, Axelsson S, Tegelberg A. Effect of educational intervention on adoption of new endodontic technology by general dental practitioners: a questionnaire survey. International Journal of Dentistry2009;42(4):313–21.

22.Gound TG, Sather JP, Kong TS, Makkawy HA, Marx DB. Graduating dental students' ability to produce quality root canal fillings using single- or multiple-cone obturation techniques. Journal ofDental Education 2009;73(6):696–705.

23. Hale R, Gatti R, Glickman GN, Opperman LA. Comparative analysis of carrier-based obturation and lateral compaction: a retrospective clinical outcomes study. International journal of dentistry. 2012;2012.

Knowledge of Undergraduates in Dental Radiology

M.Indhumathi[1], Shantha Rajan[2],Hemavathy.O.R[3]
Graduate Student[1],Senior Lecturer[2], Department of Pedodontics,Reader[3], Department of Oral and Maxillofacial Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Dental radiographs are important tools in the diagnosis of oral diseases.It helps the dentist for appropriate treatment for patients and dental conditions and play a continual role in dental practice.So it's availability in dental clinics are very important for dental procedures.Radiation dosage from such dental radiographs are low and it shows minimal risk in patients. The aim of the study is to evaluate the knowledge of under graduates in dental radiology.

Materials and Methods: The study was conducted in Saveetha dental college, chennai, India. The study was conducted among78 final year dental students, of which 46 were females and 32 were males. This was cross-sectional questionnaire based study on radiation protection among dental students. Correct responses to the questions were allocated 1 mark while wrong response received no mark. Statistical analysis was performed to analyse the study.

Results: Among 78 final year students, who participated in the following study,only57(83.3%)students were aware of radiation protection and about 21(27.2%) students should improve their knowledge of dental radiation.

Conclusion: There is need to expand the curriculum to provide better exposure to radiation protection and its practice, so that the dental students on graduation will be well grounded with the principle governing dental radiography.

Key Words: *Assessment, dental students, knowledge, radiation protection, radiation practice*

Introduction

Dental radiographs are important tools in the diagnosis of oral diseases. It helps the dentist to employ appropriate treatment regimen for patients' dental conditions, and plays a continual important role in dental practice. Hence its availability in most dental clinics is considered crucial. Radiation dosage from such dental radiographs are low and are thought to present minimal risk.It is estimated that about 480 million dental radiographs are performed annually accounting for approximately 15% of all diagnostic X-ray examinations.[1]Consequently concern radiation biology and protection and radiology of oral diseases among other topics.These students are also exposed to clinical postings in the oral diagnosis and radiology clinic for a period of 4 weeks in the 5th year . On completion of the course, the undergraduate student is expected to be able to explain the production of ionizing radiation, image formation and principles of radiation biology and protection from effects of ionizing radiation to oneself, clinical staff and patients.[3] Eventually these dental students would metamorphose to dentists and may perform dental radiography in their clinics. Their knowledge and practice on dental radiography are consequently crucial. [2]

Dentists take radiographs in the evaluation of their patients. During undergraduate and post graduate training dentists are trained to perform dental radiographic exposures particularly during clinical rotations. In India students are allowed to take dental radiographs under supervision. The undergraduate dental training in Saveetha dental college is divided into the pre-clinical levels ($1^{st}$and 2^{nd}year) of study and the clinical levels (3rd, 4th and 5thyear) of study. In the institution where this study was carried out the fifthyear curriculum of dental students includes two schedules each of lectures in dental radiology with the second schedule of the fifthyear having basic radiation physics, the production of X-rays, radiation biology and protection and radiology of oral diseases among other topics.[3] These students are also exposed to clinical postings in the oral diagnosis and radiology clinic for a period of 4 weeks in the 5th year.. On completion of the course, the undergraduate student is expected to be able to explain the production of ionizing radiation, image formation and principles of radiation biology and protection from effects of ionizing radiation to oneself, clinical staff and patients.[3] Eventually these dental students would metamorphose to dentists and may perform dental radiography in their clinics. Their knowledge and practice on dental radiography are consequently crucial. [4]

Materials and Methods

This is a cross-sectional study on radiation protection conducted at Saveetha dental college. A four section questionnaire was administered to volunteering final year dental students at the Saveetha dental college. The questionnaires were collected after being filled by the students within the hour of administration with all the students seated and interaction between students discouraged. Correct responses were allocated 1 mark while wrong response receives no mark. [5] No point was subtracted for wrong responses. There was no student identifier in the questionnaires. The first section consists of socio-demographic data and questions bordering on if the students had previous exposure to formal lectures on radiation protection and pre-medical school graduate certificate. The second section consists of questions on radiobiology while the third section was on radiation protection. The fourth section was meant to test good radiation protection practice and also contains other questions such as if the students feels that their knowledge on radiation protection was adequate and the kVp of the machine they use. Each of the second, third and fourth sections have five questions each for assessing that section. Radiobiology, radiation protection and practice scores were categorized into; 0 to 2 points (poor), 3 points and above (good).The correct responses of radiobiology (section 2) and radiation protection (section 3) were summated to yield ‘‘total knowledge on radiation protection’’ which was subsequently graded into ‘‘poor’’ (0 to 4 points) and ‘‘good’’ (5 to 10 points). Statistical analysis was performed using SPSS version 17 (Chicago, IL). Pearson’s coefficientcorrelation analysis was performed to establish relationship between various variables with the significant level set at 5%.[6]

Results

The study was conducted among78 final year dental students, of which 46 were females and 32 were males. The mean score of the students on knowledge of radiobiology was 1.85 ± 1.19 (of 5 points). The proportion of students with a score of 2 points was the most, accounting for 22 (28.2%) of all the students. None of the students correctly answered all the questions on radiobiology. In comparison to the knowledge of radiobiology, knowledge of radiation protection was abysmally poor as the mean score was 0.92 ± 0.80 (of 5 points) while the modal score was zero point (accounting for 24 (30.8%) of the students). Only one student correctly ranked the effectiveness of the methods of protecting patients. Surprisingly,

none of the student knew the correct annual radiation dose limit for a dentist and only 14(17.9%) of the total number of students correctly knew what each of the acronyms of ALARA stood for. [8]Overall, the mean of the total (sum of radiobiology and radiation protection scores) score by the students was 5.45 ± 2.66 (of 10 points) and the modal score was 3 points (23.1%). No student was observed to have a score of 10. On categorizing the total knowledge of radiation protection, 12(15.4%) students had ‘‘good’’ score while 66(84.6%) students had ‘‘poor’’ score. Although none of the students used film badge, the other indices tested showed that they had good radiation protection practice knowledge — [Table 1]. The mean score of radiation protection practice was 2.69 ± 1.42 (of 5 maximal points) and modal score of 4 points (39.7%). The proportion of students with ‘‘good’’ radiation protection practice rating was 50 (64.1%) whereas 28 (35.9%) had ‘‘poor’’ rating. Undermining that 52 (66.7%) of the students responded that they used the lowest possible settings during dental radiographic exposure on patients, only 2 (2.6%) actually knew the kVp of the machine used in the department. The mean radiographs taken by these students monthly were 12.87 ± 8.8 with 53 (67.9%) students taking an average radiographs of 0 to 9 per month (‘‘poor’’ rating) and 25 (32.1%) took over 9 radiographs/month (‘‘good’’ rating). There was no significant correlation between the number of radiographs taken and knowledge of radiation protection or practice. A greater proportion of students with ‘‘good’’ radiation protection knowledge in comparison to those with ‘‘poor’’ knowledge kept a distance of over 3 metres from the patients/X-ray tube [7 (58.3%) vs. 34 (51.5%)], wore lead apron [12 (100%) vs. 54 (81.8%)], used the lowest possible settings on the x-ray machine [11 (91.7%) vs. 41 (62.1%)] and used collimators [10 (83.3% vs. 40 (60.6%)] — Table 1. Of the preceding tested parameters of radiation protection practice those that used the lowest possible settings was at statistical significant level, P = 0.046. Most 59 (75.6%) of the students thought they did not have adequate knowledge on radiation protection.

Radiological investigations should only be prescribed by medical personnel for specific purposes when the benefit outweighs the risk. Thus, it is imperative that the dentist ensure that adequate justification for the dental radiograph is met and that minimal permissible exposure is given. These dentists should be knowledgeable on radiation protection in order to properly protect the patients, themselves and others around them and their knowledge of ionizing radiation from medical devices makes explanation of the benefitand risk to the patients easy. In order to assess knowledge on radiation protection it is pertinent to establish the level of awareness on radiobiology. These students had an appreciable degree of

knowledge on radiobiology. Radiation causes cell damage by ionization with the consequent formation of ions that can produce free radicals, break chemical bonds, creates cross-linkage between macromolecules or damage molecules and genes. [9]

Table 1: Questionnaire

Radiation causes cell injury by?	Thermal effects 18 (10.3%)	Radio frequency 20 (24.4%)	Ionization 35(43.6%)	I don't know 10 (21.8%)
Susceptible cells to radiation are usually;	Somatic cells 18 (23.1%)	Rapidly dividing unspecialised cells 41 (52.6%)	I don't know 19 (24.4%)	
Cell damaged by radiation:	Cannot be repaired 25 (32.1%)	Is reparable 15(19.2%)	Always leads to cell death 18 (23.1%)	I don't know 20 (25.6%)
Which of the following radiation sources affects normal people the most?	Industry 33 (42.3%)	Medical 13 (16.7%)	Natural sources 15 (19.2%)	I don't know 17 (21.8%)
Foetal tissues are susceptible to radiation especially during: Radiation protection	<20 weeks 42 (53.8%)	20 to 30 weeks 11 (14.1%)	30 weeks to term 2 (2.6%)	I don't know 23 (29.5%)

Table 2: Effectiveness of protection

Ranking of the methods of protecting the	Reducing repeat rate: 9 (11.5%)	Modern fast film: 6 (7.1%)	Thyroid shield: 13 (16.7%)	Lead apron: 3 (3.8%)

patient, starting with the most affective:				
What is the annual radiation dose limit for a dentist?	I don't know 74 (94.9%)	12 mSv 1 (1.3%)	250mSv 1 (1.3%)	No limit 2 (2.6%)
Do you know what ALARA means:	Yes: 14 (17.9%)	No: 64 (82.1%)		
Which head and neck organ will you like to protect from radiation?	Thyroid 53 (67.9%)	Brain 8 (10.3%)	Nostril 1 (1.3%)	I don't know 16 (20.5%)
How much radiation does a patient absorb during a single peri-apical x-ray exposure?	0.2mSv 2 (2.6%)	4 (5.1%)	0.005mSv 8 (10.3%)	I don't know 62 (79.5%)

Discussion

Table 3: Radiation protection practice

	Yes	**No**
Keep a distance of greater than 3 meters from the patient:	41 (52.6%)	37 (47.4%)
Wear a lead apron:	66 (84.6%)	12 (15.4%)
Use lowest possible setting possible:	52 (66.7%)	26 (33.3%)
Use film badge:	0 (0%)	78 (100%)
Use cone collimator:	50 (64.1%)	28 (35.9%)

Examples of such rapidly proliferating cells are found in foetuses; hence the developing foetus is particularly sensitive to the effects of radiation especially 2-9 weeks after conception which is the period of organogenesis.[15] for health care workers is 50 millisieverts (mSv) and the maximum permissible life time dose is 10 mSv multiplied by a person's age.[1] Concerning peri-apical radiograph dose, only 10.3% of these students could estimate the average radiation dose during a single peri-apical exposure. The effective dose from intra-oral peri-apical radiograph is 0.005 mSv.[14] Gijbels et al.[15] further determined the effective dose from peri-apical radiographs with rectangular collimation as 0.001 mSv (anterior), 0.001 mSv (lateral) and 0.003 mSv (posterior) while for round collimation the values were 0.001 mSv (anterior), 0.002 mSv (lateral) and 0.005 mSv (posterior). Hence, rectangular collimators reduce the radiation dose to patients when compared to conical collimators. Contrary to the earlier observation that these students have poor knowledge on radiation protection, majority (67.9%) of the students correctly opted to protect the thyroid gland rather than the brain or nostril during head and neck radiography. In the head and neck region the thyroid gland is one of the most sensitive organs.[6] Sikorski et al.[13] documented that radiation exposure reduction by thyroid shield varied from 5% to 56% for a complete-mouth survey, 2% to 18% for a bitewing survey, and 10% to 79% for a panoramic survey. In addition, their study showed that thyroid skin exposures measured on adults were 33% to 84% lower in offices in which thyroid shield was used as compared to offices in which it was not used and thyroid skin exposures measured on children were 63% to 92% lower. As a result it is highly recommended that leaded thyroid shields should be employed in children.[5] In comparison, knowledge on radiation protection was poor. Only one student knew the correct order of sequence for the effective radiation protection of patients for dental radiography. The most effective method is to reduce repeat rate, followed by the use of modern fast fi lm, thyroid shield utilization and finally lead apron. Reducing repeat rate comes with experience, adequate training and application of standard radiation protection practices with proper departmental quality control programme.[18] If no repeat radiograph is taken then there will be no additional undue exposure to radiation of the patients. Consequently, the exposing health professional should apply the appropriate settings and be familiar with the optimal settings to use. The use of faster fi lm speed can also reduce radiation exposure to about 50% while maintaining the diagnostic quality.[12] Poor knowledge of the meaning of ALARA was observed, as only 17.9% of the students actually

knew what the acronyms meant. It can be deduced that these students who were unfamiliar with the term may not be able to apply the principle of ALARA in practice. Consequently, patients may receive unnecessary radiation dose if the ALARA principle is not put into practice. Furthermore, none of the students knew the annual radiation dose limit for dentist. The maximum permissible annual dose of ionizing radiation Sixty-four percent of the student had good radiation protection practice while 367% had poor knowledge, an observation incongruous to knowledge on radiation protection assessment. None of the students had a thermolumincent dosimeter (TLD) badge despite some performing up to an average of 40 dental radiographs per month. Although 52.6% of these students kept a distance of at least 3 meters to the radiation beam, there is still some radiation dose absorbed from scatter radiation.[17] The direct and scatter radiation dose to the patient as well as the scatter radiation to the imager are considered very low, however some studies showed evidence of increased risk of brain, salivary gland, and thyroid tumours from dental radiographic exposure.[10] It is therefore crucial that other means of reducing radiation dose be employed. The use of collimation further reduces patients' primary and scattered radiation. Collimators are like aperture of a camera that reduces the field of irradiation and are typically conical/circular or rectangular. Rectangular collimators are five times more effective at reducing the radiation dose compared to conical collimators.[4,13] Consequently collimators should be provided in every dental radiographic unit, especially rectangular type. In the center where this study was conducted, rectangular and circular collimators are available, but the circular type is more commonly used.

Table 4: Cross-tabulations of total knowledge of radiation protection against radiation protection practise indices

Total knowledge classification	**Good**	**Poor**	**Total**
Keep a distance of >3m from patients			
Yes	7 (58.3%)	34 (51.5%)	41 (52.6%)
No	5 (41.7%)	32 (48.5%)	37 (47.4%)
Total	12 (100%)	66 (100%)	78 (100%)
			P=0.663

Wear lead apron Yes No Total	 12 (100%) 0 (0%) 12 (100%)	 54 (81.8%) 12(18.2%) 66 (100%)	 66 (84.6%) 12(15.4%) 78 (100%) P = 0.108
Use of lowest possible setting Yes No Total	 11 (91.7%) 1 (8.3%) 12 (100)	 41 (62.1%) 25 (37.9%) 66 (100%)	 52 (66.7%) 26 (33.3%) 78 (100%) P = 0.046
Use collimator Yes No Total	 10 (83.3%) 2 (16.7%) 12 (100%)	 40 (60.6%) 26 (39.4%) 66 (100%)	 50 (64.1%) 28 (35.9%) 78 (100%) P = 0.131

In this study, knowledge of radiation protection positively affected the students' practice of taking dental radiographs with reduced exposure to radiation.[15] A higher proportion of students with good radiation protection knowledge use the lowest possible setting in acquiring dental radiographs which was at statistical significant level (P = 0.046). Similarly a higher proportion of these students with good knowledge of radiation protection kept a distance of more than 3 metres from the patients, wore lead aprons and used collimators (although not at statistical significant level). [16]About 86.4% of the students wore lead apron while all those with good knowledge did wear lead apron which sharply contrast with the 8.7% that did so in the study on dentists in Turkey.[19,20] Methods to protect the imaging health personnel include education, implementation of radiation protection program and usage of barrier shielding. The radiation protection program should also limit the life time and annual radiation exposure within the allowable threshold. Training in radiation protection should be a continuous process even after graduation from dental school because studies have shown that substantial amount of knowledge is lost by 6-12 months following course attendance, and to achieve long-term knowledge retention, early or repeated reinforcement

may be necessary.[16,21] Attendance at such refresher courses is crucial as about two-third of these students were aware that their knowledge of radiation protection was inadequate.

Conclusion

In conclusion, the dental students in this study did have good knowledge on radiation biology but show relatively poorer knowledge on radiation protection. Despite their poor radiation protection knowledge their practice was better. We recommend that the curriculum of dental school be expanded further to provide better exposure to radiation protection and its practice, so that these students on graduation will be well grounded with the principle governing dentalradiography, its justification, quality control practice and the correct practise of the ALARA concept.

Corresponding Author

Hemavathy.O.R,

Reader, Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai -600077, Tamil Nadu, India.

Email: hemadocmd@gmail.com,

Mobile Number: 9626033600

References

1. Wenzel A, Kirkevang L. Students' attitudes to digital radiography and measurement accuracy of two digital systems in connection with root canal treatment. *Eur J Dent Educ 2004;8:167–171.*
2. Scarfe WC, Potter BJ, Farman AG. Effects of instruction on the knowledge, attitudes and beliefs of dental students towards digital radiography. *Dentomaxillofac Radiol 1996;25:103–108.*
3. Al-Rawi WT, Jacobs R, Hassan BA, Sanderink G, Scarfe WC. Evaluation of web-based instruction for anatomical interpretation in maxillofacial cone beam computed tomography. *Dentomaxillofac Radiol 2007;36:459–464.*
4. Freeman JP, Brand JW. Radiation doses of commonly used dental radiographic surveys. *Oral Surg Oral Med Oral Pathol 1994;77:285-9.*

5. Bury B. X-ray dose training: Are we exposed to enough. Clin Radiol 2004;59:926.
6. Royal College of Radiologists and National Radiological Protection Board. Patient dose reduction in diagnostic radiology. Document of the National Radiological Protection Board. 1990; 1 (2).
7. Prabhat MP, Sudhakar S, Praveen B, Ramaraju K. Knowledge, attitude and perception (KAP) of dental undergraduates and interns on radiographic protection — A questionnaire based cross-sectional study. *J Adv Oral Res 2011;2:45-50.*
8. Mubeen SM, Abbas MQ, Nisar N. Knowledge about ionising radiation among medical students. *J Ayub Med Coll Abbottabad 2008;20:118-21.*
9. Ilguy D, Ilguy M, Dincer S, Bayirli G. Survey of dental radiological practice in Turkey. *Dentomaxillofac Radiol 2005;34:222-7.*
10. Shiralkar S, Rennie A, Snow M, Galland RB, Lewis MH, GowerThomas K. Doctors' knowledge of radiation exposure: Questionnaire study. *BMJ 2003;327:371-2.*
11. Zewdneh D, Dellie ST, Ayele T. A study of knowledge and awareness of medical doctors towards radiation exposure risk at TikurAnbessa Specialized Referral and Teaching Hospital. Addis Ababa, Ethiopia. *J Pharm Biol Sci 2012;2:1-5. 12.*
12. Mojiri M, Moghimbeigi A. Awareness and attitude of radiographers towards radiation protection. *J Paramed Sci 2011;2:2-5.*
13. Smith NJ. Continuing education in radiation protection: Assessment of a one-day course. *Br Dent J 1991;170:186-8.*
14. Soye JA, Paterson A. A Survey of Awareness of Radiation Dose among Health Professionals in Northern Ireland.*Br J Radiol 2008;81:725-9.*
15. Streffer C. Radiation effects of exposure during prenatal development. *Radiologe 1995;35:141-7.*
16. Scarfe WC, Farman AG, Sukovic P. Clinical applications of cone-beam computed tomography in dental practice. *J Can Dent Assoc 2006;72:75–80.*
17. Scarfe WC, Farman AG. What is cone-beam CT and how does it work? *Dent Clin North Am 2008;52:707–730.*
18. White SC. Cone beam imaging in dentistry. *Health Phys 2008;95:628–637.*
19. Feldkamp LA, Davis LC, Kress JW. Practical cone-beam algorithm. *J Opt Soc Am 1994;1:612–619.*

20. TamizhPaavaiTha, Jayanth Kumar V. To Study Awareness about Radiation Protection among Dental students of Chennai- A questionnaire based study. Department of Oral Medicine and Radiology, SIMATS. *Int J Pharm Bio Sci 2017 Jan ; 8(1): B 542-551.*
21. Jembulingam, Geo Mani, Deepa Gurunadhan. Awareness and Attitude about Dental treatments among Middle school students of Chennai – A questionnaire based study. Department of Pedodontics, SIMATS. *Int J Pharm Bio Sci 2016 Oct ; 7(4): (B) 637 – 64.*

Knowledge, Attitude and Perception of Dental Students towards Black Board versus Smart BoardTeaching- A Survey

Sri Varsha.L [1], Thenmozhi M S [2], Asha Ramesh [3],
Graduate Student [1],Professor and Head [2],Department of Anatomy,
Senior Lecturer [3],Department of Periodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: For decades, black board has been the only way to teach school and college students in class but as years passed by, technology has been very efficient in bringing new electronic gadgets. Now with the new technologies, many colleges and schools have started using smart boards instead of blackboard. This study is being done to find out which is more efficient - black board or smart board, from the students of Saveetha dental college, Chennai.

Material and Methods: This research was done by interacting with 100 students from different batches and analysing which was - either smart board or black board, was more efficient and user- friendly.

Results:61% of the students preferred smart board and 39% preferred black board. 59% of students felt that smart board was more user friendly. 31% of the students felt that smart board was more complex when compared with black board

Conclusion: From this research, it will be evident which system is more approachable to the students and thus, it can be implemented in their respective schools and colleges.

Key Words*: Smart board; Black board; User-friendly; Efficiency; Animations*

Introduction

From earlier days, blackboard has been used in schools and colleges for teaching purposes.A blackboard or chalkboard is a reusable writing surface on which text or drawings are made with sticks of calcium sulphate or calcium carbonate, known, when used for this purpose, as chalk. Blackboards are manufactured by thin sheets of slate stones which is of black or dark colour. These blackboard are made in a way that these thin sheets are smooth as well.A blackboard can simply be a piece of board painted with dark paint (usually black or dark green). The marks made by the chalk can be easily cleaned off by using a wet cloth, a sponge or a duster which is made up of a blackboard eraser containing a wooden block covered by a pad to hold with hand. However, chalk marks made on some types of wet blackboard can be difficult to remove.

The smart board is starting to be used as a replacement for blackboards in many schools and colleges.The Smart Board interactive whiteboard operates as part of a system that includes the interactive whiteboard, a computer, a projector and white boarding software - either Smart Notebook collaborative learning software for education, or Smart Meeting Pro software for business. The components are connected wirelessly or via USB or serial cables. A projector connected to the computer displays the desktop image on the interactive whiteboard [1]. In this study, a questionnaire is prepared asking various questions regarding blackboard and smart board and the students are asked to fill it out to find out which is more approachable-either black board or smart board, and which system they prefer better.

There has been a constant shift in teaching methodologies for the students in various fields with the advent of technology. The innovative teaching has gained significant impetus as it is centred around better understanding in the student community and the teaching fraternity find it less cumbersome to the traditional methodologies. This study will be helpful in making changes accordingly in schools and colleges to make education and teaching more approachable and easier for students. The aim of this study is to compare the difference between black board and white board methodology of teaching and alsoto find out which system is better to approach to students so that it can be implemented in near future.

Materials and Methods

A set of questionnaires was prepared and given to 100 students of Saveetha dental college, Chennai. The questionnaire was on the topic smart board vs black board. The questionnaire

consisted of many questions whether the student preferred smart board or black board, if the student felt smart board was complex, if they understood black board drawings better or smart board animations better, if smart board has decreased the teacher's interaction with the students, which is not up tothe mark – black board or smart board, which is user-friendly: black board or smart board, if some information is cut out in smart board teaching. The questionnaire also listed questions asking about the advantages and disadvantages of both black board and smart board. From these questionnaires given to 100 students, the results can be analysed and the percentage will be more helpful for schools and colleges to understand which method of teaching reaches the student best and which should be set up in their respective schools and colleges.

Results

From the set of questionnaires answered by 100 students, 39% of students believed that black board was much better while the rest 61% of students preferred smart board as shown in the [Figure 1]. Just above half of the students who participated in the study (59%) felt that black board was more user-friendly since they have got more exposure to black board due to many years of usage as they have learned from it since their childhood times where the smart board is just at the beginning stage of emerging in schools and colleges. On the other hand, remaining 41% of students felt that smart board was more user-friendly than blackboard as illustrated in the [Figure 2]. 31% of students felt that the smart board technique was more complex and confusing to adapt whereas the rest 69% believed that it was much simple, convenient and is easy to access as illustrated in the [Figure 3].

On questioning the participants regarding the establishment of both methods of education, the black or white board system in accordance to their level of satisfaction or expectation, 35% of students admitted that they felt black board was not quite up to their expectations, while 45% of students felt that smart board was not up to their expectations. Whereas the rest 20% of students had a dilemma as they had little idea in distinguishing whether both the systems were good or not which is depicted in [Figure 4]. Now for the question about the use of colours while teaching, Seventy Seven out of hundred participants preferred colourful teaching while nineteenparticipants preferred conventional black and white teaching. The remaining four participants found comfort in both the methods.

Then the students were as asked about their preference of diagrams or animations for black board and white board respectively. 27% of students responded with their ability to understand with black board diagrams was better. On the other hand, 61% of students believed smart board animations were way more attractive and easier to remember when compared to black board diagrams. The rest 12% of students believed that both were helpful.

Discussion

This study was conducted among 100 dental students in order to assess the different teaching methodologies employing black board and smart board. It was conducted in the form of survey for evaluating the general pulse among the dental students towards the different teaching methods utilizing black board and smart board. Certain questions were included to gather knowledge about smart board teaching its merits and demerits. One of them is the information obtained by the students through smart board teaching. Regarding the amount of information delivered through smart board teaching, 46% of students believed that manyinformationis cut incase of smart board teaching whereas the rest 54% believed that enough information was provided in smart board and there were no cuts. A question was raised about the interactions among the teachers and students in classrooms during smartboard teaching. Total of 47 participants felt that there was less interaction between the teacher and students with smart boards teaching and rest 53 participants believed that there is enough interaction between the teacher and students using smart board similar to that of conventional method of teaching.

For the questions regarding the merits and demerits of both the system blackboard and white board, students responded about their views regarding both the system respectively. Considering the advantages of black board, most of the students said that it was user friendly and does not cost much for its set up and that black board was eco-friendlier. Whereas regarding its disadvantage, the students felt that it was more time consuming and it was lot of work to write on board and that it did not have colour images or animations. On the other hand, about the whiteboard, the students said that it can incorporate images and videos and can be viewed like it is real. When asked about the disadvantages of smart board, many said that it caused stress and strain to eyes and that it caused lot ofpower consumption and is complex to handle.

Dori and Kurtz et al checked to what degree the use of technological means contributes to understanding the study material and to students' motivation. Their research findings indicate that most students report that learning in a technological environment boosts their motivation and enhance their learning experience. The major contribution of technology as perceived by students is in increasing access to a variety of tools that contribute to understanding the study material, organization of the information, efficacy in carrying out assignments, and to development of knowledge [2].

The functions of smart board includedtouch feature where the mouse requires the touch to the surface of smart board or a screen. Apart from using the mouse, users are used to open and close documents with Windows applications, to be clicked web links, and also objects or pictures can be moved by finger swiping on the board [3, 4]. The feature of writing and deletion in smart board images and text to help existing users to write, draw and delete. Written text and images can be erased with a magnetic erase. It works like a hand eraser. In addition, the intelligent keyboard may be brought up on the board when needed [5, 6]. There is also drag and download feature that is suitable for any course that requires drawing, such as mathematics or engineering. These techniques are achieved by moving the stylus [7, 8].

One of the advantage of using smartboard technology is that users can record anyapplication seamlessly onto the board. These files which are recorded can be played again if required also on any computer. The files which are recorded can be played using Windows Media Player. Usersare able to save speeches and even their own voices, and areable to listen to them again. In addition, video files, whichwould play on all types of computers, can be created by users [9]. This creates ample time for the students to listen to the videos and also revise in the process of learning. This aids in student-centred

Many researches have been carried out to find out the use of smartboard to extend its limits. These researches have proven that educational advantage of use of smartboard for the students with special needs, it states that those types of students gain an opportunity study and obtain knowledge in a differentiated way which enables them to learn in a multi-sensory approach. This is a huge advantage to the students, particularly to those who have learning disability. The use of smartboard changes their disability to potentially able through increasing their attention, motivation to improve their learning process and also helps them to engage in the classroom activity [10].

Whiteboard has many merits and demerits. The merits are to keep students engaged, the teachers can create lessons which has incorporated videos, moving diagrams, animations and certain contents from online which helps in explaining difficult matters to students. This is possible by the use of interactive whiteboard. Whiteboard has a capability known as multimediawhere a teacher can explain an information in many possible different ways. This makes the lecture more interesting a lot more as it involves student participation [11]. For instance, if a teacher is teaching a lesson on some country like India, they can show the maps through Google Earth, making PowerPoints,regarding the country's population and certain histories and also an education video from YouTube regarding the wildlife present in India [12,13]. This makes the student to involuntary participate in topic discussion. As mentioned earlier, the students can participate in the classroom activity by writing on them, using certain objects as a part of activity session, playing games related to topic, or by voting an answer to the question by using handheld devices. These activities makes the students engaged and interested during lecture hours [14]. These boards helps teachers to show the students who had made projects by using PowerPoint software in order to motivate the student to participate and involve in learning process. Showcasing their projects helps the student enjoy their work as they take it as an appreciation when their work is displayed on a big screen in front of other classmates.

Regardless of the pros of whiteboards, their cost becomes a major concern. These boards are not cheap and there is an idea is that paying salary will the do the job of teaching rather than spending it on these boards [15]. Use of whiteboard depends upon the teacher's adaptation and use. Certain teachers like to stick to the traditional way of teaching using black boards preferably than adapting to the technology advancement. This makes the use of white board a major concern as the school may lose experienced teacher who has been in teaching conventional method due to sudden change in teaching methodology [16]. And also use of whiteboard requires some area of expertise in operating them to provide some knowledge to the students.

At last while teaching, the teachers does not like the students being distracted from listening to lecture for the sake of notes or important points of the topic [17]. Hence, the key points of the topic can be prepared in advance by the teacher by means of a PowerPointslide or other methods like chart, tables, maps etc., as it allows the students to pay attention to the lecture

by following the teacher and those points can be noted after the class hours during leisure time [18, 19]. This can be used as an alternative strategy for making the students actively participate in the learning process but cannot be used as a substitute for not attending the class lecture [20].

From the above data, it is evident that the students prefer smart board over black board. But both had equal advantages and disadvantages. A general consensus can be obtained from this study as it gauged the pulse of the students towards the different teaching methodologies whereas, a conclusion cannot be made because it is a survey with limited samples. It can be observed that while the smart board has lot of power consumption and is complex to use, black board consumes a lot of time and it cannot incorporate animations and colourful images. But when we look at the bright side, smart board has the ability to bring many images and animations virtually that is more helpful in understanding concepts. When it comes to black board, it is eco-friendlier and it helps the students to understand the diagrams better as the teachers will draw it in front of the students and in that way, the students will learn the technique to draw the diagram. So, if the institution is planning on setting up smart boards, the students will look forward for learning as the learning session will better with images and animations or if the institution is planning on setting up black boards, we can save lot of electricity and it will also be easy for the students to understand.

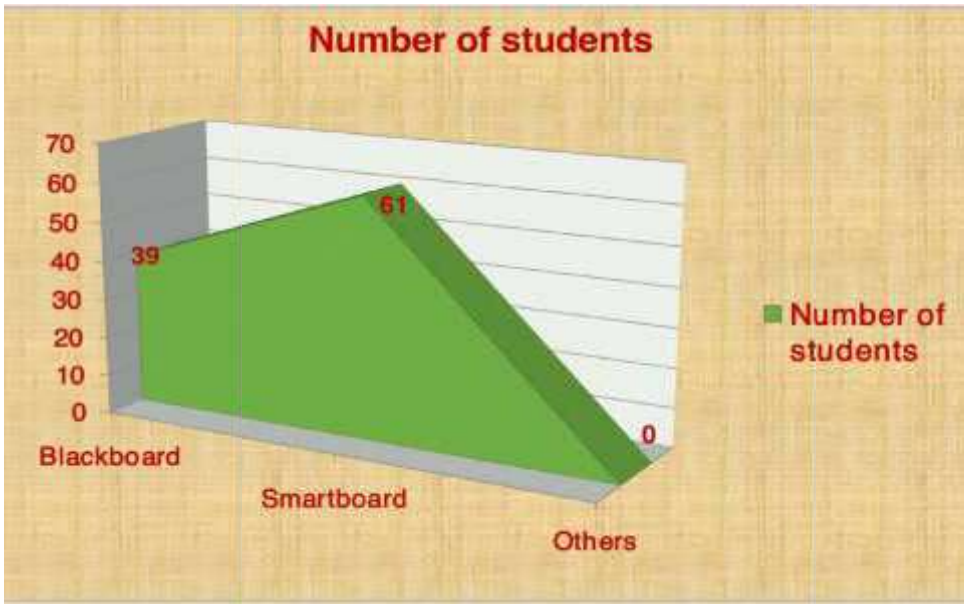


Figure 1: Percentage of students' preference for black board or smart board

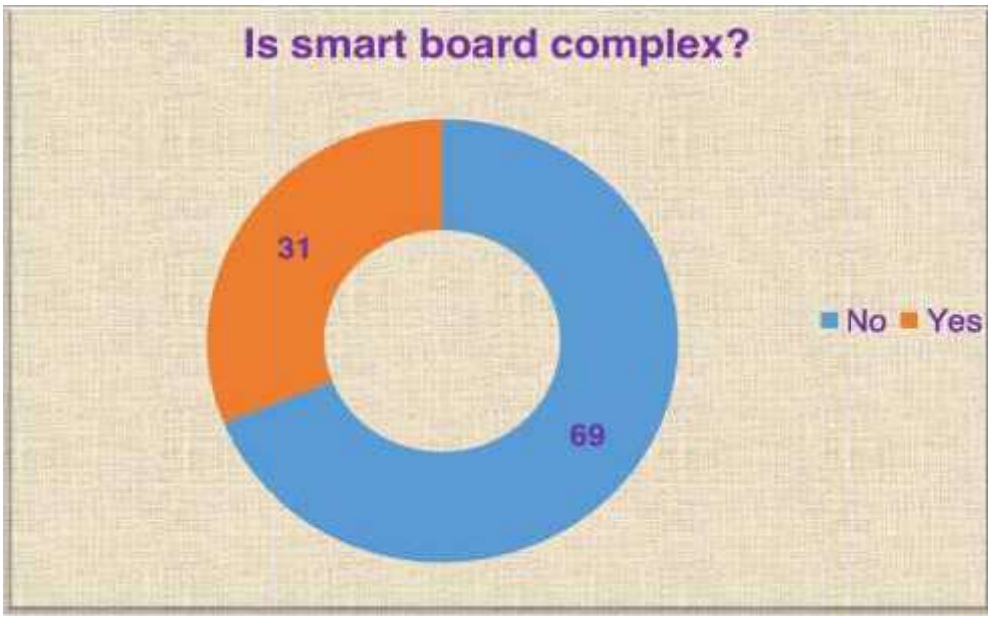


Figure 2: Percentage of students assessing the complexity of smart board

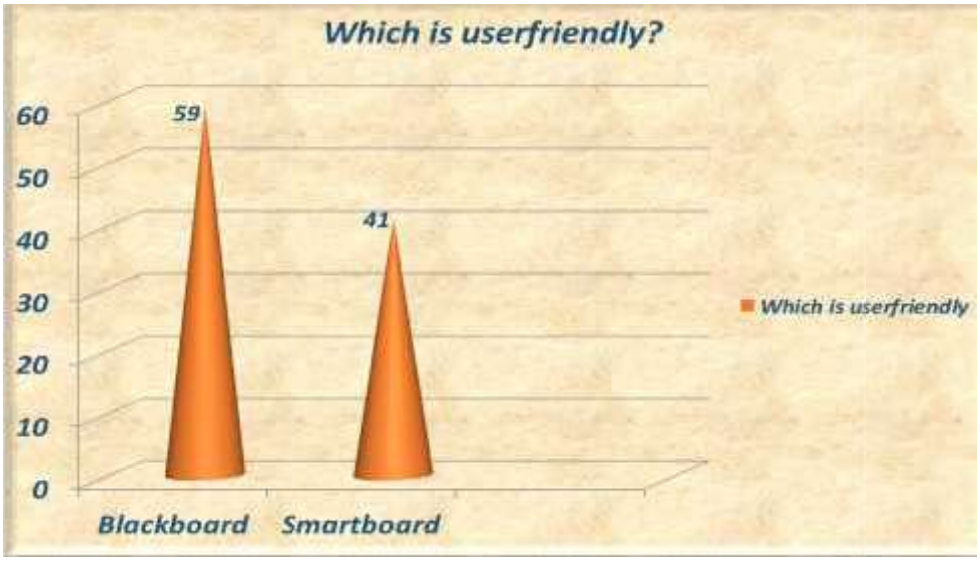


Figure 3: User-friendliness assessment of black board and smart board

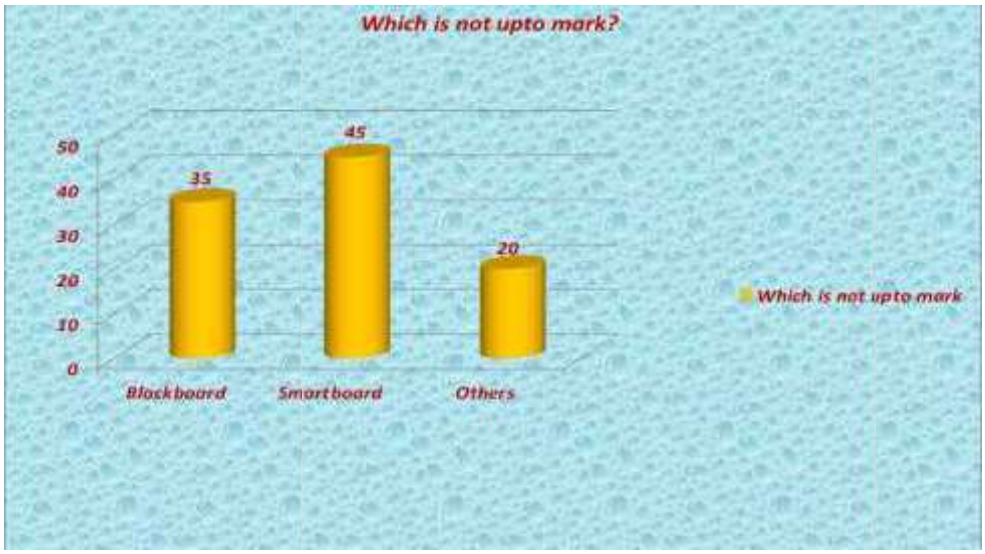


Figure 4: Percentage of student satisfaction using black board and smart board

Conclusion

Teachers play a major role as they influence the students towards the path of acquiring knowledge. Hence, a teacher should be aware in order to deliver the correct information through a technology so that it does not misguide the students. They should be comfortable and left to their own choice on selecting the method of teaching. If doubt arises, setting up both black board and smart board would be the finest option were the teaching can be done in smart boards while the diagrams can be taught using black board. By this way, teaching will be more approachable and efficient to the students.

Corresponding Author

Asha Ramesh

Senior Lecturer,Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India

Phone Number: +91-9841722113

E-mail id: doc.ash4587@gmail.com

References

1. Brenner S. Law in an era of smart technology. Oxford University Press; 2007 Dec 31.
2. Davidovitch N, Yavich R. The Effect of Smart Boards on the Cognition and Motivation of Students. Higher Education Studies. 2017 Feb 2;7(1):60.
3. Meral ŞE, Ahmet AĞ. The effects of using an interactive white board in teaching English on the achievement of primary school students. HasanÂliYücelEğitimFakültesiDergisi. 2014;11(2):29-40.
4. Tekelioğlu S, Sürücü M, Uğur B, Sönmez A, Ok M, Eren F. Smart Board (AkıllıTahta)'un EğitimeEntegrasyonuSunumRaporu. In9th International Educational Technology Conference 2009.
5. Morgan H. Teaching with the interactive whiteboard: An engaging way to provide instruction. Focus on Elementary. 2010;22(3):3-7.
6. Weimer M. The influence of technology such as a SMART Board interactive whiteboard on student motivation in the classroom. Retrieved October 21, 2007.
7. Smith HJ, Higgins S, Wall K, Miller J. Interactive whiteboards: boon or bandwagon? A critical review of the literature. Journal of Computer Assisted Learning. 2005 Apr 1;21(2):91-101.
8. Kratcoski A, Bates C, Hopkins A. Using SMART Boards to enhance student learning. Journal of the Research Center for Educational Technology. 2009 Sep 11;3(2):47-9.
9. Bell MA. Teacher feature: Why use an interactive whiteboard? A baker'sa dozen reasons! Teachers. net Gazette, 3 (1). Retrieved November 22, 2009.
10. Handler M. An evaluation of the effectiveness of smart board technology by evaluating the students' ability of completing their work with focus on students with disabilities.
11. Krippel G, McKee AJ, Moody J. Multimedia Use in Higher Education: Promises and Pitfalls. Journal of instructional Pedagogies. 2010 Mar;2.
12. Collins J. Education techniques for lifelong learning: giving a PowerPoint presentation: the art of communicating effectively. Radiographics. 2004 Jul;24(4):1185-92.
13. Jones AM. The use and abuse of PowerPoint in Teaching and Learning in the Life Sciences: A Personal Overview. Bioscience Education. 2003 Nov 1;2(1):1-3.
14. Craig RJ, Amernic JH. PowerPoint presentation technology and the dynamics of teaching. Innovative Higher Education. 2006 Oct 1;31(3):147-60.
15. Nouri H, Shahid A. The effects of PowerPoint lecture notes on student performance and attitudes. The Accounting Educators' Journal. 2008 Dec 30;18.
16. Romiszowski AJ. The Selection and Use of Teaching Aids. 1968

17. De Groote SL, Doranski M. The use of personal digital assistants in the health sciences: results of a survey. Journal of the Medical Library Association. 2004 Jul;92(3):341.
18. Devitt P, Palmer E. Computers in medical education 1: evaluation of a problem-orientated learning package. ANZ Journal of Surgery. 1998 Apr 1;68(4):284-7.
19. Krishna Mohan S. Does The Smart Board Improve Skills Of The First Year Medical Students In Learning Biochemistry?. INTI Journal: Special Issue on Teaching and Learning. 2009:152-60.
20. McLachlan JC, Patten D. Anatomy teaching: ghosts of the past, present and future. Medical education. 2006 Mar 1;40(3):243-53.

Knowledge, Attitude and Practice Regarding the Use of Precision Attachments in Fixed Partial Denture among the Dental Students – A Pilot Survey

R.Sharmila [1], Dhanraj[2],M. S. Nivedhitha[3],
Graduate Student[1], Senior Lecturer[2], Department of Prosthodontics,Professor and Head[3],
Department of Conservative Dentistry and Endodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Precision attachments are small interlocking devices to connect prosthesis and abutments that offer a variety of solutions to the challenge of balance between functional stability and cosmetic appeal. Precision attachments have wide applications, used in fixedremovable bridge, removable partial dentures, over dentures, implant retained over dentures, and maxillofacial prosthesis.

Materials and Methods: A closed ended questionnaire comprising 10 questions were prepared.

A total of 100 dental students were asked to complete the questionnaire. The completed questionnaire forms were collected personally. The data from the questionnaires were analyzed statistically.

Results: Out of 100 dental students,74% of the respondence were aware of precision attachment in FPD. 72% felt precision attachment improve chewing efficiency.63% expressed precision attachment is more expensive. 73% felt precision attachments offers better retention. 38% felt it repairs and modifications were easy with precision attachment and 71% opinioned precision attachments preserved the underlying hard and soft tissues.

Conclusion:Precision attachments present a challenge in the technical skill. A thorough understanding of the biomechanics of maxilla mandibular function, different attachments and knowledge, attitude and practiceis essential in treating a case of precision attachment in FPD.

Key Words:*Precision attachment, Fixed partial denture, Attitude, Implant and Over dentures.*

Introduction

A precision attachment is a precision-machined male and female (key and lock) housing that connects a removable partial denture to fixed bridgework. The female part of the attachment is soldered to fixed crowns , the male part is attached to the partial denture. The male is machined by the manufacturer to fit the female with such precision that any male out of the box will fit any female with an exact degree of accuracy. All of the precision attachments in a partial denture are positioned so that they are exactly parallel to each other.

Precision attachments in fixed partial dentures are the restorationswhere fixed restorations are contraindicated. There are many different kinds of attachments that will avoid the need for unsightly clasps, but only one type of precision attachment can claim has the greatest documentation when used according to basic principles of engineering. It was invented in 1906. When used properly, this type of precision attachment case will promote periodontal health and longevity of the abutments, prevent bone resorption of the ridges under the partial denture, and confer the highest degree of patient comfort.

An approach to eliminate the display of the clasp assembly with removable partial dentures is to consider the use of precisionor semi precision attachments. Precision attachments are generally poorly understood and overused in the profession.

Attachments have been classified as intracoronal or extracoronal and resilient or nonresilient, and each type of attachment has significant inherent associated deficiencies. With intracoronal attachments, room must be created for the attachment within the crown. This must be recognized at the time of tooth preparation and often results in removal of a considerable amount of sound tooth structure to accommodate the attachment. This loss of sound tooth structure represents a significant intervention into the structural integrity of the abutment tooth and exacts a biologic price that is often paid at a later date.

Extracoronal attachments by definition result in alteration ofthe physiologic crown contour of the abutment tooth and frequently establish a clinical situation in which it is extremely

difficult if not impossible for patients to maintain adequate oral hygiene beneath the attachment. This, again, usually exacts a biologic price, inthe form of periodontal disease or recurrent caries or both.

There are a large number of extra- coronal attachments available, and they vary considerably in their design and size. In general, it is the opinion of the authors that extra coronal attachments should be avoided with natural abutment teeth. If an extracoronal attachment is being considered for use, it is suggested that one of minimal size be chosen, to minimize the effect on physiologic crown contour, and that the patient be educated and encouraged to practice meticulous oral hygiene, to prevent the development of pathology. In general, resilient attachments are undesirable because they do not necessarily work in the manner in which they are intended, are usuallythe form of periodontal disease or recurrent caries or both.

There are a large number of extra- coronal attachments available, and they vary considerably in their design and size. In general, it is the opinion of the authors that extra- coronal attachments should be avoided with natural abutment teeth. If an extracoronal attachment is being considered for use, it is suggested that one of minimal size be chosen, to minimize the effect on physiologic crown contour, and that the patient be educated and encouraged to practice meticulous oral hygiene, to prevent the development of pathology.

The success of precision attachmentsin fixed partial dentures depends on creating an ideal architecture for the mouth. All the teeth that support bridgework are prepared with full shoulder preparations (flat ledges) in three-dimensions. In other words, the prepared teeth relate to each other, the gingiva and the underlying bone. Precision attachments must be used in a precise manner in order to maintain a high percentage of longevity. Great care must be taken to ensure precision at every step; and steps cannot be skipped or the final result will be compromised.

The entire chain can only be as strong as the weakest link. Precision attachment cases must fit with precision—the abutments must be stable and the frameworks must fit properly without rocking.Aim of the study is to assess the knowledge, attitude and practice regarding the use of precision attachments in fixed partial denture among the dental students.

Materials and Methods

The closed ended questionnaire approach was used to assess the Knowledge, attitude and practice regarding the use of precision attachments in fixed partial denture amongthe dental studentsin Dental college and hospital, Chennai. The questionnaire comprised 10 questions. A total of 100 dental students were asked to complete the questionnaire.(FIG – 1) The completed questionnaire forms were collected personally. The data from the questionnaires were analyzed statistically.

Results

Table 1: Responses to the questionnairein percentage (%)

QUESTIONS	YES(%)	NO(%)
1) Are you aware about the use of precision attachments in FPD ?	74% (74)	26% (26)
2) Do you think that the usage of precision attachments offers better chewing efficiency?	72% (72)	28% (28)
3) Do you think precision attachments are more expensive ?	63% (63)	37% (37)
4) Do you think that the caries occurrence rate is less with the use of precision attachments ?	58% (58)	42% (42)
5) Do you think precision attachments offers better retention ?	73% (73)	27% (27)

6) Do you think precision attachments provides better esthetics ?	74% (74)	26% (26)
7) Do you think the use of precision attachments offers a greater life span in FPD ?	41% (41)	59% (59)
8) Do you think precision attachments are easy to repair in FPD ?	38% (38)	62% (62)
9) Do you think the wear resistance is improved with the use of precision attachments ?	55% (55)	45% (45)
10) Do you think precision attachments preserve the hard and soft tissue ?	71% (71)	29% (29)

Discussion

In this study, the closed ended questionnaire approach was used to assess the knowledge, attitude and practice regarding the use of precision attachments in fixed partial denture among the undergraduate students in saveetha dental college and hospital, chennai. The questionnaire comprised 10 questions. A total of 100 dental students were asked to complete the questionnaire. The completed questionnaire forms were collected personally.

In which 74% of them were aware about the use of precision attachments in FPD and 26% were not aware about the use of precision attachments in FPD. 72% think that the usage of precision attachments offers better chewing efficiency and 38% don't think that the usage of precision attachments offers better chewing efficiency. 63% think precision attachments are more expensive and 37% don't think precision attachments are more expensive. 58% think that the caries occurrence rate is less with the use of precision attachments and 42% don't think that the caries occurrence rate is less with the use of precision attachments. 73% think precision attachments offers better retention.

27% don't think precision attachments offers better retention. 74% think precision attachments provides better esthetics and 26% don't think precision attachments provides better esthetics. 41% think the use of precision attachments offers a greater life span in FPD and 59% don't think the use of precision attachments offers a greater life span in FPD. 38% think precision attachments are easy to repair in FPD and 62% don't think precision attachments are easy to repair in FPD. 55% think the wear resistance is improved with the use of precision attachments and 45% don't think the wear resistance is improved with the use of precision attachments. 71% think precision attachments preserve the hard and soft tissue and 29% don't think precision attachments preserve the hard and soft tissue.(TABLE – 1)

Precision filled and semi precision attachments, improved impression materials, improved techniques and designs which would eventually attain a comprehensive treatment. In case of partially edentulous mouth, retention provided by the usage of precision attachments which may be related to comfort, satisfaction, chewing ability, as well as adequate distribution of occlusal loads to, and preservation of abutment teeth in patients with removable partial dentures. Retentive ability increases significantly over time in the metal- alloy precision attachments group.

There are many advantages of precision attachment. They are uncomplicated, precision-fitting, inter- changeable attachments. which are popular for RPD retention. Many technicians routinely stock these attachments and are familiar with their application. The labor cost is less than for laboratory made attachments (semi- precision attachment) because theyare prefabricated, and the retention is better. In addition, are placement that will fit exactly can be purchased andsoldered in place if breakage of an attachment occurs.Other attachments of similar design.such as the Stern Type 7 and the Stern G/A attachments.The technician must

not alter the configuration because the attachments are precision milled, therefore,the components of the attachment should not be air abraded or polished.

The milled guiding planes serve two purposes.They augment,the retention obtained from the attachments to help prevent undesirable attachment wear, and they guide the prosthesis during insertion and removal to facilitate handling. The combination of these guidingplanes and the parallel side attachments ensure rigidly fixed prosthesis with predictable retention that can also be easily removed and reinserted. Thus, the patient can clean the restoration daily although the prosthesis feels thesame as an FPD when seated in the mouth.

This fixed detachable prosthesis is versatile and can solve the problem of patient access for oral hygiene in most clinical situations.While the technique is especially usefulwhere there is extensive loss of residual bone and the softtissue must be replaced with the prosthesis, this attachment retained prosthesis analso beused when the implants were inadvertently inserted in lingo -version and the prosthesis is must be designed with facially placed artificial teeth. The patient may experience difficultiescleaning thetissue surface of a fixed prosthesis supported by lingually placed implants; however, this detachable design is a viable option to compensate for the unfavourable implant positions.

Charwaki HG et al(1) evaluated and quantified changes in retention ability and weight change over time and thus reported Metal-alloy and plastic inserts precision attachments preserve supporting teeth and alveolar bone ridges when associated with at least two splinted abutments.

Holst et al (2) cited as it is difficult to evaluate precision attachments' effects on treatment longevity based solely on *in vitro* results, since other factors such as continuous ridge resorption, changes in saliva flow and composition, and occlusal considerations may affect its long-term success. However, if an attachment removable partial denture is the treatment of choice because of esthetics, abutment alignment, or the need for greater cross-arch bracing, it must be used with a thorough knowledge and understanding of prosthodontic principles and attachment use, as well as an awareness of the intricacies and special problems associated with attachments.

The attachment is essentially a tube with a sleeve joint that provides bulk to the connector. For clinical cases where the extra coronal cast clasp displays the retention arm of a cast partial denture, the precision attachment of such types provide an alternative option for patients to be treated with a fixed partial denture(3,4).

In addition to clasps used to prevent the dislodgment of RPDs from the rest position during functional movements,(5) devices such as adhesive attachments, crowns, and FPDs with intra- or extracoronal attachments, telescopes, root-caps, and/or pre- fabricated intraradicular retainers may also be used to retain these prostheses. (6-9)

Most of the studies have shown that attachment retained cast partial dentures gives better comfort, function, esthetics ,less adjustments, protect abutment teeth, easy to clean and can worn most of the time by the patient. Its use in fixed prostheses, over dentures and in implant therapy contributes to the success of prosthesis in terms of esthetics, comfort and function(10).

Edentulism leads to an acknowledged impairment of oral function with both, aesthetic and psychological changes. Depending upon the clinical need and demand, restoration of the lost structure can be achieved by using conventional methods. (11) A full-arch fixed prosthesis can be fabricated, if sufficient and properly situated abutments remain orsufficient number of implants can be placed. However, both extensive fixed dental prosthesis and implant-supported prosthesis can be financially burdensome to patients (12,13).Retainer selection for removable dental prosthesis mainly depends on the remaining tooth structure, the intra- and inter-maxillary relationships, aesthetics, and financial aspects(14,15).

Preiskel first reported the inventions of attachments in early 20^{th} century. To the late 20^{th}century ,with growing technology the attachment has been applied to the superstructure of implant.

Nishimura et al (16) reported that the least stress was observed when using a nonrigid connector, and a rigid connector in particular situations caused slightly higher stresses in the supporting structure. The authors thought that limiting the stress would help protect the alveolar ridge from excessive load; that under certain occlusal forces, the resilience of the

attachment could be adjusted to distribute the force properly between the abutment and alveolar ridge.

Chou et al. (17) reported that a rigid-precision attachment produced great stresses and caused more movement of abutment teeth than did clasps, and they pointed out the risk of rigid-design dentures. However, the high stress, especially in apical areas, does not always cause damage to the tooth, because a tooth can withstand greater forces along the long axis than horizontal forces.

Kratochvil et al. (1981), Pezzoli et al. (1986) and Igarashi et al. (1999).(18,19,20) Reported that the frequency of relining of rigid-design denture was less. There was no significant difference between stresses on the denture bases of attachment dentures and telescopic dentures.

Feingold et al. (1988) (21) reported that the base movement of a rigid attachment was less than that of a resilient attachment. Saito et al. (1998) (22) investigated the vertical movements of modified clasp- and telescopic crown-retained denture, and they found that the displacement of the denture base decreased with an increase in the rigidity of the connection of the retainer to abutment teeth. However, the amount of displacement was influenced by the fulcrum line of rotation of the RPD.

The success of prosthesis depends on careful treatment planning and attention to the prosthodontic problems; the mechanical ingenuity of the attachment is important, but must take second place. Precision attachments present a challenge in the technical skill. A thorough understanding of the biomechanics of maxilla mandibular function, different attachments and knowledge of material science is essential in treating a case of precision attachment. Unfortunately, most often precision attachments are chosen from descriptions in manufacturer’s catalogues which leads to failure of precision attachment cases. Precision attachments serve the function of retention, stress distribution and aesthetics successfully provided the case is planned based on sound biological and technical grounds and proper care is rendered by the dentist and the patient during the maintenance phase. Hence precision

attachments should be employed for use in the fabrication of fixed prosthesis in the dental practice.

Conclusion

This study inferred the knowledge, awareness and practice regarding precision attachments in fixed prosthodontics is inadequate among the participants..This study alsohighlights the need for further awareness about the clinical applications of precision attachments in fixed – removable partial dentures among the undergraduate dental students in saveetha dental college, chennai.

Corresponding Author

M. S. Nivedhitha,

Professor and Head,Department of Conservative Dentistry and Endodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai- 600077,Tamil Nadu, India.

Telephone Number- +91 9840912367

E-mail Id- nivea16@hotmail.com

Reference

1. Charkawi HG, Wakad MT. Effect of splinting on load distribution of extracoronal attachment with distal extension prosthesis in Vitro. *J Prosthet Dent* 1996; 76(3):315-320.
2. Holst S, Blatz MB, Eitner S, Wichmann M. In Vitro wear of different material combinations of intracoronal precision attachments. *Int J Prosthodont* 2006; 19(4):330- 332.
3. Preiskel HW. Precision attachment: Uses and abuses. *J. Prosthet. Dent*., 1973; 30: 491-492.
4. XxRushford CB. A technique for precision removable partial denture construction. *J. Prosthet. Dent.*, 1974; 31: 377-383.
5. Leupold RJ, Faraone KL: Etched castings as an adjunct to mouth preparation for removable partial dentures. *J Prosthet Dent* 1985;53:655-658

6. Hirschman BA: Extracoronal precision attachments for removable partial dentures. *J Mich Dent Assoc* 2000;82:30-36
7. Owall B: Precision attachment retained removable partial dentures: 1. Technical long-term study. *Int J Prosthodont* 1991;4:249-257
8. Wang HY, Zhang YM, Yao D, Chen JH. Effects of rigid and nonrigid extracoronal attachments on supporting tissues in extension base partial removable dental prostheses: a nonlinear finite element study. *The Journal of prosthetic dentistry*. 2011 May 31;105(5):338-46.
9. Zitzmann NU, Rohnerb U, Weiger R, Krastl G. When to choose which retention element to use for removable dental prostheses. *International Journal of Prosthodontics.* 2009 Mar 1;22(2).
10. Shakeel SK removable prosthesisusing extra coronal precision attachment: A case reportGMJ, *ASM* 2013; 2(S1):S126-S129.
11. Mh Siegfried, AlexanderS Mandibular two-implant telescopic overdentures- 10 year clinical and radiographical results. *Clin Oral Implant Res.* 2004;15:560–69.
12. MA Polly S, Brudvik James S. Managing the maxillary partially edentulous patient with extensive anterior tooth loss and advanced periodontal disease using a removable partial denture: A clinical report. *JPD*. 2008;100:259–63.
13. Zitzmann Nicola U, Rohner Urs, Weiger Roland, Krastl Gabriel. When to choose which retention element to use for removable dental prosthesis. *Int J Prosthodont.*
14. Zinner ID, Miller RD, Parker HM,Panno FV: Prefabricated metal intracoronal semiprecision attachments for removable partial dentures. *Int J Prosthodont* 1989;2:357-364
15. Burns DR, Ward JE: Review of attachments for removable partial denture design: 1. Classification and selection. *Int J Prosthodont* 1990;3:98-102
16. Nishimura RD, Ochiai KT, Caputo AA, Jeong CM. Photoelastic stress analysis of load transfer to implants and natural teeth comparing rigid and semirigid connectors. *J Prosthet Dent* 1999; 81:696-703.
17. Chou, T.M., Caputo, A.A., Moore, D.J. & Xiao, B. (1989) Photoelastic analysis and comparison of force-transmission characteristics of intracoronal attachments with clasp distal- extension removable partial dentures. *Journal of Prosthetic Dentistry*, 62, 313.

18. Kratochvil, F.J., Thompson, W.D. & Caputo, A.A. (1981) Photo- elastic analysis of stress patterns on teeth and bone with attachment retainers for removable partial dentures. *Journal of Prosthetic Dentistry*, 46, 21.
19. Pezzoli, M., Rossetto, M. &Calderale, P.M. (1986) Evaluation of load transmission by distal-extension removable partial den- tures by using reflection photoelasticity. *Journal of Prosthetic Dentistry*, 56, 329.
20. Igarashi, Y., Ogata, A., Kuroiwa, A. &Wang, H. (1999) Stress distribution and abutment tooth mobility of distal-extension removable partial dentures with different retainers: an in vivo study. *Journal of Oral Rehabilitation*, 26, 111.
21. Feingold, G.M., Grant, A.A. &Johnson, W. (1988) Abutment tooth and base movement with attachment retained removable partial dentures. *Journal of Dentistry*, 16, 264.
22. Saito, M., Miura, Y., Notani, K., Aizawa, T., Matsui, T. &Kawasaki, T. (1998) The influence of clasp structures on load distribution of abutment teeth and ridges and displacement of denture base. *The Journal of the Japan Prosthodontic Society*, 42,641.

FIG- 1 This questionnaire is to assess the knowledge, attitude and practice regarding the use of precision attachments in fixed partial denture among the dental students

Name- Age/sex-

1) Are you aware about the use of precision attachments in FPD ?
   a) Yes b) No

2) Do you think that the usage of precision attachments offers better chewing efficiency?
   a) Yes b) No

3) Do you think precision attachments are more expensive ?
   a) Yes b) No

4) Do you think that the caries occurrence rate is less with the use of precision attachments ?
   a) Yes b) No

5) Do you think precision attachments offers better retention ?
   a) Yes b) No

6) Do you think precision attachments provides better esthetics ?
   a) Yes b) No

7) Do you think the use of precision attachments offers a greater life span in FPD ?
   a) Yes b) No

8) Do you think precision attachments are easy to repair in FPD ?
   a) Yes b) No

9) Do you think the wear resistance is improved with the use of precision attachments ?
   a) Yes b) No

10) Do you think precision attachments preserve the hard and soft tissue ?
   a) Yes b) No

Measuring Orthognathic Surgery Satisfaction in Orthodontic Patients- A Questionnaire Survey

Nandhini.S[1], Saravana Pandiyan[2], Mahathi.N[3]

Graduate Student[1], Reader[2], Department of Orthodontics, Reader[3], Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: In orthodontic surgical treatment the motivation of the patient for seeking treatment and her/his expectations of the results should be considered carefully in treatment planning. Therefore the patient satisfaction will be improved. The objective of this study was to develop a reliable self-report measure of consumer satisfaction with orthodontic treatment, and to preliminarily assess its validity. The aim of this study was to assess patients who have undergone orthognathic surgery.

Material and Method: A patient centered questionnaire to assess the satisfaction of the patients undergoing orthognathic surgery was being prepared and given to 50 patients in Saveetha dental college and the results are being discussed.This questionnaire is easily and quickly applicable and economic, however, it should be kept in mind that it is subjective.

Results: 50 participants who attended the O.P of Department of orthodontics, Saveetha Dental college, were included in the study. When questions were asked about their experience in the department, 98% felt the treatment was hygienic, 96% felt the treatment was expensive 86% felt they were well informed. And only 62% had discomfort during the treatment. when asked about treatment outcomes, 97% had a smile satisfaction, 95% said they would recommend to friends. 88% felt they had experienced pain. Finally questions were asked about how it affects the quality of life of the patients.

Conclusion: In conclusion, orthognathic surgery causes significant improvement in patients' quality of life. This improvement was seen in emotional, oral function, psychological, and social domains of quality of life. The maximum influence was in emotional and the least in

functional aspect. Our study assessed quality of life in a limited period postoperatively and obviously there is a need for longitudinal studies in this area of healthcare services.

***Key Words**: Orthognathic surgery, satisfaction, smile, confidence, measurement.*

Introduction

Orthodontists, like other health care professionals, rely on patient cooperation for successful treatment outcomes, and an important factor in this success is patient's satisfaction with the delivery of care [1].Patient satisfaction after orthodontic treatment isinfluenced by a number of factors. To illustrate,psychological traits of individual patients affect theirsatisfaction with orthodontic treatment outcomes.Factors like gender, age, duration of treatment,compliance, and dentofacial improvement also seemto contribute to the level of satisfaction.Patientsatisfaction is important for ensuring patient adherencein orthodontic treatment. In previous studies, differentfactors have been explored and different instrumentshave been used to assess satisfaction after orthodontic treatment. Not surprisingly, investigations of patientsatisfaction after orthodontic treatment have shown awide range of satisfaction levels.Thismakescomparisons between studies difficult.Almost a decade ago, in a study in the Netherlands,the patient satisfaction questionnaire developed by Boset al.

The factors like doctor-patient relationship, situational aspects, dento-facial improvement, psychosocial improvement, anddental function, as well as a residual category, wereexplored. It was shown that the most important factorcontributing to patient satisfaction was the patient'ssatisfaction with the doctor-patient relationship.Gender was shown to be a significant predictor of thepatient's satisfaction with the doctor-patient relationshipand the situational aspects of the treatment.However, dental attitudes of patients have changedover time.Over the past decade, the number ofpatients seeking orthodontic treatment has increasedconsiderably with socioeconomic development andchanging opinions on appearance.[2,3,4]

Orthodontic tooth movement (OTM) is mainly a biological response towards mechanical force .Skeletal disfigurement of the face has a negative effect on many aspects of life. These include social interactions, opportunities, choice of profession, choice of partner, and personality characteristics. Subjects with malocclusion, particularly those in need of surgical

corrections, have a lower health-related quality-of-life and are more anxious .It therefore seems reasonable to offer orthognathic surgery as appropriate treatment to correct a disfigurement if it is subjectively perceived as a handicap, in part to improve the psychology of the patient. This leads to the consideration that patients' perception of the quality of orthognathic surgery is dependent upon several factors. These are not only aesthetics and function, but also psychological aspects. As a consequence, patients' preoperative expectations of orthognathic surgery and postoperative outcome could offer discrepancies if patients are not clearly informed of what is possible and what is not.However, without patient cooperation, few medical or dental therapies, including orthodontics, will achieve optimum results.It has been shown that a desire for orthodontic treatment, together with a sound understanding by the patient of the nature of the malocclusion, auger well for future compliance.Hence, compliance does not remain the sole responsibility of the patient.

Rather, orthodontists need to inform and instruct their patients to such a level as to ensure their full commitment. It is of concern that patients show a very low recall rate with regard to any risks associated with orthodontic treatment. Lack of communication between the orthodontist and the patient and insufficient information about orthodontics can lead to premature termination of the treatment. Orthodontists should therefore look at the way theyeducate patients, ensuring that full comprehension has been achieved. Measuring treatment satisfaction is a complex task.Patient satisfaction is higher when visible treatment outcome goals are met and when their expectancy with regard to psychosocial benefits is lower. The key to success is to discover the actions that will produce the most positive response from the patient. Orthodontists should strive to achieve the correct bite and an excellent smile, but they have not been truly successful if their patients have not also benefitted psychosocially.The fulfillment of patient expectations has become one of the main objectives of health care systems.[5,6]

Patients' expression of desires is important because health professionals often underestimate them for care. The clinician with an awareness of a patient's demands is better able to satisfy the patient's justified desires and to initiate frank discussion about those expectations that are unrealistic, leading to more productive clinical negotiations.Assessment of patients' expectations is central to understanding oral health needs, patient satisfaction with treatment, and ultimately the perceived overall quality of health systems.Mismatch between patient

desires and the service received is relatedto decreased satisfaction.Patient satisfaction is a multifaceteddimension; however, people may have a complexset of important and relevant beliefs.Interest in-patient satisfaction with various aspects of their healthcare has grown significantly for surgeons and orthodontists. The benefits provided by a combined orthodontic-surgical treatment,as well as the potentialrisks and negative side effects regarding this therapy.[7,8,9]

Orthodontists and oral maxillofacial surgeons shouldthe informed consent process and properlytemper patients' expectations by limiting false impressions of a "new face" after such complex treatment.Ithas been observed that patients tend to expect theirnew profile to fit more closely to socially acceptedpatterns than what should really be expected.Another major factor to be considered is that theperceived care and attention the orthodontist, surgeon,and staff provided to the patient increased the patient'sconfidence in the orthodontic/surgical treatment out-comes. However, perception of care is a broadcategory that was sometimes only assessed as qualityof care in the studies examined.The overall aim of orthodontic care should be good treatment results and satisfied patients but at a reasonable cost. To reach this goal it is important that the quality of care is continuously and systematically evaluated and documented by means of professional clinical assessmentsand patient questionnaires or interviews.Patients with inappropriately high expectations may be dissatisfied with the optimal care and those with inappropriately low expectations may be satisfied with deficient care.From a policy perspective, understanding patients' concerns and requests is important for the measurement of health care quality, the delivery of health services, and the costs of care.Correlations between satisfaction with dental and facial appearance and expectations of orthodontic treatment appear to be age but not gender related.[10,11]

The aim of this study was to assess patients.Orthodontists should recognize and respond to these needs, for as caring professionals they may be the patient's only source of positive reinforcement.

The main goals of orthodontic treatment are to correct aesthetic impairment, improve oral function, and help patients restore their socio psychological well-being A number of factors may influence patient satisfaction, including physical comfort, emotional support, patients expectations, and respect for patient preference.This research is being designed to measure the satisfaction of orthodontic patients undergoing orthodontic surgery.[12]

Materials and Methods

A patient-centered questionnaire to assess satisfaction of the patients undergoing orthognathic surgery was being prepared and given to 50 patients in Saveetha dental college and the results are being discussed.

Inclusion criteria -This questionnaire was distributed only to patients who have undergone orthognathic surgery.

This questionnaire is easily and quickly applicable and economic, however, it should be kept in mind that it is subjective because replies can be effected by patients' moods at the time of quastionnaire.The questionnaire is as follows:

Table 1: Questionnaire

1-Was the treatment hygienic and sanitary?
2- Do you feel like it's an expensive treatment?
3-Did the doctor keep you informed about the procedure?
4-Do you feel orthognathic surgery along with the orthodontic treatment takes a longer time?
5- Did you feel any discomfort in this long term treatment?
6-Did you have pain during the treatment?
7-Considering everything, do you feel the orthognathic treatment increases your self-confidence?
8-Do you feel treatment has improved your chewing ability?
9-Do you feel treatment has improved your speech quality?
10-Are you satisfied with the alignment of your teeth?
11-Are you satisfied with your final facial appearance?
12-Are you satisfied with your smile?
13-Are you satisfied with the overall results of the treatment?
14- Do you feel this orthodontic treatment along with orthognathic surgery is useful?
15- Would you recommend your family, relatives and friends to go through the same kind of treatment?

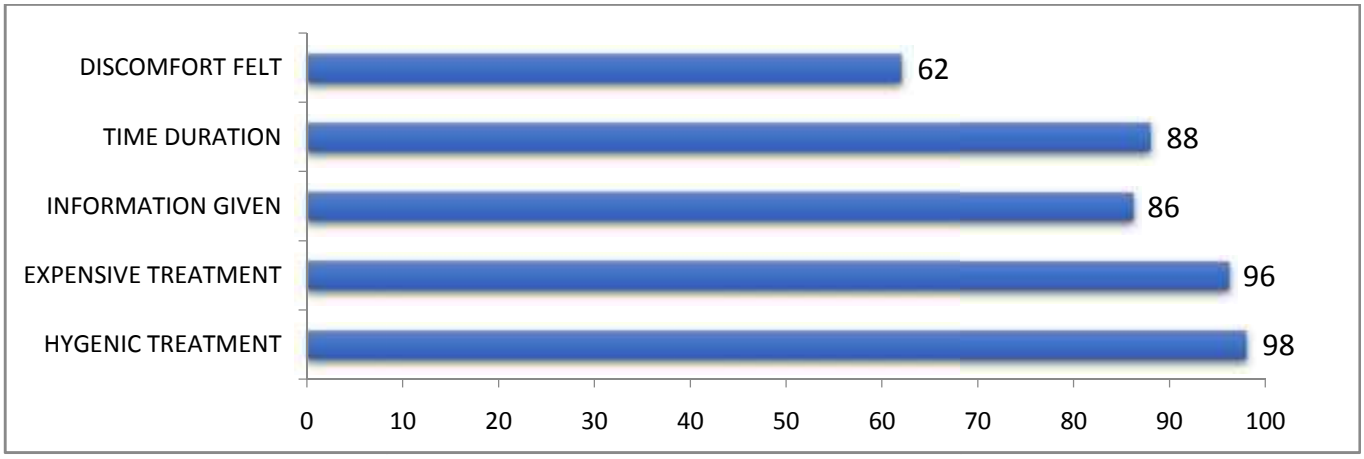
Results

50 participants who attended the O.P of Department of orthodontics, Saveetha Dental college, were included in the study. When questions were asked about their experience in the

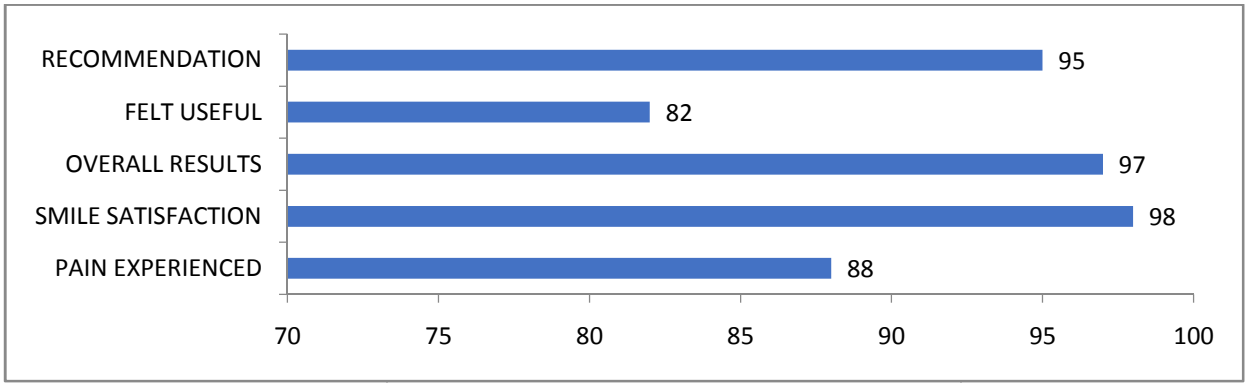
department, 98% felt the treatment was hygienic, 96% felt the treatment was expensive 86% felt they were well informed. And only 62% had discomfort during the treatment (Graph I).

when asked about treatment outcomes, 97% had a smile satisfaction, 95% said they would recommend to friends. 88% felt they had experienced pain (Graph II). Finally questions were asked about how it affects the quality of life of the patients, and the outcomes are listed in Graph III.

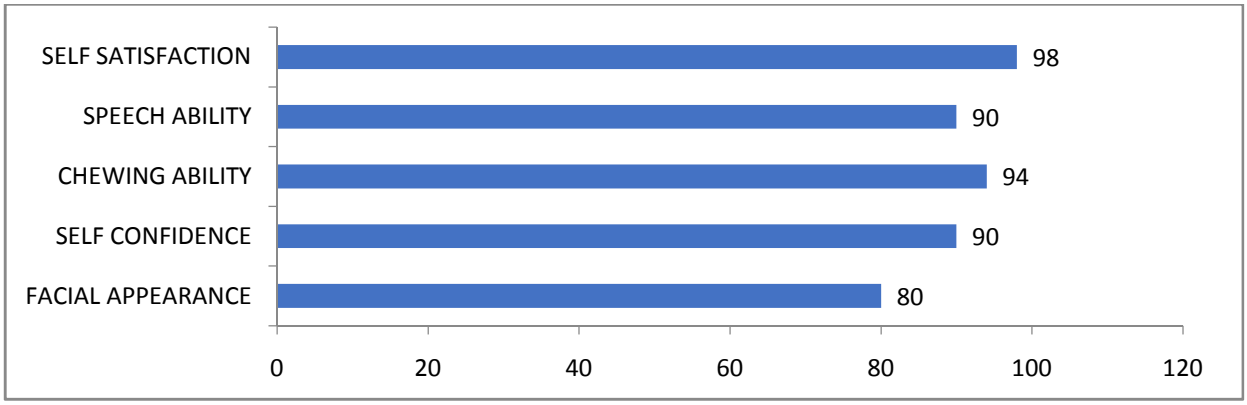
Graph 1: Factors experienced by patients regarding orthognathic treatment



Graph 2: Patients experience regarding treatment outcome



Graph 3: Improvement in patients quality of life post orthognathic therapy



Discussion

Patient satisfaction with orthodontic treatment hasno definitive outcome in the literature.This can beexplained by the different motivations and expectations of patients receiving orthodontic treatment. Moststudies report functional, esthetic, and social reasons as the main motives to seek orthodontic treatment.Satisfaction level may be affected by the severity of themalocclusion at the beginning of the treatment or bythe final result of the treatment. However, a recentstudy showed that patient satisfaction is not necessarily bound to the severity of malocclusion beforetreatment or the final result of the treatment.Thecurrent status of the dentition may be more relevant forpatients than the benefits obtained just after orthodontic treatment.Dentofacial deformities drastically affect patient's health related qualities of life. Especially unaesthetic appereances of soft tissue and skeletal architecture in class 3 deformities may cause psychological problems.[13,14]

Therefore, the most crucial step of preoperative planning in orthognathic surgery candidates is psychological assessment. Ideally, a psychiatrist or psychologist should undertake this assesment. If this cannot be provided, an orthodontist or surgeon should evaluate the patient carefully. Patient's self-perception of facial appearance can differ from physicians perception. Therefore, patient's subjective complaints and expectations should be analyzed carefully. [15]

Physicians should decide whether they can meet these expectations .Studies show that even if there are minor disparities in the results, patients are satisfied with the results of orthognathic surgeries. There are many factors that might cause postoperative dissatisfaction. Most of them are secondary to miscommunication of patient and physician rather than poor postoperative results or lack of surgical skills.Many measurement indexes are used for assessing the benefits of orthognathic surgery and usually questionnaires are preferred. The questionnaire was assessed for the satisfaction of the patients.50 Orthognathic patients

completed the questionnaire. And 98% of them feel that the treatment is hygienic and sanitary.So it is understood that the treatment is a better one. But 96% of them feel it is an expensive treatment.86% of the doctors kept the patients well informed about this procedure and 62% of them feels discomfort and major percentage of them that is nearly 80% feels that it is an expensive procedure.94% patients have reported that it improves their chewing ability and expect few everyone are happy about the smile and facial appearance. Nearly 98% of patients are satisfies with speech quality and the teeth alignment and nearly 96% of people are satisfies with this procedure.94% patients would like to recommend it to their friends, relatives and family.This study has several limitations. More patients with longer follow-up and a survey before the institution of orthodontics would be useful to better correlate the results with lasting benefits obtained through orthognathic surgery.

Conclusion

In conclusion, orthognathic surgery causes significant improvement in patients' quality of life. This improvement was seen in emotional, oral function, psychological, and social domains of quality of life. The maximum influence was in emotional and the least in functional aspect. Our study assessed quality of life in a limited period postoperatively and obviously there is a need for longitudinal studies in this area of healthcare services.This report focuses on patient satisfaction with orthognathic treatment as an outcome measure of the quality of the treatment. Based on the results, it is very well clear that almost 94% of the patients are satisfied with this treatment, inspite of the discomfort.There are many studies which proves the quality and success rate of the orthognathic treatment. Though it is expensive, the effectiveness of the treatment overcomes it. This survey reveals the existing scenario of orthognathic practice and highlights the importance of it.

Corresponding Author:

N.Mahathi,

Reader, Department of Oral Surgery and Maxillofacial Surgery,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email- nerallamahathi@gmail.com

Mobile Number: +91 9840958142

References

1. Kress G, Shulman JD. Consumer satisfaction with dental care: Where have we been, where are we going. J Dent Educ 1997;64:9-15.
2. Kress GC. Patient satisfaction with dental care. Dent Clin North Am 1998;32:791-802.
3. Newsome PRH, Wright GH. A review of patient satisfaction. Concepts of satisfaction. Br Dent J 1999; 186:161-165.
4. Croucher R, Robinson P, Zakrewska JM, Cooper H, Greenwood I. Satisfaction with care of patients attending a dedicated dental clinic.1987;64:9-17.
5. Shilpa.R, Self Perceived Orthodontic Treatment Need in Dental Students, Saveetha Dental College. Int. J. Pharm. Sci. Rev. Res.,2017;46(2): 39-44.
6. MuthuLaakshmiGanesh,Saravana Pandian. Acceleration of Tooth Movement during Orthodontic Treatment-A F, MuthuLaakshmi Ganesh et al /J. Pharm. Sci. & Res. 2017;Vol. 9(5)741-744.
7. Batmaraj,Umashankar. The effects of commonly used drugs on orthodontic tooth movement : a systematic review, Asian Journal Of Pharmaceutical And Clinical Research 2014; 1990;64:9-05.
8. Butters JM, Willis DO. A comparison of patient satisfaction among current and former dental school patients. J Dent Educ 2000;64:409-4.
9. Sitzia J, Wood N. Patient satisfaction: A review of issues and concepts.SocSci Med 1997;45:1829-1843.
10. .D.A.CampellReid,A.H.K.Price, Orth, RCS. Digital Deformities and Dental Malocclusion Due To Finger Sucking. Br. Journal of Plastic Surgery Volume 1984; 445-452.
11. Valeria Luzzi, MarinellaGuaragna, Gaetano Lerardo, MatteoSaccucci, GiulianaConsoli, Anna Rita Vestry, Antonella Polimeni. Malocclusions And Non-Nutritive Sucking Habits: A Preliminary Study.Int J Adult OrthodOrthognath Surg.2011;17:75–81.

12. Zhou YH, Hagg U, Rabie AB. Concerns and motivations of skeletal Class III patients receiving orthodontic-surgical correction. Int J Adult OrthodOrthognath Surg. 2001;16:7–17.
13. Shell TL, Woods MG. Perception of facial esthetics: a comparison of similar class II cases treated with attempted growth modification or later orthognathic surgery. Angle Orthod. 2003;73:365–7.
14. Gerzanic L, Jagsch R, Watzke IM. Psychologic implications of orthognathic surgery in patients with skeletal Class II or Class III malocclusion. Int J Adult OrthodOrthognath Surg. 2002;17:75–81.
15. Lazaridou-Terzoudi T, Kiyak HÁ, Moore R, Athanasiou AE, Melsen B. Long-term assessment of psychological outcomes of orthognathic surgery. J Oral Maxillofac Surg. 2003;61:545–52.
16. Motegi E, Hatch JP, Rugh JD, Yamaguchi. Health-related quality of life and psychosocial function 5 years after orthognathic surgery. Am J OrthodDentofacialOrthop. 2003;124:138–43.
17. Jedrzdrzejewski M, Smektała T, Sporniak-Tutak K, Olszewski R. Preoperative, intraoperative, and postoperative complications in orthognathic surgery: a systematic review. Clin Oral Investig. Int J Adult OrthodOrthognathSurg 2015;19(5):969–77.
18. Ruiz LP, Lara JC. Facial nerve palsy following bilateral sagittal split ramus osteotomy for setback of the mandible. Int J Oral Maxillofac Surg. 2011;40:884–886.
19. Chrcanovic BR, Custódio AL. Optic, oculomotor, abducens, and facial nerve palsies after combined maxillary and mandibular osteotomy: case report. J Oral Maxillofac Surg. 2011;69:234–41.
20. Maple JR, Vig KWL, Beck FM, Larsen PE, Shanker S. A comparison of provide'sanda consumer's perception of facial-profile attractiveness. Am J OrthodDento Facial Orthop. 2005;128:690–96.
21. Aravind Kumar.S, Lakshmi.T.Efficacy of Natural Products in Fixed Orthodontic Appliances (FOA) Treatment International Journal of Drug Development & Research 2011 ; 3(4) 0975-9344.

.

Mental Health Inventory

Kalaivani Natarajan[1] , K. Karpagam[2], S.Sangeetha[3]
Graduate student[1],Reader[2],Department of Anatomy,
Reader[3], Department of Prosthodontics,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

IntroductionAll the people donot possess all these characters at a same degree. The higher the degree , the better mental health. They are independent and responsible and also is giving and productive. The lower the degree they are depressed and are under mental stress. This survey was conducted to assess the mental health of individuals.

Aim and objective : To assess the mental health status of individuals of Saveetha University.

Materials and Methods:A survey based on mental health questionnaire by Augustes was used to evaluate the mental health of individuals.. A total of 50 individuals studying I BDS from a Reputed college were selected. They were made to sit comfortably and were given the questionnaire to fill . Once done , the results were collected and tabulated

Results: Mental health is a condition of psychological maturity a relatively constant and the enduring function of personality. It is the adjustment of human beings to the world and to each other with a maximum of effectiveness and happiness.This study concludes that individuals differ in their mental health values.

Key Words: *Social behaviour, personality, emotion, psychological maturity, socially well being.*

Introduction

According to Menninger, mental health refers to "the adjustment of human beings to the world and to each other with a maximum of effectiveness and happiness. It is the ability to maintain an even temper , an alert intelligence , socially considerate behaviour and a happy disposition". Mental health is a state of physiological maturity–a relatively constant and enduring function of personality [1]. According to the World Health Organization, mental health includes "subjective well-being, perceived self-efficacy, autonomy, competence, inter-generational dependence, and self-actualization of one's intellectual and emotional potential, among others [2].

Mental health is important at every stages of life, from childhood and adolescence toadulthood .

In general there are five primary senses to attain satisfaction and social adjustment.

1. A sense of responsibility: it is an awareness of what consequences he / she will dues to their behaviour and its effect on others
2. A sense of self – reliance a confidence of one's own way of facing problems rather than to display anger or anyother emotional outbursts.
3. A sense of direction – setting good goals and directing efforts towards success.
4. A set of personal values –having a philosophy about life based on their own beliefs and goals. The philosophy of life will tend to increase their social status.
5. A sense of individuality –recognising oneself from others. The person endeavours to develop attitudes and patterns of behaviour that entail neither blind conformityto the desires and demands of others nor a rebellious detachment and isolation from others.

All the people donot possess all these characters at a same degree. The higher the degree , the better mental health. They are independent and responsible and also is giving and productive. The lower the degree they are depressed and are under mental stress. They require motivation and counselling [3].

Particularly students in late adolescence are introduced to professional course with very minimal or no mental preparation is bound to cause a lotof adjustment, personality, behaviour, anxiety and depression among students. They also undergo a lotf of physical and

hormonal changes being a teenager. Social changes like being away from home, stressful and demanding work schedule, excessive competition etc [4-7].

Also, the changing global demographic, Economical, cultural, political, educational scenarios has created a lot of mental stressto students thus making assessment of mental health status and providing care and support is a need of the hour for a better and healthy society [8].

Materials and Methods

A survey based questionnaire was used to evaluate the mental health of individuals. It was carried out in the month of November 2015. A total of 50 individuals studying I BDS from a reputed dental college were selected. They were made to sit comfortably and were given the questionnaire to fill . Once done , the results were collected and tabulated.

The questionnaire is as follows:

I. How happy, satisfied or pleased have you been with your personal life during the past month.?

1. Extremely happy, could not be more pleased or satisfied
2. Very happy most of the time.
3. Generally, satisfied,pleased.
4. sometimes fairly satisfied, sometimes fairly unhappy
5. generallydissatidfied, unhappy
6. verydissatisfied,unhappy most of the time

II.How much of the time have you felt lonely during the past month?

1. None of the time
2. a little of the time
3. some of the time
4. a good bit of the time
5. most of the time
6. all the time

III.How often did you become nervous or jumpy when faced with excitement or unexpected situations during the past month?

1. Always

2. Very often
3. Fairly often
4. Sometimes
5. Almost never
6. Never

IV.During the past month, how much time have you felt that the future looks hopeful and promising?

1. All the time
2. Most of the time
3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

V.How much time, during the past month, has your daily life been full of things that were interesting to you?

1. All the time
2. Most of the time
3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

VI.How much time, during the past month, did you feel relaxed and free from tension?

1. All the time
2. Most of the time
3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

VII.During the past month, how much of the time have you generally enjoyed the things you do?

1.All the time

2.Most of the time

3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

VIII.During the past month, have you had any reason to wonder if you were losing your mind or losing control over the way you act, talk, think, feel or any of your memory?

1. Yes, I am very much concerned about it
2. Yes, I am quite concerned about it
3. Yes, and I have been a little concerned
4. Yes, but not enough to be concerned or worried about it
5. May be a little
6. Not at all

IX.Did you feel depressed during the last month?

1.No, never felt depressed at all

2.Yes, a little depressed now and then

3.yes, quite depressed several times

4.yes, very depressed almost all the time

5. yes to the point that Ididnot care about anything for days at a time

X.During the past month, how much time have you felt loved and wanted?

1. All the time
2. Most of the time
3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

XI.How much time, during the past month, have you been a very nervous person.?

1. All the time

2. Most of the time
3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

XII.When have you got up in the morning, this past month, about how often did you expect o have an interesting day?

1. Always
2. Very often
3. Fairly often
4. Sometimes
5. Almost never
6. Never

XIII.During the past month, how muchof the time have you felt tense or “high strung “?

1. All the time
2. Most of the time
3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

XIV. During the past month, how often did your hands shake when you tried to do something?

1. Always
2. Very often
3. Fairly often
4. Sometimes
5. Almost never
6. Never

XV. During the past month, how often did you feel that you had nothing to look forward to?

1. Always
2. Very often
3. Fairly often
4. Sometimes
5. Almost never
6. Never

XVI. How much of the time, during the past month, have you felt calm and peaceful?

1. All the time
2. Most of the time
3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

XVII. How much of the time, during the past month, have you felt emotionally stable?

1. All the time
2. Most of the time
3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

XVIII. How much of the time, during the past month. Have you felt downheartened and blue?

1. All the time
2. Most of the time
3. A good bit of the time
4. Some of the time
5. A little of the time
6. None of the time

XIX. How often have you felt like crying, during the past month?

1.All the time

2.Most of the time

3.A good bit of the time

4.Some of the time

5.A little of the time

6.None of the time

XX. During the past month, how often have you felt that others would be better off if you were dead?

1. Always
2. Very often
3. Fairly often
4. Sometimes
5. Almost never
6. Never

Results

The scores were calculated as per the options and the scores were tabulated as below 40, 40 to 80 and above 80.

Table 1:Shows the scores of individuals that are tabulated in ranges from 0-40, between 40-80 and 81-120

S.No	**Low 0 - 40**	**In between 40 - 80**	**High 81 - 120**
1.	34	55	89
2.	22	43	93
3.	27	67	99
4.	33	66	103
5.	39	74	107
6.	29	56	100
7.	25	71	82
8.	30	60	97
9.	24	50	110
10.	37	55	105

11.	35	73	88
12.	28	68	108
13.		67	90
14.		45	
15.		72	
16.		61	
17.		70	
18.		48	
19.		51	
20.		49	
21.		68	
22.		43	
23.		73	
24.		78	
25.		42	
Average	30.25	60.2	97.76

Table 2:Shows the lowest value and highest value of each categoriesrr

S. No	**Low 0 - 40**	**Inbetween 40 - 80**	**High 81 – 120**
Lowest value	22	42	82
Highest value	39	78	110

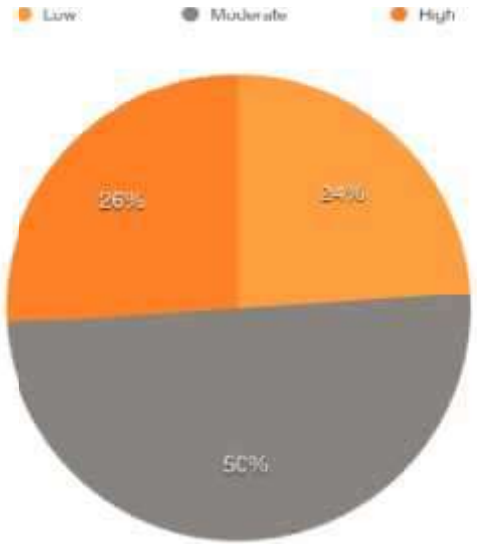


Figure 1:Showspie chart of the scores categorised as low, moderate and high

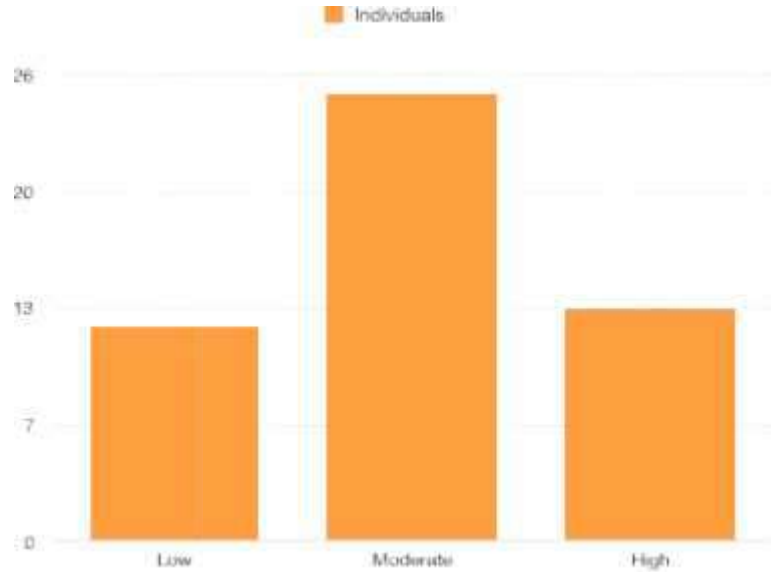


Figure 2:Shows the bar graph the scores categorised as low, moderate and high.

Discussion

Stress is the basic reason for any psychological issue. Many factors determine the healthy mental status, say from peer pressure , family , academics , society and socioeconomic status. Physical health also contributes to mental health to progress positively. This study

throws vision upon creativity and awareness among counsellors to give notice upon the mental well being and f the individual. This can help them to guide the individuals to improve, modify or to have a balanced mental health, according to the values scored by them [9].

A total of about 50 individuals were assessed. Among that , the mental health of 12 people was low with an average of 30.25. The highest value is 39 and the lowest value is 22. The mental health of 13 people were extremely high with an average of 97.76. The highest value in this group is 110 and the lowest value is 82. The mental health of 27 people were moderate and the average value is 60.89. The highest value is 78 and the lowest value is 42. Thus, mental health varies among the individuals of the university .

Several studies have been conducted in different parts of the world like Pakistan and USA. Similar study conducted by Sherina MS et-al from Malaysia, the prevalence of depression was33.6% using the CES-D scale [10]. Another study conducted by Rael D Strous et al found 55.5% of the students had reported poor mental health status [11]. In a study conducted by shah Navas P and Dahlin M et al revealed that the prevalence was as low as 23% and 12% respectively [12,13].Various international studies conducted in different parts of world like Pakistan and United States of America reported the prevalence of depression to be 15- 65% among medical students [15,16]

Mental illnesses are serious disorders which can affect your thinking, mood and behaviour. There are many causes of mental disorders. The genes and family history of individuals play an important role. Several life experiences such as stress or history of abuse also matter to a greater extent. Biological factors can also be a part of the cause. Hence mental disorders are common among these people. Treatment and therapies are available for these disorders that include Meditation, Relaxation, Yoga For Health etc [17-20]

Conclusion

Mental health is a condition of psychological maturity a relatively constant and the enduring function of personality. It is the adjustment of human beings to the world and to each other with a maximum of effectiveness and happiness. It is the ability to maintain an even temper, an alert intelligence, socially considerate behaviour and a happy disposition. I am interested to analyse the different levels of maturity of various individuals and also interested to help in

improving their maturity levels.This study also concludes that individuals differ in their mental health values.

Corresponding Author

S.Sangeetha

Reader, Department of Prosthodontics,

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

162 Poonamallee High Road,Chennai 600077,Tamil Nadu, India

Email ID:dr.sangeethakiruba@gmail.com

Telephone Number: 9894067220

References MINIMUM 20 REFERENCES

1.World Health Organization. The World Health Report 2001: Mental health: new understanding, new hope. World Health Organization; 2001.

2. World Health Organization, World Health Organization. Management of Substance Abuse Unit.Global status report on alcohol and health, 2014.World Health Organization; 2014.

3. Nojomi M, Gharayee B. Medical students and mental health by SCL-90-R. Medical Journal of The Islamic Republic of Iran (MJIRI). 2007 Aug 15;21(2):71-8.

4.Shaheena P, Shafiq M. Mental Health and coping among youth in Kashmir: Implication of psychological implications. Delhi Psychiatry J. 2014;17(2):295-302.

5. Kumar GS, Jain A, Hegde S. Prevalence of depression and its associated factors using Beck Depression Inventory among students of a medical college in Karnataka. Indian journal of Psychiatry. 2012 Jul;54(3):223.

6. Shaikh BT, Kahloon A, Kazmi M, Khalid H, Nawaz K, Khan N, Khan S. Students, stress and coping strategies: a case of Pakistani medical school. EDUCATION FOR HEALTH-ABINGDON-CARFAX PUBLISHING LIMITED-. 2004 Nov 1;17:346-53.

7.Inam SN, Saqib A, Alam E. Prevalence of anxiety and depression among medical students of private university. JOURNAL-PAKISTAN MEDICAL ASSOCIATION. 2003 Feb;53(2):44-6.

8.Manjunath R, Kulkarni P. Mental health status and depression among medical students in Mysore, Karnataka-an untouched public health Issue. Depression. 2013;96(45.5):38-7.

9.Dyrbye LN, Thomas MR, ShanafeltTD.Systematic review of depression, anxiety, and other indicators of psychological distress among US and Canadian medical students.Academic Medicine. 2006 Apr 1;81(4):354-73.

10.Sherina MS, Rampal L, Kaneson N. Psychological stress among undergraduate medical students. Medical Journal of Malaysia. 2004 Jun;59(2):207-11.

11.Strous RD, Shoenfeld N, Lehman A, Wolf A, Snyder L, Barzilai O. Medical students' self-report of mental health conditions. International journal of medical education. 2012;3:1.

12.Shah Navas P. Stress among Medical Students. Kerala Med J. 2012;5(2):53-5.

13.Dahlin M, Joneborg N, Runeson B. Stress and depression among medical students: A cross- sectional study. Medical education. 2005 Jun 1;39(6):594-604.

14.Rael D Strous, Shoenfeld N, Lehman A, Aharon W, Leah S, Barzilai O. Medical students self-report of mental health conditions. Int J Med Edu. 2012;3:1- 5.

15.Shaikh BT, Kahloon A, Kazmi M, Khalid H, Nawaz K, Khan N, Khan S. Students, stress and coping strategies: a case of Pakistani medical school. EDUCATION FOR HEALTH-ABINGDON-CARFAX PUBLISHING LIMITED-. 2004 Nov 1;17:346-53.

16.Inam SN, Saqib A, Alam E. Prevalence of anxiety and depression among medical students of private university. Journal-Pakistan Medical Association. 2003 Feb;53(2):44-6.17.Yuvaraj BY, Poornima S, Rashmi S. Screening for overall mental health status using mental health inventory amongst medical students of a government medical college in North Karnataka, India. International Journal Of Community Medicine And Public Health. 2016 Dec 22;3(12):3308-12.

18.Dyrbye LN, Thomas MR, Shanafelt TD. Systematic review of depression, anxiety, and other indicators of psychological distress among US and Canadian medical students. Academic medicine. 2006 Apr 1;81(4):354-73.

19.Safiri S, Khanjani N, Kusha A, Narimani MR, Karamzad N. Prevalence of depression and its associated factors using Beck Depression Inventory among students of School of Health and Nutrition, Tabriz, Iran in 2009. Journal of Analytical Research in Clinical Medicine. 2013 Dec 30;1(2).

20. Whiteford HA, Degenhardt L, Rehm J, Baxter AJ, Ferrari AJ, Erskine HE, Charlson FJ, Norman RE, Flaxman AD, Johns N, Burstein R. Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. The Lancet. 2013 Nov 9;382(9904):1575-86.

Parents Attitude and Knowledge towards Dental Radiography in Children

Priyadharshni S[1], Shantha Rajan[2], E. Roja[3]
Graduate Student[1], Senior Lecturer[2], Department of Pedodontics,Tutor[3], Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction:Radiographs are an essential part of most clinical dental examinations and diagnoses. Radiographs often add critical information to the clinical examination revealing developmental and eruption problem in addition to caries, pulp, and peri apical pathology. Radio graphic guidelines exist to avoid unnecessary exposure,as well as to identify individuals for whom radiographic examination will be beneficial.It is unknown whether radiation fears are exaggerated or whether parents recognise and accept the associated risks.The aim of this study is to determine the parental knowledge and attitude towards dental radiography in children.

Material and Method: A self-administered questionnaire is prepared and distributed to 42 parents with children who are to be radiographed in Chennai population and covering parental level of radiation and socio-demographics was applied which is used to assess attitude towards dental radiographs.

Results: 65% parents have a positive attitude towards dental radiographs on their children. However, the majority of parents lack knowledge(20%) regarding dental radiography, especially regarding the risks involved.

Conclusion: Most parents had a positive attitude towards dental radiographs although they had limited knowledge about radiography. This study emphasises the importance of providing accurate and appropriate information so patients and parents have a better knowledge and understanding of dental radiographs.

Key Words*: Radiograph, children, radiation, knowledge, health.*

Introduction

Professor Wilhelm Conrad Roentgen, a Bavarian physicist, accidentally discovered X-rays on November 8, 1895. The first dental bitewing radiograph, whose exposure time was about 25 minutes due to combination of unreliable generator output and relatively low sensitivity of receptor, was performed on himself by Dr. Otto Walkhoff in 1896, which was glass plated it by hand with photographic silver emulsion [1]. This cumulative effect of long and repetitive radiation exposure led to the irreparably damaged tissues of the radiography's early pioneers [2], this raised an issue to decrease the radiation exposure to the public and the operator. As a result the radiation doses have been greatly reduced needed to make radiographic images over the past century [1]. Today various factors such as fast lm, collimation, filtration, standardisation of x-ray lm processing, have greatly helped us in quality assurance programmes and achieving a reasonable radiographs by keeping radiation hazards at bay [2]. As technology advances in dental radiology, operators must maintain current knowledge and adapt their skills for the best treatment of the patient. Radiography is a highly technical held, essential to the modern dental practice. The intraoral radiographic image, when correlated with the case history and clinical examination, is one of the most useful and powerful diagnostic aids available to the dental practitioner. Although the radiation doses used by dentists might be low for individual examinations, patients are exposed to repeated examinations overtime, and many people are exposed during the course of dental care [3]. Broad range of exposures used in dental offices ranging from 5 μSv bitewing to 14-24 μSv for extra-oral imaging such as panoramic radiography are encountered. Thanks to such varied techniques, per capita dose of radiation has increased in today’s population, nonetheless our awareness regarding the same has also increased [4]. Hence the major concern of dentist is to reduce the radiation exposure to the patient and surrounding environment, without compromising the quality of image [4]. Therefore proper techniques must be employed to reduce radiation exposure to the patient through the use of protective aprons, digital radiology, high speed lms, and proper technique; thus decreasing radiographic retakes and additional exposure.

Due to the faith entrusted by the parents on the dentists regarding their children’s care, it is the moral responsibility of the dentist to inform about the biohazards associated with radiation to the parents. Children are more vulnerable to radiation than adults, which is due to the fact that there is a longer life expectancy and thus a greater potential for radiation-induced

cancers to manifest. Furthermore, the cumulative nature of radiation exposure over a patient's lifetime increases the importance of explaining radiation risks to parents[5].

Dental radiographs play an important role in the detection and management of oral diseases and a useful diagnostic aid in oral examination of children. While radiation exposure in the dental setting is relatively low, it is one of the most frequently undertaken radiographic procedures, and is often repeated several times during childhood and adolescence. When visiting the dentist, parents entrust the dentist with the care of their children and therefore they have the right to understand the often complex risks and benefits of a procedure, including the taking of radiographs.Radiographs often add critical information to the clinical examination, revealing developmental and eruption problems in addition to caries, pulp and periapical pathology. Radiographic guidelines exist to avoid unnecessary exposure, as well as to identify individuals for whom radiographic examination will be beneficial[6].

Radiographs should be taken only when there is an expectation that the diagnostic yield will affect patient care. The AAPD recognises that there may be clinical circumstances for which a radiograph is indicated, but a diagnostic image cannot be obtained. For example, the patient may be unable to cooperate or the dentist may have privileges in a health care facility lacking intraoral radiographic capabilities. If radiographs of diagnostic quality are unobtainable, the dentist should confer with the parent to determine appropriate management techniques (eg, preventive/restorative interventions, advanced behaviourguidance modalities, deferral, referral), giving consideration to the relative risks and benefits of the various treatment options for the patient.Because the effects of radiation exposure accumulate over time, every effort must be made to minimise the patient's exposure. Good radiological practices (eg, use of lead apron, thyroid collars, and high-speed film; beam collimation) are important. The dentist must weigh the benefits of obtaining radiographs against the patient's risk of radiation exposure.Dental X-rays are very safe and expose your child to a minimal amount of radiation. When all standard safety precautions are taken, today's X-ray equipment is able to prevent unnecessary radiation and allows the dentist to focus the X-ray beam on a specific part of the mouth. High-speed film enables the dentist to reduce the amount of radiation the patient receives. A lead body apron or shield will be placed over the child's body. Make sure the shield covers your child's neck to protect the thyroid gland.Keywords for good practice are appropriate selection criteria for the use of radiography, optimised radiation protection and

utilisation of the total amount of information in each radiographs[7]. The aim of this study is to determine parents attitude and knowledge towards dental radiography in children.

Material and Method

Data was collected from 42 parents with children in North Chennai population using a structured questionnaire consisting of 8 survey items. The survey was conducted in Saveetha Dental College, Chennai. Patientswho visited the hospital and requiredradiographic examination were selected for the survey. In the questionnaire,details were asked about their knowledge on dental radiograph.

Inclusion Criteria

1. Father
2. Mother
3. Guardian

Exclusion Criteria

1. Dental practitioners
2. Dental assistant

In the questionnaire, details were asked about the experience in exposing X-rays in children, parents level of education, knowledge about the risk and the importance of X-rays. The data thus obtained was subjected to statistical evaluation.

Result

A total of 42 parents with children participated and answered the questionnaire.The majority of respondents accompany their children to their dentist and were aware that exposure from a dental radiograph was too small to put their children at any significant harm, whereas most were not aware that radiation exposure from the environment is higher than radiation from dental radiographs. Of those respondents where parents accompanied their child and radiographs had been taken, 60% felt that the risks of dental radiographs were not explained to them, but 40% said that the dentist explained the reasons for taking dental radiographs(Tab.1).

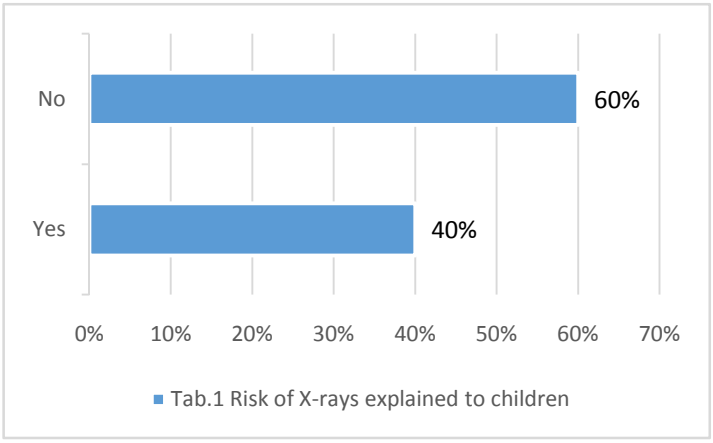


Figure 1: Risk of X-rays explained to children

Parents with children who have had previous dental radiographs were more likely to perceive dental radiographs as ‘useful’ , ‘pleasant’, ‘good’, ‘unpleasant’ (Tab.2). Parents with children who visited the dentist regularly (every six months) were also more likely to perceive that the benefits outweighed the risks.

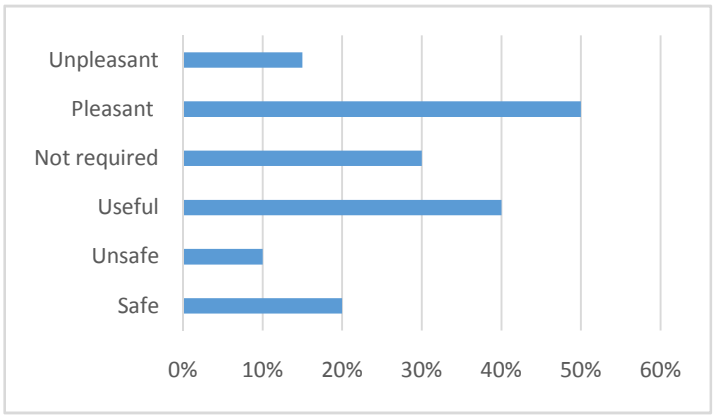


Figure 2: X-rays on Children

A higher level of parental radiographic knowledge was associated with a higher level of formal education(65%) and having children who have had previous dental radiographs reported as ‘useful’(40%) and parents who admitted to lacking knowledge about radiographs perceived them as unpleasant. Parents with average knowledge and who has done undergraduate have reported that X-rays are ‘unpleasant’(Tab.3).Regularity of dental visits

by the parent,type of dental service were found to have no significant associations with attitude or knowledge.

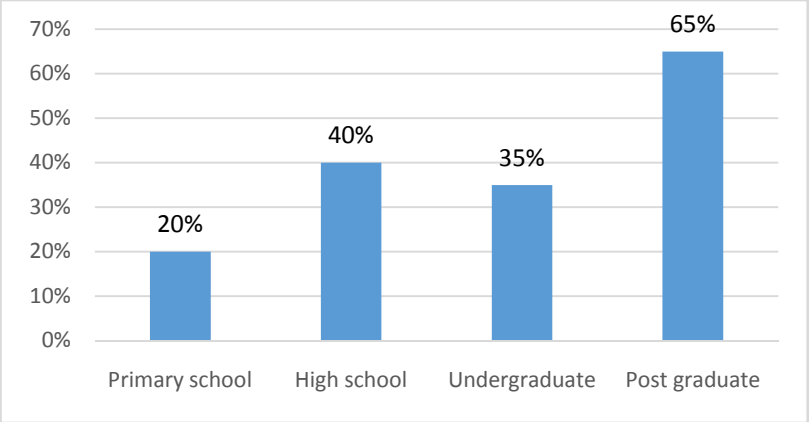


Figure 3: Level of education

Discussion

A key insight gained from this study is that while knowledgeaboutdentalradiographsislow,parentshaveapositive attitudetowardsradiographs.This suggeststhatattitudeisnotprimarilyderivedfromknowledge. On study, a survey done by chiri et al inAustralia reported that Parents with high levels of knowledge about radiographs stated that they thought dental radiographs were safe and beneficial. Conversely, parents who answered the knowledge-based questions incorrectly perceived radiographs as being 'harmful'. Attitudesdevelopovertimeandareorganisedaroundthreemaintypes of beliefs: 1- descriptive beliefs thesearebasedondirect experiences andareofmost value; 2-inferential beliefs these are based on an inferenceprocess, wherebyabeliefisinferredfromother beliefs; 3-informationalbeliefs these arebasedoninformationderivedfromanoutsidesource[8]. Thisstudyfoundthatdescriptive beliefs, basedonparentsexperienceswithradiographstakenfortheirchildren,mayhaveshapedparentalattitudes[9].

Thisstudyindicatedthatparentswhoparticipatedinthesurveyhavehadpositiveexperienceswhenradiographshavebeentaken,andthesepreviousexperienceshaveimpactedontheparentalattitudes.Forexample,parentswithchildrenwhohavehadpreviousdental radiographsandparentswhohavetheperceptionthatradiographsare'good'and'useful'weresignificantlyassociatedwitheachother.

Whilethemajorityofparentshadtheimportanceofdentalradiographsexplainedto them, significantlyfewerparentsreported thattheyhad been informedoftheradiationrisks(40%).Furthermore, over half the respondents were university educated and approximately 60% had experience of dental radiographs. This skewed demographic may have affected the results and therefore the relatively positive attitudes towards dental radiographs found in this population may not extend to the general population[10,11]. The low response rate may have been improved if direct communication was made with the parents and follow-up reminders had been placed in newsletters. It is unknown how many children were giving their parents the newsletter or how many parents read the school newsletter.Reassuringly however, parents who believed the risks were explained to them also agreed that the benefits of the radiographs outweigh the risks. Dentists need to assure patients that they are committed to obtaining excellent clinical results with the lowest possible radiation risk, and that the potential benefits of modern medical imaging procedures almost always far outweigh the associated risks[12,13].

Dental radiographs play an important role in diagnosis and treatment planning. The modalities at the disposal of dentists range from intraoral radiography to cone beam computed tomography. In the field of dentistry, radiation exposure for diagnostic purpose is minimal[14]. However, it is one of the most frequently undertaken radiographic procedures, which is repeated several times during childhood and adolescence. Thus, it is the parent's right to know the associated radiation risks. As per literature available, there are not many studies done to assess the knowledge and attitude of parents regarding dental radiography for their children. Thus, it is unknown whether the fears associated with dental radiography are overstated or the parents are aware about the risks and accept them[15,16].

In a study done by Babu et al.The knowledge of the parents toward dental radiography was found to be relatively low. More than half of the participants were oblivious to the facts such as the damage to the body from dental radiographs is not permanent (58%) or the exposure from dental X-rays is too small to put their child at any significant harm (54%). 56% of the participants were unaware that the exposure to radiation from the environment (e.g. the sun) is higher than the radiation from dental X-rays. However, 58% of the parents knew that radiation from other medical procedures such as chest X-ray is more and, also that if the child wore a lead apron during dental X-ray procedure, it would provide protection against possible

radiation damage[17]. Chiri et al. in their study also reported a low knowledge of the parents regarding dental radiography[6].

Children from low-income and disadvantaged families have a disproportionately higher prevalence of untreated dental caries and lower dental care utilization than higher income group children. A reduction of oral health disparities requires a comprehensive oral health promotion strategy for better oral health among low-income group children. This strategy includes programs such as oral health education, preventive and comprehensive dental care, and social and organizational interventions to address multiple barriers to access and utilise dental care[8]. For these interventions and programs to be successful, the caregivers should be motivated to take action by being aware of their children's oral health conditions and the need for prevention and early interventions. A better understanding of how well caregivers perceive their children's oral health status may improve utilization of dental care services[18].

The radiation dose should be kept as low as can reasonably be achieved both for patient and operator. Usually there will be no damage of clinical significance caused by low level X–rays used in dental radiography. However, the hypothesis in modern radiation protection is that any dose of radiation has the potential to cause biological harm. "It is impossible to relate any specific dental exposure to any specific cancer. All we can say is that the evidence indicates that even very small doses carry the potential for causing cancer" [Smith, 1987]. The probability of long-term effects (stochastic effects) of radiation increases with the dose of exposure, but the severity of the consequential effect when it occurs, such as cancer, is not affected. That means that the probability for cancer is related to radiation dose, but when the disease unfortunately breaks out, the severity of the disease will not depend on the radiation dose. The younger the individual, the higher the vulnerability to radiation is because of the large number of cell divisions occurring in small children. Children also have a higher proportion of the bone marrow located in the skull than adults have. Smith [1992] has shown in a calculation of risk estimates that about one induction of malignant disease per 1,000,000 dental exposures of 5 year old children can be expected. The International Commission on Radiological Protection [ICRP, 1991] has proposed the estimate for a single small dose at the age of 5, which is used in calculations. The risk is reduced when the fastest films available, or digital radiography, are used due to the lower dose needed[19,20]. The image quality could be as good as that of conventional films, but depends on the digital system used. At the

moment there are large variations in quality between the different systems. In a recent study dentists considered the user-friendliness of the handling of the two different digital systems before taking a radiograph as less than for the conventional film [Berkhout et al., 2002]. The patient's comfort was also mentioned as unfriendly especially when the systems were used for children. In the case of digital radiography, the elimination of the chemistry of film processing after taking the radiograph was considered an advantage. Digital images are best viewed on a good computer screen and often loose quality when printed. Such images are like any computer files and may be stored on disks and easily transferred to other computers. In the future, "expert" systems may provide decision support based on automated image analysis [Firestone et al., 1998; White, 1999]. In conclusion, digital radiography has advantages over conventional radiography, but the bulky sensor systems with attached cable and the need for a computer are clinical inconveniences. No studies concerning the use of digital radiography in children are available, but it seems likely that at present time the advantages of these systems are cancelled out by the disadvantages such as acceptance of the sensor or phosphate plate by the child.

Conclusion

Most parents had a positive attitude towards dental radiographs although they had limited knowledge about radiography. This study emphasises the importance of providing accurate and appropriate information so patients and parents have a better knowledge and understanding of dental radiographs. It is imperative for dental health professionals to understand their role in shaping positive attitudes towards dental radiographs.Dentists need to assure patients that they are committed to obtaining excellent clinical results with the lowest possible radiation risk, and that the potential benefits of modern medical imaging procedures almost always far outweigh the associated risks.

Corresponding Author

E.Roja,

Tutor,Department of Public Health Dentistry,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600 077, Tamil Nadu, India

Email Id: itsrojaa@gmail.com

Contact Number: +91 99941 74480.

References

1. Farman TT and Farman AG. Evaluation of a new F-speed dental X-ray lm.The effect of processing solutions and a comparison with D- and E-speed lms.Dentomaxillofacial Radiology. 2000;29:41-44. サ

2. Campbell DJ. A brief history of dental radiography. N Z Dent J. 1995;91:127-133. サ

3. Lee BD and Ludlow JB. Attitude of the Korean dentists towards radiation safety and selection criteria.Imaging Science in Dentistry. 2013;43:179-184. サ

4. Madalli VB, et al. The evaluation of effect of developer age in the detection of proximal caries using three speed dental x-ray lms: an in-vitro study. J Clin Diagn Res. 2014;8:236-239.

5. Frush DP. Radiation risks to children from medical imaging. Rev Med Clin Condes 2013;24:15-20.

6. R Chiri, S Awan, S Archibald,PV Abbott. Parental knowledge and attitudes towards dental radiography for children,Aust Dent J 2013; 58: 163–169.

7.Espelid I, Mejare I, Weerheijm K. EAPD guidelines for use of radiographs in children. Eur J Paediatr Dent 2003;1:40–48.

8.Fishbein M, Ajzen I. Belief, attitude, intention, and behavior: an introduction to theory and research. Reading: Addison-Wesley, 1975.

9.Ludwigk.looe.kParental knowledge and attitudes towards dental radiography for children. British Dent J 215, 561 (2013).

10.Aravind BS, Joy ET, Kiran MS, Sherubin JE, Sajesh S, Manchil PR. Attitude and awareness of general dental practitioners toward radiation hazards and safety. J Pharm Bioallied Sci 2016;8 Suppl 1:S53-8.

11. Chiri R, Awan S, Archibald S, Abbott PV. Parental knowledge and attitudes towards dental radiography for children. Aust Dent J 2013;58:163-9.

12.kelly SE, Binkley CJ, Neace WP, Gale BS. Barriers to care-seeking for children's oral health among low-income caregivers. Am J Public Health 2005;95:1345-51.

13. Sohn W, Taichman LS, Ismali Al, Reisine S. Caregivers perception of child's oral health status among low-income African Americans. Pediatric Dent 2008;30:480-7.

14. Ashok NG, kumar V J. Patients perception on dental radiographs: A questionaire-based study. Int J Orofac Biol 2017;1:28-31.

15.Looe HK, Pfaffenberger A, Chofor N, Eenboom F, Sering M, Rühmann A, et al. Radiation exposure to children in intraoral dental radiology. RadiatProtDosimetry 2006;121:461-5.

16. Ludlow JB, Davies-Ludlow LE, White SC. Patient risk related to common dental radiographic examinations: The impact of 2007 International Commission on Radiological Protection recommendations regarding dose calculation. J Am Dent Assoc 2008;139:1237-43.

17. NetaG, Rajaraman P, Berrington de Gonzalez A, Doody MM, Alexander BH, Preston D, et al. A prospective study of medical diagnostic radiography and risk of thyroid cancer. Am J Epidemiol 2013;177:800-9.

18. Preston-Martin S, White SC. Brain and salivary gland tumors related to prior dental radiography: Implications for current practice. J Am Dent Assoc 1990;120:151-8.

19. Ludwig RL, Turner LW. Effective patient education in medical imaging: Public perceptions of radiation exposure risk. J Allied Health 2002;31:159-64.

20. Lee CI, Haims AH, Monico EP, Brink JA, Forman HP. Diagnostic CT scans: Assessment of patient, physician, and radiologist awareness of radiation dose and possible risks. Radiology 2004;231:393-8.

Patient Expectations from Dentist - A Questionnaire Based Study

Divyadharshini V[1], Loganathan[2], Dinesh Prabu[3]
Graduate student[1,] Reader[2], Department of Oral and Maxillofacial surgery,
Senior Lecturer[3], Department of Oral and maxillofacial Surgery
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Aim: To assess the patientsopinion towards dentist appearance, clinical attire, cross infection control, years of experience and their expectations from their dentist.

Materials and method : Patients attending a dental hospital for a consultation appointment were asked to complete a close ended questionnaire comprising 12 questions. A representative sample of patients completed 100 questionnaires over a period of 4 weeks. Their responses were tabulated and analysed statistically.

Result : The study found that majority of patient preferred their own gender dentist and they preferred young dentist with 1-5 years of experience. Some patients preferred dentist wearing white coat while some patients were comfortable with whatever the dentist was wearing. The majority of patients preferred dentist wearing name badge and personal protective equipment. The majority of patients also preferred treatment from colleges and hospitals and preferred dentist who has done MDS/ specialisation. The majority of patients were more comfortable with dentist who is smiling, welcoming and being friendly. Some patients preferred their dentist to talk more while some preferred their dentist to talk appropriately and few patients preferred their dentist to talk less. Dentist having long nails and malaligned or fractured teeth bothered some patient while it didn't bother some patients and some patients didn't notice it.

Conclusion :A considerable amount of research dealing with the professional image of healthcare providers has been produced, but very little of this has been specific to the practice of dentistry. So it is hoped that this study will be informative for dental team and the results should be taken into consideration.

Keywords-*Dentist, Dental Hospitals, Patient, Preferences, Professionalism*

Introduction

Professionalism is an image that promotes a successful relationship with the patient and thus enables the foundation of effective patient care[1]. This fundamental connection is created during the first meeting where the patient forms a initial impression of the clinician[2]. Walsh suggested that proper appearance formed part of the essentialelements for development of this successful professional relationship[3]. First impression can make a difference. How a doctor dresses may be important in determining the success of the patient-doctor relationship[2].Over the years professionalism in health care has had several definitions, many of which are very ambiguous[4].Professionalism is expressed in various ways including the dimensions of competence, ranging from comprehension of basic biology principles to clinical skill; engagement, which includes behaviours and attributes pertaining to empathy and communication; reliability, which pertains to timely access to competence; dignity, which includes treatment of patient, clinical staff, and self; the health care providers dedication to placing the needs of the patient above his or her own needs; and concern for the quality of clinical care[5]. Three broad components of professionalism were introduced by Walsh, suggesting that proper appearance, behaviour, and conversation are essential for a successful relationship[3]. This study also found that patients are more apt to discuss medical issue with a health care provider who is well groomed, has a professional voice, and a confident expression. Physical attractiveness also positively relates to increased patient willingness to disclose symptoms[6, 7].

Dentist attire has been moulded by tradition and fashion over centuries. There have been many changes in the dental field and patient expectations and with increased proportion of female doctors entering the profession that have led to changes in dentist dress code. White coats were worn by dentist for professionalism, identification and hygiene, yet white coats may be a source of, rather than a barrier to cross infection.

Patients will come to dental appointments with some level of expectation regarding their likely outcome. Patients can have a very clear and detailed outcome in mind or it may be more broad and open. Most of the patient's expectation will be realistic; however some won't be.Unrealistic expectations from patient'spresent very real challenges for dentists. If a patient with unrealistic expectations undergoes treatment, it's very unlikely those expectations are going to be met. If a patient's expectations haven't been met, then the patient is going to be unhappy or dissatisfied with the treatment. These unhappy and dissatisfied patients are the ones more likely to complain about the treatment and expect

further corrective treatment or compensation. It's therefore vital that dentists do all they can to help the patient fully understand treatment and the likely and possible treatment outcomes before treatment begins.

A key step in making sure a patient has realistic outcomes regarding treatment is to have an open and honest conversation with them. This will not only provide the patient with further information about their treatment, but will also give the dentist a clearer understanding of the patient's expectations. A dentist's clinical skills are vital to what they do, however effective communication goes a long way in providing positive outcomes.

In order to make the patient'shave realistic expectations, dentists must ensure they explain the treatment and its outcomes to patients using simple and clear terms. Technical and clinical words should be avoided as many patients will not be able to understand these terms. Dentists should also consider how they tailor their language and the information for each individual patient. For a person with language or literacy challenges, they may need information presented in a more detailed manner than other patients. Dentists should also consider using diagrams, pictures or models to assist with understanding where appropriate.

When discussing treatment with a patient, it's important that dentists don't make assumptions about what the patient will or won't understand. It's easy for dentists to become so familiar with what they do and know that they sometimes forget how foreign that knowledge can be to other people. Patients may have varying degrees of knowledge and experience regarding dental treatment. Therefore, what they understand about their treatment will also vary.

When a patient requests a particular form of treatment, this is an occasion when a dentist should be especially careful of the patient's expected outcome. When a patient has requested a form of treatment, the patient has clearly has a idea around what treatment they need to get the outcome they desire. In case if what the patients demanded is not best for them, then the dentist need to be sure they don't rush into providing the patient with the requested treatment and explain them about various treatment options available. As with all patients, there needs to be a thorough assessment and diagnosis process. Then the patient is to be provided with their treatment options, as well as the risks and benefits of those options. There may be treatment options which are more suitable for that patient which the patient isn't aware of. The patient also needs to be made aware of the likely treatment outcomes for each of those treatment options.

The dentist needs to be sure the patient has all required information before consenting to treatment and this includes understanding the likely outcomes. And dentists need to

remember that they’re always responsible for the treatment they’ve provided, regardless of whether it was requested by a patient.

In this study we focused on the importance of patient’s opinion towards their dental professional based on their physical appearance, gender preferences, age preferences, years of experience, dental care system, clinical attire and dentist attitude. In the modern world of clinical dental practice the dentist-patient relationship is moving towards a customer- based service, with increasing demands and pressure. Patient’s opinion keeps changing so it is important for the dental professionals to keep an update on patient preferences which will improve dentist-patient relationship and aids in satisfying the patient. Dentist should groom themselves in a way that they feel is acceptable to their patients.

Materials and methods

A questionnaire based study was conducted of new patients attending an outpatient dental facility for consultation appointments. A self-completion questionnaire consisting of 12 question was developed to gather data on patientopinions on dental clinical attire, name badges and a number of cross-infection control procedures.

Inclusion criteria

Patients attending Saveetha dental college and hospital.

Exclusion criteria

Patients unable to speak English,

Uneducated patients or

For any other reason which would impede their understanding of the questionnaire.

The questionnaires were completed in the waiting area prior to the participants' appointments. A total of 100 questionnaires were distributed over afour weekperiod of which 50 were given to males and 50 were given to females.

The demographic information collected from the questionnaire included the respondent's age, sex, address, occupation and educational qualifications. Of the 100 questionnaires 50 were given to males and 50 were given to females.

Completed patient questionnaires were collected and were analysed for response frequency and the results tabulated. The chi-squared analysis for non-parametric data with the appropriate degrees of freedom was performed on the data to assess responses to the

questionnaire items across patient age groups and gender. Statistical significance was determined at p <0.05.

Results

The surveyed patient pool consisted of 50 males (50%) and 50females (50%). The age of the patients was between 18-50 years age. The mean age of the sample was 29.04 years.

Gender preferences

Of those 50 male patients, 60% of the patients preferred male dentists and 30% of the patients preferred female dentists. 10% of the male patients were comfortable with both the male and female dentists (Figure 1).

Of those 50 female patients, 54% of the patients preferred female dentists and 32% of the patients preferred male dentists. 14% of the female patients were comfortable with both the male and female dentists (Figure 2).

A Chi squared test indicated statistically significance (p<0.05) with the difference between male and femalepatientpreferences.

Figure 1: Male Patient's Preference over Dentist's GenderOver Dentist's Gender

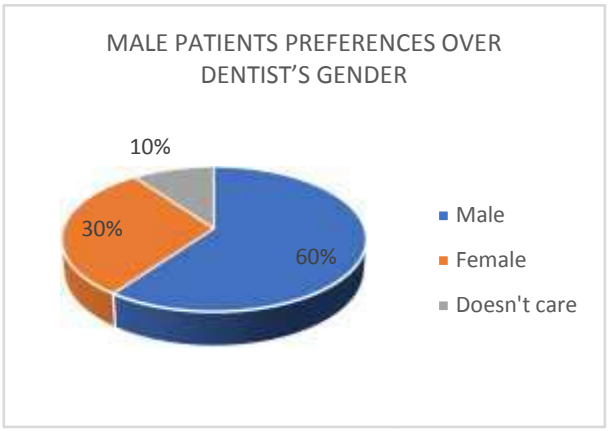
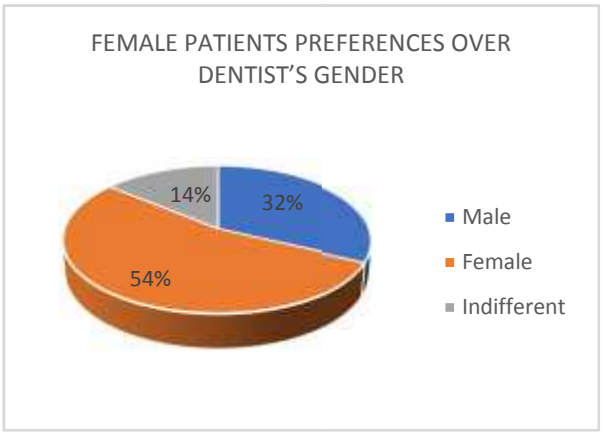


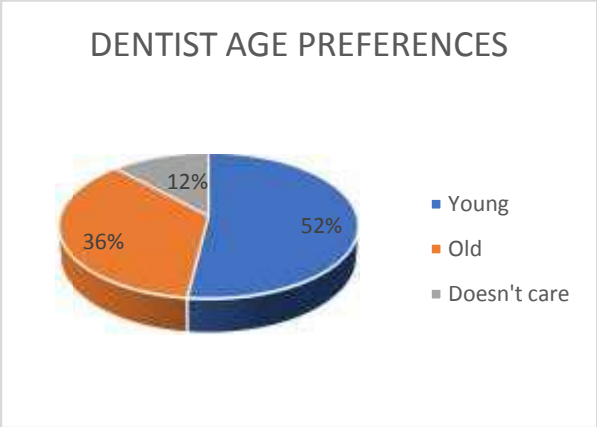
Figure 2: Female Patient's Preference



Dentist's age preferences

Of those 100 patients, 52% of the patients preferred young dentists, 36% of the patients preferred old dentists and 12% of the patients were comfortable with both young and old dentists (Figure 3).

Figure 3: Dentist's Age Preference



Dentist's years of experience

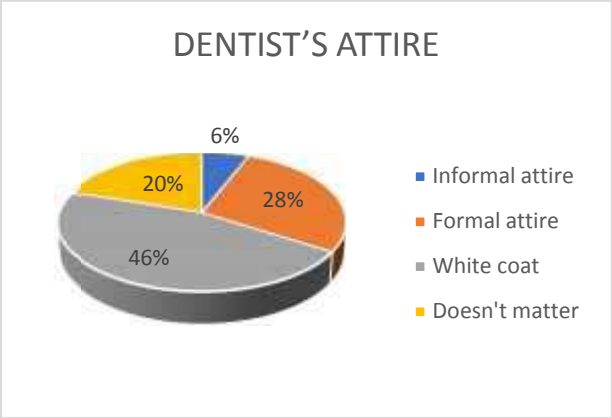
Of those 100 patients, 45% of the patients preferred dentists with 1-5 years of experience, 20% of the patients preferred dentistswith 5-10 years of experience and more than 10 years of experience and 15% of the patients preferred dentists with less than 1 year of experience (Figure 4).

Figure 4: Dentist's Years of Experience

Dentist's attire

Of those 100 patients, 46% of the patients preferred dentists wearing white coat, 28% of the patients preferred dentistswearing formal attire, 20% of the patients didn't care about dentist's attire and 6% of the patients preferred dentists wearing informal attire (Figure 5).

Figure 5: Dentist's Attire



Dentist's with long nails and malaligned/ fractured tooth

Of those 100 patients, dentistshaving malaligned, stained or fractured teethbothered43% of the patients, did not bother 31% of the patients, 26% of the patients didn't notice it (Figure 6). Of those 100 patients, dentists having long nails bothered60% of the patients, did not bother 22% of the patients and 18% of the patients didn't notice it (Figure 7).

Figure 6: Dentist's with Long Nails and Malaligned/Fractured Tooth Malaligned

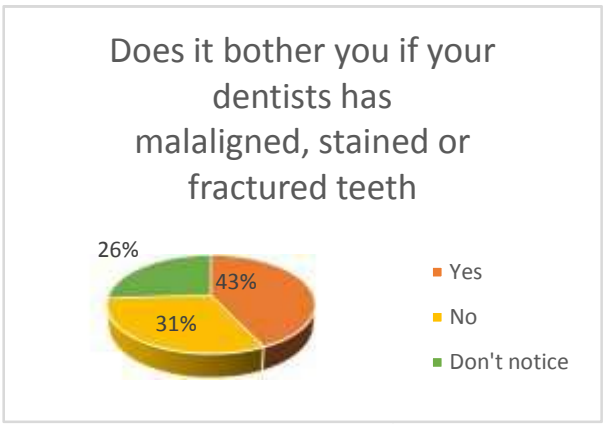
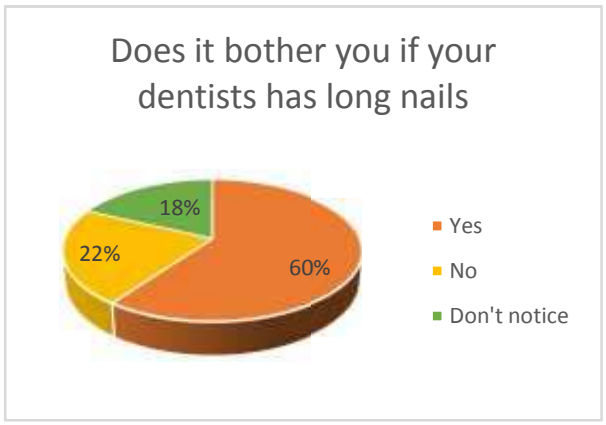


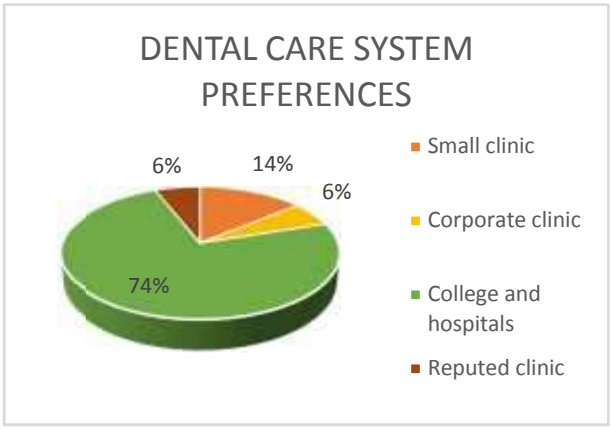
Figure 7: Dentist's with Long Nails



Dental care system preferences

Of those 100 patients, 74% of the patients preferred getting treated in college and hospitals, 14% of the patients preferred going to small clinics and 6% of the patients preferred going to corporate and reputed clinics (Figure 8).

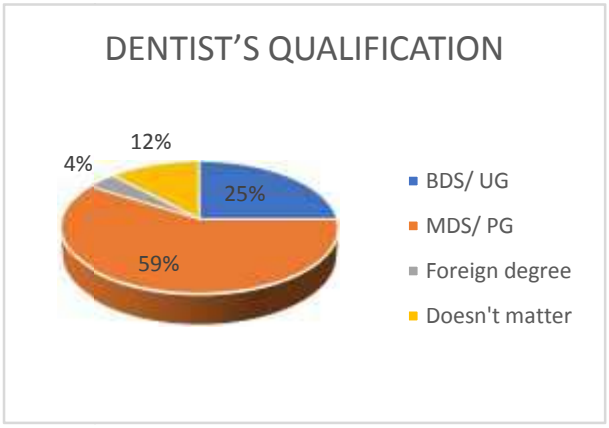
Figure 8: Dental Care System Preferences



Dentist's qualification

Of those 100 patients, 59% of the patients preferred dentists with MDS degree, 25% of the patients preferreddentistswith BDS degree and 4% of the patients preferred dentists with foreign degree. For12% of the patients, dentist's qualification didn't matter (Figure 9).

Figure 9: Dentist's Qualification



Personal protective equipment

Of those 100 patients, 79% of the patients preferred dentists wearing personal protective equipment, 11% of the patients preferred dentistsnot wearing personalprotective equipment and 10% of the patients didn't have any idea about personal protective equipment (Figure 10).

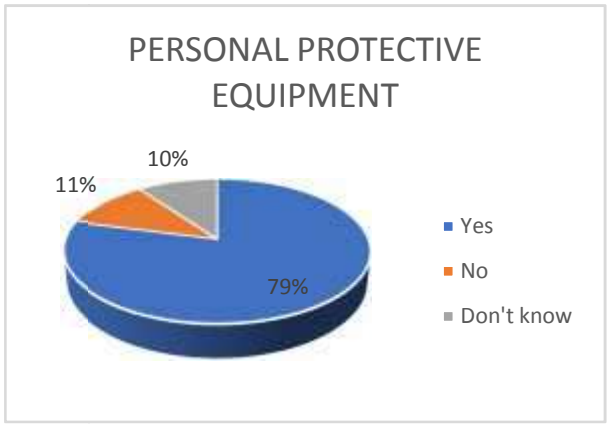
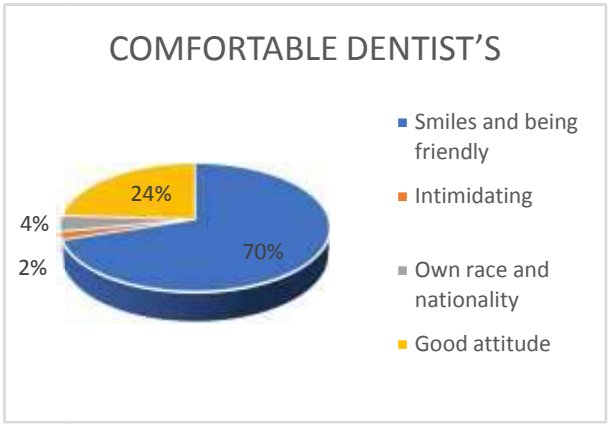


Figure 10: Personal Protective Equipment

Comfortable dentists

Of those 100 patients, 70% of the patients preferred dentists who is smiling, welcoming and being friendly, 24% of the patients preferred dentists with good attitude, 4% of the patients preferred dentists who belongs to their own race and nationality and 2% of the patients preferred dentists who seemed intimidating (Figure 11).

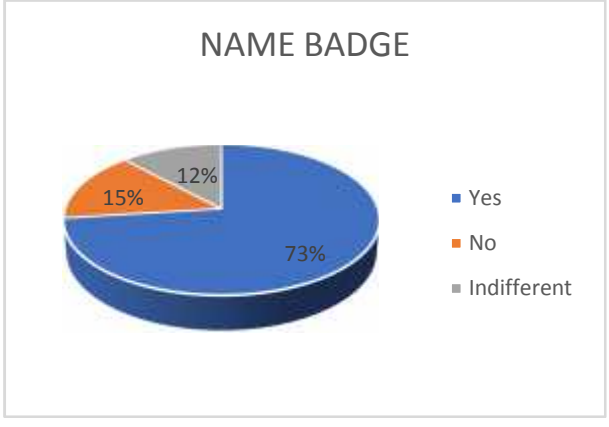
Figure 11: Comfortable Dentist's



Name badge

Of those 100 patients, 73% of the patients preferred dentists wearing name badge, 15% of the patients preferred dentists not wearing name badge and 12% of the patients didn't care about name badge (Figure 12).

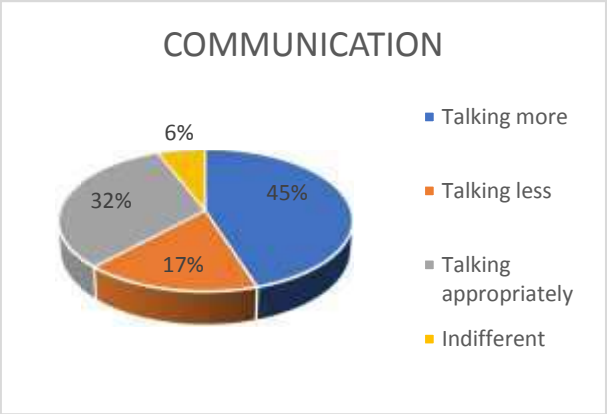
Figure 12: Name Badge



Communication

Of those 100 patients, 45% of the patients preferred dentists talking more, 32% of the patients preferred dentiststalking less, 17% of the patients preferred dentists talking appropriately and 6% of the patients didn't care (Figure 13).

Figure 13: Communication



Discussion

We measured opinions using a questionnaire based study of patients attending Saveetha Dental College and Hospital. Over a four week period of time, sample sizes of 100 patients were achieved. The demographics indicated that 50% consisted of male patients and 50% consisted of female patients with a mean age of 29.04 years.

From the results, it was found that the female patients mostly preferred the female doctors and the male patients preferred the male doctors. From Ackerman-Ross and Sochat studies in 1980, Women seem to prefer female physician and men seem to prefer male physician, so that there is a similar gender preference[8].The patients preferred younger doctors with 1-5 years of experience and with a MDS degree.

The results indicate that patients have strong opinion about dental professional's dress.When the patients were asked about the dentist's attire, majority of the patients wanted the dentists to wear a white coat and they were more comfortable with the formal attire.It is widely reported that patients prefer medical personnel to be smartly dressed, despite debate amongst medical colleagues about the wearing of white coats[9, 10, and 11].

Majority of the patients were concerned about the dentist having long nails and malaligned, stained or fractured tooth.

About 74% of the patients preferred getting treated in the collage and hospitals rather than going to a private clinic.The majority of the respondents (79%) preferred the dentist to wear

headcap, mouthmask, safety glasses and gloves.These results were supported by the findings of Shulman's study in 2001[12].

The patient preferred the doctor who welcomes them with a pleasant greetings and they prefer the dentists to talk more or in an appropriate amount. The study conducted by Deepthi K stated that 55% of the dentists were friendly[13].

The results indicated that majority of the patients preferred dental professionals to wear a name badge.This is in keeping with previous studies found in medical literature[14,15].

In the study conducted by G.N.Karpagam et al. stated that the patient felt secure and safe when the dentist gives proper respect to them[16].

Conclusion

This study has implications for all branches of dentistry where dental professionals strive to deliver a patient-centred service. We have drawn attention to the importance of physical appearance and its effect upon first impressions and interpersonal relationships. From this study we can conclude that patients place a great deal of emphasis on dental professionalsway of dressing and attire. They are supportive of the appropriate use of protective equipment such as face masks and safety glasses and prefer their dentists to wear them. They also prefer dental professionals to wear name badges. Those sampled in this study expressed a strong preference for smartly dressed dental consultants and specialists who wear a white coat. Having this insight into patient preferences should enable individuals and organisations to set standards for appropriate dress codes for dental professionals.

Dentists should be doing all they realistically can to improve the outcomes for their patients and reduce the likelihood of poor outcomes and complaints. Dentists should use their clinical knowledge in conjunction with practical terminology to assist patients to develop realistic expectations.

Future research could focus on patient opinions towards dress codes for other members of the dental team including dental care professionals. It may also be useful to determine patient opinions on dress codes in the general practice and community dental services. In addition, it may be informative to survey patient attitudes towards dental student’s attire. Students operate in an environment where the development of a successful working relationship is paramount given the relative inexperience of the clinician. This is also the setting where our younger colleagues begin to understand the values of professionalism which will positively influence their future practising careers.

Corresponding Author

Dinesh Prabu M,

Senior Lecturer,Department of Oral and Maxillofacial Surgery,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University,

162, Poonamallee High Road,Chennai 600077, Tamil Nadu, India.

E-Mail: rogerprabu@gmail.com

Telephone Number: +919790098719

Reference

1. Brosky M, Keefer O, Hodges J, Pesun I, Cook G. Patient perceptions of professionalism in dentistry. J Dent Educ 2003; 67(8): 909-915.

2. Lill, Wilkinson. Judging a book by its cover: descriptive survey of patients' preferences for doctors' appearance and mode of address. Bmj. 2005 Dec 22; 331(7531):1524-7.

3. Walsh K C. Projecting your professional image. Imprint 1993; 40(5): 46-49.

4. Stevens RA. Themes in the history of medical professionalism. Mt Sinai J Med 2002; 69(6):357-62.

5. Barondess JA. Medicine and professionalism. Arch Intern Med 2003; 163(2):145-9.

6. Young JW. Symptom disclosure to male and female physicians: effect of sex, physical attractiveness, and symptom type. J Behav Med 1979; 2(2):159-69.

7. Gjerdingen DK, Thorpe W, List-Holt P. The physician’s appearance and professionalism. Res and Staff phys 1990; 36(1):65-74.

8. Ackerman-Ross FS, Sochat N. Close encounters of the medical kind: attitudes toward male and female physicians. Social Science & Medicine. Part A: Medical Psychology & Medical Sociology. 1980 Jan 1;14(1):61-4.

9. Matsui D, Cho M, Rieder M J. Physicians' attire as perceived by young children and their parents: the myth of the white coat syndrome. PediatrEmerg Care 1998; 14(3): 198–201.

10. Zwart D L, Kimpen J L. The white in paediatrics: link between medical history and preference for informally dressed physicians. Ned TijdschrGeneeskd 1997; 141(3): 2020–2024.

11. Gonzalez Del Ray J A, Paul R I. Preferences of parents for paediatric emergency physicians' attire. PediatrEmerg Care 1995; 11(6): 361–364.

12. Shulman E R, Brehm W T. Dental clinical attire and infection-control procedures. J Am Dent Assoc 2001; 132(4): 508–516.

13. Deepthi K, Pradeep D. A study on Dental Care Satisfaction among Adult Patients. International Journal of Innovative Research and Advanced Studies.2016; 3(9): 100-101.

14. Kanzler MH, Gorsulowsky DC. Patients' attitudes regarding physical characteristics of medical care providers in dermatologic practices. Archives of Dermatology. 2002 1; 138(4):463-6.

15. Gjerdingen D K, Simpson D E, Titus S L. Patients' and physicians' attitudes regarding the physician's professional appearance. Arch Intern Med 1987; 147(7): 1209–1212.

16. Karpagam G N, Mahesh, Shanmugavel. Patients Needs and Expectations on Dental Treatment. Research J. Pharm. and Tech. 2016; 9(7):933-936.

Patients Acceptance for Orthodontic Tooth Extraction

Preethi Pavithra.R [1], Asha Ramesh [2]

Graduate student [1],Senior Lecturer [2],Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,

Saveetha University, Chennai, India

Abstract

Introduction: All orthodontic treatment requires patient compliance and hence it is necessary to assess the patient opinion for success of treatment

Aim: The purpose of this study was to assess the rate of acceptance for therapeutic tooth extraction as part of orthodontic treatment. The aim of this study was to analyse the various responses of patients for the extraction of tooth in orthodontic treatment.

Material and Method: This survey was conducted among hundred patients who reported to the dental hospital for orthodontic treatment. A questionnaire containing twenty questions related to orthodontic tooth extraction was formulated.

Result:Among 100 patients, 70% of patients accepted for orthodontic tooth extraction in order to achieve aesthetic results.

Conclusion: This study concluded that patient acceptance and co-operation towards diagnosis and proposed treatment planning is very essential for successful treatment outcome.

Key Words: *Extraction; Patient Acceptance; Orthodontic treatment; Survey; Aesthetics*

Introduction

The most frequent method to gain space for orthodontic purpose is by extraction of one or more teeth. Premolars are most frequently extracted teeth as a part of orthodontic treatment. Extraction of one premolar from each quadrant of jaw provides sufficient space to correct the malocclusion. Extraction of selective teeth can be utilized for correction of malocclusion in both the anterior as well as the posterior segments of the arch. Ideally the arch length and tooth material should be in harmony with each other [1]. Discrepancy in the size of the dentition and arch length can result in crowding of teeth or proclamation of anterior region. In some situations, careful time of extractions may result in spontaneous correction of the malocclusion.

The factors which affect the decision to extract include the patients' medical history, the attitude to treatment, the oral hygiene, caries rates and the quality of the teeth. The decision to extract teeth or not and the number of teeth to be extracted can influence the final result of orthodontic treatment, including esthetics, occlusion, satisfaction of the patients and their families, as well as treatment time. Hence patient's acceptance and cooperation towards diagnosis and proposed treatment planning is essential for successful treatment outcome. This survey was conducted among hundred patients who reported to the dental hospital for orthodontic treatment [2].

All orthodontic treatment requires patient compliance in, for example, maintaining adequate oral hygiene, not breaking or damaging the orthodontic accessories, or simply attending regular appointments. Certain types of malocclusion, however, require additional compliance to ensure treatment success. To correct certain types of Class II malocclusion, especially those of a skeletal origin, patients must wear a headgear [3]. Moreover, in the treatment of Class III malocclusion with maxillary deficiency (patient with growth potential), the use of maxillary protraction face mask is also indicated. In most treatments, the regular use of intermaxillary elastics as an aid in the correction of malocclusion or in the final treatment stage for intercuspation also requires patient compliance. All the resources mentioned above pose patient compliance difficulties involving potential aesthetic concerns [4].

At first, it is extremely difficult to determine whether or not a patient will cooperate, but by observing certain criteria, such as patient behavior in the office, the nature of their relationship with their escort and through an interview with the parents, we can venture some predictions

regarding compliance [5]. These remarks apply mainly to adolescent patients. Overall, adult patients are more compliant than youths because they are more emotionally mature and can, therefore, better understand the importance of this factor in their treatment. When significant cooperation is required it is suggested that a restudy be conducted after a certain period of time since, if compliance is indeed an issue, the orthodontist will not be able to fully rely on this factor to resolve borderline cases [6]. Sometimes lack of compliance can extend treatment time and even lead to reviews of the initial planning, requiring dental extractions.

Class II malocclusions with an adequate lower arch can be corrected by moving the upper teeth distally with the use of elastics or headgear. Both require substantial patient compliance. Alternatively, distal movement can be achieved with mini-implant support, or orthodontic correction can be accomplished by extracting upper premolars, which requires virtually no patient cooperation [7].Some treatment plans can achieve similar results whether conducted with or without extractions (especially borderline cases). However, others may have their treatment outcome jeopardized if planning was based on patient dependent mechanics and the patient failed to respond accordingly.

Extractions and Facial Aesthetics

One of the main indications for orthodontic extractions is to achieve a more harmonious profile in patients with excessive facial convexity secondary to dental biprotrusion. It must be noted, in this regard, that the concept of the ideal profile has changed notably throughout the last century. Several decades ago, the ideal Caucasian profile was flat or even slightly biretrusive, with relatively thin lips; while in recent times more convex profiles have become more popular with a marked lip relief and a wide smile with buccal corridors [10]. This change in tastes for greater facial convexity is mainly for women and in Caucasians; whereas in the male and in oriental races, the flat pro- file is still considered more harmonious. Obviously, this is not the case in Negroid races, one of whose most characteristic features is precisely biprotrusion [11].

The greater tolerance to convexity in our environment has naturally reduced the need for extractions due to biprotrusion and DOD. For example, Proffit performed a study on the changes in the pattern of extractions in the treatment of malocclusions during the last 60 years. It showed that the frequency of extractions was around 30% for the years 1953 and 1993: 40 years apart.

However, interestingly, the analysis in 1968 gave a result of 76%. The explanation given for this high percentage was the trend at the time for removing all teeth outside of the arch. At present, this pro- portion is limited to 5%; 20% down on most studies [12].

However, there are some facial features linked to excessive convexity which are objectionable in any aesthetic framework and put a limit on the extraction option. One of those features is the hyperactivity of the muscles of the chin associated with biprotrusion which, in an effort to close the lips, gives the chin a kind of "golf ball" appearance [13].

The positive effect on the profile of extracting the bicuspids in patients with a normal vertical dimension or a little short and a marked biprotrusion, especially if associated with crowding, is generally clear; thus, there is usually agreement among authors for its indication [14]. This does not occur in the biprotrusive patients with a pattern of mandibular poster rotation and dolicofacial growth. The aesthetic result in these patients of resolving biprotrusion with extractions is unpredictable, if not clearly wrong; so, the clinician is often faced with the choice of obtaining good occlusion at the risk of worsening facial aesthetics, or not altering the profile and accepting the limitations in the resolution of the malocclusion. Obviously, in cases where the dentofacial deformity is more severe, orthognathic surgery allows for both goals, facial and occlusal [15].

Materials and Methods

This survey was conducted among hundred patients who reported to the dental hospital for orthodontic treatment. A questionnaire containing twenty questions related to orthodontic tooth extraction was formulated. Oral examination such as presence of the malocclusion, type of anterior discrepancy such as proclination, crowding, spacing, and presence of impacted tooth, retroclination, and anterior cross bite was included. The treatment plan was explained to the patients and they were requested to complete the questionnaire. Finally, their opinion for extraction of tooth is concluded. If the patient is willing for extraction they were further investigated whether they have any choice to tooth extraction. If the patient hesitates to extract, they were questioned for reasons. Some reasons such as anxiety, pain and discomfort, complications or any health problems. The results of hundred patients are concluded by statistics method.

Result

Among 100 patients 70%of patient accepted for tooth extraction in accordance to treatment plan .30%people refused for extraction due to factors like due to anxiety, discomfort, space, refuse to extract original tooth, fear of loose vision and some health problems.

Table 1: It shows no of patients not willing for treatment due to various reasons ,83.9% avoid extraction due to anxiety,41.9% due to discomfort,19.4% due to fear due to spacing,74.2 due to fear to lose original tooth,45.2% due to health related problems.

Discussion

Tooth arch discrepancy should be evaluated in both the upper and lower arches. But for diagnostic purposes, the lower arch is a priority because of greater difficulty in obtaining space.

When orthodontists are faced with a marked negative tooth-arch discrepancy (TAD) in the lower arch, they will be hard pressed to treat the patient by performing tooth extractions. Small negative discrepancies can, in most cases, be treated without extractions [16]. Thus, space can be obtained by using leeway space (if still possible), stripping, correction of pronounced mesial tipping of lower posterior teeth and small expansions and/or protrusions with the goal of restoring normal tipping to the lower teeth, especially if accompanied by rapid maxillary expansion (RME)[17].

The clinical case 1 illustrates the situation of using leeway space to avoid extractions. The 9-year-old patient had a negative discrepancy in the upper and lower arches. To solve this case, we could choose for upper and lower premolar extractions. Although the profile was slightly convex, we opted for treatment using leeway space in the lower arch, placement of lingual arch during the mixed dentition and rapid maxillary expansion in the upper arch [18]. With this therapeutic approach, we achieved tooth alignment without the need to perform extractions and obtained a straight profile, which probably would have been in worse shape if the case had been conducted with tooth extractions [19].

Another situation typical of negative discrepancy cases is when the need arises to perform tooth extractions but no changes can be made to the facial profile. In the clinical case 2, the patient's facial profile was straight with negative discrepancy in the upper and lower arches and asymmetry in the lower arch with lower midline shift to the right. To solve this case, we chose to extract three premolars (14, 24 and 34). To avoid excessive retraction of anterior teeth towards lingual and deepening of the profile, we used resistant torque in the upper and lower teeth during retraction and avoiding incisor up- righting. The result at the end of treatment was dental harmony in the existent space, with maintenance of the facial profile. Zero or positive model discrepancies require that treatment be performed without extractions, unless the patient has some other associated problem that indicates extraction.

In a clinical study they assessed Some pathologies play a key role in defining orthodontic treatment planning. Patients can have half-formed teeth, ageneses, ectopias, abnormal shapes or even carious processes, and endodontic lesions that indicate tooth extraction. During diagnosis these conditions should be considered as they may change in certain situations the choice of the tooth or teeth to be extracted.

In patients with an indication for premolar extraction due to a sharp negative model discrepancy, but with extensive decay in the first permanent molars, these teeth are a viable extraction alternative for the premolars. In asymmetric malocclusions, where only one tooth must be extracted, if the patient happens to have an anomalous tooth, this tooth should be selected for extraction. Many other pathological conditions such as cysts, abnormal roots and periodontal problems indicate the extraction of teeth. Thus, the different pathologies greatly contribute to orthodontic treatments involving extraction.

Clinical case 8 is of a female 10 year-old patient and illustrates the importance of pathologies in deciding which tooth to extract. She was in the mixed dentition phase and had an Angle Class I malocclusion, 3 mm anterior open bite, mouth breathing, upper midline shifted due to a missing tooth and skeletal Class II relationship. The maxilla was slightly contracted with no crossbite and she had a 6 mm lower arch model discrepancy.

An analysis of the lateral radiograph. Showed skeletal Class II (ANB = 6o), vertical facial growth pattern (SNGoGn = 42o and Y axis-SN = 74o), upper incisors retroclined (1. NA = 16o)

and linguoversion (1-NA = 3 mm) and lower incisors protruding and in labioversion (1. NB = 29o and 1-NB = 5 mm), although the latter were well established in the mandible (IMPA = 89°). The profile was straight (S-UL=+1/S-LL=+1).

In malocclusions with skeletal discrepancies it is crucial for the diagnosis and prognosis of the case to check whether the patient is still undergoing significant facial growth. Maximum pubertal growth spurt occurs approximately at around 11-12 years in girls and 13-14 years in boys, subject to individual variations. The most widely used method for assessing skeletal age is through a hand and wrist radiograph, by analyzing the size of the epiphyses relative to the diaphyses. If a patient is in his/her development period it is not possible to correct a skeletal dysplasia with the use of appliances that produce orthopedic effects.

If a malocclusion can be corrected with growth response (growth redirection), clinicians can handle the case without extractions.

In a case with these characteristics. We achieved skeletal and dental correction using headgear associated with a fixed orthodontic appliance. Initially, this 11 year-old patient had a convex profile, Skeletal Class II (ANB = 8o), Angle Class II, division 1, 2 mm lower TAD, 8 mm overjet, 5% overbite, well positioned upper incisors (1.SN = 101o), protruding lower teeth (IMPA = 99) and in- creased lower facial third. As an aggravating factor, the patient had a thumb-sucking habit, mouth breathing and a predominantly vertical resultant growth (SN.GoGn = 40o).

In this case, we opted for the use of combined pull headgear with a greater vertical component to correct the Class II by differential anterior displacement of the mandible (due to growth) associated with the use of Class III elastics to reposition the lower incisors.

Proffit and Fields developed a guide of contemporary procedures for evaluating extraction in Class I cases with crowding and/ or protrusion. The authors reported that in negative lower arch discrepancies below 4 mm tooth extraction is rarely required, except in cases of incisor protrusion or posterior vertical [20].

Table 1: Chi-Square test to compare proportions

Factors affecting tooth extraction	Q12. Willing for extraction if required						P-Value
	Yes		No		Total		
Q03. Gender	N	Row %	N	Row %	N	Row %	0.234
Male	31	75.6	10	24.4	41	100.0	
Female	38	64.4	21	35.6	59	100.0	
Total	69	69.0	31	31.0	100	100.0	
Q04. Occupation							
Student	51	72.9	19	27.1	70	100.0	0.315
Working	11	55.0	9	45.0	20	100.0	
Housewife	7	70.0	3	30.0	10	100.0	
Total	69	69.0	31	31.0	100	100.0	
Q07. Malocclusion							
Yes	69	69.7	30	30.3	99	100.0	
No	0	.0	1	100.0	1	100.0	0.310
Total	69	69.0	31	31.0	100	100.0	
Class I							
Yes	48	69.6	21	30.4	69	100.0	

No	21	67.7	10	32.3	31	100.0	0.855
Total	69	69.0	31	31.0	100	100.0	
Class II							
Yes	21	80.8	5	19.2	26	100.0	
No	48	64.9	26	35.1	74	100.0	0.131
Total	69	69.0	31	31.0	100	100.0	
Division I							
Yes	14	77.8	4	22.2	18	100.0	
No	55	67.1	27	32.9	82	100.0	0.374
Total	69	69.0	31	31.0	100	100.0	
Division II							
Yes	7	87.5	1	12.5	8	100.0	
No	62	67.4	30	32.6	92	100.0	0.429
Total	69	69.0	31	31.0	100	100.0	
Class III							
Yes	0	.0	4	100.0	4	100.0	
No	69	71.9	27	28.1	96	100.0	0.008
Total	69	69.0	31	31.0	100	100.0	
Proclination							

Yes	18	78.3	5	21.7	23	100.0	
No	51	66.2	26	33.8	77	100.0	0.274
Total	69	69.0	31	31.0	100	100.0	
Crowding							
Yes	39	76.5	12	23.5	51	100.0	0.099
No	30	61.2	19	38.8	49	100.0	
Total	69	69.0	31	31.0	100	100.0	
Spacing							
Yes	10	52.6	9	47.4	19	100.0	
No	59	72.8	22	27.2	81	100.0	0.087
Total	69	69.0	31	31.0	100	100.0	
Q08. Why interested in orthodontic treatment							
Recommended by doctor	36	64.3	20	35.7	56	100.0	0.250
Recommended by friends	33	75.0	11	25.0	44	100.0	
Total	69	69.0	31	31.0	100	100.0	
Q09. Treatment plan explained prior to starting the treatment							
Yes	69	69.0	31	31.0	100	100.0	–

No	0	.0	0	.0	0	.0	
Total	69	69.0	31	31.0	100	100.0	
Q10. Have undergone extraction before							
Yes	37	82.2	8	17.8	45	100.0	0.010
No	32	58.2	23	41.8	55	100.0	
Total	69	69.0	31	31.0	100	100.0	
Q11. Have knowledge on the extraction process							
Yes	40	65.6	21	34.4	61	100.0	0.354
No	29	74.4	10	25.6	39	100.0	
Total	69	69.0	31	31.0	100	100.0	

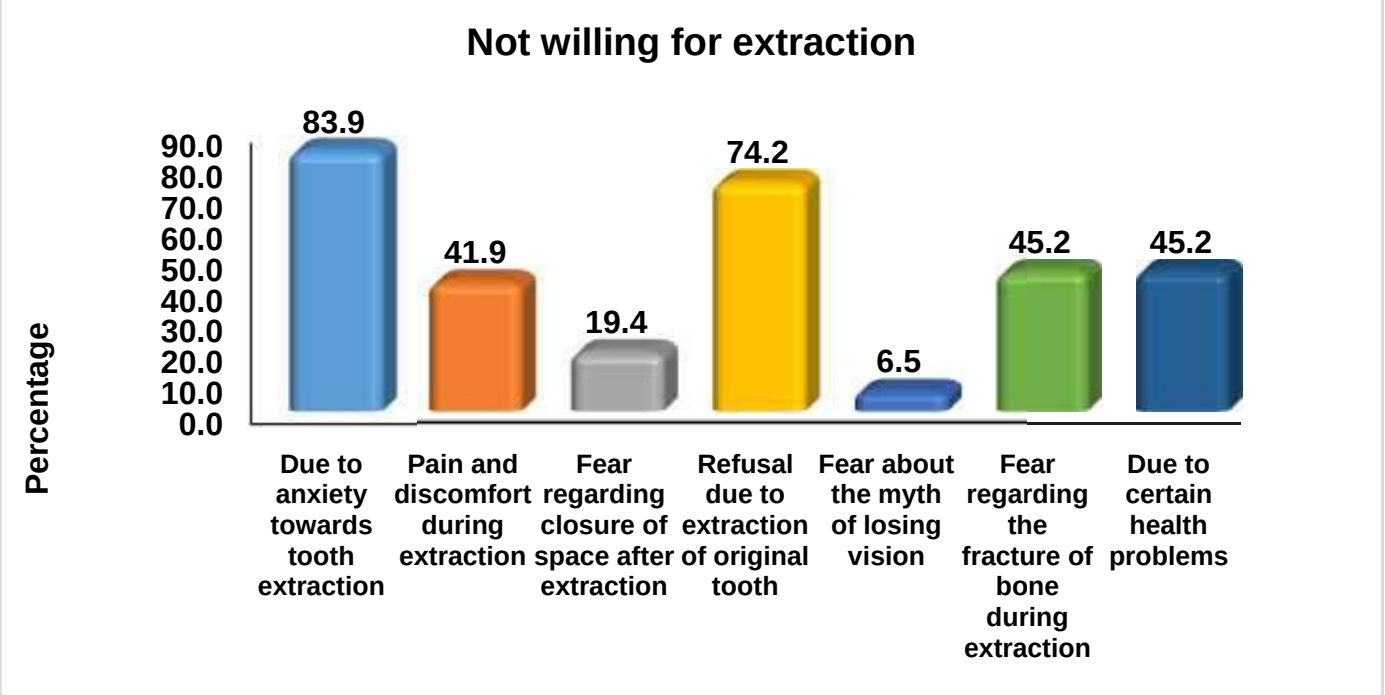
		Q12. Willing for extraction if required					
		Yes		No		Total	
		N	Col %	N	Col %	N	Col %
Q13. In accordance to the treatment plan	Yes	69	100.0	0	.0	69	100.0
	No	0	.0	0	.0	0	.0
	Total	69	100.0	0	.0	69	100.0
Q13Y. Choice to tooth extraction	Yes	23	33.3	0	.0	23	33.3
	No	46	66.7	0	.0	46	66.7
	Total	69	100.0	0	.0	69	100.0

Q14. Due to anxiety towards tooth extraction	Yes	0	.0	26	83.9	26	83.9
	No	0	.0	5	16.1	5	16.1
	Total	0	.0	31	100.0	31	100.0
Q15. Pain and discomfort during extraction	Yes	0	.0	13	41.9	13	41.9
	No	0	.0	18	58.1	18	58.1
	Total	0	.0	31	100.0	31	100.0
Q16. Fear regarding closure of space after extraction	Yes	0	.0	6	19.4	6	19.4
	No	0	.0	25	80.6	25	80.6
	Total	0	.0	31	100.0	31	100.0
Q17. Refusal due to extraction of original tooth	Yes	0	.0	23	74.2	23	74.2
	No	0	.0	8	25.8	8	25.8
	Total	0	.0	31	100.0	31	100.0
Q18. Fear about the myth of losing vision	Yes	0	.0	2	6.5	2	6.5
	No	0	.0	29	93.5	29	93.5

	Total	0	.0	31	100.0	31	100.0
Q19. Fear regarding the fracture of bone during extraction	Yes	0	.0	14	45.2	14	45.2
	No	0	.0	17	54.8	17	54.8
	Total	0	.0	31	100.0	31	100.0
Q20. Due to certain health problems	Yes	0	.0	14	45.2	14	45.2
	No	0	.0	17	54.8	17	54.8
	Total	0	.0	31	100.0	31	100.0

Graph 1: It shows no of patients willing for extraction of tooth according to treatment plan,it shows 33.3% of patients are willing for extraction

Graph 2: It shows no of patients not willing for treatment due to various reasons ,83.9% avoid extraction due to anxiety,41.9% due to discomfort,19.4% due to fear due to spacing,74.2 due to fear to lose original tooth,45.2% due to health related problems.



Questionarrie format:

Patients acceptance for orthodontic tooth extraction

1)Name:

2)Age:

3)Sex:

4)Occupation:

5)Income:

6) Chief complaint:

7) Malocclusion:Yes / No

Angle's Class I / Class II- Division I/DivisionII. /Class III

Type of anterior discrepancy:

Proclination/Crowding/Spacing/Presence of impacted tooth/Retroclination/Anterior cross bite

8) Why were you interested in orthodontic treatment?

9) Was the treatment plan explained to you prior to starting the treatment : Yes/No.

10)Have you undergone extraction before: Yes/No

11) Do you have any knowledge on the extraction process : Yes/No

12) Are you willing for extraction if required: Yes/No

If yes:

13)In accordance to the treatment plan: Yes/No

Choice to tooth extraction: Yes/No

If yes:

Lower incisor:

First premolar:

Second premolar:

Lingually blocked upper lateral canine:

Severely blocked out canine:

Figure 1: Questionnaire for assessing patient acceptance for orthodontic extraction

CONCLUSION:

Due to the process of the evolution there is a great incidence of arch length tooth size discrepancy. This survey was conducted among hundred patients who reported to the dental hospital for orthodontic treatment. A questionnaire containing twenty questions related to orthodontic tooth extraction was formulated in different types of malocclusion. This necessitates the need for tooth extraction to gain space. However, patient reporting orthodontic practice are not aware of same. Hence this questionnaire is formulated to know the patient response to tooth extraction as part of orthodontic treatment. This study concluded that patient acceptance and co-operation towards diagnosis and proposed treatment planning is very essential for successful treatment outcome.

Corresponding Author

Asha Ramesh

Senior Lecturer, Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamalle High Road, Chennai 600077,Tamil Nadu, India

Mobile Number: +91 9841722113

Email Id:doc.ash4587@gmail.com

References

1. Ngan P, Yiu C, Hu A, Hägg U, Wei SH, Gunel E. Cephalometric and occlusal changes following maxillary expansion and protraction. The European Journal of Orthodontics. 1998 Jun 1;20(3):237-54.
2. Timms DJ. The burden of proof: a critical review of orthodontic claims made by some general practitioners. American journal of orthodontics and dentofacial orthopedics: official publication of the American Association of Orthodontists, its constituent societies, and the American Board of Orthodontics. 1995 Jul;108(1):17A.
3. McNamara Jr JA, Bookstein FL, Shaughnessy TG. Skeletal and dental changes following functional regulator therapy on Class II patients. American Journal of Orthodontics. 1985 Aug 1;88(2):91-110.
4. Richardson ME. Lower molar crowding in the early permanent dentition. The Angle Orthodontist. 1985 Jan;55(1):51-7.
5. Aras A. Vertical changes following orthodontic extraction treatment in skeletal open bite subjects. The European Journal of Orthodontics. 2002 Aug 1;24(4):407-16.
6. Bernstein L, Edward H. Angle versus Calvin S. Case: extraction versus nonextraction. Historical revisionism. Part II. Am J Orthod Dentofacial Orthop. 1992;102(7):546-61.
7. Boley JC, Pontier JP, Smith S, Fulbright M. Facial changes in extraction and nonextraction patients. The Angle Orthodontist. 1998 Dec;68(6):539-46.

8. Burstone CJ. Diagnosis and treatment planning ofpatients with asymmetries. InSeminars in orthodontics 1998 Sep 1 (Vol. 4, No. 3, pp. 153-164). WB Saunders.
9. Camargo ES, Mucha JN. Moldagem e modelagem em Ortodontia. Rev. dent. press ortodon. ortop. maxilar. 1999;4(3):37-50.
10. Ruellas AC, Ruellas RM, Romano FL, Pithon MM, Santos RL. Tooth extraction in orthodontics: an evaluation of diagnostic elements. Dental Press Journal of Orthodontics. 2010 Jun;15(3):134-57.
11. Kusnoto J, Kusnoto H. The effect of anterior tooth retraction on lip position of orthodontically treated adult Indonesians. American Journal of Orthodontics and Dentofacial Orthopedics. 2001 Sep 1;120(3):304-7.
12. Dhiman S, Maheshwari S. A dilemma in orthodontics: Extractions in borderline cases. J Adv Clin Res Insights. 2015 Jan;2:36-9.
13. Massahud NV, de Souza Totti JI. Estudo Cefalométrico Comparativo das Alterações no Perfil Mole Facial Pré e Póstratamento Ortodôntico com Extrações de Prémolares. Jornal Brasileiro de ORTODONTIA & Ortopedia Facial. 2010 Aug 20;9(50).
14. Kokich VG, Shapiro PA. Lower incisor extraction in orthodontic treatment: four clinical reports. The Angle Orthodontist. 1984 Apr;54(2):139-53.
15. Ruellas AC, Ruellas RM, Romano FL, Pithon MM, Santos RL. Tooth extraction in orthodontics: an evaluation of diagnostic elements. Dental Press Journal of Orthodontics. 2010 Jun;15(3):134-57.
16. de Oliveira Ruellas AC, de Oliveira Ruellas RM, Romano FL, Pithon MM, dos Santos RL. Tooth extraction in orthodontics: an evaluation of diagnostic elements. LILACS-1998 BBO-1998 National Library of Medicine-1999 SciELO-2005. 2010 Jun.
17. Ramos AL, Tatsuei Sakima M, dos Santos Pinto A, Bowman SJ. Upper lip changes correlated to maxillary incisor retraction—a metallic implant study. The Angle Orthodontist. 2005 Jul;75(4):499-505.
18. Roberts CA, Subteiny JD. An american board of orthodontics ease report, use of the face mask in the treatment of maxillary skeletal, retrusion. American Journal of Orthodontics and Dentofacial Orthopedics. 1988 May 1;93(5):388-94.
19. Rauch ED. Torque and its application to orthodontics. American Journal of Orthodontics. 1959 Nov 1;45(11):817-30.

20. Talass MF, Tollaae L, Baker RC. Soft-tissue profile changes resulting from retraction of maxillary incisors. American Journal of Orthodontics and Dentofacial Orthopedics. 1987 May 1;91(5):385-94.

Patients Knowledge and Attitude in Endodontic Treatment-A Questionnaire Based Study

Priyadharshni S[1], Nivethithan[2], E. Roja[3]

Graduate Student[1], Tutor[2], Department of Public Health Dentistry,Tutor[3], Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: The basic aspect of modern endodontics is painlessness and effectiveness of the treatment of diseased dental pulp. The disease of dental pulp is mainly caused due to dental caries. Dental caries is a common chronic disease prevalent all over the world. It is a microbiologic disease which manifests as demineralization of hard tissues of the tooth. If detected earlyits management can be done easily via conservative techniques like restorations. As it progresses to involve the vascular tissue of the tooth it may cause symptoms such as pain and swelling. This leads patients to seek dental care. Additionally, dental decay may cause tooth loss which can reduce the ability to eat fibrous foods, leading to dietary deficiencies. Root Canal Treatment is a procedure that uses biologically acceptable chemical and mechanical treatment in the root canal system to eliminate pulpal and peri-radicular diseases and to promote healing and repair of peri-radicular tissue.The aim of this research is to study the patients knowledge and attitude in Endodontic treatment.

Material and Method: A total of 13 questions was prepared and distributed to 100 patients in Saveetha Dental College which included to record patients knowledge and attitude towards root canal therapy.

Result: 68% participants responded positively, but when asked in detail what root canal treatment does and whether it is a complicated the responses were mixed and majority didn't know or were not sure.

Conclusion: From the self-prepared questionnaire and based on the responses acquired, it can be seen that on an average 58% of responses for all fifteen questions turned out to be positive and patients who had prior knowledge about root canal treatment were interested in proceeding post endodontic treatments to increase the success of RCT

Key Words:*Endodontics, knowledge, treatment, root canal, crown*

Introduction

Endodontics is the branch of dentistry that deals with diseases of the tooth root, dental pulp, and surrounding tissue in human. It is a profession based on the work with other people, so several factors should be considered during clinical decision-making process. The process of clinical decision-making is the essence of everyday clinical practice. This process involves an interaction of application of clinical and biomedical knowledge, problem-solving, weighing of probabilities and various outcomes, and balancing risk-benefit. Although most clinical deci- sions are based on 'traditional' clinical criteria, they are also influenced by a wide range of non-clinical factors. Non-clinical influences on clinical decision-making affects medical decisions and it includes patient-related factors such as socioeconomic status, quality of life and patient's expectations and wishes, physician-related factors such as personal characteristics and interaction with their professional community, and features of clinical practice such as private versus public practice as well as local management policies.

Endodontic treatment is performed to save the natural tooth. Most of the dentists agree that there is no substitute for healthy and natural teeth in spite of many advanced restorations. Publications on patient's awareness of endodontic procedures are rarely presented. Patients perceived problems related to endodontically treated teeth are an important consideration for all dental practitioners. Contemporary endodontics is developing continuously. It concerns not only the instruments, but also the treatment procedures. The basic principle of modern endodontics painlessness and effectiveness of the treatment (1,2). The conviction of difficulties related to root canal treatment, including pain associated with it has been functioning among patients. It raises many concerns, which often lead to cancelling the visits to the dental office(3,4).Many patients admit that they feel the stress related with the planned endodontic treatment. In all professions based on the work

with other people, understanding of motivational processes and skills of using knowledge is very important.(5,6).

In dentistry, RCT is the most feared procedures of all treatments. Dentist asserts that modem root canal treatment is relatively painless because pain can be controlled. Major setback of root canal treatment is that most patients lack the knowledge. They do not know what root canal treatment entails and what the benefits of such treatment are. Most patients fear root canal treatment because it is painful.(7)

Indications of root canal treatment are irreversible pulpitis, periapical periodontitis, crown fractures involving the pulp, acute per apical abscess, when want to create post space, construction of an over denture and when the vitality of pulp is doubtful. Root canal treatment is contraindicated in patient with poor oral hygiene, poorly motivated patients, patients with poor general health, and the very old and in patients with infective endocarditits.

Material and Method

A self-administered questionnaire was prepared and distributed to 100 patients in Saveetha Dental College (convenience sampling) during their regular dental visits in order to assess the knowledge and attitude towards endodontic treatment. A total of 13 questions were included to record patients knowledge and attitude towards root canal therapy. These ranged from personal details such as name, age etc to specific questions relating to awareness about root canal therapy, perceived barriers to undergo the same and sources of knowledge regarding Endodontics.The findings of this study will be used to come up with strategies to improve knowledge and practice of patients towards endodontic treatment.

1.Are you aware of Root Canal Treatment (RCT)?
A) Yes
B) No
C) Not sure
2.Do you know that RCT is a treatment involving cleaning and restoring up to the root of the
tooth
A) Yes
B) No
C) Not sure
3.Do you think RCT is a complicated procedure?
A) Yes
B) No
C) Not sure
4.Have you undergone RCT procedure?
A) Yes
B) No
C) Not sure
5.Was it painful during treatment?
A) Yes
B) No
C) Not sure
6.Do you know how long will take it to finish the RCT procedure?
A) Yes
B) No
C) Not sure
7.Would you prefer root canal treatment to tooth extraction?
A) Yes
B) No
C) Not sure
8.Do you know the price of RCT procedure?
A) Yes
B) No
C) Not sure
9.Do you know that procedures like permanent filling or crowns after root canal treatment determine the success of RCT done?
A) Yes
B) No
C) Not sure
10.Do you intend to continue treatment procedures after RCT
A) Yes
B) No
C) Not sure
11.What do you do in case of toothache?
A) Go to a dentist
B) Use home remedies or pain killers
C) Not sure
12.How do you know about endodontic treatment?
A) Through Media or internet
B) Parents or friends
C) Dentist
D) Don't know
13.Why do you think root canal treatment is necessary?
A) Toothache or Broken tooth
B) Change in tooth colour
D) Not sure

Figure 1: Questionnaire

Results

The study records based on the questionnaire showed that 90 out of 100 patients contacted regarding the study responded positively and agreed to participate. Among the 90 patients, 22 patients were of 18-24 age group, 34 patients were of 25-30 age group, 19 of them were 31-40 age group, 15 of them 41-60 age group of the total participants.

Table 1: Awareness of Root canal treatment.

Responses	Number Out Of 90 Participants
Yes	61
No	15

Out of 90 participants, 61(68% of 90 participants) respondents were aware that of root canal treatment, other 15(17% of 90 participants) of them were not aware and 14(15%)of them were not sure.

Table 2: Response for RCT treatment involving cleaning and restoring up to the root of the tooth.

Responses	Number Out Of 90 Participants
Yes	31
No	42

31 (34%) participants responded positively , 42(47%) didn't know and the remaining 17(19%) were not sure.(Tab.2)

Table 3: Patients response towards RCT procedure.

Responses	Number Out Of 90 Participants
Yes	30
No	19

30 (33%) out of 90 participants think RCT is complicated procedure and 19(21%) of them responded that RCT is not a complicated procedure and 41(46%) of them did not know about it.(Tab.3)

Table 4: Patients who have undergone RCT procedure.

Responses	Number Out Of 90 Participants
Yes	43
No	10

43 out of 90(48%) participants responded that they have undergone RCT procedure, 10(11%) of them did not undergo RCT procedure and 37(41%) of them were not sure if they had undergone RCT procedure. (Tab.4)

Table 5: Patients response during treatment.

Responses	Number Out Of 90 Participants
Yes	25
No	18

25 out of 43(28%) participants who underwent RCT procedure felt that RCT was painful, 18(20%) of them felt it was not painful.

Table 6: Patients knowledge about the duration of the procedure

Responses	Number Out Of 90 Participants
Yes	31

No	42

Out of 90 participants, 31(34%) of them responded that they know how long will it take to finish RCT procedure, 42(47%) of them agreed that they do not know about the time taken to finish the RCT procedure and 17(19%) of them were not sure about it.

Table 7: Patients preference on root canal treatment.

Responses	Number Out Of 90 Participants
Yes	30
No	21

More than 44% of the respondent preferred root canal treatment and 23% consider extraction as the ultimate method. The remaining 40% had doubts about their preference. (Tab.7)

Table 8: Patients response about the cost of RCT procedure.

Responses	Number Out Of 90 Participants
Yes	28
No	22

Twenty-eight (31%)of respondents out of 90 participants admitted to knowing the price of root canal treatment(Tab.8).

Table 9:Patients success rate on permanent filling or crowns after root canal treatment.

Responses	Number Out Of 90 Participants
Yes	23
No	19

Table 9 shows that 23 (26%) of 90 participants knew that procedures like permanent filling or crowns after root canal treatment are mandatory to determine the successof RCT .

Table 10: Patients response on continuing procedures after RCT.

Responses	Number Out Of 90 Participants
Yes	26
No	19

Out of 90 participants, 26(29%) respondents said that they intend to continue treatment procedures after RCT (Tab.10)

Table 11: Patients response in case of toothache

Responses	Number Out Of 90 Participants
Go To A Dentist	60
Use Home Remedies Or Pain Killers	20

Tab.11 shows the behaviour of patients in case of a sudden, severe toothache. The majority of patients 67% would go immediately to a dentist after the first symptoms and 22% of respondents admit to using the “home remedies” to combat ailments.

Table 12: Patients knowledge on endodontic treatment.

Responses	Number Out Of 90 Participants
Through Media Or Internet	49
Parents Or Friends	18
Dentist	9
Don’t Know	14

Tab.12 shows about the knowledge of the patient about endodontic treatment. Out of 90, 49(54%) of the participants responded that they knew endodontic treatment through media or internet , 18(20%) knew from friends and family, remaining 9(10%)through dentist and 14(26%) participants did not know.

Table13: Patients opinion on Root canal treatment.

Responses	Number Out Of 90 Participants
Toothache Or Broken Tooth	60
Change In Tooth Colour	23

Tab.13 shows attitude of the respondents. Out of 90 participants, 60(66%)of them responded that RCT is necessary in case of toothache or broken tooth, 23(26%)of them responded that RCT is necessary in case of change in tooth color.

Discussion

The study was conducted based on a self-prepared questionnaire circulated among 100 patients visiting Saveetha dental college, Chennai. Out of 100, 90 patients answered the questionnaire and 10 patients were not interested to answer the questionnaire. There are 15 direct questions focused on the awareness and attitude towards endodontic procedures in the participants. Majority of participants belonged to the age group 25-30 years (38%). When asked pertaining to their awareness maximum (68%) participants responded positively, but when asked in detail what root canal treatment does and whether it is a complicated the responses were mixed and majority didn't know or were not sure(Tab.1). The participants were then questioned about their previous experiences with root canal treatment, a maximum of 48% participants had prior experience, and about 28% responded they didn't have pain during the procedure and 20% had pain during treatment. When asked about the duration to complete root canal treatment and would they prefer it over extraction of tooth, only 33% - 34% responded positively and majority had doubts. To the next questions about the cost, success and further treatment, about 25% - 31% responded positively that they knew about the cost and were aware and willing to follow up treatments like permanent restoration and

crowns to improve the success rate, but still a majority didn't know the cost and further treatment options. A large percentage of participants 67% responded that they would go to a dentist when there is incidence of pain and remaining 33% chose either home remedies or were not sure. About 84% of participants had prior interest in knowing about endodontics either through media, friends, family and dentists; majority of participants believed root canal treatment is necessary in case of broken tooth or pain associated with the tooth.

The main aim of a Root canal treatment is the complete removal of the connective tissue and the destruction of residual microorganisms found in infected root canals and an effective seal in order to prevent recolonization of the root canal system with bacteria. Thus the primary endodontic treatment goal must be to ensure root canal disinfection and to prevent re-infection. Irrigating solutions play a very large role in disinfecting the root canals. To remove smear layer and disinfect canals are the main objective of cleaning and shaping. Today, use of laser to remove smear layer and to disinfect root canals has increasingly attracted the attentions. (8)The complexity of the root canal system, presence of numerous dentinal tubules in the roots, invasion of the tubules by microorganisms, formation of smear layer during instrumentation and presence of dentin as a tissue are the major obstacles in achieving the primary objectives of complete cleaning and shaping of root canal systems.(9)

In recent years, endodontics has been one of the fastest growing areas of dentistry. The use of modern tools, rubber dam, and the microscope in daily practice, significantly influence the quality and durability of treatment effects. All endodontic procedures should be performed with a rubber dam, which should be considered standard care. The purpose of rubber dam protection in dentistry on whole cannot be overemphasized (10). Patients more often do not identify the endodontic treatment as the most unpleasant procedure. Endodontic procedures with associated pain is felt as moderate, rarely sharp. In addition, patients can benefit from many forms of local anesthesia, and the use of nitrous oxide, which according to recent reports, enhances the effect of their actions. Another form of premedication are preoperative intravenous sedatives and analgesics. In extreme cases, general anesthesia is the alternative. A study conducted by Klages et al. demonstrates that patients visiting the dental office expect pain usually larger than that actually experiencing during treatment [11].One of the fastest growing areas of dentistry in the recent years is observed to be endodontics. The quality and

durability of the treatment has become more effective after the use of Modern tools (the rotary nickel-titanium files which has enabled a quicker root canal preparation, as these instruments produce a uniformly tapered canal configuration with minimum canal transportation and also reduces the incidences of periradicular irritation and post-operative discomfort but only about one fifth of respondents use these rotary instruments in root canal preparation whereas the others use Non-rotary manual endodontic files), rubber dam, and microscope in daily practise. (12)

The conviction of difficulties related to root canal treatment, including pain associated with it has been functioning among patients. It raises many concerns, which often lead to cancelling the visit to the dental office. Many Patients admit that they feel the stress related with the planned endodontic treatment. Publications on patients' awareness of this type of procedures are rarely presented. Contemporary endodontics is developing continuously. It concerns not only the instruments, but also the treatment procedures. The basic principle of modern endodontics is painlessness and effective of the treatment. There are many methods providing the patient with comfort by reducing pain during treatment: local and conduction anesthesia, nitrous oxide or even general anesthesia. Successful RCT depends not only on specific factors like root canal infection, complexity of root canal morphology, etc. Majority of RCT in India is provided by general dental practitioner. Various investigations are therefore, carried out to explore the standard of RCT carried out by them.The antibiotics should be used judiciously for well defined indications in endodontics. The antibiotics use should only be considered as an adjunct to non surgical endodontic therapy. At the end the dentists should have a sound knowledge about the endodontic indications for the prescription of antibiotics to restrict and prevent the misuse and overuse of antibiotics.(13)

According to the study done bySisodia N et al, it indicates that most of the barriers reported to acceptability of root canal therapy could be overcome by patient education and creating increased general awareness about oral health and different dental procedures through school dental health education programs.(14) As the dentist is the most common source of information for most patients, the onus of appropriate dissemination of knowledge lies with them. This can be done most effectively in the Dental office by placing pictoral pamphlets which describe RCT, crowns in the waiting area. Non-operating office staff can additionally

help in increasing awareness by explaining the importance of retaining and replacing teeth while the patient is in the waiting area.(15,16)

In all professions based on the work with other people, understanding of motivational processes and skills of using knowledge is very important. These factors are particularly important in everyday practice of a dentist(17). Properly conducted interview with the patient helps in deciding on the appropriate therapeutic treatment, especially with regard to endodontics, which often prevents tooth loss(18).Since the beginning of modern-day endodontics, there have been numerous concepts, strategies, and techniques for performing pain-free and reasonable cost root canal treatment. The level of patients' knowledge and awareness regarding root canal treatment may influence their decision-making and choice; it may be obstacle to get the treatment(19,20).

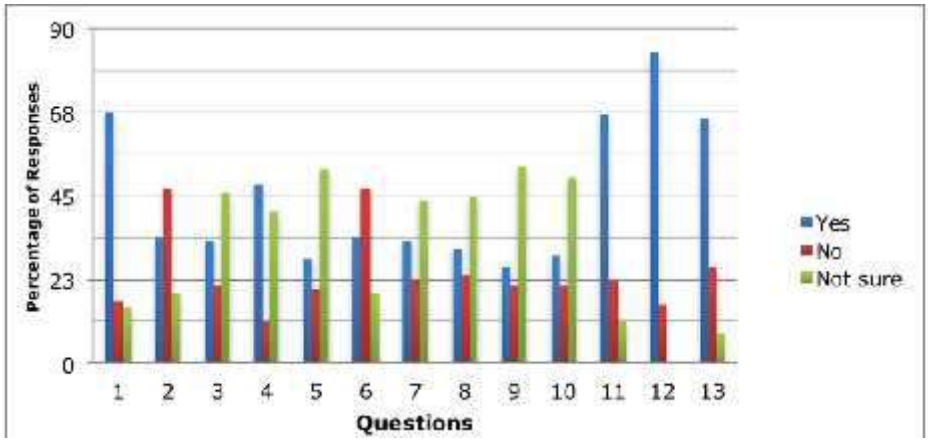


Figure 2: Representing patient's knowledge on Root Canal Treatment.

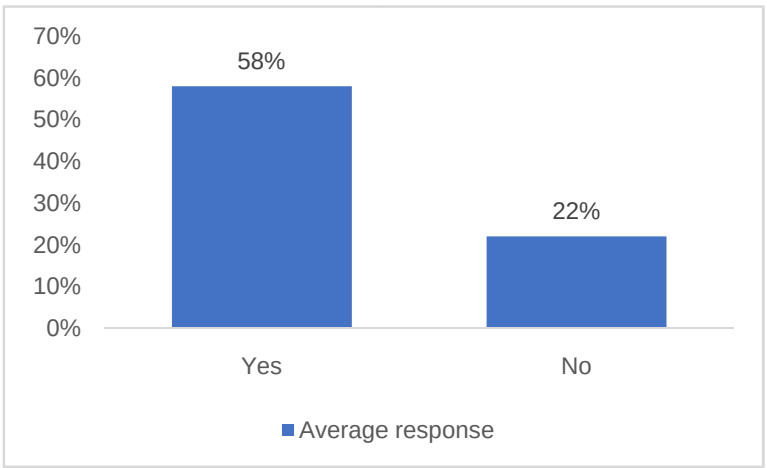


Figure3: Graph representing majority of 58% had prior experience, and 22% of the patients were not aware of the procedure, 30% were not sure.

Conclusion

From the self-prepared questionnaire and based on the responses acquired, it can be seen that on an average 58% of responses for all fifteen questions turned out to be positive and patients who had prior knowledge about root canal treatment were interested in proceeding post endodontic treatments to increase the success of RCT. The next major focus was the 30% of participants who responded doubtfully, steps can be taken to clear their doubts by means of explaining what and how Root Canal Treatment and other endodontic procedures done, it can be done during the diagnosis and treatment planning or even during dental camps and awareness programs conducted separately.

Corresponding Author

E. Roja,
Tutor,Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India
Email Id: itsrojaa@gmail.com
Contact Number: 91 99941 74480.

References

1. Hamedy R, Shakiba B, Fayazi S. Patient-centered endodontic outcomes, a Narrative Review. Iran Endod J. 2013;8(4):197-204.
2. Pak JG, White SN,Pain prevalence and severity before, during, and after root canal treatment: a systematic review. J Endod. 2011;37(4):429-38.
3. Georgelin-Gurgel M, Diemer F, Nicolas E. Surgical and nonsurgical endodontic treatment-induced stress. J Endod. 2009;35(1):19-22.
4. Bracha HS, Vega EM, Vega CB. Posttraumatic dental-care anxiety (PTDA): Is dental phobia a misnomer Hawaii Dent J. 2006;37(5):17-9.

5. Klages U, Ulusoy Ö, Kianifard S. Dental trait anxiety and pain sensitivity as predictors of expected and experienced pain in stressful dental procedures. Eur J Oral Sci. 2004; 112(6): 477-83.
6. Wellstood K, Wilson K, EylesJ Reasonable access' to primary care: assessing the role of individual and system characteristics. Health and Place 2006;12(2):121-30.
7. Hajjaj FM, Salek MS, Basra MKA, Finlay AY Non-clinical influences on clinical decision-making: a major challenge to evidence-based practice. Journal of royal Society of Medicine. 2010;103: 178–187.
8. Soumya Abraham et al Endodontic Irrigants: A Comprehensive Review /J. Pharm. Sci. & Res. Vol. 7(1), 2015, 5-9.
9. PreethaParthasarathy. Awareness and Knowledge of Dentists about Laser Technique Used in Endodontic Disinfection J. Pharm. Sci. & Res. Vol. 9(4), 2017, 476-479.
10. Sruthi Srinivasan,S.pradeep,The attitude of dentists towards the prescription of antibiotics during endodontic treatment- a questionaire based study,International Journal of Pharmacy & Technology;IJPT,Sep-2016,Vol. 8, Issue No.3,15895-15900.
11. Bamgartner JC. Antibiotics in endodontic therapy. Antibiotics and antimicrobial use in dental practice. Carol Stream 2001;143-57.
12. PoojaUmaiyal.M, Awareness of root canal treatment among people, Research journal of pharmacy and technology.vol.9,7:2016.0974-3618.
13. Jyothi,Pradeep, Practice of endodontic therapy among practitioners: a Questionnaire based study, International journal of pharmacy and technology,IJPT| Sep-2016 | Vol. 8 | Issue No.3 | 16546-16558.
14. Janczarek M, Cieszko-Buk M, Bachanek T, Chałas R Survey-based research on patients' knowledge about endodontic treatment. Polish Journal Public Health 2014;124(3): 134- 137.
15. SisodiaN,YadavS,NangiaT,SinghP,YadavM,SinghHP Dental Patients' Knowledge and Attitude towards Endodontics – A Survey. Journal of Pharmaceutical and Biomedical Sciences 2015;05(01): 80-83.
16. Eli I, Schwartz-Arad D, Bartal Y Anxiety and ability to recognize clinical information in dentistry. Journal of Dental Research 2008; 87.

17. Gatten DL, Riedy CA, Hong SK, Johnson JD, Cohenca N Quality of life of endodontically treated versus implant treated patients: a University-based qualitative research study, Journal of Endodontics 2011;37(7):903-9.
18. Butani Y, Weintraub JA, Barker JC Oral health-related cultural beliefs for four racial/ethnic groups: Assessment of the literature. BMC Oral Health 2008;15; 8:26.
19. N. Mehata, D. Raisingani, S.Gupta, M. Sharma. Endodontic Trends: Where We Are And Where We Should Be-A Survey Report. People's Journal of Scientific Research. 2013.Vol.6 (2) : 30-37
20. Magdalena Janczarek, mariacieszko-Buk, Teresa Bachnek, renaTachalasSurvey-based research on patients knowledge about endodontic treatment.Pol J Public Health 2014;124(3):134-137.
21. NehaSisodia, ShardaYadav,TanuNangia, Preetinder Singh, ManishaYadav, HP Singh. Dental patients knowledge and attitude towards Endodontics-A survey journey of Pharmaceutical and biomedical science 2015;05(01)80-83.
22. Armfield JM, Heaton LJ Management of fear and anxiety in the dental clinic: a review. Australian Dental Journal 2013;58:390- 407.
23. Armfield JM, Slade GD, Spencer AJ Dental fear and adult oral health in Australia. Community Dentistry and Oral Epidemiology 2009;37:220-230.
24. GullifordM, Figueroa-Munoz J, Morgan M, Hughes D, Gibson B, Beech R, Hudson M what does' access to health care' mean. Journal of Health Services Research and Policy 3002;7(3):186- 188.
25. Baumgartner JC, Smith JR. Systemic antibiotics in endodontic infections. Endodontic Microbiology 1st ed 2009;225-41.

Perception of Exodontia

N.S.Harini[1], Loganathan[2], Sankari M[3]

Graduate Student[1], Reader[2], Department of Oral Surgery[2],

Professor[3], Department of Periodontics,

Saveetha Dental College, Saveetha University,

Saveetha Institute of Medical and Technical Sciences.

Abstract

Aim: The aim of the study is to study and analyse the perception of exodontias (tooth extraction).

Objective: The objective of this study is to assess the most difficult tooth to extract, and the frequent complications faced by the dentists during dental extraction, the instruments used for extraction of upper and lower third molar, and the preference of antibiotics and analgesic by the dentists.

Materials And Methods: A Survey had been conducted among 50 dentists to assess the perception of exodontia. Questionnaire survey which includes 9 questions. The analysis was statistically analysed and represented with bar graph diagram.

Results: Among 50 dentists, 13 dentists(p value=0.069) that the most difficult tooth to extract according to them was upper left second molar and another 13 dentists (p value=0.069) opted that upper left third molar was the most difficult tooth to extract. Root fracture is the most common complication according the dentists conducted in the survey(p value=0.015). Cryers are mostly been selected during extraction of upper third molar(p value=0.126) and coupland elevators are mostly used for exodontia of lower third molar(p value=0.713). The normal blood glucose level range to select patients for extraction lies mostly on 70-110mg/dL where 41 dentists among 50 of them opted for this range(p value=0.152). The normal blood pressure for extraction tends to be 120/80-160/94mmHg where 23 dentists preferred in the survey(p value=0.620). The most preferred antibiotic in

therapeutic extraction is broad spectrum antibiotics where 33 dentists among 50 opted for this(p value=0.011). The most common alternative way preferred by the dentists when local anaesthesia does not work is through intra ligamental way where 25 dentists opted in this study(p value=0.035). Penicillin and metronidazole is the most preferred antibiotic in dento alveolar abscess where 34 among 50 dentists opted for this(p value=0.035).

Conclusion: Our study answers the difficulties and common questionable issues that arise in dental extraction. Awareness must be created about the medical issues like normal blood pressure range among dentists to be increased.

Keywords:

Exodontia, assessment, dentists, technique, instruments, anaesthesia, perception.

Introduction

Tooth extraction is one of the dental treatments which should be considered the last option. A decrease in the number of teeth may result in poor dietary habit and deterioration of quality of life[1]. The number of extracted teeth can serve as an indicator of socio-economic and oral hygiene level[2]. Extraction of permanent teeth is performed for several reasons including dental caries, periodontal disease, orthodontic reasons, impacted teeth, failed dental treatment, prosthetic indications and other reasons.

An understanding of the reasons why teeth are extracted is essential to improve oral health outcomes. A large number of cross-sectional studies have investigated for tooth loss in different countries. Dental caries was the main cause for tooth loss[3-9], but a few studies revealed that a greater proportion of tooth extractions were due to periodontal disease[10-12].Extraction of teeth is the commonest surgical procedure carried out in the dental surgery setting by the practicing dental surgeon[13]. From a historical perspective, dental extractions have been used to treat a variety of diseased conditions, and as a method of severe punishment or torture to obtain forced confessions from suspected criminals[13-15]. Also, before the advent of antibiotics, chronic tooth infections were sometimes linked to a variety of health-related problems and removal of such diseased tooth was therefore a common treatment option for various medical conditions[14]. Likewise, it was once a common practice

to remove the front teeth of institutionalized psychiatric patients who had a history of biting[15]. Tooth mortality, which is mainly a reflection of untreated dental caries and periodontal disease, is considered as a crude but useful measure for the dental status of a community[16]. Causes of tooth extractions had large geographical and cultural differences between various regions in a country and from one country to another[16,17]. Extraction of teeth is the most common procedure carried out in oral surgery clinics. The final consequence of most dento-alveolar diseases is tooth loss, mostly through routine tooth extraction[18].Reasons for routine tooth extractions have been widely reported in medical literature[18-22]. In addition, postoperative pain and discomfort, loss day at work as well as healing complications have been well reported in the immediate postoperative period following non-surgical (routine) tooth extraction[23-25].

Materials And Methods

Study Designand Sample

To address the research purpose, a survey study is been implemented by conducting a questionnaire survey which contains 9 questions. All the questions were most relevant for the perception of exodontia. The sample size for this study is 50. The questionnaire survey was then distributed among 50 dentists in Saveetha Dental College in the department of oral and maxillofacial surgery. The participants answered the survey without any bias and after the survey was over, data was collected and statistically analysed and the results were tabulated.

PERCEPTION OF EXODONTIA – RESEARCH QUESTIONNARE

AGE:

SEX: MALE / FEMALE

YEAR OF PRACTICE:

1. **WHICH IS THE MOST DIFFICULT TOOTH TO EXTRACT?**

18 17 16 15 14 13 12 11 21 22 23 24 25 26 27 28

48 47 46 45 44 43 42 41 31 32 33 34 35 36 37 38

2. WHAT IS THE MOST COMMON COMPLICATION ENCOUNTED IN EXODONTIA?

A. BLEEDING
B. ORO-ANTRAL COMMUNICATION
C. DENTOALVEOLAR FRACTURE
D. TUBEROSITY FRACTURE
E. ROOT FRACTURE
F. OTHERS

3. WHICH INSTRUMENT DO YOU USE FOR EXTRACTION OF UPPER THIRD MOLAR?

A. STRAIGHT ELEVATORS
B. FORCEPS
C. CRYERS
D. COUPLAND ELEVATORS
E. OTHERS

4. WHICH INSTRUMENT DO YOU USE FOR EXTRACTION OF LOWER THIRD MOLAR?

A. STRAIGHT ELEVATORS
B. FORCEPS
C. COUPLAND ELEVATORS
D. OTHERS

5. WHAT IS THE NORMAL BLOOD GLUCOSE LEVEL DO YOU PREFER FOR EXTRACTION?

A. 40 - 60 mg/dL (fasting)
B. 70 – 110 mg/dL (fasting)
C. 120 – 140 mg/dL (fasting)

6. WHAT IS THE NORMAL BLOOD PRESSURE DO YOU PREFER FOR EXTRACTION?

A. 100/70- 140/90mm/Hg
B. 90/60- 140/94 mm/Hg
C. 120/80-160/94 mm/Hg

7. WHICH ANTIBIOTIC DO YOU PREFER IN THERAPEUTIC EXTRACTION?

A. NARROW SPECTRUM
B. BROAD SPECTRUM
C. METRONIDAZOL
D. NO ANTIBIOTICS
E. OTHERS

8. HOW WILL YOU MANAGE IF THE LOCAL ANASTHESIA IS NOT ACHIEVED?

A. MAIN TRUNK ANASTHESIA
B. INTRA PULPAL INJECTION
C. INTRA LIGAMENTAL INJECTION
D. ANTIBIOTIC AND RECALL

9) WHAT IS YOUR PREFERENCE OF ANTIBIOTICS IN DENTO ALVEOLAR ABSCESS CASES?

A. PENICILLIN AND METRONIDAZOLE

B. ORNIDAZOLE AND OFLOXIN

C. CLINDAMYCIN

D. CIPROFLOXACIN

E. OTHERS

DATA ANALYSIS

To analyze the data, counts and tabulation were calculated. Results were obtained for survey responses, and was estimated according to the study. Bar charts were used to present the results.

Results

FIGURE 1:RESULTS SHOWING THE MOST DIFFICULT TOOTH TO EXTRACT:

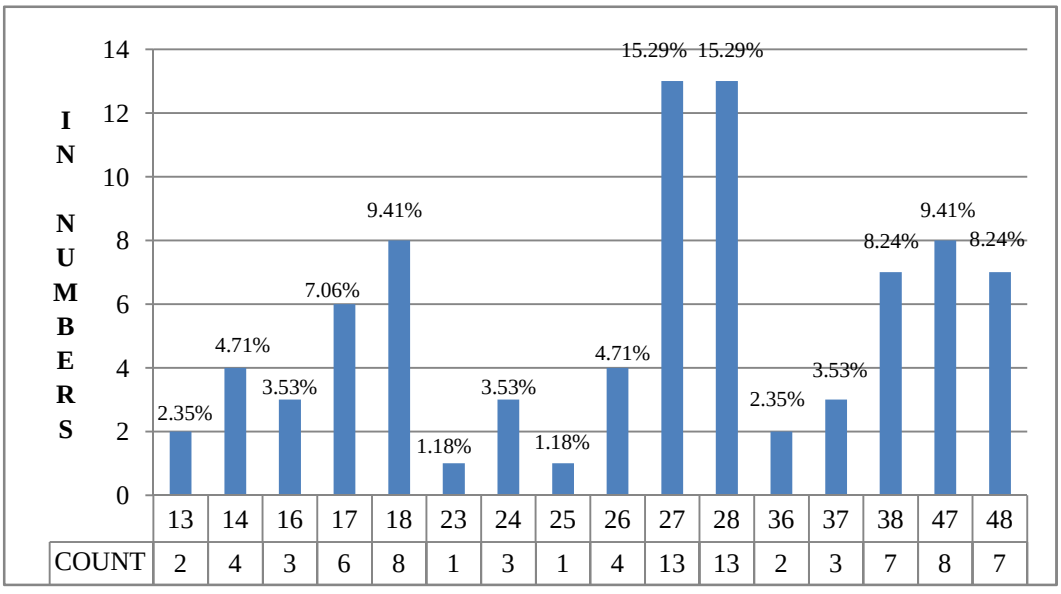


FIGURE 2: BAR GRAPH SHOWING MOST COMMON COMPLICATION ENCOUNTED:

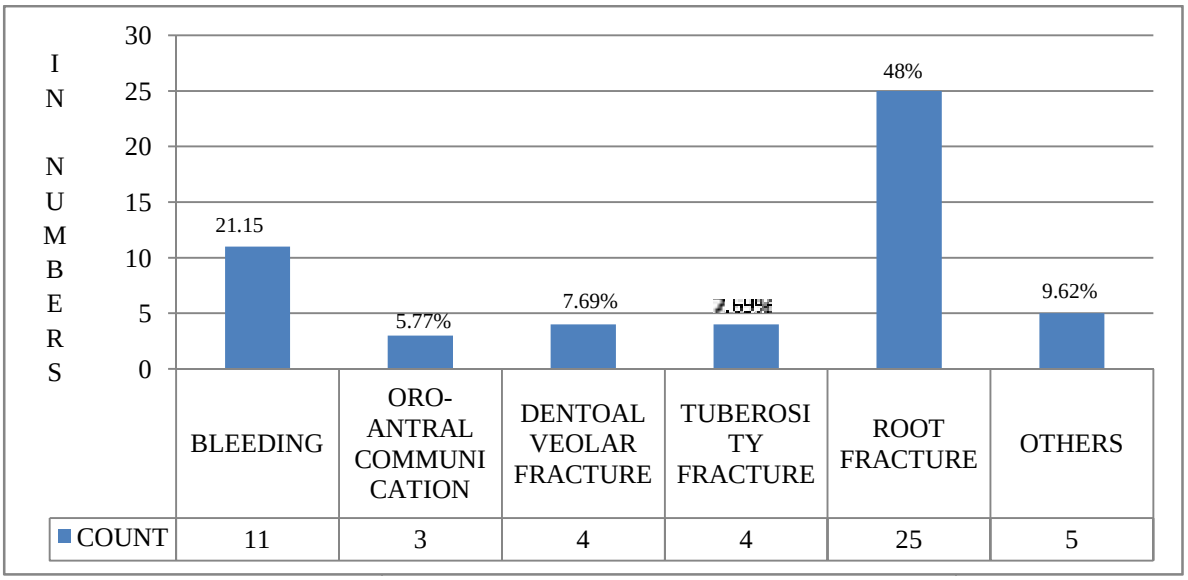


FIGURE3: BAR GRAPH REPRESENTING THE MOST COMMONLY USED INSTRUMENT FOR EXTRACTION OF UPPER THIRD MOLAR:

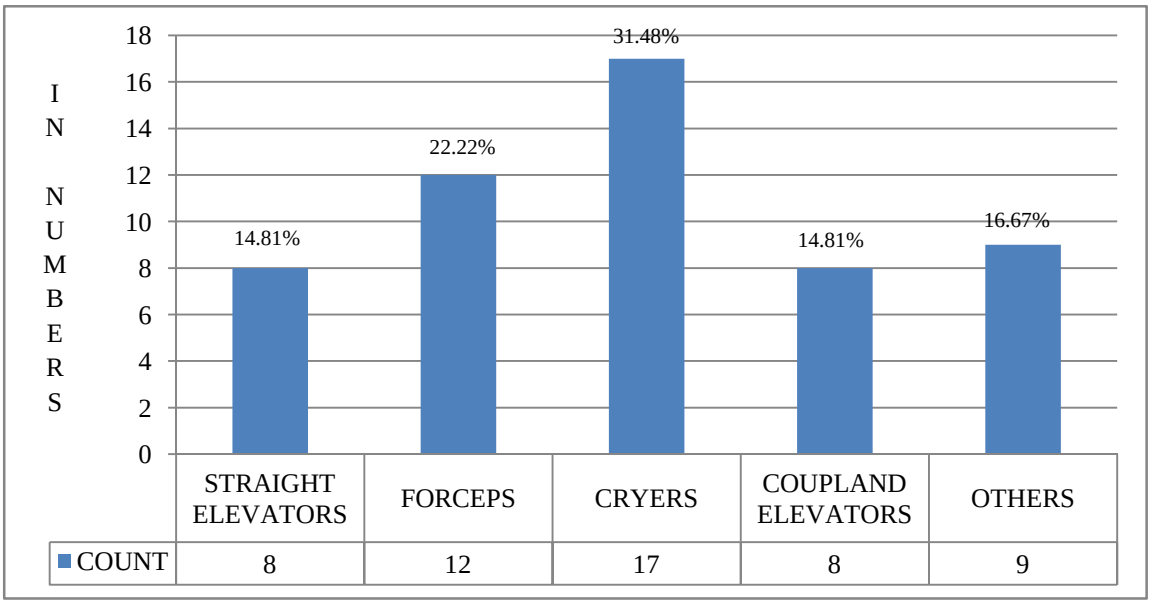


FIGURE 4: BAR GRAPH REPRESENTING THE MOST COMMONLY USED INSTRUMENT FOR EXTRACTION OF LOWER THIRD MOLAR:

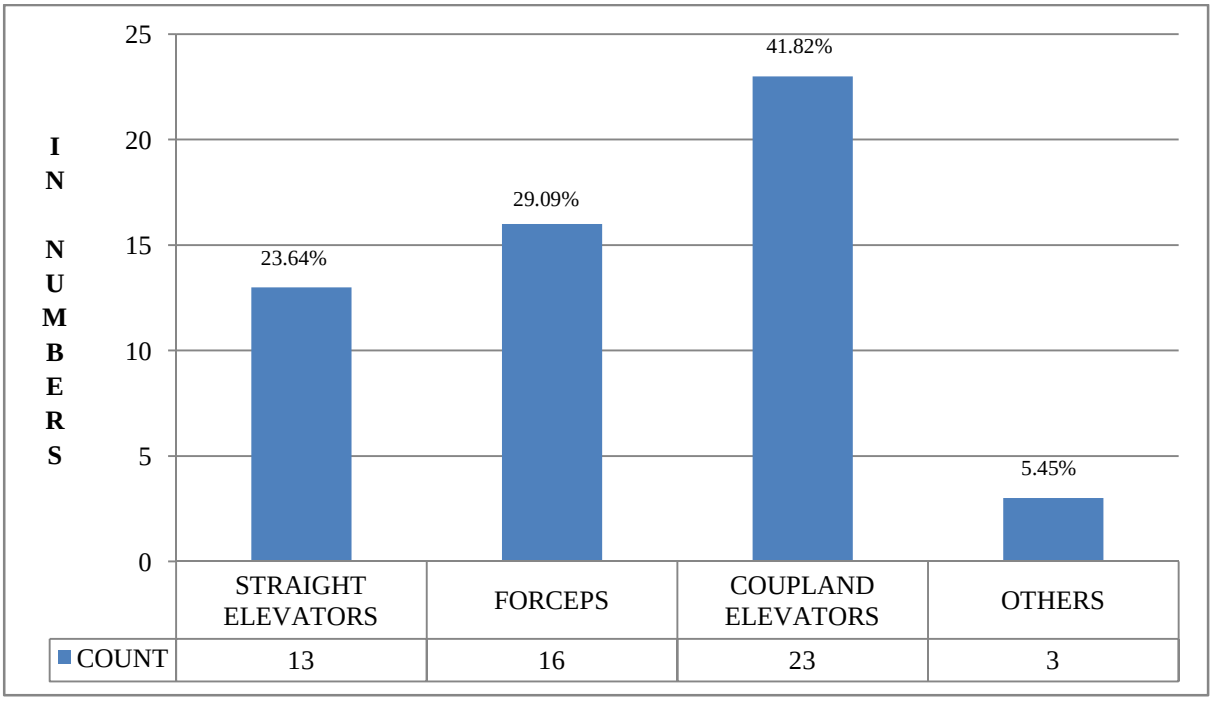


FIGURE 5: BAR GRAPH SHOWING NORMAL BLOOD GLUCOSE LEVEL PREFERRED FOR EXTRACTION BY THE DENTISTS:

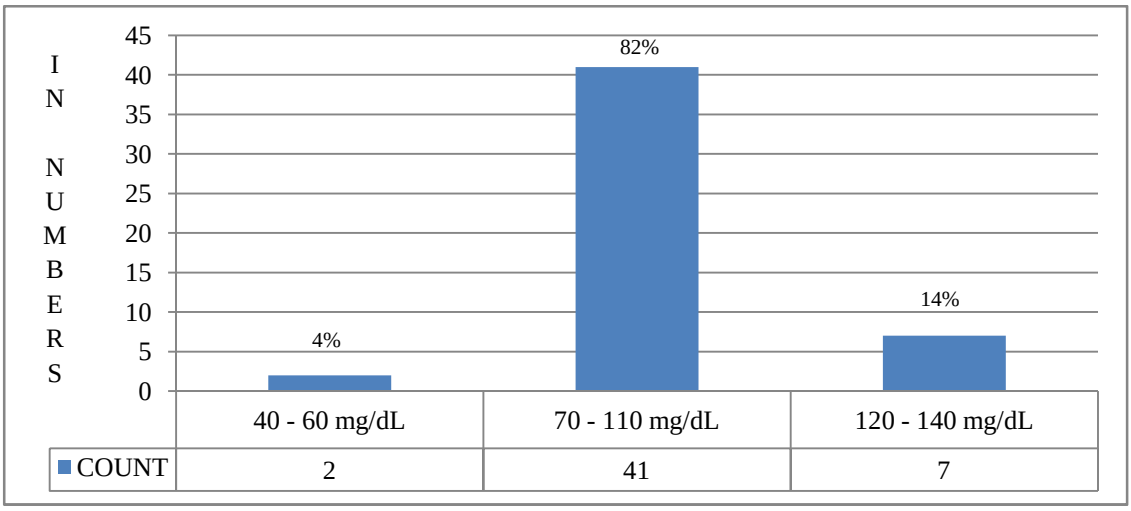


FIGURE 6: BAR GRAPH SHOWING NORMAL BLOOD PRESSURE PREFERRED FOR EXTRACTION:

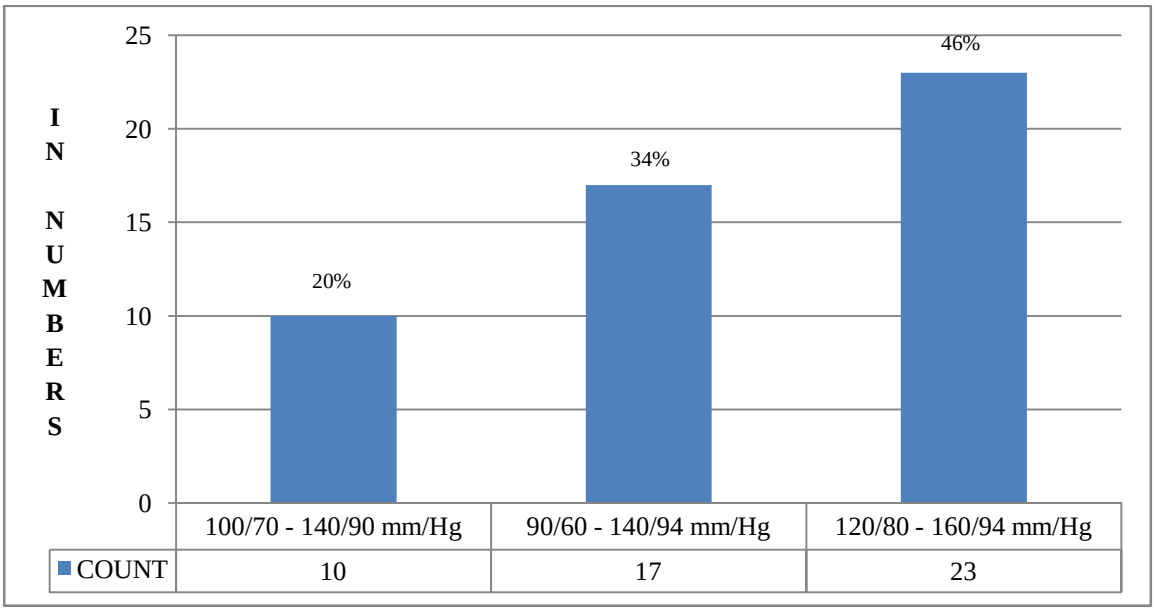


FIGURE 7: BAR GRAPH SHOWING THE MOST PREFERRED ANTIBIOTIC IN THERAPEUTIC EXTRACTION:

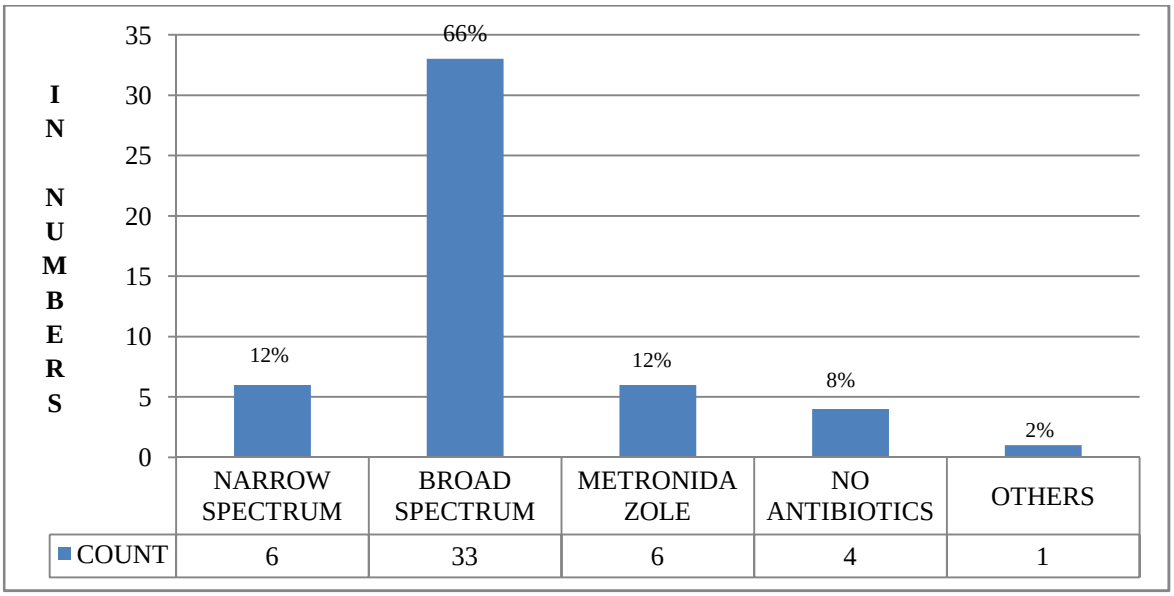


FIGURE 8: WAYS PREFERRED WHEN LOCALANAESTHESIA DOES NOT WORK:

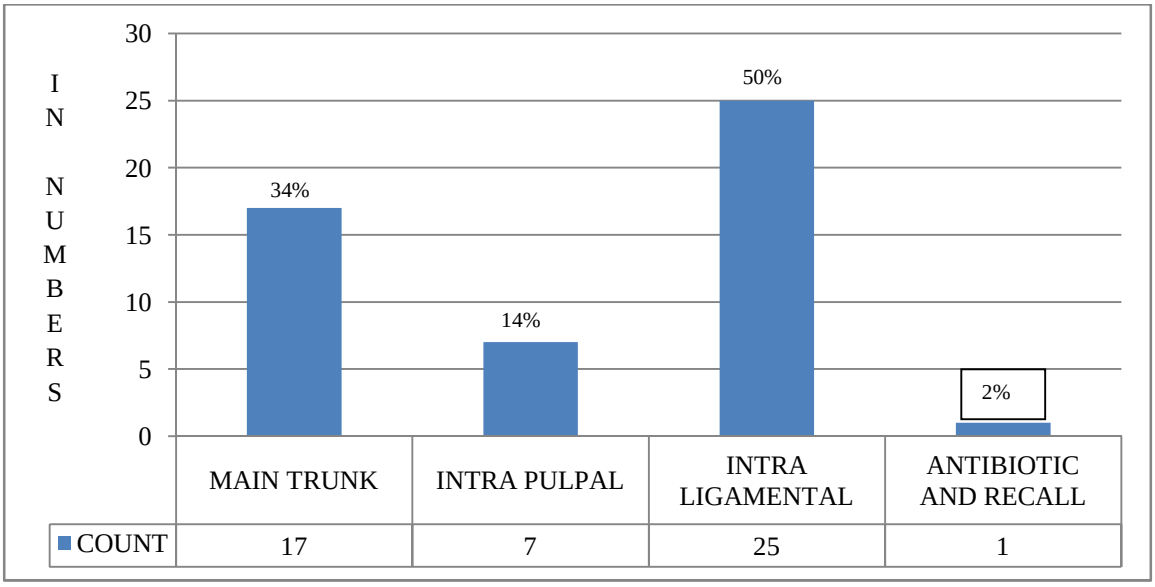
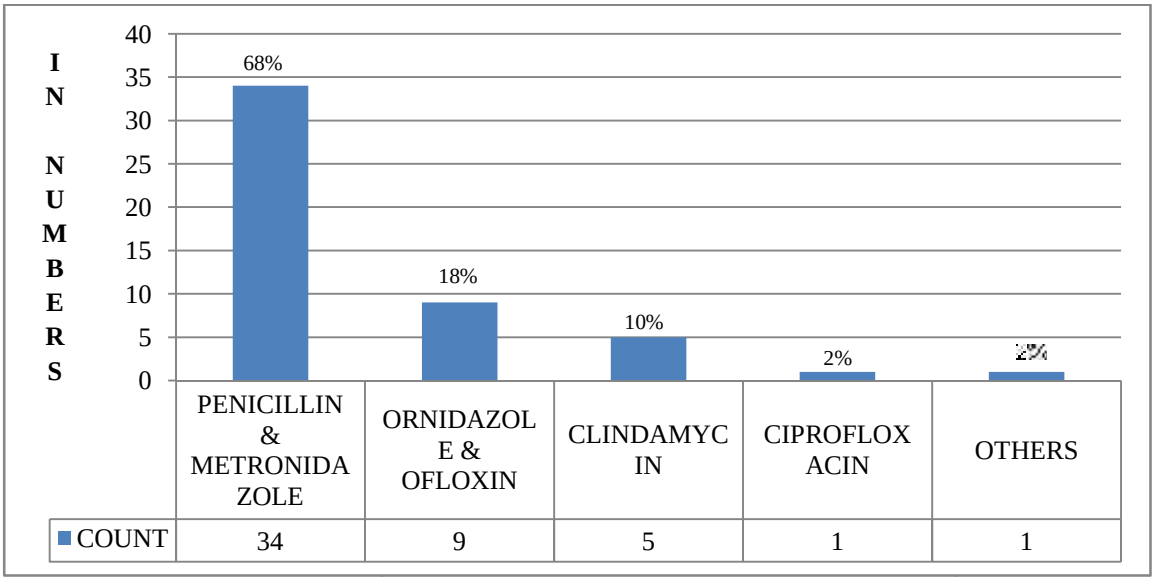


FIGURE 9: PREFERENCE OF ANTIBIOTICS IN DENTO ALVEOLAR ABSCESS:



Discussion:

Tooth extraction remains a major cause commonly performed procedure in developing countries[26,27] Tooth loss has significant socioeconomic, quality of life general health, and psychological consequences[27,28] Indeed, tooth loss has become a global public health concern of immense proportion[28,29]. Despite being preventable, dental caries and periodontal disease remain the most common reasons for tooth extraction, especially developing nations.[30,31]

The reasons for tooth extraction and the number of teeth extracted in a population have been linked to the oral hygiene, level of education, socioeconomic status, and individual quality of life[28,30,32].Degree of urbanization has also been found to affect the pattern of tooth extractions[33,34]. Moreover, oral disease burden and its etiological factors exhibit inter- and intra-regional variations[35]. Dental extraction has a psychological influence on the patients both before and after the surgery. It is always a delicate task in the hands of dentists to prepare the patients before any dental extraction procedure and it is also critical to provide appropriate expectations of the discomfort the patients would experience[36]. Local anesthetic solutions are utmost importance in the field of dentistry. The main use of the local anesthetic drug is that they eliminate pain[37]. Extraction is the most common procedure done in

dentistry in india, and a knowledge about post extraction complications and their management can prevent the occurrence of untoward sequelae following extraction[38]. Third molar impactions are common. The impaction rate for third molars is higher than for any other teeth[39].Root fracture is the most common complication according the dentists conducted in the survey. Thus, extracting a tooth without fracturing the root is technique sensitive and thus need to be practiced well by the dentists to avoid further complication. Cryers and coupland elevators are the most preferred instruments for extraction of upper and lower third molar respectively,which makes us understand that these instruments makes extraction more easier. 70-110mg/dL is the normal range of blood glucose during exodontia and 120/80-160-94mmHg is the normal blood pressure for exodontia, thus making it significant before initiating extraction.The most commonly preferred antibiotic in therapeutic extraction is broad spectrum antibiotics, the other alternative when LA does not work is intra ligamental and penicillin an metronidazole are the most preferred in dento alveolar abscess. Awareness must be created about the normal blood pressure range among dentists to be performed in cases of exodontia.

Conclusion:

Thus, exodontia is very well understood from the survey that 27 and 28 are the most difficult teeth during exodontia, root fracture is the most common complication, cryers and coupland elevators are the most preferred instruments for extraction of upper and lower third molar respectively, 70-110mg/dL is the normal range of blood glucose during exodontia and 120/80-160-94mmHg is the normal blood pressure for exodontia, the most commonly preferred antibiotic in therapeutic extraction is broad spectrum antibiotics, the other alternative when LA does not work is intra ligamental and penicillin an metronidazole are the most preferred in dento alveolar abscess. Awareness must be created about the normal blood pressure range among dentists to be performed in cases of exodontia.

Corresponding Author:

Dr.M.Sankari,

Professor, Department of Periodontics,

Saveetha dental college,

Saveetha University,

Saveetha Institute of Medical and Technical Sciences,

162,Poonamallee High Road,

Chennai-600077,

Tamil Nadu,India.

REFERENCES:

1) **Aida J, Ando Y, Akhter R, Aoyama H, Masui M, Morita M:** ***Reasons for permanent tooth extractions in Japan***. J Epidemiol 2006;16:214-219.
2) **Chrysanthakopoulos NA**: ***Reasons for extraction of permanent teeth in Greece: a five-year follow-up study.*** Int Dent J 2011;61:19-24.
3) **Agerholm DM, Sidi AD:** ***Reasons given for extraction of permanent teeth by general dental practitioners in England and Wales.*** Br Dent J 1988;164:345-348.
4) **Al-Shammari KF, Al-Ansari JM, Al-Melh MA, Al-Khabbaz AK**: ***Reasons for tooth extraction in Kuwait.*** Med Princ Pract 2006;15:417-422.
5) **Chen SC, Chueh LH, Hsiao CK, Wu HP, Chiang CP**: ***First untoward events and reasons for tooth extraction after nonsurgical endodontic treatment in Taiwan***. J Endod 2008;34:671-674.

6) **Chestnutt IG, Binnie VI, Taylor MM:** ***Reasons for tooth extraction in Scotland***. J Dent 2000;28:295-297.

7) **Corbet EF, Davies WI:** ***Reasons given for tooth extraction in Hong Kong***. Community Dent Health 1991;8:121-130.

8) **Da'ameh D:** ***Reasons for permanent tooth extraction in the North of Afghanistan.***J Dent 2006;34:48-51.

9) **Jovino-Silveira RC, Caldas AF Jr, de Souza EH, Gusmão ES**: ***Primary reason for tooth extraction in a Brazilian adult population.*** Oral Health Prev Dent 2005;3:151-157.

10) **Haddad I, Haddadin K, Jebrin S, Ma'Ani M, Yassin O**: ***Reasons for extraction of permanent teeth in Jordan.*** Int Dent J 1999;49:343-346.

11) **Matthews DC, Smith CG, Hanscom SL:** ***Tooth loss in periodontal patients.***J Can Dent Assoc 2001;67:207-210.

12) **Murray H, Locker D, Kay EJ**: ***Patterns of and reasons for tooth extractions in general dental practice in Ontario, Canada.*** Community Dent Oral Epidemiol 1996;24:196-200.

13) **Ainamo J, Sarkki L, Kuhalampi ML**. ***Thefrequency of periodontal extractions in Finland.*** Community Dent Health. 1984;1:165–72.

14) **Razak IA, Jaafer N, Matnor G;** ***The causes of tooth mortality of permanent teeth in a Malaysian population.*** J Ir Dent Assoc. 1989;35:39–41.

15) **Trott JR, Cross HG;** ***An analysis of the principle reasons for tooth extractions in1813 patients in Manitoba.*** Dent Pract Dent Rec. 1966;17:20–7.

16) **Farsi JM;** ***Common causes of extraction of teeth in Saudi Arabia***. Saudi Dent J. 1992;4:101–5.

17) **Chestnutt IG, Binnie VI, Taylor MM;** ***Reasons for tooth extraction in Scotland***. J Dent. 2000;28:295–7.

18) **Kay EJ, Blinkhorn AS;*****The reasons underlying the extraction of teeth in Scotland.*** Br Dent J. 1986;160:287–90.

19) **Angelillo IF, Nobile CG, Pavia M;** ***Survey of reasons for extraction of permanent teeth in Italy.*** Community Dent Oral Epidemiol. 1996;24:336–40.

20) **Murray H, Locker D, Kay EJ;** ***Patterns of and reasons for tooth extractions in general dental practice in Ontario, Canada.*** Community Dent Oral Epidemiol. 1996;24:196–200.

21) **McCaul LK, Jenkins WM, Kay EJ;** ***The reasons for extraction of permanent teeth in Scotland: A 15-year follow-up study.*** Br Dent J. 2001;190:658–62.

22) **Aida J, Ando Y, Akhter R, Aoyama H, Masui M, Morita M;*****Reasons for permanent tooth extractions in Japan.*** J Epidemiol. 2006;16:214–9.

23) **Adeyemo WL, Ladeinde AL;*****Ogunlewe MO. Clinical evaluation of post-extraction site wound healing.*** J Contemp Dent Pract. 2006;7:40–9.

24) **Adeyemo WL, Ladeinde AL;** ***Ogunlewe MO. Influence of trans-operative complications on socket healing following dental extractions***. J Contemp Dent Pract. 2007;8:52–9.

25) **Oginni FO, Fatusi OA, Alagbe AO;*****A clinical evaluation of dry socket in a Nigerian teaching hospital.*** J Oral Maxillofac Surg. 2003;61:871–6.

26) **Saheeb BD, Sede MA;*****Reasons and pattern of tooth mortality in a Nigerian Urban teaching hospital.*** Ann Afr Med. 2013;12:110–4.

27) **Jafarian M, Etebarian A;*****Reasons for extraction of permanent teeth in general dental practices in Tehran, Iran.*** Med Princ Pract. 2013;22:239–44.

28) **Taiwo OA, Sulaiman AO, Obileye MF, Akinshipo A, Uwumwonse AO, Soremi OO;** ***Patterns and reasons for childhood tooth extraction in Northwest Nigeria.*** J Pediatr Dent. 2014;2:83.

29) **Alesia K, Khalil HS;** ***Reasons for and patterns relating to the extraction of permanent teeth in a subset of the Saudi population.*** Clin Cosmet Investig Dent. 2013;5:51–6.

30) **Dixit LP, Gurung CK, Gurung N, Joshi N;** ***Reasons underlying the extraction of permanent teeth in patients attending Peoples Dental College and Hospital.*** Nepal Med Coll J. 2010;12:203–6.

31) **Kashif M, Mehmood K, Ayub T, Aslam M;** ***Reasons and patterns of tooth extraction in a tertiary care hospital-A cross sectional prospective survey.*** LiaquatUni Med Health Sci. 2014;13:125–9.

32) **La Torre G, Romeo U, Iarocci G, Brugnoletti O, Semyonov L, Galanakis A;** ***Socio-demographic inequalities and teeth extraction in the last 12 months in Italy.*** Ann Stomatol (Roma) 2015;5:131–5.

33) **Luan WM, Baelum V, Chen X, Fejerskov O;** ***Tooth mortality and prosthetic treatment patterns in urban and rural Chinese aged 20-80 years.*** Community Dent Oral Epidemiol. 1989;17:221–6.

34) **Varenne B, Petersen PE, Ouattara S;** ***Oral health behaviour of children and adults in urban and rural areas of Burkina Faso, Africa.*** Int Dent J. 2006;56:61–70.

35) **Odai CD,Azodo CC, Ezeja EB, Obuekwe ON;*****Reasons for exodontia in rural Nigerian children.*** Odontostomatol Trop. 2010;33:19–24.

36) **M. Hari Priya, Santhosh Kumar;*****Dental Extraction – Post Operative wellness Survey***/Int.J. PharmTech Res. 2015,8(4),pp 666-669.

37) **Balamurugan.R;*****Efficacy of Local Anesthesia;***International Dental Journal of Student's Research| December 2013-June 2014 | Volume 2| Issue 2.

38) **M.P.Santhosh Kumar, Lavenya ;*****Knowledge about Post Extraction Complications among Undergraduate Dental Students*** ; J. Pharm. Sci. & Res. Vol. 8(6), 2016, 470-476.

39) **ArshiyaShehanaz;** ***The incidence of types of mandibular third molar impactions in different skeletal face types: A clinical study;***International Journal of Orofacial Research; Year : 2017 | Volume : 2 | Issue : 1 | Page : 24-27.

Perception of Pain during Orthodontic Treatment among School Students Undergoing and not Under Going Orthodontic Treatment

BhavaniGanesan[1], Jaiganesh Ramamurthy[2]

Graduate Student[1], Reader[2], Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: This study helps to understandthe perception of pain which is felt by the school students, who are undergoing orthodontic treatment and also the perception of pain related to orthodontic treatment who are not undergoing orthodontic treatment. This will help the dentist to understand the perception of pain much better.The aim and objective of the study was to evaluate the perception of pain in orthodontic treatment among school children who are undergoing and not undergoing orthodontic treatment.

Materials and Methods: The school student consists of two groups: group 1 consist of students who are undergoing orthodontic treatment and group 2 consist of students who are not undergoing orthodontic treatment. Questionnaire forms weregiven to both the group of students. In Group 1 questions were based on the difficulties they underwent during treatment. In Group 2 questions were based on their perception about orthodontic treatment. The data was analysed statistically to achieve the results.

Results: From the results it has been found that 64% of the people had experienced a pain during orthodontic treatment most probably at day1, 22% of the people had experienced a pain at day3 and 14% of the people had experienced pain beyond 5th day. Student who were not undergoing orthodontic treatment; 68% of the people felt orthodontic treatment was painful and 32% of the people felt that orthodontic treatment was not painful.

Conclusion: This study helps the orthodontist to understand the perception of pain towards the orthodontic treatment and conduct the treatment accordingly to make the treatment as painless as possible.

Key Words: *Pain, treatment, perception, students, Orthodontics*

Introduction

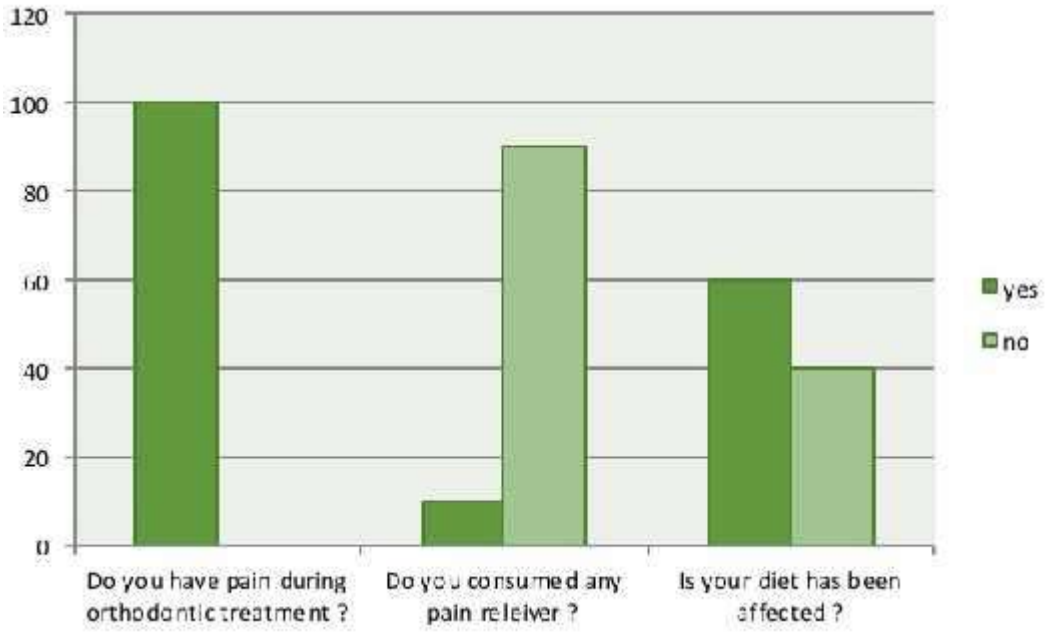
Pain and discomfort to the patient are more common during orthodontic treatment Malaligned teeth often result into food lodgment esthetic and psychological problems in patients.The main cause of pain during orthodontic treatment is the application of forces to induce tooth movement, separator placement, and during debonding[1,2] Pain is influenced by gender, personality factors, individual pain threshold, magnitude of force applied, and motivation.[3-5]TMJ, lips, gingiva and tongue are not affected by fixed appliances.[6-8] Discomfort is mainly localized at the teeth. Among the soft tissues, Ulcer sites are localized at the inner side of the cheeks[9,10] Ithas been found that well-informed patients has lesser pain perception and require fewer medications.The aim of this study was to evaluate perception of pain towards orthodontic treatment by questionnaire survey.

Materials and Methods

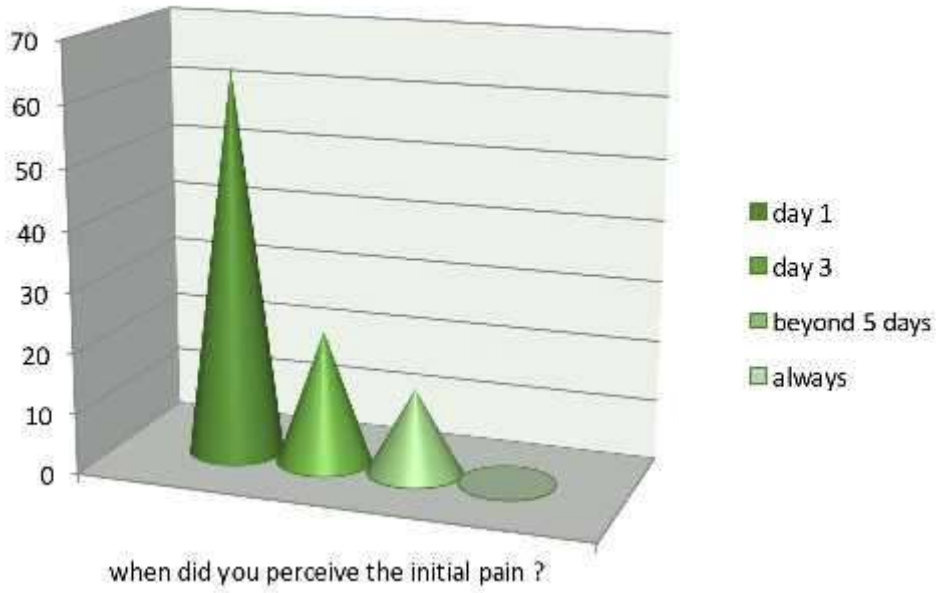
The questionnaire study was conducted on 100 patients with Group I consist of 50 people who were undergoing orthodontic treatment and Group II consist of 50 people who weren't undergoing orthodontic treatment. The questionnaireform was relevant to pain perception toward orthodontic treatment. For evaluation of perception pain, there were twoquestionnaires independently given for each group. The questionnaire forms will be differs from both the groups, questions related to their experience towards orthodontic treatment was given to the group of people undergoing orthodontic treatment, whereas questions about their perception towards malocclusion and orthodontic treatment was given to people who are not undergoing orthodontic treatment.The data was analyzed statistically to achieve the results.

Results

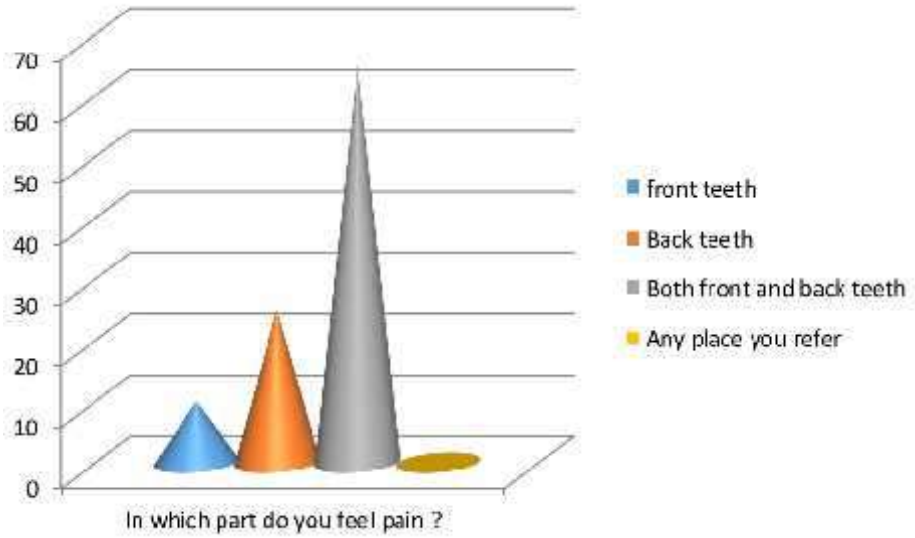
Group 1



Graph 1: Presents 100% of the people reported that there was pain during the orthodontic treatment.10% of the people have consumed pain reliever during orthodontic treatment and 90% of the people have not consumed pain reliever during treatment.60% of the people has a disturbance in their diet and 40% of the people has no disturbance in their diet.

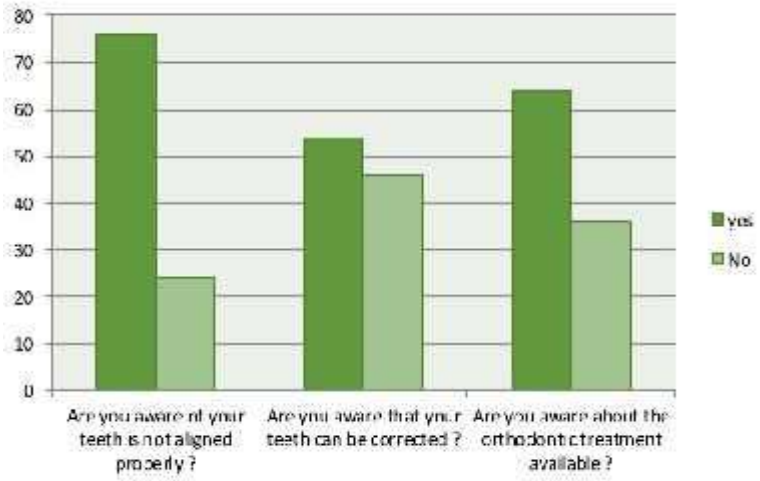


Graph 2: Represents 64% of the people feel that they perceived the initial pain at day1, 22% of the people feel that they perceived the initial pain at day 3, 14% of the people feel that they perceived the initial pain at beyond day 5and no one feel that they had pain always.

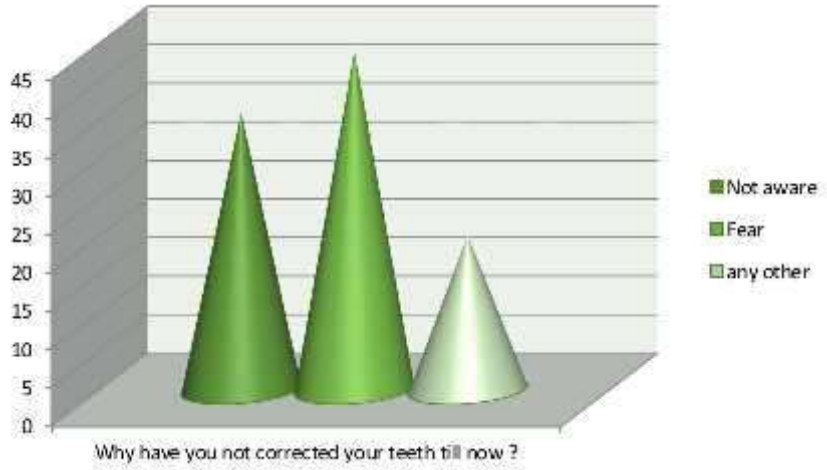


Graph 3: Represents 10% of the people feel that they had a pain in front teethregion ,25% of the people feel that they had a pain in back teeth region,65 % of the people that they had experienced a pain in both front and back teeth region.

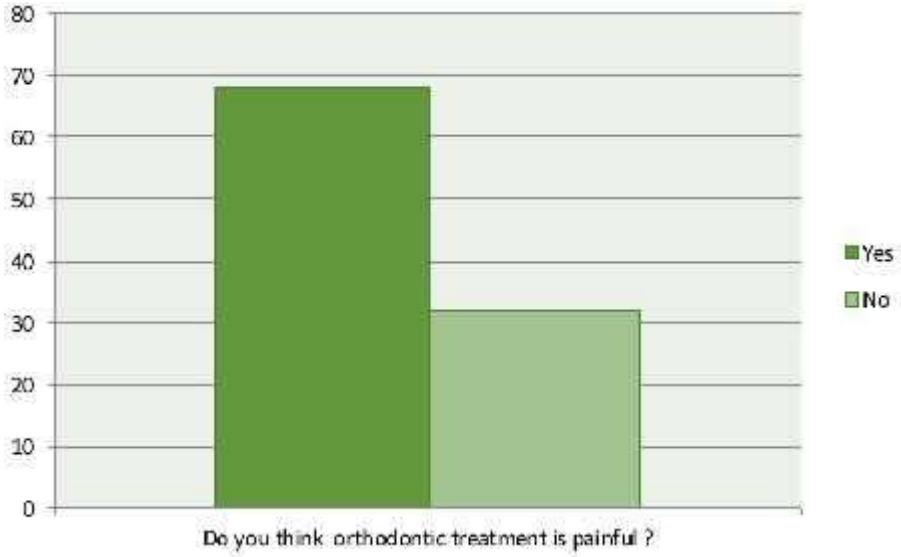
Group II



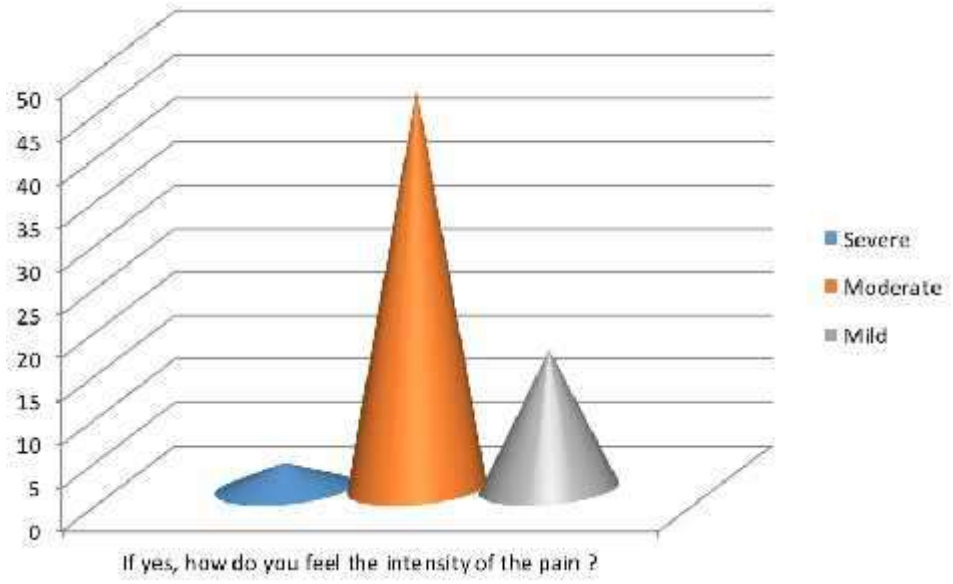
Graph 4: Represents 76% of the people reported that they were aware of their teeth were not aligned properly and 24% of the people reported that they were not aware about their teeth is not aligned properly.54% of the people reported that they were aware of their teeth can be corrected and 46% reported that they were not aware of their teeth can be corrected.64% of the people reported that they were aware about the orthodontic treatment available and 36% of the people reported that they were not aware about the orthodontic treatment available.



Graph 5: Represents 36% of the people reported that they were not aware of correcting their teeth, 22% of the people reported that they had fear of correcting their teeth, 10% of the peoplehad some other reason of not correcting their teeth.



Graph 6: Represents 68% of the people feel orthodontic treatment is painful and 32% of the people feel that orthodontic treatment is not painful.



Graph 7: Represents 6% of the people reported the intensity of the pain issevere, 46% of the people reported the intensity of the pain ismoderate, 16% of the people reported the intensity of the pain is mild.

Discussion

The study was performed on 100 people from which 50 people who have undergoing orthodontic treatment and another 50 people who have not undergoing orthodontic treatment.From the results it has been found that 64% of the people had experiencedapainduring orthodontic treatment most probably at day1, 22% of the people had experienced a pain at day3 and 14% of the people had experienced a pain at beyond 5 day.

The people felt the pain has reduced as progress the weeks, no one experienced a pain always till orthodontic treatment completed.Periodontal pain is caused by a10% of the people feel that they had a pain in front teeth region,25% of the people feel that they had a pain in back teeth region, 65 % of the people that they had a pain in both front and back teeth region.Now the reason of this could be that individual's perception of pain varies from person to person.The pain seems to more initially because of the inflammatory reaction andmovement of the tooth and also the pressure were felt maximum on the first day.

The forces applied could start coming down after the day 1 because of fatigueof elastic materials which are used and as the tooth moves the pressure on the tooth also reduce.Theresults have been found from the people who are not undergoing orthodontic treatment.68% of the people felt orthodontic treatment is painful and 32% of the people felt that orthodontic treatment is not painful.68% of the people reported that orthodontic treatment is painful from that 6% of the people reported the intensity of the pain is severe, 46% of the people reported the intensity of the pain ismoderate, 16% of the people reported the intensity of the pain is mild.

Every treatment in the dental specialty has its own set of complications orthodontic therapy being no exception. Dental aesthetics are a key factor in overall physical attractiveness, which also contributes to self-esteem. This is one of the main reasons for patients to undergo Orthodontic treatment.[11-15] Although Orthodontic treatment has many recognized benefits, including improvement in dental health, function, appearance, and self-esteem, nevertheless Orthodontic appliances can cause unwanted complications if adequate care is not taken during the treatment. It is important that the patients are aware of these potential risks, so that they can know their responsibilities and the expectations placed on them during the treatment. This ensures in achieving successful results without any adverse effects during and after completion of orthodontic treatment.[16-20].

Conclusion

This study helps the orthodontist to understand the perception of pain towards the orthodontic treatment and conduct the treatment accordingly to make the treatment as painless as possible.

Corresponding Author

Jaiganesh Ramamurthy

Reader, Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Phone Number: +91 98404 43463,

Email: dr.r.jaiganesh@gmail.com

References

1. Jones M. An investigation into the initial discomfort caused by placement of an archwire. European Journal of Orthodontics.1984; 6: 48-54.
2. Feinmann C, Ong M, Harvey W, Harris M. Psychological factors influencing post-operative pain and analgesic consumption. British Journal of Oral and Maxillo-Facial Surgery.1987; 25:285-92.
3. Kvam E, Gjerdet N, Bondevik. Traumatic ulcers and pain during orthodontic treatment. Community Dentistry and Oral Epidemiology.1987; 15:104-7.
4. Kvam E, Bondevik, Gjerdet N. Traumatic ulcers and pain in adults during orthodontic treatment. Community Dentistry and Oral Epidemiology.1989; 17:154-7.
5. Oliver R, Knapman Y. Attitudes to orthodontic treatment. British Journal of Orthodontics.1985; 12:179-88.
6. Brown D, Moerenhout R. The pain experience and psychological adjustment to orthodontic treatment of pre- adolescents, adolescents, and adults. American Journal of Orthodontics and Dentofacial Orthopedics.1991; 100:349-56.
7. Soltis J, Nakfoor P, Bowman D.Changes in ability of patients to differentiate intensity of forces applied to maxillary central incisors during orthodontic treatment. Journal of Dental Research.1971; 50:590-6.
8. White L. Pain and cooperation in orthodontic treatment. Journal of Clinical Orthodontics.1984; 18: 572-5.
9. Wilson S, Ngan P, Kess B. Time course of the discomfort in young patients undergoing orthodontic treatment.Pediatric Dentistry.1998;11: 107-10.
10. Huskisson E. Measurement of pain. Lancet.1974; 2:1127-31.
11. Jones M, Chan C. The pain and discomfort experienced during orthodontic treatment. A randomised controlled clinical trial of two initial aligning arch wires. American Journal of Orthodontics and Dentofacial Orthopedics.1992; 102: 373-81
12. Priyanka.S, Saravana Dinesh, Jaiganesh Ramamurthy.Ortho-perio relationship: Is it a nuptial knot. International Journal of Pharma and Bio Sciences.2014; 5: B100-5.
13. Meenakshi Priyanka, Jaiganesh. Periodontally Accelerated Osteogenic Orthodontics. International Journal of Pharma and Bio Sciences.2013; 5(4):49-51.

14. Suhashini Ramanathan, Navaneethan Ramasamy. Avoidable Complication and Patient Care during Orthodontic Treatment. Journal of Pharmaceutical Science and Research.2015; 7(12):1096-8.
15. Shrada Kumar. Pain and discomfort in patients undergoing orthodontic treatment with crowding. International Journal of Scientific Research.2017; 6(10):148-150.
16. Breece GL, Nieberg LG: Motivations for adult orthodontic treatment.J Clin Orthod.1986;20:166-71.
17. Ferreira SH, Moncada S, Vane JR: Prostaglandins and the mechanism of analgesia produced by aspirin-like drugs. Br J Pharmac.1973; 49:86-97.
18. Jones ML: An investigation into the initial discomfort caused byplacement of an arch wire. Eur J Orthod.1984;6:48-54.
19. Jones ML, Richmond S: Initial tooth movement: force application and pain -- a relationship? Am J Orthod 1988:111-16.
20. Kamogashira K, Yanabri M, Ichikawa K, Itoh T, Matsumato M, Ishibashi K, Abe K: The effects of upper incisor separation on the submandibular and sublingual glands of rats. J Dent Res 1988; 67:602-10.

Perception of Pain during Orthodontic Treatment among School Students Undergoing and not Under Going Orthodontic Treatment

Bhavani Ganesan [1], Jaiganesh Ramamurthy[2]
Graduate Student[1], Reader[2], Department of Periodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: This study helps to understandthe perception of pain which is felt by the school students, who are undergoing orthodontic treatment and also the perception of pain related to orthodontic treatment who are not undergoing orthodontic treatment. This will help the dentist to understand the perception of pain much better.The aim and objective of the study was to evaluate the perception of pain in orthodontic treatment among school children who are undergoing and not undergoing orthodontic treatment.

Materials and Methods: The school students consists of two groups: group 1 consist of students who are undergoing orthodontic treatment and group 2 consist of students who are not undergoing orthodontic treatment. Questionnaire forms weregiven to both the group of students. In Group 1 questions were based on the difficulties they underwent during treatment. In Group 2 questions were based on their perception about orthodontic treatment. The data was analysed statistically to achieve the results.

Results: From the results it has been found that 64% of the people had experienced a pain during orthodontic treatment most probably at day1, 22% of the people had experienced a pain at day3 and 14% of the people had experienced pain beyond 5th day. Student who were not undergoing orthodontic treatment; 68% of the people felt orthodontic treatment was painful and 32% of the people felt that orthodontic treatment was not painful.

Conclusion: This study helps the orthodontist to understand the perception of pain towards the orthodontic treatment and conduct the treatment accordingly to make the treatment as painless as possible.

Key Words*: Pain, Orthodontics, Treatment, School students, Perception.*

Introduction

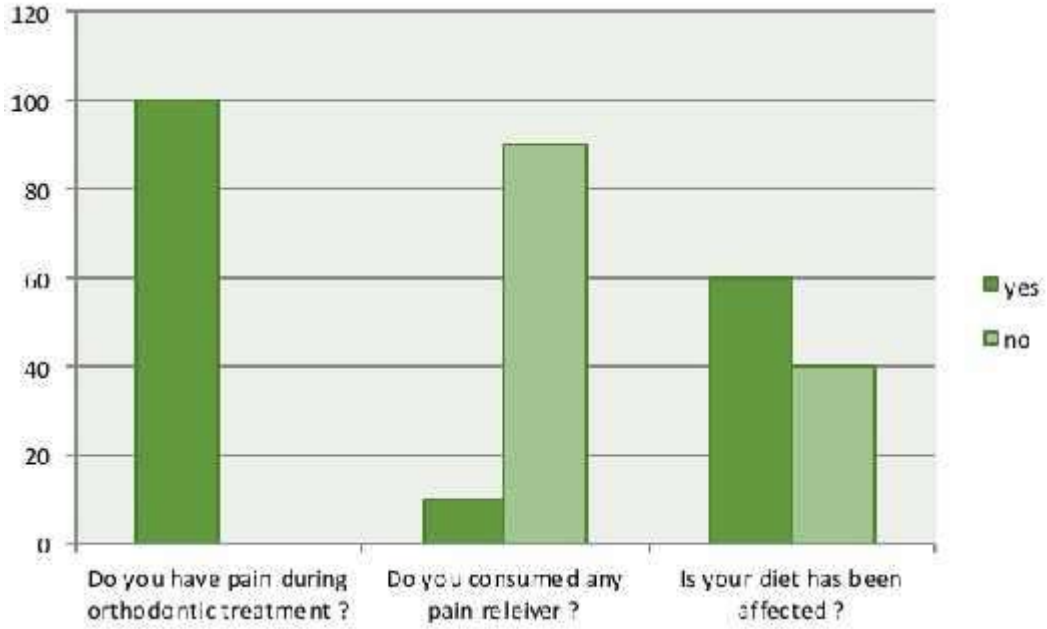
Pain and discomfort to the patient are more common during orthodontic treatment Malaligned teeth often result into food lodgment esthetic and psychological problems in patients.The main cause of pain during orthodontic treatment is the application of forces to induce tooth movement, separator placement, and during debonding[1,2] Pain is influenced by gender, personality factors, individual pain threshold, magnitude of force applied, and motivation.[3-5]TMJ, lips, gingiva and tongue are not affected by fixed appliances.[6-8] Discomfort is mainly localized at the teeth. Among the soft tissues, Ulcer sites are localized at the inner side of the cheeks[9,10] Ithas been found that well-informed patients has lesser pain perception and require fewer medications.The aim of this study was to evaluate perception of pain towards orthodontic treatment by questionnaire survey.

Methods and Materials

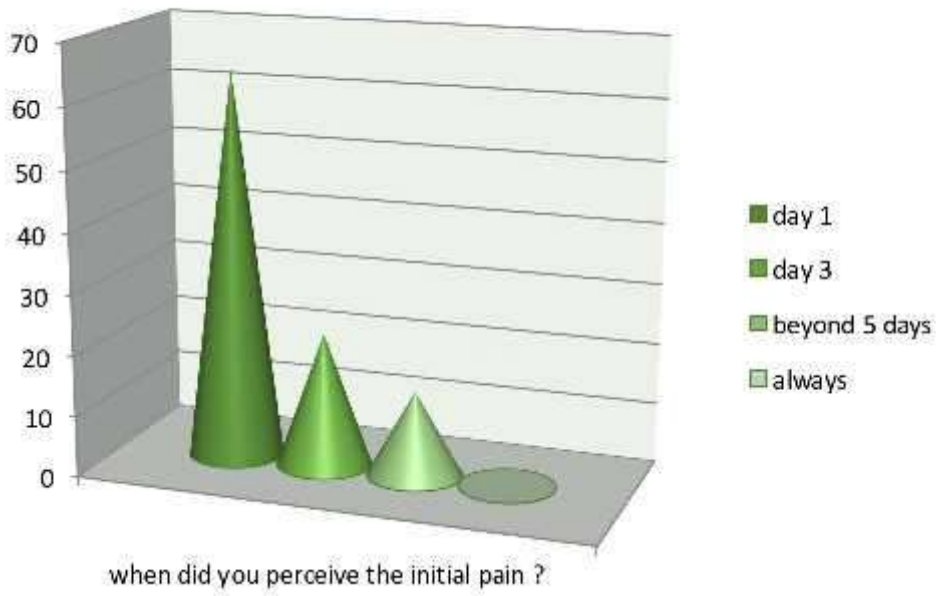
The questionnaire study was conducted on 100 patients with Group I consist of 50 people who were undergoing orthodontic treatment and Group II consist of 50 people who weren't undergoing orthodontic treatment. The questionnaireform was relevant to pain perception toward orthodontic treatment. For evaluation of perception pain, there were twoquestionnaires independently given for each group. The questionnaire forms will be differs from both the groups, questions related to their experience towards orthodontic treatment was given to the group of people undergoing orthodontic treatment, whereas questions about their perception towards malocclusion and orthodontic treatment was given to people who are not undergoing orthodontic treatment.The data was analyzed statistically to achieve the results.

Results

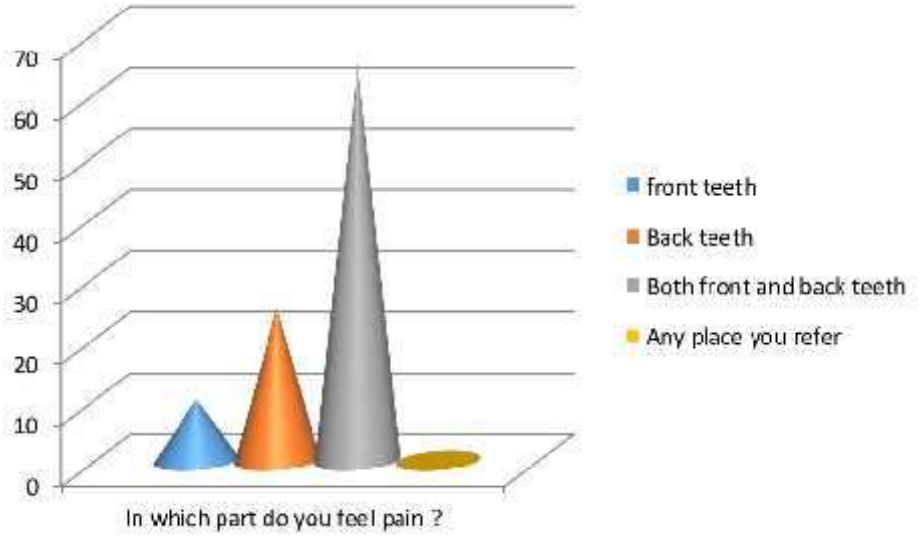
Group I



Graph 1: Presents 100% of the people reported that there was pain during the orthodontic treatment.10% of the people have consumed pain reliever during orthodontic treatment and 90% of the people have not consumed pain reliever during treatment.60% of the people has a disturbance in their diet and 40% of the people has no disturbance in their diet.

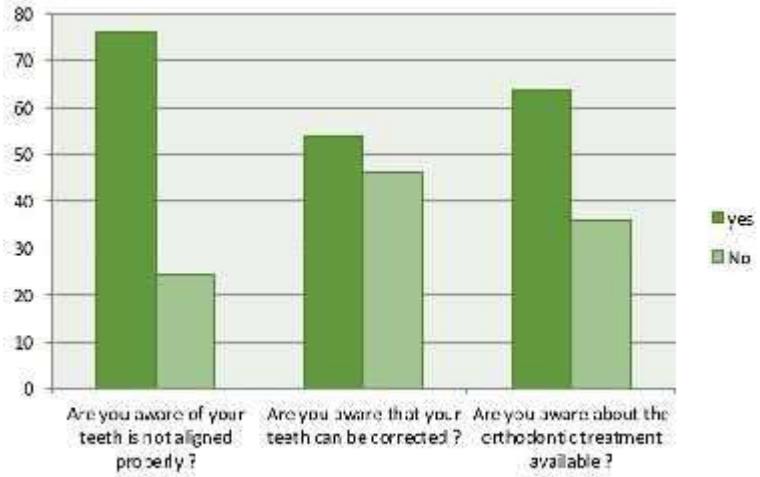


Graph 2: Represents 64% of the people feel that they perceived the initial pain at day1, 22% of the people feel that they perceived the initial pain at day 3, 14% of the people feel that they perceived the initial pain at beyond day 5and no one feel that they had pain always.

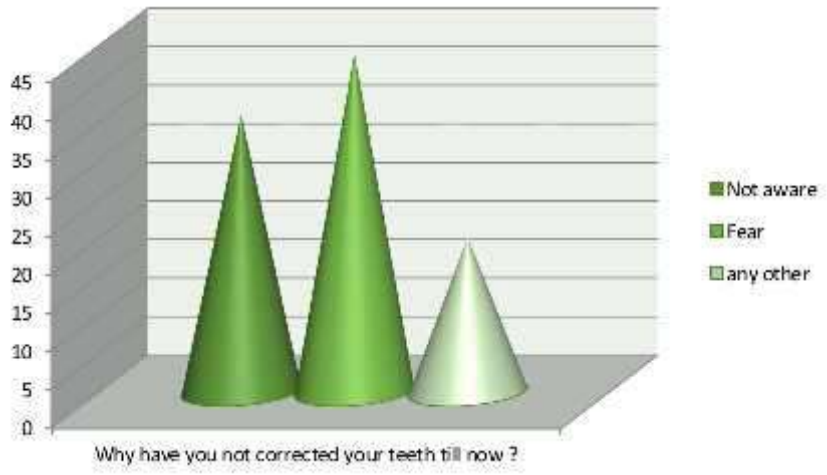


Graph 3: Represents 10% of the people feel that they had a pain in front teethregion ,25% of the people feel that they had a pain in back teeth region,65 % of the people that they had experienced a pain in both front and back teeth region.

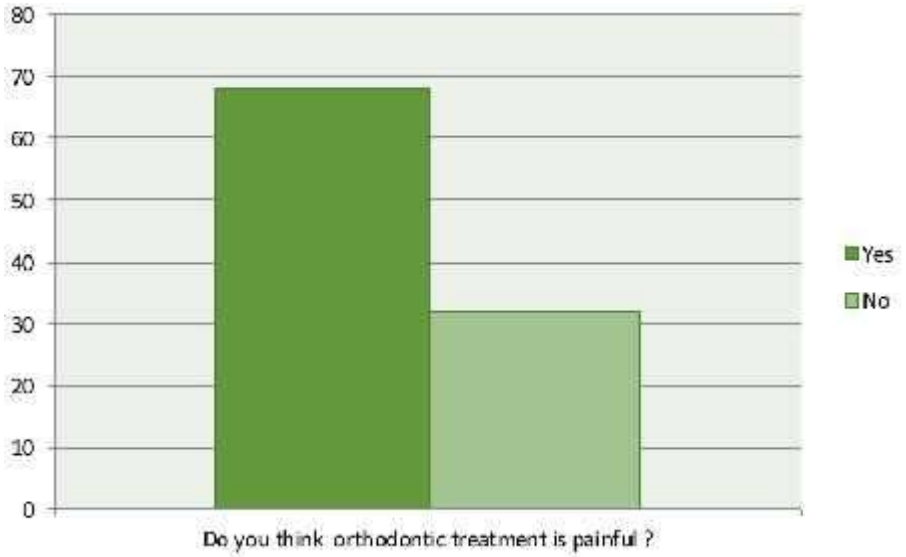
Group II



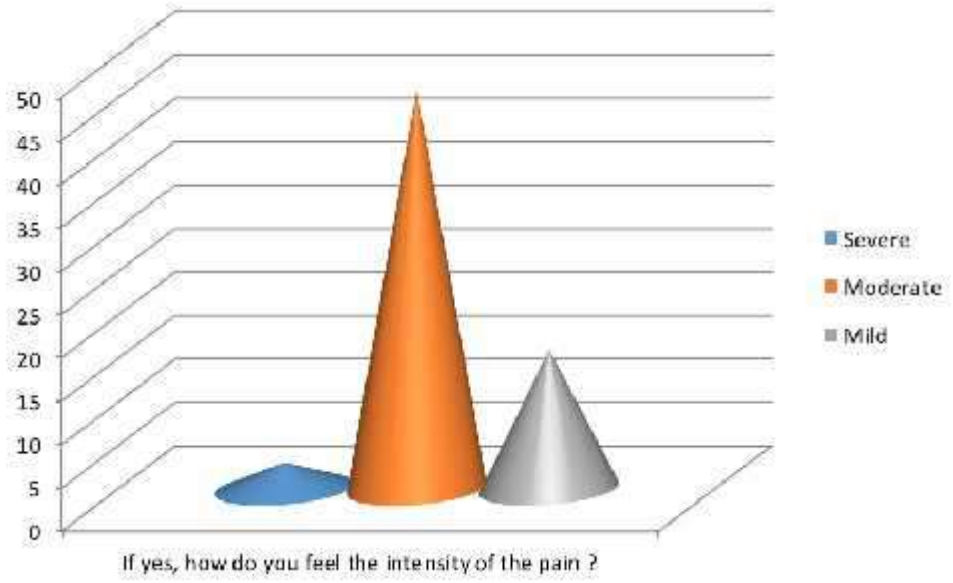
Graph 4: Represents 76% of the people reported that they were aware of their teeth were not aligned properly and 24% of the people reported that they were not aware about their teeth is not aligned properly.54% of the people reported that they were aware of their teeth can be corrected and 46% reported that they were not aware of their teeth can be corrected.64% of the people reported that they were aware about the orthodontic treatment available and 36% of the people reported that they were not aware about the orthodontic treatment available.



Graph 5: Represents 36% of the people reported that they were not aware of correcting their teeth, 22% of the people reported that they had fear of correcting their teeth, 10% of the peoplehad some other reason of not correcting their teeth.



Graph 6: Represents 68% of the people feel orthodontic treatment is painful and 32% of the people feel that orthodontic treatment is not painful.



Graph 7: Represents 6% of the people reported the intensity of the pain issevere, 46% of the people reported the intensity of the pain ismoderate, 16% of the people reported the intensity of the pain is mild.

Discussion

The study was performed on 100 people from which 50 people who have undergoing orthodontic treatment and another 50 people who have not undergoing orthodontic treatment.From the results it has been found that 64% of the people had experiencedapainduring orthodontic treatment most probably at day1, 22% of the people had experienced a pain at day3 and 14% of the people had experienced a pain at beyond 5 day.

The people felt the pain has reduced as progress the weeks, no one experienced a pain always till orthodontic treatment completed.Periodontal pain is caused by a10% of the people feel that they had a pain in front teeth region,25% of the people feel that they had a pain in back teeth region, 65 % of the people that they had a pain in both front and back teeth region.Now the reason of this could be that individual's perception of pain varies from person to person.The pain seems to more initially because of the inflammatory reaction andmovement of the tooth and also the pressure were felt maximum on the first day.

The forces applied could start coming down after the day 1 because of fatigueof elastic materials which are used and as the tooth moves the pressure on the tooth also reduce.Theresults have been found from the people who are not undergoing orthodontic treatment.68% of the people felt orthodontic treatment is painful and 32% of the people felt that orthodontic treatment is not painful.68% of the people reported that orthodontic treatment is painful from that 6% of the people reported the intensity of the pain is severe, 46% of the people reported the intensity of the pain ismoderate, 16% of the people reported the intensity of the pain is mild.

Every treatment in the dental specialty has its own set of complications orthodontic therapy being no exception. Dental aesthetics are a key factor in overall physical attractiveness, which also contributes to self-esteem. This is one of the main reasons for patients to undergo Orthodontic treatment.[11-15] Although Orthodontic treatment has many recognized benefits, including improvement in dental health, function, appearance, and self-esteem, nevertheless Orthodontic appliances can cause unwanted complications if adequate care is not taken during the treatment. It is important that the patients are aware of these potential risks, so that they can know their responsibilities and the expectations placed on them during the treatment. This ensures in achieving successful results without any adverse effects during and after completion of orthodontic treatment.[16-20].

Conclusion

This study helps the orthodontist to understand the perception of pain towards the orthodontic treatment and conduct the treatment accordingly to make the treatment as painless as possible.

Corresponding Author

Jaiganesh Ramamurthy, Reader, Department of Periodontics

Saveetha Dental College, Saveetha University, Saveetha Institute of Medical and Technical Sciences

162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Phone Number: +91 98404 43463, Email: dr.r.jaiganesh@gmail.com

References

1. Jones M. An investigation into the initial discomfort caused by placement of an archwire. European Journal of Orthodontics.1984; 6: 48-54.
2. Feinmann C, Ong M, Harvey W, Harris M. Psychological factors influencing post-operative pain and analgesic consumption. British Journal of Oral and Maxillo-Facial Surgery.1987; 25:285-92.
3. Kvam E, Gjerdet N, Bondevik. Traumatic ulcers and pain during orthodontic treatment. Community Dentistry and Oral Epidemiology.1987; 15:104-7.
4. Kvam E, Bondevik, Gjerdet N. Traumatic ulcers and pain in adults during orthodontic treatment. Community Dentistry and Oral Epidemiology.1989; 17:154-7.
5. Oliver R, Knapman Y. Attitudes to orthodontic treatment. British Journal of Orthodontics.1985; 12:179-88.
6. Brown D, Moerenhout R. The pain experience and psychological adjustment to orthodontic treatment of pre- adolescents, adolescents, and adults. American Journal of Orthodontics and Dentofacial Orthopedics.1991; 100:349-56.
7. Soltis J, Nakfoor P, Bowman D.Changes in ability of patients to differentiate intensity of forces applied to maxillary central incisors during orthodontic treatment. Journal of Dental Research.1971; 50:590-6.
8. White L. Pain and cooperation in orthodontic treatment. Journal of Clinical Orthodontics.1984; 18: 572-5.
9. Wilson S, Ngan P, Kess B. Time course of the discomfort in young patients undergoing orthodontic treatment.Pediatric Dentistry.1998;11: 107-10.
10. Huskisson E. Measurement of pain. Lancet.1974; 2:1127-31.
11. Jones M, Chan C. The pain and discomfort experienced during orthodontic treatment. A randomised controlled clinical trial of two initial aligning arch wires. American Journal of Orthodontics and Dentofacial Orthopedics.1992; 102: 373-81
12. Priyanka.S, Saravana Dinesh, Jaiganesh Ramamurthy.Ortho-perio relationship: Is it a nuptial knot. International Journal of Pharma and Bio Sciences.2014; 5: B100-5.
13. Meenakshi Priyanka, Jaiganesh. Periodontally Accelerated Osteogenic Orthodontics. International Journal of Pharma and Bio Sciences.2013; 5(4):49-51.

14. Suhashini Ramanathan, Navaneethan Ramasamy. Avoidable Complication and Patient Care during Orthodontic Treatment. Journal of Pharmaceutical Science and Research.2015; 7(12):1096-8.
15. Shrada Kumar. Pain and discomfort in patients undergoing orthodontic treatment with crowding. International Journal of Scientific Research.2017; 6(10):148-150.
16. Breece GL, Nieberg LG: Motivations for adult orthodontic treatment.J Clin Orthod.1986;20:166-71.
17. Ferreira SH, Moncada S, Vane JR: Prostaglandins and the mechanism of analgesia produced by aspirin-like drugs. Br J Pharmac.1973; 49:86-97.
18. Jones ML: An investigation into the initial discomfort caused byplacement of an arch wire. Eur J Orthod.1984;6:48-54.
19. Jones ML, Richmond S: Initial tooth movement: force application and pain -- a relationship? Am J Orthod 1988:111-16.
20. Kamogashira K, Yanabri M, Ichikawa K, Itoh T, Matsumato M, Ishibashi K, Abe K: The effects of upper incisor separation on the submandibular and sublingual glands of rats. J Dent Res 1988; 67:602-10.

Perception of Prosthodontic Need of Patients During and After Extraction – A Survey

.

S.Renuka[1], S. Sangeetha Kirubagaran[2],M.Sankari[3],
Graduate student[1],Reader[2], Department of Prosthodontics, Reader[3],Department of Periodontics,
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Tooth loss in patients may cause oral discomfort, mastication problems, displacement of adjacent and opposing teeth in order to cover edentulous spaces and also the alignment of the teeth. Loss of teeth not only adversely affects the body physiology but also disturbs the psychology of the individual**.** The aim of this study is to identify the perception of patients about the impacts and needs of prosthesis at the time and after extraction of the tooth.

Materials and Methods: Teeth play an important role in mastication. Tooth loss can be treated with various types of prosthetics, depending on the severity which aids in maintaining the mastication properly. Most of the patients are not seeking treatment immediately after tooth loss evenwhen it is needed because of unawareness about the treatment. The purpose of this study is to evaluate the patients' perception about the impacts and prosthesis need at the time and after extraction of the tooth. A convenient sample of 101 patients were clinically evaluated and answered for the questionnaire about prosthetic needs. Data were collected at the time of extraction and at the time interval of onemonth. Statistical analysis has been done with the collected data.

Result: This study is designed comparatively to evaluate and analyze the patients' perception about the impacts and needs of prosthesis at the time and after tooth loss. The reason of this study is to aware people about their knowledge on benefits, risk and impact on prosthesis and about impacts on untreated tooth loss.

Conclusion: Self- perception about tooth loss and prosthodontic needs at the time and after an interval of 3 month from extraction didn't have much significant change among patients. Better function and oral discomfort were mentioned as major benefit and risk of prosthesis. Treatment demand was low due to lack of knowledge and awareness, poor interest and motivation.

Key Words: *Perception, prosthesis, treatment, mastication, extraction.*

Introduction

Tooth loss in patients may cause oral discomfort, mastication problems, displacement of adjacent and opposing teeth in order to cover edentulous spaces and also the alignment of the teeth. Loss of teeth not only adversely affects the body physiology but also disturbs the psychology of the individual [1]. Even though the knowledge about the impacts of tooth loss and the importance of tooth loss on general health [2, 3] among the patients is less, the psychological disturbances are directly related to the acceptance of the artificial prostheses by the patient [2]. Prosthodontics is concerned with the replacement of partial or complete loss of teeth and oral function due to tooth or tissue damage [4, 5]. The patient's self- perceived prosthodontic desire is based on number of absent teeth, age, gender, position of the missing tooth, oral discomfort, functional disability and socio-economical status especially when anterior tooth are not involved [6, 7]. If anterior tooth involved, the prosthodontic treatment is indicated to restore the patient aesthetics and self-esteem [5]. In a recent study, most patients gave financial constraints & rumours for not replacing teeth. Cost was the main barrier for obtaining dentures [8, 9]. Another issue is the time taken by the patients between tooth losses and seek for prosthodontic treatment. Some of the patient’s perception might change after a period of time from extraction and may seek prosthodontic treatment or implants. The physical effects and emotional status of the clinical conditions are widely commonly explained for both partially dentate and edentulous individuals [10-13]. Prosthodontic rehabilitation of partially or completely edentulous patients is important, since the definitive treatment can improve oral function, aesthetics and quality of life of most patients [14]. This study is done to assess the patient's self-perceived prosthodontic need at the time of extraction and short interval after tooth loss.

Materials and Methods

A convenient sample of 101 patients were reported to our dental institution Saveetha dental college about tooth loss are examined and interviewed. Data's are collected at two stages: at the time of extraction and 3 month after extraction. Patient's exclusion criteria were third molar surgery (complete dentition), extraction for orthodontic purposes, emergency dental problems with acute symptoms, and patients unable to answer the questionnaire or who refuse to participate. All patients scheduled for elective dental extraction were included. Clinical examination was performed at the time of tooth extraction before surgery. It includes socio-demographic variables and clinical variables such as position, extension and number of previous edentulous areas, and reasons for extraction, location of edentulous spaces and number of teeth indicated for extraction. After 3 months, patients were contacted by phone and/or mail. Patients were questioned if they had replaced the extracted teeth and, if not, the reason why they did not demand for immediate replacement.

Results

A total convenient sample of 101 patients who visited our dental institution with age group range from 16 to 70 years were included in the study (45.5% male; 54.5% female). 43.6% patients are at low educational level and 57.4% patients have completed their basic education.

At the Time of Extraction

Table 1 shows that, at the time of extraction, most patients (91.1%)had prior edentulous spaces. Most of the edentulous spaces were present in both anterior and posteriorly of about 42.6%. There ismultiplenumbers of edentulous spaces of about 50.5% present in both the arches (34.7%). Only 52.4% of patients undergone prosthodonticrehabilitation for previously extracted tooth.86.2% of patients optedfixed partial dentures in both anterior and posterior teeth(37.9%) or only posterior teeth (36.2%), and 34.5% of dentures were inthe both arches or upper arch respectively.

In Table 2 shows that patients' perceptions of impacts associated totooth loss were considered high at the time of extraction (33.7 to61.4%). Although 51.5% of patients felt immediate replacement ofextracted teeth is more important, only 51.5% intended to treat edentulous spaces shortly after extraction. 61.4% patients felt thatprosthodontics replacement is necessary to solve the problems followedby tooth extraction, 51.5% considered that

immediate placement isnecessary and 51.5% of patients intended to seek treatment immediatelyafter extraction. 50.5% of patients believed that prosthesis does not have potential risk. 60.4% of patients concern about their dentalcondition and 52.5% of patients are satisfied with their dentalcondition. The patients felt that loss of teeth does not affect theirgeneral health, appearance and digestion of food of about 66.3%, 55.4% and 55.4% respectively. Only 58.4% of patients felt difficulty to chewthe food after tooth loss.

Table 3 shows frequency of patient's perceptions of benefits and riskof prosthodontic treatment and reason for not treating. 63.6% and53.5% of patients felt better function and aesthetics has a benefitrespectively. 60.4% patients felt oral discomfort as a risk and only 34.4% of patients have poor adaptation to dentures. 32.6% of patientsdon't know the reason and didn't feel necessary to undergone treatmentafter tooth loss.

After Extraction

After 3 months of tooth extraction, all the patients were recalledfor the second evaluation. Table.4 shows only 53 patients had undergone prosthodonticrehabilitation on the second evaluation. 28.7% of patientsrestoredtheir teeth in the posterior region and only 16.8% in their anteriorregion. The demand for treatment was greater in the upper arch(30.7%) with small extension (34.7%) and single (32.7%) edentulousspaces.

After 3 months of interval of extraction, 51.5% of patients donotperceive any difficulty in chewing food. Most of the patientsperceived that tooth loss does not worsen the digestion, generalhealth and appearance of the patient, of about 72.3%, 67.3% and 58.4%respectively. 54.5% of patients perceived that they are not satisfiedwith their dental condition and 57.4% of patients concern about theirdental condition. Hence, 57.4% of people believed that prosthesissolve all the problems caused by tooth loss. Even though 50.5% ofpatients considered immediate replacement is necessary and 61.4% ofpeople believed that prosthesis does not have potential risk, 51.5% ofpatientsdo not seek immediate replacement of teeth.

Table 1. Initial clinical characteristics of patients and edentulous spaces (ES) at the time of teeth extraction.

	n	%
All Patients (n=101)		
Presence of prior ES		
Yes	92	91.1%
No	9	8.9%
Number of absent teeth/patient		
0	9	8.9%
1	34	33.7%
2 – 4	33	32.7%
5 – 10	20	19.9%
11 – 20	4	4.0%
21 – 30	1	1.0%
Patients with ES (n=53)		
Position of ES		
Anterior	19	18.8%
Posterior	30	29.7%
Both	43	42.6%
Arch		
Upper	30	29.7%
Lower	27	26.7%
Both	35	34.7%
Number of ES		
Single	41	40.6%
Multiple	51	50.5%
Treatment of ES		
Treated	53	52.4%
Untreated	48	47.5%
Patients with Prosthodontics (n=53)		
Prosthodontic treatment		
Fixed	50	86.2%
Removable	5	8.6%
Both	3	5.2%
Position		
Anterior	15	25.9%
Posterior	21	36.2%
Both	22	37.9%
Arch		
Upper	20	34.5%
Lower	18	31.0%
Both	20	34.5%

Table 2. Comparison of self-perception of patients at the time and after teeth extraction.

	Before Extraction (n=53)		1 month after extraction		Z	Asymp.sig
	yes	No	Yes	No		
1. Difficult to chew any food	59(58.4%)	42(41.6%)	49(48.5%)	52(51.5%)	1.019	0.363
2. Digestion became worsened	45(44.6%)	56(55.4%)	28(27.7%)	73(72.3%)	1.489	0.256
3. General health became worsened	34(33.7%)	67(66.3%)	33(32.7%)	68(67.3%)	0.419	0.704
4. Appearance became worsened	45(44.6%)	56(55.4%)	42(41.6%)	59(58.4%)	1.071	0.322
5. No satisfaction with dental condition	48(47.5%)	53(52.5%)	55(54.5%)	46(45.5%)	0.615	0.541
6. Concern about dental condition	61(60.4%)	40(39.6%)	58(57.4%)	43(42.6%)	0.415	0.698
7. Believed that prosthesis solve all potential problems caused by tooth loss	62(61.4%)	39(28.6%)	58(57.4%)	43(42.6%)	0.540	0.620
8. Believed that prosthesis have potential risk	50(49.5%)	51(50.5%)	39(38.6%)	62(61.4%)	0.763	0.508
9. Considered that immediate replacement is necessary	52(51.5%)	49(48.5%)	50(49.5%)	51(50.5%)	0.968	0.416
10. Intended to seek treatment shortly after extraction	52(51.5%)	49(48.5%)	49(48.5%)	52(51.5%)	0.506	0.636

*Statistically significant ($p<0.05$)

Table 3. Frequency of patients' perception of benefits and risks of a prosthodontic treatment and reasons for not treating edentulous spaces previously, at the time of extraction.

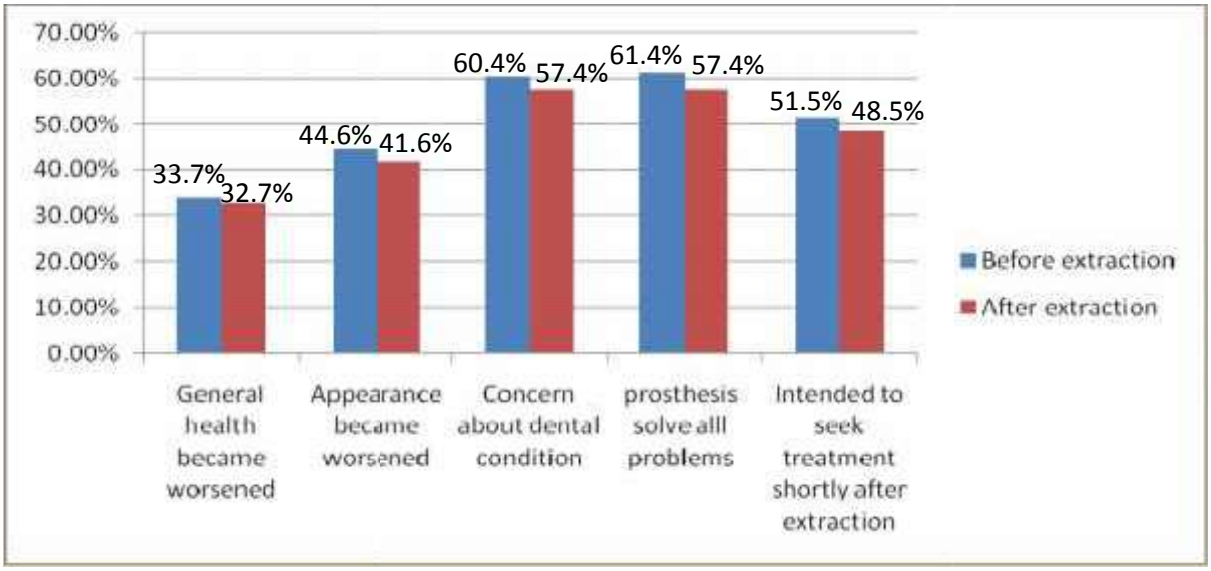
	n %
Benefits of prosthodontic treatment (n=53)	
Esthetics	31(53.5%)
Oral comfort	11(18.8%)
Better function	37(63.6%)
Better self-esteem	9(15.3%)
Better general health	5(8.6%)
Psychological benefits	20(34.3%)
Social benefits	7(11.9%)
Risks of prosthodontic treatment (n=53)	
Oral discomfort	35(60.4%)
Poor adaptation to dentures	20(34.4%)
Others	10(17.2%)
Injuries	5(8.6%)
Rejection	4(6.8%)
Iatrogenic	4(6.8%)
Harm to general health	6(10.4%)
Reason for not treating edentulous spaces previously (n=58)	
Financial limits	9(26.74%)
Don't know	11(32.68%)
Poor interest	7(20.19%)
Poor motivation	5(14.85%)
Negative past experience	2(5.94%)
Didn't feel necessary	11(32.67%)
Others	2(5.94%)

Table 4. Clinical aspects of edentulous spaces and replacement of extracted teeth (n=53).

	n %
Position	
Anterior	17(16.8%)
Posterior	29(28.7%)
Both	7(6.9%)
Arch	
Upper	31(30.7%)
Lower	15(14.9%)
Both	7(6.9%)
Extension	
Large	3(3.0%)
Medium	15(14.9%)
Small	35(34.7%)
Number	
Single	33(32.7%)
multiple	20(19.8%)

*Untreated patients after one month interval = 48(47.5%)

Graph 1: Comparison of self-perception of patients at the time and after teeth extraction.



Discussion

Decrease in self-perceived prosthodontic needs in patients has determined effect on oral health related to quality of life and measures [15, 16]. Hence, patients should understand the potential impacts of tooth loss. This study assess the patients self perception of prosthodontic need during and after extraction. The self perception of patient's impacts was higher at the time of extraction from 33.7% to 61.4% than after 3 months interval of extraction. Almost

half of the patient's undergone prosthodontic treatment of edentulous spaces at the time of extraction and after extraction. Most of the patients preferred fixed prosthodontic treatment. Before extraction, 52% of patients are satisfied with the dental condition and 54% of patients were not satisfied with the dental condition. Most of the patients believed that tooth loss does not affect the digestion, General health and appearance before and after extraction. Their result was in-accordance with the previous studies by Lineia Tavares.et al in 2007 [17]. Even though patients believed that prosthesis solve all problems and does not have potential risk, only half of the patients considered immediate replacement are necessary before and after extraction. Intention of prosthodontic need was greater only at the time of extraction. It is in accordance with the study by Trovik et al [18]. The patients believed that aesthetics and better function as the benefit of prosthodontic treatment whereas oral discomfort and adaptation to dentures as the risk. The patients are more reluctant and don't feel necessary to treat edentulous spaces. This result was not in accordance with the previous study by McGrath and Bedi [19]. It may be because of poor knowledge about the impacts of tooth loss and socio economical status of the patients [20].

Conclusion

Self- perception about tooth loss and prosthodontic needs at the time and after an interval of 3 month from extraction didn't have much significant change among patients. Better function and oral discomfort were mentioned as major benefit and risk of prosthesis. Treatment demand was low due to lack of knowledge and awareness, poor interest and motivation.

Corresponding Author

M.Sankari,

Professor,Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University,Chennai, India

162,Poonamallee High Road,Chennai-600077,Tamil Nadu,India.

Email Id: dr.sankarim@gmail.com

Contact Number: 9176098012

References

1. Gupta S, Mantri SS, Bhasin A. Knowledge and Attitude towards Prosthodontic Rehabilitation and Utilization of Dental Services by Central India Population of Jabalpur City, India. Annals of Medical and Health Sciences Research. 2018.Raj N, Reddy N, Japatti S, Thomas M, Uthappa R. Knowledge, attitudes towards prosthodontics rehabilitation and utilization of dental services among Songadh and Amargadh Population. J Dent Med Med Sci. 2014;3(1):1-6.
2. Akeel R. Attitudes of Saudi male patients towards the replacement of teeth. J. Prosthet. Dent. 2003;90:571-577.
3. Maupome G, Mac Entee MI. Prostho-dontic profiles relating to economic status, social status, social network and social support in an elderly population living independently in Canada. J. Prosthet. Dent. 1998;80:598-604.
4. Allen PF, McMillan AS, Walshaw D. A patient-based assessment of implant-stabilized and conventional complete dentures. Journal of Prosthetic Dentistry. 2001;85:141-147.
5. MDSMDACUVARMA AR. Evaluation of Esthetic Rehabilitation of Teeth with Severe Fluorosis Using Direct and Indirect Laminate Veneer: A Case Report. Biology and Medicine. 2016;8(7):1.
6. Lath V, Jena P, Vaidya NA, Vaidya VN, Sharma S, Baid GC, Singh S, Gupta N. Knowledge, awareness, and perception towards prosthodontic treatment and services among the natives of Durg, Chhattisgarh. JOURNAL OF EVOLUTION OF MEDICAL AND DENTAL SCIENCES-JEMDS. 2017 Apr 10;6(29):2377-82.
7. Balsi TJ, WolfingerGJ . Patient attitude before and after dental implant rehabilitation. Implant Dent. 1994;3:106-109.
8. Chowdhary R, Mankani N, Chandraker NK. Awareness of dental implants as a treatment choice in urban Indian population. Int. J. Oral Maxillofacial Implant. 2010;25:305-308.
9. Macek MD, Cohen LA, Reid BC, et al. Dental visits among older U.S. adults, 1999: the roles of dentition status and cost. J Am Dent Assoc 2004;135(8):1154- 62.

10. Witter DJ, HeldermanWHvP, Creugers NHJ, Kayser AF. 1378 RidwaanOnuiretnl E.D.J. Vol. 49. No. J The shortened dental arch concept and its implications for oral health care. Community Dent Oral Epidemiol 1999:27:249-258.
11. Carlsson GE. Clinical morbidity and sequelae of treatment with complete dentures. J Prosthel Dent 1998;79:17-23.
12. Davis DM. The shift in the therapeutic paradigm: Os-seointegration. J Prosthet Dent 1998; 79: 37-42.
13. Davis DM, Fiske J, Scott B, Radford DR. The emotional effects of tooth loss in a group of partially dentate people: A quantitative study. Eur J Prosthodont Restor Dent 2001; 9:53-57.
14. Leles CR, Freire MCM. A sociodental approach in prosthodontic treatment decision making. J Appl Oral Sci 2004; 12:127-132.
15. Gilbert GH, Meng Xiaoxian M, Oucan RP, Shelton BJ. Incidence of tooth loss and prosthodontic dental care: effect on chewing difficulty onset, a component of oral health-related quality of life. J Am Geriatr Soc 2004; 52:880-888.
16. John MT, Koepsell TO, Hujoel P, Miglioretti OL, LeResche L, Micheelis W. Demografic factors, denture status and oral health-related quality of life. Community Dent Oral Epidemiol 2004; 32:125-132.
17. Teófilo, Linéia Tavares, &Leles, Cláudio Rodrigues. (2007). Patients' self-perceived impacts and prosthodontic needs at the time and after tooth loss. Brazilian Dental Journal, 18(2), 91-96. https://dx.doi.org/10.1590/S0103-64402007000200001.
18. Trovik TA, Klock KS, Haugejorden O. Predictors of Norwegian adult patient' perceived need for replacement of teeth at the time of extraction. Community Dent Health 2002; 19:79- 85.
19. McGrath C, Bedi R. Severe tooth loss among UK adults - who goes for oral rehabilitation? J Oral Rehabil 2002; 29:240-244.
20. Mishra D, Singh HP, Kuppuswamy S. Socioeconomic status scale - A revision. Indian J. Pediatr. 2003;70:273-274.

Survey:-Premedication and Post-Operative Complication in Conscious Sedation

K. Pavithra[1], Dr.Kanthaswamy[2], M S Nivedhitha[3]

Graduate Student[1], Senior Lecturer[2],Department of Pedodontics[2],Professor[3],Department of Conservative and Endodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: Dental patients with generalised anxiety where treated with nitrous oxide sedation method to reduce the anxiety level. The use of premedication before proceeding nitrous oxide sedation will fasten the onset of sedationDental fear and anxiety is a common problem in pediatric patients. There is considerable variation in techniques used to manage them. Various sedation techniques using many different anesthetic agents have gained considerable popularity over the past few years. Children are not little adults; they differ physically, psychologically, and emotionally. The purpose of this review is to survey recent trends and concerning issues in the rapidly changing field of pediatric sedation. We will study the topic from the perspective of an anesthesiologist. It will also provide information to practitioners on the practice of conscious sedation in dentistry and will also outline the route of administration, pharmacokinetics, and pharmacodynamics of various drugs used.. Knowledge about the post-operative care and management of post-operative complications are needed for the post care of the proper procedure.

Materials and Methods: Questionnaire were framed regarding suggestion of the drug, its usage, preferred route of administration,common complications encountered and circulated among the dentist and postgraduate students.100 dentist and postgraduate students participated in the questionnaire survey and they gave their opinion regarding the premedication and post operative complication of conscious sedation based on their clinical experience.The data collected from these 100 dentist and post graduate students of the same department were then statistically analysed to generate appropriate results regarding the questionnaire study.

Result: Based on the statistical analysis,60% of the dentist assure that premedication is safe for conscious sedation,whereas 20% of the dentist and post graduates state that only after proper history and other have no awareness about premedication for conscious sedation.

Conclusion: Over view of this survey is about the knowledge of premedication and post-operative complication towards dentist,since dentist practise in their clinicare aware about the premedication of conscious sedation and the survey shows that dentist have knowledge about the premedication and post-operative complication of conscious sedation.

Key Words:*Conscious sedation uses, Premedication, Post-op complication, Nitrous oxide and Midazolam.*

Introduction

Current understanding of pediatric oral health includes absence of dental fear and anxiety as well as healthy oral structures with the aim of forming the basis for good oral health throughout life. This implies two main dimensions in pediatric oral care which is to keep the oral environment healthy and to keep the patient capable of, and willing to utilise the dental service.[1]Dental anxiety can be managed either by non-phamacological methods like behavior therapy, desensitization or by pharmacological means that include conscious sedation techniques using inhalation sedation (nitrous oxide/oxygen mixture), oral or intranasal sedation (midazolam), intravenous sedation (midazolam) and general anaesthesia. The goal of conscious sedation is to alleviate fear and anxiety in order to facilitate treatment and it serves only as an adjunct to behavioural shaping techniques, and not a replacement.

It is widely recognised that the level of caries in children of various nations has dropped substantially over the last few decades[2].Unfortunately a significant proportion of these children still have caries which remains untreated.This represents a significant problem, if dentine caries is left it will usually lead to pain and sepsis which can often only be managed by extraction or extensive restoration of the affected teeth[3]Either conscious sedation or pre-medication with pharmacological agents is often recommended to children who are particularly fearful or anxious or for whom a disability prevents their being able to cooperate ,during the dental treatment.[3]The treatment and alleviation of pain is a basic human right

that exists regardless of age and demands treatment for this reason alone. Therefore all children should expect painless, high quality dental care.[4].

Sedation is required for some pediatric patients in order for the dentists to be able to deliver high quality, pain-free dental care. Sedation is used for pain control in form of local anesthesia, and for behaviour management [4]. Sedation is the production of a depressed state involving a lack of total consciousness. In sedation, the protective pharyngeal and laryngeal reflexes are not dulled, so the patient can maintain his or her own airway. In this sedated state, the functional activity of the higher centre of the central nervous system isreduced without distortion of the vital functions. The aim of this study is premedication and post-operativecomplications in conscious sedation.

Result:

Table 1: Premedication is safe for conscious sedation

Agree	After proper history	No	No idea
60%	20%	0.9%	9.1%

Table 2: Choice of drug for premedication

Anti emetics	Sedative	Antibiotic	Anticholinergic
75%	20%	15%	2%

Table 3: Major outcome of premedication

Fast onset of sedation	Post operative easiness	Both	None of the above
60%	23%	15%	2%

Table 4: Route of administration for conscious sedation

Inhalation	Intravenous

98%	2%

Table 5: Drug for conscious sedation

Nitrous oxide	Midazolam	Ketamine
95%	3%	2%

Table 6: Postoperativecomplications

Nausea	Vomiting	Post operative sleepiness	Diffusion hypoxia	Hypersensitivity
55%	25%	15%	3%	2%

Discussion

The majority of studies involved sedation in children less than 6 years of age, because this age range belongs to a 'pre-cooperative' group. Treatment needs and management of children will vary as they grow and develop[5].Overall goal of premedication of conscious sedation in pediatric dentistry is to provide a pain free treatment and to relieve the fear and anxiety of the pediatric patients so that the patient feels comfortable in the dental environment and willingly comes back to the clinic for regular checkups without any reluctance[5].The premedication of conscious sedation could increase the success rate of the treatment with ease of operation for the dentist while performing the dental procedure[6].Based on the statistic analysis,60% of the dentist assure that premedication is safe for conscious sedation,whereas 20% of the dentist and post graduates state that only after proper history and other have no awareness about premedication for conscious sedation.(Table 1), A.L. Halosthane survey 95.6% dentist have said that only after proper history of the patient Premedication can be given but in this survey 60% of the dentist agree with premedication is safe for conscious sedation. Drug of choice for premedication is sedation,anti emetics, antibiotic and anticholinergic among this anti emetics is widely preferred drug for premedication which is of 75%, next choice of drug is sedation which is 20%whereas antibiotic and anticholinergic is of 3% and 2% which is less used drug for premedication(Table 2). In Hosey F.G survey most commonly used drug of choice is nitrous oxide with opioids midazolam to increase the concentration.Based on

statistic analysis of major outcome for premedication among dentist and post graduate is 60% fast onset of sedation,23% post operative easiness,15% both whereas 2% have no awareness about major outcome of premedication(Table 3). The Most prevalent route of administration of the sedative drug used. 98% of the drug was administered by inhalation method whereas 2% of the drug was administered by intravenous route. The dentist also mentioned that during administration of the drug assistance from specialised anesthetist was also taken so that to increase the efficacy of the sedative drug and to maintain thevital signs of the patient in a safer manner(Table 4). Based on the statistical analysis,95% of the dentist and the postgraduate students prefer nitrous oxide for conscious sedation during their dental treatment.It is most commonly used drug and it is preferred by large number of the dentist population. 3% of the dentists prefer Midazolam to control their pediatric patients, whereas 2% of them prefer ketamine for conscious sedation in pediatric patients.(Table 5).Post operative complications in conscious sedation is nausea,vomiting,hypersensitivity,diffusion hypoxia and post operative sleepiness in this 55% nausea is the common post operative complication of conscious sedation next common most complication is vomiting which is 25% and others 15% post operative sleepiness, 3% hypersensitivity,2% diffusion hypoxia is less common post operative complication of conscious sedation(Table 6)

Routes of administration Oral Midazolam is the most commonly used oral agent. It produces earlier sedation, more complete amnesia and improved awakening when compared with diazepam. The onset is 60- 90 seconds and the duration of action for small doses is 10-15 minutes. Oral sedation is easy to administer and monitor, and it costs less. But the level of sedation cannot be easily changed and there is no analgesic effect. Inhalation It is a dependable and simple route of drug administration. As a general rule, the drugs delivered through this route have a very rapid onset and short recovery period[7]. Their effect may be rapidly reversed by lowering the concentration of the agent or discontinuing it entirely and administering only oxygen or room air.Nitrous oxide is the most popular agent used through this route. But it has an inherent disadvantage of being the weakest agent available today. Nitrous oxide administration requires special equipment and training. Although it has a wide safety margin, it could prove dangerous and sometimes even fatal at the hands of an untrained individual. Parenteral administration Intravenous sedation The standard technique is the use of titrated dose of a single benzodiazepine or opiod, like fentanyl, etomidate and propofol. The actions can be reversed by using agents like naloxone, a competitive antagonist of

opiodreceptors and flumazenil, a pure benzodiazepine antagonist[7]. Other routes include intramuscular and subcutaneous administration. But they are not commonly practiced.

Nitrous oxide

It is a gas and used as the inhalational anesthetic agent. It has anxiolytic and sedative properties with varying degree of analgesia and muscle relaxation. Recent studies suggest both gamma-aminobutyric acid type A (GABA A) and N-methyl-D-aspartate (NMDA) receptors are affected[8]. It has a long history of safe use providing moderate sedation for minimally moderately painful procedures. Care must be taken when used in addition to other sedatives where deep sedation can easily result. Currently, available nitrous oxide/oxygen delivery systems are manufactured with oxygen fail-safe devices that stop the flow of nitrous when the flow of oxygen is stopped, thus preventing this catastrophe. It should be the first choice for pediatric dental patients who are unable to tolerate local anesthesia alone and have sufficient understanding to accept the procedure[8]. It may be offered with mild to moderate anxiety to better accept the treatment which may require a series of visits. It can also facilitate the provision of more complex time-consuming procedures and dental extractions particularly for young and anxious patients undergoing orthodontic extractions.

Ketamine

It is a dissociative agent, which makes a state of catalepsy that gives sedation, control of pain and amnesia. Ketamine has advantages over other drugs in its relative cardiovascular steadiness and restricted affect on the respiratory mechanics. Recovery occurs in 30–120 min, which allows for patient discharge in a reasonable time after the procedure. It is a dose-related cardiovascular stimulant. Even in children with congenital heart disease, it caused clinically only minor increases in heart rate and mean pulmonary artery pressure during catheterization. More than 11,000 reported cases of its use in children with no reported fatalities have been described in the literature by Green. The most frequently cited disadvantage is the emergence phenomenon, seen more commonly in adults (5–50%) than children (0–5%)[9]. Ketamine causes an increase in salivary and tracheobronchial mucus gland secretions, so an antisialogog is recommended for use with ketamine for GA.Emesis is the third most common side effect of ketamine. In Green's review, he found that the reported incidence of vomiting in children was 10%, and was associated with dental

procedures..Ketamine can be given intramuscularly at 3–4 mg/kg or intravenously at 1–2 mg/kg.

Benzodiazepine

They provide anxiolysis, sedation/hypnosis, skeletal muscle relaxation, anterograde amnesia, respiratory depression, and an anticonvulsant effect[10].but have no analgesic properties. Mechanism of action is through GABA-mediated opening of chloride channels. Benzodiazepines (BZD) have a wide margin of safety between therapeutic and toxic doses. They have high lipid solubility so have a rapid onset of action[11]. They have been widely used in dentistry. They are usually combined with nitrous oxide/oxygen for conscious sedation as additive effect of nitrous oxide to BZD produces analgesic properties.[12] The most common drug used is midazolam, which is having a short duration of action. It is considered as BZD of choice for conscious sedation during treatment in pediatricdentistry.It is given in the form of sweetened syrup given either via a drinking cup or drawn in a needleless syringe and deposited in the retromolar area or oral tablets[13].

Propofol

It is a water-immiscible oil which is formulated as an emulsion with a soya oil base to facilitate injection[14]. The elimination half-life is between 2 and 24 h. However, its duration of clinical effect is much shorter because Propofol is rapidly distributed into peripheral tissue, and its effects, therefore, wear off considerably within even a half hour of injection. This, together with its rapid effect and the moderate amnesia it induces makes it an ideal drug for IV sedation.Sub-anesthetic doses of propofol used for IV conscious sedation infusion facilitated operative dental treatment in anxious children[15]. IV induction by ketamine or propofol remains a problem because of the difficulty in obtaining vascular access in the awake and frightened child. Potent volatile anesthetic agents are used for induction of anesthesia to avoid the struggle to get IV access before the child is asleep[16]. In a study by Arya and Damle in comparison of propofol with midazolam it was found that propofol exhibit rapid onset while having involuntary movements as side effect while midazolam had an edge over propofol in being good anxiolytic and anticonvulsant. With sevoflurane, dose of propofol used is an initial loading dose, (usually 1 mg/kg body weight) and the maintenance dosage needed to achieve satisfactory sedation, ranging from 0.3 to 4 mg/kg/h[17].

Complications in Conscious Sedation

Surgical Factors

As airway is shared by the anesthesiologist and dentist, it may be soiled with blood or debris and stimulation of trigeminal nerve increases chances of arrhythmia during surgery.

Pediatric Issues

1. They may have enlarged tonsils and adenoids thus increasing chances of respiratory obstruction
2. They are uncooperative and communication may be challenging
3. Many medical conditions can co-exist such as epilepsy, reflux, and cardiac anomalies
4. They are needle phobic and highly anxious
5. High autonomic activity thus increasing chances of arrhythmias and vasovagal response
6. Gastric emptying may be delayed
7. Problems of ambulatory anesthesia.

Keeping all the above factors, one should thoroughly prepare the patient after complete pre-anesthetic check-up and after proper examination of airways, cardiorespiratory system, and any congenital abnormalities. Another one of the most important problems is position in the dental chair[17,18,19]. It becomes very difficult to resuscitate the patient if something unwanted happens suddenly[20]. All types of drugs and resuscitative measures should be there in case of any emergency.

Current Status of Conscious Sedation in Pediatrics

Sedation for dental procedure carries high risks for both patient and anesthesiologists. The sedation techniques offer alternative for patients where the use of general anesthesia (GA) is unavoidable.[21] Oversedation or undersedation is unreasonable or unacceptable in some circumstances.[22] In order to decrease dependency of the patient to sedation other psychological methods can be used, e.g. Cognitive reconstructing, hypnosis, relaxation and distraction techniques, systematic desensitization, and conditioning

Conclusion

Over view of this survey is about the knowledge of premedication and post operative complication towards dentist,since dentist practise in their clinicare aware about the

premedication of conscious sedation and the survey shows that dentist have knowledge about the premedication and post operative complication of conscious sedation.

Corresponding Author

M .S.Nivedhitha,

Professor and Head, Department of Conservative and Endodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Science, Saveetha University, Chennai, India

162,PoonamalleeHigh Road, Chennai 600077, Tamil Nadu, India,.

Email Id: nivea16@hotmail.com

Phone Number:9840912367

References

1. A.-L. Hallonsten, B. Jensen, M. Raadal, J. Veerkamp, M.T. Hosey, S. Poulsen. EAPD guidelines on sedation in paediatric dentistry.Pediatric Dentistry. A clinical approach. Copenhagen: Munksgaard, 2001: 226-8.
2. Matharu L, Ashley PF. Sedation of anxious children undergoing dental treatment. Cochrane Database of Systematic Reviews 2006, Issue 1. Art. No.: CD003877. DOI: 10.1002/14651858.CD003877.pub3.
3. M. O. Folayan, A. Faponle& A. Lamikanra. A review of the pharmacological approach to the management of dental anxiety in children. International Journal of Paediatric Dentistry 2002; 12: 347–354.
4. Joseph P. Cravero, MD, and George T. Blike, MD. Review of Pediatric Sedation. International Anesthesia Research Society, Anesth Analg 2004;99:1355–64.
5. HOSEY, M. T.Managing anxious children: the use of conscious sedation in paediatric dentistry. International journal of paediatric dentistry Volume 12(5) September 2002; 359–372
6. Michael F. Hasty, MS William F. Vann, Jr., Diane C. Dilley, Jay A. Anderson. Conscious sedation of pediatric dental patients:an investigation of chloral hydrate, hydroxyzine pamoate,and meperidine vs. chloral hydrate and hydroxyzine pamoate. The American Academy of Pediatric Dentistry Volume 13, Number 1, 10-1
7. JamesA. Roelofse,* LouisR.Louw, and PieterG.Roelofse. A Double Blind Randomized Comparison of Oral Trimeprazine Methadone and Ketamine Midazolam

for Sedation of Pediatric Dental Patients for Oral Surgical Procedures.American Dental Society of Anesthesiology 1998 AnesthProg 45: 3-11

8. Stephen Wilson. A survey of the American Academy of Pediatric.Dentistry membership: nitrous oxide and sedation. American Academy of Pediatric Dentistry. 1996 Pediatric Dentistry - 18:4, 287-293
9. Stephen Wilson, Kelly Farrell, Ann Griffen, Dan Coury. Conscious sedation experiences in graduate pediatric dentistry programs. American academy of pediatric dentistry. Pediatric dentistry 2001, 23:3 307-314
10. Wes McCann, Stephen Wilson, Peter Larsen, Bill Stehle. The effects of nitrous oxide on behavior and physiological parameters during conscious sedation with a moderate dose of chloral hydrate and hydroxyzine. American academy of pediatric dentistry. Pediatric dentistry 1996, 18:1, 35-41
11. PattarawadeeLeelataweewud, William F. Vann, Diane C. Dilley, Warner J. Lucas.
12. The physiological effects of supplemental oxygen versus nitrous oxide/oxygen during conscious sedation of pediatric dental patients.American academy of pediatric dentistry. Pediatric dentistry 2000, 22:2, 125-133
13. Paterson SA, Tahmassebi JF. Pediatric dentistry in the new millennium: Use of inhalation sedation in pediatric dentistry. Dent Update 2003;30(7):350-6, 358.
14. Crowe S, Lyons B. Herbal medicine use by children presenting for ambulatory anesthesia and surgery. PaediatrAnaesth. 2004;14:916–919. [PubMed]
15. Kearon C, Hirsh J. Management of anticoagulation before and after surgery. N Engl J Med. 1997;336:1506–1511. [PubMed]
16. Fleisher LA. Risk of anesthesia. In: Miller RD, editor. Anesthesia. 5th Edition. Philadelphia: Churchill-Livingstone; 2000. pp. 795–823.
17. Cohen MM, Duncan PG, Tate RB. Does anesthesia contribute to operative mortality? JAMA. 1988;260:2859.[PubMed]
18. Khuri SF, Daley J, Henderson W, et al. The National Veterans Administration Surgical Risk Study: risk adjustment for the comparative assessment of the quality of surgical care. J Am Coll Surg. 1995;180:519–531.[PubMed]
19. Klotz HP, Candinas D, Platz A, et al. Preoperative risk assessment in elective general surgery. Br J Surg. 1996;83:1788–1791.
20. Eagle KA, Berger PB, Calkins H, et al. ACC/AHA Guideline Update for Perioperative Cardiovascular Evaluation for Noncardiac Surgery—Executive

Summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Update the 1996 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery) AnesthAnalg. 2002;94:1052–1064.

21. Marickar RF, Geetha RV, Neelakantan P. Efficacy of contemporary and novel intracanal medicaments against Enterococcus faecalis. Journal of Clinical Pediatric Dentistry. 2014 Sep 1;39(1):47-50
22. Rennotte MT, Baele P, Aubert G, Rodenstein DO. Nasal continuous positive airway pressure in the perioperative management of patients with obstructive sleep apnea submitted to surgery. Chest. 1995;107:367–374.
23. Aishwarya AS, Gurunathan D. Stress level in dental students performing pedodontic procedure.Journal of Advanced Pharmacy Education & Research| Jan-Mar. 2017;7(1).

Prevalence and Predictive Factors Of Dental Anxiety Among Adolescent Patients Attending A Dental Hospital In Chennai, India

Anitha.D.R[1], Saravana pandian.K[2], Sri Sakthi.D[3]
Graduate Student [1],Senior Lecturer [2],Department of Orthodontics,
Reader [3],Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Dental anxiety affects a significant proportion of people of all ages from different social classes.It remains to be a serious concern for both dentists and patients for the provision of routine dental care on day to day basis.Anxiety and fear towards dental treatment are common problems frequently experienced by adolescent patients. Occurrence of dental anxiety has attributed to factors like personality characteristics; traumatic dental experiences in childhood, vicarious learning from family members anxious to dental treatment, blood injury fears and pain reactivity.To determine the prevalence and predictive factors of dental anxiety among adolescent patients attending a dental hospital

Materials and Methods: A pretested, interview administrated questionnaire was used among 50 adolescent patients, attending the pedodontic clinic of Saveetha Dental College, Chennai. Descriptive statistics were computed using frequency, inferential statistics were done using Chi square test.

Results: Total of 50 participants completed the questionnaire. Out of 50, 24(48%) were males and 26(52%) were females. Comparing the genders, dental fear was high in female patients 25(50%), and it was high after treatment under local anaesthesia28(56%) and for patients who had appointment within last year 33(66%).

Conclusion: Analysing the results of the study, it can be concluded that factors like, number of previous dental treatments,post traumatic dental experiences, personality, gender, age and education level significantly affected dental anxiety. Dentists attending such young patients should bear all these factors in mind, to provide a quality treatment which will be totally acceptable by the patient.

Keywords: *outpatients, anxiety, phobia, traumatic experience, adolescents*

Introduction

Anxiety can be defined as a state of unpleasantness associated with fear of danger from within or a learned process of one's own environment. It depends on the ability to imagine[1]. Dental anxiety has been mainlyassociated with poor oral health.Avoidance of the dental treatment was high in anxious patients and they also had increased caries rate and DMFS scores. The long-term consequences for the dentition explains about the role of dental anxiety which leads to increased in usageof antibiotics and analgesics[2].

Dental anxiety is defined as state of anxiety as it occurs due to the dental treatment procedure and it is related with negative expectations which are often linked to earlier traumatic experiences, negative attitudes in the family, fear of pain and trauma and perceptions of an painful previous dental treatment. Anxiety is one of the most common problems encountered in the dental operatory and it is a challenge for the Paediatric Dentist, as many children who are extremely anxious totally refuse the dental treatment[3].

Dental anxiety is defined as "an abnormal fear or dread of visiting the dentist for preventive care or therapy and unwarranted anxiety over dental procedures" and may have psychological, cognitive and behavioural consequences. Dental anxiety can prevent patients from cooperating fully during dental treatment[4]. Moreover, anxious people tend to overestimate pain and discomfort caused by dental treatment and may also postpone or miss appointments, with negative consequences for their oral health and often having to incur more complex interventions, thereby entering a vicious cycle that tends to intensify anxiety with regard to treatment[5].

Dental anxiety is a common problem that affects people of all ages and appears to develop mostly in childhood and adolescence. Childhood dental anxiety is not only distressing for the child and family but is also associated with poor oral health outcomes and an increased reliance on costly specialist dental services.Thus, the frequency of dental diseases and unpleasant dental experiences is greater among children with more anxious and uncooperative behaviour in comparison to non-anxious children[6].

Dentists need to understand the anxiety and its repercussions in dental care and need to know how to identify behaviour that indicates anxiety so that a relationship of trust may be established with the patient for the implementation of strategies aimed at minimising the anxiety caused by dental treatment.Questionnaires or scales constitute the most common means of assessing dental anxiety. [7], [8]

Although the aetiology of dental anxiety in children is still not completely understood, it has been a subject of concern for countless researchers over numerous years[9]. Anxiety is “a multi-system response to a supposed threat or danger; it comprises a combination of biochemical changes in the body and aspects of the patient's personal history, memory and the social situation”[10]. “Dental anxiety is associated with symptoms that include at least four of the following: palpitations, sweating, trembling, shortness of breath, sense of choking, chest pain, nausea or other stomach upset, dizziness, a feeling of being detached from the world, being unable to think, fear of dying, numbness or tingling, cold or hot flushes, easily tired, trouble concentrating, irritability, muscle tension, restlessness, and sleep problems”[11].

Dental anxiety is expected to start during childhood and increase over time in the absence of diagnosis and management[12]. This anxiety is relatively common in the dental setting: “the British national children's dental health survey found that the proportion of children who were dentally anxious steadily increased through the primary school years and then levelled off during the secondary school years to about 50% of the population”[13]. “A study of eight European countries found that 35% of 5-year-olds and 21% of 12-year-olds were fearful before visiting the dentist, whereas 48% of children in Dubai were anxious even before visiting the dentist”[14]. Most people feel that their dental fears began in childhood[15].

The disturbing natures of some dental procedures could be considered as the supreme anxiety-provoking stimuli[7]. A study in 2002 found that children with many carious lesions at the age of five years are likely to be dentally anxious at 10 years of age, probably because they have had pain and other negative treatment experiences[15]. Dental anxiety is the basis of avoiding dental visits, as reported by numerous studies[16]. Furthermore, dental anxiety has been reported to decrease with repeated exposure to dental treatment, possibly due to habituation.

Negative thoughts were associated with patients with dental anxiety; anxious and calm patients were found to differ dramatically on a sum of other cognitive reasons[14]. Anxious patients are supposed to be less capable of continuing treatment plans that could help to reduce the anxiety during dental treatment; they expect “dental treatment to be more painful and more traumatic, they expect themselves to be more difficult to treat, and expect to be more nervous and helpless”[13]. Several studies reported that dental anxiety is more prevalent in females than in males[17]. Consequently, the objective of this study was to assess dental anxiety and the potential causative factors in a female population.

The prevalence of dental anxiety among children varies according to the methodology employed and the age of the subjects. A study with Taiwanese children aged 5-8 years found a 20.6% prevalence rate of dental anxiety[9]. Dental anxiety can be considered a common condition, affecting approximately 9% of children and adolescents in Europe and in countries such as Australia, Canada and the United States [10].

In a study by Ost, it was reported that almost 20% of dental phobic were in the age group of 14[18]. similarly, Milgrom reported that 33.3% of the individuals whom he studied, became anxious during adolescence [19]. The nature of dental anxiety is more often related to age of onset; and it is believed that child-onset are more often due to past traumatic dental experiences and the later-onset are more likely to be constitutional vulnerability to the anxiety disorder [20].

Voluminous work has been conducted, correlating fear, anxiety and dental treatment. But the age group between 10 – 17 years has not been studied much in a hospital environment in India. Hence this study was planned to conduct in outpatients attending Pedodontic O.P of Saveetha Dental College in Chennai. Once the prevalence and predictive factors for dental anxiety are listed out, it would help the clinician to execute a tailored intervention and behaviour management for each patient.

Materials and Methods

Study Design: An observational study

Study Area: Pedodontic outpatient clinic, Saveetha Dental College, Chennai, Tamil Nadu.

Study Population: Adolescent patients aged between 10 to 17 years visiting the hospital were included in the study

Inclusion Criteria: Patients attending the O.P during the scheduled days of the study, who were in the designated age group, those who were willing were included in the study

Exclusion Criteria: Patients who had already participated in similar studies. Participants who dint produce a written informed consent were excluded from the study.

Survey instrument: A pretested, interview administrated questionnaire was circulated among 50 adolescentpatients visiting pedodontic outpatient clinic i.e.24 male children and 26 female children, age of 10 to 17 years, who visits saveetha dental hospital. The 7 item questionnaire was designed to elicit the prevalence and predictive factors regarding dental anxiety.

Scheduling: Data collection was scheduled for a period of one week.

Sample and Sampling methodology: Sample size was calculated based on the study by Arshia [22] at 80% power and 5% alpha. A convenience sampling methodology was adopted.

Statistics: Data was entered using micro soft excel. Descriptive statistics (frequency and percentage) were used to project the results

Results

Out of the 50 patients who participated in the study, 26 (52%) were females and 24(48%) were males. 22(44%) belonged to the age group 10 – 13 years and 28(56%) belonged to the age group 14-17 years.

Table– 1- Gender wise predictive factors for causing dental anxiety among study subjects

Factors responsible for Dental anxiety	Yes		No	
	Male n (%)	Female n(%)	Male n(%)	Female n(%)
Fear unrelated to dentistry	1(2)	5(10)	43(86)	1(2)
Previous dental treatment with	32(64)	8(16)	2(4)	8(16)

local anaesthesia				
Present dental treatment with local anaesthesia	20(40)	8(16)	17(34)	5(10)
Dental appointment within last year	20(40)	13(26)	10(20)	7(14)

Table – 2- Degree of Dental Fear among Study Subjects and Their Parents

	High		Low	
Parent's dental fear	40%		20%	
Children's dental fear	Male n(%) 10(20)	Female n(%) 25(50)	Male n(%) 5(10)	Female n(%) 10(20)

Discussion

Current study had totally50 respondents, 24 were males and 26 were females,the response rate was 80%. In our study 30% of the adolescents have high degree of dental anxiety, where as in the study by Krista Baier [21]20% of the children had high dental fear. This discrepancy could be due to the difference in type of and number of invasive procedures undergone by the study subjects in both studies.The children and adolescents were most often accompanied by their mothers during dental care (87%). Maternal dental anxiety is likely to be associated with the avoidance of dental treatment, which could prevent children/adolescents from receiving appropriate dental care[20].This aspect gains further importance when considering the fact that women exhibit higher levels anxiety than men and are generally in charge of accompanying their children in health-related matters.

The tendency toward an association between the need for invasive treatment and the anxiety scores of children and adolescents regarding dental treatment (P = 0.08) demonstrates that, even with the progress gained in minimally invasive dentistry, the use of local anaesthesiaremains closely associated with unpleasant experiences regarding dental treatment and dental anxiety [21],[22].

Fear reported by other family members is one of the factors most often associated with dental fear and anxiety in children. Indeed, negative attitudes and experiences transmitted by mothers and their opinions regarding dental treatment are considered etiological factors of dental fear and anxiety in children.A previous study found that children whose mothers exhibited a moderate or high level of dental anxiety were more likely to have untreated dental caries in comparison to children whose mothers had a low degree of dental anxiety.

When dental anxiety prevalence and gender was taken in to consideration. Our study results were in agreement with other studies [22]. In the current study 12% of the adolescents had phobia unrelated to dentistry, this was in contrast and lower when compared to the study by Corah NL [23]. The difference could be due to the difference in culture, geographical variations and socio demographic factors between the study populations.

In our study, children who received treatment under local anaesthesia behaved negatively 4 times higher than those who dint receive treatment under local anaesthesia. This was twice higher than the study results by Nakai [24]. Children who had painful experience at an early age are an important factor related to the dental anxiety.In our study we found that children with previous dental experience had significantly has dental fear than those without. This result was in accordance with the study byNicolas [25].Dental fear or anxiety may be described as a subjective state of feeling or reaction to a known source of danger which lies in the subconscious mind [14]. The most commonly reported source of fear in the present study was ‘fear of specific stimuli’ mainly pain, while other categories such as fear of catastrophe, fear of reprimand for neglect of oral hygiene and generalized fear cause less concern to the surveyed children. Such fear of pain may be a major obstacle to seeking dental care [15] and appears to be strongly associated with extreme deterioration of oral and dental health [16]. The finding that painful dental experiences and expectations of trauma were identified as the major sources of adverse reactions to dentistry in other reports [12, 17].

Another interesting finding in the current study was that, there was an association between the type of treatment done in the first dental visit and dental fear scores in the second dental visit. This was again in accordance with the study by Nicolas [25].

Despite the results obtained from the current study, it carried few limitations in terms of sample selection and sample size, making the results non generalizable to whole adolescent population. Hence a study on larger sample size with a probability sampling technique will ensure generalizable result, which can be used in evidence based practice.

Conclusion

Despite technological advances in modern Dentistry, anxiety and fear are common among children and adults, constituting a significant barrier to dental care, interfering with regular oral health care.Fear is part of childhood development. It is usually transitory and does not produce major disruptions in child's daily life. Fear can generate anxiety, which, in turn, can be defined as a response to a situation in which the source of a threat to the subject is not clearly defined. Although the ability to experience fear is an innate biological function,fear responses to certain objects and situations are mostly acquired through learning.Fear and anxiety can make it difficult, at different levels, for performing children dental treatment, creating an atmosphere of tension for both the dentist and their patients. It can also result in an unsatisfactory treatment or even prevent its fulfilment. Anxiety and dental fear are considered multifactorial conditions; there are several variables that can influence these conditions, such as family interference, socioeconomic factors, age, child's temper, among others [1,8,13,23]. However, few studies assess, in the same group of people, the relationship between fear and anxiety among children and their caregivers, as well as the caregiver's prediction of fear and anxiety experienced by children during dental treatment.

Based on the results of the study, it can be concluded that females have more dental anxiety than male patients and its occurrence is dependent on so many factors like, personality, traumatic dental experiences in child hood, vicarious learning from dentally anxious family members, pain reactivity and etc.

Corresponding author

D.Sri Sakthi

Reader,Department of Public Health Dentistry,

Saveetha Dental College,Saveetha University,

Saveetha Institute of Medical and Technical Science,

Chennai, India.

Email: drsrisakthiphd@gmail.com

Reference

1. Deepak vishwanath, Mahesh kumar, Prabhuji M.L. Dental anxiety, fear and phobia in children: International Journalof DentalResearch and Development. IJDRD 2014; 2(1):2321.

2.James P Zacny, Rebecca J Hurst, Lou Graham, Debra J Janizewski. Preoperative dental anxiety and mood changes during nitrous oxide inhalation. J Am Dent Assoc133; 1: 82-8.

3.Youn-Soo Shim, Ah-Hyeon Kim, Eun-YoungJeon, So-Youn An. Dental fear & anxiety and dental pain in children and adolescents. J Dent Anesth Pain Med 2015; 15(2):53-61.

4.Torriani DD, Teixeira AM, Pinheiro R, Goettems ML, Bonow ML. Cross-cultural adaptation of instruments to measure anxiety and child behaviour in the dental clinic. Archieves in Dentistry 2008;44:17-23.

5.Spielberger CD, Gorsuch RL, Luhehe RE. State-Trait Anxiety Inventory - STAI. Rio de Janeiro: Tradução Biaggio, A.M.B., Natalicio, L. CEPA 1979;55:6.

6.Corah NL. Development of a dental anxiety scale. J Dent Res 1969;48:596.

7.Biaggio AM. Development of the Spielberger State-Trait Anxiety Inventory experimental form in Portuguese. In: Research in Psicology: Development and Personality Ed. 1 Porto Alegre: Federal University of Rio Grande do Sul, UFRGS, Brazil1984;67:7-8.

9.Gorenstein C, Andrade L. Validation of a Portuguese version of the beck depression inventory and the state-trait anxiety inventory in Brazilian subjects. Braz J Med Biol Res 1996;29:453-7.

10. Brazil. Children and Adolescents Statute. Statute of Children and Adolescents and other measures. Eur J Oral Sci 1978;22:5-6.

11.Luoto A, Tolvanen M, Rantavuori K, Pohjola V, Lahti S. Can parents and children evaluate each other's dental fear? Eur J Oral Sci 2010;118:254-8.

12.Ribas T, Guimarães VP, Losso EM. Dental anxiety assessment in children during dental treatment. Archieves in Dentistry. 2006;42:191-8.

13.A.K. Bolin. Children's dental health in Europe. An epidemiological investigation of 5- and 12-year-old children from eight EU countriesSwed Dent J Suppl1997;122: 1-88.

14. Al-Namankany, P. Ashley, A. Petire. Development of the first Arabic cognitive dental anxiety scale for children and young adultsWorld J Meta Anal 2014;2(3): 64-70.

15. M.Raadal, G.V. Strand, E.C. Amarante, G. Kvale. Relationship between caries prevalence at 5 years of age and dental anxiety at 10Eur J Paediatr Dent 2002;3(1): 22-26.

16.Stenebrand A, Wide Boman U, Hakeberg M. Dental anxiety and temperament in 15-year olds. Acta Odontol Scand 2013;71:15-21.

17.Bottan ER, Oglio D, Araújo SM. Dental Anxiety in schoolchildren. Pesq Bras Odontoped Clin Integr, João Pessoa 2007;3:241-6.

18. Ost L. Age of onset in different phobias. Journal of Abnormal Psychology 1987; 96:223-29.

19. Milgrom P, Fiset L, Melnick S, Weinstein P. The prevalence and practice management consequences of dental fear in a major US city. J Am Dent Assoc1988; 116(6):641-47.

20. Moore R, Birn H, Kirkegaard E, Brodsgaard I, Scheutz F. Prevalence and characteristicsof dental anxiety in Danishadults. Commun Dent Oral Epidemiol 1993; 21: 292-6.

21. Krista Baier, BSc, DMD, MSD Peter Milgrom, DDS Lloyd Mancl, PhD Stephen Russell, BSc Toshiko Yoshida, PhD. Children's Fear and Behaviour in Private Paediatric Dentistry Practices: Paediatric Dentistry. 2004; 26:4.

22. Dr.Arshiya Shehenaz, assessment of dental anxiety among 12-15 years old school children, IJRT.2001; 2(8): 2456-3315.

23. Corah NL. Development of a dental anxiety scale. J Dent Res. 1969;48:596.

24. Nakai Y, Milgrom P, Mancl L, Coldwell S, Domoto P, Ramsay D. Effectiveness of local anesthesia in pediatric dental practice. J Am Dent Assoc. 2000;131:1699-1705.

25. Nicholas H, Melamed BG, Hawes RR, Heiby E, Glick J. The use of filmed modelling to reduce uncooperative behaviour of children during dental treatment. J Dent Res. 1975; 67:797-801.

To determine the prevalence and predictive factors of Dental anxietyamong adolescent.	Survey

Prevalence of Chronic Fatigue Syndrome among Dental Graduates-A Study

Priyadharshni S[1], Abilasha[2], E. Roja[3]
Graduate Student[1], Reader[2], Department of Oral Pathology,Reader[3], Department of Public Health Dentistry,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Chronic Fatigue Syndrome also known as Myalgia Encephalomyelitis is a neurological illness. It is a condition that presents long term physical and mental fatigue with associated symptoms of pain and sensitivity across a broad range of systems in the body. The etiology is unknown. The aim of this study is to estimate the prevalence of Chronic Fatigue Syndrome(CFS) in students and relate the incidence to various factors such as sleep, rest, relaxation, diet using a questionnaire .

Materials and Methods: Questionnaire will be prepared using the established factors affecting Chronic Fatigue Syndrome in students of age group of 19-24years will be included in this study. Those who are not willing to participate will be excluded.

Result: The survey found that 49 out of 100 participants are not aware of Chronic Fatigue Syndrome. It is important to know about CFS which helps to reduce the prevalence.

Conclusion: Chronic fatigue is one of the most Perplexing conditions in the domain of medical science.People with CFS who have a thorough knowledge of the illness tend to manage their condition better and have a more positive outlook. Whereas, people in general are not aware of this condition and it is important to know about CFS which helps to reduce the prevalence. Several studies have described how living with a chronic invisible illness results in a multiplicity of stressful consequences and considerable impact on daily life

***Key Words**: fatigue, stress, symptoms, health, illness.*

Introduction

The chronic fatigue syndrome is a clinically defined condition characterised by severe disabling fatigue and a combination of symptoms that prominently features self-reported impairments in concentration and short-term memory, sleep disturbances, and musculoskeletal pain. The extent to which the features of the chronic fatigue syndrome are generic features of chronic fatigue and deconditioning due to physical inactivity common to a diverse group of illnesses must also be established[1,2].

The onset of the condition is usually sudden. is means that the main symptom of fatigue (along with other symptoms) may appear only from a particular point of time before which they did not exist. The onset is usually preceded or accompanied by a " u-like" disease, quite similar to a common cold or viral infection. is has promoted the association of virus and viral infection with the etiopathogenesis of the disease with the postulation that such viral infections somehow cause meningoencephalitis resulting in CFS. Both pathogenic as well as nonpathogenic viruses are sought to be associated with the etiopathogenesis of the disease.In some cases, the onset of the disease follows a prolonged period of adverse stress. The period of stress may be of several months duration and this stress can be either physical or mental, or both. Some authorities consider these as precipitating factors whereas some consider them as actiological factors. The main symptom is fatigue or exhaustion. The fatigue is always of new onset which means that it was not present to any significant degree in the past. The duration of the fatigue is prolonged and to qualify as CFS it must be of more than 6 months duration. The fatigue is usually unexplained. It cannot be attributed to any known or immediate cause. It is not directly related to exertion. It is not as a result of physical exertion, but physical exertion always exacerbates it.It is not significantly relieved by rest unlike the usual fatigue that follows exertion. The level of fatigue is such that it causes significant reduction of previous activity levels of the person.Chronic unexplained fatigue unrelieved by rest that incapacitates the patient is the hallmark of CFS.Pathophysiology of CFS is largely unknown at the present moment. ere are no diagnostic laboratory tests for CFS. In fact, no abnormal lab findings characteristic of CFS has been identified so far. There are two hypotheses which explains the Pathogenesis of CFS which includes two categories: a) Biomedical

abnormality, oxidative stress, genetic predisposition, infection by viruses both pathogenic and non-pathogenic, infection by bacteria, immune dysfunction b)Hypothalamic pituitary adrenal axis abnormality, psychological factors, psychosocial factors.

Evidence of viral infection with cause and effect relationship has not been found in CFS so far. Similar is the cause of bacterial infection. But that an inflammatory (may not be infective or postinfective) condition exists along with CFS has been surmised from such findings as arthralgia, myalgia, u-like prodrome and tender lymphadenopathy.The role of oxidative stress in CFS is an emerging focus of research. Although it is uncertain whether oxidative stress is a cause or a result of this illness, recent studies have demonstrated that oxidative stress contributes to the pathology and clinical symptoms of CFS. Theoretically, oxidative stress can be caused by an increase in the generation of reactive oxygen species, of which mitochondrial dysfunction is believed to be a main source, or it can be caused by a decline in the efficiency of antioxidant enzyme systems. Recent studies have examined both of these possibilities by looking for markers of oxidative stress and protective antioxidant systems[3]. To identify theprevalence of chronic fatigue is critically essential andimportant in theprocess of chronicfatiguetargetingstudies andtherapeuticdevelopments.Thisstudy aimed toreviewthecurrentstudiesfortheprevalenceof Chronic Fatigue Syndrome among dental graduates[4].

Methodology

Study Design

In order to compare the features of prevalence of chronic fatigue syndrome, among dental graduates,questionnairewas prepared using the established factors affecting this syndrome in students of age group of 19-24yrs. A total of 100 students (convenience sampling) participated in this research of which 42 were males and 58 were females.

Gender-
Age-
Weight-
Height-

1. What are your views on chronic fatigue syndrome?
A) Complex disorder characterised by profound fatigue that is not improved by bed rest
B) Severe fatigue lasting at least 6 months
C) Significant impairment of short-term memory or concentration
D) All of the above

2. .Have you experienced fatigue or tiredness for a prolonged period of time (6 months or more) despite adequate rest and not working overtly hard?
Yes / No

3. How long ago did your problem with fatigue begin?
Less than 6 months / More than 6months / Not having a problem with fatigue

4. Do you currently have a diagnosis of chronic fatigue syndrome(CFS)?
Yes / No

5. Have any of your family members been diagnosed with CFS?
Yes / No

6. I am prone to pimples.
Yes / No / In between

7. Do you have difficulty in concentrating?
Often / Sometimes / Hardly ever

8. I am unable to work full-time.
True / Uncertain / False

9. I am frequently irritable.
True / Uncertain / False

10. Do you feel unrefreshed after sleep?
Yes / No

11. Do you experience headaches?
Often / Sometimes / Hardly ever

12. Do you experience muscle pain?
Often / Sometimes / Hardly ever

13. How frequently do you get sore throat?
Often / Sometimes / Hardly ever

14. Burning sensations in the face, hands or feet.
Yes / no / uncertain

15. Do you feel dizzy when sitting up or standing?
Frequently / occasionally / hardly ever

16. I react badly to. Many chemicals / few chemicals / hardly any chemical

17. How many hours do you sleep daily?
8hours / more than 8 hours / less than 8 hours

18. Are you currently engaging in any form of exercise?
Yes /no

19. Have you ever consulted a medical doctor about your fatigue/energy problem?
Yes / no

20. Do you currently have a medical doctor overseeing your fatigue problem?
Yes / no

21. Are you currently taking any medications for CFS?
Yes / no. If yes.what medicines are you taking?

Figure 1: Questionnaire

Data Analysis

The main objective of this research was to explore and identify the main experiences of CFS for the individual sufferer. The research aimed to explicate the major experiences and events core to the experience of CFS in order to enable psychologists and counsellors to more richly understand the illness effects. 10% of the people have reported that they have muscle pain or myalgia, because of the similarity of symptoms, fibromyalgia and CFS are considered to be overlapping syndromes. In fact, CFS can affect people of any gender, age, race, or socioeconomic group. Only 12% of them are undergoing diagnosis for this syndrome. CFS is usually diagnosed by evaluating symptoms and eliminating other causes of fatigue. Unexplained continuing or recurring chronic fatigue for at least six months that is of new or definite onset, is not the result of ongoing exertion, and is not mainly relieved by rest, and causes occupational, educational, social, or personal activities to be greatly reduced. The survey found that 10% are suffering from Chronic Fatigue Syndrome with fatigue problem for more than 6months. 5% of the people are undergoing medical treatment and medical doctor overseeing this fatigue problem. For patients with CFS, learning to manage activity levels is a key part of managing the illness. This requires a new way of defining exercise and thinking about daily activities. 25% of them are engaged in doing exercise which can manage the illness. It's important, however, not to avoid activity and exercise altogether. Such avoidance leads to serious deconditioning and can actually worsen other symptoms. Sore throat is one common sign of Chronic Fatigue Syndrome. 26% of them reported that they frequently get sore throat. It is often accompanied by a feeling of tenderness in the lymph nodes in your neck. However, to be diagnosed with Chronic Fatigue Syndrome, symptoms must last for six months or more because sore throat is a symptom for many other illnesses. Headache is associated with many of these symptoms as well. This survey says 15% of the CFS patients reported that they feel less refreshed and restored after sleep than they felt before they became ill. Unrefreshing sleep can be present even though medications may help patients achieve required hours of sleep. A sleep specialist should evaluate patients whose sleep remains non-restorative following standard interventions. Memory and concentration complaints are two of the more distressing symptoms reported by people with CFS. Relaxation and meditation training and memory aids, such as organizers, schedulers, and written resource manuals, can be helpful in addressing cognitive problems. Some patients with CFS may also exhibit symptoms of orthostatic instability, in which symptoms are triggered or made worse by standing upright, in particular frequent dizziness and light-

headedness. The survey found that 10% of them feel dizzy when sitting up or standing. When clinicians judge these symptoms to be severe enough to warrant additional assessment, they should refer patients for evaluation by a cardiologist or neurologist. Specific treatment for orthostatic instability should only be started following confirmed diagnosis and by clinicians experienced in evaluating therapeutic results and managing possible complications.The reported incidence of acne contributes to the CFS which Is caused by inflammation and oxidative stress.

Result

The survey found that 49 out of 100 participants are not aware of Chronic Fatigue Syndrome. It is important to know about CFS which helps to reduce the prevalence.

DIAGNOSIS:

CFS can resemble many other disorders, it's important not to self-diagnose CFS. It's not uncommon for people to mistakenly assume they have chronic fatigue syndrome when they have another illness that will respond to treatment.Diagnosis is established through the exclusion of other diseases causing fatigue. This syndrome is rare in childhood and adolescence, although the fatigue symptom per se is quite commonin paediatric patients. Currently, no curative treatment exists for patients with chronic fatigue syndrome[5,6]. The therapeutic approachto thissyndrome requires a combination of different therapeutic modalities. The specific characteristics of the symptomatology of patients with chronic fatigue require a rapid adaptation of the educational, healthcare and social systems to prevent the problems derived from current systems.Despite the concerns of some medical professionals that diagnosing a patient with CFS could be unhelpful, this study revealed overwhelmingly that receiving a diagnosis was very positive for CFS sufferers[7,8,9].

Prevention and Management of Fatigue

It is important to manage fatigue in the context of each patient suffering with it. Treatment of CFS with its various major clinical and functional impacts, should be associated with a ‘biopsychosocial model’ of management. Educating patients about their diagnosis is crucial. Physicians should emphasise distinction among factors that may have predisposed patients to develop, trigger, or perpetuate the illness. Progressive muscular rehabilitation, combined with

behavioural and cognitive treatment, and appropriate choice of medications are essential parts or therapy.[10]Various treatment of CFS has been subjected to randomised controlled trials. However, recent reviews conclude that only cognitive behavioural therapy (CBT) and graded exercise therapy (GET) have a scientifically proven beneficial effect[11,12].Important components of CBT are explanation of pathophysiologic theories on CFS, challenging of fatigue-related cognitions and gradual increase of physical activity. In this way, simply speaking, the patients learn to acquire control over their symptoms.Management of CFS patients should also include attention to possible complications, like secondary depression and dietary deficiencies in the severely disabled. Further, patients need appropriate assistance on social and economical issues. Treatment modalities for patients with CFS are aimed at relief of symptoms rather than curing the disease[13,14]. Common pharmacologic therapies that may be prescribed by a physician include the administration of non-steroidal anti-inflammatory drugs (NSAIDs) for pain relief, low-dose tricyclic antidepressants to improve sleep and relieve mild pain, and anxiolytic agents to treat anxiety. In some cases, serotonin reuptake inhibitors prescribed to non-depressed patients have been found to be beneficial. Furthermore, evidence-based systematic reviews have illuminated the beneficial role of light exercise[15]. Aims of treatment are to reduce levels of fatigue and associated symptoms, to increase levels of activity, and to improve quality of life[16].

Discussion

Fatigue of the dentist is mainly caused by his forced working posture. Although several studies of the cause of the fatigue and its recovery way have been done, it seems that they are not practically applied to the dental field[17]. In this study authors obtained some basic information for the fatigue of dentists and analysed them in view point of the experience year and working posture of dentists.The chronic fatigue syndrome (CFS) is fundamentally characterized by intense fatigue of unknown cause, which is permanent and limits the patient's functional capacity, producing various degrees of disability. It is advisable to differentiate fatigue from other medical concepts with which the symptom is often confused: first, from asthenia, defined as the lack of strength or feeling of inability to carry out daily tasks, which is more intense at the end of the day, and usually improves after a period of sleep; second, from weakness, which is the reduction or loss of muscular strength, and the key symptom in muscular diseases. In addition to fatigue, CFS is associated to a

wide spectrum of symptoms, including arthralgia, muscle pain, headaches, anxiety, depressive symptoms, cognitive disorders, sleep disorders, or intolerance to physical exertion, among the most frequent[18]. CFS is sometimes seen in members of the same family, but there is no evidence that it is contagious; instead there maybe a familial predisposition or a genetic link.Studies suggest that close to 80 percent of people who are living with chronic fatigue syndrome have never been formally diagnosed and are not getting the treatment they need, but data also implies that getting a diagnosis early in the course of the disease before you've had it for two years gives you the best chance of improving your symptoms through treatment[19].

Research suggests that chronic stress is associated with deregulation of the neuroendocrine and sympathetic nervous systems causing changes in the immune system and consequently resulting in an increased risk of periodontal diseases, recurrent aphthous ulcerations, herpes virus in- fections, etc[20]. Moreover, it is known that chronic stress can mediate the risk of periodontitis through changes in health- related behavior, such as oral hygiene, diet and smoking[21,22]. There could be several explanations why the current study failed to find an association between oral hygiene and stress. First of all, our study did not aim to distinguish short-term stress from chronic stress. It is likely that students experienced short-lived stress related to the examination period. Short-term stress, as opposed to chronic stress, has a stim- ulating effect, possibly resulting in unchanged oral health- related behavior. Secondly, stress is often associated with perfectionist personality type[23]. A perfectionist person, especially in the field of dental studies, is self-conscious about personal oral health status and, therefore, such a person is likely to have sufficient oral hygiene habits.Unrefreshing sleep is extremely common in people with CFS. Patients usually report a longer time to fall asleep, an increased time in bed awake, and a broken and restless sleep pattern. A shift from regular night-time sleep to daytime naps and a late-night to late-morning sleep cycle is some- times noted. It is known that chronic disruption of the normal sleep pattern can induce symptoms in healthy volunteers, including fatigue, musculoskeletal pains, irritability and impairment of concentration.The general goals of sleep management are to establish a regular, unbroken, night-time sleep pattern and to improve perceptions of the quality of sleep.

No medication has yet been shown to provide long term remission or “cure” in people with CFS. However, there is a place for symptomatic treatment for relief of specific symptoms if

they are sufficiently distressing. As such treatments for CFS are empirical, each patient should be monitored carefully to ensure that the symptomatic benefits outweigh any side effects.

Many people with CFS report an increased susceptibility to drug side effects, and it is advisable to begin with small doses when introducing new agents[24].

Although depression is a common symptom in people with CFS, the disorder as a whole cannot be regarded simply as a “somatised” variant of a depressive illness. Overall, clinical trials of antidepressant drugs show no consistent pattern of improvement. However, judicious use of particular agents may provide symptomatic improvement in subjective energy (moclobemide), sleep disturbance (amitryptyline, nefazodone), muscle and joint pain (ami-tryptyline), and depressed mood (sertraline, paroxetine, nefazodone). A reasonable approach is to consider under- taking an “N = 1” therapeutic trial of a selected drug based on this broad pattern of effects on brain function. Given that these drug therapies are increasingly varied and complex, there is an important role for the specialist physician or psychiatrist to guide the choice of drugs and their monitoring[25].In people with the overlapping syndrome of fibromyalgia, the use of symptomatic treatments such as analgesics and NSAIDs, in combination with tricyclic agents, can be effective in reducing pain and improving sleep.

Conclusion

Chronic fatigue is one of the most Perplexing conditions in the domain of medical science.People with CFS who have a thorough knowledge of the illness tend to manage their condition better and have a more positive outlook. Whereas, people in general are not aware of this condition and it is important to know about CFS which helps to reduce the prevalence. Several studies have described how living with a chronic invisible illness results in a multiplicity of stressful consequences and considerable impact on daily life. Some people are thought to be more susceptible to the condition because of their genes, as the condition is more common in some families.More research is needed to confirm exactly what causes the condition.

Corresponding Author

E. Roja,

Tutor,Department of Public Health Dentistry,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162 Poonamallee High Road, Chennai 600 077, Tamil Nadu, India
Email Id: itsrojaa@gmail.com
Contact Number: 91 99941 74480.

REFERENCES

1. Alan C. Logan, ND, Cathy Wong, ND (Cand.)Chronic Fatigue Syndrome: Oxidative Stress and Dietary Modifications Altern Med Rev 2001;6(5):450-459)
2. James P.Griffith, M.D.,F.A.C.P.,and Fahd A.Zarrouf,MD A systematic review of chronic fatigue syndrome:don't assume it's depression.(prim care companion J clinc Psychiatry 2008;10:120-128)
3. Vegard Bruun Wyller.The chronic fatigue syndrome –an update ActaNeurolScand2007:115(Suppl.187):7–14.
4. Chang-GueSon.Review of the Prevalence of Chronic Fatigue Worldwide2012. Vol.33. No.2. 25-33 The Journal of Korean Oriental Medicine
5. Kathleen Light2, Alan Light2, Lucinda Bateman3 and Lisa A Cannon-Albright4,5 Evidence for a heritable predisposition to Chronic Fatigue Syndrome Frederick Albright1*, Albright et al. BMC Neurology 2011, 11:62
6. Samantha Johnston1 Ekua W Brenu1 Donald Staines1,2 Sonya Marshall-Gradisnik The prevalence of chronic fatigue syndrome/ myalgic encephalomyelitis: a meta-analysisClinical Epidemiology2013:5 105–110
7. Chronic fatigue syndrome: aetiology, diagnosis and treatment Alfredo Avellaneda Fernández*1, Álvaro Pérez Martín2, MaravillasIzquierdo Martínez3, Mar Arruti Bustillo4, Francisco Javier Barbado Hernández5, Javier de la Cruz Labrado6, Rafael Díaz-Delgado Peñas7, Eduardo Gutiérrez Rivas8, Cecilia Palacín Delgado9, Javier Rivera Redondo10 and José Ramón Ramón Giménez11BMC Psychiatry 2009, 9(Suppl 1):S1
8. Wyller VB. The chronic fatigue syndrome –an update. ActaNeurolScand2007:115(Suppl.187):7–14. 2007 The Author. Journal compilation 2007 Blackwell Munksgaard.
9. MXPatel, D G Smith, T Chalder, S Wessely Chronic fatigue syndrome in children: a cross sectional surveyArch Dis Child 2003;88:894±898

10. Esther M Crawley,1 Alan M Emond,1 Jonathan A C Sterne2Unidentified Chronic Fatigue Syndrome/ myalgic encephalomyelitis (CFS/ME) is a major cause of school absence: surveillance outcomes from school-based clinics BMJ Open 2011;1: e000252.
11. Kecklund G, Åkerstedt T. The psychometric properties of the Karolinska Sleep Questionnaire. J Sleep Res. 1992;1:113.
12. Reeder LG, Chapman JM, Coulson AH. Socioenvironmental stress, tranquilizers and cardiovascular disease. Proceedings of the Excerpta Medica International Congress Series. 1968;182:226–238.
13. De Vries J, Michielsen HJ, Van Heck GL. Assessment of fatigue among working people: A comparison of six questionnaires. OccupEnviron Med. 2003;60:i10–i15.
14. De Vries J, Michielsen HJ, Van Heck GL, Drent M. Measuring fatigue in sarcoidosis: The fatigue assessment scale (FAS). Br J Health Psychol. 2004;9:279–291.P
15. Goštautas A. Psichologiniaistresoaspektai. Sveikata. 1999;6:55–58.
16. Dahlin M, Joneborg N, Runeson B. Stress and depression among medical students: A cross-sectional study. Med Educ. 2005, 39:594–604.
17. Suen LK, Tam WW, Hon KL. Association of sleep hygiene-related factors and sleep quality among university students in Hong Kong.Hong Kong Med J. 2010;16:180–185.
18. Afari N, Buchwald D. Chronic fatigue syndrome: a review. Am J Psychiatr. 2003;160(2):221-36.
19. Carruthers BM, Jain AK, De Meirleir KL, et al. Myalgic encephalomyelitis/ chronic fatigue syndrome: clinical working de nition, diagnostic and treatment protocols (PDF). Journal of Chronic Fatigue Syndrome. 2003;11(1):7-97.
20. Chambers D, Bagnall AM, Hempel S, et al. Interventions for the treatment, management and rehabilitation of patients with chronic fatigue syndrome/myalgic encephalomyelitis: an updated systematic review. Journal of the Royal Society of Medicine. 2006;99(10):506-20.
21. Thenmozhi, P. and Dr. Aruna, S, RISK FOR FALL AMONG PATIENTS IN THE ACUTE CARE SETTINGS, International Journal of Recent Advances in Multidisciplinary Research,Vol. 03, Issue 05, pp.1463-1465, May, 2016

22. Kimberly RW, Teodor TP, Maureen EG, Omar P, Deanna LK, Mark AR. Role of chronic stress and depression in periodontal diseases. Peri- odontol 2000. 2014;64:127–138.
23. Aleksejuniene J, Holst D, Eriksen HM, Gjermo P. Psychosocial stress, lifestyle and periodontal health. J Clin Periodontol. 2002;29:326–335.
24. Ravishankar TL, Ain TS, Gowhar O. Effect of academic stress on plaque and gingival health among dental students of Moradabad, India. J Periodontol. 2014;16:115-120.
25. Luyten P, Kempke S, Van Wambeke P, Claes S, Blatt SJ, Van B. Self- critical perfectionism, stress generation, and stress sensitivity in patients with chronic fatigue syndrome: relationship with severity of depression. Psychiatry. 2011;74:21–30.

Prevalence of Dental Caries Among 9 To 13 Years School Children of Tiruvallur District-Chennai Tamil Nadu

Indhulekha Vimalakshan[1], Deepa Gurunathan[2], Pradeep Kumar[3]
Graduate Student[1], Professor[2], Department of Pedodontics, Professor[3], Department of Community Dentistry
Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Oral health plays an important role in general health of body. Poor oral hygiene can also lead to many systemic diseases. Dental caries is a disease with multifactorial causes. The prevalence and occurrence of dental caries in a population is influenced by many factors such as sex, age, socioeconomic status, dietary patterns and oral hygiene habits. Hence, the present study was designed to assess the prevalence of dental caries in Tiruvallur district Chennai, Tamil Nadu.

Material and Methods: The survey was carried out among school going children of age 6 years and 13 years. 399 healthy subjects including 219 boys and 129 girls from 7 different schools were examined in day light with the help of basic diagnostic instruments like mouth mirror and probe.

Result: In this study it was observed that the 9 and 10 age group of children had high percentage of caries when compared to 11 to 13 years age group of children. The mean dmft score was also high in was also high in 9 and 10 age group of children. In total dental caries was observed in 253(72.21%) study population. Restoration is the most required treatment in both the group.

Conclusion: The result of this study reveals that there are still large amounts of population that lack awareness on the effect of poor oral hygiene and oral health status and also are ignorant on the advantages of having a good health.

Key Words*: oral hygiene, dental caries, school children, prevalence, mouth mirror, probe*

Introduction

Health is a basic human right and oral health is a significant component of general health. Although oral diseases are mostly not life-threatening, they are important public health problems. [1] Dental caries is a multifactorial disease where many risk factors are involved, and it occurs because of an imbalance between the environment and external factors. Accumulation of bacteria especially cariogenic streptococcus mutants, diet, oral hygiene and time play an important role in carious lesion. However, dental caries is not a typical classical infectious disease. [2] Dental caries is specifically increasing in children. 80 to 85% children suffer from this disease and the average number of decayed, missing and filled teeth per child at the age of 12 years is about 4 in rural areas and 5 in urban areas of India with almost no dental restorative help available particularly in the rural areas. [3] Carlos and Gittelsohn (1965) were among the first to group the teeth from most susceptible to least susceptible. According to them the mandibular molars were the most susceptible and the lower incisors were least susceptible. The most frequently affected site is the occlusal surface of first and second molars. Molars are susceptible to dental caries, not only because of their location but also because of their anatomy. [4]

The prevalence of dental caries was of great interest for long and is a principal subject of many epidemiological researches being carried out worldwide. This significant but a preventable public health problem interferes with normal food intake, speech, self–esteem, and routine activities affecting overall health status of the children. [5]The prevention of dental caries has long been considered as an important labor for health profession. The world health organization (WHO) recognizes dental caries as a pandemic and reports that the prevalence of dental caries among school going children is about 60 to 90%. The risk factors for dental caries are many and they can be classified into oral and non-oral risk factors. Oral risk factors include anatomy of the tooth, composition of dental plaque, restoration and oral hygiene status. Non-oral risk factors include age, irregular tooth brushing, dietary habits and medication history. [6]School years cover a period that runs from childhood to adolescence. These are influential stages in people’s lives where lifelong substantial oral health-related behaviors, as well as beliefs and attitudes, are being developed. Children are particularly receptive during this period, and the earlier habits are established, the long-lasting, and the

impact. [7]Children who suffer from poor oral health are 12 times more likely to have more restricted activity days including missing schools than those who do not. [8]

.Studies have reported missed school hours, toothache, and several impairments of daily life activities associated with a high decayed component in both primary and permanent dentition. [9]Similar findings have been reported in Brazilian preschool children and in a school survey of American native children. [10]

Voluminous literature exists on the status of the dental caries in the Indian population. In 1997, 22.7% of Indian population was estimated to be 5 to 14 years. This being such a high proportion of the population, the prevalence of dental caries among this age group needs to be assessed. It has been observed that during 1940 the prevalence of dental caries in India was 55.5%, during 1960 it was reported to be 68%. Overall the general impression is that dental caries has increased in the prevalence severity in urban and cosmopolitan population over the last couple of decades. However there is no definite picture as yet regarding the disease status in rural and backwards areas of the country in the comparison where 80% of the population inhabits. [11]

The “Prevalence” of dental caries in an individual is obtained by calculating DMFT, which is the most common index used and for deciduous dentition dft index is used. An epidemiological study was planned as very few studies have been conducted in Chennai city. Hence, this present study was conducted as a part of the screening, to assess the prevalence among school going children of Tiruvallur district using DMFT index.

Materials and Method

The study received the approval from university review board (STP/SD 12 BDS 103).

A study of prevalence of dental caries among school going children of Tiruvallur district Chennai was undertaken by the Department of Public Health Dentistry, Saveetha Dental College and Hospitals, Chennai to evaluate prevalence of dental caries in relation to various risk factors.

The survey was conducted in 348 school children residing in Tiruvallur district, Chennai. Out 139 government schools in Tiruvallur district 7 schools were selected for the survey. The

sample consisted of 348 individuals of both gender (219 were male and 129 are females); their ages ranged from 9 to 13 years. Age of 12 years has been universally accepted as a global monitoring age for caries since all permanent teeth except third molars would have been erupted by this age. Consent for examining of the children was obtained from the respective head master. The criteria for selection of the study subjects were that the children should be permanent residents of Tiruvallur.

Depending on the physical condition of the school, the exact arrangement for conducting the examination was determined. The school children were examined under daylight with the help of plain mirror and standardized dental probes. Examination of the child was done by only one examiner to avoid interexaminer variability. Recording of data was done by a trained person who assisted throughout the study. Dental caries was evaluated and the DMFT indices were recorded respectively [12]

Result

This study aims to measure the distribution of dental caries among school going children from 9 to 13 years old in Tiruvallur district. The sample size was 348 school children of different age groups.

Table 1 shows the distribution of sample according to source of population, age and gender. The sample is divided into four groups according to age and also was divided into groups according to gender in which each group has both the gender.

Table 2 shows the prevalence of dental caries in which the highest is seen in the age group of 9 with a percentage value of 88.24%. Comparatively the other age group had a percentage value of 85.28%, 74.24%, 52.78%, 60.32%, and 72.21% respectively. Table 2 also shows the mean DMFT for the study subject which was 3.24 while the mean DMFT for different age groups (9-13 years) was 5.03, 4.76, 2.20, 2.18, 2.06, and 3.24 respectively.

Table 3 shows the prevalence of dental caries in different age groups (9-13 years old) for females. The highest percentage of dental caries prevalence was found in the age 10 and the lowest percentage of was found in the age 12.

Table 4 shows the prevalence of dental caries in different age groups (9-13 years old) for males. The highest percentage of dental caries prevalence was found in the age 9 and the lowest percentage of was found in the age 12.

Table 1: Shows the source population according to age and gender.

Age	Female	Male	Total
9 years	51	34	85
10 years	32	29	62
11 years	35	33	66
12 years	32	39	72
13 years	34	29	63

Table 2: Shows the prevalence and severity of dental caries mean DMFT in different age groups (9-13 years) old.

Age Group	Total number	Affected number	Caries prevalence %	DMFT(mean)
9	85	75	88.24	5.03
10	62	53	85.48	4.76
11	66	49	74.24	2.20
12	72	38	52.78	2.18
13	63	38	60.32	2.06
Total	348	253	72.21	3.24

Table 3: The prevalence and severity of dental caries mean dmft in different age groups (9-13 years) old females

Age	Total number	Affected number	Caries prevalence %	Dmft(mean)
9	51	42	82.35	1.53
10	32	30	93.75	2.80
11	35	27	77.14	0.50

12	32	16	50.00	0.91
13	34	22	64.70	0.52

Table 4: The prevalence and severity of dental caries mean DMFT in different age groups (9-13 years) old males

Age	Total number	Affected number	Caries prevalence %	Dmft(mean)
9	34	33	97.05	3.5
10	29	23	79.31	1.96
11	33	22	66.67	1.78
12	39	22	56.41	1.27
13	29	16	76.19	1.54

Discussion

Untreated oral diseases in children frequently lead to serious general health, significant pain, and interference with eating and loss of school time. [13] Despite incredible scientific advances and the fact that caries is preventable the disease continues to be a major health problem. The World Health Organization (WHO) has ranked dental caries, as number three among all chronic noncommunicable diseases that require worldwide attention for prevention and treatment and the fact that caries One of the factors to be considered when planning for the required growth in dental in dental facilities is the prevalence of dental diseases and their treatment need in the population. A Who Health Organization of global DMFT for 12-year-old children reported that in the 188 countries included in their database, that on global basis, 200,335,380 teeth were decayed, filled or missing among just that age group. This was based on the data available in 2004 from the WHO Oral Health Database, Country/Area Profile program (CAPP) [13]. Therefore, WHO continues to advocate that efforts to improve the overall situation are still highly indicated?[14]

The use of DMFT and dft indices has been accepted practice for assessing the prevalence and severity of caries in a population. Hence an attempt has been made in the present study which was conducted as a part of the outreach screening to assess the prevalence of dental caries among primary school going children of Chennai using dft/DMFT index.

In this study, the prevalence of dental caries has increased with age from group 9-13 years. Oral health is an integral part of our health. Lack of awareness and limited access to dental specialist has made the children in Tiruvallur district to be more prone to disease. The 9-13-year age group was chosen for the study as it is the global monitoring age for dental caries, for international comparisons and monitoring of disease trends. [15] In this study it was observed that the caries prevalence of 11 to 13 years old children was lower as compared to 9 and 10 years old age group. This shows that as age advances prevalence of dental caries decreases. This finding corresponds with the study conducted by Misra F.M (1979)[16] among 6-16 year old children in urban area of South Orissa. He observed an increase in caries level between 5 to 12 year (56% to 81%) and a decrease in caries level in 13-15year (41.4%).Similarly in the study conducted by Peterson P.E,et al (1991) [17], Retna Kumari N (1999) [18], Dash J.K (2002) [19], Saravanan S,.et al (2003) .[20]The increased prevalence of caries in boys compared to girls confirms the view that there is a marked preference for sons regardless of the socio-economic class, which manifests itself in the longer feeding of sons compared to daughters .

With the limitation of this study, further studies are required to correlate dental caries prevalence in the target population with parent's literacy level and other socio behavioral factors. Although severity of dental caries is not assessed separately in the present study; the data regarding treatment need gives a glimpse of the severity of dental caries in the study subjects.

Hence oral hygiene education and motivation are the pertaining goal to achieve proper oral hygiene health among school going children. Oral health consciousness programs should be administered by dental college and association to increase consciousness of oral and dental health. The government should provide oral health through primary health-care system and appoint dental surgeons up to primary health center level in this region.

A preventive program including pit and fissure sealant application and topical fluoride application which would be an ideal measure in the prevention of dental caries in the permanent dentition of schoolchildren would aid in protecting the permanent dentition which has to remain lifelong.

Conclusion

1. The prevalence of dental caries was high in 9-11 years of age group with highest prevalence seen in 9 years.
2. Caries prevalence showed variation in relation to sex in which male population had high prevalence of dental caries compared to female population

Conflict of Interest: Nil

Source of Funding: Self

Corresponding Author

Deepa Gurunathan

Professor, Department of Pedodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Email id: drgdeepa@yahoo.co.in

Telephone Number: +919994619386

Reference

1. Naziya KB, Kumar RP, Arumughamm IM, Srisakthi D. Prevalence of dental caries among primary schoolchildren in Chennai-A cross-sectional study. Journal of Advanced Pharmacy Education & Research| Apr-Jun. 2017; 7(2).
2. Goyal A, Gauba K, Chawla HS, Kaur M, Kapur A. Epidemiology of dental caries in Chandigarh school children and trends over the last 25 years. Journal of Indian Society of Pedodontics and Preventive Dentistry. 2007 Jul 1; 25(3):115.
3. Sunayana G, John J, Saravanan S, Arumugham IM. Prevalence of dental caries among 12 and 15 year old school children in Chennai city. Journal of Indian Association of Public Health Dentistry. 2009 Jan 1; 7(13):54.
4. Kotsanos N, Darling AI. Influence of posteruptive age of enamel on its susceptibility to artificial caries. Caries research. 1991; 25(4):241-50.
5. Selwitz RH, Ismail AI, Pitts NB. Dental caries. The Lancet. 2007 Jan 6; 369(9555):51-9.

6. Gathecha G, Makokha A, Wanzala P, Omolo J, Smith P. Dental caries and oral health practices among 12 year old children in Nairobi West and Mathira West Districts, Kenya. Pan African Medical Journal. 2012; 12(1).
7. Jürgensen N, Petersen PE. Promoting oral health of children through schools-Results from a WHO global survey 2012. Community Dent Health. 2013 Dec 1; 30(4):204-18.
8. World Health Organization. Oral health surveys: basic methods. World Health Organization; 2013.
9. Ogola JA. Oral Health Knowledge, Attitudes and Practices among 9-12 Year Old Children Attending Primary Schools in Westlands, Nairobi County, Kenya (Doctoral dissertation).
10. Dixit LP, Shakya A, Shrestha M, Shrestha A. Dental caries prevalence, oral health knowledge and practice among indigenous Chepang school children of Nepal. BMC oral Health. 2013 Dec;13(1):20.
11. Moses J, Rangeeth BN, Gurunathan D. Prevalence of dental caries, socio-economic status and treatment needs among 5 to 15 year old school going children of Chidambaram. J Clin Diagn Res. 2011 Feb;5(1):146-51.
12. Sudha P, Bhasin S, Anegundi RT. Prevalence of dental caries among 5-13-year-old children of Mangalore city. Journal of Indian Society of Pedodontics and Preventive Dentistry. 2005 Apr 1; 23(2):74.
13. Prabakar J, John J, Srisakthi D. Prevalence of dental caries and treatment needs among school going children of Chandigarh. Indian Journal of Dental Research. 2016 Sep 1; 27(5):547.
14. Das UM, Beena JP, Azher U. Oral health status of 6-and 12-year-old school going children in Bangalore city: An epidemiological study. Journal of Indian Society of Pedodontics and Preventive Dentistry. 2009 Jan 1; 27(1):6.
15. Bagramian RA, Garcia-Godoy F, Volpe AR. The global increase in dental caries. A pending public health crisis. Am J Dent. 2009 Feb 1; 22(1):3-8.
16. Misra FM, Shee BK. Prevalence of dental caries in school going children in an urban area of South Orissa. Journal of the Indian Dental Association. 1979 Sep; 51(9):267-70.

17. Petersen PE, Poulsen VJ, Ramahaleo J, Ratsifaritara C. Dental caries and dental health behaviour situation among 6-and 12-year-old urban schoolchildren in Madagascar. African dental journal: official publication of the Federation of African Dental Associations Journal dentaire africain. 1991; 5:1-7.
18. Retnakumari N. Prevalence of dental caries and risk assessment among primary school children of 6-12 years in the Varkala municipal area of Kerala. Journal of the Indian Society of Pedodontics and Preventive Dentistry. 1999 Dec; 17(4):135-42.
19. Dash JK, Sahoo PK, Bhuyan SK, Sahoo S. Prevalence of dental caries and treatment needs among children of Cuttack (Orissa). Journal-Indian Society of Pedodontics and Preventive Dentistry. 2002 Dec 1;20(4):139
20. Saravanan S, Anuradha KP, Bhaskar DJ. Prevalence of dental caries and treatment needs among school going children of Pondicherry, India. Journal of the Indian Society of Pedodontics and Preventive Dentistry. 2003 Mar; 21(1):1-2.

Prevalence of Midline Spacing among School Students in Chennai Region- A Survey

Saarusree.E [1], A.V. Arun [2], Asha Ramesh [3]
Graduate Student [1],Professor [2],Department of Orthodontics
Senior Lecturer [3],Department of Periodontics
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,
Saveetha University, Chennai, India

Abstract

Introduction: The ideal occlusion and spacing in primary dentition acts as mirror for the prevalence of malocclusion in the permanent dentition. The properly placed teeth in dental arch help in maintaining the better health of oral cavity and the supporting structures, but also influence the personality of the children. Malocclusion is a problem affecting a disproportionately large number of Indian children.The objective of the study was to assess spacing (physiological and primate spaces) in anterior region of both maxilla and mandible and to determine the molar and canine relation in Chennai school children.

Materials and Methods: 517 female school students of Velammal Matriculation Higher Secondary School, Ponneri and local schools of Chennai populations aged between 15-19yrs were examined. Prevalence of spacing was assessed by taking photographs.

Result: From the result, it has been proved that out of 517 female students who were included in this study, 51 female students had upper anterior spacing. It involved not only midline diastema but also generalised spacing wasalso considered for this study.

Conclusion: This study provides information of the prevalence of spacing and closed dentition, occlusion relation and its relation to a malocclusion. Hence, determining the malocclusion at its early age and early intervention procedure helps in preventing the problems at later stage.

Key Words: *Malocclusion; Midline Diastema; Early intervention; Preventive Orthodontics; Survey*

Introduction

Malocclusion exhibits itself mainly in three forms, one is crowding and irregularly arranged teeth, another is proclination and third is spacing. The reasons may be due todiscrepancy between arch size and tooth materials or because of habits or any other environmental aetiology. There are various studies conducted to assess prevalence of spacing in various populations.However, no such studies have been conducted in Chennai school populations to assessthe prevalence of spacing within our population. So, we have taken up such studies to examine the prevalence of spacing in school students of our population. This will help the orthodontists to get a better idea of the incidence of upper anterior spacing in our populations and in their clinical practise. study was conducted among female school students of Chennai populations. The occlusal relationship and spacing in deciduous dentition is known to have a vital bearing on the setting up of the normal occlusal relationship in permanent dentition. The ideal occlusion and spacing in primary dentition acts as mirror for the prevalence of malocclusion in the permanent dentition. The properly placed teeth in dental arch help in maintaining the better health of oral cavity and the supporting structures, but also influence the personality of the children. Malocclusion not only compromises maintaining better hygiene and also the health of investing tissue, but can also lead to behavioural (psychological) and social problems[1]. Malocclusion is a problem affecting a disproportionately large number of Indian children. The canine relation in the primarydentition is known to influence the canine relation in permanent dentition because it is thought to be a stable relation in the deciduous period. Dental aesthetics plays a major role in determining facial appearance of a person. Facial appearance of a person contributes to his self- confidence and thereby provides social recognition to a person.

The aesthetic factors are not easily evaluated and are generally determined subjectively. Identification of malocclusion by dental professional is primary factor in motivating individual to seek orthodontic care. The following patient factors appearance improvement, age, gender, environment influence and social class and orthodontist factors appreciation of treatment need, access to services, cost of treatment and treatment priority can be motivating factor to receive orthodontic treatment.The prevalence of malocclusion in India varies from 20% to 43%. Although there is plenty of literature concerning the prevalence and type of malocclusion is quoted in literature, the paucity of information is present on malocclusion in preschool children and its correlation with crowding, spacing, and closed dentition.Hence, the present study was carried out to known the impact of occlusal relationship and spacing on

malocclusion in the primary dentition. Malocclusion may cause unpleasant appearance, impaired oral function, speech problems, temporomandibular disorders, increased susceptibility to trauma and periodontal disease[2]. Identifying occlusal status in particular population provides important information on treatment needs and enables the government to draw the appropriate preventive and treatment programs. Malocclusion is regarded as an irregularity of the teeth or a malrelationship between the dental arches beyond the normal range. The aetiology of malocclusion is multifactorial, including genetic and environmental causes as well as harmful oral habits. Malocclusion represents a developmental disorder of the craniofacial complex that affects the jaws, tongue and facial muscles and is known as one of the three major oral diseases that affect human oral function, aesthetics, social interactions and health-related quality of life.

Face and dentition reflect an individual's most intimate expression such as elaborating speech and communicating; they also play a role in respiratory, masticatory, and swallowing functions. Malocclusions may cause psychosocial problems, especially concerning aesthetics, which is highly valued in current society in both personal and professional levels, lowering self-esteem and triggering a negative impact on the quality of life of children.Identifying the aetiology of malocclusions is essential for the success of any orthodontic procedure. Anthropological studies have shown changes in occlusal patterns of the population, most likely caused by softer foods in their dietary habits, non-nutritivesucking habits such as the use of pacifier and thumb sucking, respiratory problems, and early weaning. Thus, the alleged increase in malocclusion indexes between primary, mixed, and permanent dentition is considered.In addition, extensive untreated caries lesions and premature primary tooth loss contribute to compromising the occlusion of children, ranking malocclusions third in the scale of oral health problem priorities[3].

Considering the changes caused by modern living conditions and the consequent development of certain types of malocclusion, several studies prove the occurrence of malocclusion in the young population. However, data referring to primary dentition are rare in Brazil and originate from studies of distinct focus, making comparability difficult.Understanding the oral health conditions of children through epidemiological studies is fundamental for the development of adequate action proposals that may reduce the incidence of occlusion abnormality. This knowledge may also contribute to an ultimate evaluation of the impact of these actions.Regarding population, prevention is an attractive

and viable alternative for the treatment of occlusal changes, considering there is the possibility of controlling environmental variables that contribute to its occurrence.Moreover, prevention simplifies or even avoids corrective orthodontic treatments which are often inaccessible to the majority of the population. In modern countries, many sophisticated attention are given to various number of dentofacial deformities and treatment of malocclusion at an earlier stage. Treatment usually takes place at an adolescent period when the patients cooperation and motivation are at a high peak. Epidemiological investigation in the permanent dentition have become a difficult job since the significant number of populations cannot be recorded because of orthodontic treatment [4]. The aim of this study is to access spacing in both maxilla and mandible in primary dentition for treatment of malocclusion.

Materials and Methods

The study was approved by the Institutional Review Board of Saveetha Dental College and Hospital. 517 female school students of Velammal Matriculation Higher Secondary School, Ponneri and local schools of Chennai populations aged between 15-19 years were selected and screened for this study.Prevalence of spacing was assessed by taking photographs shown in [Figure 1]. The inclusion criteria were female students with upper anterior spacingand between the age group of 16-20 years. Students who had crowding and proclination without spacing were excluded from the study. Apart frommidline diastema, generalised spacing was also included in theassessment [5].

Results

From the result, it has been proved that out of 517 female students who were included in this study, 51 female students had upper anterior spacing shown in [Figure 2]. It should be noted that only midline diastema but also generalised spacing in the dentition was assessed among these students.

Discussion

This study was conducted among female school children between ages of 16-20years. Oral screening was done to 517school students between ages of 16-20years out of which 51 students has upper anterior spacing. Generalised spacing was assessed for female students apart from midline diastema.Spacing may be due to developmental causes and physiological causes. Developmental causes are spacing in deciduous teeth, ugly duckling stage and

microdontia. Physiological causes are tongue thrusting, thumb sucking, periodontal problems. These spacing can be corrected by wearing orthodontic braces, direct and indirect veneers and crowns and bridges.Spacing is the most common causes in deciduous or milk teeth.Many studies were conducted in various populations to assess prevalence of spacing among school students. According to various studies, Spacing is more common in Caucasians than Africans. Spacing of at least 2mm was found in 9.1-55.8% of the African and 7.1-59.2% of the Caucasian childrenduring different emergence stages of the dentition[6].

Early recognition of conditions predisposing young children to malocclusions is in the hands of primary care providers who for practical purposes are general practitioners and the paediatric dentist. It is important that conditions that predispose to develop a malocclusion of the permanent dentition be detected early in the primary dentition so the early interceptive procedure can be applied to prevent its further consequences. The presence of spacing or crowding and occlusion in the primary dentition and its relation to the developmentof malocclusion has been the long subject of discussion. There are many studies done regarding correlation of spacing or crowding with the malocclusion in pre-school children but still the knowledge of it is scares. The development of malocclusion starts from the primary dentition, so it is very important to know the occlusion in the primary dentition, as well as the changes of occlusal pattern, during the period of deciduous dentition. According to Bishara et al., marked arch width (inter canine and intermolar width) expansion occurs significantly between 3 and 5 years of age in both maxilla and mandible [7]. Therefore, the school children included in current study was limited to 20 years. Joshi and Makhija conducted study on 3-6 years' school children in Gujarat and concluded that non-spaceddentition was less common in school children when compared to spaced dentition.

A significant difference was noticed in which amount of spacing was found to be greater in males.Almost similar results were obtained in various studies [8]. Im et al., concluded that maxilla showed more spacing when compared to mandible irrespective of sex. ElNofely et al., assumed that spacing can be either due to small mesiodistal or wide inter canine width. Leighton's hypothesis suggests that for the proper alignment of permanent teeth in mandibular arch, the amount of spacing in primary dentition should be of 6 mm or more. The study concluded that flush terminal molar relation recorded highest (74%) followed by mesial step (22%) and then distal step (4%) in all the age groups but with increase mesial step molar relation increased to 33% at the age of 6 years[9]. Similar results were obtained by Baume

showed that flush terminal plane (76%) is more commonly seen followed by mesial step (14%) and then distal step molar relation (10%). The study results shown highest bilateral flush terminal plane molar relation in children from Hyderabad (72.5%) and Chennai (74%) as concluded by Sriram et al., On contradictory, several other studies have shown that mesial step relation recorded highest followed by flush terminal plane and distal step. Bahadure et al., shown highest the rarest occurrence of mesial step molar relation (57.3%) followed by flush terminal plane (31.1%) and then the distal step molar relation (11.7%) [10].

The changes in the occlusion can be attributed to a combination of mesial migration of the lower arch and a mesial shift of the mandible, which is probably caused by growth. Considering canine relation, the most common relation found was Class I (90%) canine relation followed by Class II (6.4%) and Class III (3.6%) canine relation at the age of 2-3 years. At the age interval of 5-6 years Class I relation was found to be 82%, Class II relation was 9.8%, and Class III was 7.6%. Baidaset al., concluded that among the canine relation, Class I (90.1%) showed peak value succeeded by Class III(7.4%), and Class II (2.5%) relationship, which are similar to the present study. On the contradictory, Bahadure et al. concluded that among the canine relation, Class I (47.20%) showed highest value followed by Class II (42.83%) and Class III (9.97%) [11]. There were studies done on how spacing and occlusion is related to malocclusion and what were the possible reasons for the malocclusion. Howe concluded that malocclusions were discrepancy in arch length and tooth size.

The major reasons for the malocclusion may be due to the modern diet, which results in decreased inter-proximal wear and jaw growth[12]. According to Profit, oral habits such as mouth breathing, thumb sucking might also be responsible for crowding in the arch.Prevention and early treatment in orthodontics is still the subject of continuous debate and controversy regarding cost effectiveness analysis and functional and psychosocial benefit[13]. In recent articles, Gianelly et al., favoured treatment in the late mixed dentition for 90 percent of the children while Viazis et al., concluded that early orthodontic/orthopaedic treatment would be profitable and desirable in younger patients. In this study, it was observed that malocclusion is actually commonplace in the temporary dentition[14].

Obvious similarities could be seen and also in with other investigations of occlusal traits in the primary dentition. The available documentation also suggests that development of the

occlusion is a continuum for many aspects: most of the major occlusal trends characterizing the adult dentition were detectable at early stages. Lack of space was common, as well as lateral crossbites, excessive overjet or Class II relationships; the striking difference from data concerning the permanent dentition was the high prevalence of open bites which are expected to decrease with maturation. In the absence of longitudinal documentation and with due reservation to the possible biases inherent to cross-comparison of epidemiological data on malocclusion, we are inclined to conclude that, provided the patient'scooperation is established early, consideration may be given to treatment of the malocclusion. This concern should be focused mainly on lateral crossbites and sagittal relationship.

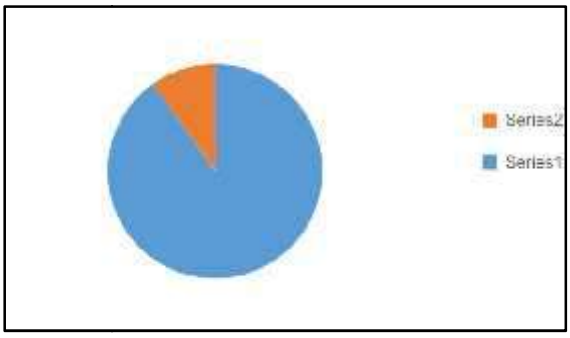
The most common trait of malocclusion was revealed to be a deep overbite (33.66%), which is consistent with a previous study. However, deep bite in primary dentition may be temporary, and spontaneous correction may occur due to vertical growth of the mandibular ramus and the full eruption of permanent molars, as a longitudinal study supported [15]. Scholars have recommended that an increased overbite in primary dentition is rarely treated unless the lower incisors impinge the maxillary palatal mucosa, leading to pain or other clinical symptoms. Physiological spaces, including the developmental spaces and the primate space, were the second most common characteristic in primary dentition, with a prevalence of 27%. Spacing indicates proper alignment of the permanent dentition; thus, early effective treatment is not necessary in such cases. Cross bite (25.29%), mainly associated with genetically inherited characteristics and environmental factors, was observed at a higher prevalence than that reported in India [16]. Self-correction was noted for anterior crossbite, whereas posterior crossbite was transferred from the deciduous to the permanent dentition. Likewise, crossbite warrants increased early check-ups and treatment to decrease the long-term effects on growth and development. Relative to the reported prevalence of increased overjet in the primary dentition in different countries, which ranges from 3% to 16%, the prevalence rate in the present study was moderate, at 10.16%. Because increased overjet was alleviated during the transition from primary to permanent dentition, it was not a precise indicator of a similar increase in the permanent dentition. In addition, a standard of 3 mm is acceptable to clinicians. The prevalence of crowding (8.03%) was much lower than that reported in Colombia (52.1%)[17]. This difference might be the result of a sampling error secondary to the small sample size in the former study.

There are clear indications that crowding in deciduous dentition, mostly due to the modern diet, contributes to the disproportion between the jaw and tooth size in the permanent stage; thus, early interceptive treatment is necessary to enhance the favourable growth and development of the jaw and teeth. Moreover, the discrimination between temporary malocclusion and pathological malocclusion is crucial in this period.Some study revealed a dramatic increasing trend in the prevalence of malocclusion over time among children in mainland China, which suggests the need for the early recognition and treatment of deciduous-dentition malocclusions in further diagnostic and treatment procedures. There are several potential reasons for this observation[18]. First, the rapid development of China's economy has changed lifestyles and diet structures among its inhabitants, and the overconsumption of refined and high-sugar foods has increased the risk of malocclusion. Second, a lack of proper oral hygiene knowledge, attitudes or behaviours among parents has likely contributed to the increasing prevalence of malocclusion among children. For example, there is a general misunderstanding that malocclusion in primary dentition is temporary and should resolve with the eruption of permanent teeth. Additionally, the government's investment in oral health services and resources, as well as health education programmes, is insufficient, especially in rural areas[19].

The present study demonstrated that the majority of children in mainland China had a flush terminal plane (47.10%), followed by a mesial step relationship (43.24%) and a distal step relationship (5.04%). In India, a survey of 1,000 pre-schoolers revealed similar results; the molar relationship prevalence was the flush terminal plane (66.0%), mesial step (12.8%), distal step (2.4%) and bilateral symmetry (18.8%). There was a consensus that the distal surfaces on the second deciduous molars, i.e., the primary molar relationship in the deciduous dentition, predicted the identification of the permanent molar relationship[20]. A previous surveyrevealed that most cases of flush and mesial terminal plane developed into Angle Class I in the permanent dentition due to a combination of forward movement of the mandible and mesial migration of the mandibular arcus. Additionally, a study by Ravn et al., demonstrated that a distal step molar relationship could develop into an Angle Class II molar relationship in permanent dentition. Therefore, additional longitudinal studies are necessary to identify the changes in the molar relationship from primary dentition to permanent dentition[21].

Figure 1: Assessment of midline spacing and generalised spacing

Figure 2: Prevalence of Spacing among Chennai School Children



Series1- 517 female students
Series 2- 51 female students

Conclusion

This study provides information of the prevalence of spacing and closed dentition, occlusion relation and its relation to a malocclusion. In the study, spaced dentition was more frequent the non-spaced dentition. In this study female populations had upper anterior spacing in their early stage which can be corrected by further orthodontic treatment. Hence, determining the malocclusion at its early age and early intervention procedure helps in preventing the problems at later stage.

Corresponding Author

Asha Ramesh

Senior Lecturer,Department of Periodontics

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

162, Poonamallee High Road, Chennai 600077,Tamil Nadu, India

Mobile Number: -+91-9841722113

Email id: ash.periopg@gmail.com

References

1. Lux CJ, Ducker B, Pritsch M, Niekusch U, Komposch G. Space conditions and prevalence of anterior spacing and crowding among nine-year-old schoolchildren. Journal of orthodontics. 2008 Mar 1;35(1):33-42.
2. Mugonzibwa EA, Eskeli R, Laine-Alava MT, Kuijpers-Jagtman AM, Katsaros C. Spacing and crowding among African and Caucasian children. Orthodontics & craniofacial research. 2008 May 1;11(2):82-9.
3. Baidas L. Occlusion characteristics of primary dentition by age in a sample of Saudi preschool children. Pakistan Oral & Dental Journal. 2010 Dec 1;30(2).
4. Bahadure RN, Thosar N, Gaikwad R. Occlusal traits of deciduous dentition of preschool children of Indian children. Contemporary clinical dentistry. 2012 Oct;3(4):443
5. Alhaija ES, Al-Khateeb SN, Al-Nimri KS. Prevalence of malocclusion in 13-15 year-old North Jordanian school children. Community dental health. 2005 Dec;22(4):266.
6. Heimer MV, Tornisiello Katz CR, Rosenblatt A. Non-nutritive sucking habits, dental malocclusions, and facial morphology in Brazilian children: a longitudinal study. The European Journal of Orthodontics. 2008 Sep 4;30(6):580-5.
7. Bishara SE, Ortho D, Jakobsen JR, Treder J, Nowak A. Arch width changes from 6 weeks to 45 years of age. American Journal of Orthodontics and Dentofacial Orthopedics. 1997 Apr 1;111(4):401-9.
8. Joshi MR, Makhija PG. Some observations on spacing in the normal deciduous dentition of 100 Indian children from Gujarat. British journal of orthodontics. 1984 Apr 1;11(2):75-9.

9. Vinay S, Keshav V, Sankalecha S. Prevalence of spaced and closed dentition and its relation to malocclusion in primary and permanent dentition. International journal of clinical pediatric dentistry. 2012 May;5(2):98.
10. Sriram CH, Priya VK, Sivakumar N, Reddy KM, Babu PJ, Reddy P. Occlusion of primary dentition in preschool children of Chennai and Hyderabad: A comparative study. Contemporary clinical dentistry. 2012 Jan;3(1):31.
11. Nanda RS, Khan I, Anand R. Age changes in the occlusal pattern of deciduous dentition. Journal of dental research. 1973 Mar;52(2):221-4.
12. Peres KG, Barros AJ, Peres MA, Victora CG. Effects of breastfeeding and sucking habits on malocclusion in a birth cohort study. Revista de saude Publica. 2007 Jun;41(3):343-50.
13. del Conte Zardetto CG, Rodrigues CR, Stefani FM. Effects of different pacifiers on the primary dentition and oral myofunctional structures of preschool children. Pediatric dentistry. 2002;24(6):552-80.
14. Viggiano D, Fasano D, Monaco G, Strohmenger L. Breast feeding, bottle feeding, and non-nutritive sucking; effects on occlusion in deciduous dentition. Archives of disease in childhood. 2004 Dec 1;89(12):1121-3.
15. Priyanka MJ, Jaiganesh D. Periodontally accelerated osteogenic orthodontics. Int J Pharm Pharm Sci. 2013; 5:49-51.
16. Kumar RP, John J, Saravanan S, Arumugham IM. Oral health knowledge, attitudes and practices of patients and their attendants visiting College of Dental Surgery, Saveetha University, Chennai. Journal of Indian Association of Public Health Dentistry. 2009 Jan 1;7(13):43.
17. Bichara LM, Aragón ML, Brandão GA, Normando D. Factors influencing orthodontic treatment time for non-surgical Class III malocclusion. Journal of Applied Oral Science. 2016 Oct;24(5):431-6.
18. Bugaighis I. Prevalence of malocclusion in urban libyan preschool children. Journal of orthodontic science. 2013 Apr;2(2):50.
19. Reddy S, John J, Sarvanan S, Arumugham IM. Normative and perceived orthodontic needs among 12-year-old school children in Chennai, India-A comparative study. Applied Innovations and Technologies. 2010;3(3):40-7.
20. Baccetti T, Franchi L, McNamara JA. Longitudinal growth changes in subjects with deepbite. American Journal of Orthodontics and Dentofacial Orthopedics. 2011 Aug 1;140(2):202-9.

21. Bhayya DP, Shyagali TR. Gender influence on occlusal characteristics of primary dentition in 4-to 6-year-old children of Bagalkot City, India. Oral Health and Preventive Dentistry. 2011 Jan 1;9(1):17.

Prevalence of Musculoskeletal Disorders among Dental Students-A Questionnaire Survey

Nandhini.S[1], Mahesh[2], Mahathi.N[3]

Graduate Student[1], Senior Lecturer[2], Department of Pedodontics, Reader[3], Department of Oraland Maxillofacial Surgery.

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: The most important causes of this disorder can be awkward postures, repetitive and rapid movements, excessive force, psychological factors, genetic and generally inappropriate body condition. Damage or injury can be defined as a disorder of the musculoskeletal system (muscles, ligaments, tendons, joints, nerves, blood vessels and soft tissues). The symptoms of MSDs increase with the number of years of practice. The prevention and reduction of MSDs among dentists should include their knowledge in dental ergonomics and awareness regarding the importance of work-related risk factors. This study aimed to identify risk factors for musculoskeletal disorders among dentists and identify its effective risk factors, in order to reduce musculoskeletal problems and is designed to improve the work environment.

Materials and Methods: The target population of this investigation was the students of dental school.A sample of 300 questionnaires were distributed.To determine the prevalence of musculoskeletal disorders, questionnaire was prepared.The Questionnaire assessed the musculoskeletal disorders, pain in different organs of the body.Finally the results are evaluated in percentage

Results: Out of 300 questionnaires distributed, the female prevalence was higher.86% of the people don’t do physical exercise daily.71% of the dental students take rest after each patient, 15% take rest once an hour and 13% of the students never take rest at all.95% of the dental students involve frequent bending of elbows and joints.91% of them feel some kind of musculoskeletal discomfort in the body and other 9% are free from it. Most discomfort is felt

in the neck region that is 41% and 23% in one or both elbows,15% in one or both shoulders and 20% in back/spine.66% of the students feel the pain most of the time and 22% always and 11% at times.50% of the students follow periodic stretching and breaks as the pain relief strategy.36% follow postural awareness strategy and 13% follow strengthening exercises.

Conclusion: Therefore dentists should implement right ergonomic designs in their dental clinics along with doing regular exercise and relaxation techniques, which help them to combat stress, thus improving the quality of life and resulting in consistent long-lasting work efficiency of the dentists.

Key Words: *musculoskeletal,dentists,pain, long lasting work,occupational.*

Introduction

Today, work related musculoskeletal disorders (WMSDs) are one of the most common occupational diseases and causes of disability in developing countries.[1]The most important causes of this disorder can be awkward postures, repetitive and rapid movements, excessive force, psychological factors, and genetic and generally inappropriate body condition. Damage or injury can be defined as a disorder of the musculoskeletal system (muscles, ligaments, tendons, joints, nerves, blood vessels and soft tissues)[2].Studies have shown that the best strategy to prevent WMSDs is to reduce exposure to risk factors such as repetitive movements, excessive force, awkward postures, vibration and static work.

This study aimed to identify risk factors for musculoskeletal disorders among dentists and identify its effective risk factors, in order to reduce musculoskeletal problems and is designed to improve the work environment.Musculoskeletal disorders have become increasingly common worldwide during the past decades. It is a common cause of work-related disability among workers with substantial financial consequences due to workers' compensation and medical expenses. Various work-related factors have been established as predisposing the disorders. In dentists, overstrained and awkward back postures for back pain, repetitiveness for neck and shoulder disorders, and psychosocial stressors for back, neck and shoulder complaints. A slight hand neuropathy has also been reported caused by exposure to high frequency vibration tools.In most studies only a few of these risk factors have been taken into account simultaneously.

This makes it difficult to appreciate the impact of specific risk factors since most studies did not control appropriately for concurrent risk factors. In Greece, very few studies have been undertaken in occupational groups with respect to the simultaneous occurrence of different musculoskeletal complaints and their interrelationships .The first aim of this study was to investigate associations between personal characteristics, physical load, psychosocial factors and general health status with complaints of back, neck, shoulder and hand/wrist. The second aim was to analyses interrelations between these musculoskeletal complaints and its effect on associations between work-related risk factors and musculoskeletal complaints.Dental surgeons often cannot avoid prolonged static postures. Even in optimal seated postures, more than one-half of the muscles of the body are contracted statically and there is little movement of the vertebral joints. This may result in damaging physiological changes that can lead to back, neck, or shoulder pain or MSDs.

If regularly occurring pain or discomfort is ignored, the cumulative physiological damage can lead to an injury (macro change) or a career-ending disability.Basic operating posture is considered an important occupational health issue for dental surgeons. It is generally agreed that the physical posture of the operator should be such that all the muscles are in a relaxed, well-balanced, and neutral position. Postures outside of this neutral position are likely to cause musculoskeletal discomfort. A thorough understanding of the underlying physiological mechanism leading to these problems is necessary to develop and implement a comprehensive approach to minimize the risk of work-related injury.[3,4]In dentistry, bad working habits and repetitive tasks such as scaling, root planing, and uncomfortable physical postures contribute greatly to MSDs, stress, and loss of productivity. The key objective for clinicians is to find a position that allows them to achieve optimum access, visibility, comfort, and control at all times.

Materials and Methods

The target population of this investigation was the students of dental school.A sample of 300 questionnaires were distributed.To determine the prevalence of musculoskeletal disorders, questionnaire was prepared.The Questionnaire assessed the musculoskeletal disorders, pain in different organs of the body.Finally the results are evaluated in percentage.These questions addressed items such as tiredness after work, fatigue, lack of concentration, putting interest in

other people, the ability to recover from work, and the influence on work performance.For both general health endpoints subjects with a score above the median value were considered to have a high need for recovery or a moderate/bad general health.Musculoskeletal complaints were measured using the standardized Nordic questionnaire . Four endpoints for each body site were defined: (i) musculoskeletal complaint of back, neck, shoulder or hand/wrist was defined as pain in the past 12 months, which had continued for at least a few hours during the past 12 months, (ii) chronic musculoskeletal pain in the past 12 months, referred to a complaint that was present almost every day in the preceding 12 months with a minimal presence for at least 1 month, (iii) musculoskeletal complaint which led to a period of sickness absence in the past 12 months, and (iv) musculoskeletal complaint which led to medical care seeking in the past 12 months.

Results

Table 1: Mostly followed working position by the practitioners

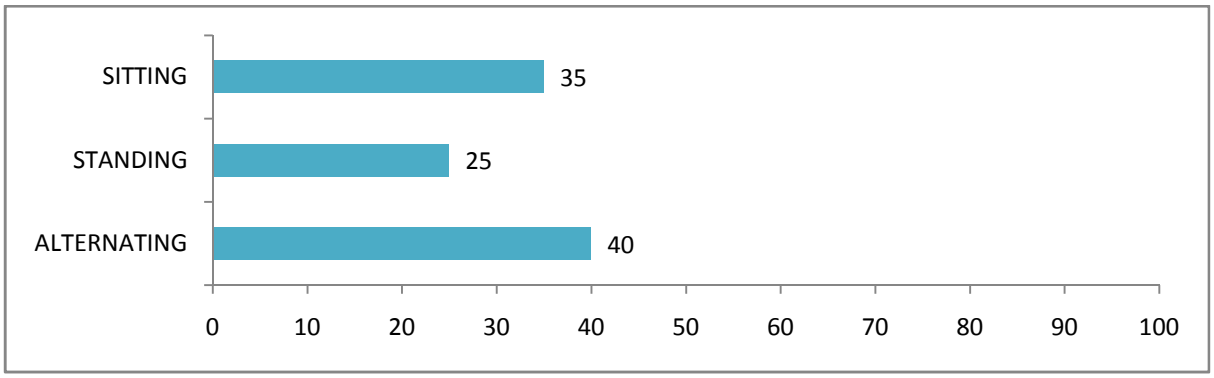


Table 2: The part of the body in which most discomfort is felt

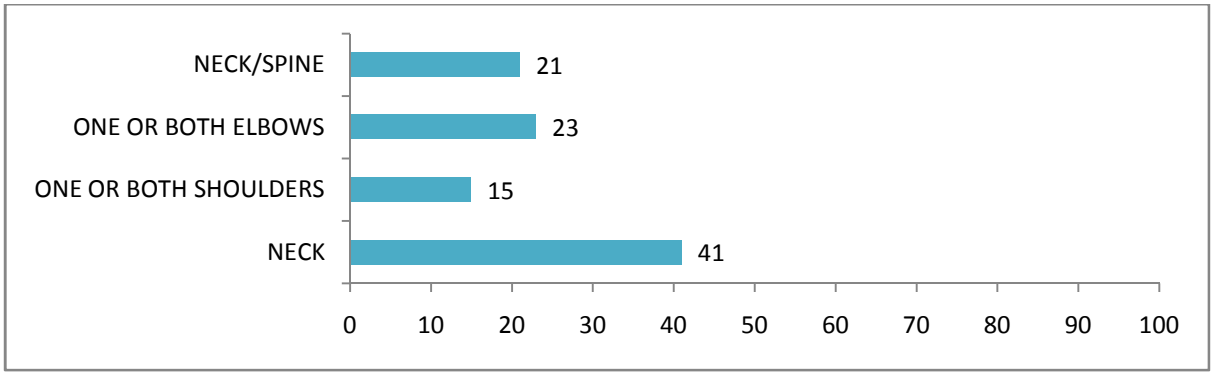
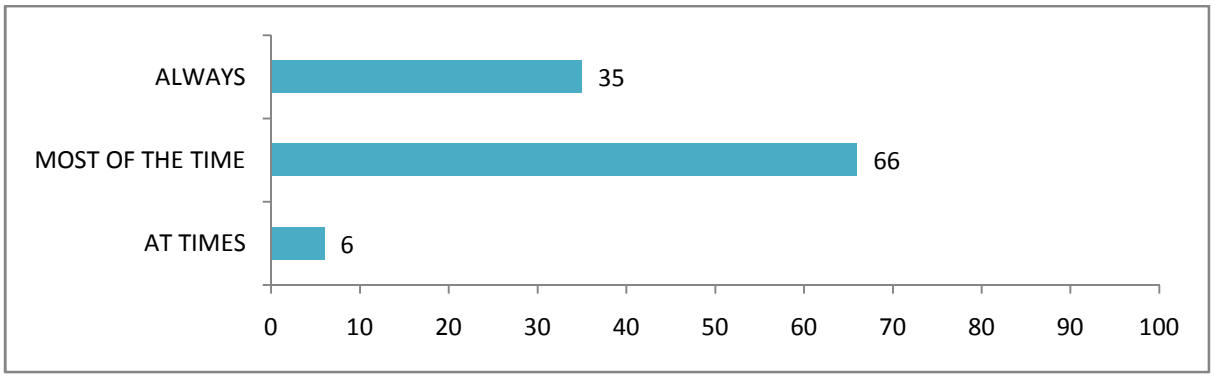


Table-3 Frequency of pain



Out of 300 questionnaires distributed, the female prevalence was higher.86% of the people don't do physical exercise daily.71% of the dental students take rest after each patient, 15% take rest once an hour and 13% of the students never take rest at all.95% of the dental students involve frequent bending of elbows and joints.91% of them feel some kind of musculoskeletal discomfort in the body and other 9% are free from it. Most discomfort is felt in the neck region that is 41% and 23% in one or both elbows,15% in one or both shoulders and 20% in back/spine.66% of the students feel the pain most of the time and 22% always and 11% at times.50% of the students follow periodic stretching and breaks as the pain relief strategy.36% follow postural awareness strategy and 13% follow strengthening exercises.

Discussion

The number of years of practice plays an important role in the occurrence of MSDs, although both younger and older dentists report the same symptoms, as confirmed in other studies also.Young general dental practitioners work very intensively in the first years of practice, often over 8 hours a day, which causes an early occurrence of MSDs, even within 3 years. Possibly due to experiencing pain and muscle stiffness they start to keep fit and work less intensively, this is why they do not experience the pain in the next few years.[6,7,8] Findings suggest that by raising awareness of these problems among students, the risk of MSDs can be reduced. It has been recommend that as a preventive measure; students should be taught, from the beginning of their undergraduate studies, about optimal working postures and good work habits. In this cross-sectional study, we found high prevalences for back pain, neck,

shoulder and hand/wrist complaints. Musculoskeletal problem was too high and a proportion of the subjects reported chronic complaints and seeking absenteeism.

Self-reported physical risk factors were important for the occurrence of musculoskeletal complaints whereas age, gender and perceived moderate/bad general health were strongly associated with chronicity of complaints and medical care seeking. Musculoskeletal sickness absence was related to perceived exertion and family situation.Inthis survey self-administered questionnaires were used to collect information about physical and psychosocial load and perceived health.Like most studies significant relations were found between self-reported physical risk factors and the occurrence of musculoskeletal disorders in various body sites. Since our study did control appropriately for concurrent risk factors, it was possible to disentangle the most important risk factors among the strongly interrelated factors of physical load, psychosocial load, and general health. The results demonstrate the importance to separate risk factors for the occurrence of musculoskeletal complaints from factors that determine their aggravation and consequences for disability. The occurrence of musculoskeletal complaints among dentists was associated with which seems to be especially important for hand/wrist complaints. Work-related psychosocial factors played an inconsistent role in the development of chronicity of complaints. The strongest impacts in all outcomes under study i.e. occurrence, chronicity, comorbidity and medical care seeking was held by perceived general health.[5,6,7]

Perceived health partly incorporate psychosocial factors as low control and working without breaks. High job demands increase the need for recovery, which is related to worst perceived health. This might reflect an influence particularly on more severe disease or an effect on people's ability to cope when symptoms occurred. The clear differences between work-related risk factors and general health with respect to the observed associations with different endpoints of musculoskeletal health calls for further exploration of these associations in various occupational groups and national settings.[8,9,10]Dental surgeons are normally included within the group of professionals at risk of suffering from MSDs due to prolonged awkward or forced postures at work and failure to adopt preventive measures.The present study found that most of the dental surgeons had some kind of musculoskeletal pain and stiffness while performing their professional work in the last 6 months.

The mechanism of musculoskeletal pain production has been studied extensively. The onset of modern dentistry, as evidenced by four-handed dentistry, has made the major part of the dentist tasks purely sedentary in nature. This has resulted in dramatic rise in musculoskeletal symptoms.Karwaski*et al*. reported that the symptoms are a product of many risk factors including prolonged static postures, repetitive movements, and poor positioning. Ratzen, on the other hand, linked musculoskeletal pain occurrence in the dentists to the frequent assumption of static postures, which usually requires more than 50% of the body's muscles to contract to hold the body motion less, while resisting gravity.[11,12] The static forces resulting from these postures have been shown to be much more tasking than dynamic forces. Repeated prolonged static postures are thought to initiate a series of events that could account for pain, injuries, or career-ending problems seen in MSDs.Lalumandir*et al*. reported that all dental specialties show a high occurrence of MSDs, but with variations in frequency and locations. In this study, we found that frequency of pain varies with the number of patients treated per day in the area of hip/thigh and with the height of the dentists in case of hip/thigh and knee joints.[13,14,15]

Conclusion

The prevalence of musculoskeletal disorders in young dentists is 41%. Neck is the most common affected region[16,17]. Stress and lack of exercise are the main associated factors for musculoskeletal problems in dentists. In conclusion, it was recognized that limited ergonomics in the work environment of the dentists' results in MSDs, and its prevalence is very high.The symptoms of MSDs increase with the number of years of practice. The prevention and reduction of MSDs among dentists should include their knowledge in dental ergonomics and awareness regarding the importance of work-related risk factors.[18,19,20]The musculoskeletal disorder is one of the disease that most related to the dentist work. Therefore dentists should implement right ergonomic designs in their dental clinics along with doing regular exercise and relaxation techniques, which help them to combat stress, thus improving the quality of life and resulting in consistent long-lasting work efficiency of the dentists.[21,22]

Corresponding Author

N.Mahathi,

Senior Lecturer,Department of Oral and Maxillofacial Surgery,
Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India
162, Poonamallee High Road, Chennai 600077, Tamil Nadu, India.
Telephone Number-9840958141
Email Id -nerallamahathi@gmail.com

References

1. Åkesson I, Johnsson B, Rylander L, Moritz U: Musculoskeletal dis- orders among female dental personnel – clinical examination and a 5-year follow-up study of symptoms. *Int Arch Occup Environ Health* 1999; 72, 395–403.
2. Åkesson I, Hansson GA, Balogh I, Moritz U, Skerfving S: Quanti- fying work load in neck, shoulders and wrists in female dentists. *Int Arch Occup Environ Health* 1997; 461–474.
3. Alexopoulos EC, Stathi IC, Charizani F: Prevalence of musculo- skeletal disorders in dentists. *BMC MusculoskeletDisord* 2004; 10(5), 1–8.
4. Al Wazzan K Almas K, Al Shethri SE: Back and neck problems among dentists and dental auxiliaries. *J Contemp Dent Pract* 2001;2, 17–30.
5. Finsen L, Christensen H, Bakke M: Musculoskeletal disorders among dentists and variation in dental work. *Appl Ergon* 1998; 29, 119–125.
6. Johnston V, Souvlis T, Jimmieson NL, Jull G: Associations between individual and workplace risk factors for self-reported neck pain and dis- ability among female office workers. *Appl Ergon* 2008; 39, 171–182.
7. Kerosuo E, Kerosuo H, Kanerva L: Self-reported health complaints among general dental practitioners, orthodontics, and office employees. *ActaOdontolScand* 2000; 58, 207–212.
8. Kierklo A, Kobus A, Jaworska M, Botuliński B, Pawińska M. Przerwyspoczynkowejako element profilaktykizagrożeń zdrowotnychlekarzystomatologów. Analiza w województwiepodlaskim. *Implanto-protStomKlin* 2008; 9, 39–42.

9. Burdorf A, Sorock G. Positive and negative evidence for risk factors of work-related back disorders. *Scand J Work Environ Health.* 1997;23:243–56.
10. Devereux JJ, Vlachonikolis IG, Buckle PW. Epidemiological study to investigate potential interaction between physical and psychosocial factors at work that may increase the risk of symptoms of musculoskeletal disorder of the neck and upper limb.*Occup Environ Med. 2002; 59:269–77.*
11. Alexopoulos EC, Burdorf A, Kalokerinou A. Risk factors for musculoskeletal disorders among nursing personnel in Greek hospital. Int Arch Occup Environ Health. 2003; 76:289–94.
12. Borg G. Psychophysical scaling with applications in physical work and the perception of exertion. Scand J Work Environ Health. 1990;16:55–58.
13. Karasek R, Brisson C, Kawakami N, Houtman I, Bongers P, Amick B. The job content questionnaire (JCQ): an instrument for internationally comparative assessments of psychosocial job characteristics. *Journal of Occupational Health Psychology.* 1998; 3:322–355.
14. VanSonsbeek JLA, De Voeg.A list of subjective health complaints. The Hague SDU/publishers (Statistical reports M37) 1990;451-471.
15. Sluiter JK, Van derBeek AJ, Frings-Dresen MHW. The influence of work characteristics on the need for recovery and experienced health: a study on coach drivers. Ergonomics. 1999; 42:573–583.
16. Ashish R Jain..A Novel Method for the Risk Assessment of Dental Practitioners in Chennai City for Occupational Musculoskeletal Disorders,Arthisri et al., *Biol Med (Aligarh*) 2016, 851-900
17. S.PaulSilvian, ArunMaiya, A.T. Resmi, Tom Page. Antecedents of work related musculoskeletal disorders in software professionals, *Int. J. of Enterprise Network Management* 2009; 65:516–24.
18. MohamadQulamZakiBin,MohamadRasidi and Gheena,Awareness On Musculoskeletal Disorder Among Dental Students In Chennai, *International Journal Of Current Research* 2016: 8, (12), 43868-43873.
19. Karahan A, Kav S, Abbasoglu A, Dogan N. Low back pain: Prevalence and associated risk factors among hospital staff. *J AdvNurs*. 2009; 65:516–24.
20. United States: Department of labour; Labour statistics Workplace injuries and illnesses in 2005; *Journal of Occupational Health Psychology.* 1998; 3:322–355.

21. Fulton-Kehoe D, Franklin G, Weaver M, Cheadle A. Years of productivity lost among injured workers in Washington State: Modeling disability burden in workers' compensation. *Am J Ind Med*. 2000; 37:656–62.
22. Stewart WF, Ricci JA, Chee E, Morganstein D, Lipton R. Lost productive time and cost due to common pain conditions in the US workforce. *JAMA*2003; 290:2443–54.
23. Subashri. A, T. N. Uma Maheshwari.Knowledge and Attitude of Oral Hygiene Practice among Dental Students,*Research Journal of Pharmacy and Technology 2016;9(11)*676-700.

APPENDIX-1:

The Questionnaire Is As Follows

PREVALENCE OF MUSCULOSKELETAL DISORDERS AMONG DENTAL STUDENTS - A QUESTIONNAIRE SURVEY

AGE- YEARS OF CLINICAL PRACTICE- NO OF CASES PER DAY-

1. Gender

a) Male b) Female

2. Will you do physical exercise daily?

a) Yes b) No

3. How long do you take rest while working?

a) Never b) Once in an hour c) Once for every patient

4. Which is your mostly followed working position?

a) Sitting b) Standing c) alternating

5. Does your working position involve frequent bending of elbows and joints?

a) Yes b) No

6. Do you feel any musculoskeletal discomfort in your body?

a) Yes b) No

7. If so, in which part of the body do you feel the most discomfort?

a) Neck b) One or both shoulders c) One or both elbows d) Back/Spine

8. Musculoskeletal symptoms are most felt in which part of the body?

a) Upper limb b) Lower limb c) Both

9. How long is the duration of pain?

a) At times b) Most of the time c) Always

10. Do you follow any pain relief measures?

a) Postural awareness strategy b) Periodic breaks and stretching c) Strengthening exercises

Questionnaire Survey about Knowledge of Incremental Techniques in Composite Placement among Dentists in South India

N.S.Harini[1], Jayalakshmi Somasundaram[2], Sankari M[3]

Graduate Student[1], Senior Lecturer[2], Department of Conservative Dentistry and Endodontics[2], Professor[3], Department of Periodontics.

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: Dental composite resins are types of synthetic resins which are used in dentistry as restorative material or adhesives. Synthetic resins evolved as restorative materials since they were insoluble, aesthetic, insensitive to dehydration. Polymerisation shrinkage is one of the major disadvantages in composites due to "c" factor. To reduce this configuration factor incremental techniques have been used. Several incremental techniquesbeing followed by dentists. Incremental techniques is one of the most comfortable and qualitative technique which reduces "c" factor in composites. This is a questionnaire survey which assess the most practiced incremental technique by various dentists.

Material and Method: A questionnaire survey about the most popularly used Incremental techniques being used in composite placement by several dentists have been analysed.

Results:70% of dentists are practicing incremental techniques in composite placement. The rest 30% of dentists are practicing bulk fill techniques. Of the dentists practicing incremental techniques 71.43% are following oblique layering technique, 14.29% are following three site novel matrix and the rest 14.29% are following successive cusp build up technique

Conclusion: Of the three incremental techniques, oblique layering technique has many advantages such as allows easier Class II build-up, with proper proximal contact and proximal smooth surfaces. Another advantage is that there is adequate light exposure for polymerization.

Key Words:*Knowledge, incremental techniques, composite placement, dentists, oblique layering.*

Introduction

We are now living in a era of aesthetic dentistry where tooth coloured restorations matching the shade of natural teeth are the most in demand. Since necessity is the mother of invention, evolution of composite resins has seen a cutting edge over other dental restorative materials.

Composite resins are widely used in dentistry to restore teeth with structural loss due to their aesthetics and physical properties. However, these restoratives have polymerization shrinkage as an inherent problem that may cause residual stresses in the tooth, even when not in function[1,2]. Clinical signs that have been associated with polymerization shrinkage stress include inadequate adaptation at tooth/restoration interface, micro-cracking, postoperative sensitivity, microleakage, and secondary caries[3,4].These issues are often responsible for replacement of composite restorations in posterior teeth[5,6]. Changes in material formulations and filling techniques, aimed at reducing volumetric contraction and shrinkage stress, have been the primary approaches for reducing the development of residual stresses[7,8].Composite restorations also hold other advantages such as they match the shade of the natural teeth, are Mercury- free, thermally non - conductive and bond to the tooth structures with the use of adhesive agents. Placing successful posterior composites is exacting, tedious and time consuming. The process includes achieving the necessary isolation, selecting and placing an appropriate matrix, precise execution of the adhesive steps, the placement of a flowable resin or resin ionomer liner and finally, the incremental placement, adaptation, and light curing of at least two or more layers of composite. Add to this sculpting, adjusting the occlusion and finishing and polishing and have a procedure which just takes too much time. This in turn can produce a profitability problem for dentists who have contracted with insurance companies. Given today's overhead per hour, dentist's need material and technology advancements so that posterior composites can be placed faster, easier, and profitably without taking compromising shortcuts[9,10].In recent years, materials have been introduced in an attempt to reduce some of the time and effort needed for layering and adaptation when placing posterior composites.

However the life of a resin composite restoration is dependent on several factors, including the cavity-composite interface sealing. From this view point factors related to gap formation mechanism are crucial in improving the clinical longevity of resin composite restorations. Although composite resins possess numerous advantages, one predominant disadvantage is polymerization shrinkage. This shrinkage can lead to failure of the restoration because of the "C" factor or Configuration factor. This "C" factor can be minimized by incremental technique. A lot of different incremental techniques have been proposed so far by various authors. Composite resins with high elastic modulus produce more rigid restorations, which increase

the effect of polymerization contraction on residual shrinkage stresses [11]. Filling techniques also influence stress distributions. The potential of incremental composite placement technique to reduce the shrinkage deformation and stress at the adhesive interface is controversial [12-14]. An incremental technique could increase shrinkage stresses due to incremental cuspal deformation by each polymerized increment. The incremental cuspal deformation also leads to a reduction in the volume of the cavity, reducing the amount of composite that is placed in subsequent increments [14].

Material and Method

A questionnaire survey containing 10 questions was taken to assess the knowledge about incremental techniques in composite placement among various dentists in south India for which the sample size was 400

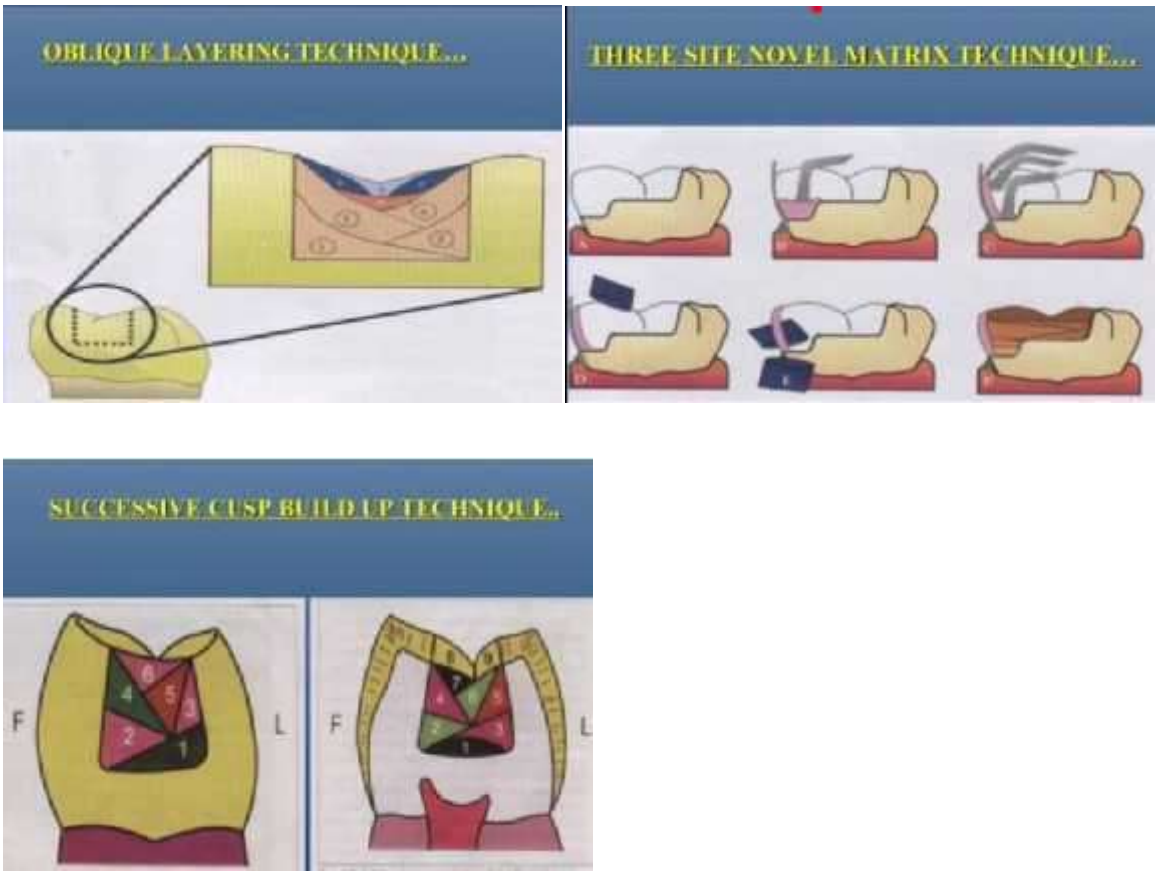


Figure 1: Diagram Showing Various Incremental Techniques in Composite Placement

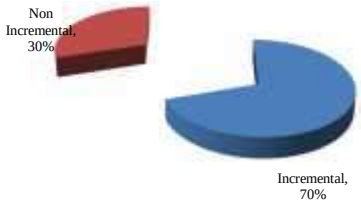
1. Years of Experience in dentistry?	10 years/15 years/> 15 years
2. Have you been trained in composite placement?	Yes/No
3. Do you prefer incremental technique or bulk placement technique?	Incremental/Bulk
4. Which incremental technique do you prefer in composite placement?	
Three site novels matrix/Oblique layering technique/Successive cusp build up	
5. Factors influencing to follow that technique?	
6. Disadvantages of that technique?	Yes/No
7. Overall grade for that technique?	Poor/Good/Excellent
8. Post treatment problems?	Present/Absent
9. Do you use any flowable composites under packable composite?	Yes/No
10. Is the patient satisfied with your composite placement?	Poor/Good/Excellent

Figure 2: Questionnaire

Results

This study is a survey which is aimed to find out the most commonest incremental techniques practiced among dentists. The results of the survey are given below:

70% of dentists are practicing incremental technique in composite placement. The rest 30% of dentists are practicing bulk fill technique. Among the dentists practicing incremental techniques, 71.43% are practicing oblique layering technique, 14.29% are practicing three site novel matrix technique,, and the rest 14.29% are practicing for successive cusp build up technique.



INCREMENTAL TECHNIQUE

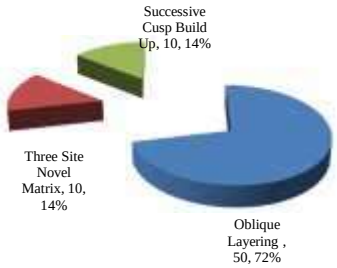


Figure 3: Pie chart showing various techniques used in composite placement among dentists

Discussion

Resin composite restorations have gained popularity because they match the shade of the natural teeth, are mercury-free and thermally non-conductive, and they bond to the tooth structure with the use of adhesive agents.

Direct dental composites can be used for:

- Filling cavity preparations
- Filling gaps which are diastemas between teeth using a shell-like veneer or
- Minor reshaping of teeth
- Partial crowns on single teeth. Although composites are now the material of choice for most restoration, their polymerisation shrinkage remains a problem.

Configuration Factor

Resin composite restorations have gained popularity because they match the shade of the natural teeth, are mercury-free and thermally non-conductive,[15] and they bond to the tooth structure with the use of adhesive agents[16]. Although composites are now the material of choice for most restoration, their polymerization shrinkage remains a problem.

Modern resin composites undergo volumetric contractions of between 2.6 and 7.1%,[17]resulting in shrinkage stress generation at the composite-tooth interface[18]. These stresses may cause the composite to pull away from the cavity margins, resulting in adhesive failure and marginal gap formation [19]. Oral fluids containing bacteria may fill these gaps, causing micro leakage and secondary caries. Other adverse consequences of shrinkage stresses include coronal deformation resulting in postoperative sensitivity, propagation of existing enamel micro cracks, and micro cracks of composite resin due to cohesive failure [20].

In 1987, Feilzer *et al*. postulated that the geometric configuration plays an important role in the adaptation of resin composite restoration [21]. The cavity configuration (C-factor) is defined as the ratio of bonded to unbonded surfaces. A high ratio denotes high polymerization stresses, which are accompanied by increased shrinkage stresses. Among many of the factors contributing to the shrinkage stresses, C-factor is an important one.

Several techniques have been suggested to improve marginal adaptation of high C-factor preparation, including adhesive systems that potentially resist composite shrinkage[22, 23] placement techniques for resin composites,[24,25]protocols for polymerisation and different cavity preparations[26].

Internal stress can be reduced in a restoration subject to potentially reduce high disruptive contraction forces by using:

- "soft-start" polymerization instead of high-intensity light curing
- incremental layering to reduce the effects of polymerization shrinkage; and
- a stress-breaking liner, such as filled adhesive, flowable composite, or resin modified glass ionomers.
- The application of non or low shrinking restorative materials[25,26].

The Incremental Layering Technique

Incremental layering has been advocated for use in large composite restorations to avoid the limitation of depth of cure, to reduce the effects of polymerization shrinkage, and to enhance the aesthetic results from the multilayering of colour. Incremental layering with successive layers of dentin and enamel composite creates high diffusion layers that allow optimal light transmission within the restoration, providing a more realistic depth of color as well as natural surface and optical characteristics. The polychromatic effect is achieved by stratifying variations in shades and opacities of the restorative composite.

Polymerization stresses generated by polymerization shrinkage may compromise the bond integrity, leading to concerns such as micro leakage, postoperative sensitivity, and ultimately secondary caries.If the composite-tooth bond remains intact, stresses transferred to tooth structure may result in cuspal flexure, enamel fracture, or fractured cusps.

Due to the variations in natural teeth, the combinations of different composite shades must be applied in relationship to the anatomy of the tooth, and are specifically adapted to different clinical situations.

In order to minimize micro leakage, different incremental insertion techniques have been suggested as a way to improve composite curing in-depth and minimize the effect of confinement on contraction stress development. Instead of using a bulk technique, layering techniques may have some advantages, that is, the use of a small volume of material, a lower cavity configuration factor and minimal contact with the opposing cavity walls during polymerization[10, 12]

Three Site Novel Matrix Techniques

The technique indicates the use of a metallic matrix band and wood wedges to provide an interproximal contour and contact with the adjacent tooth as well as to provide an adequate cervical adaptation. The first increment of the packable composite resin is applied on the gingival wall of the proximal box, packed cervically near the axial wall and, automatically, the resin climbs up in contact with the inner surface of the matrix band. This increment is sculpted and light-cured and the metallic matrix band is removed. Thus, the Class II cavities are transformed into Class I, with free access for light-curing. Small incremental layers of composite fill the remaining cavities. This technique is faster than conventional techniques and permits appropriate embrasure, better contour, and contact points[13-15].

Oblique Layering Technique

The presence of a high c-factor is a risk for debonding within the resin–dentin interface. For deep Class I cavities, horizontal layering is the most promising way to get a good bond to the cavity floor

Using four horizontal increments, achieved higher bond strengths and to reduce the possibility of cuspal flexure, a composite hybrid with a low volumetric polymerization shrinkage should be used. Opposing enamel walls should not be contacted by the same increment;this will minimize the wall-to-wall shrinkage and thus reduce intercuspal stress. The application of the composite in oblique layers results in fewer contraction gaps at the margins. It is important to continue to condense and shape the composite resin to correspond to cusp development and replacement of dentin[26].

Successive Cusp Build Up Technique

Successive cusp build-up is an improved placement technique for posterior direct resin restorations. By placing successive layers of apicoocclusal wedges of composite, the C-factor for each layer is extremely reduced. This technique gives up a glossy finish and is aesthetically much better than any other restoration.

In this study, 70% of the dentists are practicing incremental technique in composite placement. The rest 30% of dentists are practicing bulk fill technique. Among the dentists practicing incremental techniques, 71.43% are practicing oblique layering technique,14.29% are practicing three site novel matrix technique,, and the rest 14.29% are practicing successive cusp build up technique.[27]

Thus oblique layering technique is the most practiced incremental technique by various dentists in south India.

Reasons to prefer oblique layering technique:

Reduce "c"factor, High strength, Withstand maximum masticatory stress, Rapid and aesthetic, Allows easier Class II build-up, with proper proximal contact and proximal smooth surfaces. Another advantage is that there is adequate light exposure for polymerization[27].

Conclusion

Composite restorations have revolutionized restorative dentistry due to their conservative nature, adhesive bonding and patient appeal. The adhesive bonding, ability makes it unnecessary to remove tooth structure for retention, prevention, and convenience. Successful restorations can be done with less precise preparations [28].There is mercury present in the silver filling material, called amalgam, in the past century there has been no evidence showing that silver fillings are harmful to patients. But nowadays dentists doesn't use amalgam filling because of its various disadvantages like it can corrode over time, amalgam filling does not bond (hold together) with your tooth etc. Composite fillings are primarily a resin which has been" filled" with other inorganic materials. This compound makes a composite filling more resistant to wear, colour adjustable, and easier to polish [29].Composite resin, serves as esthetic alternative to amalgam and cast restorations [30].The marginal integrity of composite restoration refers to its marginal fit and marginal adaptation thereby increasing the longitivity of the restorative material [31].Resin composites have been the pinnacle of direct esthetic restorations ever since its discovery [32]. It is justifiable to speak of future dental practice because it takes many years for nascent technology emerging from scientific research laboratories to be developed and made commercially available for dentists' use. Therefore, some of the current research successes represent future improvements in patient care. The durability, reparability, and quality of the surface texture of composites are matters that are receiving considerable research. Anything which improves the strength and durability of this interaction could lead to restorations that are stronger and more durable. Future improvements in the level of oral hygiene, the use of fluorides, antiseptics, sealants, and protective coatings to prevent decay, together with improvements in composites to repair traumatically damaged or malformed teeth, can lead to better general health because of better oral health.

Corresponding Author

M.Sankari,

Professor, Department of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162 Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Telephone Number: 9176098012

Email Id: dr.sankarim@gmail.com

References

1. Marchesi G, Breschi L, Antoniolli F, Di Lenarda R, Ferracane J, & Cadenaro M (2010) Contraction stress of low-shrinkage composite materials assessed with different testing systems Dental Materials26(10) 947-953.
2. Jafarpour S, El-Badrawy W, Jazi H, & McComb D (2012) Effect of composite insertion technique on cuspal deflection using an in vitro simulation model Operative Dentistry37(3) 299-305.
3. Versluis A, & Tantbirojn D (2009) Relationship between shrinkage and stress In: Daskalaki A (ed) Dental Computing and Applications: Advanced Techniques for Clinical Dentistry.Hershey, PA: IGI Global. 45-64.
4. Burgess J, & Cakir D (2010) Comparative properties of low-shrinkage composite resins Compendium of Continuing Education in Dentistry 31(2) 10-15.
5. Mjor IA, & Toffenetti F (2000) Secondarycaries: A literature review with case reports Quintessence International 31(3) 165-179.
6. Brunthaler A, Konig F, Lucas T, Sperr W, & Schedle A (2003) Longevity of direct resin composite restorations in posterior teethClinical Oral Investigation 7(2) 63-70.
7. Ferracane JLResin composite—state of the art (2011) Dental Materials 27(1) 29-38.
8. Gao BT, Lin H, Zheng G, Xu YX, & Yang JL (2012) Comparison between a silorane-based composite and methacrylate-based composites: Shrinkage characteristics, thermal properties, gel point and vitrification pointDental Materials Journal 31(1) 76-85.

9. Manhart J, Chen H, Hickel R,Clinical Evaluation of the Posterior Composite Quixfil in Class I and II Cavities: 4-year Follow-up of a Randomized Controlled Trial, J Adhes Dent 2010; 12: 237-243.
10. Manhart J, Chen H, Hamm G, Hickel R,Review of the clinical survival of direct and indirect restorations in posterior teeth of the permanent dentition,Oper Dent 2004; 29: 481 – 508.
11. Versluis A, Tantbirojn D, & Douglas WH (2004) Distribution of transient properties during polymerization of a light-initiated restorative compositeDental Materials 20(6) 543-553.
12. Versluis A, Tantbirojn D, Pintado MR, DeLong R, & Douglas WH (2004) Residual shrinkage stress distributions in molars after composite restorationDental Materials 20(6) 554-564.
13. Campodonico CE, Tantbirojn D, Olin PS, & Versluis A (2011) Cuspal deflection and depth of cure in resin-based composite restorations filled by using bulk, incremental and transtooth-illumination techniquesJournal of the American Dental Association 142(10) 1176-1182.
14. Versluis A, Douglas WH, Cross M, & Sakaguchi RL (1996) Does an incremental filling technique reduce polymerization shrinkage stresses?Journal of Dental Research 75(3) 871-878.
15. Jordan RE, Suzuki M.Posterior composite restorations: Where and how they work best. J Am Dent Assoc. 1991;122:31–7.
16. Eakle WS.Fracture resistance of teeth restored with class-II bonded composite resin. J Dent Res. 1986;65:149–53.
17. Feilzer AJ, De Gee AJ, Davidson CL.Curing contractions of Composites and Glass ionomer cements. J Prosthet Dent. 1988;59:297–300.
18. Davidson CL, De Gee AJ, Feilzer AJ.The competition between the Composite-Dentin bond strength and the polymerisation contraction stress. J Dent Res. 1984;3:1396–9.
19. Lutz F, Krijci I, Barbkow F. Quality and durability of marginal adaptation in bonded Composite restorations. Dent Mat. 1991;7:107–13.
20. Feilzer AJ, De Gee AJ, Davidson CL.Setting stress in Composite resin in relation to configuration of the restoration. J Dent Res. 1987;66:1636–9.
21. Carvalho RM, Pereira JC, Yoshiyama M, Pashley DH.A review of polymerization contraction: The influence of stress development versus stress relief. Oper Dent. 1996; 21:17–24.
22. Swift E, Jr, Perdigao J, Heymann HO.Bonding to enamel and dentin: A brief history and state of the art. Quintessence Int. 1995;26:95–110.
23. Hansen EK.Effect of cavity depth and application technique on marginal adaptation of resins in dental cavities. J Dent Res. 1986;65:1319–21.
24. Versluis A Douglas WH, Cross M, Sakaguchi RL.Does an incremental technique reduces polymerization shrinkage stresses? J Dent Res. 1996;75:871–8.

25. Koran P, Kurschner R.Effectof sequential versus continuous irradiation of light-cured resin composite on shrinkage, viscosity, adhesion and degree of polymerisation. Am J Dent. 1998;11:17–22.
26. Mixson JM, Eick JD, Moore DL, Tira DE.Effect of two dentin bonding agents on microleakage in two different cavity designs. J Prosthet Dent. 1992;67:441–5.
27. Liebenberg WH.Successive cusp build-up: an improved placement technique for
28. posterior direct resin restorations. J Can Dent Assoc. 1996 Jun;62(6):501-7.
29. V. Susila Anand, C. Kavitha, and C. V. Subbarao; Effect of Cavity Design on the Strength of Direct Posterior Composite Restorations: An Empirical and FEM Analysis; International Journal of Dentistry; Volume 2011 (2011); p: 1-7.
30. Gayathri R Menon, G Karthikeyan, James D Raj; Recent advancement in restorative composite materials; International Journal of Recent Advances in Multidisciplinary Research Vol. 03, Issue 06, June, 2016; pp.1528-1530.
31. Rubeena Abdul Azeem, Nivedhitha Malli Sureshbabu; Clinical performance of direct versus indirect composite restorations in posterior teeth: A systematic review; 2018; 2(1); p : 2-9.
32. Priyalakshmi.S, Manish Ranjan;A Review on Marginal Deterioration of Composite Restoration; Volume 13, Issue 1 Ver. IV (Jan. 2014), PP 06-09.
33. Joshua Ng Chor Yang , James David Raj , Herald Sherlin; Effects of Preheated Composite on Micro leakage-An in-vitro Study;Journal of Clinical and Diagnostic Research : JCDR; 01 Jun 2016,10(6):ZC36-8]

Questionnaire Based Study on the Awareness of Need for Dental Treatment for the Children Administered to the Parents Visiting Saveetha Dental College and Hospitals.

S.Renuka[1], Gheena.S[2],M.Sankari[3],

Graduate Student[1],Reader[2],Department of Oral Pathology,Professor[3], Department of Periodontics

Saveetha Dental College,Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

Abstract

Introduction: The foundation for healthy permanent teeth in children and teenagers is laid during the first years of life. Poor oral hygiene and inadequate tooth brushing habits during the first two years of life can cause dental problems in future according to several studies. This study aims at evaluating the awareness of the need for dental treatment among parents of school going children visiting Saveetha dental college and hospitals.

Materials and Methods: Poor dental health in preschool-aged children can lead to pain [1,2], may affect their growth and quality of life [3-6]and may have a chance of developing future dental problems. Growing children need proper guidance for healthy growth, upkeep and hygiene of their teeth and parents need to be educated on the importance of dental treatment. Parents should receive counselling on appropriate oral hygiene procedures, fluoride supplementation,oral habits and also need for clinical intervention for their children because knowledge regarding risk amongst parents may be variable. A convenient sample of 150 participants who are parents of school going children visited Saveetha Dental College and hospitals for treatment were included.

Results: This study shows that the importance of parental knowledge on their child's dental health. Parents are not fully familiar with oral hygiene techniques and dental treatment methods but more awareness is required for them to instill good oral health in their children.

Key Words: *dental treatment, oral hygiene, fluoride, oral habits, occlusion.*

Introduction

The foundation for healthy permanent teeth in children and teenagers is laid during the first years of life. Poor oral hygiene and inadequate tooth brushing habits during the first two years of life can cause dental problems in future according to several studies[7]. Therefore, it is essential to establish proper oral hygiene early in life to ensure good oral health. Parents have to make their children aware about the importance of oral hygiene. But parents were not proactive in making sure that their children received regular dental care. Dental caries is the most common chronic disease of childhood that interferes with normal nutrition intake, speech, and daily routine activities[8,9].Parents' knowledge and attitudes about the importance of oral health care and their fears about dental treatment influenced their children's dental care [10]. Dental care has been systematically organized to improve dental health attitudes among children and the young [11]. For young children, the influence of parent's belief and behaviour is known to be strong [12-16]. Parents from low social status were found to be less accepting of the more "ultimate" techniques such as general anaesthesia [17]. The purpose of this study is to check the awareness of dental treatment among parents. It is essential for the parents to be aware of the current dental treatment milieu for their child's oral health care.

Material and Methods

This is a questionnaire based study on the awareness of the need for dental treatment for the children administered to the parents visiting Saveetha Dental College and hospitals. The participants include parents having children in both rural as well as urban side in Tamil Nadu. The questionnaire is filled in paper and pen method. After the data collection, statistical measurements are done with SPSS software version. Data is collected from a convenient sample of 150 participants under the supervision of dental students.

The questions mentioned in the Appendix have been asked to the parents through the questionnaire. The answers were marked according to their knowledge. Among 27 questions, seventeen questions are of yes or no type and the others are questions with appropriate answers given as choices.

Results

This study included a total of 150 parents visiting Saveetha dental college and hospitals with their children. Their child's ages ranged from five years to thirteen years with a mean of 8.85(S.D 2.49). The descriptive statistics of child's age of the participants are shown below in the Table 1.

General awareness about both dental and general health among the population is 105 of them are aware that the health of mouth and dentition impact the health of the body with the percentage of 70% and 24 of them felt that the health of mouth and dentition does not impact the general health of the body with percentage of 16%, 21 of them are not aware about this concept with the percentage of 14%. 55 of them are aware that the poor oral health is not good for cardiac health with the percentage of 36.7% and 73 of them felt that the poor oral health does not affect the cardiac health with percentage of 48.7%, 22 of them are not aware about this concept with the percentage of 14.7%. 75 of them are aware that the caries affect their child's dental aesthetics with the percentage of 50% and 38 of them felt that the caries affect your child's dental aesthetics with percentage of 25.3%, 37 of them are not aware about this concept with the percentage of 24.7%. 102 of them are aware that the treatment of toothache is as important as any organ in the body with the percentage of 68% and 32 of them felt that treatment of toothache is not as important as any organ in the body with percentage of 21.3%, 16 of them are not aware about this concept with the percentage of 10.7%. 60 of them care about their teeth than the body parts with the percentage of 40% and 73 of them does not care about their teeth than the body parts with percentage of 48.7%, 17 of them are not aware about this concept with the percentage of 11.3%. 65 of them are aware that the caries will affect their teeth appearance with the percentage of 43.3% and 54 of them felt that the caries will not affect their teeth appearance with percentage of 36.0%, 31 of them are not aware about this concept with the percentage of 20.7%. All data are enclosed as a form of table and graph below as Table 2 and Graph 1 respectively.

Awareness about caries attack among the population is 80 of them felt that the sweets will affects their child's dental health with the percentage of 53.3% and 41 of them felt that the sweets will not affect their child's dental health with percentage of 27.3%, 29 of them are not aware about this concept with the percentage of 19.3%.In case of soft drinks, 64 of them felt that the soft drink will affects their child's dental health with the percentage of 42.7% and 60 of them felt that soft drink will not affect their child's dental health with percentage of 40.0%, 26 of them are not aware about this concept with the percentage of 17.3%. For Ice creams,93 of them felt that ice cream will affects their child's dental health with the percentage of 62.0% and 27 of them felt that ice cream will not affect their child's dental health with percentage of 18.0%, 30 of them are not aware about this concept with the percentage of 20%. All datas are enclosed as a form of table and graph below as Table 3 and Graph 2 respectively.

The meaning of gum bleeding was asked to check their knowledge about the dental problems. In that, 11 of them said that it means a healthy gum with the percentage of 7.3% and 54 of them felt that it is an inflamed gum with percentage of 36.0%, 41 of them said that it means gum recession with the percentage of 27.3% and 44 of them are not aware about that dental term with the percentage of 29.3%. All datas are enclosed as a form of table and graph below as Table 4 and Graph 3 respectively.

The question is about how they will protect themselves from the gum bleeding. In that, 28 of them said that they will use tooth brush,paste and floss to protect themselves from gum bleeding with the percentage of 18.7% and 22 of them said that they will use soft food to protect them from gum bleeding with percentage of 14.7%, 73 of them said that they will consult doctor for gum bleeding with the percentage of 48.7% and 27 of them are not aware about that knowledge of gum bleeding with the percentage of 18.0%. All data’s are enclosed as a form of table and graph below as Table 5 and Graph 4 respectively.

The meaning of dental plaque was asked to check their knowledge about the dental problems. In that, 33 of them said that it means a soft deposit on the tooth surface with the percentage of 22.0% and 34 of them felt that it is an heavy deposit on the tooth surface with the percentage of 22.7%, 40 of them said that it means tooth discolouration with the percentage of 26.7%

and 43 of them are not aware about that dental term with the percentage of 28.7%. All data's are enclosed as a form of table and graph below as Table 6 and Graph 5 respectively.

The question is about what does the dental plaque leads to. In that, 33 of them said that it will leads to inflammation of gum with the percentage of 22.0% and 32 of them said that it will leads to staining of the teeth with percentage of 21.3%, 38 of them said that it will leads to dental caries with the percentage of 25.3% and 47 of them are not aware about that knowledge of dental plaque with the percentage of 31.3%. All data's are enclosed as a form of table and graph below as Table 7 and Graph 6 respectively.

Awareness about the brushing habit and role of parents in their child's oral hygiene among the population is 79 of them said that their children brushes twice a day with the percentage of 52.7% and 60 of them said that their children do not brush their teeth twice a day with the percentage of 40.0%, 11 of them do not care about that with the percentage of 7.3%. The next question is about whether their children are using tooth brush and paste for brushing their teeth to check that they are knowledgeable about their child's dental hygiene. In that, 96 of them said that their children are using tooth brush and paste for brushing with the percentage of 64.0% and 44 of them said that their children are not using tooth brush and paste for brushing with the percentage of 29.3%, 10 of them are not aware about that with the percentage of 6.7%. The question is asked to the participants about does brushing will prevent dental caries. In that, 70 of them said that brushing will prevent dental caries with the percentage of 46.7% and 43 of them said that brushing will not prevent dental caries with percentage of 28.7%, 37 of them are not aware about this concept with the percentage of 24.7%. The most important question is asked to parents that they supervisetheir children while brushing or not. In that, 98 of them said that they are supervising their children while brushing with the percentage of 65.3% and 44 of them said that they are not supervising their children while brushing with the percentage of 29.3%, 8 of them are not aware about this concept with the percentage of 5.3%. 48 of them felt that fluoride will strengthen their teeth with the percentage of 32.0% and 43 of them felt that fluoride will not strengthen their teeth with percentage of 28.7%, 59 of them are not aware about this concept with the percentage of 39.3%. 57 of them felt that they can decide the treatment whatever they need with the percentage of 38.0% and 73 of them felt they cannot decide the treatment whatever they need

with percentage of 48.7%, 20 of them are not aware about this concept with the percentage of 13.3%. All data are enclosed as a form of table and graph below as Table 8 and Graph 7 respectively.

The question is about how long their children are brushing their teeth. In that, 30 of them said that their children are brushing their teeth less than 1 min with the percentage of 20.0% and 56 of them said that their children are brushing their teeth 2 min with percentage of 37.3%, 43 of them said that their children are brushing more than 2 min with the percentage of 28.7% and 21 of them will not supervise their children while they are brushing with the percentage of 14.0%. All data's are enclosed as a form of table and graph below as Table 9 and Graph 8 respectively.

The question is about what their children used for brushing their teeth. In that,101 of them said that their children are using tooth brush and paste along with floss for brushing their teeth with the percentage of 67.3% and 23 of them said that their children are using mouth wash instead of brushing with percentage of 15.3%, 18 of them said that their children are using tooth picks instead of brushing with the percentage of 12.0% and 8 of them will not aware about what their children are using for brushing with the percentage of 5.3%. All data's are enclosed as a form of table and graph below as Table 10 and Graph 9 respectively.

The question is about how often they will change their toothbrush. In that, 44 of them said that they will change their tooth brush in the interval of 3 months with the percentage of 29.3% and 39 of them said that that they will change their tooth brush in the interval of 6 months with percentage of 26.0%, 44 of them said that that they will change their tooth brush only when damage occurs with the percentage of 29.3% and 23 of them will not change their brush at anytime with the percentage of 15.3%. All data's are enclosed as a form of table and graph below as Table 11 and Graph 10 respectively.

The question is about how often they will visit their dentist. In that, 41 of them said that they will regularly visit their dentist with the percentage of 27.3% and 62 of them said that that they will visit their dentist only during pain with percentage of 41.3%, 23 of them said that they will visit their dentist occasionally with the percentage of 15.3% and 24 of them will

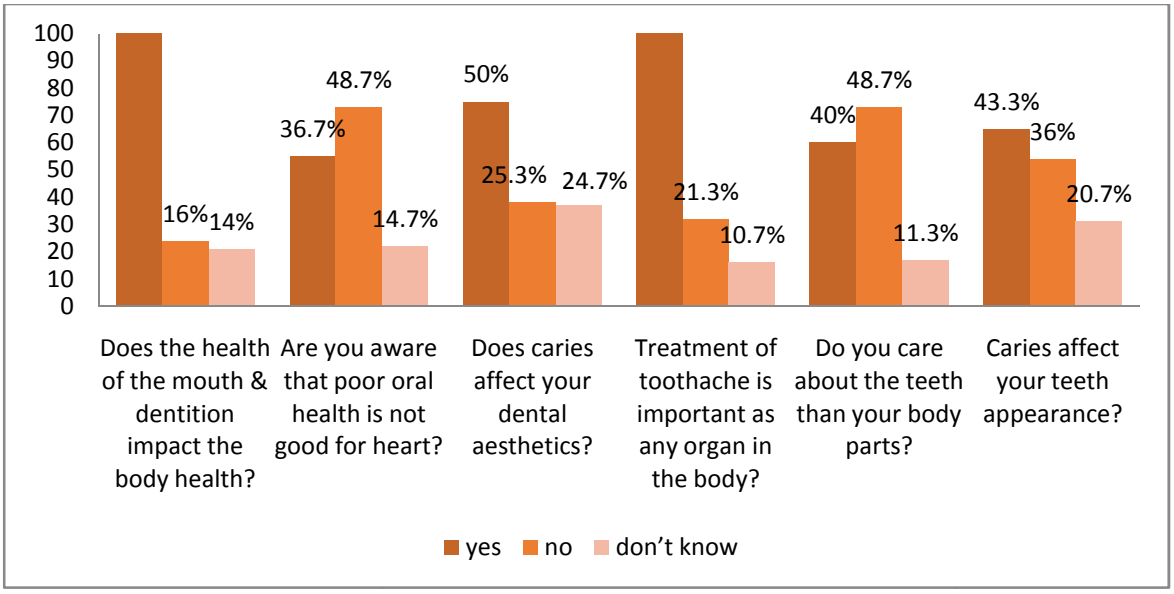
never visit the dentist with the percentage of 16.0%. All data's are enclosed as a form of table and graph below as Table 12 and Graph 11 respectively.

63 of them are aware that regular visit to the dentist is necessary with the percentage of 42.0% and 46 of them felt that regular visit to the dentist is not necessary with percentage of 30.7%, 41 of them are not aware that the regular visit to the dentist is necessary with the percentage of 27.3%. 68 of them said that dentist always explain procedures before treatment with the percentage of 45.3% and 34 of them felt that the dentist will not explain procedures before treatment with percentage of 22.7%, 48 of them are not aware as they will never visit the dentist with the percentage of 32.0%. All data's are enclosed as a form of table and graph below as Table 13 and Graph 12 respectively.

The question is about when they visited the dentist recently. In that, 48 of them said that they have visited the dentist 6 months before with the percentage of 32.0% and 27 of them said that they have visited the dentist 1-2 years before with percentage of 18.0%, 26 of them said that they have visited the dentist more than 2 years before with the percentage of 17.3% and 49 of them will never visit the dentist with the percentage of 32.7%. All data's are enclosed as a form of table and graph below as Table 14 and Graph 13 respectively.

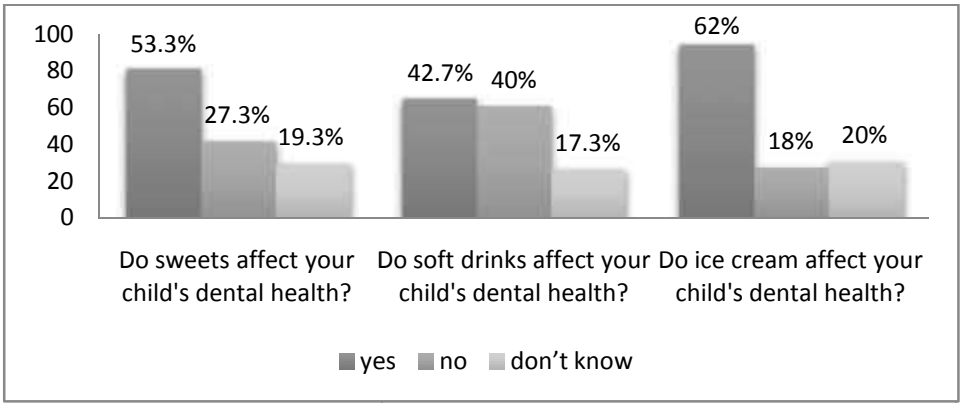
The question is about reason behind not visiting the dentist. In that, 27 of them said that they are afraid of needle with the percentage of 18.0 % and 51 of them said that they have no time to visit the dentist with percentage of 34.0%, 35 of them said that they are not interested to visit the dentist with the percentage of 23.3% and 37 of them are not aware in visiting the dentist with the percentage of 24.7%. All data's are enclosed as a form of table and graph below as Table 15 and Graph 14 respectively.

Graph 1: General awareness about both the dental and general health.



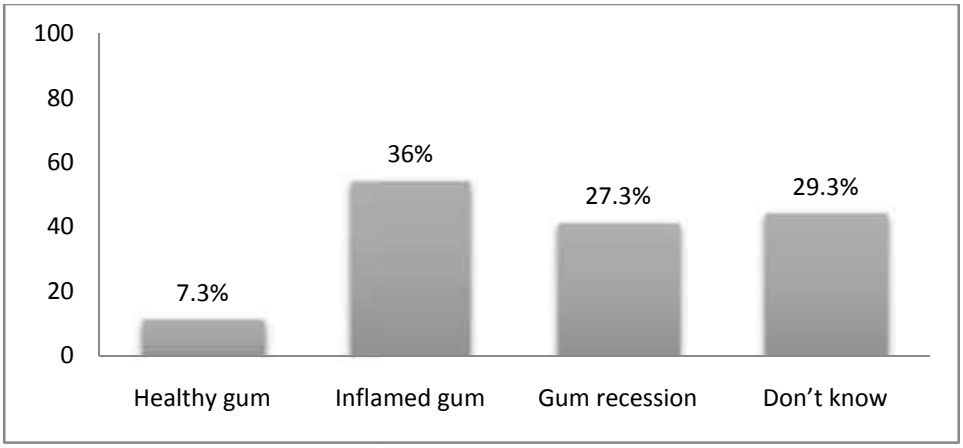
70% parents thought that body health have impact on oral health and dentition, 36.7% parents have an opinion that poor oral health affects the heart, 50% parents thought that caries affects their aesthetics and 68% parents thought that treatment for toothache is important.

Graph2: Awareness about the Caries attack.



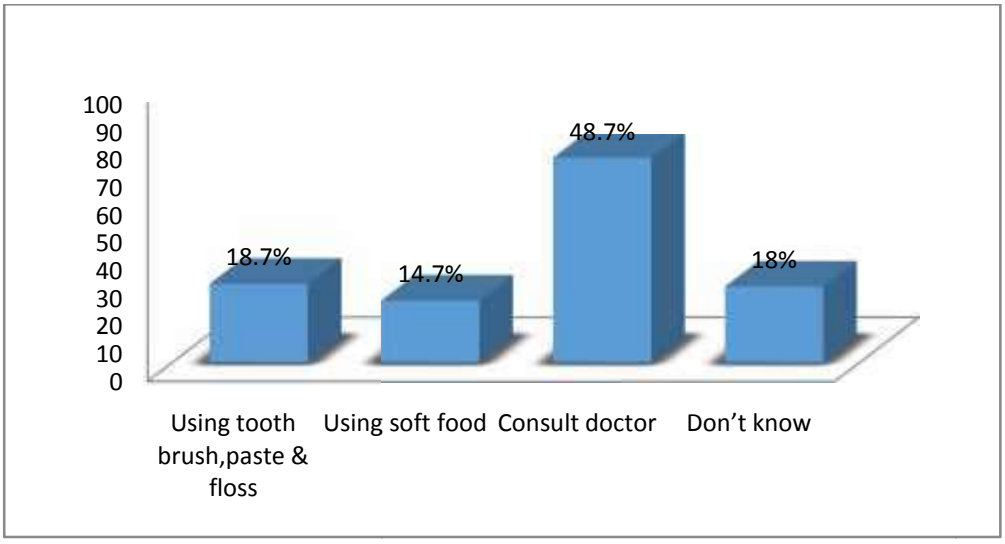
53.3% parents think that sweets, 42.7% parents think that soft drinks, 62% parents think that ice cream affects their child's oral health.

Graph 3: Knowledge about the term Gum bleeding.



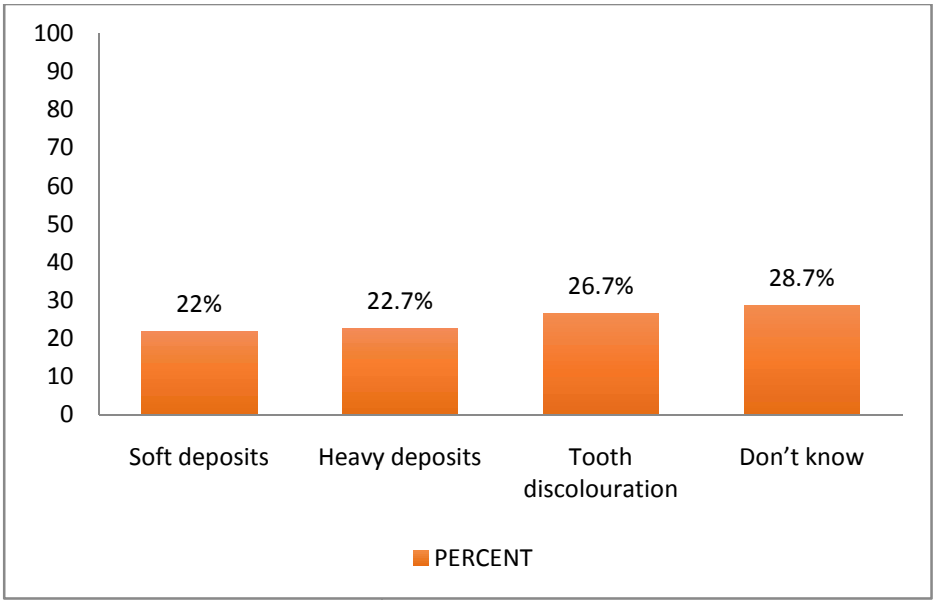
Knowledge about the term gum bleeding was significantly known only by 36% study population.

Graph 4: Knowledge about the Gum bleeding treatment.



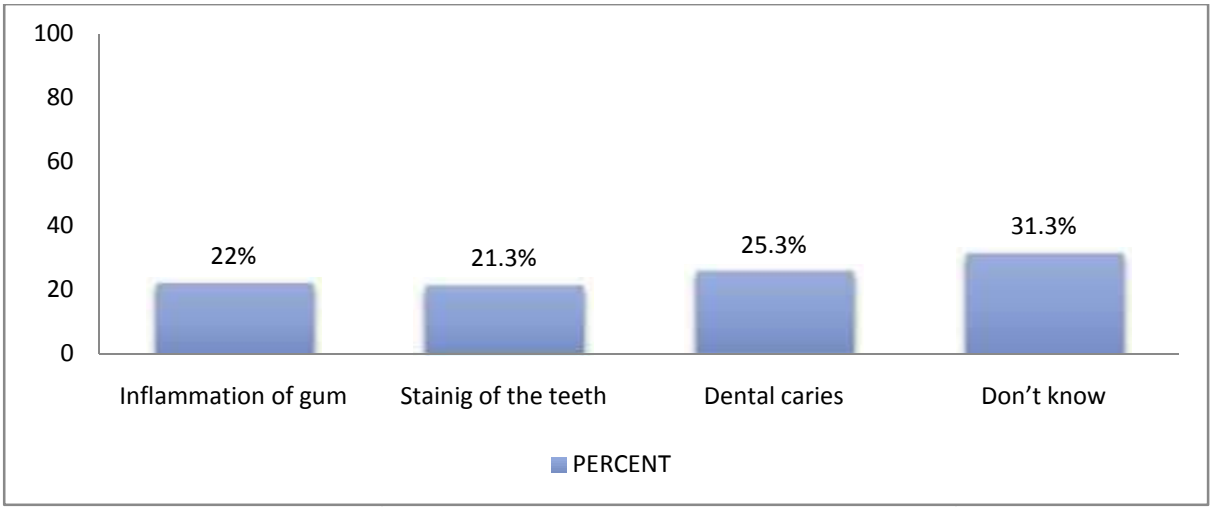
48.7% parents only think that for the treatment of gum bleeding should consult a doctor, 33.4% parents thinks that using tooth brush, paste, floss, soft brush will solve gum bleeding.

Graph 5: Knowledge about the term Plaque.



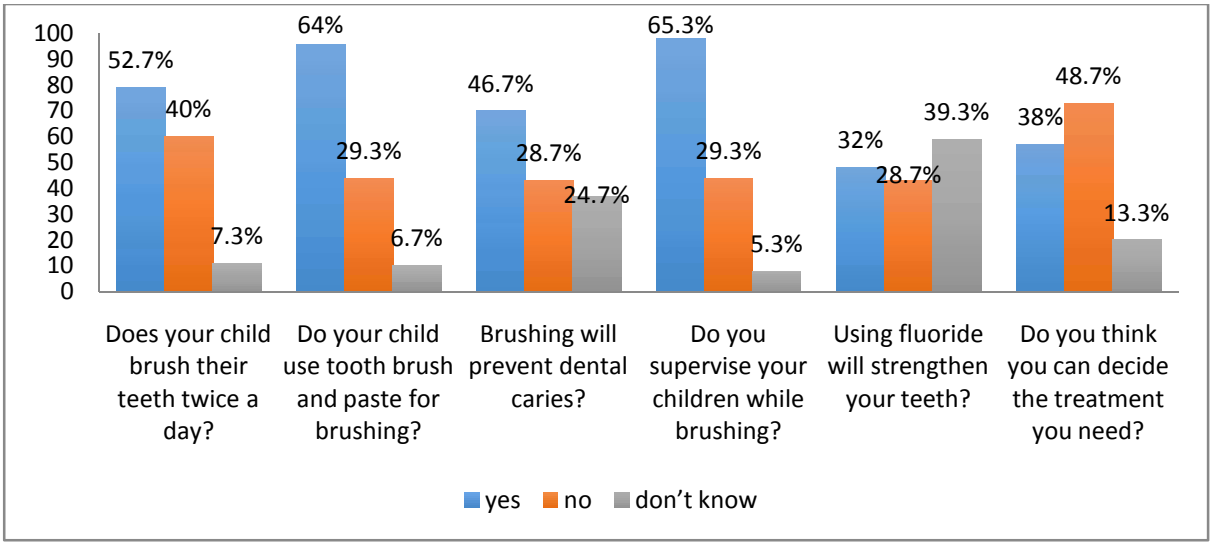
Knowledge about the term plaque was significantly known by 22% as soft deposits.

Graph 6: Knowledge about the Plaque etiology.



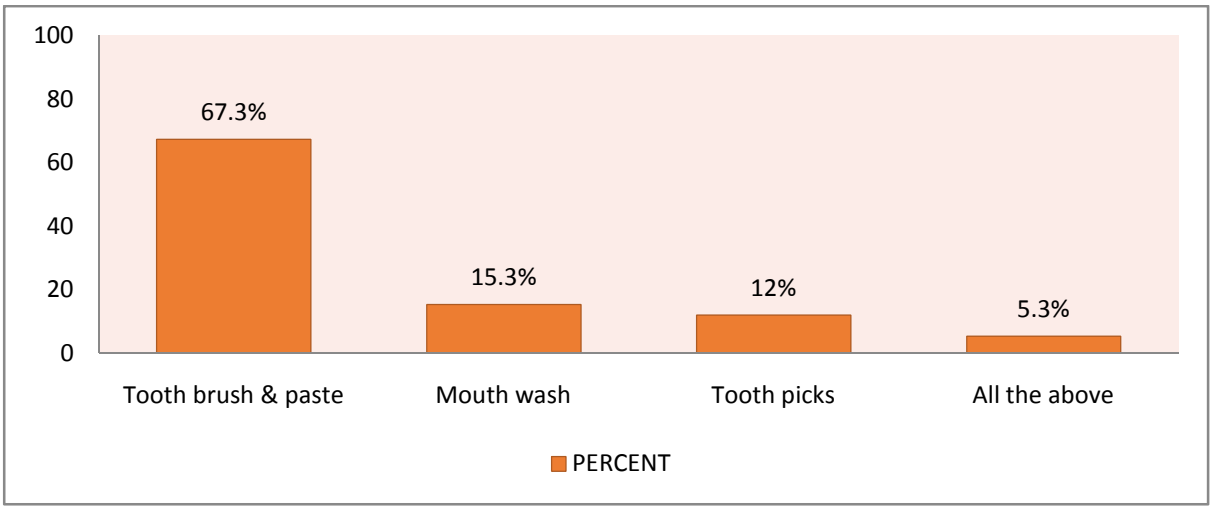
25.3% parents think that plaque causes dental caries and 22% parents think that it causes inflammation of gum and 31.3% parents had no idea about it.

Graph 7: Awareness about the brushing habits and the role of parents in their child's oral hygiene.



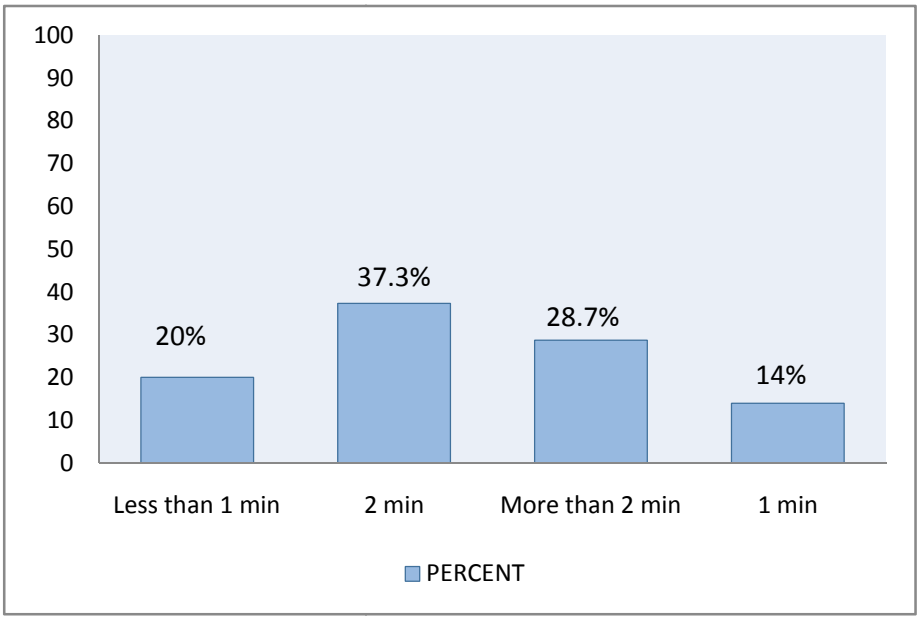
52.7% study population said that their child brushes twice daily and 64% population uses tooth brush and paste for brushing, 46.7% population believe that brushing will prevent caries, 65.3% parents supervise their children during brushing, 32% population believe that fluoride will strengthen the teeth and 48.7% population will never decide their treatment on their own.

Graph 8: Knowledge about the oral habits of their children.



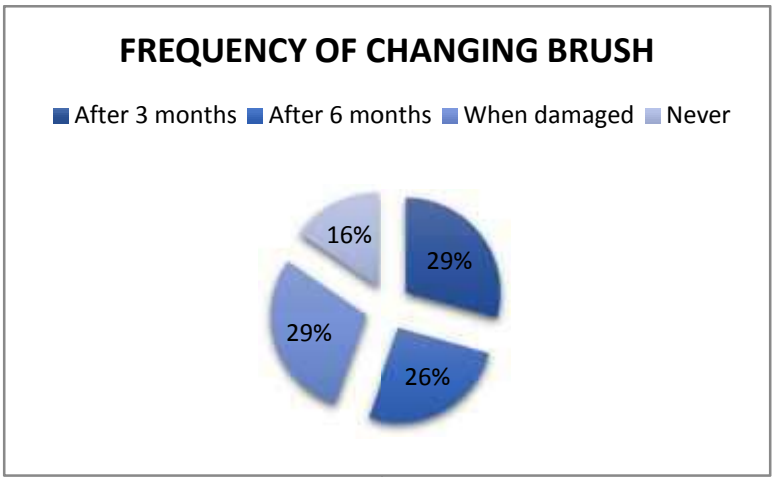
67.3% uses tooth brush and paste to maintain their oral hygiene and 12% uses tooth picks to clean their teeth.

Graph 9: Knowledge about the brushing habits of their children.



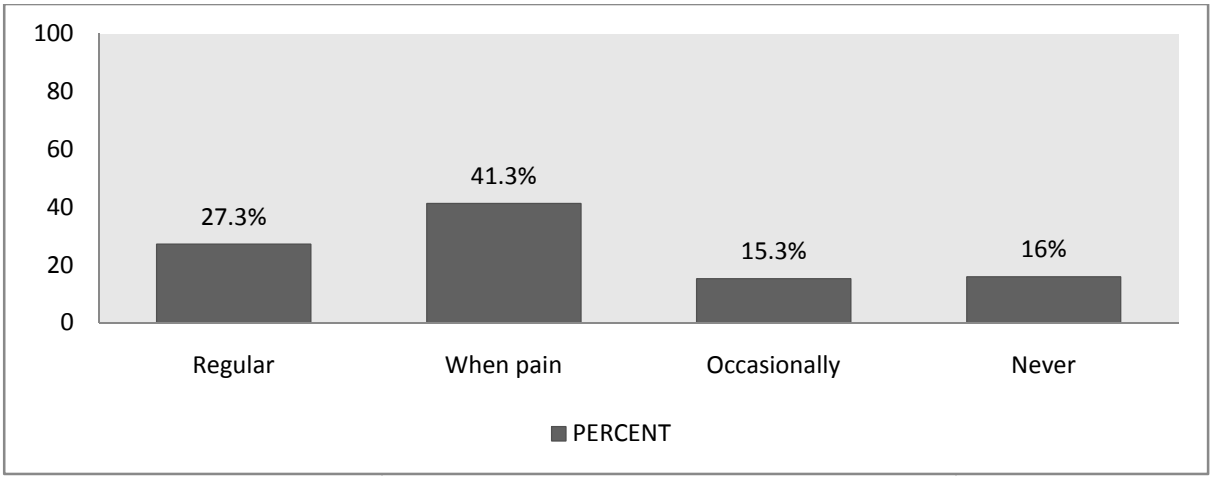
Only 37.3% parents teach their child to brush for 2 minutes.

Graph 10: About the frequency of changing the tooth brush of their child.



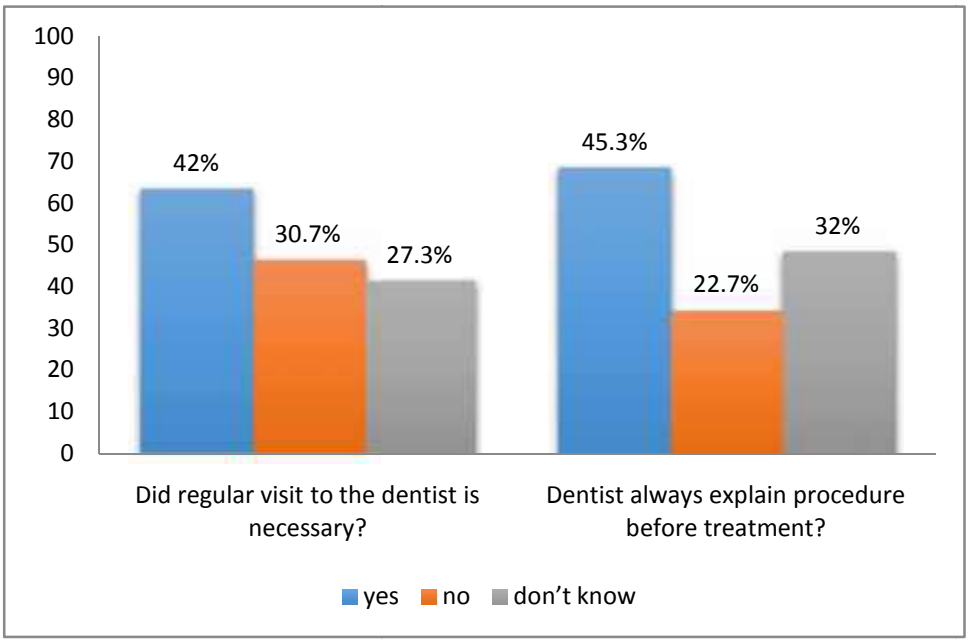
Only 55% study population care on their children dental health at regular intervals.

Graph 11: Knowledge about the dental visit.



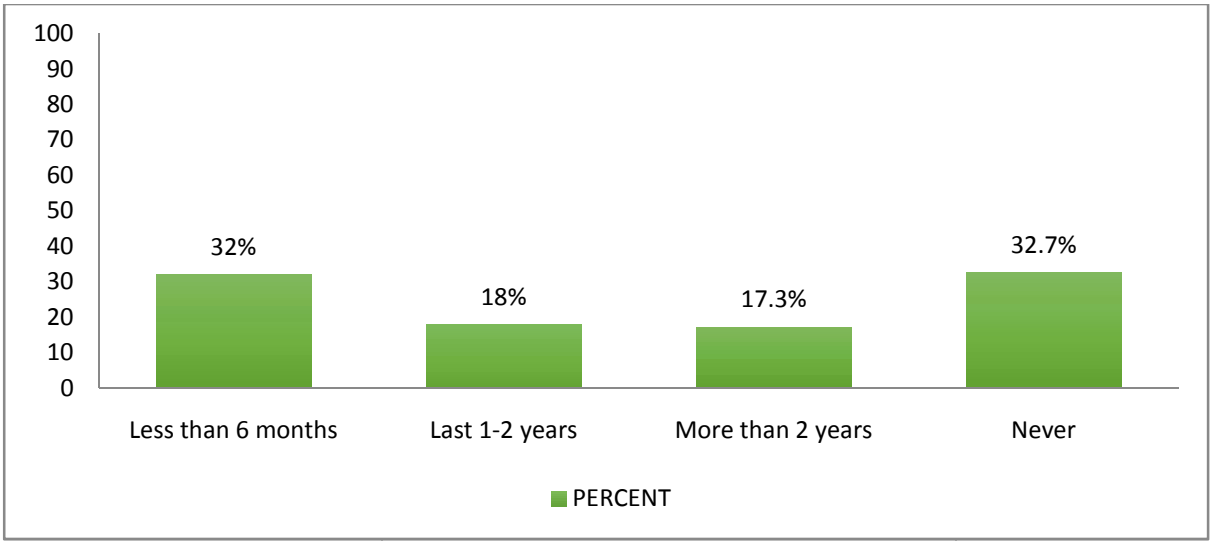
Only 27.3% study population think that regular dental check-up is important for proper oral health.

Graph 12: Knowledge about the importance of dental visit.



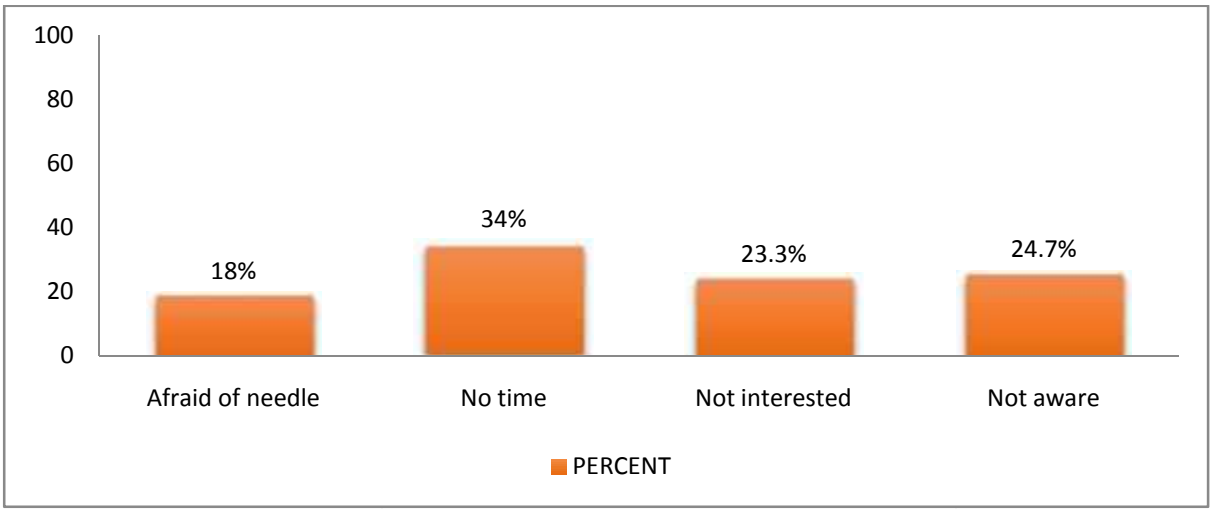
42% parents think that regular visit to dentist is important and 45.3% believe that the dentist will explain the procedure before treatment.

Graph 13: Knowledge about the regular visit to the dentist.



32% study population think that regular dental visit less than months is important.

Graph 14: Reason behind not visiting the dentist.



18% population not visiting the dentist because of needle fear and 34% is because of no time for the visit.

Table 1: The statistical description about the child's age

	N	Mean	Std. Deviation
CHILD'S AGE	150	8.85	2.494

Table 2: The statistical description about the awareness on dental and general health.

Does the health of mouth and dentition impact the health of the body?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	105	70.0	70.0	70.0
	b	24	16.0	16.0	86.0
	c	21	14.0	14.0	100.0
	Total	150	100.0	100.0	
Are you aware that poor oral health is not good for heart?					
Valid	A	55	36.7	36.7	36.7
	B	73	48.7	48.7	85.3
	C	22	14.7	14.7	100.0
	Total	150	100.0	100.0	
Does caries affect your child's dental aesthetics?					
	A	75	50.0	50.0	50.0
	B	38	25.3	25.3	75.3
	C	37	24.7	24.7	100.0
	Total	150	100.0	100.0	
Treatment of toothache is as important as any organ in the body?					
Valid	A	102	68.0	68.0	68.0
	B	32	21.3	21.3	89.3
	C	16	10.7	10.7	100.0
	Total	150	100.0	100.0	
Do you care about the teeth than your body parts?					
	A	60	40.0	40.0	40.0
	B	73	48.7	48.7	88.7
	C	17	11.3	11.3	100.0

	Total	150	100.0	100.0	
Caries affect teeth appearance?					
Valid	A	65	43.3	43.3	43.3
	B	54	36.0	36.0	79.3
	C	31	20.7	20.7	100.0
	Total	150	100.0	100.0	

Table 3: The statistical description about the awareness on caries attack.

Do sweets affect your child's dental health?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	80	53.3	53.3	53.3
	b	41	27.3	27.3	80.7
	c	29	19.3	19.3	100.0
	Total	150	100.0	100.0	
Do soft drinks affect your child's dental health?					
Valid	A	64	42.7	42.7	42.7
	B	60	40.0	40.0	82.7
	C	26	17.3	17.3	100.0
	Total	150	100.0	100.0	
Do soft drinks affect your child's dental health?					
Valid	A	93	62.0	62.0	62.0
	B	27	18.0	18.0	80.0
	C	30	20.0	20.0	100.0
	Total	150	100.0	100.0	

Table 4: The statistical description about the awareness on term gum bleeding.

What does gum bleeding mean?				
	Frequency	Percent	Valid Percent	Cumulative Percent

Valid	A	11	7.3	7.3	7.3
	B	54	36.0	36.0	43.3
	C	41	27.3	27.3	70.7
	D	44	29.3	29.3	100.0
	Total	150	100.0	100.0	

Table 5: The statistical description about the awareness on gum bleeding treatment.

How do you protect yourself from bleeding?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	28	18.7	18.7	18.7
	B	22	14.7	14.7	33.3
	C	73	48.7	48.7	82.0
	D	27	18.0	18.0	100.0
	Total	150	100.0	100.0	

Table 6: The statistical description about the awareness on the term plaque.

What does plaque mean?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	33	22.0	22.0	22.0
	B	34	22.7	22.7	44.7
	C	40	26.7	26.7	71.3
	D	43	28.7	28.7	100.0
	Total	150	100.0	100.0	

Table 7: The statistical description about the awareness on the plaque etiology.

What does dental plaque leads to?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	33	22.0	22.0	22.0
	B	32	21.3	21.3	43.3
	C	38	25.3	25.3	68.7
	D	47	31.3	31.3	100.0
	Total	150	100.0	100.0	

Table 8: The statistical description about the awareness about the brushing habits and the role of parents in their child's oral hygiene.

Does your child brush their teeth twice a day?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	79	52.7	52.7	52.7
	B	60	40.0	40.0	92.7
	C	11	7.3	7.3	100.0
	Total	150	100.0	100.0	
Do your child use tooth brush and paste for brushing?					
Valid	A	96	64.0	64.0	64.0
	B	44	29.3	29.3	93.3
	C	10	6.7	6.7	100.0
	Total	150	100.0	100.0	
Brushing will prevent dental caries / decay?					
Valid	A	70	46.7	46.7	46.7
	B	43	28.7	28.7	75.3
	C	37	24.7	24.7	100.0
	Total	150	100.0	100.0	
Do you supervise your child while brushing?					

Valid	A	98	65.3	65.3	65.3
	B	44	29.3	29.3	94.7
	C	8	5.3	5.3	100.0
	Total	150	100.0	100.0	
Using fluoride will strengthen your teeth?					
Valid	A	48	32.0	32.0	32.0
	B	43	28.7	28.7	60.7
	C	59	39.3	39.3	100.0
	Total	150	100.0	100.0	
Do you think you can decide the treatment you need?					
Valid	A	57	38.0	38.0	38.0
	B	73	48.7	48.7	86.7
	C	20	13.3	13.3	100.0
	Total	150	100.0	100.0	

Table 9: The statistical description about the awareness about the oral habits of their children.

How long do your children brush their teeth?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	30	20.0	20.0	20.0
	B	56	37.3	37.3	57.3
	C	43	28.7	28.7	86.0
	D	21	14.0	14.0	100.0
	Total	150	100.0	100.0	

Table 10: The statistical description about the knowledge on oral habits of their children.

What does your child use for brushing their teeth?					
		Frequency	Percent	Valid Percent	Cumulative Percent

Valid	A	101	67.3	67.3	67.3
	B	23	15.3	15.3	82.7
	C	18	12.0	12.0	94.7
	D	8	5.3	5.3	100.0
	Total	150	100.0	100.0	

Table 11: The statistical description about the care of the parents on their children dental health.

How long you use your tooth brush?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	44	29.3	29.3	29.3
	B	39	26.0	26.0	55.3
	C	44	29.3	29.3	84.7
	D	23	15.3	15.3	100.0
	Total	150	100.0	100.0	

Table 12: The statistical description about the knowledge on dental visit.

How often do you visit your dentist?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	41	27.3	27.3	27.3
	B	62	41.3	41.3	68.7
	C	23	15.3	15.3	84.0
	D	24	16.0	16.0	100.0
	Total	150	100.0	100.0	

Table 13: The statistical description about the knowledge on importance of dental visit.

Did regular visit to the dentist is necessary?

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	63	42.0	42.0	42.0
	b	46	30.7	30.7	72.7
	c	41	27.3	27.3	100.0
	Total	150	100.0	100.0	
Dentist always explain procedures before treatment?					
Valid	A	68	45.3	45.3	45.3
	B	34	22.7	22.7	68.0
	C	48	32.0	32.0	100.0
	Total	150	100.0	100.0	

Table 14: The statistical description about the knowledge on regular of dental visit.

Last time, you visited the dentist was?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	48	32.0	32.0	32.0
	B	27	18.0	18.0	50.0
	C	26	17.3	17.3	67.3
	D	49	32.7	32.7	100.0
	Total	150	100.0	100.0	

Table 15: The statistical description about the reason behind not visiting the dentist.

Reason behind not visiting the dentist?					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	A	27	18.0	18.0	18.0
	B	51	34.0	34.0	52.0
	C	35	23.3	23.3	75.3
	D	37	24.7	24.7	100.0
	Total	150	100.0	100.0	

Figure.1: Questionnaire of the Study

Discussion

This study provides the awareness about dental treatment for the children administered to the parents. The result showed that the parents are aware to a certain extent about the dental treatments and their children's oral hygiene. But still the parents seem to be not in touch with the oral symptoms of their children [18]. In another study, they found that parents who did not obtain dental care for themselves were less likely to bring their children for dental care [19]. A recent study states that parental knowledge and awareness are necessary prerequisite for

change in behaviour, including behaviour related to health and disease prevention [20]. Most parents believed that inadequate tooth cleaning and sweets,snacks and drinks are contributed to early child caries [21]. According to this study, 53.37%,42.7% and 62.0% parents are aware about the prevalence of caries due to sweets,ice cream and drinks. 50% and 43.3% people are aware that the caries will affect their child's teeth appearance. 46.7% of the parents believe that brushing will prevent dental caries.

Exposure to fluoride is known to be protective against caries development [22,23]. According to this study, only 32.0% of the parents felt that fluoride will strengthen their teeth. Parents must be aware of the appropriate amounts of toothpaste to apply on the brush or cleaning cloth [21]. In this study, only 64.0% of the children using tooth brush and paste for cleaning their teeth and 15.3% of the children using mouth wash instead of brushing. An another study among Hong Kong children stated that among who had a toothbrush habit, only 49% are reported using toothpaste [27].

A recent survey found that a high percentage of the children in their study brush their teeth at least once daily although this effort was not fully organized or supported by parents [9]. According to this study, 52.7% of the children brush their teeth twice a day and 40.0% of them are not brushing their teeth twice a day.

In this study, 65.3% of the parents supervise their children while brushing. But a finding by Lee, who reported that only 45% of parents helped their children to brush their teeth [26].

Parents' failure to organize or support their children's tooth brushing efforts coincided with findings from previous studies that reported lack of acceptable levels of knowledge and awareness of periodontal problems among Jordanian adults [24,25]. The participants demonstrated positive attitudes towards their dentists and high awareness of the link between oral health and systemic well-being [21]. In this study, 70% of the parents felt that the health of mouth and dentition impact the health of the body and 42.0% of the parents have positive attitudes towards the dental visit and dental treatments.

Lack of parental encouragement and advice to visit the dentist might also contribute to the irregular dental attendance. Lack of parents' regular dental attendance might be reflected in their children [21]. According to this study, 58% of the parents are not aware about regular dental visit. Fear of dental treatments was found to be high among the school children in North Jordan [9]. In this study, 18.0 % of the children are afraid of needle. It can also be a reason for lack of regular dental attendance of the parents and children.

Consequently, dental health education programs that aim to improve oral health practice among the population are very important. Hence, there is a need for educational programs to improve the oral health practice, knowledge, and attitudes of the general population [21].

Conclusion

This study shows that the importance of parental knowledge on their child's dental health. Parents are not fully familiar with oral hygiene techniques and dental treatment methods but more awareness is required for them to instill good oral health in their children. The results of this study will be helpful for the health care providers to conduct more public education programs about oral hygiene in the future. The importance of parental support in managing the oral hygiene of their children is mentioned.

Corresponding Author

M.Sankari,

Professor, Department Of Periodontics,

Saveetha Dental College,Saveetha Institute of Medical and TechnicalSciences,Saveetha University, Chennai, India

162, Poonamallee High Road,Chennai 600077, Tamil Nadu, India.

Email Id: msankari@gmail.com

Telephone Number: +91 9176098012

References

1. Slade GD. Epidemiology of dental pain and dental caries among children and adolescents. Community Dent Health 2001; 18: 219227.

2. Slade GD, Spencer AJ, Davies MJ, Burrow D. Intra-oral distribution and impact of caries experience among South Australian school children. Aust DentJ 1996;41:343–350.
3. Ayhan H, SuskanE,Yildirim S. The effect of nursing or rampant caries on height, body weight and head circumference. J ClinPediatr Dent 1996; 20: 209–212.
4. Acs G, Shulman R, Ng MW, Chussid S. The effect of dental rehabilitation on the body weight of children with early child hood caries.Pediatr Dent 1999; 21: 109–113.
5. Thomas CW, Primosch RE. Changes in incremental weight and well-being of children with rampant caries following complete dental rehabilitation. Pediatr Dent 2002;24:109–113.
6. Anderson HK, Drummond BK, Thomson WM. Changes in aspects of children oral health-related quality of life following dental treatment under general anaesthesia. Int J Paediatric Dent 2004;14:317–325.
7. Ahamed ST. Awareness of Oral Hygiene among Children in Chennai. Research Journal of Pharmacy and Technology. 2016;9(8):1055-8.
8. Prabakar J, John J, Srisakthi D. Prevalence of dental caries and treatment needs among school going children of Chandigarh. Indian J Dent Res 2016;27:547-52.
9. Naziya KB, Pradeep Kumar R, Arumughamm IM, Srisakthi D. Prevalence of dental caries among primary schoolchildren in Chennai - A cross-sectional study. J AdvPharmEdu Res 2017;7(2):150-152.
10. Mahmoud K. Al-Omiri, B.D.S., Ph.D., FDS RCS (Eng.), Jor Board, Ahed M. Al-Wahadni, B.D.S., M.D.Sc., Ph.D. and Khaled N. Saeed, B.D.S., M.Sc. Oral Health Attitudes, Knowledge, and Behavior Among School Children in North Jordan. Journal of Dental Education February 1, 2006 vol. 70 no. 2 179-187.
11. Holst D, Schuller A, Grytten J. Future treatment needs in children, adults, and the elderly. Community Dent Oral Epidemiol 1997;25:113–8.
12. Sallis JF, Nader PH. Family determinants of health behaviors. In: GochmanDS, ed. Health behavior. Emerging research perspectives. New York: Plenum Press, 1988; 107–124.
13. Bush JP, Melamed BG, Cockrell CS. Parenting children in a stressful medical situation. In: MillerTW, ed. Stressful life events. Madison: International Universities Press, 1989; 643–657.

14. Murphey DA. Constructing the child: relations between parents' beliefs and child outcomes.Dev Rev 1992; 12: 199–232.
15. Inglehart MR, Tedesco LA. The role of the family in preventing oral diseases. In: CohenLK, GiftHC, eds. Disease prevention and oral health promotion. Socio-dental sciences in action. Copenhagen: Munksgaard, 1995; 271–305.
16. Bhavna S. Talekar B.D.S., M.P.H.R. Gary Rozier D.D.S., M.P.H.Gary D. Slade B.D.Sc., D.D.P.H., Ph.D.Susan T. Ennett M.S.P.H., Ph.D. Parental perceptions of their preschool-aged children's oral health. The Journal of the American Dental Association. March 2005, Vol.136 (3):364–372.
17. Havelka C, McTigue D, Wilson S, Odom J: The influence of social status and prior explanation on parental attitudes toward behavior management techniques. Pediatr Dent 14:376–81, 1992.
18. Weyant RJ, Manz M, Corby P, Rustveld L, Close J. Factors associated with parents' and adolescents' perceptions of oral health and need for dental treatment. Community Dent Oral Epidemiol 2007; 35:321–330.
19. Inyang A. Isong, Katharine E. Zuckerman, Sowmya R. Rao, Karen A. Kuhlthau, Jonathan P. Winickoff and James M. Perrin. Association between Parents' and Children's Use of Oral Health Services. Paediatrics 2010; 125; 502.
20. Green W, Kreuter M. Health promotion planning: an educational and ecological approach. 3rd edn. Mountain View, CA: Mayfield Pub.Co., 1999.
21. MG Gussy,*§ EB Waters, EM Riggs ,SK Lo, NM Kilpatrick§. Parental knowledge, beliefs and behaviours for oral health of toddlers residing in rural Victoria. Australian Dental Journal 2008; 53:52–60.
22. Chan SC, Tsai JS, King NM. Feeding and oral hygiene habits of preschool children in Hong Kong and their care givers dental knowledge and attitudes. Int J Paediatric Dent 2002; 12:322–331.
23. Davies GM, Worthington HV, Ellwood RP,.A randomised controlled trial of the effectiveness of providing free fluoride tooth paste from the age of 12 months on reducing caries in 5–6 year old children. Community Dent Health 2002; 19:131136.
24. Rajab LD, Petersen PE, Bakeen G,. Oral health behaviour of school children and parents in Jordan. Int J Pediatr Dent 2002; 12:168-76.

25. Taani DQ. Periodontal awareness and knowledge and pattern of dental attendance among adults in Jordan. Int Dent J 2002; 52:94-8.
26. Lee AMP. Preschool oral health education programme, mid-programme surveys, part I: Survey on the oral health education delivered at maternal and child health centres, Department of Health, Hong Kong, 1997.
27. Chan, SCL; Tsai, JSJ; King, NM. Feeding and oral hygiene habits of preschool children in Hong Kong and their caregivers' dental knowledge and attitudes. International Journal of Paediatric Dentistry, 2002, v. 12 n. 5, p. 322-331.

Questionnaire Survey on Cervical Spondylosis among Dentists

N.S.Harini[1], Saravana Kumar[2], Sankari M[3]

Graduate Student[1], Professor[2], Department of Anatomy[2], Professor[3], Department of Periodontics.

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences, Saveetha University, Chennai, India

Abstract

Introduction: Most patients particularly dentists who present with neck pain have “non-specific (simple) neck pain, “where symptoms have a postural or mechanical basis. Aetiological factors are usually multifactorial, including poor posture, anxiety, depression, neck strain, and sporting or occupational activities. When mechanical factors are prominent, the condition is often referred to as “cervical spondylosis,” although the term is often applied to all non-specific neck pain. The aim of this study was to find the frequency of neck pain among dentists.

Materials and Methods:A cross sectional survey was carried out by convenience sampling in different dental hospitals of Chennai. A Total of 100 questionnaires were filled by dentists. The data was collected using a questionnaire focusing on back position, travel to clinic, hours of practice, assisting hands during practice, duration of sleep,time spent on electronic gadgets, dizziness or discomfort after work, duration of pain and if any treatment undergoing. Qualitative variables were represented as percentages and bar charts. Descriptive statistics that is mean and standard deviation were used to represent the total score.

Results: Neck pain was the most common complaint. 67% were males, 33% were females. 34% are practising dentistry above 20 years, 32% above 15 years, 26% above 10 years, and 8% above 5 years. The position of practice is that 96% of the dentists practise sitting position and the rest 4% practise standing position. 72% of dentists uses electronic gadgets more than 3 hrs, 18% of dentists uses for 3 hrs and 10 of them uses lesser than 3 hrs. 90% of dentists faces discomfort around neck and shoulder after work whereas the rest10% do not.

Conclusion: The frequency of neck pain and neck disability in dentists seemed to be high and is an area that needs further deliberation. The majority of working dentists have musculo-skeletal symptoms in the neck.

Key Words:*cervical spondylosis, dentists, neck pain, posture, Ergonomics.*

Introduction

Most patients who present with neck pain have “non-specific (simple) neck pain,” where symptoms have a postural or mechanical basis. Aetiological factors are poorly understood [(1)] and are usually multifactorial, including poor posture, anxiety, depression, neck strain, and sporting or occupational activities.[(2)] Neck pain after whiplash injury also fits into this category, provided no bony injury or neurological deficit is present.[(3)] When mechanical factors are prominent, the condition is often referred to as “cervical spondylosis,” although the term is often applied to all non-specific neck pain. Mechanical and degenerative factors are more likely to be present in chronic neck pain.

In cervical spondylosis, degenerative changes start in the intervertebral discs with osteophyte formation and involvement of adjacent soft tissue structures. Many people over 30 show similar abnormalities on plain radiographs of the cervical spine, however, so the boundary between normal ageing and disease is difficult to define.[(4)]

Even severe degenerative changes are often asymptomatic, but can lead to neck pain, stiffness, or neurological complications. Neck pain is an ache or discomfort in anatomical area between occiput and 3rd thoracic vertebra and laterally between middle margin of scapula [(5)]. Neck pain is considered to be chronic if the patient suffered more than 3 months [(6,7)].

The prevalence for neck pain varies between 16.7% and 75.1% in the general population [(8)]. Neck pain may be caused by; spondylosis, spinal stenosis, disc herniation, stress, poor posture, prolonged posture [(9,10)].

Symptoms of neck pain may include; neck soreness and headache, pain around shoulder blades, arm complaints (pain, numbness or weakness). Dental profession is one of the occupational risk factors of neck pain [(5,11)]. Studies showed that neck pain starts early in dental career even during educational training [(12, 13).]

In a recent study, 54.4% of dentists in K.S.A. had neck pain, 21.69% of them drop out of job [(14).] Recently, it was shown that there is a strong relation between work duration and neck pain in the staff of dental school [(15,16).] Another study reported that increasing weekly work hours has little effect on neck pain [(14,17).]

The most common cause of neck pain in dentists is prolonged work posture which makes high load on muscles of neck and shoulder region (trapezius more than splenius)[(16).] The main working posture of dentist is in sitting, with neck flexion, shoulder elevation and upper extremity abduction and his patient supine [(14,15,17,18).]

Materials and Methods

A cross sectional survey was carried out by convenience sampling in different dental hospitals of Chennai. A Total of 100 questionnaires were filled by dentists, in which 67 were males and 33 were females. The data was collected using a questionnaire focusing on socio-demographic data, back position, travel to clinic, consultation per day, hours of practice, assisting hands during practice, specialisation ,duration of sleep, type of mattresses and pillows, time spent on electronic gadgets, dizziness or discomfort after work, duration of pain and if any treatment undergoing. Qualitative variables were represented as percentages and bar charts. Descriptive statistics that is mean and standard deviation were used to represent the total score.

Table 1: Questionnaire survey on cervical spondylosis among dentists

	AGE:		YEARS		
	GENDER:	MALE	FEMALE		
	YEARS OF PRACTICE:		YEARS		
1	HOW DO YOU COMMUTE TO YOUR CLINIC	2 WHEELER	CAR		
2	HOW MANY HOURS DO YOU WORK A DAY?	4 HRS	8 HRS	>8 HRS	
3	ANY CONSULTANCY APART FROM YOUR CLINIC	YES	NO		
4	IF YES NUMBER OF CONSULTANCY IN DAY				
5	BACK POSITION	WITH BACK REST	W/OUT BACK REST		
6	ASSISTING HANDS DURING PRACTICE	SINGLE HANDED	TWO HANDED	FOUR	

				HANDED	
7	YOUR POSTURE DURING TREATMENT	SITTING	STANDING		
8	SPECIALIZATION	ORAL SURGERY (EXTRACTION)			
		PERIO SURGERY			
		RCT ENDODONTICS			
		PROSTHO IMPLANTS			
		PEDODONTICS			
9	DURATION OF SLEEP	<7 HRS	7 HRS	>7 HRS	
10	FREQUENCY OF TRAVEL UNDERTAKEN				
11	TYPE OF MATTRESS & PILLOWS	HARD	SOFT		
QUESTIONAIRE SURVEY ON CERVICAL SPONDYLOSIS AMONG DENTISTS					
12	NUMBER OF PILLOWS FOR HEAD DURING SLEEP	ONE	TWO		
13	TIME SPENT ON ELECTRONIC GADGETS		HOURS		
14	ANY DIZZINESS DURING UP AND DOWN MOVEMENT OF NECK	YES	NO		
15	ANY DISCOMFORT AROUND NECK AND SHOULDER AFTER WORK	YES	NO		
16	IF YES DURATION OF PAIN		MONTHS		YEA

17	TREATMENT UNDERWENT/UNDERGOING	MEDICATION	PHYSIOTHERAPHY	YOGA	EXC

The survey was conducted without any bias, among various dentists in south India. Once the survey was done, the results were tabulated according to the data obtained. Pie charts, statistics were used for concluding the results. The results obtained from the survey are estimated below using pie charts.

Results

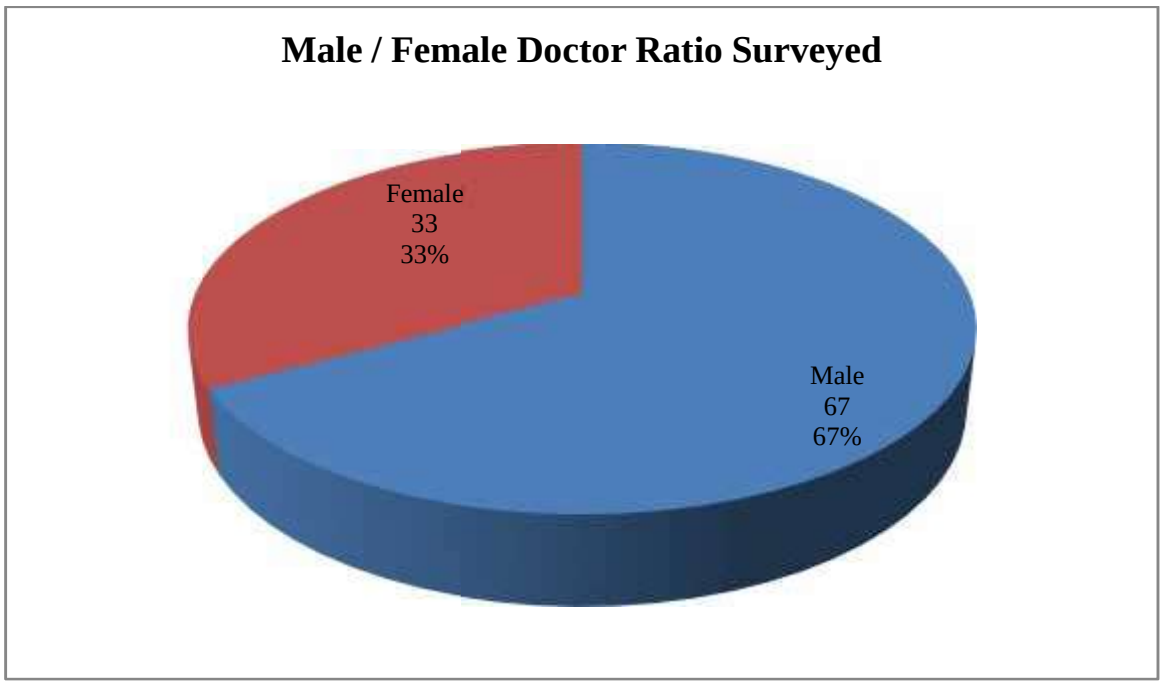


Figure 1: Pie Chart Representing Gender Distribution among Dentists

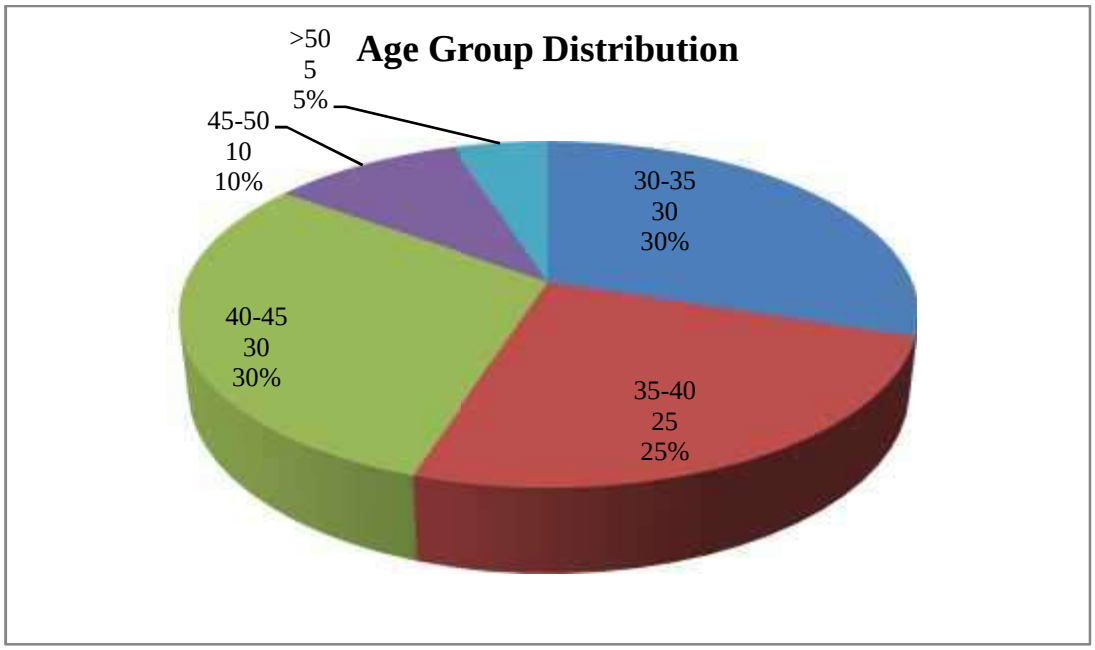


Figure 2: Pie Chart Representing Demographic Data among The Dentists

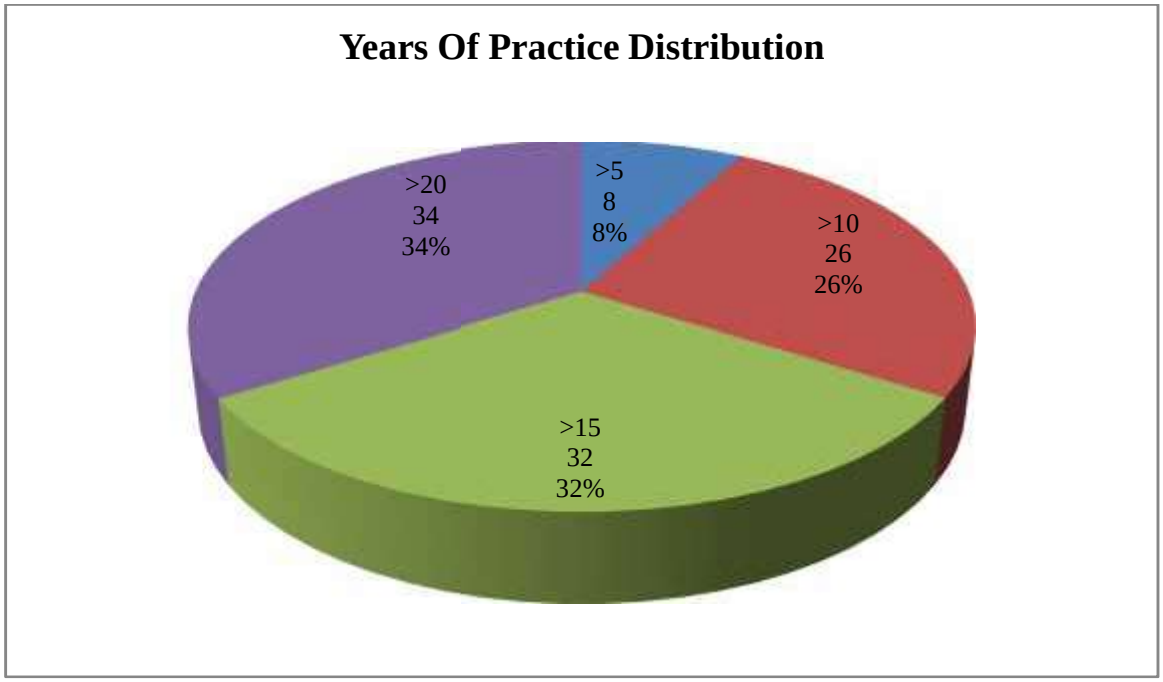


Figure 3: Pie Chart Showing the Number of Years in Practice by Several Dentists in South India

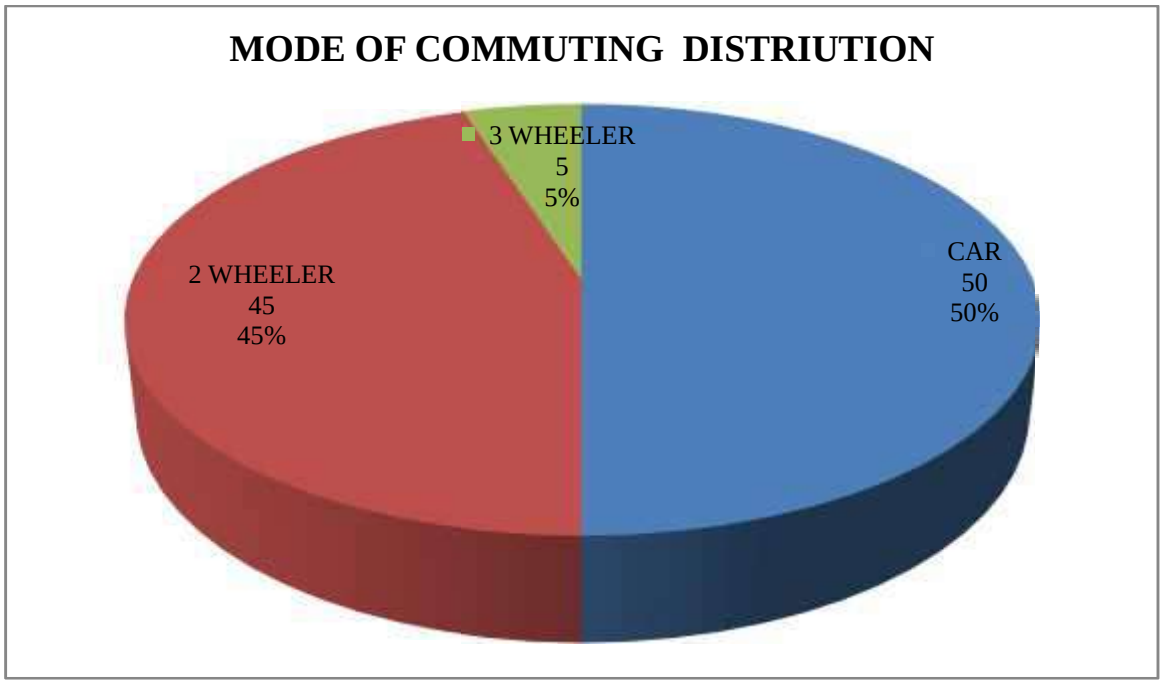


Figure 4: Pie Chart Representing the Mode of Transport for Commuting

:

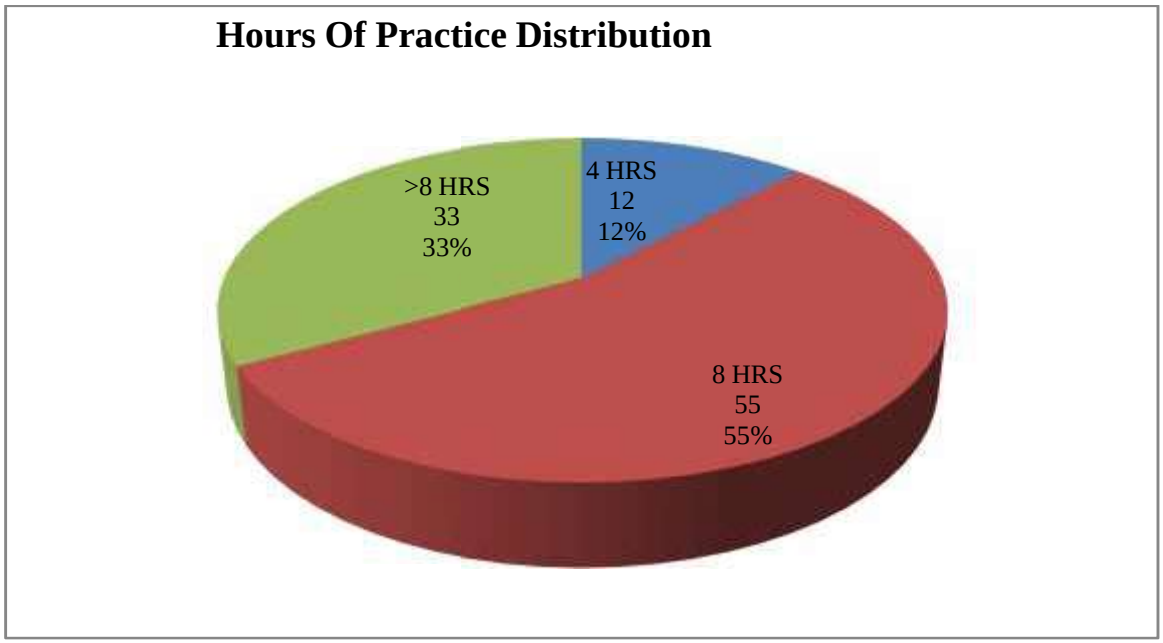


Figure 5: Pie Chart Showing the Number of Hours Practiced per Day

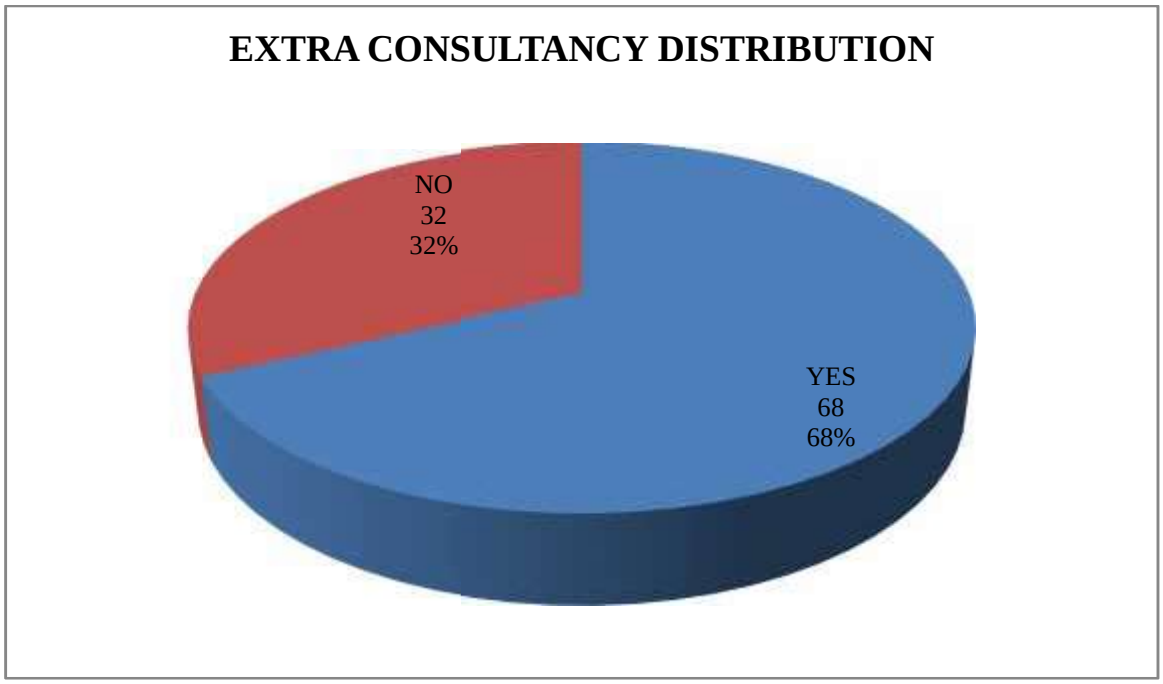


Figure 6: Pie Chart Showing the Number of Consultancy per Day

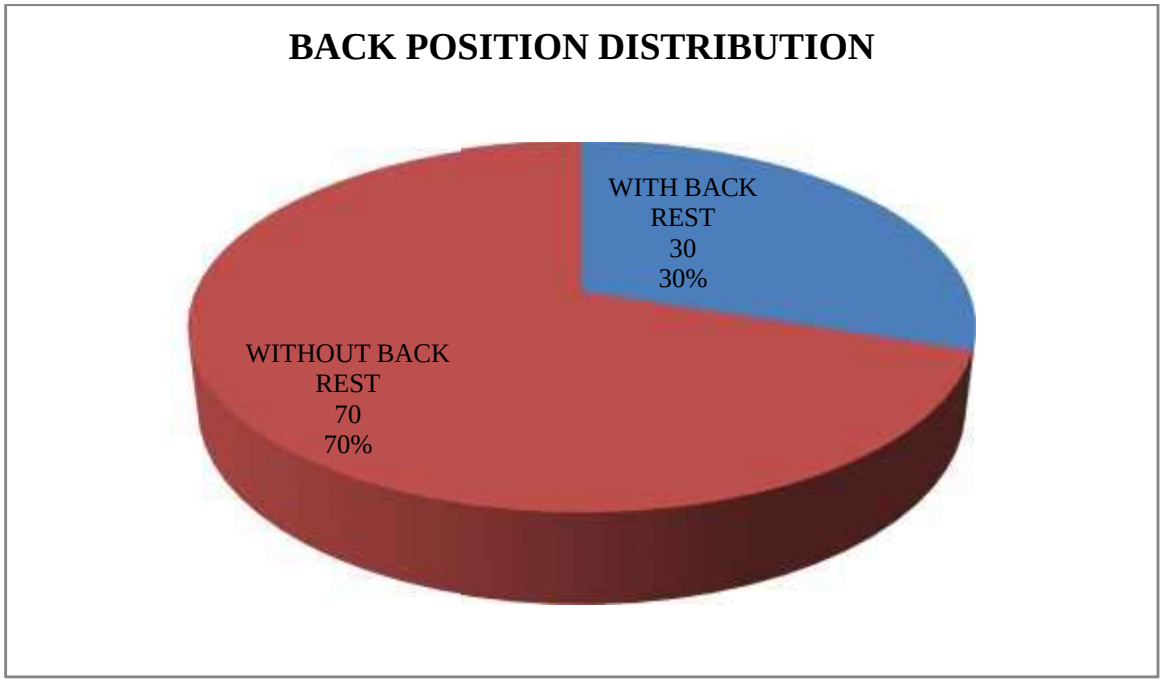


Figure 7: Pie Chart Showing the Back Rest Position Followed During Practice

:

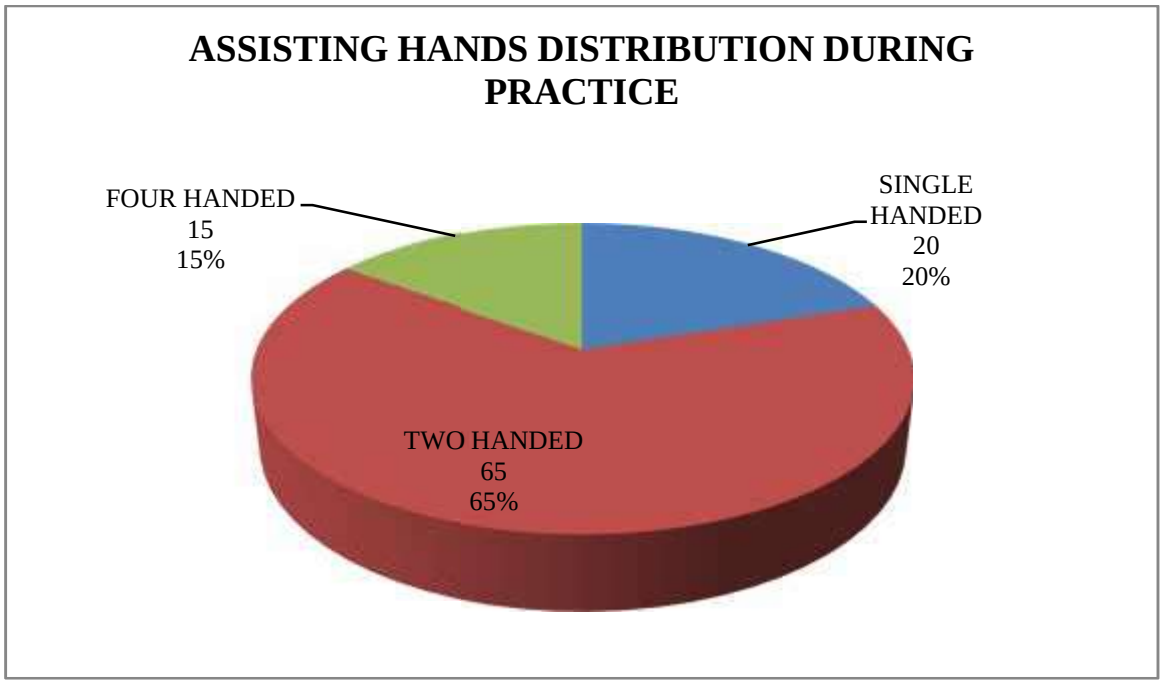


Figure 8: Pie Chart Representing the Mode of Assistance Sought During Practice

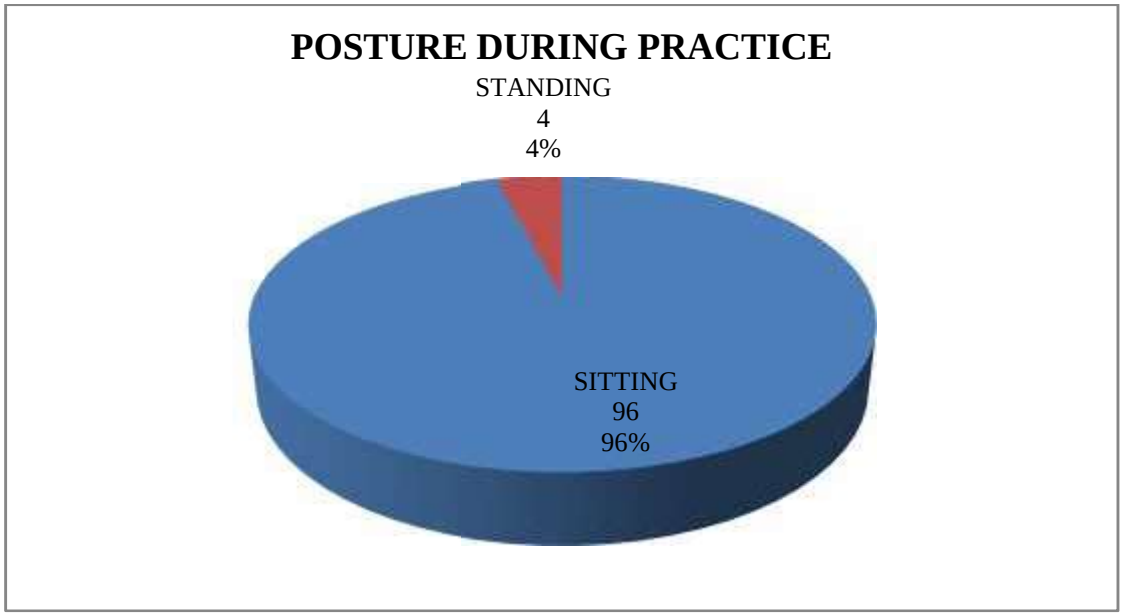


Figure 9: Pie Chart Showing the Dentists' Posture during Practice

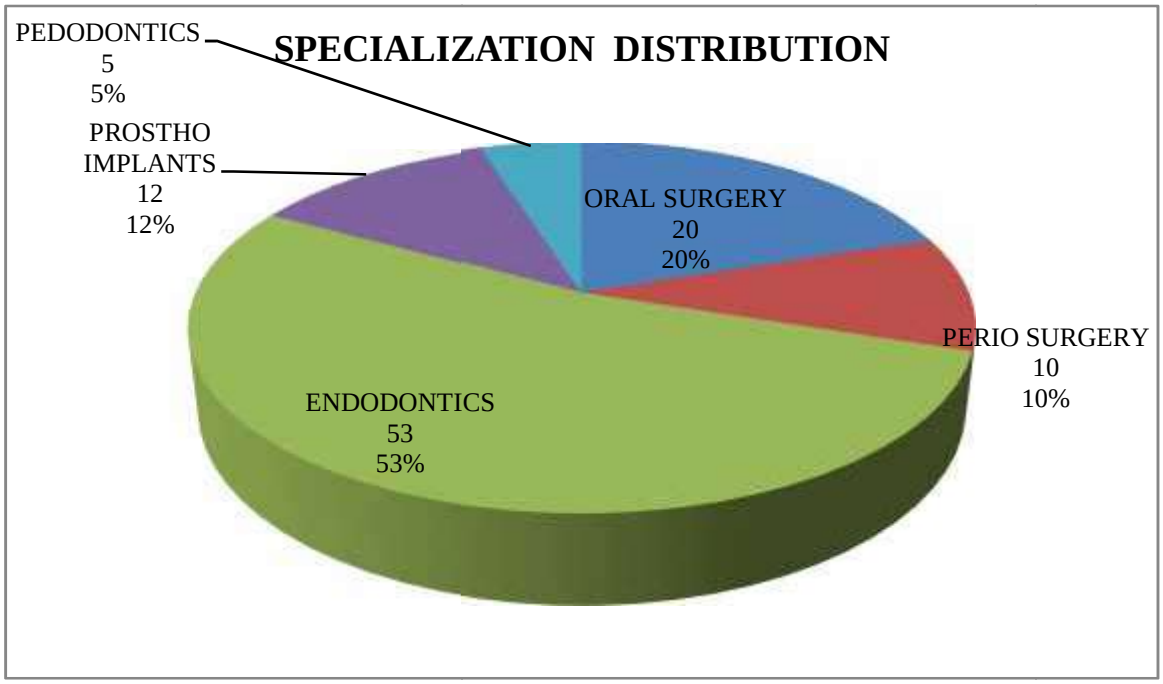


Figure 10: Pie Chart Showing the Specialization by Dentists In South India

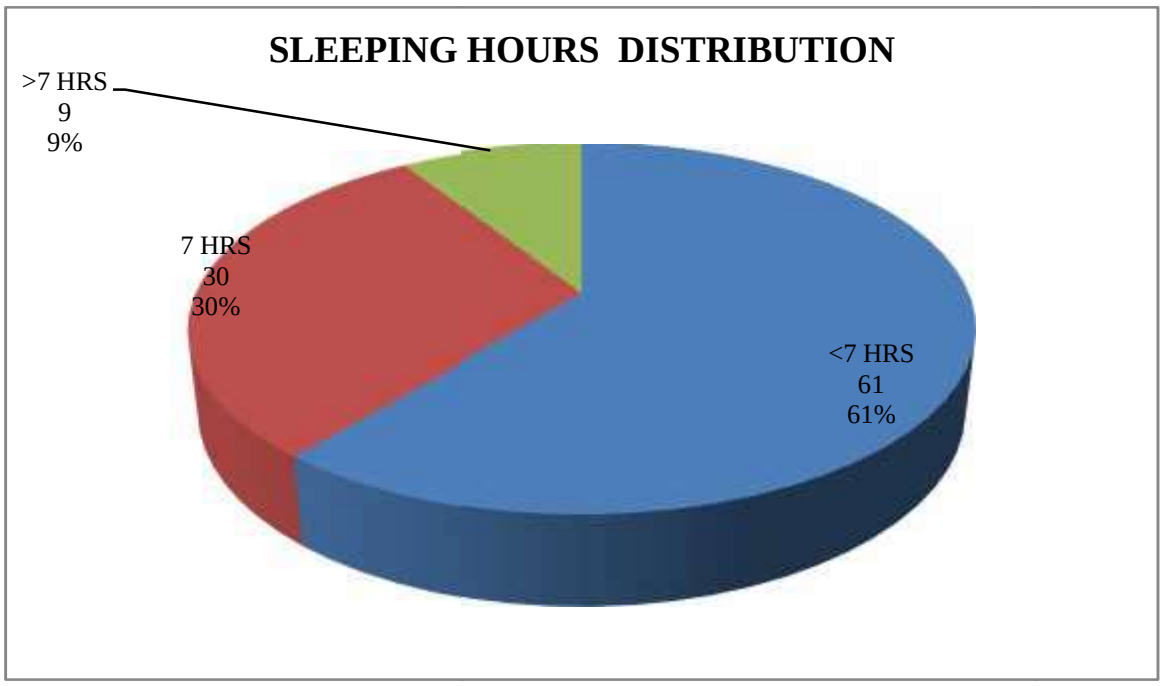


Figure 11: Pie Chart Showing Duration of Sleep

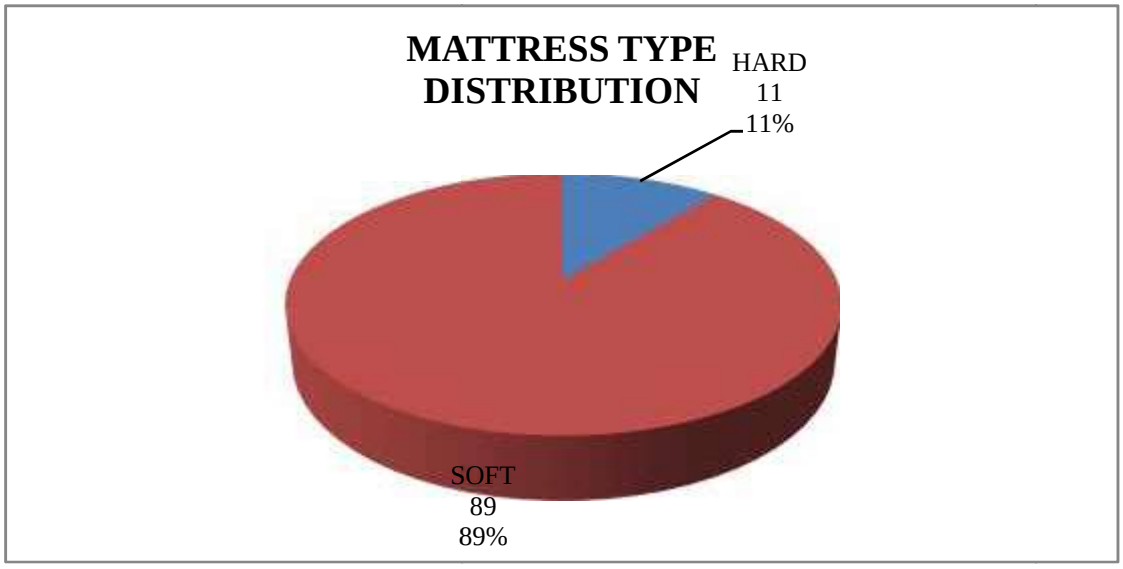


Figure 12: Pie Chart Showing the Type of Mattress Used For Sleep

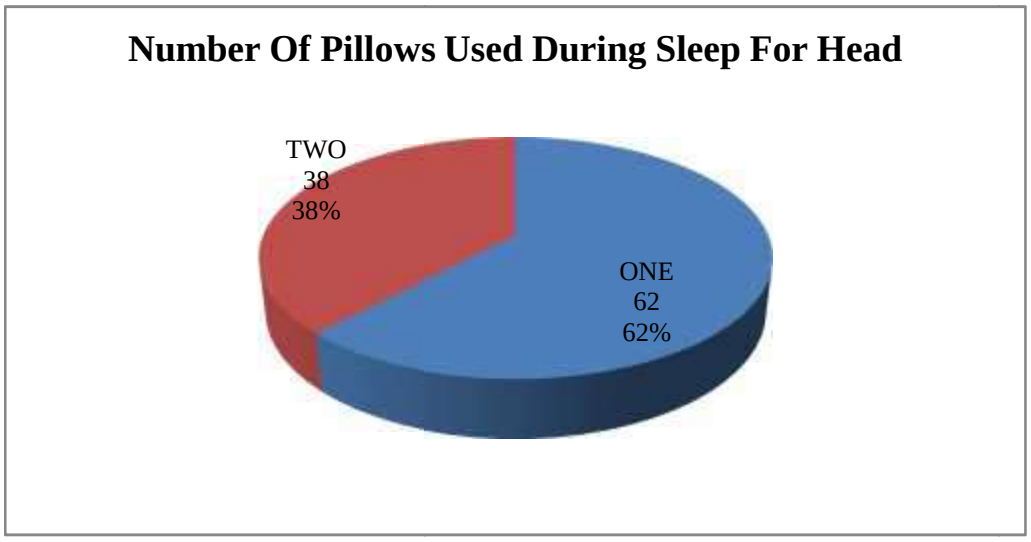


Figure 13: Pie Chart Showing the Number Of Pillows Used During Sleep

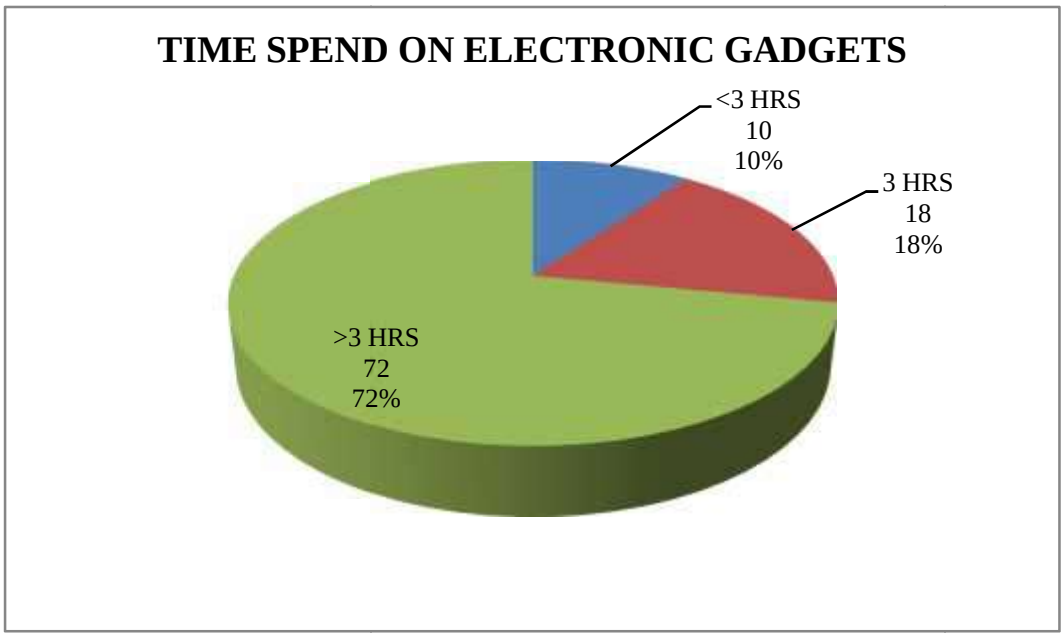


Figure 14: Pie Chart Representing the Time Spent On Electronic Gadgets per Day

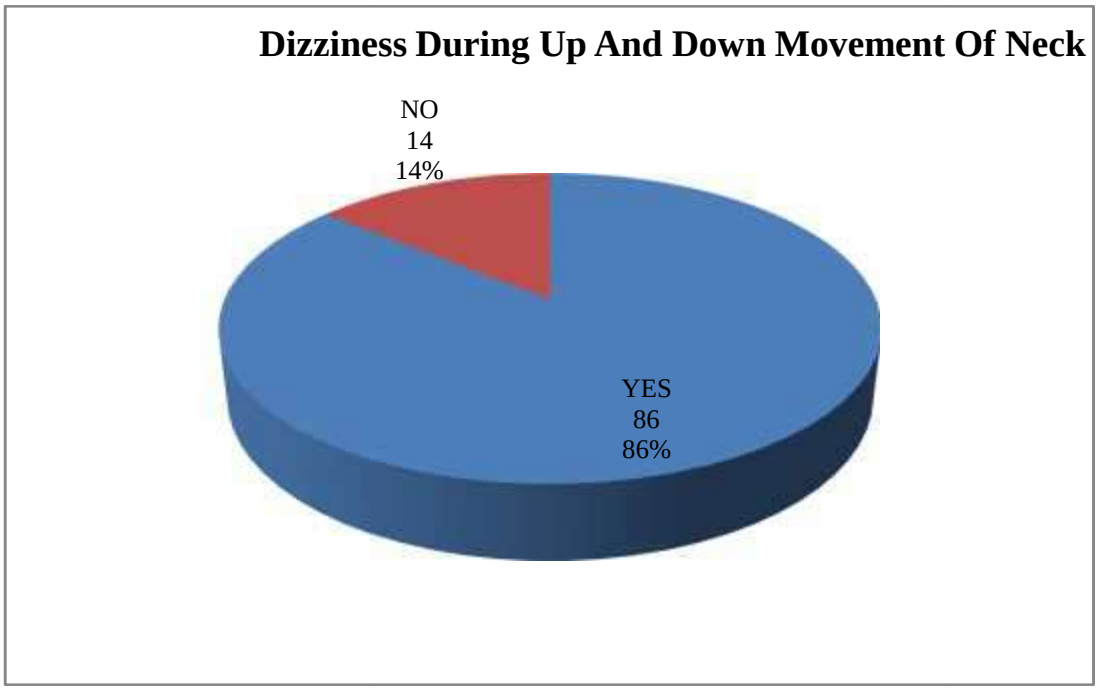


Figure 15: Pie Chart Showing Number Of Dentists Undergoing Dizziness During Movement of Neck

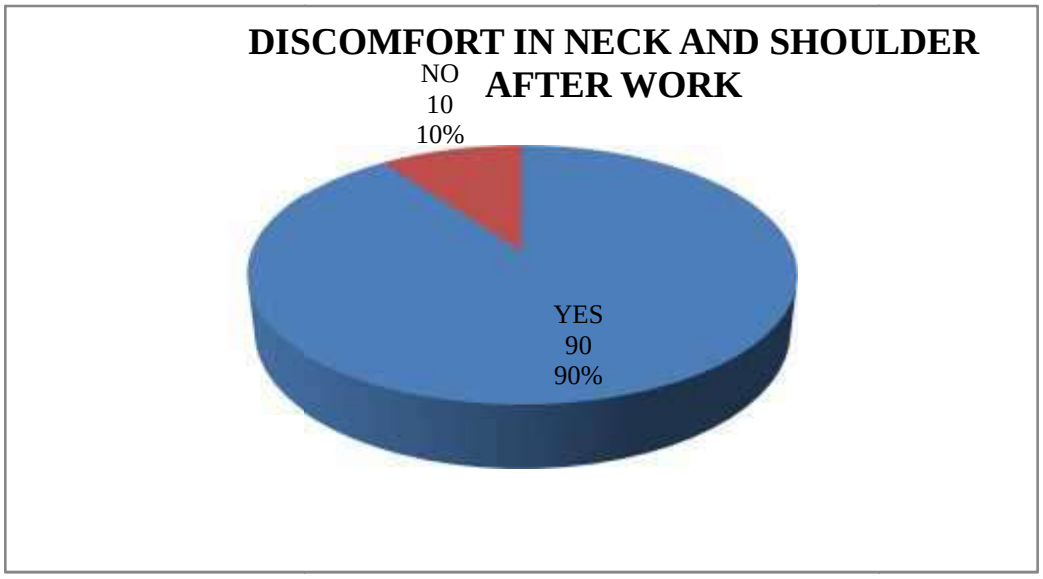


Figure 16: Pie Chart Representing The Discomfort Undergoing By Dentists In Their Neck And Shoulder After Work

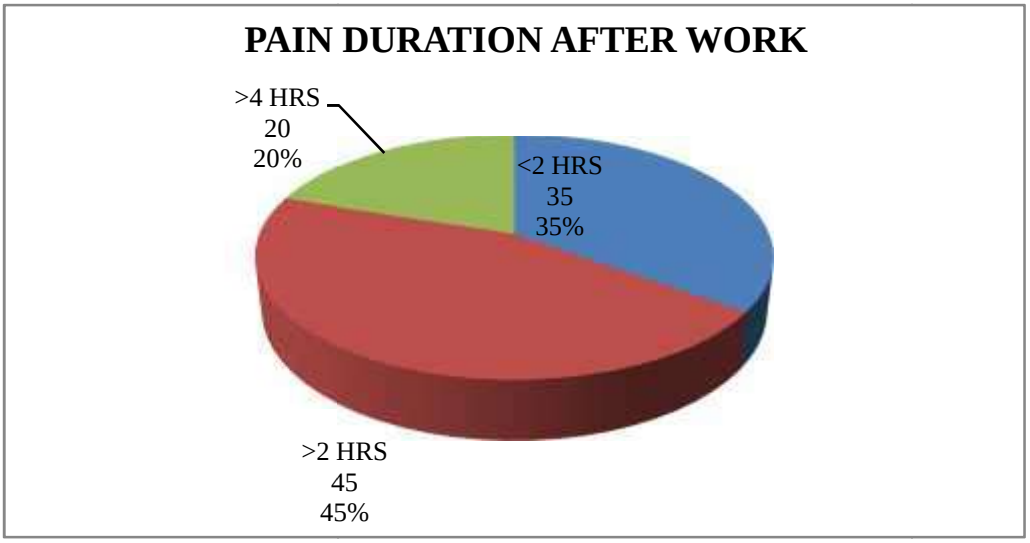


Figure 17: Pie Chart Showing the Pain Duration after Work

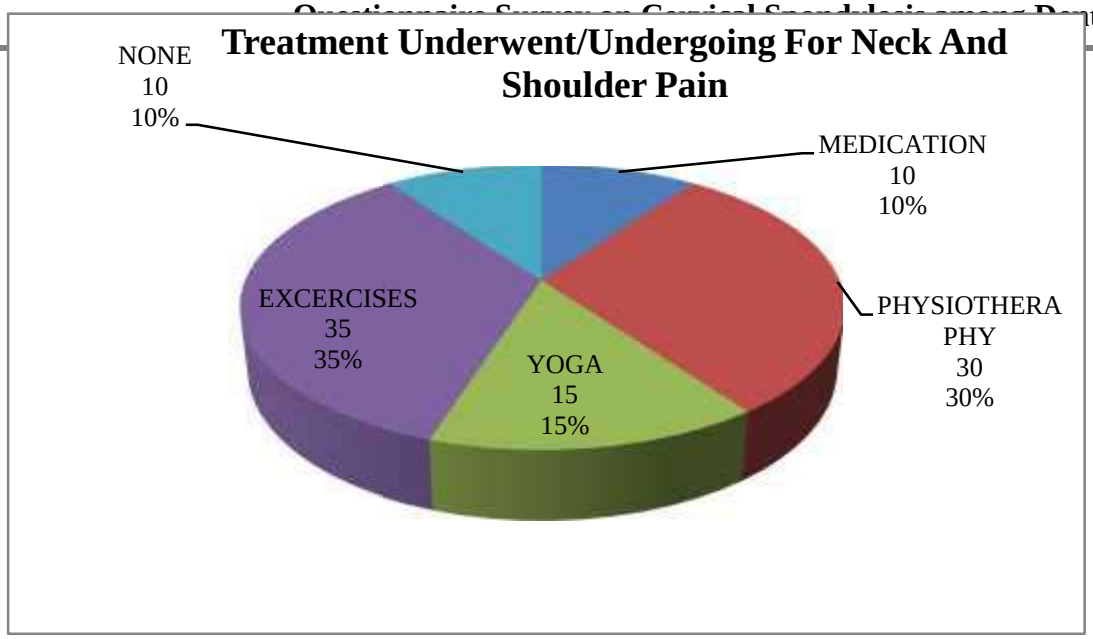


Figure 18: Pie Chart Representing Various Treatments Adopted for Neck and Shoulder Pain

Discussion

67% were males, 33% were females. 30% of people were from 30-35 age group and 40-45 age group each, 25%were from 35-40 age group, 10%were from 45-50 age group, and 5%were >50 age group. 34% are practising dentistry above 20 years, 32% above 15 years, 26% above 10 years, and 8% above 5 years. 50% of dentists use4 wheeler for travel, 45% of dentists use 2 wheeler to travel and 5% uses 3 wheeler for travel. Among these dentists, 55% of dentists works 8hrs, 33% of dentists work more than 8 hrs and 12% of dentists work 4 hrs a day.

World Health Organization defines musculoskeletal disorders (MSD) as problems of muscles, tendons, joints, inter-vertebral discs, peripheral nerves and vascular system. It is not directly a result of acute or prompt incidence but mounting slowly and frequently. During past many years it has been reported that MSD have increased in routine work. [21,22]. MSD are a frequent occurring throughout the globe and are the most common reason of chronic pain as well as disability effecting millions of people. Literature shows prevalence ratio of neck pain is high among dentists. World Health Organization describes work related musculoskeletal diseases being dependent on many factors including but not exclusive to structural, psychosocial and socio-cultural variables.

The second most common MSD in dentistry is neck pain. [23] The symptoms of MSD are categorized by occurrence of uneasiness, disablement and pain for a prolonged time period in the soft tissue structures. [24] Dentistry demands high accuracy and is frequently performed with the cervical spine being rotated and

flexed forward. This produces high static load in the neck region. Extended duration of static load and repetitive movements can result in neck pain, tension neck syndrome, muscle imbalance or cervical instability. [25]. As the oral cavity is narrow, dentists have a constrained visual field and restricted movement of neck and back leading to pain in these regions. [26]. The forward flexion of head and neck leads to cervical spine instability that causes straightening of its curvature. There is an increased risk of disc herniation and prolapse due to the lengthening and shortening of particular muscles, tendons and ligaments. Inflammation of neck muscles ensues due to overload and an unstable neck posture. [25].

As a result of tension neck syndrome (TNS), patients can have some symptoms like rigidity, pain and soreness in the region of trapezius. This is frequently associated with muscular spasm or tenderness or trigger points. It is not necessary that all symptoms must be localized in the region of neck but this can radiate into arms, skull and between shoulder blades. The most common symptoms of TNS is headache. The primary causative factor for TNS is poor posture with forward head position. The associated factors with neck pain include forward head posture or increased working hours. The symptoms of neck pain can be worse in professions where work demands extended head posture and utilization of muscles with reduced endurance that stabilize the neck [26]. Risk factors for this problem include high demands of job, poor job control, minimum social support and some personal characteristics [27]. Age related changes in vertebral column, its shape, weakness of muscles, poor practice posture / techniques of lifting and mechanical pressure are factors that contribute in neck and back pain [28]. Cervico-genic headache is a pain that refers from cervical spine to the head. Physiology of this pain is conjunction between trigeminal afferents and upper three cervical spinal nerves afferents[29].Trapezius pain is the classic stress pain and it is the most common musculoskeletal disorder [31].Neck pain is a common in the general population, with 70% of individuals affected some time in their lives [35]. Many researchers have proven scientifically and has also reported that protracted shoulder might lead to shoulder malposition, which may increase the subacromialimpingement [32]. Low Back Ache (LBA) is the second most common cause of disability [33].

The term work-related musculoskeletal disorders (MSDs) refers to the disorders to which the work environment contributes significantly to musculoskeletal disorders that are made worse or longer lasting by work conditions or workplace risk factors. Some of the examples of such workplace risk factors include jobs requiring repetitive, forceful or prolonged exertions of the hands, frequent or heavy lifting, pushing or pulling, or carrying of heavy objects and prolonged awkward postures. The level of risk depends on the intensity, frequency and duration of the exposure to these conditions. A WMSD can be defined as a condition wherein work-related tasks affect the nerves, tendons, muscles and supporting structures. Conditions can vary from mild recurrent symptoms to severe and incapacitating. Early symptoms of WMSDs include pain, swelling, tenderness, numbness, tingling sensation and loss of strength [30].Grip

strength is a force applied by the hand to pull on or suspend from objects and is a specific part of hand strength. It is the muscle power and force that can be generated by the hand.

Mechanical Neck pain may arise due to muscular tightness in both the neck and upper back. Exercise plus joint mobilization and manipulation has been found to be beneficial in both acute and chronic neck disorders. Neither mobilization nor manipulation without exercise however has been found to be helpful. Mobilization is equivalent to manipulation. Here two varieties of techniques are carried out in treating the neck pain. Recently evidence has begun to emerge for the use of manual therapy, specifically thrust manipulation procedures, directed at the thoracic spine in people with neck pain. Further decrease in the mobility of the thoracic spine has been shown to be related to the presence of neck pain symptoms.[34]

Coming to the consultation, 68% goes for consultation and the rest 32% do not. The most important part of back posture where 70% of the dentists do not use back rest whereas 30% uses back rest. Single handed dentistry is been practised by 25% of dentists, two handed dentistry is been practised by 65% of the dentists, and 10% of the dentists practice four handed dentistry. The position of practice is that 96% of the dentists practise sitting position and the rest 4% practise standing position. Among these dentists, dental surgeons were 20%, 10% periodontists,53% endodontists, 12% were prosthodontists and 5% were pedodontists. 61% of dentists sleep lesser than 7 hrs, 30%of dentists sleep 7 hrs, and the rest 9% sleeps greater than 7 hrs.

11% of dentists prefer hard mattresses and the rest 89% prefers soft type of mattresses. 62% of dentists use only one pillow during sleep and 38% of dentists uses 2 pillows for sleep. 72% of dentists uses electronic gadgets more than 3 hrs, 18% of dentists uses for 3 hrs and 10 of them uses lesser than 3 hrs. 86% of the dentists experiences dizziness during up and down movement of neck and 14% of dentists are comfortable. 90% of dentists face discomfort around neck and shoulder after work whereas the rest10% do not.

Of which 45% of dentists experiences pain >2 hrs, 20% of the dentists experiences pain >4 hrs, 35% of dentists experiences pain <2 hrs. For relief, 10% of dentists take medications, 30% of dentists undergo physiotherapy, 15% of dentists practise yoga, 35% of dentists exercise regularly, and 10% do not undergo any treatment.

Conclusion

Dentists are at the high risk of musculoskeletal symptoms in the neck. The reason for it is the position of work is difficult with cervical spine in flexion and rotation, repetitive procedure which demand accuracy; these problems can be overcome by some preventive measures like dentist’s chair is properly constructed

and the design of work unit should be appropriate, educate the dentist regarding ergonomics and should improve the work organization.[19]. Dentistry is physically and mentally a demanding profession. The physical characteristics include good psychomotor skills, hearing, visual quality, manual skill and ability to maintain good posture during work for an extended period of time. In case the dentist fails to adjust to a particular working environment, he / she can incur injury or disability. Hence dentists are at risk of work-related diseases / injuries e.g. allergies, systemic diseases, loss of hearing and musculoskeletal problems[20].

Corresponding Author

M.Sankari,

Professor, Department of Periodontics,

Saveetha Dental College, Saveetha Institute of Medical and Technical Sciences,Saveetha University, Chennai, India

162,Poonamallee High Road, Chennai 600077, Tamil Nadu, India

Telephone Number: 9176098012

Email Id: dr.sankarim@gmail.com

References

1. Binder AI. Neck pain syndromes. Clinical Evidence; December 2006; 15: 834-848.
2. Binder AI, Isenberg DA, Maddison PJ, Woo P, Glass DN, Breedveld FC; Cervical pain syndromes.Oxford textbook of rheumatology. 3rd ed. Oxford: Oxford Medical Publications, 2004:1185-95.
3. Vernon HT, Humphreys BK, Hagino CA.A systematic review of conservative treatments for acute neck pain not due to whiplash; J Manipulative Physiol Ther 2005;28:443-8.
4. Joshya A Cleland, paul Glynn, Julie M Whitman, Sarah L Eberhart et al; Short term effects of thoracic thrust manipulation and non thrust mobilization for the patient with neck pain;Volume 87, issue 5, Pg. No. 431-440.

5. FejerRNeck pain (Prevalence, genetic and environmental factors);University of Southern Denmark;2006; 29: 82-86.
6. Hogg-Johnson S, van der Velde G, Carroll LJ, Holm LW, Cassidy JD, et al; The burden and determinants of neck pain in the general population: results of the Bone and Joint Decade 2000-2010 Task Force on Neck Pain and Its Associated Disorder; 2008;Spine (Phila Pa 1976) 33: S39-51.
7. Jensen I, Harms-RingdahlKStrategies for prevention and management of musculoskeletal conditions. Neck pain; Best Pract Res Clin Rheumatol 2007; 21: 93-108.
8. Fejer R, Kyvik KO, Hartvigsen J; The prevalence of neck pain in the world population: a systematic critical review of the literature;European spine journal: official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society; 2006; 15: 834-848.
9. El KeshawiA;Neckpain and work-related factors among administrative and academic staff of the Islamic University of Gaza; The Islamic University-Gaza, Deanery of Higher Studies, Faculty Of Education 115; 2008; 18: 1532-1540.
10. Bernard M, Karnath M; Identifying the Musculoskeletal Causes of Neck Pain. The Journal of musculoskeletal medicine 2012; 29: 82-86.
11. Hush JM, Michaleff Z, Maher CG, Refshauge K; Individual, physical and psychological risk factors for neck pain in Australian office workers: a 1-year longitudinal study. European spine journal : official publication of the European Spine Society, the European Spinal Deformity Society, and the European Section of the Cervical Spine Research Society; 2009; 18: 1532-1540.
12. Yousef M, Al-ZainA;Posture Evaluation of Dental Students; 2009; 16: 51-68.
13. Morse T, Bruneau H, DussetschlegerJ;Musculoskeletal disorders of the neck and shoulder in the dental professions; 2010; 75(34); 419-429.
14. Al Wazzan KA, Almas K, Al Shethri SE, Al-QahtaniMQ;Back& neck problems among dentists and dental auxiliaries.JContemp Dent Pract 2001; 14(2): 17-30.
15. Külcü D, Güluen G, Altunok T, Küçükoulu D, NaderiS;Neck and Low Back Pain Among Dentistry Staff. Turk J Rheumatol; 2010; 25: 122-129.
16. Finsen L, Christensen H, Bakke M; Musculoskeletal disorders among dentists and variation in dental work; National Institute of Occupational Health, Department of Physiology LerseParkalle 105, Copenhagen DK-2100, Denmark; 1998; 29:119-125.
17. Harutunian K, Gargallo-Albiol J, Figueiredo R, Gay-EscodaC;Ergonomics and musculoskeletal pain among postgraduate students and faculty members of the School of Dentistry of the University of Barcelona; 2012; 29: 82-86.

18. Shrestha BP, Singh GK, NiraulaSR;Work related complaints among dentists.JNMA J Nepal Med Assoc;2008; 47: 77-81.
19. ArshiaIjaza ,IqraKhanb, Ashfaq Ahmedc , SamreenSadiqd; Frequency of neck pain among dentists; POJ 2016:8(2) 89-93.
20. Ayers K, Thomson W, Newton J, Morgaine K, Rich A;Self-reported occupational health of general dental practitioners.Occupational Medicine 2009;59(3):142-8.
21. Pargali N, Jowkar N; Prevalence of musculoskeletal pain among dentists in Shiraz, Southern Iran; The int j of occupational and environmental med. 2010;1(2)16-23.
22. Ahmed H, Batinic M; Prevalence and Risk Factors of Neck Pain Among Dentists and Dental Hygienists; 2014. M.S. Thesis. Department of Occupational and Environmental Medicine, Umeå University, Sweden.31pp:34-36.
23. Muralidharan D, Fareed N, Shanthi M; Musculoskeletal Disorders among Dental Practitioners: Does It Affect Practice? Epidemiology Research International 2013;Volume 13,issue 5; 1-6
24. Zaerian M; Neck Pain: A Scientific Look at the Dentist's Neck;Oral Health J 2011:53-6
25. Bansal A, Bansal P, Kaur S, Malik A; Prevalence of neck disability among dental professionals in North India;J of Evolution of Med and Dent Sci 2013;45(2);8682-8
26. Shetty S M, Shetty S, Hegde A, Babu N;Prevalence of neck and back pain among paediatric dentists;Nitte University. J of Health Sci 2015;5(1):45- 7
27. 27.Feng B, Liang Q, Wang Y, et al;Prevalence of workrelated musculoskeletal symptoms of the neck and upper extremity among dentists in China; BMJ Open 2014;4:e006451. doi:10.1136/bmjopen-2014- 006451
28. 28.Hayes M, Cockrell D, Smith D;A systematic review of musculoskeletal disorders among dental professionals; International j of Dent Hyg 2009;7(3):159-65
29. 29.Rao R; Neck pain, cervical radiculopathy and cervical myelopathy; The J of Bone & Joint Surg 2002;84(10):1872-81.
30. Shibu Thomas Mathew;Posturedontics in dentistry: A review; J. Dent. Oral Hyg.; Jun 2015; Vol. 7(6), pp. 78-85.
31. A.kumaresan, G.Deepthi, VaiyapuriAnandh, S.Prathap;Effectiveness Of Positional Release Therapy In Treatment Of Trapezitis; International journal of pharmacutical science and health care; 2012; 1(2);p:71-81.
32. K Kotteeswaran, K Rekha, VaiyapuriAnandh; Effect of Stretching and Strengthening Shoulder Muscles in Protracted Shoulder in Healthy Individuals;International journal of computer application 2 (2), 111-118.

33. K Kotteeswaran, JosyulaSnigdha, JagatheesanAlagesan; Effect of proprioceptive neuromuscular facilitation stretching and dynamic soft tissue mobilization on hamstring flexibility in subjects with low back ache - single blinded randomised controlled study; Int J Pharm Bio Sci 2014 July; 5(3): (B) 228 – 233.
34. R Raja , K Kotteeswaran , V Anandh; The Effects of Thoracic Thrust Manipulation and Neck Flexibility Exercises for the Management of the Patients with Mechanical Neck Pain; Indian Journal of Physiotherapy and Occupational Therapy. October-December 2015, Vol. 9, No. 4;p:168-172.
35. Srilatha K and Kotteeswaran K; Effects of cervical muscle endurance training on cervical flexor and extensors in mechanical neck pain;IJBPAS, September, 2013, 2(9): 1755-1761.