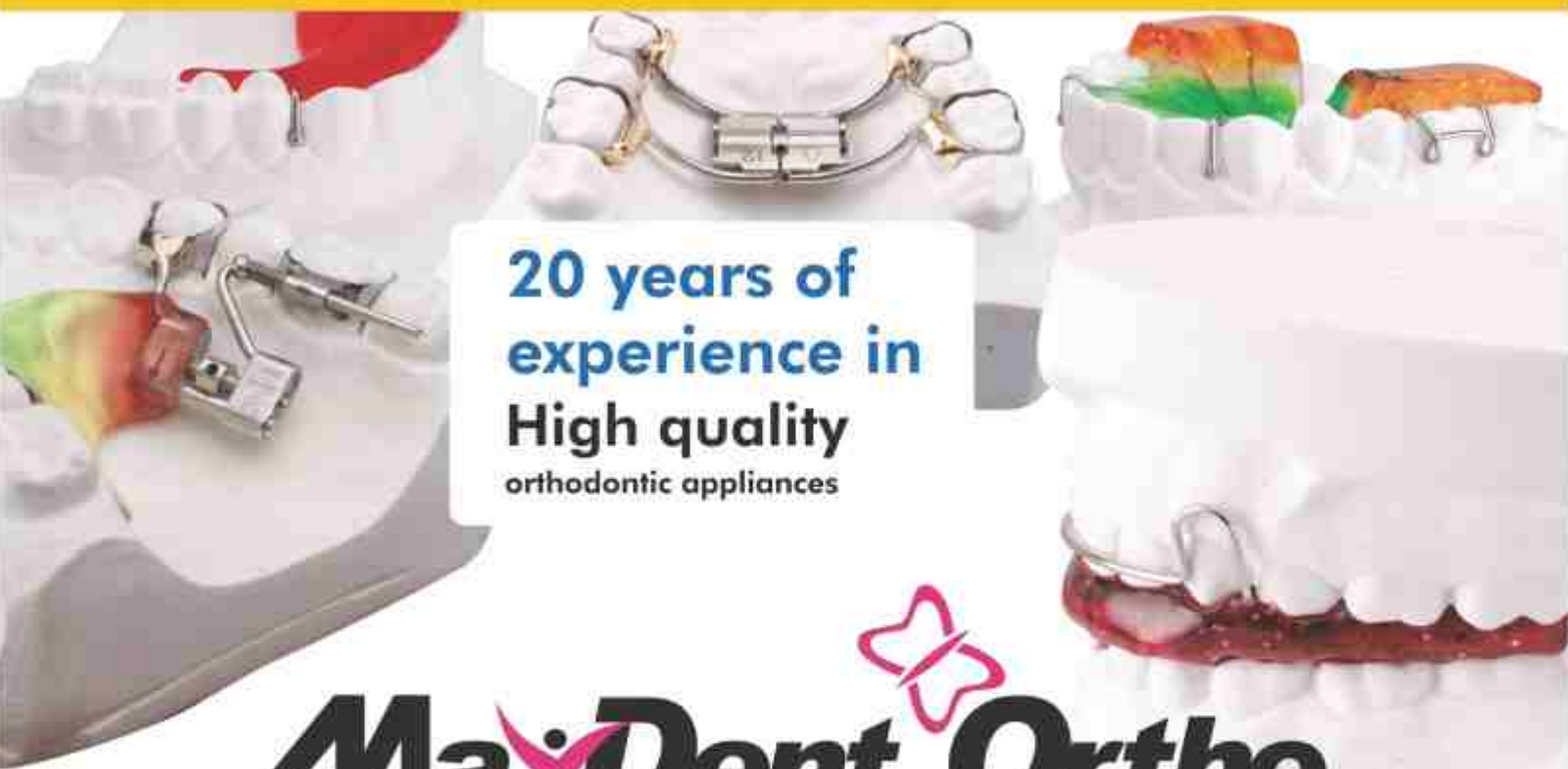
Jan. 2013 Volume I Issue I

Journal of Odontological Research

Official Publication of
Indira Gandhi Institute of Dental Sciences
Nellikuzhy, Kothamangalam 686 691, Kerala, India

Makes Orhtodontics Easy

CLEAR-ALIGNER®



20 years of experience in
High quality
orthodontic appliances

MaxDent Ortho

A Pedo/Orthodontic lab created with Ideas!

www.clearaligner-max.com www.maxdentortho.com

Journal of Odontological Research

Official Publication of
Indira Gandhi Institute of Dental Sciences
Nellikuzhy, Kothamangalam 686 691, Kerala

Journal of Odontological Research is the official publication of the Indira Gandhi Institute of Dental Sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala. It is a peer-reviewed journal published bi-annually. The journal will cover studies related to dentistry and applied basic subjects. The articles will be published under the categories of Original Research, Review, Case Reports and Guest Column. The manuscripts for publication may be sent to the journal's e-mail : jorigids@gmail.com

EXPERT PANEL OF CONSULTANTS

Dr. George Varghese
Principal
Government Dental College
Kottayam, Kerala

Dr. Chandu G. N.
Professor
Department of Preventive and Community
Dentistry College of Dental Sciences
Davangere, Karnataka

Dr. Umashankar K.
Professor
Department of Orthodontics Saveetha Dental
College and Hospital, Chennai,
Tamil Nadu

Dr. Pradeep Kumar
Professor and Head
Department of Prosthodontics
KMCT Dental College
Mukkom, Kozhikode, Kerala

Dr. B. R. R. Varma
Consultant Periodontist
Dr. Varma's Centre for Advanced Dental Care,
Cochin, Kerala

Dr. B. Shivapathasundaram
Professor and Head
Department of Oral Pathology, Meenakshi
Ammal Dental College Chennai, Tamil Nadu

Dr. Srilal
Professor
Department of Prosthodontics
Sri Mookambika Institute of Dental Sciences,
Kulasekharam, Tamil Nadu

Dr. Rezy Cheru T.
'Shalom', TC 12/639
Champion Bhasker Road,
Kunnukuzhy,
Trivandrum, Kerala

Dr. Prashant G. M.
Reader
Department of Preventive and Community
Dentistry College of Dental Sciences
Davangere, Karnataka

Dr. D. S. Mehta
Professor and Head Department of
Periodontics, Bapuji Dental College and Hospital,
Davangere, Karnataka

Dr. R. Rajendran,
Professor of Oral Pathology
College of Dentistry
King Saud University
Kingdom of Saudi Arabia

Dr. Shashikanth Hegde
Professor and Head
Department of Periodontics,
Yenepoya Dental College
Mangalore, Karnataka

Dr. Vijayalakshmi Acharya
Acharya Dental
Nungambakkam
Chennai, Tamil Nadu

Dr. U. S. Krishna Nayak
Professor and Head, Department of Orthodontics
A. B. Shetty Memorial Institute of Dental Sciences
Mangalore, Karnataka

Dr. V. Gopikrishna
Professor
Department of Conservative Dentistry and
Endodontics
Thai Moogambika Dental College, Chennai

Dr. K. Ranganathan
Professor and Head
Department of Oral Pathology,
Ragas Dental College and Hospital,
Chennai, Tamil Nadu

Dr. Sakeenabi B.
Reader
Department of Preventive and
Community Dentistry, College of
Dental Sciences Davangere, Karnataka

TABLE OF CONTENTS

editorial

It is indeed a matter of pride for all faculty members of Indira Gandhi Institute of Dental Sciences, Nellikuzhy, Kothamangalam, Kerala, that the first issue of our institution's scientific journal by name 'Journal of Odontological Research' is being released in the month of February 2013. Unrevealed knowledge is same as good textbooks kept idle at the corner of bookshelf. Scientific thoughts, ideas and expertise remain invalid unless they are conveyed, discussed and disclosed to the fellow colleagues and community.

I am delighted to share with you the joy of successfully bringing out this journal, which was the dream project of the Academic Club of the institution.

I would like to take this opportunity to thank all our dynamic faculty members for their untiring efforts to make this dream a reality. I wish all success for this noble endeavor and also hope this become a highly informative and interesting read for the professionals and dental students.

Dr. Ambika K.
Chief Editor

Indira Gandhi
INSTITUTE OF DENTAL SCIENCES

Nellikuzhy, Kothamangalam, Ph: 0485 6451630, 31, 3390000 (1000 Lines),
Fax: 2824942, Web: www.igids.org, E-mail: dental@igmt.org

GUEST COLUMN

VIRTUAL MICROSCOPY - TECHNOLOGY OF THE NEW MILLENIUM

Author:
Dr. Mithilesh Chandra, M.D., FIC Path
Consultant Histopathologist

Address for correspondence:
Email: docmchandra@yahoo.com.

INTRODUCTION

Health care in India is making rapid strides in every field. Last two decades have witnessed rapid growth and digitalization of the health care. Many aspects of patient care and record keeping are now becoming Information and Communication Technology (ICT) based. This change has resulted in world class medical care in India leading to booming medical tourism.

However digitalization not yet become part of medical and dental education. Hence there is a mismatch in the training of medical graduates and postgraduates in digital world and the requirement and expectations from them. Young dental doctors trained in India are at a disadvantage when they go for further training to other developed countries. There is an urgent need to focus on this issue and take remedial measures in this field. Similarly digitalization in diagnostic services especially in the field of telepathology has not kept pace with the developments taking place in this field particularly virtual microscopy.

VIRTUAL MICROSCOPY-TECHNOLOGICAL INNOVATION

Last decade has seen attempts at improving the methods employed in telepathology. This had led to the development of virtual microscopy which produces virtual images and these images are gradually replacing the static images as preferred toll of telepathology. Virtual images are playing greater role in e-medical and e-dental education than what initially was envisaged.

Virtual microscopy is the process of producing virtual images with the help of an advanced microscopy called virtual microscope. Virtual microscope is a trinocular microscope with robotic control of various adjustments like brightness of illumination, movement of mechanical stage in X and Y axis, change of objective, coarse and fine focusing of the section. Slide kept on the stage is scanned in the magnification selected and hundreds of pictures are taken of the section by automatic movement of the equipment which therefore is also called Digital scanner. Each field of section is autofocussed before capture of image. On compilation of scanning, the picture captured in millions of pixels is stitched and blended together with the help of software to finally produce a composite picture which is exact replica of the tissue section. The image thus created is automatically stored in the computer.

This image is called Digital slide or whole slide image, virtual Image and has the characteristics of the original section. If the staining quality of the section was not optimal, quality of the image can be improved in terms of colour saturation, brightness and contrast with the help of software. Digital slide can be viewed on the screen of the PC or laptop in any magnification just like a glass slide is viewed under the microscope and any area of the slide can be viewed. Image can be annotated to point out salient features which are very useful for undergraduate teaching.

APPLICATIONS OF VIRTUAL MICROSCOPY

1. Telepathology

The main application of virtual microscopy envisaged initially lies in telepathology for primary consultations and second opinion from expert consultants. Slides from a smaller centre equipped with the virtual microscope are converted into digital slides and these are transmitted to a centre of excellence to consultant pathologist who views the slide in digital form and sends opinion to the referring laboratory. Technology has tremendous potential for India which has a pool of excellent dental institutions at par in excellence to any other institution in the world and specialized centres for various diseases. These centres can become digital pathology hub in future just like teleradiology and medical tourism in India. Telepathology can be done using static images or the whole slide images. The limitation of telepathology using static images has limitations as the area selected for consultation is dependent upon the referring oral pathologist while whole slide images have no such bias. Telepathology allows consultation and interactive case discussion, implementation of uniform protocols extremely helpful in standardization of report and medical terminologies and creation of pool of teaching material for mutual use.

2. Medical Education

Digital whole slide images are going to bring revolution in medical education and usher in era of e-medical education. Futuristic class rooms will be devoid of microscopes and will be equipped with PCs of laptops. Professors will be showing whole slide images instead of showing microscopy glass slides and students will be viewing the images on their laptops. This technology will be highly effective for self learning.

The whole slide images will become instrument of bringing uniformity of teaching in various institutions as images will be duplicated and distributed in the collages across the country. Next few years are going to witness improvement in the quality of dental education and bring it at par with global centres of excellence.

3. Storage and cataloguing of teaching material

Digital slides will completely revolutionize

storage of slides in the departments of oral pathology. Instead of glass slides, cases will be stored in the computer disks. Compilation of cases will be computerized and retrieval will be easy. Physical space will not be required for storage of slides.

It remains to be seen how soon the resourceful institutions in the country take initiative in promoting technology adaptation and diffusion among the oral pathology and dental anatomy community to explore the full potential of this innovative technology. In the interim period private players concerned with medical education are initiating the process.

REFERENCES

1. Samraj T. Virtual microscopic method of teaching histology. JOHS 2011;2(1):43-4.
2. Betigeri AM, Aparna P, Pasupathi P: Could India become the digital pathology hub of the future? A consideration of the prospects telepathology outsourcing. Int J Biol Med Res. 2010;1(4):300-2.
3. Baruah MK. The practice of telepathology in India. J Posgrad Med. 2005;4:316-8.
4. Weinstein RS, Graham AR, Richter LC, Barker GP, Krupinski EA, Lopez AM, Erps KA, Bhattachryya AK, Yagi Y, Gilbertson JR. Overview of telepathology, virtual microscopy, and whole slide imaging: prospects for future. Hum Pathol. 2009;40:1057-69.
5. Dee FR. Virtual microscopy in pathology education. Hum Pathol. 2009;40:1112-21.

ORIGINAL RESEARCH ARTICLE

ANTIMICROBIAL MOUTH RINSES - ARE THEY POTENT AGAINST CANDIDA ALBICANS?

Authors:
Mithun Pai BH[1],
Mahesh Kumar M. [2],
Amit V. Mahuli[3],
Prashanth GM[4],
Chandu GN[5].

[1]Assistant Professor, Dept. of Public Health Dentistry, Manipal College of Dental Sciences, Mangalore 575 001, Karnataka, India.

[2]Assistant Professor, Dept. of Public Health Dentistry, CSI College of Dental Sciences and Research, East Veli Street, Madurai 625 001, Tamil Nadu, India.

[3]Senior Lecturer, Dept. of Public Health Dentistry, Dr. DY Patil Dental College and Hospital, Pimpri, Pune, India.

[4]Reader, Department of Preventive and Community Dentistry, College of Dental Sciences, Davangere. 577 004, Karnataka, India.

[5]Professor, Department of Preventive and Community Dentistry, College of Dental Sciences, Davangere. 577 004, Karnataka, India.

Address for correspondence:
Dr. Mithun Pai BH, Assistant Professor, Dept. of Public Health Dentistry, Manipal College of Dental Sciences, Mangalore, Karnataka 575 001.
E mail dr6446@yahoo.co.in

ABSTRACT

Background: There has been an increase in *Candida* infections over the period of several decades. This is probably because of the increasing number of elderly and seriously ill patients, immunosuppressive therapies, as well as the increased use of antibiotics and more invasive therapeutic medical procedures. The study was conducted with an objective of determining the antifungal efficacy of five mouth rinses on *Candida albicans* in vitro.

Methods: The study is an experimental in-vitro study conducted to evaluate antifungal efficacy of five mouthrinses against *Candida albicans.* A three pronged objective included screening the mouth washes for antifungal properties, to check the minimum inhibitory concentration (MIC) and to evaluate the fungicidal efficacy of the mouth rinses on *Candida albicans,* using disc diffusion, broth macro dilution and modified Bernstein method was done respectively. The mean and standard deviation of the diameter of inhibition zone was calculated. The data analyzed using one way Analysis of Variance (ANOVA) followed by Tukey post hoc test for pair wise comparison.

Results: The study showed that all the mouthrinses showed zones of inhibition against *Candida albicans.* Zones of inhibition for Chlorhexidine were highest and were significantly greater. All the mouth washes used in the present study showed kill times that were lesser than thirty seconds.

Conclusion: The findings of the study demonstrate that mouthrinses have sufficient antifungal properties that might represent appropriate alternative to conventional antifungal drugs in the management of *Candida albicans*.

Key words: antimicrobial Mouth rinse, *Candida albicans*, antifungal activity.

J Odontol Res 2013;1(1):4-10

INTRODUCTION

Oral Candidiasis is a relatively common opportunistic mycotic infection in man. It is usually caused by *Candida albicans,* which is predominant over other yeasts in the oral cavity. Although oral carriage of this opportunistic pathogen varies greatly, it is often present in the oral microflora of healthy individuals. A characteristic feature of candidiasis is its development when the host provides the environmental conditions and nutrients essential for attachment, growth and reproduction. [1]

In comparison to bacteria pathogenic fungi have received little attention as biocide targets. Biocides, better known as antiseptics, disinfectants, or preservatives, are commonly added to mouthwashes, toothpastes, hand soaps, and related consumer products.

C. albicans is commonly found at low levels among the normal oral flora, but its overgrowth in immuno compromised individuals or following broad-spectrum antibiotic therapy leads to oropharyngeal Candidiasis.[2] As oral microbiota not only consist bacteria but also contain other micro organisms which cause mixed infection in the oral cavity a broader spectrum of activity of oral care products are needed, the establishment and maintenance of oral microbiota is related not only to interbacterial co aggregations but also to interactions of these bacteria with yeasts such as *C. albicans*. Fungi are frequently isolated in several oral sites, including the tongue, buccal mucosa, palate, dental biofilm, sub gingival microbiota, carious lesions and prosthetic appliances. Studies have suggested a possible relation between *C. albicans* and periodontal disease, dentin and/or root caries. *C. albicans* has similar capacity of colonizing hydroxyapatite as that of *Streptococcus mutans*, however using different mechanisms. Enamel and dentin demineralization produced by fungal organic acids, as well as the presence of cells with *C. albicans* hyphae invading dentinal tubules, prove this ability of fungi to invade and destroy organic and inorganic dental tissues. This microorganism adheres to hydroxyapatite, especially through electrostatic interactions and at smaller numbers. *Candida albicans* has also the ability to dissolve hydroxyapatite at a larger rate when compared to *S. mutans* .[3]

Despite the availability of a number of effective antimycotic for the treatment of oral candidiasis, failure of therapy is not uncommon owing to the unique environment of the oral cavity where the flushing effect of saliva and the cleansing action of the oral musculature tend to reduce the drug concentration to sub-therapeutic levels. Since mouthwashes have already proved their efficacy against wide range of bacteria their anti fungal efficacy have yet to be substantiated. The study was conducted with a three pronged objectives of screening the antifungal efficacy of five mouth washes on *Candida albicans.* To check The Minimum Inhibitory Concentration (MIC) of five mouthrinses on *Candida albicans and* check the fungicidal efficacy of five mouth washes on *Candida albicans in an in vitro medium.*

MATERIALS AND METHODS

The materials and armamentarium used in the study constituted of the following:

The Test mouth rinses that were used in this study are Cetylpyridinium chloride mouth wash Crest Pro Health, Procter And Gamble (Cetylpyridinium chloride 0.06 %). Hexidine ICPA Health Product (Chlorhexidine Gluconate 0.2%). Listerine mouthwash Johnson and Johnson, (Thymol 0.6%, eucalyptol 0.09 %, Menthol 0.4%, Ethanol 26%). Betadine,Win Medicare Pvt Limited, (Povidone iodine, Absolute Alcohol 38%). Mougel ayurvedic mouth wash Immis Pharmaceuticals, (Embilica Officinalis, Terminalia chebula, Terminalia Belerica, Acacia Catechu, Borax) were used to determine antifungal activity on Candida albicans.

Filter discs uniformly loaded with 30µl of mouthwash were placed with sterile tweezers onto the prepared plates. Two filter discs were placed in each Sabouraud agar plates. Based on expected difference from pilot study, 5% level of significance (α), and 80% power of study (1-β), for each mouth rinse and control (sterile deionised distilled water). The zone of microbial inhibition was measured after the time intervals of 18 hours, 24 hours, and 48 hours on the underside of the Petri dishes using vernier

callipers. The zone of inhibition on the growth of test strains were defined by the area where visible growth had been inhibited if that is obvious; if it was not then, measurement was made to the point of 80% inhibition of growth. First the whole of the diameter of the zone of inhibition was measured and later the diameter of filter disc was deducted from it. This revealed the actual zone of inhibition around the filter disc.

All the measurements of zone of inhibition were carried out by a single examiner. Calibration of examiner was done prior to and during the study by re-examining 5% of the samples, to minimize intra examiner variability. Intra examiner agreement was determined using kappa statistics (k). Intra examiner agreement score ($\kappa = 0.94$) was almost perfect, according to Landis and Koch, thus meeting the scientific requirement for validity and reliability.[4]

Determination of minimum inhibitory concentration (MIC):

The first dilution (the highest concentration) was made by adding equal volume of stock solution to the broth in the first tube. The further dilutions were made by adding 1.5 ml of previous dilution to the next tube containing 1.5 ml broth where the v/v ratio was 1: 2, 1:4, 1:8, 1:16, 1:32, to 1:64 respectively. The final volume of broth and mouth rinse in each tube should be 1.5 ml, so the excessive amount of mouth rinse in the last test tube was discarded.[5]

Mouth rinses were thus diluted in series of exponential dilutions from undiluted to1:64 dilution using broth dilution technique. Equal volumes (1.5 ml) of fungal suspensions and mouth rinses are mixed and kept at 37^0C for 18hours. Optical density at 560nm was determined using spectro photometer. Duplicates were examined and averages of two test tubes were taken. The optical density was determined relative to fungal growth. Controls consisted equal volume of fungal suspension and distilled water. Blanks consisted of Sabouraud broth and mouth rinse results were expressed as percentage.

$$= \frac{(\text{OD CONTROL} - \text{OD BLANK}) - (\text{OD SAMPLE} - \text{OD BLANK})}{(\text{OD CONTROL} - \text{OD BLANK})}$$

Test for fungicidal activity:

The fungicidal activity was determined by modified Bernstein method, wherein, 0.5 ml of mouth rinse and fungal suspension are measured with a micropipette and kept in plastic vials at room temperature, the mixture was centrifuged after a time periods of 30 seconds, five minutes and 15 minutes in a Remi R-83 centrifuge at 1000 RPM for three minutes. Mixture was inoculated on Sabouraud s agar medium to form a lawn culture, and incubated at 37 °C for 48 hours. Average of two test tubes were taken as mean. Controls consisted of equal volumes of fungal solutions in Phosphate Buffered Saline and number of colony forming units were determined from each sample and control, and was expressed in terms of percentage.

Statistical Analysis

For each mouth rinse and control, the mean and standard deviation of the diameter of inhibition zone was calculated. The data were analyzed using one way Analysis of Variance (ANOVA) followed by Tukey's post hoc test for pair wise comparison**.** p-values <0.05 were considered statistically significant.

RESULT

The results of this study as per the differing methodologies were as follows**,**

Results of disc diffusion method:

Graph 1, demonstrates the zones of inhibition of mouth rinses at time intervals of 18 hours, 24 hours and 48 hours on the agar plates. The disc diffusion method demonstrated that all the mouthrinses used in the present study showed zone of inhibition against *Candida albicans*. The zones of inhibition was largest for Chlorhexidine mouthwash and Mougel ayurvedic mouth wash demonstrated the least zone of inhibition at 18 hours, 24hours and 48 hours respectively**.** Difference in zones of inhibition between Chlorhexidine mouthwash, Povidone iodine mouthwash, Listerine mouthwash and Mougel ayurvedic mouth wash at the time intervals of 18 hours, 24hours and 48 hours was statistically significant, but the zones of inhibition between Chlorhexidine mouthwash and Cetylpyridinium

chloride mouthwash at the end of 18 hours, 24hours and 48 hours was statistically not significant.

MIC, Optical Densities and Percentage Inhibition of the mouthwashes using Broth Macro Dilution technique is illustrated in table 1, Chlorhexidine mouthwash and Cetylpyridinium chloride mouth wash even at the dilutions of 1:64 (15.1 µg/ ml) did not demonstrate any change in their optical densities, followed by Listerine mouthwash which demonstrated changes in optical density at dilutions of 1:32(31.2 µg/ ml), succeeded by Povidone iodine mouthwash wherein changes were observed at the dilutions of 1:8 (125 µg/ ml) and Mougel ayurvedic mouth wash showed changes in optical density at dilutions of 1:2(500 µg/ ml). The table I also demonstrates the percent inhibition of mouthrinses on *Candida albicans* wherein Chlorhexidine mouthwash and Cetylpyridinium chloride mouthwash demonstrated 100 percent inhibition, and Mougel mouthwash demonstrated an inhibition of 100 percent at its original concentration only.

The number of viable colonies were recovered after the exposure of *Candida albicans* to mouthwashes to demonstrate Fungicidal Activity or the kill times at 30 seconds, 5 minutes and 15 minutes. All the mouth washes used in the present study showed kill times that were lesser than thirty seconds as there were no visible colony growth even after incubation of organisms in mouthwash for 15 minutes.

Graph 1: Comparative effect of mouthwashes against Candida albicans at 18 Hours, 24 hours and 48 hours.

Table 1: Minimum Inhibitory Concentration (MIC) and Percentage Inhibition of the five mouthrinses using Broth Macro Dilution technique.

DISCUSSION

The antimicrobial properties of individual mouthrinses were measured by testing the zone of inhibition on the microbial strains incubated on Sabourauds dextrose agar. In regards to the testing of zones of inhibition, many methods have been devised and employed with concerns for cost and time. One of the most popular methods used today, as explained by Cormican M et al, is to measure zones of inhibition by a disc diffusion method. This method is quick, easy, and inexpensive.

In the present study the zones of inhibition of Chlorhexidine was highest and was significantly greater at 18 hours, 24hours and 48 hours than Cetylpyridinium chloride mouth wash, Povidone iodine mouthwash, Listerine mouthwash Mougel ayurvedic mouth wash demonstrated least zone of inhibition at the end of 18 hours, 24hours and 48

Graph 1` :Zone of inhibition of mouthrinses at 18hours, 24 hours and 48 hours

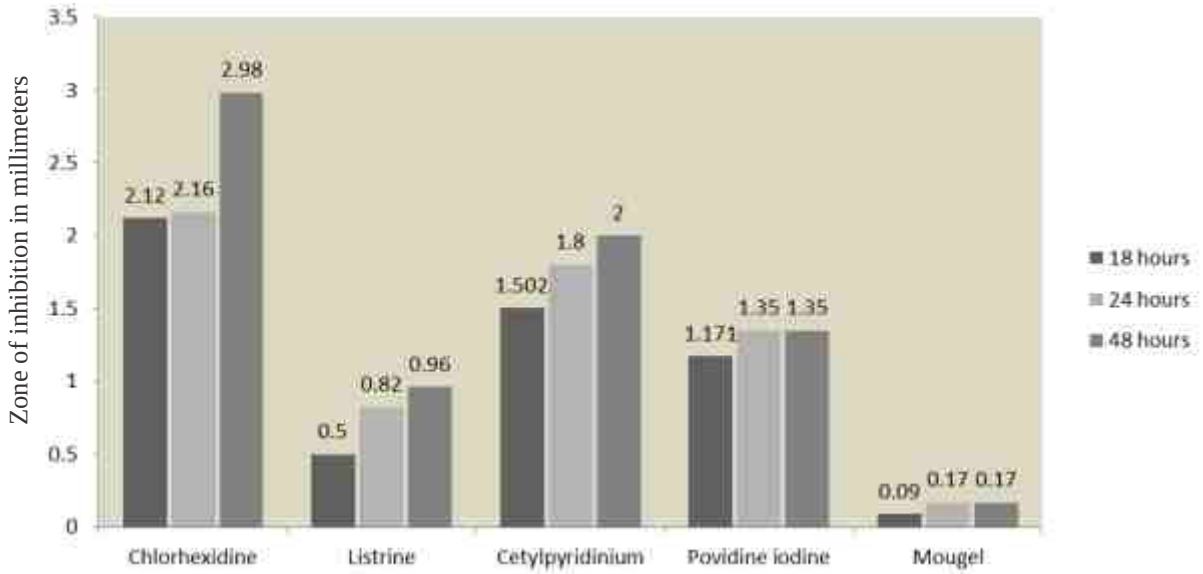


Table 1: Minimum Inhibitory Concentration (MIC) and Percentage Inhibition of the mouthwashes using Broth Macro Dilution.

	1:1	1:2	1:4	1:8	1:16	1:32	1:64
CHLORHEXIDINE % INHIBITION	100	100	100	100	100	100	100
CETYL PYRIDINIUM % INHIBITION	100	100	100	100	100	100	100
POVIDONE IODINE % INHIBITION	100	100	100	90.9	86.9	73.6	26.3
LISTERINE % INHIBITION	100	100	100	90.9	86.9	94.7	94.4
MOUGEL % INHIBITION	100	97.6	92.5	80	50	14.2	0

hours respectively against Candida albicans. In a study conducted by Nakamoto K et al in 1995 [7] chlorhexidine mouthwash did not show any zones of inhibition on cultures of Candida albicans, which is in stark contrast to the present study. The results of the study conducted by Nakamoto K et al in 1995 is similar to with the present study, where in Cetylpyridinium chloride mouth wash demonstrated zones of inhibition of less than 8mm with and without sodium benzoate at the end of 18 hours. The difference in the result may be attributed to the concentration of mouth rinses used as the methodology of both the studies are similar.

In the present study the Mougel showed the least zones of inhibition of less than 1mm even after 48 hours of incubation and is in contrast with the studies of Saeed S et al in 2007 [8], Patel M et al in 2007 [9] and Saini ML et al in 2008 [10] wherein zones of inhibition for Candida albicans were much higher than in the present study. The authors in the above mentioned studies used individual ingredients of the plants directly and used 100 percent concentrations where as in the present study the ingredients were a mixture of Embilica officinalis, Terminalia chebula, Terminalia Belerica, Acacia catechu, Borax and was commercially available as mouthwash and in the present study agar diffusion was carried out with filter paper discs according to CLSI guidelines but in the studies conducted by Saeed S et al in 2007, Patel M et al in 2007 agar diffusion by well method was performed.

In the present study the minimal inhibitory concentration of Cetylpyridinium chloride mouth wash was less than 15.1 (µg/ ml) which was similar to the studies conducted by Nakomato et al [7], Gulliana et al [11], Gulliana et al [12], Edlind MP et al [2] wherein the procedure or methods to achieve minimal inhibitory concentration varied between all the other authors except Edlind MP et al[2]. Where in the agar plates were used for determining the minimal inhibitory concentration where as in the present study differences in optical densities were considered for determining the minimal inhibitory concentration of each of the mouthwashes.

The minimal inhibitory concentration for Chlorhexidine in this study was similar to that of Cetylpyridinium chloride mouth wash where in no changes in optical density was observed at 15.1 (µg/ ml) concentration which is similar to the findings of the studies conducted by Miller et al [13], Gulliana et al [12] but in stark contrast to study conducted by

Gulliana et al [11]. where in the minimal inhibitory concentration for Chlorhexidine mouth wash was higher than the present study, this difference can be attributed to the difference in the methodology of the two studies where in Gulliana et al[11]. have used agar plates to demonstrate the minimal inhibitory concentration of the mouthwashes, whereas the differences in optical densities were considered for the present study and the incubation time for the present study was 18 hours as compared to 48 hours for the study conducted by Gulliana et al[12] the ideal incubation duration is of 16-18 hours, but prolonged incubations as in the study of Gulliana et al [11], should be avoided as antimicrobial deteriorate over time and may result in false interpretation.

The fungicidal efficacy of the mouth washes on *Candida albicans* modified Bernstein method was used where in the kill time or the fungicidal efficacy of the mouth washes were evaluated. In the studies conducted by Yamanaka et al in 1994 on listerine mouthwash and Nakamoto et al in 1995 [7] on Cetylpyridinium chloride and Waltimo et al on povidone iodine kill time or the fungicidal efficacy of the mouth washes, was similar to that of the present study, where as in the study conducted by Gulliana et al 1999[11], cetyl pyridinium chloride showed a longer kill time than the present study, this diffrence in the kill times may be attributed to their assesments of microorganisims on the cultures where in even the presence of discrete colonies were considered to asess the kill times and their kill times ranged for every 5 seconds which is contrary to the present study where the colonies were assesed for only 30 seconds, 5 minutes and 15 minutes and no intermediate readings were assessed.

CONCLUSION

From the results of the present study, it can be concluded that all the five commercially available mouthwashes demonstrated antifungal activity. Cetylpyridinium chloride mouthwash and Chlorhexidine mouthwash demonstrated significantly higher antifungal efficacy than other mouthwashes used in this study. Upon demonstration of Minimal Inhibitory Concentration two mouthwashes namely Cetylpyridinium chloride mouthwash and Chlorhexidine mouthwash demonstrated strong antifungal activity at lower concentrations. The fungicidal activities or the kill times of all mouthrinse was similar and showed that the antifungal efficacy was not time dependent. The findings of the present study suggest that mouthrinses containing antimicrobial agents might represent appropriate alternative to conventional antifungal drugs in the management of diseases caused by *Candida Albicans*. Although results of the present study show best overall antifungal activity of Cetylpyridinium chloride mouthwash and Chlorhexidine mouthwash against *Candida albicans* tested in present study, in vivo studies are needed to prove or refute its efficacy in real environmental circumstances.

REFRENCES

1. Waltimo D, Orstavik EK, Siren, Haapasalo MPP. In vitro susceptibility of Candida albicans to four disinfectants and their combination. International Endodontic Journa 1999;32:421-9.
2. Edlind MP, Smith WL, and Edlind TD. Effects of Cetylpyridinium Chloride Resistance and Treatment on Fluconazole Activity versus Candida albicans Antimicrobial Agents and Chemotherapy.2005;49 (2):843-5.
3. Vasconcelos LSD, Sampaio FD, Sampaio MCC, Pereira MSP, Higino JS, Peixoto MHP. Minimum Inhibitory Concentration of Adherence of Punica granatum Linn (pomegranate) Gel Against S. mutans, S. mitis and C. albicans. Braz Dent J 2003; 17(3):223-7.
4. http://en.wikipedia.org/wiki/ Cohen's_kappa(assessed on October 8th, 2011)
5. Flisfisch S, Meyer J, Meurman JH, Waltimo T. Effects of fluorides on Candida albicans. Oral Diseases. 2008;14(4):296-301.
6. National Committee for Clinical Laboratory

Standards. Reference method for broth dilution antifungal susceptibility testing for yeasts. Proposed standard. Document M-27-P. National Committee for Clinical Laboratory Standards, Villanova.1992.

7. Nakamoto K, Tamamoto M , Hamada T. In vitro effectiveness of mouthrinses against Candida Albicans .Int J Prosthodont 1995; 8(5):486-9.

8. Saeed S, Tariq P Antimicrobial activities of emblica officinalis and coriandrum sativum against gram positive bacteria and Candida albicans. Pak. J. Bot.2007; 39(3): 913-7.

9. Patel M, Shackleton J, Coogan MM , Galpin J, Annual Report March 2006 – March 2007 Division of Oral Microbiology The main research interest of this division is HIV and oral Candidiasis. The research projects in 2006-2007.

10. Saini ML, Saini R, Roy S, Kumar A. Comparative pharmacognostical and antimicrobial studies of acacia species (Mimosaceae). Journal of Medicinal Plants Research2008;2(12):378-86.

11. Gullian G, Pizzo G, Milici ME, Musottoc GC, Gaingreco R. Invitro antifungal properties of mouthrinses containing antimicrobial agents. J Periodontol 1997; 68:729-33.

12. Gullian G, Pizzo G, Milici ME,Gaingreco R . Invitro activities of antimicrobials against Candida species. Oral surg oral Med Oral Pathol Oral Radiol Endol 1999;87:44-9.

13. Meiller T F, Kelley J I, Jabra-Rizk MA, De Paola LG , Baqui AAMA , Falker WA. In vitro studies of the efficacy of antimicrobials against fungi. Oral Surg Oral Med Oral Path Oral Radiol Endo2001;91:663-70.

ORIGINAL RESEARCH ARTICLE

ANTIMICROBIAL EFFICACY OF OZONATED WATER AND CHLORHEXIDINE MOUTH RINSE ON PORPHYROMONAS GINGIVALIS: AN IN VITRO STUDY

Authors:
Mathew MJ[1],
Jithesh Jain[2],
Peter Simon Sequeira[3],
Vipin Jain K[1],
Sudeep CB[1],
Reshmi JK[4]

[1]Post Graduate Student,
Dept. of Public Health Dentistry,
Coorg Institute of Dental Sciences,
K. K. Campus, Magula, Virajpet
571 218, Karnataka, India.

[2]Professor and Head,
Dept. of Public Health Dentistry,
Coorg Institute of Dental Sciences,
K. K. Campus, Magula, Virajpet
571 218, Karnataka, India.

[3]Professor,
Dept. of Public Health Dentistry,
Coorg Institute of Dental Sciences,
K. K. Campus, Magula, Virajpet
571 218, Karnataka, India.

[4]Senior lecturer,
Dept. of Public Health Dentistry,
Pariyaram Dental College,
Pariyaram Medical College P. O.,
Kannur, 670 503, Kerala, India.

Address for correspondence:
Dr. Mathew MJ,
Post Graduate Student,
Dept. of Public Health Dentistry,
Coorg Institute of Dental Sciences,
K. K. Campus, Magula, Virajpet
571 218, Karnataka, India.

ABSTRACT

Background: *Porphyromonas gingivalis* is an anaerobic bacterium that resides within the biofilm community in the subgingival crevice of the oral cavity and is regarded as a major causative agent in the initiation and progression of severe forms of this disease. The established oral antiseptics for periodontal treatment include Chlorhexidine Gluconate (CHX, 0.2-2%). Regarding side-effects, it is known that Chlorhexidine may cause mucosal desquamation, impaired wound healing and fibroblast attachment to the tooth surfaces, tooth staining and altered taste sensation. The objective of this in-vitro study was to assess the efficacy of ozonated water and 0.2% Chlorhexidine mouth rinse on *Porphyromonas gingivals*..

Methods: The bacterial strains *Porphyromonas gingivalis* (ATCC 33277) were incubated in trypticasein-soy agar supplemented with 5% lamb blood. 100 ml of ozonated water and 0.2% chlorhexidine was poured in the respective well and the plates were incubated at 37^0 for 24 hrs. The zone of inhibition was measured at 1 min and 24 hrs . Independent samples t test was used and p-value <0.05 was considered statistically significant.

Results: After 1 min the effect of ozone on *P.gingivalis* was superior when compared to chlorhexidine.

Conclusion: Ozonated water significantly inhibits the growth of *P.gingivalis* in-vitro.

Key words: Chlorhexidine, Ozone, Bacteria.

J Odontol Res 2013;1(1)11-6.

INTRODUCTION

Actinobacillus actinomycetemcomitans and *Porphyromonus gingivalis* are major putative periodontopathicbacteria.[1] *Actinobacillus actinomycetemcomitans* has been closely associated with periodontitis in young individuals and with cases of refractory adult periodontitis. *Porphyromonus gingivalis* occurs in severe adult periodontitis, failing guided tissue regeneration and acute periodontal abscesses. Other organisms have also been related to advanced periodontitis, although with less evidence.[(1)] *Actinobacillus actinomycetemcomitans* and *Porphyromonus gingivalis* can also seed to and produce severe infections in extraoralsites (gastrointestinal tract, respiratory tract, colon etc.).[(2)]

Removal of dental plaque thus forms an important part of controlling and treating periodontal disease, which brings about qualitative as well as quantitative changes in the subgingival microflora. A number of chemical adjuncts have been used to improve the outcome of mechanical oral hygiene procedures, one of which is chlorhexidine broad spectrum antibiotic with antimicrobial effects on gram positive as well as Gram-negative bacteria, viruses and fungi.[(3,4)]

An alternative approach to conventional antimicrobial or antiseptic agent in the suppression of subgingival bacteria is to inhibit their growth by changing the subgingival environment, which has been shown to be highly anaerobic with a prevailing low oxygen tension.[(5)] First advocated by Dunlop in 1913, various agents such as molecular oxygen,[(6)] hyperbaric oxygenation[(7)] and hydrogen peroxide have been applied.[(8)] It has been shown that repeated sub gingival oxygen irrigation in previously untreated deep periodontal pockets resulted in a significant clinical improvement of the periodontal baseline conditions.[(9)] Recently, ozone therapy is gaining popularity in various treatment modalities in the field of medicine, dentistry, veterinary, food industry, water treatment etc. In dentistry, ozone is being successfully utilized for the treatment of dental caries.[(10)]

Recent investigations have reported antimicrobial effects on oral pathogens by both gaseous and aqueous forms of ozone, and the effectiveness of ozone in the treatment of oral diseases is currently a subject of intense research.[(11)]

The current study outlines the latest thinking regarding the efficacy of ozone therapy and Chlorhexidine 0.2% on virulent periodontal pathogenic microorganism, *Porphyromonas gingivalis.*

The objectives of this study were to assess the efficacy of ozonated water and 0.2% chlorhexidine mouth rinse on *Porphyromonas gingivalis* and to compare the efficacy of ozonated water and 0.2% Chlorhexidine mouth rinse on *Porphyromonas gingivalis* inhibition.

METHODS

The materials used in this study were:

Test Materials used:

- Ozone
- Chlorhexidine (0.2%)

  Microorganism
- *Porphyromonas gingivalis ATCC 33277*
- The microbial strains selected for the present study were collected from the American Type Culture Collection (ATCC), USA.
- Brain heart infusion agar
- Vernier Caliper.

Ozonated Water Preparation

The wells were subjected inoculated with ozonated water that was released from an irrigation device, “Aqu Ozone”. The device released a single pulsating stream of ozonated water from the nozzle, which could be adjusted for different speeds and pressures ranging from 350 to 500 kPa (kilo pascals) and an ozone output of 0.082 mg/h, at a noise output of <70 dB (decibels) and water outflow of ≥450 ml. A 20-gauge blunt needle was bent and attached to the tip of the nozzle of the ozone dental jet holder and then inoculated into the different wells.

Microbial Assay

The bacterial strains *Porphyromonas gingivalis* (ATCC 33277) was obtained by Dept of Microbiology, Coorg Institute of Dental Sciences, Karnataka. The strains were incubated in trypticasein-soy agar supplemented with 5% lamb blood at 37°C in an oxygen-free atmosphere, and stored in the fridge until use. They were preserved by freezing suspensions at -70°C in skimmed milk supplemented with a cryoprotectant. For the experiments, bacterial suspensions (pure cultures) were inoculated in TS broth and incubated for 7 days at 37^0 C, after which they were placed in the fridge. Wells of 8 mm diameter were cut into solidified trypticasein-soy agar media using a sterilized standard device. 100 ml of ozonated water and 0.2% chlorhexidine was poured in the respective well and the plates were incubated at 37%for 24 hrs. To ensure the consistency of all findings, the experiment was performed and repeated under strict aspetic conditions. The bacterial activity was expressed in terms of the mean of zone of inhibition (in mm) produced by each test product at the end of incubation period. All the measurement of zone of inhibition were carried out by a single examiner. Calibration of the examiner was done prior to and during the study by reexamining 5 % of the samples, to minimize intra examiner variability. Intra examiner agreement was determined using kappas statistics (k) and the score thus obtained (k=0.82) almost perfect, according to Landis and koch, thus meeting the scientific requirement of validity and reliability

Statistical Analysis

The collected data was classified and tabulated in Microsoft Office excel. SPSS for windows version 17 software (Chicago, USA) was employed for statistical analysis. Mean*(X)* and Standard Deviation (SD) were calculated. Independent samples t test was used for and p-value <0.05 was considered statistically significant.

RESULTS

The study was conducted to assess the efficacy of ozonated water and 0.2% Chlorhexidine mouth rinse on *Porphyromonas gingivalis.* Test products (Ozone and chlorhexidine) were tested after 2 intervals. i.e after 1 min and after 24hours. After 1 min the effect of ozone on *Porphyromonas gingivalis* was superior (24CFU/ml) when compared to chlorhexidine (22CFU/ml) as mentioned in Graph I .

DISCUSSION

Ozone is an allotropic form of oxygen. It possesses unique properties which are being defined and applied to biological systems as well as to clinical practice. As a molecule containing a large excess of energy, ozone through incompletely understood mechanisms, manifests bactericidal, viricidal and fungicidal actions which may make it a treatment of choice in certain conditions and an adjunctive treatment in others[12].

Ozone can be considered a potential therapy for periodontal disease due to its wide range of biological effects. Some of these are:

- Inactivation of bacteria, viruses, fungi, yeast and protozoa,
- Enhancement of circulation,
- Stimulation of oxygen metabolism,
- Activation of the immune system.

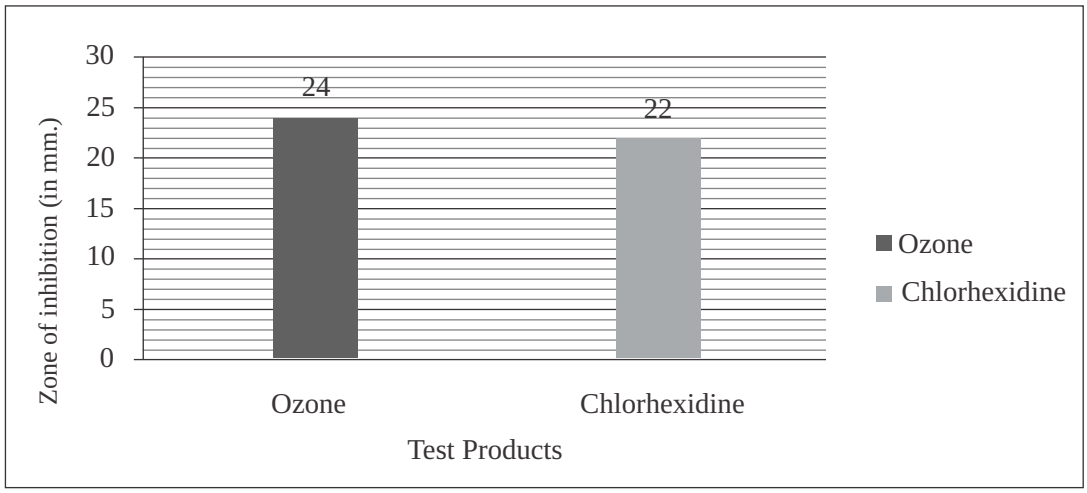
Each of these actions is without doubt beneficial in management of inflammatory periodontal disease[12].

The results of the present study showed that inhibition of the *P.gingivalis,* the specific strain selected by both ozone therapy and chlorhexidine were comparable. The difference between the inhibitory effects of both the test materials was statistically significant ($p < 0.05$).

The results of this study were thus in line with a previous study conducted by Kammer et al to examine the effect of ozonated water on oral microorganisms including *Porphyromonas gingivalis*. The viable count of the organism was found to decrease when treated with ozonated water.

This particular study had studied the effect of ozonated water on several other microorganisms implicated in the causation of periodontal disease.

Graph I : Zone of inhibition (in mm.) of the test products against *P. gingivalis* after 1 minute



After 24hrs the effect of ozone on *Porphyromonas gingivalis* was low (6CFU/ml) when compared to chlorhexidine (22CFU/ml) as mentioned in Graph II, which indicates that residual effect of chlorhexidine remains the same whereas, the effect of Ozone was depleted after 24hrs.

Graph II : Zone of inhibition (in mm.) of the test products against *P. gingivalis* after 24 hours

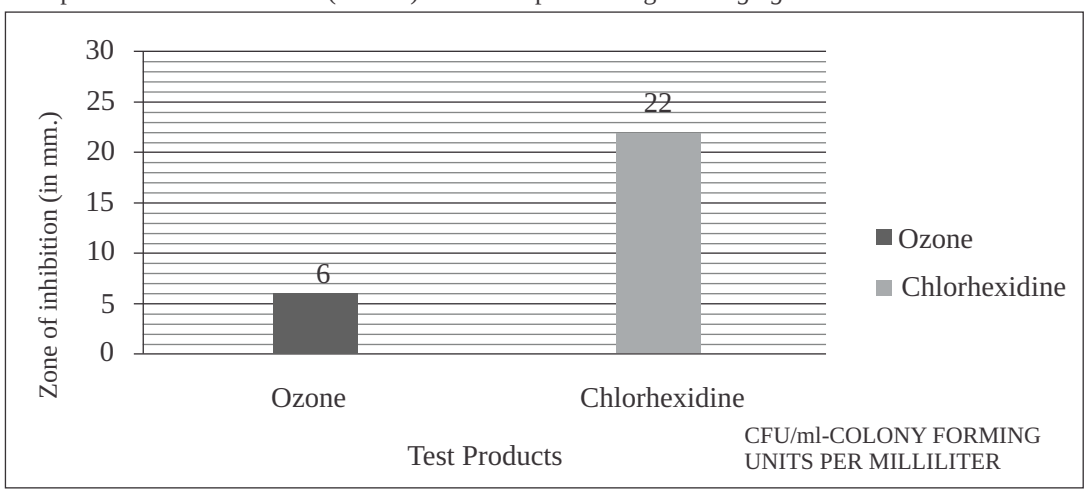


Table 1 shows antimicrobial activity of the extracts against *Porphyromonas gingivalis* after 1 min and 24 hours. Ozone showed a lowest inhibition rate against *Porphyromonas gingivalis* compared with the chlorhexidine and this finding was found to be statistically significant.

Samples	Mean zone of inhibition (in mm) ±S.D		p value
	After 1 min	After 24hrs	0.0482*
Ozone	24	6	
Chlorhexidine (0.2%)	22	22	

*- statistically significant

Nevertheless, the results for the bacterial strain *Porphyromonas gingivalis* can be compared to the present study.

Our findings are similar to that obtained by Ripolles de ramon et al.[13], who found a significant decrease in the microbiological periodontal parameters.

To compare the effectiveness of ozonated water, the present study used a positive control, namely chlorhexidine which is the standard adjunctive therapy for treatment of periodontitis.

The outcomes in both the groups (ozonated water and Chlorhexidine) with regard to the inhibition of the bacterial count were similar at 1 min after treatment. However, assessment after 24 hours showed that microbial count in the ozonated group had increased.

This differed significantly from that of Chlorhexidine group. The substantivity property of Chlorhexidine is well known and would have accounted for this difference found after 24 hours. Hence, this could be one drawback of ozonated water when compared to Chlorhexidine.

Our study considered only a single strain of a single species of microorganism known to a periodontal pathogen. The effect of ozone therapy at different concentrations on other microorganisms implicated in the red complex of periodontal disease, at various time intervals need to be determined. The inherent limitations of an in-vitro study need to kept in mind while interpreting the results.

Effectiveness trials in-vivo where the effect of other confounders like saliva, which could dilute the concentration of ozone, a number of other aerobic and anaerobic microorganisms in and around the gingival pocket, the mode and dose of administration, the cost of the therapy need to be evaluated thoroughly before making generalizing statements.

Ozone therapy significantly inhibits growth of the *Porphyromonas gingivalis* which was the selected strain in the present study. One major drawback of ozone is its unstable nature. The absorbance of water in the ozone increased almost linearly with time from 5 to approximately 60 seconds. The stability of ozone in the water is low and ozone dissipated very quickly in the ozone demand free water at room temperature over 5 mins, as described by Shechter.[14,15]

CONCLUSION

Ozonated water significantly inhibits the growth of *Porphyromonas gingivalis* in vitro. However, the inhibitory effect was pronounced at 1 min and was low at 24hours.

Ozone may be considered as an alternative management strategy due to its powerful ability to inactivate microorganisms. Also, there is growing evidence that ozone can be employed as a useful therapeutic agent in both dentistry and medicine.

REFERENCES

1. Slots J, Chen C. The oral microbiota and human periodontal disease of the normal microflora. London: Kluwer Academic Publishers, 1999:101-127.
2. Van Winkelhoff AJ, Slots J. *Actinobacillus actinomycetemcomitans* and *Porphyromonas gingivalis* in nonoral infections.Periodonto12000 1999: 20: 122-135.
3. Bernstein D, Schiff G. IN vitro viricidal effectiveness of a 0-12% chlorhexidine Gluconate mouth rinse. J Dent Res 1990;69:874-6.
4. Ellepola AN, Samaranayaka LP. Adjunctive use of chlorhexidine in oral Candidoses.A review Oral Dis 2001;7:11-7
5. Mettraux GR, Gusberti FA. Oygen tension in untreated human periodontal pockets. J Clin Periodontol 1984;55:516-21.
6. Hirsch RS, Clarke NG, Townsend GC. The effect of locally released oxygen on the debelopment of plaque and gingivitis in man. J Clin Periodontol 1981;8:21-8.
7. Guenterman RH, Bishop JG, Collings KC, Dorman HL. The effect of increased blood oxygen tensions on periodontal disease. J Periodontol 1972;43:233-6.

8. Wennstrom J, Lindhe J . Effect of hygrogen peroxide on developing plaque and gingivitis in man. J Clin Periodontol 1979;6:115-30.

9. Schlagenhauf U,Horlacher V, Netuschel L, Breck M. The effect of repeated subgingival oxygen irrigations in previously untreated periodontal pockets. J Clin Periodontol 1994;21:48-50.

10. Rickard GD, Richardson R, Johnson T, McColl D, Hooper L. Ozone therapy for treatment of dental caries. Cochrane Database Syst Rev 2004;(3):CD004153.

11. Huth KC, Jakob FM, Saugel B, Cappello C, Paschos E, Hollweck R, Hickel R, Brand K. E ect of ozone on oral cells compared with established antimicrobials. Eur J Oral Sci 2006;114: 435-440

12. Durga Kshitish, Vandana K Laxman. The use of ozonated water and 0.2% chlorhexidine in the treatment of periodontitis patients.A clinical and microbiological study.Indian Journal of Dental Research 2010;3:341-348.

13. Ripolles de ramon, J., Colmenero ruiz, C., Gallut ruiz, J. et al. Clinical, microbiological and inmunological evaluation of the ozone's therapy in patients with moderate-severe periodontal pockets. AvPeriodon Implantol. 2004;16,1:63-72.

14. Wilson M. Suceptability of oral bacterial biofilms to antimicrobial agents. J Med Microbiol 1996;44:79-87.

15. Scheter H. spectrophotometric method of determination of ozone in aqueous solution.Water Res 1973;7:729-39.

ORIGINAL RESEARCH ARTICLE

ORAL HEALTH RELATED QUALITY OF LIFE OF B.Ed STUDENTS IN DAVANGERE CITY, INDIA.

Authors:
Rama Sharma[1],
Mahesh Hiregoudar [2],
Prashant GM[3],
Naveen Kumar PG[4],
Chandu GN[5]

[1]Senior Lecturer, Department of Public Health Dentistry, Sardar Patel Post Graduate Institute of Medical and Dental Sciences, Chaudhary Vihar, Uthrathia, Raibareily Road, Lucknow 226 002, Uttar Pradesh, India.

[2]Senior Lecturer, Dept. of Public Health Dentistry, Coorg Institute of Dental Sciences, K. K. Campus, Magula, Virajpet 571 218, Karnataka, India.

[3]Reader, Dept. of Preventive and Community Dentistry, College of Dental Sciences, Davangere 577 004, Karnataka, India.

[4]Professor and Head, Dept. of Preventive and Community Dentistry, College of Dental Sciences, Davangere 577 004, Karnataka, India.

[5]Professor, Dept. of Preventive and Community Dentistry, College of Dental Sciences, Davangere 577 004, Karnataka, India.

Address for correspondence:
Dr. Rama Sharma, Senior Lecturer, Department of Public Health Dentistry, Sardar Patel Post Graduate Institute of Medical and Dental Sciences, Chaudhary Vihar, Uthrathia, Raibareily Road, Lucknow 226 002, Uttar Pradesh, India.
E mail: drramasharma30@gmail.com

ABSTRACT

Background: To assess the effect and impact of oral health on quality of life among B.Ed students of Davangere city, India.

Methods: A questionnaire study, using OHQoL-UK (W) questionnaire, which takes into consideration both 'effect' and 'impact' of oral health on life quality, incorporating an individualized weighting system. The questionnaire was administered to a sample of 269 B.Ed students, of Davangere city, India. Gender variations on the responses of their effect and impact on OHRQoL was analyzed using Chi- square test.

Results: The overall response rate was 89.67%. Of the total students 39.8% (n=107) were males and 60.2% (n=162) were females. 63.17% believed that their oral health enhanced their QoL, 30.34% believed that their oral health had no effect on their QoL and 6.48% believed that their oral health reduced their QoL. Most frequently (29.78%) the respondents have stated that the various aspects of oral health have moderate impact on their quality of life. Response related to breath odour was only found to be statistically significant between the genders.

Conclusion: To conclude, the study shows that the B.Ed students of Davangere city, India perceive oral health as affecting their QoL and also has an impact on QoL in a variety of physical, social and psychological ways.

Key words: Oral health, B.Ed students, Quality of Life

J Odontol Res 2013;1(1):17-24.

INTRODUCTION

Oral diseases such as dental caries and periodontal diseases are highly prevalent and their consequences are not only physical, they are also economical, social and psychological. They seriously impair quality of life (QoL) in a large number of individuals and can affect various aspects of life, including oral function, appearance and interpersonal relationships.[1]

Symptoms that arise from oral disease, such as toothache, are among the most common health problems and a large number of school and work days are lost due to oral health problems and/or their treatments. Over the past decade there has been an explosion of interest in conceptualizing, developing and assessing the impact of oral heath on life quality.[2]

The World Health Organisation agrees, 'QoL' is an individual's perception of their position in life in the context of culture and the value systems in which they live and in relation to their goals, expectations, standards and concerns. It is ultimately a personal and dynamic concept, a concept which respects the autonomy of the individual and acknowledges that people can provide information about what is in their own interest.[3] Consequently, there has been a tremendous growth in the literature concerned with these constructs. This is also the case in dentistry, where there has been a proliferation of instruments and scales seeking to assess what has come to be called oral health-related quality of life (OHR-QoL) and / or the quality of life of patients with various oral conditions.[4]

In dentistry, measures of oral health-related quality of life have been used in oral health surveys of adolescents, adults, and elderly populations, and as outcome measures in clinical trials of implant therapies and evaluations of dental care programs for special care populations. Their use in clinical practice and clinical decision making has yet to be reported.[5] Further more, the majority of existing instruments measure only the prevalence of the effects of oral health on life quality and fail to capture the importance or salience of their effects overall i.e. impact (weighting).[6] Hence the present study has been undertaken to explore the perception of B. Ed students who will be future school teachers towards their OHRQoL. The objective of the study thus was to assess the effect and impact of oral health on quality of life among Bachelor of Education (B.Ed) students of Davangere city, Karnataka, India.

METHODS

Study population

The target population was B.Ed. students of Davangere city. Three B.Ed colleges were randomly selected, which had a total enrolment of 300 students. Of the total B.Ed students 269 students were available on the day of survey, and thus were included in the study. Prior to the survey, the permission was obtained from the principals of the respective colleges and programme was scheduled accordingly. All questions in the questionnaire were close-ended.

The data was collected by two post graduate students posted in the Department of Preventive and Community Dentistry, College of Dental Sciences, Davangere. The questionnaires were distributed to the B. Ed students of various colleges on the scheduled dates.

Data collection

The survey is a questionnaire survey, a specially designed OHRQoL questionnaire by McGrath C and Bedi R was used[6]. It consists of a battery of 16 questions which takes into account both effect and impact of oral health on quality of life. In the beginning of the questionnaire, the personal information regarding the subject was obtained like name, sex, age and college.

The OHRQoL questionnaire was distributed to the students. The students were made to understand each and every question and about effect and impact related to each question, each of the proposed sixteen items were scored firstly on effect, with responses ranging from bad-to-good effect, on quality of life. Then students were asked to rate the "impact" of each "effect" on a scale ranging from none-to-extreme impact, in that way incorporating an individualized weighting system. (Fig 1)

What effect, if any does the condition of your teeth, gums, or mouth have on your appearance*?

Good-none-bad

How would you rate the impact of the effect on your Quality of Life?

None-little-moderate-great-extreme

*Substitute for each of the sixteen items

Fig.1:
Example of working of OHRQoL questions

Each item could thus be scored on a scale from 1 to 9.

Scoring criteria

1. Good effect of extreme impact
2. Good effect of great impact
3. Good effect of moderate impact
4. Good effect of little impact
5. No effect of no impact
6. Bad effect of little impact
7. Bad effect of moderate impact
8. Bad effect of great impact
9. Bad effect of extreme impact

Ethical clearance for conducting the study was obtained from the Ethical Committee of the college.

Statistical analysis

The data were coded and analyzed using the Statistical Package for Social Sciences version 18.0 (SPSS software). Chi square test was used to know whether there was any statistically significant difference between males and females on their responses to effect and impact on oral health related quality of life. The level of significance was set at p = 0.05.

RESULTS

The overall response rate was 89.67% with 269 students participating in the study. Of the total students 39.8% (n=107) were males and 60.2% (n=162) were females.

Table I shows the distribution response of the 269 participants. 63.17% believed that their oral health enhanced their QoL, 30.34% believed that their oral health had no effect on their QoL and 6.48% believed that their oral health reduced their QoL. Most frequently the respondents have stated that their oral health has good effect on their quality of life in all aspects except breath odour (35.3%, 95). In particular 42.8% (115) and 43.1% (116) respondents stated that their oral health has no effect on their breath odour and mood respectively. Less frequently, respondents have stated oral health as detracting from their quality of life, most often by causing bad effect.

Table II shows the distribution of responses of "OHRQoL-impacts" of these "effects"on the quality of life. Most frequently (29.78%) the respondents have stated that the various aspects of oral health have moderate impact on their quality of life. 27.5% (74) respondents stated that there is no impact of oral health on their work /usual jobs and 27.9% (75) stated there is little impact of oral health on their mood.

Table III shows the distribution of responses to "OHRQoL-impacts" of these "effects" on oral health related quality of life (gender variation). Response related to breathe odour was only found to be statistically significant.

Table IV shows the gender variations of these effects on their OHRQoL. There is no statistically significant difference observed between males and females regarding the following mentioned effects on their OHRQoL.

DISCUSSION

This study focused on future school teachers who

Table I: Distribution of responses to OHRQoL- effects

Response	Effects		
	Good effect % (n)	No effect % (n)	Bad effect % (n)
Physical aspects			
a) Eating	74.7 (201)	19.3 (52)	5.9 (16)
b) Appearance	71.4 (192)	19.3 (52)	9.3 (25)
c) Speech	74 (199)	20.4 (55)	5.6 (15)
d) General health	62.5 (168)	33.8 (91)	3.7 (10)
e) Comfort	69.5 (187)	24.2 (65)	6.3 (17)
f) Breath odour	35.3 (95)	42.8 (115)	21.9 (59)
Social aspects			
a) Social life	57.6 (155)	36.8 (99)	5.6 (15)
b) Romantic relationship	65.4 (176)	32 (86)	2.6 (7)
c) Smiling/laughing	77.7 (209)	17.1 (46)	5.2 (14)
d) Work/usual jobs	57.6 (155)	39.4 (106)	3 (8)
e) Career	59.9 (161)	36.8 (99)	3.3 (9)
Psychological aspects			
a) Confidence	71.7 (193)	22.7 (61)	5.6 (15)
b) Carefree	52 (140)	38.7 (104)	9.3 (25)
c) Sleep/ability to relax	59.1 (159)	36.4 (98)	4.5 (12)
d) Mood	51.3 (138)	43.1 (116)	5.6 (15)
e) Personality	71 (191)	22.7 (61)	6.3 (17)
Total	63.17%	30.34%	6.48%

may be involved in teaching oral health related topics and may influence the adoption or implementation of oral health programs that may benefit children.

This is one of the first attempts to understand how the future school teachers perceive oral health and its effects and impacts on their quality of life. Since not many studies have been published that measures OHRQoL among future school teachers, comparisons will be made with similar studies conducted on different populations.

The response rate to this study was 89.67% which is similar to the studies conducted by Colman McGrath et.al in 2001[6] and Manish Kumar et.al in 2007[7] using the same OHRQoL indicator, which is a good signal to incorporate oral health related quality of life components into oral health surveys.

In this study female respondents (60.2%) outnumbered the male respondents (39.8%) which is similar to the studies conducted by Colman McGrath et.al in2001[6] and Manish Kumar et.al in 2007[7] this reflects the enrollment pattern of B.Ed students. Women perceived oral health as impacting more strongly on their quality of life as compared to men[8].

A large portion of the respondents (63.17%) perceived that oral health has good effect on their quality of life in all physical, social and psychological aspects, which is comparable to a study conducted by Manish Kumar et.al in 2007[7] on first grade college students where 65.6% of the respondents reported enhanced effect. However it is in contrast to the study conducted by Colman McGrath et.al in 2001[6] on general population where

Table II: Distribution of responses of OHRQoL – impacts

Response	Impacts				
	None %(n)	Little %(n)	Moderate%(n)	Great% (n)	Extreme%(n)
Physical aspect					
a) Eating	10.8 (29)	21.6 (58)	30.5 (82)	28.6 (77)	8.6 (23)
b) Appearance	13.4 (36)	14.9 (40)	33.1 (89)	30.1 (81)	8.6 (23)
c) Speech	13.8 (37)	11.9 (32)	27.9 (75)	36.4 (98)	10 (27)
d) General health	16 (43)	21.6 (58)	30.9 (83)	23.4 (63)	8.2 (22)
e) Comfort	12.6 (34)	23 (62)	29.4 (79)	25.7 (69)	9.3 (25)
f) Breath odour	25.7 (69)	20.1 (54)	24.2 (65)	22.3 (60)	7.8 (21)
Social aspects					
a) Social life	25.7 (69)	20.1 (54)	30.1 (81)	17.5 (47)	6.7 (18)
b) Romantic relationship	21.9 (59)	12.6 (34)	35.3 (95)	19.3 (52)	10.8 (29)
c) Smiling/laughing	11.2 (30)	17.8 (48)	28.6 (77)	31.2 (84)	11.2 (30)
d) Work/usual jobs	27.5 (74)	18.6 (50)	26 (70)	23.4 (63)	4.5 (12)
e) Career	22.7 (61)	19.3 (52)	27.9 (75)	21.9 (59)	8.2 (22)
Psychological aspects					
a) Confidence	11.2 (30)	21.6 (58)	36.1 (97)	21.6 (58)	9.7 (26)
b) Carefree	23.8 (64)	21.2 (57)	32 (86)	17.5 (47)	5.6 (15)
c) Sleep/ability to relax	21.9 (59)	21.6 (58)	28.3 (76)	22.3 (60)	5.9 (16)
d) Mood	19.7 (53)	27.9 (75)	27.5 (74)	20.4 (55)	4.5 (12)
e) Personality	13 (35)	16.7 (45)	28.6 (77)	30.5 (82)	11.2 (30)
Total	18.18%	19.41%	29.78%	24.51%	8.18%

only one third of population reported a good effect of oral health on their quality of life.

Near about one-third (30.34%) of respondents stated that there is no effect of oral health on their quality of life, which is in accordance with a study conducted by Manish Kumar et.al in 2007[7] where 24.2% of respondents stated the same.

It is interesting to note that a vast majority of respondents (77.7%) agree to the fact that smiling and laughing had a good effect, while least number of respondents believed that a good smile and laugh had no impact on OHRQoL, reflecting the respondent's high concern towards their social life. This is in accordance with a study conducted by Manish Kumar et.al in 2007[7] on first grade college students where respondents reported similar result but is in contrast to the study conducted on general population by Colman McGrath et.al in 2001[6] where 61% of respondents stated that oral health has good impact on their eating, reflecting a high concern towards the physical aspect of quality of life.

Respondents claim that their oral health effects all the aspects of quality of life equally, which clearly indicates that future school teachers were oriented towards all physical, social and psychological aspects of OHRQoL, this is in contrast to the study conducted on general population by Colman McGrath et.al in 2001[6] where respondents were more inclined towards physical aspects and is in accordance to the study by Manish Kumar et.al in 2007[7].

Table III: Distribution of responses to OHRQoL – impacts (gender variation)

Response	Impacts										p-value
	None		Little		Moderate		Great		Extreme		
	M % (n)	F % (n)	M % (n)	F % (n)	M % (n)	F % (n)	M % (n)	F % (n)	M % (n)	F % (n)	
Physical aspects											
a) Eating	8.41 (9)	12.35 (20)	20.56 (22)	22.22 (36)	32.71 (35)	29.01 (47)	29.91 (32)	27.78 (45)	8.41 (9)	8.64 (14)	0.894
b) Appearance	8.41 (9)	16.67 (27)	16.82 (18)	13.58 (22)	34.58 (37)	32.09 (52)	28.97 (31)	30.86 (31)	11.22 (12)	6.79 (11)	0.248
c) Speech	13.08 (14)	14.20 (23)	14.95 (16)	9.88 (16)	27.10 (29)	28.40 (46)	36.45 (39)	36.42 (59)	8.41 (9)	11.11 (18)	0.743
d) General health	16.82 (18)	15.43 (25)	18.69 (20)	23.46 (38)	31.78 (34)	30.25 (49)	26.17 (28)	21.60 (35)	6.54 (7)	9.26 (15)	0.743
e) Comfort	10.28 (11)	14.20 (23)	24.30 (26)	22.22 (36)	28.97 (31)	29.63 (48)	28.04 (30)	24.07 (39)	8.41 (9)	9.88 (16)	0.834
f) Breath odour	15.89 (17)	32.10 (52)	19.63 (21)	20.37 (33)	28.04 (30)	21.60 (35)	28.04 (30)	18.52 (30)	8.41 (9)	7.41 (12)	0.034
Social aspects											
a) Social life	24.30 (26)	26.54 (43)	20.56 (22)	19.75 (32)	29.91 (32)	30.25 (49)	16.82 (18)	17.90 (29)	8.41 (9)	5.56 (9)	0.913
b) Romantic relationship	15.89 (17)	25.93 (42)	14.02 (15)	11.73 (19)	36.45 (39)	34.57 (56)	20.56 (22)	18.52 (30)	13.08 (14)	9.26 (15)	0.366
c) Smiling/ laughing	14.02 (15)	9.26 (15)	16.83 (18)	18.52 (30)	28.04 (30)	29.01 (47)	29.91 (32)	32.10 (52)	11.22 (12)	11.11 (18)	0.821
d) Work/ usual Jobs	23.37 (25)	30.25 (49)	21.50 (23)	16.67 (27)	25.23 (27)	26.54 (43)	25.23 (27)	22.22 (36)	4.67 (5)	4.32 (7)	0.694
e) Career	24.30 (26)	21.60 (35)	16.82 (18)	20.99	28.04 (30)	27.78 (45)	20.56 (22)	22.84 (37)	10.28 (11)	6.79 (11)	0.754
Psychological aspects											
a Confidence	11.22 (12)	11.11 (18)	20.56 (22)	22.22 (36)	40.19 (43)	33.33 (54)	20.56 (22)	22.22 (36)	7.48 (8)	11.11 (18)	0.757
b) Carefree	21.50 (23)	25.31 (41)	24.30 (26)	19.14 (31)	30.84 (33)	32.72 (53)	19.63 (21)	16.05 (26)	3.74 (4)	6.79 (11)	0.588
c) Sleep/ ability to relax	20.56 (22)	22.84 (37)	19.63 (21)	22.84 (37)	29.91 (32)	27.16 (44)	26.17 (28)	19.75 (32)	3.74 (4)	7.41 (12)	0.512
d) Mood	18.70 (20)	20.37 (33)	27.10 (29)	28.40 (46)	26.17 (28)	28.40 (46)	25.23 (27)	17.28 (28)	2.80 (3)	5.56 (9)	0.504
e) Personality	14.02 (15)	12.35 (20)	12.15 (13)	19.75 (32)	34.58 (37)	24.69 (40)	28.97 (31)	31.48 (51)	10.28 (11)	11.73 (19)	0.306

Table IV : Distribution of responses to OHRQoL - effects (gender variation)

Response	Effects						p-value
	Good effect		No effect		Bad effect		
	Male %(n)	Female %(n)	Male %(n)	Female %(n)	Male %(n)	Female %(n)	
Physical aspects							
a) Eating	75.01 (81)	74.07 (120)	15.89 (17)	21.60 (35)	8.41 (9)	4.32 (7)	0.232
b) Appearance	70.09 (75)	72.22 (117)	17.76 (19)	20.37 (33)	12.15 (13)	7.41 (12)	0.401
c) Speech	89.16 (74)	77.16 (125)	21.50 (23)	19.75 (32)	9.35 (10)	4.67 (5)	0.075
d) General health	57.94 (62)	65.43 (106)	39.25 (42)	30.25 (49)	2.80 (3)	4.32 (7)	0.283
e) Comfort	69.16 (74)	69.75 (113)	24.30 (26)	24.07 (39)	6.54 (7)	6.1 (10)	0.991
f) Breath odour	38.32 (41)	33.33 (54)	41.12 (44)	43.83 (71)	20.56 (22	22.84 (37)	0.699
Social aspects							
a) Social life	62.62 (67)	54.32 (88)	33.64 (36)	38.89 (63)	3.74 (4)	6.79 (11)	0.312
b) Romantic relationship	67.29 (72)	64.20 (104)	29.91 (32)	33.33 (54)	2.80 (3)	2.47 (4)	0.836
c) Smiling/laughing	74.77 (80)	79.63 (129)	22.43 (24)	13.58 (22)	2.80 (3)	6.79 (11)	0.078
d) Work/usual jobs	59.81 (64)	56.17 (91)	36.45 (39)	41.36 (67)	3.74 (4)	2.47 (4)	0.640
e) Career	53.27 (57)	64.20 (104)	43.93 (47)	32.10 (52	2.80 (3)	3.70 (6)	0.143
Psychological aspects							
a) Confidence	68.22 (73)	74.07 (120)	27.10 (29)	19.75 (32)	4.67 (5)	6.17 (10)	0.350
b) Carefree	50.47 (54)	53.09 (86)	39.25 (42)	38.27 (62)	10.28 (11)	8.64 (14)	0.866
c) Sleep/ability to relax	56.07 (60)	61.11 (99)	38.32 (41)	35.19 (57)	5.61 (6)	3.70 (6)	0.615
d) Mood	47.66 (51)	53.70 (87)	45.79 (49)	41.36 (67)	6.54 (7)	4.94 (8)	0.592
e) Personality	67.29 (72)	73.46 (119)	19.75(32)	19.75 (32)	5.61 (6)	6.79 (11)	0.364

CONCLUSION

To conclude, this study shows that the B.Ed students of Davangere city, India agree that their oral health affects their quality of life and also has an impact on quality of life in all the aspects i.e. physical, social and psychological. Gender variation was not significant showing that both the sexes are equally aware towards their oral health and its effect and impact on their quality of life.

Perception of future school teachers towards their OHRQoL gives an insight as to how well they understand how their oral health can affect their general well being in various aspects and the important role they can play in imparting the same knowledge to the school children.

Further in future, studies relating various socio-demographic profiles as having an effect on oral health related quality of life are recommended in India.

REFERENCES

1. Naito M, Yuasa H, Nomura Y, Nakayama T, Hamajima N, Hanada N. Oral health status and health-related quality of life: a systematic review. Journal of oral sciences 2006; 48(1):1-7.
2. McGrath C, Bedi R. Population based norming of the UK oral health related quality of life measure (OHRQoL-UK©). Br Dent J 2002;193(9):521-4.
3. McGrath C, Bedi R. A national study of the importance of oral health to life quality to inform scales of oral health related quality of life. Quality of Life Research 2004;13: 813–8.
4. Locker D, Allen F. What do measures of 'oral health-related quality of life' measure?

   Comm Dent Oral Epidemiol 2007;35:401-11.
5. Locker D, Allen FP. Developing Short-form Measures of Oral Health-related Quality of Life. Journal of Public Health Dentistry. 2002;62(1):13-20.
6. McGrath C, Bedi R. An evaluation of a new measure of oral health related quality of life – OHRQoL- UK (W). Comm Dent Health 2001; 18: 138-43.
7. Kumar M, Chandu GN, Shaffiulla Md. Oral health related quality of life (OHQoL) - views of first grade college students of Davangere, India. Journal of the Indian Association of Public Health Dentistry 2007; issue9: 7-12.
8. McGrath C, Bedi R, Gilthorpe. Oral health related quality of life - views of the public in the United Kingdom. Comm Dent Health 2000; 17: 3-7.

ORIGINAL RESEARCH ARTICLE

PERIODONTAL STATUS AND ITS RELATION TO BODY MASS INDEX AMONG UPPER PRIMARY AND SECONDARY SCHOOL PHYSICAL EDUCATION TEACHERS IN DAVANGERE CITY, INDIA

Authors:
Shwetha R[1], Subramaniam R[2], Sakeenabi B[3], Prashant GM[3], Chandu GN[4]

[1]Senior lecturer, Dept. of Public Health Dentistry, The Oxford Dental College 10th milestone, Bommanahalli, Hosur road, Bangalore 560 068, Karnataka, India.

[2]Senior Lecturer, Dept. of Public Health Dentistry, Indira Gandhi Institute of Dental sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala, India.

[3]Reader, Dept. of Preventive and Community Dentistry, College of Dental Sciences, Davangere 577 004, Karnataka, India.

[4]Professor, Dept. of Preventive and Community Dentistry, College of Dental Sciences, Davangere 577 004, Karnataka, India.

Address for correspondence: Dr. Shwetha R., Senior lecturer, Dept. of Public Health Dentistry, The Oxford Dental College, 10th milestone, Bommanahalli, Hosur road, Bangalore 560 068, Karnataka, India. E-mail: drshwetha_r@yahoo.co.in.

ABSTRACT

Background: Periodontitis is among the most common chronic disorders affecting the world population. Over the past few decades, obesity has also become a significant worldwide health problem. Being overweight and obese has been associated with an increased risk for periodontal disease. It has been suggested that obesity is second only to smoking as the strongest risk factor for inflammatory periodontal tissue destruction. The objective of this study was to determine body mass index, periodontal status and their relation, among upper primary and secondary school physical education teachers in Davangere city, India.

Methodology: A total of 109 physical education instructors from 87 upper primary and secondary schools were included in the study. Periodontal status was recorded using Community Periodontal index (CPI). Body Mass Index (BMI) was calculated as the ratio of the subject's body weight (in kg) to the square of their height (in meters).

Results: Of the 109 subjects examined, only 3.7% had healthy periodontal tissues. About 27.5% of the subjects had bleeding only. Presence of calculus was noted in 68.8% of the subjects. 74.3% of the subjects had periodontal pockets. Majority of the respondents had normal body mass index. Logistic regression analysis done to determine the association between BMI and periodontal status, revealed that there is statistically significant association between BMI and periodontal status.

Conclusion: The prevalence of periodontitis is high among the upper primary and secondary school physical education teachers in Davangere city. BMI was significantly associated with periodontal status indicating that a higher body mass index could be a potential risk factor for periodontitis.

Key words: Community Periodontal Index, Body Mass Index, physical education teachers, periodontitis.

J Odontol Res 2013;1(1): 25-31.

INTRODUCTION

Periodontitis is among the most common chronic disorders affecting the world population. It is initiated by gum colonization by pathogenic bacteria, followed by the activation of defense mechanisms. Nevertheless, the exact mechanism that describes the development of periodontitis is not yet elucidated. It is possible that many factors are associated with the development, progression and aggressiveness of the disease.[1] Periodontitis is now seen as resulting from a complex interplay of bacterial infection and host response, often modified by behavioral factors. Advances in research over recent years have led to a fundamental change in the periodontal disease model.[2]

It is evident from the scientific literature that general health has a considerable impact on oral health and vice versa. Many mediators have been postulated for this relationship, namely infection, chronic inflammation, and genetic predisposition. Apart from these mediators, nutrition has been postulated as an alternative mediator.[3]

Over the past few decades, obesity has become a significant worldwide health problem. Obesity is a complex multifactorial chronic disease that develops from an interaction of genotype and the environment. Overweight and obese adults are considered to be at high risk for various serious, life-threatening medical diseases, such as type 2 diabetes, hyperlipemia, hypertension, cholelithiasis, arteriosclerosis, cardiovascular, cerebrovascular diseases and endocrine and psychological disorders.[4,5]

Being overweight and obese also has been associated with an increased risk for periodontal disease. It has been suggested that obesity is second only to smoking as the strongest risk factor for inflammatory periodontal tissue destruction. According to current knowledge the adverse effect of obesity on the periodontium might be mediated through impaired glucose tolerance, dyslipidaemia or through increased levels of various bioactive substances secreted by adipose tissue such as serum resistin, leptin and adiponectin. Obesity enhances the risk of an individual exhibiting periodontitis, by increasing the numbers or proportions of pathogenic subgingival species.[4,6,7]

Obesity has significant association with periodontitis in terms of BMI, body fat and maximum oxygen consumption. Body Mass Index (also called as Quetelet index) which is a ratio of body weight (in kilograms) to body height (in meters) squared is highly correlated with fat mass, and morbidity and mortality therefore sufficiently reflects obesity related disease risk in a wide range of population. Numerous studies have concluded that individuals with high BMI are at an increased risk of deep periodontal pockets, loss of attachment and periodontal infection compared to individuals with normal BMI.[4-7] However, studies reporting the BMI of physical education instructors are sparse. Hence this study was conducted with the following objectives, to determine the body mass index of upper primary and secondary school physical education instructors in Davangere city, India, to determine their periodontal status and to investigate whether or not there is an association between body mass index, and periodontitis in healthy upper primary and secondary school Physical Education instructors.

METHODOLOGY

The target population was upper primary and secondary school teachers involved in physical education (PE) classes. The list of schools with PE teachers was obtained from the Block Education Officer of Davangere City. The schools in Davangere city are divided into six clusters. A total of 114 PE teachers from 87 upper primary and secondary schools from all the six clusters were included in the study.

The participants were informed about the objective of the study. Based on their approval, participants were asked to read carefully and sign a consent form.

Exclusion criteria for all subjects included: periodontal or antibiotic therapy in the previous 6 months; any systemic condition which might have influenced the course of periodontal disease or treatment (e.g. diabetes); any systemic condition which required antibiotic coverage for routine periodontal procedures. Five subjects were thus excluded from the study and the final sample size was 109.

Periodontal condition was assessed using the World Health Organization community periodontal index.

The CPI probe was used to measure the pocket depth. The subjects were examined by two post graduate students (Cohen's Kappa = 0.92) of Department of Preventive and Community Dentistry, College of Dental Sciences, Davangere. Periodontal status was recorded under five scores: score 0 (healthy), score 1 (bleeding), score 2 (calculus), score 3 (shallow periodontal pockets) and score 4 (deep periodontal pockets).

Body mass index was assessed as indicator of overall adiposity. Body mass index was computed as weight in kilograms divided by square height in meters. The subjects were classified into six groups (underweight, normal, pre-obese, obese class I, obese class II and obese class III) as recommended by the World Health Organization.

Ethical clearance for conducting the study was obtained from the Ethical Committee of the college.

Results were analyzed using the SPSS Version 17.0 software. Chi-square test was performed and the level of significance was set at p = 0.05. Multivariate logistic regression was conducted to determine the association between body mass index and the prevalence of periodontal disease after adjusting for important variables namely age, sex and smoking habit.

RESULTS

Table 1 presents the general profile of the study population. Of the 109 physical education teachers examined, 68.8% (n=75) were males and 31.2% (n=34) were females. 34.9% (n=38) were aged between 20 and 30 years and 37.6% (n=41) were aged between 31 and 40 years.

Majority of the subjects (63.3%, n=69) had normal BMI. About 6.4% (n=7) were underweight and 30.3% were overweight. Of the subjects with overweight, 25.7% (n=28) belonged to Pre-obese category. Only 1.8% (n=2) and 2.8% (n=3) of the subjects belonged to obese class I and obese class II respectively. Only 33.1% (n= 37) were smokers.

Of the 109 subjects examined, only 3.7% (n=4) had healthy periodontal tissues. About 27.5% of the subjects had bleeding only. Presence of calculus was noted in 68.8% of the subjects. (n=75). 52.3% (n=57) of the subjects had shallow pockets and 22% (n=24) of the subjects had deep periodontal pockets. (Table 2)

Chi square test used to analyze the significant relationship between CPI scores with the variables namely gender, age, smoking history and BMI revealed that there was statistically significant relationship of CPI scores with age group (p value = 0.035) and BMI (p value = 0.007).

Logistic regression analysis done to adjust the confounding variables (age group, sex and smoking history) in determining the association between BMI and periodontal status, revealed that there is statistically significant association between BMI and periodontal status. (Table 3)

DISCUSSION

The present survey included 109 physical education teachers from 87 Upper Primary and Secondary schools in Davangere City. Most of the physical education instructors were males (68.8%). Majority of the respondents were less than 40 years of age (72.5%).

The periodontal status, as indicated by the CPI scores reveals that only 3.7% of the subjects had healthy periodontal tissues. The overall prevalence of periodontal disease among the general population of India, belonging to the 35 - 44 years age group, as reported in National oral health survey and fluoride mapping is 89.6%. The higher prevalence of periodontal disease among the present study population can be attributed to a multitude of reasons like poor oral hygiene practices and lack of awareness regarding oral health. The negligence and lack of awareness towards oral health is reflected by the fact that almost a quarter of the subjects (22%) had deep periodontal pockets and more than half of the subjects (52.3%) had shallow pockets. Although no such study that determined the periodontal status of physical education teachers has been reported and the fact that the present study used a wide range of age (22-54 years), the results have been compared with studies conducted among similar age groups.

In a study conducted among adults Kesariyaji India, with age group 18-54 years, it was observed that although the percentage of subjects with healthy

TABLE 1: GENERAL PROFILE OF THE STUDY POPULATION

CHARACTERISTICS	PERCENTAGE OF SUBJECTS
GENDER	
MALE	68.8
FEMALE	31.2
AGE GROUP (IN YEARS)	
20 - 30	34.9
30 - 40	37.6
40 - 50	25.7
> 50	1.8
BODY MASS INDEX (BMI)	
UNDERWEIGHT (<18.5)	6.4
NORMAL (18.5 – 24.99)	63.3
PRE-OBESE (25 – 29.99)	25.7
OBESE CLASS I (30 – 34.99)	1.8
OBESE CLASS II (35 – 39.99)	2.8
OBESE CLASS III (> 40)	0
SMOKING HISTORY	
YES	33.1
NO	66.9

TABLE 2: PERIODONTAL STATUS OF THE SUBJECTS

CPI SCORE	PERCENTAGE OF SUBJECTS
0	3.7
1	27.5
2	16.5
3	30.3
4	22.0
SEXTANT – WISE SCORE	
CHARACTERISTICS	NO. OF SEXTANTS
Mean no. of sextants with healthy periodontal tissues	165
Mean no. of sextants with bleeding or higher score	489
Mean no. of sextants with calculus or higher score	243
Mean no. of sextants with shallow pockets or higher score	147
Mean no. of sextants with deep pockets or higher score	51

TABLE 3: MULTIVARIATE LOGISTIC REGRESSION ANALYSIS WITH CPI SCORE AS DEPENDENT VARIABLE

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta		
(Constant)	1.234	.576	-.018	2.140	.035
GENDER	-.046	.286	.171	-.162	.872
AGE GROUP	.250	.146	-.010	1.716	.089
SMOKING	-.025	.275	*.197*	-.090	.928
BMI	*.322*	*.160*		*2.010*	*.047*

periodontal tissues were similar, the prevalence of deep pockets was high (22%) in comparison with this study.[3]

The BMI status of the subjects reveals that majority of the subjects (63.3%) had normal BMI and of the 30.3% who were overweight, majority belonged to Pre-obese category. Only 1.8% (n=2) and 2.8% (n=3) of the subjects belonged to obese class I and obese class II respectively. This can be explained by the fact that the profession of the subjects demands the physical fitness and hence majority of the subjects having a normal BMI.

In the current study, obesity was positively related to periodontal status after adjusting for age, gender and smoking history. Studies that attempt to relate periodontal disease to the systemic status of an individual are often confronted with the issue that periodontal disease increases with age and most of the systemic conditions of interest such as diabetes, heart disease, stroke and obesity also increase in prevalence with age.[4] Subjects with systemic diseases were excluded from the study. After adjusting for age, gender and smoking status using a logistic regression model, BMI was still significantly associated with periodontal status.

Studies conducted by Bali et. al. in India and Ekuni et .al. in Japan [8,9] that have included either young or old subjects and data from those studies on have suggested that periodontal status deteriorates with BMI.

Numerous studies conducted by Santosh et. al, Haffajee et. al., Saitol et. al., Ylostalo et. al. and Khader YS et. al.[3-6, 10] among similar age groups have revealed significant positive association of periodontal disease with BMI. A study conducted by Johanne K et. al. in Copenhagen[11] has shown an inverse association between BMI and clinical attachment loss. The authors have explained that the observed inverse association could be due to the reason that individuals with high BMI had lost their periodontally affected teeth and thereby classified as periodontally healthy.

The underlying biological mechanisms for the association of obesity with periodontitis are not well-known; however, adipose-tissue-derived cytokines and hormones may play a key role.

Fat tissue produces a vast amount of cytokines and hormones, collectively called adipokines or adipocytokines which in turn may modulate periodontitis. Obesity increases the host's susceptibility by modulating the host's immune and inflammatory system, leaving the patient with greater risk of periodontitis. Plasminogen-activating system has been shown to play an important role in gingival inflammation. Plasminogen-activator inhibitor-1 (PAI-1) has an increased expression in visceral fat and induces agglutination of blood increasing the risk of ischaemic vascular disease. Thus, PAI-1 may also decrease the blood flow to the gingiva in obese people that encourage periodontitis progression. These are possibilities and studies are not yet conducted to reveal disease mechanisms.[12]

The association between smoking and the prevalence of periodontal disease was not statistically significant. Furthermore, there was no significant interaction between smoking and other variables on the occurrence of periodontal disease. This finding of the lack of association contradicts the findings of previous epidemiological, clinical, and in vitro studies that have provided irrefutable evidence that smoking negatively impacts periodontal health.

There were limitations to the present study that included subject selection and the cross-sectional nature of the data. Another limitation of this study is that as the periodontal status was assessed using the Community periodontal index, which does not include all the teeth and does not measure attachment loss.

Further studies with a larger sample size are required to confirm the association of body mass index and periodontal disease.

CONCLUSION

Majority of the physical education instructors in Davangere city had a normal body mass index. The prevalence of periodontitis is high among the upper primary and secondary school physical education teachers in Davangere city. After adjusting for age, gender and smoking status using a logistic regression model, BMI was significantly associated with periodontal status indicating that a higher body mass index could be a potential risk factor for periodontitis.

REFERENCES

1. Boesing F, Patiño JSR, da Silva VRG and Moreira EAM. The interface between obesity and periodontitis with emphasis on oxidative stress and inflammatory response. Obesity reviews 2009; 10: 290-7
2. Fenesy KE. Periodontal disease: an overview for physicians. Mt Sinai J Med.1998; 65: 362-9.
3. Santosh K, Rushabh JD, Chandrakant D, Parbhu D. Relationship of Body Mass Index with periodontal health status of green marble mine laborers in Kesariyaji, India. Braz Oral Res. 2009;23(4):365-9
4. Haffajee AD, Socransky SS. Relation of body mass index, periodontitis and Tannerella forsythia. J Clin Periodontol 2009; 36: 89–99.
5. Saitol T, Shimazakil Y, Kogal T, Tsuzuki M, and Ohshima A. Relationship between upper body obesity and periodontitis. J Dent Res 2001; 80(7): 1631-6.
6. Ylo¨stalo P, Suominen-Taipale L, Reunanen A, Knuuttila M. Association between body weight and periodontal infection. J Clin Periodontol 2008; 35: 297-304.
7. Pischon N, Heng N, Bernimoulin JP, Kleber BM, Willich SN, and Pischon T. Obesity, inflammation and periodontal disease. J Dent Res 2007; 86(5):400-9.
8. Bali RK, Mathur VB, Talwar PP, Chanana HB. National oral health survey and fluoride mapping 2002–2003. New Delhi: Dental Council of India; 2004.
9. Ekuni D, Yamamoto T, Koyama R, Tsuneishi M, Naito K, Tobe K. Relationship between body mass index and periodontitis in young Japanese adults. J PeriodontRes 2008; 43: 417–21.
10. Khader YS, Bawadi HA, Haroun TF, Alomari M, Tayyem RF. The association between periodontal disease and obesity among adults in Jordan. J Clin Periodontol 2009; 36: 18–24.
11. Kongstad J, Hvidtfeldt UA, Gronbaek M, Stoltze K, Holmstrup P. The relationship between body mass index and periodontitis in the Copenhagen heart study. J Periodontol 2009;80:1246-53.
12. Anne FR, Jane MR, Melissa S, Philippe H. Total body weight and waist circumference associated with chronic periodontitis among adolescents in the United States. Arch Pediatr Adolesc Med. 2006;160:894-9

ORIGINAL RESEARCH ARTICLE

KNOWLEDGE, ATTITUDE, AND PRACTICES TOWARDS DROPLET AND AIR-BORNE ISOLATION PRECAUTIONS AMONG DENTAL HEALTHCARE PERSONNEL IN A DENTAL COLLEGE IN KOTHAMANGALAM: A CROSS-SECTIONAL STUDY

ABSTRACT

Background: For long, infection control has been one of the major concerns of the dental community. Dentists use high-energy equipment, such as drills and scalers, in the presence of bodily fluids such as blood and saliva, and dental plaque. This combination has been shown to generate aerosols of oral micro-organisms, and blood. If infective aerosols persist there may be some danger of exposure in the waiting area and for subsequent patients. With this background, the study was conducted to assess the knowledge, attitude and practice towards droplet and air-borne isolation precautions among dental healthcare personnel in a dental college in Kothamangalam, Kerala.

Methods: A cross-sectional questionnaire based survey containing 23 questions to assess the knowledge, attitude and practice on airborne infections isolation precautions. The samples were the teaching faculty members and students of a dental college in Kothamangalam, Kerala. Results were expressed as a number and percentage of respondents for each question and Chi-square test was performed for inferential statistical analysis.

Results: The mean knowledge, attitude and practice scores were 3.94±1.029, 5.17±0.862, 3.62±1.596 respectively with maximum scores of 8, 6 and 7. Significant differences existed in practice scores among faculty and students.

Conclusion: The study revealed that although the attitude regarding droplet and airborne isolation precautions among faculty members and students of the institution was high, knowledge and practice remained low.

Key words: airborne infections, isolation precautions, droplet, aerosol.

J Odontol Res 2013;1(1):30-6.

Authors:

Sanjeev R[1],
Suneesh Kuruvilla[2],
Subramaniam R[2]

[1]Reader and HOD,
Dept. of Public Health Dentistry,
Indira Gandhi institute of Dental sciences, Nellikuzhy P. O.,
Kothamangalam 686 691,
Kerala, India.

[2]Senior Lecturer, Dept. of Public Health Dentistry, Indira Gandhi Institute of Dental sciences,
Nellikuzhy P. O.,
Kothamangalam 686 691, Kerala, India.

Address for correspondence:
Dr. Subramaniam R.,
Senior lecturer,
Dept. of Public Health Dentistry,
Indira Gandhi Institute of Dental Sciences, Nellikuzhy P. O.,
Kothamangalam 686 691,
Kerala, India.
E-mail: subbds@gmail.com.

INTRODUCTION

For long, infection control has been one of the major concerns of the dental community.[1] Dental healthcare workers (DHCWs) are at a high risk of cross-infection due to frequent exposure to microorganisms living in patients' blood, droplets of saliva and instruments contaminated with blood, saliva and tissue debris.[2] Transmission of infection during dental treatment or surgery can occur through several routes: direct contact with blood, saliva or tissue debris; indirect contact with contaminated instruments or surfaces that have been improperly sterilized; or contact with infective agents present in either the droplets or aerosol particles from saliva and respiratory fluids.[3]

Dentists use high-energy equipment, such as drills and scalers, in the presence of bodily fluids such as blood and saliva, and dental plaque. This combination has been shown to generate aerosols of oral micro-organisms, and blood. However, when patients harbour viruses, either blood-borne aerosol generation may prove a significant health hazard to dentists and their assistants. [4] If infective aerosols persist there may be some danger of exposure in the waiting area and for subsequent patients.[4] Thus diseases like pneumonia, influenza, hepatitis and skin and eye infections may be transmitted during dental treatment procedures from these infective bioaerosols.[1]

Aerosols are particles less than 5 micrometers in diameter. The smaller particles of aerosols have the potential to penetrate and lodge in small passages of lung are thought to carry the greatest potential for transmitting infections. Airborne transposition refers to situations where droplet nuclei or dust particles containing microorganisms can remain pendulant in air for long periods of time.[5]

With a high prevalence of tuberculosis reported in India, and diseases such as hepatitis B and acquired immune deficiency syndrome posing major threats for the dental personnel, sound knowledge regarding airborne infections and isolation precautions becomes mandatory among the dental health-care personnel.

With this background, the study was conducted to assess the knowledge, attitude and practice towards droplet and air-borne isolation precautions among dental healthcare personnel in a dental college in Kothamangalam, Kerala

METHODOLOGY

The study was a cross-sectional questionnaire based survey. The target population was the dental students with clinical exposure (the third and final year undergraduate students) and the teaching faculty members of a dental college in Kothamangalam. A prefabricated validity tested questionnaire was administered to the target population. The questionnaire was divided into two parts. The first part consisted of questions on personal and professional data including age, gender, qualification, experience and type of practice. The second part contained 23 questions on assessment of knowledge, attitude and practice regarding droplet and air-borne isolation precautions. All questions in the questionnaire were close-ended.

The questionnaires were distributed by the faculty members of Department of Public Health Dentistry and dental students posted in the department. The respondents were asked to return the questionnaire immediately.

All returned questionnaires were coded and analyzed. Results were expressed as a number and percentage of respondents for each question and were analyzed using the SPSS Version 17 software. Chi-square test was performed to assess the significant difference in response (if any) in relation to the independent variables such as gender, experience, qualification, year of study (for students) and type of practice (for faculty). The level of significance was set at $p = 0.05$.

RESULTS

Respondent's profile

Among the 111 respondents, 30.6% (n=34) were males and the rest 69.4% (n=77) were females. Seventy six percent (n=85) were dental students and the rest 24% (n=26) were teaching faculty members. Among the faculty members, 73.1% (n=19) had private practice in addition to institution based practice.

Table I:
Response to knowledge based questions on droplet and airborne isolation precautions

QUESTION	RESPONSE	n	%	Sig.
Droplet infections are airborne contamination with aerosols or splatter of respiratory fluid.	Yes	110	99.1	NS
	No	1	.9	
Are you aware of the fact that droplets/aerosols produced during routine dental treatment causes life threatening infection?	Yes	107	96.4	NS
	No	4	3.6	
If yes, which infection do u think will spread through droplets/aerosols?	Hepatitis B	9	8.1	NS
	HIV	2	1.8	
	SARS and TB	74	66.7	
	All of the above	26	23.4	
Droplet Nuclei is of the order of	1-5 µ	16	14.4	NS
	50-75µ	4	3.6	
	25-50µ	12	10.8	
	75-100µ	79	71.2	
How far do you think the aerosols during dental treatment can spread or disseminate?	0 cm.	5	4.5	NS
	50 cms.	12	10.8	
	100 cms.	15	13.5	
	150 cms.	14	12.6	
	200 cms.	65	58.6	
Which of the following procedures are known to produce maximum air-borne contamination?	Ultrasonic scalers	53	47.7	NS
	Air polishing	8	7.2	
	Air water syringe	13	11.7	
	Tooth preparation using aerotor	37	33.3	
Are you aware of the existence of a specific guideline for infection control in dental setting.	Yes	94	84.7	NS
	No	17	15.3	
Are you aware of N95 mouth masks?	Yes	15	13.5	NS
	No	94	84.7	
	Yes, I use it	2	1.8	

NS - Not Significant

Assessment of knowledge, attitude and practice on droplet and airborne isolation precautions

The results obtained have been summarized and tabulated. Table I, II and III show the response to knowledge, attitude and practice based questions on droplet and airborne isolation precautions respectively. Table IV shows mean and standard deviation (SD) score of knowledge, attitude, and practice of droplet and airborne isolation precautions among the study subjects in relation to educational qualification.

Table II:
Response to attitude based questions on droplet and airborne isolation precautions

QUESTION	RESPONSE	n	%	Sig.
If you know that your patient is suffering from disease that can potentially spread through droplets/aerosols would you like to treat that patient?	Yes	83	74.8	NS
	No	27	24.3	
If you know that your patient is suffering from a disease that can potentially spread through droplets/aerosols, when will you give him appointment?	Any time, patient's convenience	20	18.0	NS
	Any time, your convenience	13	11.7	
	First	3	2.7	
	Last	75	67.6	
If you know your patient is suffering from infection that spreads through droplets will you isolate the patient?	Yes	98	88.3	NS
	No	13	11.7	
Do you feel that droplet infection control in dental practice should compulsorily be a part of the dental undergraduate curriculum?	Yes	108	97.3	NS
	No	3	2.7	
Do you think your knowledge regarding droplet infection control in dental practice is adequate?	Yes	12	10.8	NS
	No	99	89.2	
Do you think you need any further training on droplet infection control in dental practice?	Yes	110	99.1	NS
	No	1	.9	

NS - Not Significant

DISCUSSION

Most dental treatment procedures have the potential for creating contaminated aerosols and splatter.[6] The importance of preventing airborne transmission of microorganisms in a dental setting and the risk of cross-infections between patients and DHPs (Dental health professionals) is well documented. It is therefore critical for practicing professionals to adapt proper infection control measures to protect both themselves and their patients.[7]

Knowledge, attitude and practice act as three pillars, which make up the dynamic system of life itself. Knowledge is some information that is acquired or gained. It results in congeniality and advertence about an eclectic thing or a situation. Knowledge, being the basic criterion that allows one to earmark between the right and the wrong, is a mixture of comprehension, experience, discernment and skill. Attitude accredits to thinking towards a proper situation. Practice means contemplation of rules and

QUESTION	RESPONSE	n	%	Sig.
Do you follow any specific guidelines for infection control in your routine practice	Yes	89	80.2	NS
	No	22	20.2	
Do you use mouth mask when you work on patients	Always	95	85.6	NS
	Often	12	10.8	
	Sometime	3	2.7	
	Rarely	1	.9	
	Never	-	-	
Do you change your mouth mask after every patient	Always	49	44.1	NS
	Often	26	23.4	
	Sometime	24	21.6	
	Rarely	8	7.2	
	Never	4	3.6	
Do you wear eye protection when you work on patients	Always	43	38.7	S (Signifcant difference in response among faculty and students) p=0.04
	Often	32	28.8	
	Sometime	21	18.9	
	Rarely	2	1.8	
	Never	13	11.7	
Do you clean/disinfect your eye protection after every patient	Always	18	16.2	NS
	Often	12	10.8	
	Sometime	19	17.1	
	Rarely	30	27.0	
	Never	32	28.8	
Do you wear protective clothing when you work on the patient	Always	83	74.8	S (Signifcant difference in response among faculty and students) p=0.036
	Often	7	6.3	
	Sometime	7	6.3	
	Rarely	2	1.8	
	Never	12	10.8	
Do you practice pre-procedural mouth rinsing in all your patients?	Yes	19	17.1	NS
	No	92	82.9	

NS - Not Significant, S - Significant

knowledge that lead to action. Thus, a right knowledge, a positive attitude and a good practice are imperative to guide and serve the patients.[5] Thus, this study was conducted with the objective of assessing the knowledge, attitude and practice regarding air-borne infections and isolation precautions.

Although 99 percent of the respondents were aware of what droplet were and about 96 percent believing that these aerosols could cause life threatening infections, the knowledge regarding the infections that spread through droplets, size of the droplet nuclei, distance up to which aerosols can spread through droplets during dental treatment were inad-

Table IV:
Mean and standard deviation (SD) score of knowledge, attitude, and practice of droplet and airborne isolation precautions among the study subjects in relation to educational qualification.

EDUCATION	KNOWLEDGE (Mean ± SD) Max. score = 8	ATTITUDE (Mean ± SD) Max. score = 6	PRACTICE (Mean ± SD) Max. score = 7
Students	3.92 ±1.003	5.16±0.871	4.00±1.318
Graduates	4.00±0.707	5.00±1.225	2.20±1.643
Post Graduates	4.00±1.225	5.24±0.768	2.43±1.886
TOTAL	3.94±1.029	5.17±0.862	3.62±1.596

equate. However vast majority of the respondents were aware of the existence of a specific guideline for infection control in dental setting. This indicate that majority of the respondents although not aware about the theoretical aspects regarding droplets, the respondents including students were aware of the risks of aerosol. The results are in accordance with studies conducted in Udaipur[5] and Manipal[7].

Considering the attitude related questions, it was found that a vast majority of respondents including students (74.8%, n=83) were ready to treat a patient with known history of respiratory disease, with about 67.6% agreeing that they would schedule the appointments of such patients at as last patients of the day. Almost 89% of the respondents opined that patients with infections that spread through droplets should be isolated. Moreover 97.3% of the respondents felt that droplet infection control should compulsorily be made a part of the undergraduate curriculum.

These responses indicate a positive attitude of the respondents on droplet infection control measures. The results are similar to a study conducted by Ramesh et al. in Bangalore and Chennai where it was found that the dentists had a good attitude towards the same.[8]

The respondent's lack of knowledge on this topic of interest is reflected in the response to the question regarding their adequacy of knowledge regarding the subject. Almost 90% of the respondents admitted their knowledge being adequate. Furthermore, about 99% felt they require further training in droplet infection control. These responses throw a light on the lack of proper training on airborne infections isolation precautions as a part of the dental curriculum. Inferential statistics further revealed that the observation is not just limited to the students but among the teaching faculty members too.

The questions on practice rather presented a favourable picture. Almost 90% of the respondents opined that they followed specific guidelines for infection control in routine practice. A high proportion of respondents admitted the use of personal protective equipments such as mouth masks and protective clothing while working on patients. However there was a significant difference among students and faculty members wherein students wore protective clothing more than faculty members. Nevertheless, regarding the use of eye protection, while most of the student respondents admitted its usage, this practice was significantly lower among the faculty members. These observations might be due to the fact that the students in the institution are strictly instructed to observe the universal precautions while working on patients.

On the contrary, the study revealed that the practice of pre-procedural mouth-rinse was very low. The personal protective equipments although act as barriers, these are not enough to totally eliminate the

risk of transmission of microorganisms. The current literature suggests that having patients use antimicrobial rinse before treatment may significantly reduce the microbial aerosols. Hence pre-procedural mouth rinsing should compulsorily be practiced before treating the patients.

One of the limitations to this study was the method for assessing the practice of the precaution. We could not supervise the responders' practice and, therefore, had to rely on their subjective self-assessment. Therefore, the responses might have not accurately reflected the true knowledge and attitude in practice and, therefore, the reported level of practice might be even lower than the real level.[9]

CONCLUSION

The study revealed that although the attitude regarding droplet and airborne isolation precautions among faculty members and students of the institution was high, knowledge and practice remained low. This study indicated that there is a need for creating awareness among dentists regarding droplet and airborne isolation precautions. The topic should compulsorily be made a part of the dental undergraduate curriculum. Continuing dental education programs are yet another effective method in imparting awareness among the dental practitioners.

REFERENCES

1. Shivakumar KM, Prashant GM, Madhu Shankari GS, Subba Reddy VV, Chandu GN. Assessment of atmospheric microbial contamination in a mobile dental unit. Indian J Dent Res 2007;18(4):177-180.
2. Chun Chen, Bin Zhao, Wellin Cul, Lel Dong, Na An, Xlangying Ouyang. The effectiveness of an air cleaner in controlling droplet/aerosol particle dispersion emitted from a patient's mouth in the indoor environment of dental clinics. J R Soc Interface 2010;7:1105-18.
3. Center for disease control and prevention. Guidelines for preventing transmission of mycobacterium tuberculosis in Health-Care Settings, 2005. *MMWR* 2005; *54(17):* 1-141.
4. Bennett AM, Fulford MR, Walker JT, Bradshaw DJ, Martin MV, Marsh PD. Microbial aerosols in general dental practice. Br Dent J 2000;189(12):664-7
5. Jain M, Sawla L, Mathur A, Nihlani T, Ayair U, Prabu D, Kulkarni S. Knowledge, Attitude and Practice towards droplet and airborne isolation precautions amongs dental health care professionals in India. Med Oral Patol Oral Cir Bucal. 2010 Nov 1;15 (6):e957-61.
6. Lucia B, Luminita SI, Manuela LM, Ioan D, Dana B. *Airborne Microbial Contamination in Dental Practices in Iasi, Romania. OHDMBSC - Vol. IX - No. 1 - March, 2010*
7. Abhinav S, Bharathi P, Peter S, Shashidhar A. Knowledge and attitude towards droplet and airborne isolation precautions and it's correlation among Indian dental students. Int J Infect Control 2011, v7:i1
8. Ramesh N, Anuradha KP. A Survey on infection control knowledge, attitude and practice among Bangalore and Chennai dentists. *Journal of Indian Dental Association* 2000; *71:* 116-117.
9. Askarian M, Assadian O. Infection control practices among dental professionals in Shiraz Dentistry School, Iran. Arch Iranian Med 2009;12(1):48-51.

REVIEW ARTICLE

ORAL CANCER - A JOURNEY TO UNRAVEL THE PAST

Authors:
Jithin Jose[1],
Niveditha Baiju [2],
Pramod Mathews[3],
Skariah K S[1]

[1]Senior lecturer,
Dept. of Oral Pathology & Micobiology,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

[2]Professor & HOD,
Dept. of Oral Pathology & Micobiology,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

[3]Professor,
Dept. of Oral Pathology & Micobiology,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

Address for correspondence:
Dr. Jithin Jose, Senior lecturer,
Dept. of Oral Pathology & Micobiology,
Indira Gandhi institute of Dental Sciences,
Kothamangalam 686 691,
Kerala, India.
E-mail: jitinjos@gmail.com

ABSTRACT

Oral squamous cell carcinoma (OSCC) is the most common malignant tumour in the oral and maxillofacial region. It is the sixth most common malignancy and is a major cause of cancer morbidity and mortality. OSCC accounts for 95% of malignant lesions of the mouth and is a major problem worldwide. The relative prevalence of oral SCC is 3–5% of all cancer. The purpose of this article is an attempt to review a brief frame work about epidemiology, etiology, pathogenesis, clinical presentation, staging and grading of Oral cancer.

Key words: oral cancer, staging, grading

J Odontol Res 2013;1(1):37-44.

INTRODUCTION

The term 'cancer' has originated from Greek word Karkinos, a crab, referring to an irregular jagged shape often assumed due to local spread of carcinoma.[(1)] Oral cancer is an epithelial neoplasia generally beginning as a focal clonal overgrowth of altered stem cells near the basement membrane, expanding upward and laterally, replacing the normal epithelium. The neoplastic process is a beginning with normal epithelium progressing through hyperplasia to dysplasia to carcinoma in situ and invasive carcinoma.[(2)] The term 'oral cancer' includes a diverse group of tumors arising from the oral cavity, usually includes cancers of the lip, tongue, pharynx and oral cavity.[(3)]

EPIDEMOLOGY

Globally, the varied incidence rates of oral cancer (per 100,000 cases) are seen ranging from 2.0 (UK) to 9.4 (France); 4.4 in Colombia to 13.4 in Canada; 1.6 Japan to 13.5 India; and from 2.6 New Zealand to 7.5 in South Australia. Each year, about 5, 75,000 new cases and 3,20,000 deaths occur world-wide.[4] The estimated new cancer cases in 2007 worldwide will be more than 12 million, 6.7 million will occur in economically developing countries, of which 4.7 million will result in death. On an average about 8-8.5% men and 4-8.1% women could develop oral cancer in their lifetime in developing countries.[5] Some countries with the highest incidence rates for oral cancer in the world are located in the region of South Asia. India has always been cited as the country with the highest incidence in the world, though in some recent reports Sri Lanka and Pakistan are ranked at the top. In India alone over 100,000 cases are registered every year. According to cancer incidence in five continents one district of India (Bhopal) has the highest incidence for cancers of both the tongue (10.9 per 100,000) and mouth (9.6 per 100,000) in the world.[6] It is estimated that more than one million new cases are being detected annually in the Indian subcontinent out of which 92-95% of all oral malignancies are oral squamous cell carcinoma (OSCC).[7]

ETIOLOGY

Oral squamous cell carcinoma is an age related disease, with about 90% to 95% of cases occurring in persons above the age of 40 years.[8] The etiology of OSSC is multifactorial. Genetic, environment, social and behavioral effects may all be implicated. Tobacco is by far the main risk factor for OSCC and this applies to not only smoke but also to smokeless tobacco.[9] The other risk factor for cancer is alcohol. Alcohol is oxidized to acetaldehyde and may act as a solvent and enhance the penetration of carcinogens into target tissues.[10] There is a significant joint multiplicative risk for OSCC in people who are both alcohol drinkers and heavy tobacco smokers.[11] There is also a relationship of OSCC with betel nut. The International agency for research on cancer (IARC) long ago declared that betel nut was carcinogenic to humans and that has been confirmed.[12] There is a relationship with HPV 16, HPV 18 and OSCC, particularly oropharyngeal carcinoma.[13] The dentition may also play a role in OSCC. Head and neck and oesophageal dysplasia/cancer has been found associated with dental neglect.[14] Periodontal diseases has been implicated in OSCC.[15] Other conditions associated with an increased risk of OSCC includes diabetes[16] and candidal infection.[17] Diet may play a role in OSCC development as evidenced by multiple epidemiological studies worldwide.[18] Apart from the dietary risk factors for OSCC such as alcohol and other factors, studies show that consumption of fruits and vegetables will have a protective effect.[19] Thus, environmental insults such as alcohol and/or tobacco products presumably increase DNA damage, increase p53 expression, and thereby activate clusters of genes associated with cell growth, and/or cell death.[20]

PATHOGENESIS

Tumorigenic genetic alterations consist of two major types: tumor suppressor genes, which promote tumor development when inactivated; and oncogenes, which promote tumor development when activated. Tumor suppressor genes can be inactivated through genetic events such as mutation, loss of heterozygosity, or deletion, or by epigenetic modifications such as DNA methylation or chromatin remodeling. Oncogenes can be activated through overexpression due to gene amplification, increased transcription, or changes in structure due to mutations that lead to increased transforming activity.[21] The cell of origin of OSCC is the oral keratinocyte. OSCC, as any cancer, is caused by DNA mutation, often spontaneous but increased by exposure to any of a range of mutagens like chemical, physical or microbial. The various changes in the DNA can progress from a normal keratinocyte to a pre-malignant or a potentially malignant keratinocyte that is characterized by an ability to proliferate in a less controlled fashion than normal. The cells become autonomous and a true cancer results, characterized by invasion across the epithelial basement membrane and, ultimately, metastasis to lymph nodes, bone, brain, liver and other sites.[22]

CLINICAL PRESENTATION

Symptoms: Pain is a common symptom in oral cancer patients, representing 30–40% of their main complaints. Although pain is the main symptom, it usually arises only when the lesions have reached a remarkable size, thus, early carcinomas often go unnoticed because they are asymptomatic. Other symptoms include ear pain, bleeding, and mobility of teeth, problems in breathing, difficulty in speech, dysphag, and problems using prosthesis, trismus, and paraesthesia. Occasionally patients may present with cervical lymphadenopathy without any other symptoms. In terminal stages, patients may develop skin fistulas, bleeding, severe anaemia and cachexia.[23]

Oral carcinoma presents with varied clinical features as follows:

- Exophytic (mass-forming, fungating, papillary, verruciform)
- Endophytic (invasive, burrowing, ulcerated)
- Leukoplakia (white patch)
- Erythroplakia (red patch)
- Erythroleukoplakia (combined red and white patch).[24]

Initial stages: The clinical presentation of these early malignant lesions is usually in the form of an erytholeukoplastic lesion. It consists of red or red and white areas with a slight roughness and is well-demarcated.

Advanced stages: The classic features of oral malignancy include ulceration, nodularity and fixation to underlying tissues.

Ulceration: This is one of the most common and best known features of OSCC. The ulceration has an irregular floor and margins, and is elevated and hard on palpation. When the lesion is large the patient often has severe pain, radiating from the lesion to the ipsilateral ear.

Lump: In these advanced stages, exophytic tumours with warty surfaces, poorly defined boundaries and hard to palpation may be seen.

Less common presentations: OSCC may manifest with paraesthesia or numbness of the chin. Others manifest with delayed healing after a dental extraction, or sometimes a lump with abnormal supplying blood vessels, dysphagia or weight loss.[23]

TNM STAGING

The stage of the disease depends on several factors, including the size of the primary lesion, local extension, lymph node involvement, and evidence of distant metastasis. Tumor size, the organ or tissue affected, and the extent of spread are considered to be the best indicators of the patient's prognosis. (TNM) classification of oral cancer has 3 basic clinical features: the size (in centimeters) of the primary tumor; the presence, number, size, and spread (unilateral or bilateral) to the local lymph nodes; and the presence or absence of distant metastasis. Among the commonly applied schemes in oncology are the classifications proposed by the International Union against Cancer (UICC) and the American Joint Committee on Cancer Society (AJCCS).[25]

The *tumor-node-metastasis (TNM)* staging system was first reported by Pierre Denoix in the 1940s. The *International Union against Cancer (UICC)* eventually adapted the system and compiled the first edition of the TNM staging system in 1968 later it was modified in *1974* and was given as follows.[26]

T – Extent of primary tumor

N – Condition of regional lymph nodes

M – Absence or presence of distant metastasis

T – Primary Tumor

TIS – Carcinoma in situ

T1 – Tumor 2 cm or less in greatest diameter

T2 – Tumor greater than 2 cm but not greater than 4 cm in greatest diameter

T3 – Tumor greater than 4 cm in greatest diameter

N – Regional lymph nodes

N0 – No clinically palpable cervical lymph nodes; or palpable nodes but metastasis not suspected

N1 – Clinically palpable homolateral cervical lymph nodes that are not fixed; metastasis suspected

N2 – Clinically palpable contralateral/ bilateral cervical lymph nodes that are not fixed; metastasis suspected

N3 – Clinically palpable lymph nodes that are fixed; metastasis suspected

M – Distant metastases

M0 – No distant metastases

M1 – Clinical and/or radiographic evidence of metastases other than to cervical lymph nodes

Clinical stage – Grouping of carcinoma of oral cavity: [27]

Stage I: T1 N0M0

Stage II: T2N0M0

Stage III: T3N0M0

T1N1M0

T2N1M0

T3N1M0

Stage IV: T1 N2M0 T1 N3M0 or any T or N category with M1

T2N2M0 T2N3M0

T3N2M0 T3N3M0

REVIEW OF HISTOPATHOLOGIC MALIGNANCY GRADING IN ORAL SQUAMOUS CELL CARCINOMA

Histologic grading has been used for many years in an attempt to predict them clinical behavior of squamous cell carcinomas in the head and neck region. Histopathologic evaluation of the degree to which the tumor e.g. oral squamous cell carcinoma resembles its parent tissue (squamous epithelium) and produces its normal product (keratin) is called *grading.* The first level of histological assessment of OSCC is usually the grading of the tumor using the method based on the degree of differentiation introduced by Broders in 1920. It takes into account the degree of keratinization, cellular and nuclear pleomorphism and mitotic activity. Tumors are generally divided into three categories: grade 1 (well differentiated), grade 2 (moderately differentiated) and grade 3 (poorly differentiated). [28]

Broders A.C. system was based on the proportion of differentiated cells to undifferentiated or anaplastic cells. Broders classification with modification is. [29]

Grade I: Well differentiated tumors – 75-100% of cells are differentiated

Grade II: Moderately differentiated tumors – 50-75% of cells are differentiated

Grade III: Poorly differentiated tumors – 25-50% of cells are differentiated

Grade IV: Anaplastic tumor – 0-25% of cells are differentiated

A reported lack of correlation between Broder's grade and prognosis of head and neck squamous cell carcinomas has been explained by the fact that squamous cell carcinomas usually exhibit a heterogenous cell population with probable differences in invasive and metastatic behavior. A new grading system of head and neck squamous cell carcinomas was originally introduced by Jakobsson et al. (*1973*) which includes the morphologic parameters "structure", "tendency to keratinization", "nuclear aberrations", and "number of mitosis", but also an evaluation of tumor-host relationship as estimated by parameters such as "mode," "stage of invasion", "vascular invasion" and "degree of lymphoplasmocytic infiltration".

ANNEROTH et al (1987) histologic grading system: [30]

It was based on Jakobsson et al. system for application to squamous cell carcinoma in the tongue and floor of mouth. One of the parameters, "vascular invasion" was omitted. Statistical analysis revealed that the reproducibility of the system was good for all morphologic variables. Mean total malignancy, tumor population and tumor-host relationship scores showed statistically significant correlation with mean rating for all the different

Jakobbson et al (1973) histologic grading system: [4]

Histologic grading of malignancy based on tumour cell population				
Tumor Cell Population	1	2	3	4
Structure	Papillary and solid	Strands	Small cords and groups of cells	Marked cellular dissociation
Differentiation	Highly; Keratinization	Moderately; some keratinization	Poorly; minimum keratinization	Poorly; no keratinization
Nuclear polymorphism	Few enlarged nuclei	Moderate number of enlarged nuclei	Numerous	Anaplastic immature enlarged nuclei
Mitoses	Single	Moderate number	Great number	Numerous
Histologic grading of malignancy based on tumor-host relationship				
	1	2	3	4
Mode of invasion	Well-defined borderline	Cords, less marked borderline	Groups of cells, no distinct borderline	Diffuse growth
Stage of invasion	Possibl	Microcarcinoma (few cords)	Nodular, into connective tissue	Massive
Vascular invasion	None	Possibly	Few	Numerous
Cellular response (plasma-lymphocytic infiltration)	Marked	Moderate	Slight	None

Anneroth et al (1987) histologic grading system:

Histologic grading of malignancy of tumor cell population				
Morphologic Parameters	1	2	3	4
Degree of keratinization	Highly keratinized (>50% of the cells)	Moderately keratinized (5-20% of the cells	Minimal keratinization (5-20% of the cells	No keratinization (0-5%)
Nuclear polymorphism	Little nuclear polymorphism (>75% mature cells)	Moderately abundant nuclear polymorphism (50-75% mature cells)	Abundant nuclear polymorphism (25-50% mature cells)	Extreme nuclear polymorphism (0-25% mature cells)
Number of mitoses/HPF	0-1	2-3	4-5	>5

Histologic grading of malignancy of tumor-host relationship				
Morphologic Parameters	1	2	3	4
Pattern of invasion	Pushing, well delineated infiltrating borders	Infiltrating, solid cords, bands and or strands	Small groups or cords of infiltrating cells (n>15)	Marked and widespread cellular dissociation in small groups of cells (n<15) and/or in single cells
Stage of invasion (Depth)	Carcinoma in situ /or Questionable invasion	Distinct invasion, involving lamina propria only	Invasion below lamina propria adjacent to muscles, salivary gland tissues and periosteum	Extensive and deep invasion replacing most of the stromal tissue and infiltrating jaw bone
Lympho-plasmacytic infiltrate	Marked	Moderate	Slight	None

Bryne's (1989, 1992) (ITF) Invasive Tumor Front Grading System:

Morphologic Feature	1	2	3	4
Degree of keratinization	Highly keratinized (>50% of the cells)	Moderately keratinized (5-20% of the cells)	Minimal keratinization (5-20% of the cells)	No keratinization (0-5%)
Nuclear polymorphism	Little nuclear polymorphism (>75% mature cells)	Moderately abundant nuclear polymorphism (50-75% mature cells)	Abundant nuclear polymorphism (25-50% mature cells)	Extreme nuclear polymorphism (0-25% mature cells
Number of mitoses (high - power field)	0-1	2-3	4-5	>5
Pattern of invasion	Pushing, well delineated infiltrating borders	Infiltrating, solid cords, bands and or strands	Small groups or cords of infiltrating cells (n > 15)	Marked and widespread Cellular dissociation in small groups of cells(n<15) and or in single cells
Host response (lympho-plasmacytic infiltrate)	Marked	Moderate	Slight	None

morphologic parameters with certain specified exceptions.

BRYNE'S (1989, 1992) (ITF) Invasive tumor front grading system: [4]

Bryne M. (1998) presented a hypothesis suggesting that molecular and morphological characteristics at the invasive front area of various squamous cell carcinomas may reflect tumor prognosis better than other parts of the tumor. He further states that several molecular events of importance for tumor spread like gains and losses of adhesion molecules, secretion of proteolytic enzymes, increased cell proliferation and initiation of angiogenesis occur at the tumor host interface. Consequently, they have developed a simple morphological malignancy grading system that restricts the evaluation to the deep invasive front of the tumor. Several studies have shown that this system is a significantly better predictor of prognosis.

CONCLUSION

Oral cancer constitutes the most life threatening of all dental conditions. Oral cancer has a prolonged natural history with premalignant and early invasive phases. The etiological factors are well outlined. There is a long natural history of development of the disease running in to several years, cases treated in localized stages of the disease are completely curable and for the incurable persons pain relief and palliative care can be offered with considerable improvement of the quality of life. Early diagnosis is critically important to improve survival and to reduce morbidity and mortality associated with oral cancer. The treatment outcome of this disease is much better if detected at an early stage.

REFERENCES

1. Garcia M, Jemal A, Ward EM, Center MM, Hao Y, Siegel RL, Thun MJ. Global Cancer Facts & Figures 2007. Atlanta, GA American Cancer Society;2007:1-20.
2. Jatin K, Nagpala, Bibhu R Das. Oral cancer: reviewing the present understanding of its molecular mechanism and exploring the future directions for its effective management. Oral Oncology 39;(2003):213-21.

3. DTW Wong, R Todd, T Tsuji, RB Donoff. Molecular biology of human oral cancer. Crit Rev Oral Biol Med 1996;7(4):319-28.

4. Ankur Bhargava, Sonal Saigal, Monali Chalishazar. Histopathological Grading Systems in Oral Squamous Cell Carcinoma: A Review. Journal of international oral health JIOH, Volume 2 (Issue 4);December 2010:1-10.

5. Antony George, Sreenivasan BS, Jubin Thomas, Devi Gopakumar. Potentially malignant disorders of oral cavity. Oral & Maxillofacial Pathology Journal [OMPJ] Vol 2.No 1;Jan-Jun 2011:95-100.

6. Saman Warnakulasuriya. Review Global epidemiology of oral and oropharyngeal cancer. Oral Oncology45;2009:309-16.

7. SR Moore, NW Johnson, IS Pierce, DF Wilson. The epidemiology of mouth cancer: a review of global Incidence: REVIEW .Oral Oncology Oral Diseases 2000;6:65-74.

8. Howell R E, Wright B A, Dewar R. Trends in the incidence of oral cancer in novascotia from 1983 to 1997. Oral Surg Oral Med Oral Path oral Radiol Endol. 2003; 95: 205-10.

9. Johnson N. Tobacco use and oral cancer: a global perspective. J Dent Educ. 2001; 65: 328-39.

10. J. Reidy, E. McHugh, L.F.A Stassen. A review of the relationship between alcohol and oral cancer the surgeon 9; 2011:278-83.

11. Hashibe M, Brennan P, Chuang S C et al. Interaction between tobacco and alcohol use and risk of head and neck cancer: pooled analysis in International head and neck cancer epidemiology consortium. Cancer Epidemiol Biomarkers Prev. 2009;18:541-550.

12. Chen Y J, Chang J T, Liao C T et al. Head and neck cancer in betel quid chewing area:recent advances in molecular carcinogenesis. Cancer Sci. 2008;99: 1507-14.

13. D'Souza G, Kreimer A R, Viscidi R et al. Case-control study of human papilloma virus and oropharyngeal cancer. N Eng J Med. 2007;356:1944-56.

14. Dye B A, Wang R, Lashley R. using nhanes oral health examination protocols as a part of an esophageal cancer screening study conducted in a high risk region of china. BMC Oral Health 2007; 17:7-10.

15. Meyer MS, Joshipura K, Giovannucci E. A review of the relationship between tooth loss, periodontal disease and cancer. Cancer Causes Control. 2008;19:895-907.

16. Dikshit R P, Ramadas K, Hashibe M. Association between diabetes mellitus and pre-malignant oral diseases: a cross sectional study in Kerala, India. Int J Cancer,2006;118:453-7.

17. Marina Mohd Bakri, Haizal Mohd Hussaini, Ann Rachel Holmes, Richard David Cannon and Alison Mary Rich. Revisiting the association between candidal infection and carcinoma, particularly oral squamous cell carcinoma. Journal of Oral Microbiology 2010,2:5780.

18. Winn D M. Diet and nutrition in the etiology of Oral Cancer. Am J Clin Nutr.1995;61:437S-45S.

19. Suzuki T, Wakai K, Matsuo K. Effect of dietary anti oxidants and risk of oral, pharyngeal and laryngeal squamous cell carcinoma according to smoking and drinking habits. Cancer 2006;97:760-7.

20. J. Khalili. Oral cancer, risk factors, prevention and diagnostic: Review. Experimental Oncology 30;2008 (December):259-64.

21. S. Choi, J.N. Myers. Molecular Pathogenesis of Oral Squamous Cell Carcinoma: Implications for Therapy, Critical reviews in oral biology and medicine. J Dent Res 2008;87(1):14-32.

22. Crispian Scully and Jose Bagan. Oral squamous cell carcinoma overview. Oral

Oncology 2009;45:301-8.

23. Jose Bagan, Gracia Sarrion, Yolanda Jimenez. Oral cancer: Clinical features Review. Oral Oncology 46;(2010):414-7.

24. Neville, Damm, Allen, Bouquot. Allergies and Epithelial pathology. In Oral and Maxillofacial Pathology 2ndedition. Saunders,USA,2004.p-413.

25. J.B.Lippincott. Manual for staging of cancer. American Joint Committee on Cancer 4th ed. Philadelphia, 1993:45-55.

26. Snehal G. Pal, Jatin P. Shah. TNM Staging of Cancers of the Head and Neck. CA Cancer J Clin 2005;55:242-58.

27. Rajendran R. Benign and malignant tumors. In: Shafer, Hine, Levy, Editor. Shafer's Text book of Oral Pathology, 5thedition.New Delhi, India:Elsevier, 2006.p-108.

28. Ibrahim O. Bello, Ylermi Soini, Tuula Salo. Prognostic evaluation of oral tongue cancer: Means, markers and perspectives: Review. Oral Oncology 46;(2010): 636-43.

29. Doshi Neena P, Shah Siddharth A, Patel Keyuri B, Jhabuawala Munira F. Histological grading of oral cancer: A comparison of different systems and their relation to lymph node metastasis. National journal of community medicine 2011;2(1):136-42.

30. M .Akhter, S.Hossain, Quazi.B.Rahman, Motiur.R.Molla. A study on histological grading of oral squamous cell carcinoma and its co-relationship with regional metastasis. Journal of oral and maxillofacial pathology May-August 2011;15(2):168-76.

REVIEW ARTICLE

CALCIUM HYDROXIDE IN DENTISTRY: A REVIEW OF LITERATURE.

Authors:
Nebu Philip[1],
Bharat Suneja [2],
Reeba Mary Isaac[3]

[1]Reader, Dept. of Pedodontics and Preventive Dentistry, Indira Gandhi institute of Dental sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala, India.

[2]Reader and HOD, Dept. of Pedodontics and Preventive Dentistry, BJS Dental College and Hospital, Ludhiana, Punjab, India.

[3]Senior Lecturer, Department of Orthodontics, Indira Gandhi institute of Dental sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala, India.

Address for correspondence:
Dr. Nebu Philip, Reader, Dept. of Pedodontics and Preventive Dentistry, Indira Gandhi institute of Dental sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala, India.
E-mail: drnebu@gmail.com.

ABSTRACT

Calcium hydroxide is one of the most widely used medicaments in dentistry. This article aims to review the material based on its history, properties, mode of action, clinical use of its different formulations, significance of the vehicles used in $Ca(OH)_2$ pastes, and the role $Ca(OH)_2$ plays in various clinical procedures. The review of literature enabled us to conclude that its application chiefly related to its ability to stimulate dentin bridge formation and its antimicrobial properties. The review will also enable clinicians to make informed judgements about which formulations of calcium hydroxide should be used for specific clinical procedures.

Key words: calcium hydroxide, mode of action, clinical applications

J Odontol Res 2013;1(1):45-55.

INTRODUCTION

Calcium hydroxide has played a major role in dentistry, being one of the most widely used medicaments, ever since its use was popularized by the pioneering studies of Hermann.[(1)] Well conducted research about the properties of calcium hydroxide such as its biocompatibility, antimicrobial potential, hard tissue deposition, physical-chemical aspects, give credibility to the choice of this materials in several clinical procedures.[(2)] This retrospective of literature aims to review the material under the following general headings: history, physical & chemical properties, its mechanism of action, the different formulations of $Ca(OH)_2$, vehicles used in $Ca(OH)_2$ pastes, and the role of $Ca(OH)_2$ in various clinical procedures where its use is indicated.

HISTORY

The initial reference to the use of calcium hydroxide in dentistry has been attributed to Nygren (1838)[3] for the treatment of a dental fistula, whilst Codman (1851)[4] was the first to use the material to preserve involved pulp. It was however through the studies of Hermann (1930)[1] that calcium hydroxide was introduced to dentistry as a successful pulp capping agent. Before this pulp therapy consisted of devitalization with arsenic and other fixative agents. Hermann demonstrated the formation of secondary dentin over the amputation sites of vital pulps capped with calcium hydroxide. Hermann's introduction of calcium hydroxide to dentistry had begun a new era of successful vital pulp therapy.[5]

The first reports dealing with successful pulp capping using calcium hydroxide appeared in literature between 1934 and 1941, since then the clinical indications for its use have expanded greatly. In 1938, Teuscher and Zander introduced calcium hydroxide in the United States and they histologically confirmed complete dentinal bridging with healthy radicular pulp under the calcium hydroxide medicament.[6] $Ca(OH)_2$ was most favoured as a pulpotomy agent in the 1940s and mid-1950s because it was thought to be more biologically acceptable owing to the fact that it promoted reparative dentin bridge formation and pulp vitality was maintained.

PHYSICAL PROPERTIES

· $Ca(OH)_2$ is a white odourless powder with a molecular weight of 74.08

· It has low solubility in water (1.2 g/l at 25°c), which further decreases as temperature rises. This low solubility is a good clinical characteristic, because a long period is necessary before it becomes soluble in tissue fluids, prolonging its duration of action when in contact with vital tissues.

· $Ca(OH)_2$ has been classified as a strong base with a pH of 12.5-12.8.[7,8,9] The high pH of calcium hydroxide plays a vital role in the various actions of calcium hydroxide.

CHEMICAL PROPERTIES

The main actions of calcium hydroxide come from its dissociation into Ca^{2+} ions and OH^- ions. The percentage of these ions is about 45.89% Ca^{2+} and 54.11% OH^- ions. The actions of these ions on vital tissues and bacteria induce hard tissue deposition and its antibacterial effect.[9]

Changes in the biological properties of $Ca(OH)_2$ can also be understood through its chemical reactions. For example, $Ca(OH)_2$ in the presence of carbon dioxide (from tissues or atmosphere) forms calcium carbonate (a weak acid oxide), and this product does not have $Ca(OH)_2$ hard tissue depositing and antibacterial properties.

Thus a chemical analysis of the essential aspects of calcium hydroxide is important in order to use it correctly. Factors such as the influence of the vehicle on its rate of ionic dissociation, time necessary for reaching appropriate pH for microbial control, the action of carbon dioxide that interferes with its properties etc., have to be taken into account when using the material in different clinical situations.[2]

MECHANISM OF ACTION

The mechanism by which calcium hydroxide brings about its various actions has been broadly divided into two categories:[2]

1. Biological Effects
2. Antibacterial Effects

1) Biological Effects:

When calcium hydroxide is applied directly to the pulp (for example in a direct pulp cap or pulpotomy procedure), the tissue immediately adjacent to the medicament is necrotized by the high pH of calcium hydroxide. The necrosis is accompanied by acute inflammatory changes in the underlying tissue. In 4-9 days, three distinct histological zones can be identified under the calcium hydroxide medicament-

1. Zone of coagulation necrosis.
2. Zone of variable osteodentin.
3. Zone of relatively normal pulp under a hyperemic odontoblastic layer.

Holland et al. have showed that formation of mineralized dentin starts between 7-10 days of applica-

tion, at the junction of the necrotic zone and vital inflamed tissue.[10]

Calcium Hydroxide Induced Mineralization

It seems that calcium hydroxide has the unique potential to induce mineralization even in tissues which are not programmed to mineralize. [11] Although calcium hydroxide works effectively in forming a dentin bridge, its exact mechanism is not fully understood. The following routes have been suggested as to how calcium hydroxide induces the reparative process:

- Calcium hydroxide is believed to maintain a local state of high alkalinity and thereby has the capability of activating tissue enzymes such as alkaline phosphatase, which is postulated to play an important role in hard tissue formation.[12] Alkaline phosphatase, which acts best in an alkaline pH of 8.6 to 10.3, is associated with providing phosphate ions at the mineralization sites. It does this by hydrolyzing phosphate ions from organic radicals at an alkaline pH.[13] These phosphate ions react with Ca^{2+} ions from the circulating blood, creating a sediment of calcium phosphate on the organic matrix, and indeed this sediment is the molecular unit of hydroxyapatite, starting the process of dentin bridge formation.

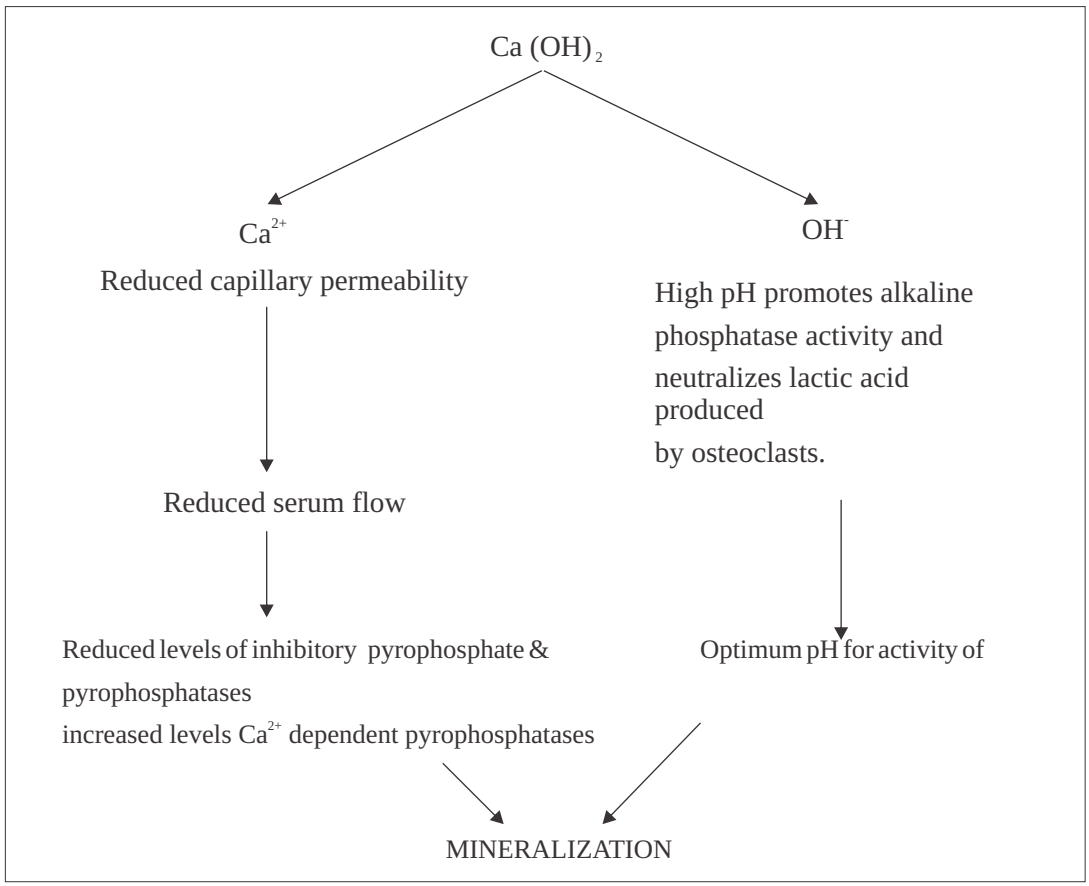
- Heithersay suggested that calcium ions may reduce the permeability of capillaries, so that less intercellular serum is produced, thus increasing concentration of Ca^{2+} ions at the mineralization site.[13] The reduced capillary permeability and the increase in the number of Ca^{2+} ions, could further reduce serum flow within the dental pulp and consequently the concentration of the inhibitory pyrophosphate ion would be reduced. The pyrophosphate ions play a role in inhibiting the mineralization process. Not only is its concentration reduced, but it is also metabolized at the mineralization sites, due to an increase in the levels of calcium dependent pyrophosphatases, and this could result in uncontrolled mineralization of pulp tissues. This could also possibly explain the high incidence of mineralized canals following pulpotomy or direct pulp capping with $Ca(OH)_2$.[14] Uncontrolled mineralization of pulp is therefore dependent on a reduced blood supply to the remaining vital tissue and not necessarily on the amount of reparative dentin formed with time.[15]

- It has been suggested that a rise in pH, as a result of the free OH^- ions, may favour mineralization.[16] An alkaline pH may neutralize the lactic acid secreted by osteoclasts and this may help to prevent further destruction of mineralized tissue. The high pH is also optimum for pyrophosphatase activity, thereby reducing levels of inhibitory pyrophosphate; leading to or favouring mineralization. There is however a problem in accepting the hydroxyl ions as the sole initiator of the mineralization process, as it has been shown that other highly alkaline compounds such as barium hydroxide and calcium phosphate fail to initiate mineralization.[11]

It has been speculated that $Ca(OH)_2$ exerts a mitogenic and osteogenic effect. The high pH combined with the availability of Ca^{2+} and OH^- ions have an effect on enzymatic pathways and hence mineralization.[17]

Yoshiba et al. provided immunofluoroscence evidence of the possible contribution of calcium hydroxide to odontoblastic differentiation. They found increased amounts of fibronectin (an extracellular glycoprotein linked to cell differentiation), among migrating fibroblasts and newly formed odontoblasts, in areas of initial calcium hydroxide induced calcification. They noted that while calcium hydroxide was not unique in initiating reparative dentinogenesis, it demonstrated the most rapid tubular dentin formation in comparison to calcium phosphate ceramics and tricalcium phosphate.[18]

Summary of Ca (OH)$_2$ induced mineralization



The Dentin Bridge

A mineralized barrier or 'dentin bridge' is usually produced following the application of calcium hydroxide to vital pulp. This repair material appears to be the product of odontoblasts and connective tissue cells. A discussion on this critical dentin bridge, which is the product of the repair process induced by calcium hydroxide, is important to understand the result of the mineralization process induced by $Ca(OH)_2$.

Source of Ca^{2+} ions in the Dentin Bridge

There is some controversy as to whether the calcium ions necessary for dentinal bridge formation comes exclusively from the circulating blood or whether the calcium hydroxide medicament also makes a contribution. Radioisotope studies have established that Ca^{2+} ions present in the applied $Ca(OH)_2$ do not become incorporated in the mineralized repair tissue.[19,20] Rather the dentin bridge derives its mineral content solely from the dental pulp, via the blood supply. These studies indicate that calcium hydroxide is an initiator rather than a substance for repair and its role in dentin bridge formation is limited to maintaining a low pH and producing a low grade irritation and inflammation.

However other studies showed that the Ca^{2+} ions from the medicament may also enter into the dentin bridge by forming calcium salts and calcium protein complexes.[21,22] What these investigators did was to use pastes such as barium hydroxide and strontium hydroxide, instead of calcium hydroxide. When they evaluated the bridge formed later, it showed deposits of barium carbonate and strontium carbonate grains, resembling graining observed with calcium hydroxide. Since barium and strontium are not normally present within the circulating blood, they concluded that these deposits could only come from the capping medicament used.

What can be tentatively concluded is that, it is the circulating calcium from the blood that is mainly responsible for the calcium phosphate dentin bridge, while other calcium complexes and salts, such as calcium carbonate in the dentin bridge, may be formed by calcium coming from the medicament. What is undisputed is the role calcium hydroxide plays in maintaining a localized state of alkalinity, stimulating alkaline phosphatase activity, and reducing capillary permeability, all factors that favour mineralization and pulpal repair.

2.) Antibacterial effects

Along with its biological effects on tissues, calcium hydroxide has an important antimicrobial action, and this property is important in many of the clinical procedures. These antibacterial effects are thought to be related to pH and directly proportional to the rate of release of OH^- ions from $Ca(OH)_2$. The various ways by which $Ca(OH)_2$ brings about its antimicrobial action are:

- There is liberation of ions OH^- from the alkaline $Ca(OH)_2$ and these ions act on the enzymatic sites, proteins and phospholipids located on the bacterial cytoplasmic membrane. The effect of high pH alters the integrity of the cytoplasmic membrane by interfering with enzymatic activity, chemical injury to organic components and transport of nutrients. The extreme values of pH cause the uncoiling of many bacterial proteins with loss of their biological activities. Estrela et al. suggested the hypothesis of an irreversible bacterial enzymatic inactivation if extreme pH conditions are maintained for a long period of time and also a temporary bacterial enzymatic inactivation, with the restoration of normal activity when the pH returns to the ideal level for enzymatic activity.[23] This suggests the importance of maintaining a high pH of the $Ca(OH)_2$ dressing used as an intracanal medicament so has to give it enough time to manifest its potential of action on microorganisms present in endodontic infections.

- Another essential aspect in the antimicrobial action of $Ca(OH)_2$ is its neutralization of bacterial toxins. Buck et al. evaluated the detoxification of endotoxin by different endodontic irrigants and calcium hydroxide. The results showed that the biologically active portion of endotoxin, Lipid A, is hydrolyzed by highly alkaline chemicals like $Ca(OH)_2$.[24]

- Safavi and Nichols showed that $Ca(OH)_2$ is capable of hydrolyzing the lipid moiety of the bacterial lipopolysaccharide cell wall (LPS), resulting in the release of free hydroxyl fatty acids. They suggested that the calcium hydroxide mediated degradation of LPS may be an important reason for the beneficial effects obtained with calcium hydroxide used in clinical endodontics.[25]

- Estrela et al. suggested that $Ca(OH)_2$ is effective against anaerobic organisms, which are the most abundant organisms in the infected root canals, through its ability to absorb carbon dioxide, essential for the survival of these organisms.[26]

What can be concluded in the search for explaining the antimicrobial mechanism of calcium hydroxide is that the enzymes in the cytoplasmic membrane were the primary targets of pH changes, which can lead to reversible or irreversible inactivation of a wide range of microorganisms. As the site of action of OH^- ions of calcium hydroxide is the bacterial cytoplasmic membrane, and since in the microbial world, cytoplasmic membranes are similar irrespective of the microorganisms other characteristics, calcium hydroxide has a wide range of action with similar effect on aerobic, anaerobic, Gram+ve and Gram-ve bacteria. This is clinically significant as in an infected canal system all these organisms are encountered.[2] It is to be noted that calcium hydroxide kills only the bacterial on the surface of the pulp and not those that have penetrated necrotic tissue.[27] Thus the material has no beneficial effect on the healing of an inflamed pulp and its use would appear to be indicated for the treatment of healthy or superficially contaminated pulps where bacteria have not penetrated into the deeper parts.[28] The main issue is not 'how bacteria are killed' but 'how vital tissues can be protected from the toxicity of calcium hydroxide'. The separation of the material from the vital tissues by a zone of necrosis is probably what prevents gross tissue damage.[14]

DENTAL FORMULATIONS OF $Ca(OH)_2$:
Setting vs. Non-setting pastes

Several calcium hydroxide containing pastes are currently being used in dentistry. These can broadly be classified according to whether they are setting (e.g. Dycal) or non-setting materials (e.g. Pulpdent). Clinicians tend to develop their own preferences according to ease of manipulation and apparent clinical success, however there are some procedures where one formulation is recommended over the other.

Non-setting pastes have a higher pH than hard setting pastes, and thus have a better antibacterial effect. However non-setting calcium hydroxide pastes tend to undergo dissolution at a much faster rate and are likely to leach out from beneath the restoration. Therefore it is essential to establish a balance between a material that is sufficiently soluble to exert a therapeutic effect, yet which is not so soluble that it dissolves away, thus vitiating its desired use. Thus non-setting $Ca(OH)_2$ pastes, like ApexCal (Ivoclar, Schaan, Liechtenstein.), are generally used for root canal dressings, where as setting $Ca(OH)_2$ pastes are used as root canal sealers, e.g. Apexit (Ivoclar, Schaan, Liechtenstein).[29] In an IPC procedure, where the antimicrobial action of $Ca(OH)_2$ is more important, a non-setting calcium hydroxide paste is recommended. British researchers have confirmed this, by finding significantly more bacteria under the hard setting version versus the non-setting pastes.[30] In a DPC or pulpotomy procedure both pastes are equally effective in inducing reparative dentin formation. The advantage a paste like Dycal (Dentsply, Milford, USA) has is that being less alkaline, it is less caustic to the remaining vital pulp, and being stronger, condensation of the permanent filling can be done directly over it. The advantage a non-setting paste like Pulpdent (Pulpdent Corp., Brookline, USA) has is that the dentin bridge it forms is more visible radiographically, allowing better clinical judgment of whether the procedure is a success or not. The reason for the better radiographic contrast under a non-setting paste, is that a necrotic zone of chemically altered tissue is formed between the material and the dentin bridge, as a result there is a gap between the bridge and the medicament, making it easier to distinguish the dentin bridge. Although the dentin bridge formed under the setting pastes are histologically similar, the necrotic zone is resorbed prior to the formation of the dentin bridge, which then comes to lie directly under the capping material making it difficult to distinguish radiographically.

VEHICLES USED IN CALCIUM HYDROXIDE PASTES

Many substances have been added to calcium hydroxide to improve it antibacterial action, radiopacity, flow, consistency, and to maintain its high pH.[31] An ideal vehicle should allow slow diffusion of Ca^{2+} and OH^- ions, have low solubility in tissue fluids, and permit hard tissue deposition.[32] The differences in the velocity of ionic dissociation of $Ca(OH)_2$ are directly related to the vehicle used in the paste, the lower the viscosity of the paste the higher will be the ionic dissociation.[23] In general 3 types of vehicles are used - aqueous, viscous and oily.[32]

Aqueous Group Vehicles: This group is represented by water-soluble vehicles including water, saline, dental anaesthetics, and methylcellulose. These types of vehicles promote rapid release of Ca^{2+} and OH^- ions and a high degree of solubility, causing $Ca(OH)_2$ to be rapidly solubilized and resorbed. Examples: Calxyl (Otto & Co., Frankfurt, Germany) - oldest manufactured paste introduced by Hermann and is basically a solution of $Ca(OH)_2$ in water, Pulpdent (Pulpdent Corp., Brookline, USA) - $Ca(OH)_2$ in an aqueous suspension of methylcellulose,& Calcipulpe (Septodont, Saint-Maur, France) - vehicle used is carboxymethylcellulose.

Viscous Group Vehicles: The pastes in this category use viscous vehicles such as glycerin, polyethylene glycol, and propylene glycol. The viscous vehicles cause a much slower release of Ca^{2+} and OH^- ions over extended periods. The higher molecular weight of these vehicles minimizes the solubility of $Ca(OH)_2$ into the tissues and maintains the paste in the required areas for longer intervals, prolonging the action of the paste. Example: Calen (S.S. White-Artigos Denta´rios, Rio de Janeiro, Brazil) - polyethylene glycol is the viscous vehicle in which $Ca(OH)_2$ is dispersed.

Oily Group Vehicles: The oily vehicles like silicone oil, camphor, & eugenol are non-water soluble substances that promote the lowest solubility and diffusion of $Ca(OH)_2$ within the tissues. Pastes containing this type of vehicle remain in the root canal for a longer time as compared to aqueous or viscous vehicles. Example: Vitapex (Neo Dental Chemical Products Co., Tokyo, Japan) - composed of calcium hydroxide, iodoform, and silicone oil as the oily vehicle.

Clinical situations that require a rapid liberation Ca^{2+} and OH^- ions at the beginning of treatment require an aqueous vehicle containing $Ca(OH)_2$ paste, whilst in clinical situations that require gradual and more uniform liberation of ions, a viscous vehicle containing paste should be used. Pastes containing oily vehicles have restricted use and are employed in those clinical situations that require a very slow ionic dissociation.[32] For example, in cases of dental replantation, as soon as treatment is performed, a paste with an aqueous vehicle should be used, because of the need for rapid ionic release. Subsequently a $Ca(OH)_2$ paste with a viscous vehicle should be used in the following dressings, because the paste must remain in the root canal for a longer period, during which high pH will be maintained and slow ionic release will occur. Pastes containing oily vehicles, like Vitapex, are used for apexification procedures, where the $Ca(OH)_2$ dressings have to last for 6-24 months.

ROLE OF CALCIUM HYDROXIDE IN CLINICAL PROCEDURES

1) Lining of cavities

The setting calcium hydroxide pastes are now in general use as lining materials. Their perceived advantages in addition to their therapeutic effects are as follows:[30]

i) They have a rapid initial set in the cavity, under the accelerating effect of moisture in the ambient air of the oral cavity.

ii) They do not interfere with the setting reaction of BIS-GMA resins, and are therefore the lining material of choice under composite resin materials.

iii) It is generally considered that the initial set of the material is sufficiently hard to resist the applied condensation pressure required for amalgam alloys.

2) Indirect Pulp Capping (IPC)

There is evidence that $Ca(OH)_2$ stimulates an increase in mineralization, within the affected dentin that remains at the base of the cavity.[29] The mechanisms by which mineralized dentin formation is initiated by calcium hydroxide, cannot hold true in an IPC procedure, as the medicament is not in direct contact with the pulp (as occurs is an direct pulp cap or pulpotomy procedure). The dentin bridging effects of calcium hydroxide occur only when the agent is in direct contact with healthy pulp tissue.[33] So the role of $Ca(OH)_2$, in an IPC procedure is limited to acting as an antimicrobial agent and a mild pulp stimulant with the pulp responding physiologically to the carious process to produce reparative dentin.[10]

Clinical studies have shown no significant differences in the ultimate success of this procedure, regardless of whether $Ca(OH)_2$ or some other medicament like Zinc oxide-eugenol is used over the residual carious dentin.[34,35] So while some clinicians recommend Zinc oxide-eugenol because of its better sealing and obtudent properties, others recommended calcium hydroxide because of its ability to stimulate a more rapid formation of reparative dentin and also it can stimulate healing of minute pulpal exposure which may have been overlooked.

3) Direct Pulp Capping (DPC)

DPC is undertaken in an attempt to maintain the health of an exposed vital pulp. Calcium hydroxide works very effectively in inducing the formation of a mineralized dentin bridge over the vital pulp. If bacterial contamination is minimal, calcium hydroxide pulp capping procedures can produce successful results in 96% of cases.[36] Just like in the IPC procedure, the importance of preventing bacterial microleakage and maintaining a proper apical seal for the success of a direct pulp capping procedure is vital.

4) Pulpotomy

This is a standard clinical procedure, which differs from DPC in that, surgical removal of the coronal

pulp is undertaken, prior to application of a medicament like $Ca(OH)_2$ The healing process is similar to that which occurs in direct pulp capping, i.e. a mineralized dentin bridge, is formed over the amputated pulp.

Calcium hydroxide was most favoured as a pulpotomy agent in the 1940s and mid-1950s because it was thought to be more biologically acceptable owing to the fact that it promoted reparative dentin bridge formation and pulp vitality was maintained.[6] Inspite of the great success of $Ca(OH)_2$ in forming a dentin bridge, a calcium hydroxide pulpotomy technique is generally not recommended for primary teeth owing to its low clinical success rate. Most of the long-term clinical studies have suggested this failure was the result of chronic inflammation and internal resorption calcium hydroxide causes.[37,38] At present calcium hydroxide pulpotomy cannot be recommended for primary teeth owing its low clinical success rate.[39]

Because of improved clinical outcomes, $Ca(OH)_2$ is the recommended pulpotomy agent for carious and traumatic exposures of young permanent teeth, particularly for the apexogenesis procedure. It is also recommended that once the apex has formed, the $Ca(OH)_2$ dressing be removed and conventional root canal obturation be done so has to prevent the long term canal calcification and obliteration it causes.[40]

***5)* Primary root canal filling material**

A study conducted by Mani & Chawala found $Ca(OH)_2$ mixed with lodoform to be a near ideal primary root canal filling material. The main advantages were its biocompatibility, anti-microbial actions, any over fill resorbs quickly, and it can be more easily applied or if necessary removed from the roots as compared to Zinc oxide-Eugenol.[42] One disadvantage with the use of $Ca(OH)_2$ as a root canal filling material was that it resorbs at a slightly faster rate that the deciduous root.

6) Intra canal medicament

Calcium hydroxide has been found to be the most effective intra canal medicament to obtain canal sterilization between endodontic appointments.[43] Although the cleaning, debridement and shaping of the root canal is the most important step of eliminating bacteria from the canal system, use of an intra canal medicament like $Ca(OH)_2$ can reduce treatment time and number of appointments. It is important to maintain a high pH as well as to allow enough time for the medicament to manifest its antimicrobial potential on the microorganisms present in endodontic infections. Heithersay recommends that $Ca(OH)_2$ be used as a root canal dressing in teeth with large periapical lesions and in cases where it is necessary to control the seepage of periapical exudates into the canal.[13] The mechanism whereby reduction of the seepage occurs is probably due to the fibrous barrier that is formed when $Ca(OH)_2$ is placed in direct contact with host tissues[44] or due to the constriction of capillaries.[13] $Ca(OH)_2$ is also believed to accelerate the natural healing of the periapical lesions regardless of the bacterial status of the root canal system.[45] The ability of $Ca(OH)_2$ to dissolve necrotic tissue is also useful, as anatomical problems may make it difficult for irrigating solutions to reach all areas of the root canal.[46]

7) Apexification

When $Ca(OH)_2$ is used for the apical closure of a non-vital young permanent tooth it forms a calcific

$Ca(OH)_2$ induced internal resorption

Calcium hydroxide induced internal resorption has been a subject of interest because of the implications it may have when used in different procedures like DPC or pulpotomy. Internal resorption occurs because the highly alkaline $Ca(OH)_2$ causes an over stimulation of the remaining vital pulp tissue, which results in undifferentiated mesenchymal cells giving rise to odontoclasts, that cause the resorption.[41] Internal resorption usually occurs when $Ca(OH)_2$ is in contact with the highly cellular vital primary pulp, and it is for this reason that DPC or $Ca(OH)_2$ pulpotomies are not recommended in deciduous teeth. Internal resorption does not appear to be a problem in permanent teeth, probably because of its lower cellular content and decreased inflammatory response of the permanent tooth pulp.

barrier of mineralized scar tissue across the apical foramen, in a manner similar to how it brings about dentinal bridging in DPC or pulpotomy. The difference here being that the hard tissue barrier it forms consists mainly of osteoid tissue (as there is no vital pulp and odontoblasts to differentiate and form reparative dentin). Because $Ca(OH)_2$ is inherently soluble, it must be replaced at 3-6 month intervals, until closure of the apex has occurred. It has also been suggested that role of $Ca(OH)_2$ in apexification is secondary, and what is more important in the success of this procedure is complete debridement and asepsis of the canal system, as apical closure has been found to occur even without a root-end filling material.

CONCLUSION

Ever since the introduction of calcium hydroxide as a pulp capping agent by Hermann, its role in dentistry has been almost unparalleled, and the indications for its use has expanded greatly. This review of literature enables us to conclude that its application in various dental procedures chiefly relate to its ability to stimulate dentin bridge formation and its antimicrobial properties. Considering the immense potential of the medicament it is truly a 'panacea in dentistry'.

REFERENCES

1. Hermann BW. Dentinobliteration der Würzelkanälen nach Behandlung mit Kalzium. *Zahnarztliche Rundschau*. 1930;39:888-99.
2. Estrela C, Holland R. Calcium hydroxide: study based on scientific evidences. J Appl. Oral Sci. 2003; 11(4):269-82.
3. Nygren J. *Radgivare Angaende Basta Sattet Att Varda Ah Bevara Tandernas Fuskhet. 1838 Osv.* Stockholm.
4. Codman, WW 1851 Ossification of the pulp of a tooth. *Newsletter* IV, 90 (printed in Malo PRT, Kessler Nieto F, Vadillo MVM) Hidroxido de calcio y apicoformación. *Revista Espa Ola de Endodoncia*. 1987;*5*:41-61.
5. Stanley HR. Calcium hydroxide and vital pulp therapy. *In* Seltzer and Bender's dental pulp, Hargreaves KM, Goodis HE (eds.). Quintessence books, Chicago. 2002,pp.309-24.
6. Teuscher GW, Zander HA. A preliminary report on pulpotomy. Northwest Univ Grad Bull 1938;39:4.
7. Maisto OA . *Endodoncia*. 3rd ed. Buenos Aires: Mundi.1975
8. Cvek M. *Calcium Hydroxide in the Treatment of Traumatized Teeth.* Stockholm: Eastman Institute. 1989.
9. Estrela C. *Análise química de pastas de hidróxido de calcio frente é liberacão de ions calcio,de ions hidroxila e formacão de carbonato de calcio na presenç a de tecido con i untivo de* cão (Thesis). 1994. São Paulo.
10. Holland R, Mello W, Nery MJ, Souza V, Bernabé PFE, Otoboni-Filho JA. Healing process of Dog's dental pulp after pulpotomy and pulp covering with calcium hydroxide in powder or paste form. Acta Odontol Pediatr 1981;2:47-51.
11. Mitchell DF, Shankwalker GB Osteogenic potential of calcium hydroxide and other materials in soft tissue and bone wounds. *J Dent Res* 1958;37:1157-63.
12. Guo J, Messer B. Properties of Ca^{2+} and Mg^{+} activated adenosine Triphosphate from rat incisor pulp Arch Oral Biology 1976;21(11):637-40.
13. Heitersay GS. Calcium hydroxide in the treatment of pulpless teeth with associated pathology. J Brith Endod Soc 1975;8:74-93.
14. Seltzer S, Bender IB. The dental pulp-biologic considerations in dental procedures. 3rd ed. Philadelphia: JB Lippincott. 1984.
15. Heide S, Kerkes GS. Delayed pulp capping in permanent incisors of monkey. Int Endod J 1987;20(2):17-21.

16. Tronstad L, Andreassen JO, Hasselgren G, Kristerson L, Riis I. pH changes in dental tissues after root canal filling with calcium hydroxide. J Endod 1981;7:17-21.

17. Torneck CD, Moe H, Hawley TP. The effect of Calcium hydroxide on porcine pulp fibroblasts in vitro. J Endod 1983; 9(4):131-6.

18. Yoshiba K, Yoshiba N, Nakamura H. Immunocolonization of fibronectin during reparative dentinogenesis in human teeth after pulp capping with calcium hydroxide. J Dent Res 1996; 75:1590.

19. Sciaky I, Pisanti S. Localization of calcium placed over amputated pulps in dogs teeth. J Dent Res 1960;39:11-28.

20. Attalla MN, Noujaim AA. Role of calcium hydroxide in the formation of reparative dentin. J Can Dent Assoc. 1969;35:267-52

21. Stark MM. The localization of radioactive $Ca(OH)_2$ over exposed pulps in rhesus monkey teeth: a preliminary report. J Oral Ther Pharmacol 1964; 3:290-97.

22. Holland R. Histochemical analysis of the dog's dental pulp after pulp capping with calcium, barium, and strontium hydroxides. J Endod. 1982;8:444-9.

23. Estrela C, Sydney GB, Bammann LL, Fellipe-Jr O. Estudo do efeito biológico do pH na atividade enzimática de bactérias anaeróbias. Rev Fac Odontol Bauru 1994;2:31-38.

24. Buck RA, Cai J, Eleazer PD, Staat RH, Hurst HE. Detoxification of endotoxin by endodontic irrigants and calcium hydroxide. J Endod 2000;27:325-327.

25. Safavi KE, Nichols FC. Effect of calcium hydroxide on bacterial lipopolysaccharide. J Endod 1993; 19:76-78.

26. Estrela C, Fabiana CP, Yoto I, Bammann LL. In vitro determination of direct antimicrobial effect of calcium hydroxide. J Endod. 1998;24:15.

27. Cox CF, Bergenholtz G, Heys DR, Syed SA, Fitzgerald M, Heys RJ. Pulp capping of dental pulp mechanically exposed to microflora: a 1-2 year observation of wound healing in the monkey. J Oral Pathol. 1985;-14:156-168.

28. Watts A, Paterson RC. Bacterial contamination as a factor influencing the toxicity of materials to the exposed pulp. Oral Surg. 1987;64:466.

29. Eidelman E, Finn SB, Koulourides T. Remineralization of carious dentin treated with calcium hydroxide. J Dent Child 1965;32(4):218-225.

30. Lim M, McCabe J. Lining material for amalgam restorations. Br Dent J 1982; 152(9): 313-5.

31. Leonardo MR, Leal JM, Simêes Filho AP. *Endodontia Tratamento Dos Canais Radiculares.* 1982 São Paulo: Panamericana.

32. Fava LR, Saunders WP. Calcium Hydroxide pastes: classification and clinical indications. Int Endod J 199; 32(4)257-69.

33. Stanley HR. Criteria for standardizing and increasing credibility of direct pulp capping studies. Am. J Dent 1998;11:S17-S34.

34. Ehrenreich DV. A comparison of the effects of zinc oxide-eugenol and calcium hydroxide in carious dentin in human primary molars. J Dent Child 1968;35:451.

35. Stanley HR. Pulpal response to dental techniques and materials. Dent Clin North Am 1971;15:115-26.

36. Cvek M Clinical report on partial pulpotomy and capping with calcium hydroxide in permanent incisors with complicated root fractures. J Endod.1978;4(8):232-7.

37. Via WR Jr. Evaluation of deciduous molars

treated by pulpotomy and calcium hydroxide. J Am Dent Assoc 1955;50:34-7.

38. Law DB. An evaluation of the vital pulpotomy technique. J Dent Child 1956; 23:40.

39. Mathewson RJ, Primosch R. Pulp treatment. In: Fundamentals of pediatric dentistry. 3rd ed. Chicago: Quintessence. 1995 p257-84.

40. Patterson SS. Pulp calcification due to operative procedures - pulpotomy. Int Dent J 1967;17:490-4.

41. Ingle IJ, Bakland L. Endodontics. In: Dummett C O Jr and M. Kopel H M Pediatric Endodontics. 5th ed. B C Decker;2002.

42. Mani A, Chawala H. Evaluation of Calcium hydroxide as a root canal filling material in primary teeth. J Dent Child 2000;67(2):142-7.

43. Byström A, Claesson R, Sundqvist G. The antibacterial effect of camphorated paramonochlorophenol, comphorated phenol and calcium hydroxide in the treatment of infected root canals. Endod Dent Traumatol 1985;1:170-75.

44. Rasmussen P, Mjor IA Calcium hydroxide as an ectopic bone inductorin rats Scan. J Dent Res 1971;79(1):24-30.

45. Matsumiya S, Kitamura M. Histopathological and histobacteriological studies of the relation between the condition of sterilization of the interior of the root canal and the healing process of periapical tissues in experimentally infected root canal treatment. Bulletin of Tokyo Dental College 1960;1:1-19

46. Hasselgren G, Olsson B, Cvek M. Effects of calcium hydroxide and sodium hypochlorite on the dissolution of necrotic porcine muscle tissue. J Endod 1988;14:125-7.

REVIEW ARTICLE

SCOPE OF PHOTODYNAMIC THERAPY IN THE FIELD OF PERIODONTICS: A REVIEW

ABSTRACT

Photodynamic therapy (PDT) is a form of phototherapy using nontoxic light-sensitive compounds that are exposed selectively to light, whereupon they become toxic to targeted malignant and other diseased cells. It involves the use of low power lasers with appropriate wavelength to kill micro organisms treated with a photosensitizer drug. PDT could be a useful adjunct to mechanical as well as antibiotics in eliminating periopathogenic bacteria. Applications of photodynamic therapy in dentistry are growing rapidly for the treatment of oral cancer, bacterial and fungal infections and photodynamic diagnosis of malignant transformation of oral lesions, and are recognized as a treatment strategy which is both minimally invasive and minimally toxic.

Keywords: photodynamic therapy, photosensitizers, gingivitis, periodontits, peri-implantitis, Flap surgery, microbial resistance, systemic antibiotics.

J Odontol Res 2013;1(1): 56-62.

Authors:
Noorudeen AM [1],
Mohammed Shereef[2],
Mathew T Joy[2]

[1]Professor &HOD, Dept. of Periodontology and Oral Implantology, Indira Gandhi institute of Dental sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala, India.

[2]Senior lecturer, Dept. of Periodontology and Oral Implantology, Indira Gandhi institute of Dental sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala, India.

Address for correspondence:
Dr. Mohammed Shereef, Senior lecturer, Dept. of Periodontology and Oral Implantology, Indira Gandhi institute of Dental Sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala, India.
E-mail: mohammedshereef@yahoo.co.uk

INTRODUCTION

Photodynamic therapy (PDT) is an effective and innovative microbicidal method which involves the combination of a non-toxic dye (photosensitizer) and a visible light source. It shows a great microbicidal effect in addition to better access to sites that are inaccessible to conventional therapy. The use of PDT as an antimicrobial control method has local and specific effects, and also selectiveness for the pathogens[(1)]. The word photodynamics means the application of dynamics of photons of light on the biological molecules[(2)]. Photodynamic therapy (PDT) matured as feasible medical technology in 1980s at several institutions in the world basically as a treatment for cancer. Photodynamic ther-

apy (PDT) has emerged in recent years as a non – invasive therapeutic modality for the treatment of various infections by bacteria, fungi, and viruses[3]. This therapy is defined as an oxygen-dependent photochemical reaction that occurs upon light – mediated activation of a photosensitizing compound leading to the generation of cytotoxic reactive oxygen species, predominantly singlet oxygen[4].

Most modern PDT applications involve three key components[5]: a photosensitizer, a light source and tissue oxygen. The wavelength of the light source needs to be appropriate for exciting the photosensitizer to produce reactive oxygen species. The combination of these three components leads to the chemical destruction of any tissues which have either selectively taken up the photosensitizer or have been locally exposed to light.

HISTORY

The origin of light as a therapy in medicine and surgery are traced from antiquity to the modern day. Phototherapy began in ancient Greece, Egypt, and India, but disappeared for many centuries, only being rediscovered by the Western civilization at the beginning of the 20th century. The use of contemporary photodynamic therapy was first reported by the Danish physician, Niels Finsen. He successfully demonstrated photodynamic therapy by employing heat - filtered light from a carbon - arc lamp (The Finsen Lamp) in the treatment of a tubercular condition of the skin known as Lupus Vulgaris. [3]

A German physician Friedrich Mayer-Betz performed the first study, with what was first called photoradiation therapy (PRT) with porphyrins in 1913 in humans. But it was John Toth, who acknowledged the photodynamic chemical effect of the therapy with early clinical argon dye asers and renamed it as photodynamic therapy (PDT). It received even greater interest as Thomas Dougherty formed the International Photodynamic Association. Its use first started in dermatology (1992), then oncology (1995), and recently in microbiology (1996) [6]. PDT was approved by the Food and Drug Administration in 1999 to treat precancerous skin lesions of the face or scalp.[4,7] The first light sources used in PDT were conventional lamps with no-coherent and polychromatic light, and a strong thermal component associated with light emission. They were later replaced by light-emitting diodes and low-level diode lasers[1].

MECHANISM OF ACTION

The basis of PDT is the interaction of light with photosensitive agents to produce an energy transfer and a local chemical effect. Here, many photosensitizers work together to harvest light energy to produce chemical reactions. Of the many photosensitizers that have been used in PDT, each has its own unique excitation properties. Usually, the photosensitizer is excited from a ground singlet state to an excited singlet state. It then undergoes intersystem crossing to a longer-lived excited triplet state. One of the few chemical species present in tissue with a ground triplet state is molecular oxygen. When the photosensitizer and an oxygen molecule are in proximity, an energy transfer can take place that allows the photosensitizer to relax to its ground singlet state, and create an excited singlet state oxygen molecule. Singlet oxygen is a very aggressive chemical species and will very rapidly react with any nearby biomolecules. Ultimately, these destructive reactions will kill cells through apoptosis or necrosis. PDT can be considered a form of targeted singlet oxygen chemotherapy, where the targeting is achieved with the combination of the photosensitizer (functioning as a catalyst) and intense light.

PHOTOSENSITIZER

Requirements of an optimal photosensitizer include following characteristics:

1. Biologically stable [1,8]
2. Must be photochemically efficient [1,8]
3. Selectively retained in the target tissue [1,8]
4. Low toxicity and fast elimination from the skin and epithelium[6]
5. Should have minimal toxicity to other than the target area [1,8]
6. High quantum yield of singlet oxygen production in vivo[6]

7. Cost effectiveness and commercial availability[6]

8. High solubility in water, injection solutions, and blood substitutes[6]

9. Storage [9]

More than 400 compounds are known with photosensitizing properties including dyes, drugs, cosmetics, chemicals and many natural substances.[10] Most of the sensitizers used for medical purposes belong to the following basic structures:

· Tricyclic dyes with different meso-atoms. Acridine orange, proflavine, riboflavin, methylene blue, fluorescein, eosine, erythrosin, rose bengal.

· Tetrapyrroles. Porphyrins and derivatives, chlorophyll, phylloerythrin, phthalocyanines.

· Furocoumarins. Psoralen and its methoxy-derivatives xanthotoxin, bergaptene.

ADVANTAGES OF PDT[6]

Therapy has only localized effects as the photosenstizer is selectively absorbed at a greater rate by target tissues, can be performed in outpatient or day-case settings[16], is more economical than radiation and surgical therapy for cancer patients, shows faster post-operative healing with no long term side effects, less invasive and can be repeated many times at the same site if needed, unlike radiation[6].

PDT presents some advantages over conventional antibiotic therapy, such as rapid elimination of target microorganisms (within seconds or minutes, depending on energy density and power used) and absence of maintenance of high concentrations of dye on lesions during hours or days as observed in conventional therapy. Due to production of singlet oxygen and free radicals, which are responsible for mediating bacterial killing, the development of resistance to lethal photosensitization by the target organisms would be a very unlikely event. Another advantage relates to the restriction of antimicrobial effects to the lesion through careful application of the dye and light source, without affecting the adjacent normal microflora. Also, PDT acts eliminating disease-causative microorganisms and their virulence factors[1].

LIMITATIONS[6]

Light needed to activate photosensitizer cannot penetrate more than 1cm of tissue depth using standard laser and low powered LED technology and hence is less effective in treatment of large tumors and

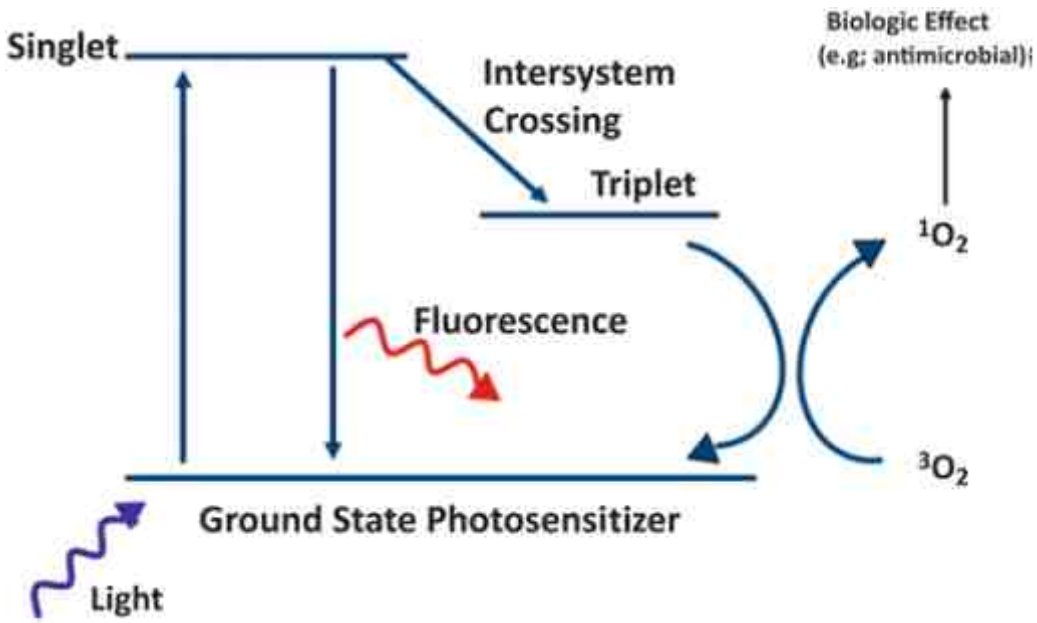


Figure1. Principles of PDT[15].

metastasis. It may leave many people very sensitive to light post therapy and cannot be used in people allergic to porphyrins.

Application of photodynamic therapy in dentistry.

Photodynamic therapy has been used in (i) photodynamic diagnosis of malignant transformation of oral lesions, (ii) treatment of premalignant and malignant oral lesions, (iii) chemotherapy (PACT) of bacterial and fungal infections, (iv) prevention of alveolar ostitis and postextraction pain, (v) decontamination of implant surface and prevention and treatment of peri-implantitis, (vi) endodontic treatment[13].

Photodynamic antimicrobial chemotherapy therapy[4]

Antimicrobial PDT can be considered as an adjunctive to conventional mechanical therapy. The liquid photosensitizer placed directly in the periodontal pocket can easily access the whole root surface before activation by the laser light through an optical fiber placed directly in the pocket.[17] As a result of the technical simplicity and the effective bacterial killing, the application of PDT in the treatment of periodontal diseases has been studied extensively. Antimicrobial PDT not only kills the bacteria, but may also lead to the detoxification of endotoxins such as lipopolysaccharide. These lipopolysaccharides treated by PDT do not stimulate the production of pro-inflammatory cytokines by mononuclear cells. Thus, PDT inactivate endotoxins by decreasing their biological activity.[4]

It has been demonstrated that bacteria associated with periodontal disease can be killed through photosensitization with toulidine blue O by irradiating with helium – neon soft laser. Data from an *in vitro* study indicated that PDT could kill bacteria organized in a biofilm. In an animal study, it was found that PDT was useful in reducing the redness, bleeding on probing, and porphyromonas gingivalis levels.[18]

Yilmaz *et al*. randomly assigned a total of 10 patients to receive repeated application of scaling and root planing with photodynamic therapy the other groups were receiving only scaling and root planing, photodynamic therapy, and oral hygiene instructions. Methylene blue served as the photosensitizer and was used as a mouth rinse. Significant clinical and microbiological improvement was seen within groups receiving scaling and root planing with photodynamic therapy and the scaling and root planing alone. However, improvement in groups receiving photodynamic therapy alone, as well as those receiving only oral hygiene instructions, did not reach significant levels. The reduced effectiveness of PDT may be the due the application of PDT from the external surface of the gingiva.[19]A study on 10 patients with aggressive periodontitis, in a split-mouth design to compare PDT using a laser source with a wavelength of 690 nm associated with a phenothiazine photosensitizer or scaling and root planning (SRP) with hand instruments; [20] to compare the CAL at baseline and three months after treatment with an automated periodontal probe, concludes that PDT and SRP show similar clinical results in the non-surgical treatment of aggressive periodontitis.

PDT has advantages such as reducing the treatment time, no need for anesthesia, destruction of bacteria in a very short period of time (<60 seconds), unlikely development of resistance by the target bacteria, and avoidable damage to the adjacent host tissues. Further studies using larger sample sizes are warranted to confirm these results[10].

An Adjunct In Non Surgical Periodontal Treatment[10]

Twenty-four subjects with chronic periodontitis were randomly treated with scaling and root planning followed by a single episode of PDT (test) and scaling and root planing alone (control). Gingival recession, and clinical attachment level (CAL) were measured at baseline and three, six months after therapy and it was concluded that the additional application of a single episode of PDT to scaling and root planing failed to result in an additional improvement in terms of pocket depth reduction and CAL gain, but it resulted in a significantly higher reduction in bleeding scores compared to scaling and root planning alone. Although mechanical removal of the

periodontal pathogens is the current gold standard of treatment in periodontics, antibiotics are also known to be effective. The use of antibiotics to destroy microorganisms (MO) selectively represents one of the most revolutionary progresses made in scientific medicine, resulting in the treatment and sometimes complete eradication of earlier incurable diseases. However, bacteria have developed resistance mechanisms against antimicrobial drugs which were previously highly effective. Besides, bacteria replicate very rapidly and a mutation that helps a MO to survive in the presence of an antibiotic will quickly become predominant in the microbial population. The use of photoactivable compounds or photo sensitizers (PS) to cause photodestruction of oral bacteria has been demonstrated, indicating that photodynamic therapy (PDT) could be a useful alternative to mechanical means as well as antibiotics in eliminating periopathogenic bacteria. Antimicrobial photodynamic therapy (aPDT) represents a potential alternative methodology to inactivate microbial cells and has already shown to be effective *in vitro* against bacteria, fungi, viruses, and protozoa.[13,21,22]

EFFECTS OF PHOTODYNAMIC ANTIMICROBIAL CHEMOTHERAPY ON ORAL BIOFILMS

A wide range of persistent human infections are due to microbial biofilms. Periodontal diseases result from accumulation of subgingival bacterial biofilms on tooth surfaces. Although mechanical removal of the periodontal pathogens is the current gold standard of treatment, antibiotics are also known to be effective. However, development of resistance in the target organisms is a problem associated with the use of such drugs. The use of photoactivatable compounds or photosensitizers (PS) to cause photodestruction of oral bacteria has been demonstrated, indicating that photodynamic therapy (PDT) could be a useful alternative to mechanical means as well as antibiotics in eliminating periopathogenic bacteria. The antimicrobial activity of photosensitizers is mediated by singlet oxygen, which, because of its high chemical reactivity, has a direct effect on extracellular molecules. Thus, the polysaccharides present in EMP of a bacterial biofilm are also susceptible to photodamage. Such dual activity, not exhibited by antibiotics, represents a significant advantage of PACT. Breaking down biofilms may inhibit plasmid exchange involved in the transfer of antibiotic resistance, and disrupt colonization. The photosensitive compounds are topically applied in the gingival sulcus and the laser is used to activate the compounds and complete the disinfection. Studies done by Braun et al., 2008 in patients with chronic periodontitis showed better clinical outcomes when PDT was used along with conventional therapy.[6,23]

Effect of PDT on Periodontal Bone Loss in Dental Furcations.[10]

The use of PDT in furcation involvement in induced periodontitis shows some advantages over the use of conventional antimicrobials, such as the reduced need for flap procedures and shorter treatment time; as local therapy, with lack of micro flora disturbance in other sites of the oral cavity. PDT is also beneficial during the maintenance of periodontal therapy because it may act on the biofilm and eliminate the need for the removal of additional root substance by mechanical retreatment. Thus, the patient may experience less dentinal hypersensitivity. This therapy also serves as an adjunct to mechanical therapy in sites with difficult access.

Effect of a PDT on peri-implantitis

Peri-implantitis seems to occur in 5-10% of all implant cases. In this way, photodynamic therapy can be used successfully to decontaminate the implant surface.[13, 24] Laser PDT can be used in implantology to promote osseointegration and to prevent peri-implantitis. Studies have shown that laser photobiomodulation can be successfully used to improve bone quality around dental implants, allowing early wearing of prostheses. The results of a study showed significant differences on the concentration of calcium hydrxyapatite on irradiated and control specimens and concluded that infrared laser photobiomodulation does improve bone healing. The percentage of bone fill and re-osseointegration also improved with photobiomodulation.[25]

One of the most interesting developments over the last years has been the introduction of the 9.6-µm CO_2 laser. It has been shown in the recent literature that the use of this new device can preserve tissue, with almost no adverse effects at the light microscopic level. Intraoperatively used PDT or peri-implant care of ailing implants with the CO_2 laser seems to be more of value than the conventional methods. Data suggest that lethal photosensitization may have potential in the treatment of peri-implantitis.[13,24]

CONCLUSION

Antimicrobial PDT seems to be a unique and interesting therapeutic approach towards periodontal therapy[4]. PDT application has an adjunctive benefit besides mechanical treatment at sites with difficult access (e.g. furcations, deep invaginations, concavities). Necessity for flap operations may be reduced, patient comfort may increase and treatment time decrease. PDT removes the biofilm in residual deep pockets during maintenance; no more root substance is removed by mechanical retreatment. Thus the patient may experience less dentinal hypersensitivity. PDT may decrease the risk of bacteremia, which routinely occurs after periodontal treatment procedure.[10] Antimicrobial photodynamic therapy may hold promise as a substitute for currently available chemotherapy in the treatment of periodontal and peri-implant diseases[4]. Its nonsurgical profile improves the comfort of treatment and thus makes the process more attractive to patients. Its ease of use makes it suitable for dentists.

Treatment regimens still have to be optimized and standardized for better therapeutic effectiveness. Severe side effects have been reported when using inappropriate PDT schedules.

Appropriate choices of drug type and dose, light wavelength, and drug-light interval can improve the efficacy and safety of PDT. Furthermore, careful attention to the physics and dosimetry of light will help to minimize toxicity[16].

REFERENCES

1. Fabiano Dalla Lana Mattiello, Alan Augusto Kalife Coelho, Odair Pimentel Martins et al . *In Vitro* Effect of photodynamic therapy on *Aggregatibacter actinomycetemcomitans* and *Streptococcus sanguinis.* Braz. Dent. J.2011;22(5)
2. Mark Wainwright. Photodynamic antimicrobial chemotherapy (PACT). Journal of antimicrobial chemotherapy 1998;42:13-28.
3. Kornman KS, Page RC, Tonetti MS. The host response to the microbial challenge in periodontitis: Assembling the players. Periodontol 2000. 1997;14:33-53.
4. S. Rajesh, Elizabeth Koshi, Koshi Philip, and Aparna Mohan. Antimicrobial photodynamic therapy: An overview. J Indian Soc Periodontol. 2011 Oct-Dec; 15(4):323–327.
5. Wang SS, J Chen, L Keltner et al. "New technology for deep light distribution in tissue for phototherapy". *Cancer Journal* 2002;8 (2):154-63.
6. Veerendra Nath Reddy, Rekha Rani K, Chandana G et al. Photodynamic Therapy. IJDA 2009;1(1):46.
7. Lui H, Anderson RR. Photodynamic therapy in dermatology: Shedding a different light on Skin disease. Arch Dermatol. 1992;128:1631-6.
8. Ackroyd R, Kelty C, Brown N, Reed M. The history of photodetection and photodynamic therapy. Photochem Photobiol 2001;74:656-69.
9. TM Busch. Biophysical determinants of photodynamic therapy and approaches to improve outcomes. Dept of Radiation Oncology, University of Peninsylvania, Philadelphia 2006.
10. Rajvir Malik, Anish Manocha, DK Suresh. Photodynamic therapy - A strategic review: Indian Journal of Dental Research. 2010;21(2):285-91.

11. V Shivakumar, M Shanmugam, G Sudhir, S Pavithra Priyadarshoni . Scope of photodynamic therapy in periodontics and other fields of dentistry. Journal of interdisciplinary dentistry 2012;2(2):78-83.

12. Allison RR, Mota HC, Sibata CH. Clinical PD/PDT in North America: An historical review. Photodiagn Photodyn Ther 2004;1:263-77.

13. Sruthima NVS Gottumukkala, Satyanarayana Raju Mantena. Photodynamics in antimicrobial periodontal therapy. Indian J Oral Sci 2012;3 (1):8-12

14. Nitzan Y, Shainberg B, Malik Z. Photodynamic effects of deuteroporphyrin on Gram positive bacteria. Curr Microbiol 1987;15:251-8.

15. Amir Azarpazhooh, Prakesh S. Shah, Howard C. Tenenbaum and Michael B. Goldberg. The Effect of Photodynamic Therapy for Periodontitis: A Systematic Review and Meta-Analysis. J Periodontol January 2010;81(1):4-14.

16. Sudhakara Reddy R, Ramya Kotha, Ramesh Tatapudi et al. Photo Dynamic Therapy in Oral Diseases. Int J Biol Med Res. 2012;3(2):1875-1883

17. Andersen R, Loebel N, Hammond D, Wilson M. Treatment of periodontal diseases by photodisinfection compared to scaling and root planing. J Clin Dent. 2007;18:34-8.

18. Sigusch BW, Pfitzner A, Albrecht V, Glockmann E. Efficacy of photodynamic therapy on inflammatory signs and two selected periodontopathogenic species in a beagle dog model. J Periodontol. 2005;76:1100-5.

19. Yilmaz S, Kuru I, Kuru L, Noyan U, Argun D, Kadir T. Effect of gallium arsenide diode laser on human periodontal diseases; a microbiological and clinical study. Lasers Surg Med. 2002;30:60-6.

20. de Oliveira RR, Schwartz-Filho HO, Novaes AB Jr, Mario Taba Jr. Antimicrobial photodynamic therapy in the non-surgical treatment of aggressive periodontitis: A preliminary randomised controlled clinical study. J Periodontol 2007;78:965-73.

21. Alves E, Costa L, Carvalho C, Tome J, Faustino M, Neves M, *et al*. Charge effect on the photoinactivation of Gram-negative and Gram-positive bacteria by cationic meso-substituted porphyrins. BMC Microbiol 2009;9:70-83.

22. Alves E, Carvalho CM, Tomé JP, Faustino MA, Neves M, Tomé AC, *et al*. Photodynamic inactivation of recombinant bioluminescent Escherichia coli by cationic porphyrins under artificial and solar irradiation. J Ind Microbiol Biotechnol 2008;35:1447-54.

23. Braun A, Dehn C, et al. Short term clinical effects of adjunctive antimicrobial photodynamic therapy in periodontal treatment: a randomized clinical trial. Journal of Clinical Periodontology 2008; 35:878-884.

24. Dortbudak O, Haas R, Bernhart T. Lethal photosensitization for decontamination of implant surfaces in the treatment of peri-implantitis. Clin Oral Implants Res 2001; 12:104-8.

25. Shibli JA, Martins MC, Nociti FH Jr, Garcia VG, Marcantonio E Jr. Treatment of ligature-induced peri-implantitis by lethal photosensitization and guided bone regeneration: a preliminary histologic study in dogs. J Periodontal 2003;74:338-45.

REVIEW ARTICLE

DIGITAL IMPRESSIONS IN DENTISTRY

Authors:
Meenu Merry C Paul[1],
Sanu Tom Abraham[2],
Pius AV[3],
Shibu George[3]

[1] Professor &HOD,
Dept. of Prosthodontics,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

[2]Professor &HOD,
Dept. of Orthodontics,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

[3]Professor, Dept. of Prosthodontics,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

Address for correspondence:
Dr. Meenu Merry C Paul,
Professor and Head, Dept. of Prosthodontics,
Indira Gandhi institute of Dental Sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.
E-mail: meenumerry@yahoo.com

ABSTRACT

The quest for innovations and improvisations in all the areas of restorative dentistry includes impression materials as well. Precision and accuracy of master impressions are critical to the overall excellence and marginal fit of definitive fixed restorations &implants. In the present scenario, conventional (traditional) techniques are being replaced by the newer, precise and accurate digital impressions. Chairside digital impression (CAD/CAM) systems create accurate and precise laboratory models and restorations, involving less chairside time, and achieve fine-tuned esthetics. This article gives an insight into the latest impression materials/techniques used to fabricate a fixed partial denture or implant prosthesis.

Key words: CAD-CAM Impressions, Digital Dental Impressions.

J Odontol Res 2013;1(1):63-7.

DIGITAL IMPRESSIONS

The key to success in restorative dentistry is the fabrication of a healthy, maintainable, aesthetic and functional prostheses. The long chain of positive and negative dimensional pitfalls between completion of the preparation and cementation of a fixed prosthesis *begins with the introduction.*

What is an impression - Is it an effect? or a belief? or an imprint?....

Innovations and improvisations in all the areas of restorative dentistry include impression materials as well. In the present scenario, conventional techniques are being replaced by the newer digital impressions.

1. Impressions with Conventional (traditional) materials

Agar and Alginate hydrocolloids were used initially. But, their usage has been limited due to many drawbacks[1,5]. *Elastomers* have proved to be the successful based on their clinical performance.[2, 3, 4]

Among the rubber base materials, Vinyl Poly Siloxanes (VPS) and Polyethers are recommended materials for FPD and implant impressions.[6] Improvements in flow characteristics and hydrophilic properties have made them popular. *Polyether materials* are ideal in situations where tissue management is difficult.[7,8].

Vinylsiloxanether(2009) a hybrid silicone material - is a combination of a polyether material and Polyvinylsiloxane having properties equivalent to or superior to those of polyether material.[19]

Variability in accuracy has been found in impressions and the resulting casts depending on the technique and material used[9]

2. Digital impressions

Digital impression is the newest innovation in this field. The dental industry changed when the University of Zurich introduced the original CEREC machine in 1980. It was the first to use digital impression technology to scan a patient's mouth, virtually design a restoration, and then mill it for immediate seating in the patient's mouth.[10,11] (Figure -1).

Advantages of digital impressions[12-14]

- Greater precision and accuracy.
- Can be used in a patient who is a gagger or cannot tolerate impression material in his or her mouth for several minutes
- Takes less time than conventional impressions, including the bite registration (scanning takes only three to four minutes).
- Easy to make impressions when mandibular or maxillary tori or other undercuts are present. Removal of a traditional impression is difficult or impossible without causing the patient discomfort and/or tearing the margins on the impression.
- No need of repeated impressions (no material wastage).
- Shade guide stumps can be photographed overlaid on the tooth, which helps to highlight similarities and differences in areas of the tooth for custom shading and provides information on the initial preparation shade.
- Preparation is instantly visible and enlarged on-screen.
- No disinfection protocol is required before it is sent to a laboratory, as there is no physical impression.
- Compatibility of impression material with specific die materials is not a concern.

Disadvantages of digital impressions[12-14]

- CAD/CAM scanners require a dry, visible field for scanning (as in a traditional impression).
- Digital scanning must include proper tissue management to ensure accuracy. Soft tissue retraction and moisture control are essential in this process (these are also essential for traditional impressions).
- Expensive - Cost/the initial investment in the machine needs to be considered. (The real savings is in the indirect costs associated with reduced seating time, fewer remakes, and fewer less-than-ideal impressions and subsequent restorations).

CAD/CAM systems are available which either digitally scan and create fixed restorations chairside (in-office CAD/CAM) or capture chairside digital impressions that are then sent to a laboratory.

In-office CAD/CAM:

- allows the clinician to provide single-visit indirect fixed restorations that are accurate and esthetically pleasing.
- does not require any communication with a laboratory

Chairside digital impression

- allows the creation of accurate models that can then be used for either traditional or CAD/CAM fabrication of restorations, and involves less chairside time.
- They enable seamless communication between the clinician and the laboratory technician.

The procedure for digitizing the impression is:-

- After the tooth preparation, the area is captured with an optical device and displayed on the computer. (Using different technologies, each system uses a camera to capture the desired image.)
- The impression surface is studied and if needed, modification of preparation /additional images can be made.
- The final image is digitally sent to the fabrication center
- The impression is analysed by the technician and he digitally trims the model and the dies.
- Then the actual physical model is made.
- This physical model is sent to the laboratory for fabrication of prosthesis.

Scanning the impression The model and intraoral scanners scan the tooth and model surface contours, regardless of the particular technology employed. Light in one form or another is projected onto a tooth or model surface. One or more cameras record the reflected light, and the sophisticated software interprets the data, producing a digital model on which restorations can be designed.

A digital scan should capture the entire restorative margin as well as approximately 0.5 mm of the tooth/root surface apical to the margin. This information is required by the ceramist or milling machine in order to reproduce the correct emergence profile, or "egression silhouette" for the final restoration.[15]

Depending on whether the restorative margin is supracrevicular (above the gingival tissues), equicrevicular (at the free gingival margin) or intracrevicular (in the gingival sulcus), either a traditional single- or double-cord technique, laser technique, chemical retraction technique, or a combination of these can be used to achieve a dry and visible field. For intracrevicular and equicrevicular margins, a double-cord tissue retraction technique can be used, with the more superficial cord removed gently just prior to scanning. If using a laser to trough the area, thereby creating a space between the preparation margin and the tissue (which will also aid hemostasis), it is important to consider the patient's tissue type and the principles of biologic width first; there must be sufficient horizontal tissue thickness to avoid loss of vertical tissue height.[16, 17, 18]

One of the biggest challenges in scanning an impression is the technology's ability to "see" inside the voids that the teeth create in the impression material. Many older model scanners are unable to see inside these voids because the angle between the light source and the camera is too great. So in order to scan impressions, manufacturers decrease the angle between the light source and the camera, giving them a better view inside those voids. Due to their size, intraoral scanners have highly condensed optics and adapt quite well to scanning impressions.

One difference between the various CAD/CAM systems is the requirement for powdering. Some systems require a coating of reflective powder or light powdering on the dry preparation prior to scanning. Whereas, some do not require powdering.

After Capturing the data Next step is to convert the scanned impression data into a physical model so that a traditional restoration or CAD/CAM-based restoration can be fabricated.

Once the virtual model has been created, it still needs to be manufactured. This requires a CAM program to take the model data and a CAM milling or 3-D printing system to produce the physical model . (Fig-3)

CAD/CAM dentistry is changing the way in which clinicians provide indirect restorations to patients, with fabrication of highly precise, accurate models and restorations; increased chairside productivity; and improved clinic-laboratory communication.

CONCLUSION

Digitizing the oral environment is a recent innovation, both chairside and in the laboratory. CAD and CAM software works with scanned impressions, and model printing and milling systems are being developed. Chairside digital impression systems

(Figure -1).

(Fig-2)

(Fig-3)

allow for the creation of accurate and precise laboratory models and restorations, involve less chairside time, and achieve fine-tuned esthetics. It is a promising future, since the CAD/CAM revolution is in full swing...

REFERENCES

1. Craig RG. Review of dental impression materials. Adv Dent Res 1988;2:51
2. Chee WWL, Donovan TE. Polyvinyl siloxane impression materials: A review of properties and techniques. J Prosthet Dent 1992;68:728.
3. Pratten DH, Novetsky M. Detail reproduction of soft tissue: A Comparison of impression materials. J Prosthet Dent 1991;65:188
4. Salem NS., Combe EC., Watts DC. Mechanical properties of elastomeric impression materials. J Oral Rehab 1988;15:125
5. Miller MW: Syneresis in alginate impression materials. Br Dent J 1975;139:425.
6. Lee H et .al: The accuracy of implant impressions. J Prosthet Dent 100(4):285-91,2008
7. Mondon M, Ziegler Ch., Reusch B : Contact Angle Studies on Unset Light Body

Impression Materials AADR(American Association for Dental Research) 618 2001

8. Klettke Th., Kuppermann B., Fuhrer C., Richter B : Hydrophilicity of Precision Impression Materials During Working Time CED/IADR 141,2004
9. Alhouri N, McCord JF, Smith PW. The quality of dental casts used incrown and bridgework. Br Dent J. 2004;197(5):261-4.
10. Beuer F, Schweiger J, Edelhoff D. Digital dentistry: an overview of recent developments for CAD/CAM generated restorations. Br Dent J. 2008;204(9):505-11.
11. Giordano R. Materials for chairside CAD/CAM-produced restorations. J Am Dent Assoc. 2006;137(suppl):14S-21S.
12. Otto T, Schneider D. Long-term clinical results of chairside CEREC CAD/CAM inlays and onlays: A case series. Int J Prosthodont.2008;21(1):53-9.
13. Sjogren G, Molin M, Van Dijken JW. A 10-year prospective evaluation of CAD/CAM-manufactured (CEREC) ceramic inlays cemented with a chemically cured or dual-cured resin composite. Int J Prosthodont. 2004;17(2):241-6.
14. Posselt A, Kerschbaum T. Longevity of chairside CEREC inlays and onlays. Int J Comput Dent. 2003;6:231-48.
15. Shavell HM. The periodontal-restorative interface in fixed prosthodontics: tooth preparation, provisionalization, and biologic final impressions. Part I. Pract Periodontics Aesthet Dent. 1994;6(1):33-44.
16. Gunay H, Seeger A, Tschernitschek H, Geurtsen W. Placement of the preparation line and periodontal health: A prospective 2-year clinical study. Int J Periodontics Restorative Dent. 2000;20(2):171- 81.
17. Padbury A Jr, Eber R, Wang HL. Interactions between the gingiva and the margin of restorations. J Clin Periodontol. 2003;30(5):379-85.
18. Robert A L. CAD/CAM Dentistry and chairside digital impression making.
19. Enkling N et. al.Vinylsiloxanether: a new impression material. Clinical study of implant impressions with vinylsiloxanether versus polyether materials. Clin Implant Dent Relat Res 14(1):144-51, 2012

REVIEW ARTICLE

WOMEN'S ORAL HEALTH: AN OVERVIEW

Authors:
Amit V Mahuli[1],
Simpy Mittal[1],
Prashanth GM[2],
Chandu GN[3],
Saurabh Kakade[4].

[1]Senior Lecturer,
Dept. of Public Health Dentistry,
Dr. DY Patil Dental College and
Hospital, Pimpri, Pune, India.

[2]Reader,
Department of Preventive and
Community Dentistry,
College of Dental Sciences,
Davangere. 577 004,
Karnataka, India.

[3]Professor,
Department of Preventive and
Community Dentistry,
College of Dental Sciences,
Davangere. 577 004,
Karnataka, India.

[4]Post Graduate Student,
Dept. of Public Health Dentistry,
Dr. DY Patil Dental College and
Hospital, Pimpri, Pune, India.

Address for correspondence:
Dr. Amit V. Mahuli,
Senior Lecturer, Dept. of Public
Health Dentistry, Dr. DY Patil Dental
College and Hospital, Pimpri, Pune,
India.
E mail: amitmahuli@gmail.com.

ABSTRACT

Women's oral health demands special attention at different times during their lifespan. In this present article the main oral health needs and their dental management have been highlighted. Women's oral health needs during, puberty, menses, oral contraceptive taking phase, pregnancy & post-menopausal period have been explained. In women, certain diseases or conditions are unique, more prevalent, more serious, or have different manifestations or different outcomes than in men. This dimorphism is influenced greatly by the hormonal fluctuations that women experience and is reflected in the health of their oral tissues. Few of the social factors affecting oral health of the Indian women are also been discussed. Education and poor socio-economic status are the major barriers in women's oral health in India. There are gender differences in the culture, practice, attitudes and disease patterns in India, there are acute need of studies related to gender differences of oral diseases and behavior of Indian women towards oral hygiene.

Key words: Women's oral health, temporomandibular joint disorders, Sjogren's syndrome.

J OdontolRes 2013;(1)1:68-75

INTRODUCTION

Women's health is one of the most important public health challenges. As interest in women's health issues grows, there is increasing concern that today's practice of medicine may not meet the health needs of women. A primary reason is the gender bias that has been inherent in medical education, research and clinical practice. The prevailing medical viewpoint has often been that the male body is considered to be the norm and that the female body exactly the same except for the reproductive function. This attitude has led to a lack of interest in researching gender differences and a consequent lack of knowledge of women's health issues.

Being a man or a woman has a significant impact on health, as a result of both biological and gender-related differences. The health of women and girls is of particular concern because, in many societies, they are disadvantaged by discrimination rooted in socio-cultural factors. For example, women and girls face increased vulnerability to HIV/AIDS.[1-4]

Some of the socio-cultural factors that prevent women and girls to benefit from quality health services and attaining the best possible level of health include:

- Unequal power relationships between men and women;
- Social norms that decrease education and paid employment opportunities;
- An exclusive focus on women's reproductive roles; and
- Potential or actual experience of physical, sexual and emotional violence

Women have special oral health needs and considerations. Hormonal fluctuations have a surprisingly strong influence on the oral cavity. Puberty, menses, pregnancy, menopause and use of contraceptive medications all influence women's oral health and the way in which a dentist should approach treatment.

The revolution of women's health care began during the early years of the feminist movement in the 1960s. At that time little was known about gender-specific health issues even among medical professionals. The activism of that decade, ranging from grass-roots demonstrations to Congressional hearings on oral contraceptives to the development of the National Women's Health Network, brought attention to a wide range of gender-specific health issues.[2-9]

In 1983, the federal government established the Public Health Service Task Force on Women's Health Issues.[3]

In 1990, the National Institutes of Health (NIH) created the Office of Research on Women's Health (ORWH). The ORWH was given a tripartite mandate:

1. To determine gaps in scientific knowledge regarding gender- specific conditions and diseases and to establish a research agenda to address these gaps;
2. To ensure that women are represented appropriately in research studies, especially clinical trials supported by the NIH;
3. To create specific initiatives to increase the number of women in biomedical careers and to facilitate their advancement and promotion.

As one part of updating the NIH agenda on women's health, a working group was identified specifically to examine the progress and to assess needs related to women's oral health as a significant aspect of women's general health. This working group focused on developing strategies to integrate women's oral health issues into the overall women's health agenda to improve health and the quality of life for women.[11-14]

Table1: WOMEN'S ORAL HEALTH ACROSS THE LIFESPAN[15-31]

Woman's Medical History	Oral Manifestation	Dental Management
Puberty	● Oral changes due to poor nutrition, fad diets, and eating disorders:caries, dental erosion, delayed healing, increased bone loss,other changes in lips, oral mucosa, periodontium ● Puberty gingivitis	Local preventive care, including a vigorous program of good oral hygiene. Mild gingivitis - scaling and improved oral hygiene. Severegingivitis - aggressive treatment, including antimicrobial therapy. If the patient's gingivitis does not respond, more frequent recall during puberty may be indicated.[15-17]
Menstruation	Menstruation gingivitis ● Buccalmucosal ulcerations ● Increased sensitivity to oral pain	Local preventive care, including a vigorous program of good oral hygiene is vital. Topical and/or systemic antiherpetic medication may be beneficial for patients experiencing recurrent herpetic outbreaks. Topical corticosteroids may also be indicated for severe aphthous ulcers. Palliative treatment, such as topical anesthetic agents and/or systemic analgesics, may be necessary for the discomfort associated with the aphthous ulcerations and herpetic lesions[18-21]
Oral Contra-ceptives	Gingivitis ● Concern over effectiveness of oral contraceptives with use of antibiotics ● Increased risk of post-surgical dry socket late in pill cycle	A comprehensive medical history and assessment of vital signs, including blood pressure, are extremely important in this group of patients. Treatment of gingival inflammation exaggerated by oral contraceptives should include establishing an oral hygiene program and eliminating all local predisposing factors. Periodontal surgery may be indicated if there is inadequate resolution after initial therapy (scaling, root planing and curettage). Antimicrobial mouthwashes may be indicated as part of the home care regimen. A recent report from the ADA Council on Scientific Affairs[22] noted that, considering the possible consequences of an unwanted pregnancy, when prescribing antibiotics to a patient using oral contraceptives, the dentist should: advise the patient to maintain compliance with oral contraceptives when concurrently using antibiotics. advise the patient of the potential risk for the antibiotic's reduction of the effectiveness of the oral contraceptive. recommend that the patient discuss with her physician the use of an additional nonhormonal means of contraception.

		Although in the literature, oral manifestations have been attributed to oral contraceptive use, it can be presumed that the same effects could occur with the use of other contraceptive medications (e.g., implants, transdermal patches).
Pregnancy	● Pregnancy gingivitis ● Increased risk of pre-term, low-birth-weight babies with periodontal disease ● Increased risk of delayed tooth formation and maturation with heavy smoking ● Concern on timing of prescribed medications and breast feeding	Elective treatment should be avoided during the first trimester and the last half of the third trimester. The first trimester is the period of organogenesis when the fetus is highly vulnerable to environmental influences. There is a hazard of premature delivery in the last half of the third trimester, and the patient may also be very uncomfortable in the dental chair. The second trimester is the safest period for treatment, and the focus should be on controlling active disease and eliminating potential problems that could occur later in the pregnancy Irradiation should be avoided if possible, especially in the first trimester. The patient's physician should be consulted if there is a need to prescribe medications or use general anesthesia.[23,24]
Menopause	● Menopausal gingivostomatitis ● Xerostomia (dry mouth) Osteoporosis	Preventive oral prophylaxis. *Sialogogue* A concern for dentists, especially with regard to removable prosthodontics, is the condition of the mandibular residual ridge. When patients exhibit rapid continuing bone resorption under a well-fitting dental prosthesis, osteoporotic bone loss may need to be considered as contributing to the etiology and pathogenesis of the resorptive process. Postmenopausal osteoporotic women may require new dentures more often after age 50 than women without osteoporosis. The bone loss may become so severe that fabrication of a functional prosthesis may become difficult. Bone regeneration techniques and dental implants may be of significant benefit to an osteoporotic patient who has experienced decreased function of a denture. Because most dental implants depend on sufficient bone volume and density for success, bone regeneration therapy may be necessary prior to implant placement. It appears that there is no contraindication for osseointegrated implant therapy in the osteoporotic patient.[18,21,25-31]

ORAL HEALTH ISSUES UNIQUE TO WOMEN

In women, certain diseases or conditions are unique, more prevalent, more serious, or have different manifestations or different outcomes than in men. This dimorphism is influenced greatly by the hormonal fluctuations that women experience and is reflected in the health of their oral tissues. Oral health statistics, however, are generally more favorable for women.[21,32]

Oral diseases are complex in that they involve multiple genes and gene susceptibility factors interacting with behavioral and environmental variables. Because of the interaction of these variables, significant numbers of women are at risk for developing oral diseases. Because women generally live longer than men, women are mere likely to experience multiple chronic conditions, multiple medications (polypharmacy), cognitive impairments, compromised functional status, and physical confinement. Significant numbers of women live in poverty or are the single head of a family without the ability to pay for dental care. Also, many women defer their own dental care for the sake of attending to the care of their children and other family members. Certain behavioral patterns, including smoking, unprotected sexual activity, and binging and purging, are increasing for women and these behaviors will probably lead to increases in oral cancer, AIDS-related oral lesions, tooth erosion, and tooth decay.[29,31,33,34]

Certain diseases that affect the oral cavity are more common in women: temporomandibular joint disorders (TMD) and myofacial pain, trigeminal neuralgia, Sjogren's syndrome (90% are women), burning mouth syndrome, and eating disorders. Gingivitis during pregnancy and oral changes associated with menopause certainly are unique to women. In older women, osteoporosis, degenerative rheumatoid arthritis, and diabetes mellitus are more prevalent.[35-39]

Because the health of women's body and oral cavity is bidirectional, a woman must adopt strategies that promote both her general health and that of her oral cavity. Such an approach will allow her to maintain her oral health and maximize the quality of her life.

WOMEN'S ORAL HEALTH: INDIAN SCENARIO

Women's oral health in India is dependent on lots of factors, education being one the most important factor. Educated women have good knowledge for maintenance of the oral hygiene.

The various factors responsible for the deteriorating state of affairs regarding women,s health and oral health in India are:

- India still has one of the lowest female literacy rates in Asia.
- Large Differences in Literacy Among the States
- Three Out of Five Girls Attend School Versus Three Out of Four Boys
- Beyond Literacy: Although there are numerous studies demonstrating a link between education and a variety of demographic indicators (i.e., fertility, infant and child mortality and morbidity), more recent studies are finding that there is a minimum threshold of education (more than 5 or 6 years) that must be achieved before there are significant improvements in female autonomy[3], particularly in a percent of all Indian women, have more than a primary education.
- Gender Gaps in University Education
- Barriers to Education: There are several reasons for the low levels of literacy in India, not the least of which is the high level of poverty. Over one-third of the population is estimated to be living below the poverty line (The World Bank, 1997). Although school attendance is free, the costs of books, uniforms, and transportation to school can be too much for poor families. Poor families are also more likely to keep girls at home to care for younger siblings or to work in family enterprises. If a family has to choose between educating a son or a daughter because of financial restrictions, typically the son will be chosen.[42]Negative parental attitudes toward educating daughters can also be a barrier to a

girl's education. Also, daughters with higher levels of education will likely have higher dowry expenses as they will want a comparably educated husband. However, education sometimes lowers the dowry for a girl because it is viewed as an asset by the husband's family.

- Inadequate School Facilities
- India Has a Shortage of Female Teachers
- Gender Bias in Curriculum Still Exists: a study of Indian textbooks done in the 1980s found that men were the main characters in the majority of lessons. In these lessons, men held high-prestige occupations and were portrayed as strong, adventurous, and intelligent. In contrast, when women were included they were depicted as weak and helpless, often as the victims of abuse and beatings (Kalia, 1988). These depictions are strong barriers for improving women's position in society.

Education and poor socio-economic status are the major barriers in women's oral health in India. There are gender differences in the culture, practice, attitudes and disease patterns in India, there are acute need of studies related to gender differences of oral diseases and behavior of Indian women towards oral hygiene.

CONCLUSION

Health needs of women are different mainly because of the distinct changes that occur over their lifetime: puberty or menopause, as well as specific times, such as pregnancy. A woman's oral health needs can also change at these times, thus affecting their dental treatment plans. In the past, research on women's health has been unfairly neglected. More research should be encouraged to address the gender differences in the various aspects of general and oral health. Women's oral health is dependent on social factors like education, equality etc. Hence, improvement of women's oral health requires a multipronged approach which addresses these issues and leads uplifting of women's status in the society on the whole.

REFERENCES

1. Women's health. World Health Organization. [Online] Cited on 2008 Dec 21 Available from: URL: http://www.who.int/topics/womens_health/en/(accessed on 26th Jan 2010)
2. National woman's health information center web site. [Online] Cited on 2008 Dec 21 Available from: URL: www.4women.gov/owh/pub/history/healthmvmt.htm. (accessed on 26th Jan 2010)
3. PINN VW. Women's health research for the twenty-first century. J Dent Educ. 1999;63 223-230.
4. National Institute of Health, Division of Research Grants: NIH guide to grant and contracts. January 23,1987.
5. Executive Summary: Agenda for Research on Women's Health for the 21st Century. In Report of the Task Force on the NIH Women's Health Research Agenda for the 21st Century, Volume 1. Bethesda, MD, US Department of Health and Human Services, Public Health Service, National Institutues of Health,1999, NIH Publication No. 99-4385.
6. Sinkford JC, Valachovic RW, Harrison SG. Women's Oral Health: The Evolving Science. J Dent Educ. 2008; 72(2):131-134.
7. Sumaya CV, Pinn VW, Blumenthal SJ: Women's health in the medical school curriculum: Report of a survey and recommendations. Bethesda, MD: Health Resources and Services Administration, National Institutes of Health, Department of Health and Human Services, 1996.
8. Silverton S, Sinkford J, Inglehart M, et al: Women's health in the dental school curriculum: Report of a survey and recommendations. Bethesda, MD: US Department of Health and Human Services, Health Resources and Services Administration, and the National Institutes of Health, 1999, Publication No. 99-4399.

9. Slavkin HC. The Human Genome, Implications for Oral Health and Diseases, and Dental Education. J Dent Educ. 2001;65(5):463-479.

10. Jeffcoat MK, Hauth JC, Geurs NC, et al. Periodontal disease and preterm birth: Results of a pilot intervention study. J. Periodontol. 2003;74:1214-18.

11. Offenbacher S, Katz V, Fertik G, et al. Periodontal infection as a possible risk factor for preterm low birth weight. J Periodontol 1996;67:1103-13.

12. Centers for Disease Control and Prevention. Recommendations for using fluoride to prevent and control dental caries in the U.S. 2001. Morb Mort Wkly Rep. 2001;50:1-41.

13. American Dental Association/U.S. Department of Health and Human Services. The Selection of Patients for Dental Radiographic Examinations-2004. (Accessed on November 2010) Available from: URL: http://www.ada.org/prof/resources/topics/topics radiography examinations.pdf

14. Ciancio, SG. ADA guide to dental therapeutics. 3rd ed. Chicago: American Dental Association;2003.

15. Briggs GG, Freeman RK, Yaffe SJ. Drugs in pregnancy and lactation. 4th ed. Baltimore: Williams and Wilkins; 1994.

16. Olin BR, ed. Drug facts and comparisons. St. Louis: Walters Kluwer; 1994.

17. Family Violence Prevention Fund, San Francisco, 2005. Enhancing Dental Professionals' Response to Domestic Violence. Website: www.endabuse.org/health Go to Health Material Index, then Dental Folio.(accessed on 26th Jan 2010)

18. National Domestic Violence Hotline Website: www.ndvh.org (accessed on 26th Jan 2010)

19. Brown S, Bonifaz DZ. An overview of anorexia and bulimia nervosa and the impact of eating disorders on the oral cavity. CompendContinEduc Dent 1993; 14(12):1594-1608.

20. Mandel L, Kaynar A. Bulimia and parotid swelling: a review and case report. J Oral MaxillofacSurg 1992; 50:1122-5.

21. Amar S, Chung KM. Influence of hormonal variation on the periodontium in women. Periodontol 2000.1994;6:79-87.

22. American Dental Association. Council on Scientific Affairs. Antibiotic interference with oral contraceptives. JAm Dent Assoc 2002;133:880.

23. Hatch JP, Rugh JD, Sakai S, Saunders MJ. Is use of exogenous estrogen associated with temporomandibular signs and symptoms? JAm Dent Assoc 2001;132:319-26.

24. Friedlander AH. The physiology, medical management and oral implications of menopause. JAm Dent Assoc 2002;133:73-9.

25. Jeffcoat MK, Chestnut CH, III. Systemic osteoporosis and oral bone loss evidence shows increased risk factors. JADA 1993;124:49-56.

26. Ruggiero SL, Mehrotra B, Osteonecrosis of the jaws associated with the use of bisphosphonates: a review of 63 cases. J Oral Maxillofac Surg. 2004;62:527-34.

27. Jeffcoat MK, Lewis CE, Reddy MS, Wang CY, Redford M. Postmenopausal bone loss and its relationship to oral bone loss. Periodontol 2000;23:94-102.

28. Ship J, Grushka M, Lipton JA, Mott AE, Sessle BJ, Dionne RA. Burning mouth syndrome: an update. JADA 1995; 16:842-53.

29. Epstein JB, Gorsky M. Burning mouth syndrome. Am Fam Physician 2002; 65:615-622.

30. Baum BJ. Salivary gland fluid secretion during aging. J Am GeriatrSoc 1989; 37:453-8.

31. Navazesh M. How Can Oral Health Care Providers Determine if Patients Have Dry Mouth? JADA 2003; 134:613-20.

32. Al-Hashimi I. The management of Sjogren's syndrome in dental practice. JADA 2001;132:1409-17.

33. Pintos A, Glick M. Management of patients with thyroid disease. Oral health considerations. JADA 2002; 133:849-58.

34. American Academy of Orthopaedic Surgeons: Antibiotic prophylaxis for dental patients with total joint replacements. J Am Dent Assoc 1997; 82:16-7.

35. American Academy of Periodontology: Tobacco use and the periodontal patient. J Periodontol. 1999;70:1419-27.

36. Beck J, Garcia R, Heiss G, et al: Periodontal disease and cardiovascular disease. J Periodontol. 1996;67:1123-37.

37. Center for Disease Control and Prevention: Fetal alcohol syndrome-United States, 1979-1992. MMWR Morb Mortal Wkly Rep. 1993;42:339-41.

38. Genco RJ: Current view of risk factors for periodontal diseases. J Periodontol 1996;67:1041-9.

39. Chernoff R: Nutritional need of elderly women. In Krummel DA, Kris-Etherton PM (eds): Nutrition in Women's Health. Gaithersburg, MD, Aspen Publishers, 1996,p:299-321.

40. Mifflin MD, St. Jeor ST, Hill LA, et al: A new predictive equation for resting energy expenditure in healthy individuals. American Journal of Nutrition. 1990;51:241-247.

41. Mattana D, Zarkowsi P, Wyche C, et al: Perimenopausal women: Oral health status and preventive strategies. Journal of Women & Aging 1996;8:21-32.

42. Velkoff VA. International Programs Center. Women's Education in India.Women of the World. Issued on October 1998.

REVIEW ARTICLE

BLEACHING, THE BIOLOGIC ASPECTS - A REVIEW

Authors:
Romel Joseph[1],
Dinesh Kamath[2],
Robin James[2],
Meera Gopalakrishnan[3]

[1]Professor and Head,
Dept. of Conservative Dentistry and Endodontics,
Indira Gandhi Institute of Dental Sciences,
Nellikuzhy P. O.,
Kothamangalam 686 691, Kerala, India.

[2]Professor,
Dept. of Conservative Dentistry and Endodontics,
Indira Gandhi Institute of Dental Sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

[3]Reader,
Dept. of Conservative Dentistry and Endodontics,
Indira Gandhi Institute of Dental Sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

Address for correspondence:

Dr. Romel J.,
Professor and Head,
Dept. of Conservative Dentistry and Endodontics, Indira Gandhi Institute of Dental Sciences, Nellikuzhy P. O.,
Kothamangalam 686 691, Kerala, India.
E mail: romeljoseph99@gmail.com.

ABSTRACT:

Present tooth-bleaching techniques are based upon hydrogen peroxide as the active ingredient. It is applied directly, or produced in a chemical reaction from sodium perborate or carbamide peroxide. Among the successfully bleached teeth 10 to 40% require repeated bleaching. Cervical root resorption is a possible consequence of internal bleaching and is more frequently observed in teeth treated with the thermo-catalytic procedure. Tooth sensitivity is a common side-effect of external tooth bleaching. A selective use of tooth bleaching based on high ethical standards and professional judgement is preferred to reduce complications. The purpose of this paper was to critically evaluate the biologic aspects of tooth bleaching.

Key words: Toxicity, esthetics, bleaching.

J Odontol Res 2013;(1)1:76-82.

INTRODUCTION

In the last decades, tooth whitening or bleaching has become one of the most popular esthetic dental treatments. Although published studies tend to suggest that bleaching is a relatively safe procedure, investigators continue to report adverse effects on hard tissue, soft tissue, and restorative materials. Concerns regarding the safety of all bleaching treatments and products have long existed, but were heightened since the introduction of at-home bleaching.[1]

A variety of peroxide compounds, including carbamide peroxide, hydrogen peroxide (H_2O_2), sodium perborate and calcium peroxide, have been used as active ingredients for bleaching materials.

HISTORY

Bleaching of discoloured, pulpless teeth was first described in 1864, and a verity of materials such as sodium hypochlorite (NaOCl), sodium perborate and H_2O_2 has been used, alone, in combination, and with and without heat activation[2]. "Walking bleach" technique that was introduced in 1961 involved the placement of a mixture of sodium perborate and water into the pulp chamber that was sealed off between the patient's visits to a clinician.[3] The method was later modified and water was replaced by 30-35% H_2O_2, to improve the whitening effect[4]. The observation that carbamide peroxide caused lightening of the teeth was made in the late1960s by an orthodontist who had prescribed an antiseptic containing 10% carbamide peroxide to be used in a tray for the treatment of gingivitis[5].

SAFETY CONCERNS

Free radicals, that are produced by the peroxides are known to be capable of reacting with proteins, lipids and nucleic acids, causing cellular damage. Because of the potential of H_2O_2 to interact with DNA, concerns with carcinogenicity and co-carcinogenicity of hydrogen peroxide have been raised, although these concerns so far have not been substantiated through research[6,7,8].

Similar to other dental and medical interventions, questions have been raised about the safety of tooth whitening treatments during pregnancy. In the absence of such evidence, clinicians may consider recommending that a tooth whitening be deferred during pregnancy.

The safety of tooth bleaching for children and adolescents is also a consideration. If possible, delaying treatment until after permanent teeth have erupted is recommended, as is use of a custom-fabricated bleaching tray to limit the amount of bleaching gel[9]. Close professional and parental/guardian supervision are needed to maximize benefits and minimize adverse effects and overuse.

A "bleaching light" is sometimes used with in-office bleaching procedures. Reports suggest that pulpal temperature can increase with bleaching light use, depending on the light source and exposure time. Pulpal irritation and tooth sensitivity are higher with use of bleaching lights or heat application, and caution has been advised with their use.

However, studies have shown that H_2O_2 is an irritant and also cytotoxic. It is known that a concentrations of 10% H_2O_2 or higher, the chemical is potentially corrosive to mucous membranes or skin, and can cause a burning sensation and tissue damage[6,10]. The amount of products applied during office bleaching treatment and other formulation variables can change the potential to cause damage.

MECHANISM OF BLEACHING

The colour producing materials in solution or on a surface are typically organic compounds that possess extended conjugated chains of alternating single or double bonds and often include heteroatoms, carbonyl, and phenyl rings in the conjugated system and are often referred to as a chromophore. Bleaching and decolouration of the chromophore can occur by destroying one or more of the double bonds in the conjugated chain, or by oxidation of other chemical moieties in the conjugated chain[11].

H_2O_2 oxidises a wide variety of organic or inorganic compounds. The mechanisms of these reactions are varied and dependent on the substrate, the reaction environment, and catalysis. In general, the mechanism of bleaching by H_2O_2 is not well understood and it can form a number of different active oxygen species depending on reaction conditions, including

temperature, pH, light and presence of transition metals[12].

Under alkaline conditions, H_2O_2 bleaching generally proceeds via the perhydroxyl anion (HO_2^-). Other conditions can give rise to free radical formation, for example, by haemolytic cleavage of either an O-H bond or the O-O bond in H_2O_2 to give $H^0 + {}^0OOH$ and 2^0OH (hydroxyl radical), respectively[12]. Under photochemically initiated reactions using light or lasers, the formation of hydroxyl radicals from H_2O_2 has been shown to increase[13].

As peroxide diffuses into the tooth , it can react with organic coloured materials found within the tooth structures leading to reduction in colour[14]. For tetracycline stained teeth, the colour is derived from photo-oxidation of tetracycline molecules bound within the tooth structures[15]. The mechanism by which peroxide affects the tetracycline stain is considered to be by chemical degradation of the unsaturated quinone type structures found in tetracycline leading to less coloured molecules[16,17].

ADVERSE EFFECTS

Cervical root resorption is an inflammatory-mediated external resorption of the root, which can be seen after trauma and following intracoronal bleaching[18]. A high concentration of H_2O_2 in combination with heating seemed to promote cervical root resorption[18,19]. The underlying mechanism for this effect is unclear, but it has been suggested that the bleaching agent reaches the periodontal tissue through dentinal tubules and initiates an inflammatory reaction[20]. It has also been speculated that the peroxide, by diffusing through the dentinal tubules, dentures the dentin, which then becomes an immunologically different tissue and is attacked as a foreign body[21]. Frequently, the resorption was diagnosed several years after the bleaching[19,21]. Invitro studies using extracted teeth showed that H_2O_2 placed in the pulp chamber penetrated the dentin[22] and that heat increased the penetration[23]. H_2O_2 also increased dentin permeability[24], and that may enhance the effects of H_2O_2 following repeated exposures. Based on the cited literature, the use of a thermo-catalytic bleaching procedure in teeth with cervical defects of the cementum constitutes a risk factor for the development of cervical defects of the cementum constitutes a risk factor for the development constitutes a risk factor for the development of cervical resorption.

Tooth crown fracture has also been observed after intracoronal bleaching, most probably due to extensive removal of the intracoronal dentin. In addition, intracoronal bleaching with H_2O_2, has been found to reduce the micro-hardness of dentin and enamel[25] and weaken the mechanical properties of dentin[26].

LOCAL SIDE EFFECTS

A - Tooth sensitivity

Tooth sensitivity is a common side-effect of external tooth bleaching. Higher incidence of tooth sensitivity (from 67 to 78%) was reported after in-office bleaching with H_2O_2 in combination with heat. Tooth sensitivity normally persists for up to 4 days after the cessation of bleaching treatment, but a longer duration of up to 39 days has been reported.

The mechanisms that would account for the tooth sensitivity after external tooth bleaching have not yet been fully established. Invitro experiments have shown that peroxide penetrated enamel and dentin and entered the pulp chamber[27], and that the penetration of restored teeth was higher than that of intact teeth. The amount of peroxide detected in the pulp chamber was related to the concentration of H_2O_2 in the preparations applied[28], and also varied among different brands of bleaching agents with the same declared concentration of carbamide peroxide[27]. The concentration of peroxide in the pulp chamber was not determined in the above studies, and the clinical significance of the findings is therefore unclear.

Tooth sensitivity was also a common symptom in patients who had not bleached their teeth, and their symptom was correlated with gingival recession[29]. Patients with a previous history of tooth sensitivity may thus have a higher risk for such an adverse effect from external tooth bleaching, and this should be taken into account before treatment begins.

B- Mucosal irritation

A high concentration of H_2O_2 (from 30 to 35%) is caustic to mucous membranes and may cause burns

and bleaching of the gingiva. In clinical trials that used 10% carbamide peroxide in custom-made trays, from 25 to 40% of the patients reported gingival irritation during treatment. It is therefore advisable that the tray be designed to prevent gingival exposure by the use of a firm tray that has contact with solely the teeth. In this respect, bleaching strips may be unfavourable, since the bleaching gel will come into contact with the gingiva.

C – Alteration of enamel surface

Morphological alteration of enamel following tooth bleaching has been addressed in several studies. By infrared spectroscopic analysis, it was found that in vitro treatment of extracted teeth with 35% carbamide peroxide for 30 min/day for 4 days changed the inorganic composition of the enamel, whereas 10% and 16% concentrations did not[30]. A high concentration of carbamide peroxide was detrimental to enamel surface integrity, but the damage was less than that seen after phosphoric acid etch. A clinical implication of these findings may be that the teeth are more susceptible to extrinsic discolouration after bleaching due to increased surface roughness.

D - Effects on restorations

Data from laboratory studies documented, increased mercury release from dental amalgams exposed to carbamide peroxide solutions for periods ranging from 8 hours to 14-28 days. The amount of mercury released varied with type of amalgam and type of bleaching agent and ranged from 4 times to 30 times higher than in saline controls. It has been suggested that the bleaching may increase the solubility of glass ionomer and other cements. Furthermore, the bond strength between enamel and resin-based fillings was reduced in the first 24 hours after bleaching. After 24 hours, there was no difference in the strengths of dental composite resin cement bonds to bleached and non-bleached enamel[31]. Following bleaching, H_2O_2 residuals in the enamel inhibit the polymerization of resin-based materials and thus reduce bond strength[32]. Therefore, tooth-bleaching agents should not be used prior to restorative treatment with resin-based materials.

GENOTOXICITY AND CARCINOGENICITY

The genotoxicity of H_2O_2 and of tooth whiteners containing carbamide peroxide has been evaluated[33]. The consensus arising from these evaluations was the direct contact with H_2O_2 induced genotoxic effects in bacteria and cultured cells. When H_2O_2 was administered to bacteria or cultured cells in the presence of catalase or other metabolizing enzymes, the effect was reduced or abolished. Since the hydroxyl radicals, perhydroxyl ions, and superoxide anions formed from H_2O_2 are capable of attacking DNA, the genotoxic potential of H_2O_2 is dependent on the accessibility of free radicals to target DNA. This may explain why H_2O_2 induces genotoxicity in the presence of metabolizing enzymes neither in vitro nor in vivo. Tooth whiteners containing carbamide peroxide were mutagenic in certain bacterial strains and non-mutagenic in the presence of additional activating enzymes.

The International Agency for Research on Cancer (IARC) concluded that there is limited evidence in experimental animals and inadequate evidence in humans for the carcinogenicity of H_2O_2 and classified the chemical into Group 3: Unclassifiable as to carcinogenicity to humans[34]. The genotoxicity of H_2O_2 in oral health products has been evaluated by The Scientific Committee on Cosmetic Products and Non-Food Products. It appears unlikely that oral health products containing or releasing H_2O_2 up to 3.6% H_2O_2 will enhance cancer risk in individuals except in those who have an increased risk of oral cancer due to tobacco use, alcohol abuse, or genetic predisposition.

TOXICITY OF HYDROGEN PEROXIDE AND CARBAMIDE PEROXIDE DUE TO HUMAN EXPOSURE

The acute effects of H_2O_2 ingestion are dependent of the amount ingested and the concentration of the H_2O_2 solution. The outcomes of accidental ingestion, or intentional ingestion for suicide, of solutions of 10% H_2O_2 and higher were more severe than those seen at lower concentrations accidental ingestion of 35% H_2O_2 has resulted in several fatal or near-fatal poisionings. In children ingestion of H_2O_2 and

carbamide peroxide produce ulceration of the oral mucosa, esophagus, and stomach, accompanied by symptoms such as nausea, vomiting, abdominal distention, and sore throat. It is therefore important to keep syringes with bleaching agents out of the reach of children, to prevent any possible accident).

RISK ASSESSMENT OF TOOTH BLEACHING

Risk assessment is traditionally considered to consist of 4 steps: the hazard identification, the dose-response relationship, the exposure assessment, and the risk characterization. The risk characterization is founded on critical comparison of the data on exposure and the dose-response relationship. The amount of bleaching agent used for bleaching one arch of teeth has been calculated to be 900 mg per application when administered according to the manufacturer's instruction[36], and an average of 500 mg per application based on clinical experiments. At least 25% of the bleaching agent administered in bleaching trays is ingested during 2 hours of bleaching[37]. This safety factor is not reached in preparations that deliver or contain more than 12.6% H_2O_2 when 500 mg of bleaching agent is used for bleaching one arch.

Longer bleaching periods per day, multiple applications, bleaching maxillary and mandibular teeth at the same time, and overfilling the tray increase the exposure and reduce the safety factor. For example, if both maxillary and mandibular teeth are bleached at the same time, the minimum required safety factor will not be reached for preparations that contain or deliver more than 7.9% H_2O_2, which corresponds to 22% carbamide peroxide. According to the exposure data from a previous evaluation (900 mg / application) [36], the concentration of H_2O_2 should not exceed 3.5%, which correspond to 10% carbamide peroxide. Based on the risk assessment, it must be concluded that selection of preparations with a low concentration of carbamide peroxide is recommended for the optimum safety of the patient. In addition, overfilling the tray without removing excess material, biting on the tray, and bleaching both maxillary and mandibular teeth at the same time are not advisable.

CONCLUSION

Tooth bleaching is one of the most conservative and cost-effective dental treatments to improve or enhance a person's smile. However, tooth bleaching is not risk-free and only limited long-term clinical data are available on the side effects of tooth bleaching. Accordingly, tooth bleaching is best performed under professional supervision and following a pre-treatment dental examination and diagnosis.

REFERENCES

1. Truman J. Bleaching of non-vital discoloured anterior teeth. Dent Times 1864;1:69-72.
2. Howell RA. Bleaching discoloured root-filled teeth. Br Dent J 1980;148:159-62.
3. Spasser HF. A simple bleaching technique using sodium perborate. NY State Dent J 1961;27:332-4.
4. Nutting EB, Poe GS. A new combination for bleaching teeth. J So CA Dent Assoc 1963;31:289-91.
5. Haywood VB. Nightguard vital bleaching, a history and product update. Part I. Esthet Dent Update 1991;2:63-6
6. Li Y. Biologic properties of peroxide-containing tooth whiteners. Food and Chemical Toxicology 1996;34:887-904.
7. Munro IC, Williams GA, Heymann HO, Kores R. Tooth whitening products and the risk of oral cancer. Food Chemical Toxicol 2006;44:301-15.
8. Munro IC, Williams GA, Heymann HO, Kores R. Use of hydrogen peroxide-based tooth whitening products and the relationship of oral cancer. J Esthet and Rest Dent 2006;18:119-25.
9. Lee SS, Zhang W, Lee DH, Li Y. Tooth whitening in children and adolescents: a literature review. Pediatric Dentistry 2005;27:362-8.
10. Scientific Committee on Consumer Products

(European Commission). Opinion on hydrogen peroxide in tooth whitening products. SCCP/0844?04, March 15,2005.

11. In:Howe-Grant M,editor. Encyclopedia of chemical technology, 4th ed., vol. 4. New York: John Wiley and Sons; 1992.p. 290-1.
12. In:Howe-Grant M,editor. Encyclopedia of chemical technology, 4th ed., vol. 4. New York: John Wiley and Sons; 1992.p. 13-5.
13. Kashima-Tanaka M, Tsujimoto Y, Kawamoto K, Senda N, Ito K, Yamzaki M. Generation of free radicals and/or active oxygen by light or laser irradiation of hydrogen peroxide or sodium hypochlorite. J Endod 2003;29:141-3.
14. mcCaslin AJ, Haywood VB, Potter BJ, Dickson GI, Russell CM. Assessing dentin colour changes from nightguard vital bleaching. JADA 1999;130:1485-90.
15. Mello HS. The mechanism of tetracycline staining in primary and permanent teeth. Journal of Dentistry for Children 1967;34:478-87.
16. Fienman RA, Madray G, Yarborough D. Chemical, optical, and physiologic mechanisms of bleaching products: a review. Practical Periodontics and Aesthetic Dentistry 1991;3:32-6.
17. Davis AK, McKellar JF, Philps GO, Reid AG. Photochemical oxidation of tetracycline in aqueous solution. Journal of the Chemical Society Perkin Transactions 1979;2:369-75.
18. Friedman S, Rotstein I, Libfelt H, Stabholz A, Heling I. Incidence of external root resorption and esthetic results in 58 bleached pulpless teeth. Endod Dent Traumatol 1988;4:23-6.
19. Baratieri LN, Ritter AV, Monterio S, de Andrada MAC, Vieira LCC. Nonvital tooth bleaching: guidelines for the clinician. Quintessence Int 1995;26:597-608.
20. Cvek M, Lindvall A-M. External root resorption following bleaching of pulpless teeth with oxygen peroxide. Endod Dent Traumatol 1985;1:56-60.
21. Lado EA, Stanley HR, Weisman MI. Cervical resorption in bleached teeth. OOO 1983;55;78-80.
22. Rotstein I. In vitro determination and quantification of 30% hydrogen peroxide penetration through dentin and cementum during bleaching. OOO 1991;72:602-6.
23. Rotstein I, Torek Y, Lewinstein I. Effect of bleaching time and temperature on the radicular penetration of hydrogen peroxide. Endod Dent Traumatol 1991d;7:196-8.
24. Heling I, Parson A, Rotstein I. Effect of bleaching agents on dentin permeability to Streptococcus faecalis. J Endod 1995;21:540-2.
25. Lewinstein I, Hirschfeld Z, Stabholz A, Rotstein I. Effect of hydrogen peroxide and sodium perborate on the microhardness of human enamel and dentin. J Endod 1994;20:61-3.
26. Chng HK, Palamara JEA, Messer HH. Effect of hydrogen peroxide and sodium perborate on mechanical properties of human dentin. J Endod 2002;28:62-7.
27. Thitinanthapan W, Satamanont P, Vogsavan N. In vitro penetration of the pulp chamber by three brands of carbamide peroxide. J Esthet Dent 1999;11;259-64.
28. Gokay O, Yilmaz F, Akin S, Tuncbilek M, Ertan R. Penetration of the pulp chamber by bleaching agents in teeth restored with various restorative materials. J Endod 2000;2692-4.
29. Jorgensen MG, Carroll WB. Incidence of tooth sensitivity after home whitening treatment. J Am Dent Assoc 2002;133:1076-82.
30. Oltu U, Gurgan S. Effects of three concentrations of carbamide peroxide on the

structure of enamel. J Oral Rehabil 2000;27:332-40.

31. Homewood C, Tyas M, Woods M. Bonding to previously bleached teeth. Austr Orthodont J 2001;17:27-34.

32. Lai SC, Tay Fr, Cheung GS, mak YF, Carvalho RM, Wei SH, et al. Reversal of compromised bonding in bleached enamel. Dent Res 2002;81:477-81

33. International Agency on Research on Cancer. Monographs on the evaluation of carcinogenic risks to humans. Hydrogen peroxide. 1985;Vol. 36. Lyon: IARC.

34. International Agency on Research on Cancer. Monographs on the evaluation of carcinogenic risks to humans. Re-evaluation of some organic chemicals, hydrazine and hydrogen peroxide.1999; Vol.71. Lyon:IARC.

35. Rackoff WR, Merton DF. Gas embolism after ingestion of hydrogen peroxide. Paediatrics 1990;85:593-4.

36. Dahl JE, Becher R. Acute toxicity of carbamide peroxide and a commercially available tooth bleaching agent in rats. J Dent Res 1995;74:710-4.

37. Matis BA, Yousef M, Cochran MA, Eckert GJ. Degradation of bleaching gels in vivo as afuction of tray design and carbamide peroxide concentration. Oper Dent 2002;27:12-8.

REVIEW ARTICLE

INTRACANAL MEDICAMENTS - REVISITED

Authors:
Prasanth Dhanapal[1],
Sheena B Prasanth [2]

[1]Professor,
Dept. of Conservative Dentistry and Endodontics,
KMCT Dental College,
Mukkam, Kozhikode, Kerala, India.

[2]Dental Surgeon,
Quality Dental Care,
Kadavanthra, Cochin, Kerala, India

Address for correspondence:
Dr. Prasanth Dhanapal T., 'Sahyadri',
NJRA-233, North Janatha Road,
Palarivattom, Cochin 682 025, Kerala, India.
E mail:
prasanthdhanapal@gmail.com

ABSTRACT

The main objective of endodontic treatment of pulpless necrotic teeth is removal of maximum possible bacteria and their byproducts from the root canal system and then finally completely seal the disinfected root canals in three dimensions. In treatment of infected root canals the number of appointments needed to achieve disinfection of the root canal system is controversial. The concept of interappointment dressing in multiple visit endodontic therapy is well documented in endodontic literature. Many materials have been suggested for use as interappointment dressing. Research on endodontic microbiology and biofilms have demystified few earlier concepts and opened up new ideas with regard to interappointment dressing material and the method of application. This literature review is an attempt to rationalise the use of interappointment dressing based on scientific evidence.

Key Words: Intracanal medicaments, Interappointment dressing, Calcium hydroxide, Antibiotic Pastes.

J Odontol Res 2013;(1)1:83-8.

INTRODUCTION

The pathogenesis of pulpal and periapical disease is attributed to bacteria. The microenvironment of the root canal is a complex one which provides excellent condition for microbial growth[1]. The objective of endodontic therapy in a necrotic teeth is to eliminate the maximum number of bacteria from the root canal systems and finally seal the disinfected root canal in all dimensions so that microorganism if any left out in the canal system would not survive. The success rate of endodontic therapy is significantly increased when the infectious bacteria is effectively eradicated prior to root canal filling [2,3,4]

Proper disinfection of the root canal system is achieved by proper asepsis during procedure, chemico mechanical preparation of the root canal, use of irrigating solutions and judicious use of intracanal interappointment medicaments [5]

INTRACANAL MEDICAMENTS- WHY AND WHEN

Medicament is an effective antimicrobial agent placed inside the root canal between treatment appointments in order to destroy remaining microorganisms and prevent the growth of any new arrivals [6]

Many materials have been suggested to be used as an intracanal medicament in multiple visit endodontic therapy. The status of the tooth at the time of start of treatment, the diagnosis and the scientific knowledge about the most likely organism involved with the pathology being treated plays a key role in selection of the medicament for intracanal usage. It is also imperative to have knowledge about the microbiology of endodontic infection such as the mechanism of growth of the microorganism and its virulence including biofilm. Chong et al have suggested bacteriological sampling if a tooth does not respond to treatment, to help in the choice of intracanal medicament.

Biomechanical preparation of the root canal along with copious irrigation during the process remarkably reduces the number of bacteria from the root canal system. This reduction is however temporary since the remaining microorganisms proliferate quickly between appointments [7,8] the maintenance and enhancement of root canal cleansing is achieved by the use of intracanal medicament between appointments. Predictable disinfection of the root canal system is only achieved by placement of interappointment intracanal medicaments [9,10] . In contemporary endodontic practice cleaning and shaping of the root canal is accorded greater importance and the role of usage if intracanal medicaments are being neglected for the worst. Mechanical instrumentation of the root canal leaves significant portions of the root canal walls untouched [11]. So there is less likely chance of complete elimination of bacteria from the root canal system only by instrumentation.[12]

ROLE OF INTRACANAL MEDICAMENTS

Bacteria particularly anaerobes is implicated in periapical lesions.[13] For an intracanal medicament to be effective in teeth with periapical infection the used substance should be germicidal in action. In an infected root canal the intracanal medicament is used for

1) Eliminate any residual bacteria remaining in the root canal system after biomechanical preparation of the root canal.
2) To make the root canal contents inert and neutralize the bacterial toxins and tissue remnants.
3) Reduce the periapical inflammation.
4) Aids in drying weeping canals.
5) Temporarily fills the canal and prevents leakage and percolation into the canal from the coronal aspect.

SINGLE VISIT ENDODONTICS

Performing endodontic therapy in a single visit is not now contraindicated and numerous studies have shown that the clinical outcomes between single- and multiple- visit endodontics are similar. Selection of cases for such protocol is the key. The general consensus is that in case of vital pulp the treatment should be ideally completed in one visit provided factors such as time, operator skill and anatomical considerations are favourable. Single visit endodontics of necrotic pulp with periradicular

lesion is one of the main controversial issues in endodontics[14]. One of the main advantages of single visit endodontics is the elimination of risk of recontamination of the cleansed root canal in between appointments. In such a protocol intracanal interappointment dressings are not used.

INTRACANAL DRESSINGS

The concept of open dressing in infected endodontic teeth is a common practice among a section of clinicians. This does not carry any scientific basis. The only indication when the access of a teeth being left open is when there is continued drainage from the canal after instrumentation and copious irrigation. Leaving a canal open allows for ingress of bacteria from the oral cavity along with salivary contamination. This provides a niche medium for the bacterial growth within the canal. The colonised bacteria undergo complex interactions and forms colonies which are totally different from the microbiota which inhabits the root canal at the start of endodontic therapy. This change in characteristics makes the microbiota less vulnerable to elimination by conventional means leading to higher chance of resistant or persistent root canal and periapical infection. Providing an open dressing is not within the acceptable limits of contemporary endodontic practice with very few exceptions.

When multiple visits endodontic therapy is planned or needed based on the clinical scenario intracanal dressings is recommended. Biocompatibility and stability are essential properties for intracanal medicaments. The more modern meaning of intracanal dressing is for a blockade against coronal leakage from the gap between filling materials and cavity wall.

Commonly used medicaments include calcium hydroxide, antibiotic preparations / combinations, Phenolic compounds, Non phenolic biocides and iodine compounds. Each has advantages and disadvantages, no single preparation has been found to be completely predictable or effective in all cases.

CALCIUM HYDROXIDE

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.

The mode of action of calcium hydroxide can be categorised into two prominent effects. Antibacterial effect by virtue of its high pH (approximately 12.5 to 12.8). The released hydroxyl ion produces a lethal effect on bacterial cells by damage to the bacterial cytoplasmic membrane; protein denaturation; and damage to the DNA. It also affects bacterial endotoxins. Its effect on microbial biofilm is controversial. Another notable and favourable effect of calcium hydroxide as an intracanal medicament is its biological activity. Calcium hydroxide activates tissue enzymes and thereby mineralization.

The dissociation coefficient of $Ca(OH)_2$ is 0.17 which allows a slow, controlled release of both calcium and hydroxyl ions. This low solubility proves to be a good clinical characteristic as it remains insoluble in tissue fluids for a longer period of time when in direct contact with vital tissues.

There is limitation in its effectiveness to disinfect the entire root canal system owing to its inherent physiochemical properties. It is also reported that calcium hydroxide alone is not effective against all the species of bacteria present within the root canal system. The efficacy of calcium hydroxide paste as an intracanal medicament is mainly depended on the vehicle used. Research have suggested association of calcium hydroxide with other medicaments to enhance its activity to obtain pronounced effect against certain pathogenic bacteria often observed in persistent infection. One of the recommended combinations is to mix calcium hydroxide with chlorhexidine gluconate so as to have an enhanced antibacterial effect against Enterococcus faecalis.[15]

ANTIBIOTIC PASTE

Antibiotics play a key adjunct role in bacterial infection control. Systemic administration of antibiotic has the potential risk of adverse effects. Moreover systemic antibiotics are often ineffective in necrotic pulpless teeth and in infected periradicular region[16]. Local application of antibiotics has been used in endodontic practice for long and is believed to a

more effective mode for delivery to the area of bacterial colonization within the root canal and periradicular tissues.

Local application of a single antibiotic as well as paste made of combination of antibiotics has been tried in endodontics as intracanal medicaments.

Tetracycline group of antibiotic are bacteriostatic and has been used as intracanal medicament either alone or in combination with corticosteroid to produce an antibacterial, anti inflammatory and antiresorptive effect. It has been suggested for use in teeth associated with traumatic injury to minimise inflammatory root resorption.

Metronidazole a synthetic antimicrobial agent is bactericidal in action. It has exceptional antianaerobic activity and has been tried as an intracanal medicament in paste form. The efficacy of this medicament alone on eradication of persistent bacteria within the root canal is questionable.

A combination of Metronidazole, Ciprofloxacin and Minocycline has been tried as triple antibiotic paste in cases with persistent weeping canal and non healing periradicular lesions[17]. Triple antibiotic mixture (TAM) was found to be effective against *E. faecalis* a common organism isolated from persistent root canal and periapical infection[18]. Triple antibiotic paste is also recommended and used to disinfect the canal in an immature tooth in revascularisation procedure. One of the draw backs of this triple antibiotic paste is the occurrence of tooth discolouration owing to the presence of minocycline in the mixture[19].

PROPOLIS

Propolis (Bee glue) is a by-product of honeybee and has been in use in traditional medicine for years. It is a natural antimicrobial agent which is found to have a pronounced antibacterial, antifungal and antiviral properties. Few studies have evaluated the effect of propolis on root canal flora particularly E.faecalis and has been recommended as an intracanal medicament[20]. It has been concluded by Maryam et al.[21] that propolis was more effective than calcium hydroxide after 10 day incubation period within the root canal. Stepanovic et al have observed a synergistic action when propolis is used with antibiotic. More research is needed on this natural antimicrobial for suggesting it as a routine intracanal medicament.

FORMOCRESOL

Until recently, formocresol and its relatives were frequently used as intracanal medicaments, but it was pointed out that such bactericidal chemicals dressed in the canal distributed to the whole body from the root apex and so might induce various harmful effects including allergies. It was also observed that formocresol has potent genotoxic effect[22] and mutagenic potential[23] Formaldehyde the primary component in formocresol, is considered a probable human carcinogen by many researchers and statutory bodies. Present day contemporary endodontic therapy does not indicate this chemical in endodontic practice.

CHLORHEXIDINE GLUCONATE

Chlorhexidine is a positively charged hydrophobic and lipophilic molecule which interacts with phospholipids and lipopolysaccharides on the cell membrane of bacteria and then enters the cell[5] leading to cell death.

At a higher concentrations of 2%, which is mainly used for endodontic purpose chlorhexidine is bactericidal. At this concentration there will be precipitation of cytoplasmic content of the bacteria cell resulting in cell death. A paste of calcium hydroxide mixed with chlorhexidine[24] has been found to very effective against E.faecalis and is recommended as an intracanal medicament in retreatment cases in place of calcium hydroxide alone as a medicament.

Chlorhexidine is also an effective antifungal agent and its comparison with sodium hypochlorite in antifungal activity is not studied much.

ENDODONTIC MICROBIOTA AND ENDOTOXINS

The microbial flora of the root canal exists as a biofilm of co aggregated communities in an extracellular matrix. Microbiota of an infected root canal before start of treatment usually consist of a mix of approximately equal proportions of Gram-

positive and Gram-negative species, dominated by obligate anaerobes. On the other hand the microbial flora of root-filled tooth with persistent apical periodontitis is very different with the presence of one or very few species, predominantly Gram-positive micro-organisms with an equal distribution of facultative and obligate anaerobes[25]. In most of the studies Enterococcus faecalis is found in persistent infection of the root canal. Gram negative bacteria play an important role in primary endodontic infections. Endotoxins, a virulence factor of these bacteria leads to initiation and perpetuation of apical periodontitis. The use and selection of the intracanal medicament should keep in mind this complexity of the root canal microflora and the variations that occur with persistent infections. This implies that one medicament may not produce the desirable result in all infected cases.

CONCLUSION

The successful outcome of endodontic therapy mainly depends on elimination of the cause viz bacteria from the root canal system. Total disinfection of the canal is difficult to achieve by mechanical means alone considering the complexity of the root canal system and the microbial interactions that occur in the infected root canal. Usage of intracanal medicament that has a deleterious effect on the bacteria, their endotoxins and biofilm is an important adjunct in contemporary endodontic practice. Intracanal medicaments such as calcium hydroxide are popular but have its own limitations in certain infections. Other medicaments such as chlorhexidine, propolis have been documented to have better antibacterial efficiency. Use of antibiotic paste which has earlier been overlooked may be required in many persistent infection cases to eradicate the microbiota prior to obturation of the root canal.

REFERENCES

1. Nair PNR, Henry S, Cano V, Vera J. Microbial status of apical root canal system of human mandibular first molars with primary apical periodontitis after one-visit-endodontic treatment. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2005; 99: 231-52
2. Sjögren U, Figdor D, Persson S, Sundqvist G. Influence of infection at the time of root filling on the outcome of endodontic treatment of teeth with apical periodontitis. Int Endod J 1997;30:297-306.
3. Sjögren U, Hägglund B, Sundqvist G, Wing K. Factors affecting the long-term results of endodontic treatment. J Endod 1990;16:498-504.
4. Trope M, Delano EO, Orstavik D. Endodontic treatment of teeth with apical periodontitis: single vs. multivisit treatment. J `Endod 1999;25:345-350.
5. Athanassiadis B, Abbott PV, Walsh LJ. The use of calcium hydroxide , antibiotics and biocides as antimicrobial medicaments in endodontics : Aust Dent J: 2007: Mar :52(1 suppl): s 64-82
6. Weine F. Endodontic Therapy. 6th Edition. St. Louis: CV Mosby; 2004. pp. 226–228
7. Chong BS, Pitt Ford TR. The role of intracanal medication in root canal treatment. Int Endod J. 1992;25:97-106.
8. Shih M, Marshall FJ, Rosen S. The bactericidal efficiency of sodium hypochlorite as an endodontic irrigant. Oral Surg Oral Med Oral Patholol. 1970;29:613-9.
9. Byström A, Sundqvist G. The antibacterial action of sodium hypochlorite and EDTA in 60 cases of endodontic therapy. Int Endod J 1985;18:35-40.
10. Siqueira JF Jr, Magalhães KM, Rôças IN. Bacterial reduction in infected root canals

treated with 2.5% NaOCl as an irrigant and calcium hydroxide/camphorated paramonochlorophenol paste as an intracanal dressing. J Endod 2007;33:667-672.

11. Peters OA, Laib A, Gohring TN, Barbakow F. Changes in root canal geometry after preparation assessed by high-resolution computed tomography. J Endod. 2001;27(1):1-6.

12. Bystrom A, Sundqvist G. Bacteriologic evaluation of the efficacy of mechanical root canal instrumentation in endodontic therapy. Scand J Dent Res. 1981;89(4):321-8.

13. Sundqvist G. Taxonomy, ecology, and pathogenicity of the root canal flora. Oral Surg Oral Med Oral Pathol. 1994;78:522-30.

14. Bergenholtz G, Spangberg L. Controversies in endodontics: Crit Rev Oral Biol Med 2004;15:99-114.

15. Gomes BP, Souza SF, Ferraz CC, Teixeira FB, Zaia AA, Valdrighi L, Souza-Filho FJ. Effectiveness of 2% chlorhexidine gel and calcium hydroxide against Enterococcus faecalis in bovine root dentine in vitro. Int Endod J:2003:Apr:36(4): 267-75.

16. Mohammadi Z. Antibiotics as intracanal medicaments: a review. J Calif Dent Assoc;2009:Feb;37(2): 98-108

17. Taneja S , Kumari M. Use of triple antibiotic paste in the treatment of large periradicular lesions. J Investig Clin Dent 2012:Feb:3(1):72-6.

18. Madhubala MM, Srinivasan N, Ahamed S. Comparative evaluation of propolis and triantibiotic mixture as an intracanal medicament against Enterococcus faecalis: J Endod. 2011;Sep 37(9):1287-9.

19. Kim JH, Kim Y, Shin SJ, Park JW, Jung IY. Tooth discoloration of immature permanent incisor associated with triple antibiotic therapy: a case report. J Endod 2010;Jun 36(6):1086-91.

20. Oncag O, Cogulu D, Uzel A, Sorkun K. Efficacy of propolis as an intracanal medicament against Enterococcus faecalis. Gen dent 2006;Sep-Oct:54(5): 319-22.

21. Maryam Zare Jahromi, Hasan Toubavani, Majid Rezaei. Propolis : A new alternative for root canal disinfection; Iran Endod J. 2012 Summer; 7(3):127-133.

22. Lucas Leite AC, Rosenblatt A, Da Silva Calixto M, Da Silva CM, Santos N. Genotoxic effect of formocresol pulp therapy of deciduous teeth: Mutat Res: 2012:Aug 30:747(1):93-7.

23. Zarzar PA, Rosenblatt A, Takahashi CS, Takeuchi PL, Costa Junior LA. Formocresol mutagencity following primary tooth pulp therapy: an in vivo study: J Dent 2003;Sep 31(7):479-85

24. Basson NJ, Tait CM. Effectiveness of three root canal medicaments to eliminate Actinomyces israelii from infected dentinal tubules in vitro ;SADJ. 2001; Nov 56(11):499-501.

25. Figdor D, Sundqvist G. A big role for the very small – understanding the endodontic microbial flora: Aust Dent J: 2007; Mar 52(1 Suppl):S38-51.

CASE REPORT

PROSTHETIC REHABILITATION OF AN ORBITAL DEFECT: A CASE REPORT

Authors:
Sanju John Jolly[1],
Pius AV[2],
Seema George[1],
Cinil Mathew[3]

[1]Reader,
Dept. of Prosthodontics,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

[2]Professor,
Dept. of Prosthodontics,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

[3]Senior lecturer,
Dept. of Prosthodontics,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

Address for correspondence:
Dr. Sanju John Jolly, Reader, Dept. of Prosthodontics, Indira Gandhi Institute of Dental Sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala, India. E-mail: drsanjujj@rediffmail.com.

ABSTRACT

Any damage or disfigurement to the face can lead to psychological and social problem. Trauma to the eye is a severe handicap to a patient because the most important sensory organ of communication is lost. Restoring these congenital as well as acquired defects is a very demanding task. When appropriately made it is the finest service that can be rendered to a patient who feels socially secluded because of facial deficiency. A sequence of steps for construction of custom-made ocular prosthesis is outlined.

Keywords: Maxillofacial prosthesis, Orbital defect, Custom-made ocular prosthesis, Acrylic resin, Iris

J Odontol Res 2013;(1)1:89-92.

INTRODUCTION

The removal of an eye and management of the anophthalmic socket requires the combined efforts of the ophthalmologist and the maxillofacial prosthodontist. The goal of any ocular prosthetic procedure is to return the patient to society with a normal appearance and reasonable mobility of the prosthetic eye. The disfigurement resulting from loss of an eye can cause significant psychological as well as social, consequences. However, with the advancement in ophthalmic surgery and ocular prosthetics the anophthalmic patient can be rehabilitated very effectively. Although implant supported eye prosthesis has a superior outcome, economic reasons may make it in unacceptable to many patients. The goal is to give an eye prosthesis having a comfortable fit that will in turn motivate the patient to use the prosthesis.

CASE REPORT

A patient reported to the department of Prosthodontics with a history of missing right eye due to a road traffic accident (fig-1, a). On examination,[1] the tissue was free of inflammation. The patient did not complain of pain or discomfort in the defect area. A custom-made acrylic resin ocular prosthesis was planned. [2,3]

An impression of the ocular defect was made with custom acrylic resin tray (fig-1, b). The patient was seated in an erect position with the head tilted backward at approximately 45 degrees. The socket was initially filled with alginate (Tropicalgin, Badia Polesine, Italy) with the help of a 5ml disposable syringe (Dispo van, HSMD, Faridabad, India). The remainder of the impression material was placed on the tray to support the alginate in the socket. The patient was instructed to move back the head to the vertical position and both eyes up and down. This facilitated the flow of the impression material into all aspects of the socket. After the material had set, the cheek, nose and eyebrow regions were massaged to break the seal.[4-6] The retrieved impression was checked for accuracy and voids. The excess impression material was trimmed with scissors.

The impression was poured in 2 sections. Initially it was boxed and poured until the height of contour with type IV dental stone (fig-1, c) (Kalastone, kalabhai Pvt Ltd, Mumbai, India). Four keyways are

Fig. 1

Fig. 2

Fig. 3

made for proper orientation of the mould. A layer of separating medium was applied and allowed to dry. Then a second layer was poured, to obtain a two-piece mould. (fig-1, d).

The wax pattern was fabricated using modelling wax (fig-2, a) (Rolex, Ashoo Sons, Ashok Vihar, Delhi). The pattern was tried in the socket repeatedly and sculpted to the correct shape until the eyelids closed completely over the wax pattern. The wax pattern was inserted by lifting the upper lid and the superior edge of the pattern was placed behind the lid and gently pushed upward. The lower lid was pulled down to place the inferior border of the pattern into the inferior fornix. The wax will not move as freely as the finished acrylic resin prosthesis, but the application of an ophthalmic lubricant will aid the movement.

Once the appropriate contours of the wax pattern were developed, the gaze and position of the iris was determined. The size and location of the iris were matched to that of the companion live eye. The correct size of the iris was determined by measuring the diameter of the patient's natural iris (fig-2, b, c). Custom iris, which matched closely with the patient live eye, was selected. The wax was removed from the pattern and the custom iris was placed and again checked in the socket (fig-2, d).

The corrected wax sclera was now invested. White scleral acrylic resin (DPI-heat cure, dental product of India Ltd, Mumbai) was packed into the mold, using the compression moulding method. The processed sclera was again checked and the iris is fixed to the sclera by resin. The iris was modified by color to resemble the live eye. The natural sclera has veins present that are usually apparent in both the nasal and temporal corners of the eye. Red cotton fibers were used to replicate the veins in the sclera. The fibers were separated and cut from the commercially available thread. The vein patterns of the natural eye are copied by tacking the fibers to the sclera with a brush and monomer. The scleral painting begins with the application of a wash of yellow comparable to that found on the patient's natural eye. Next, blue is added which is usually located inferior and superior to the iris. The iris and scleral painting were compared with the natural eye and final corrections were made. Once complete, a thin layer of wax was applied to the sclera. The corresponding area in the flask was scrapped to make place for corneal prominence. The eye was processed finally with the application of a layer of clear acrylic resin (fig-3, a). After processing, the flask is cooled and the ocular prosthesis retrieved from the mold. Flash and irregularities are removed from the surface of the eye. The surface is smoothed with a fine white stone and polished with pumice.[7,8]

The custom ocular prosthesis was then inserted (fig-3, b). The methods of inserting and removing as well as caring for the prosthesis are demonstrated to the patient. The prosthesis should not be allowed to come into contact with alcohol or solvents of any kind, as this could cause crazing of the acrylic resin. Follow up was done after 1 day, 3 days, and 1 week.

DISCUSSION

The rehabilitation of orbital defects is a challenging aspect of Maxillofacial Prosthodontics. It requires that the practitioner constant practice to gain confidence and expertise.[9] The goals of the surgeon and the prosthetic specialist regarding the prosthetic rehabilitation of the patient with an ocular or orbital defect are closely allied.

Literature has suggested many techniques for the fabrication of ocular prosthesis.[10-12] In 1575, Pare fabricated artificial eyes made of glass as well as porcelain. The glass eye was considered the state of the art until the advent of World War II, which made it impossible to obtain glass or glass eyes from Germany. Acrylic resins had replaced vulcanite as a denture base material by the early 1940's and the Naval Dental School tested the use of acrylic resin in fabricating a custom-fitted ocular prosthesis. Unlike a glass eye, an acrylic resin eye was easy to fit and adjust, unbreakable, inert to ocular fluids, aesthetically better, longer lasting, and easier to fabricate.[13] Though undoubtedly implants are the best choice for retention of an orbital prosthesis, the high financial costs involved are the limitation for implant.

A simple procedure for fabricating the ocular prosthesis has been discussed here. Attention to details is mandatory in each step to bring out a satisfactory result.

REFERENCES

1. Jebreil K. Acceptability of orbital prosthesis. J Prosthet Dent 1980;43:82-5.
2. Brown KE. Fabrication of an ocular prosthesis. J Prosthet Dent 1970;24:223-5.
3. Kale E, Mese A, Izgi AD. Atechnique for fabrication of an interim ocular prosthesis. J prosthodont 2008;17:654-61
4. Alice Katz BS, Gold HO. Open eye impression technique for orbital prosthesis. J Prosthet Dent. 1976;36:88-94.
5. Mathew FM, Smith MR, Suttion JA. The ocular impression: a review of literature and presentation of alternate technique. J prosthodont 2000;9:210-6.
6. Supriya M, Ghadiali B. Prosthetic rehabilitation of a patient with an orbital defect using a simplified approach. J Indian prosthodont Soc 2008;8:116-8.
7. Taylor DT. Clinical maxillafacial prosthetics. Quintessence publishing co, Inc;2000.p.233-44.
8. Beumer J, Curtis TA. Maxillofacial rehabilitation: prosthodontic and surgical consideration. St Louis, Ishiyaku Euro America, 1996. p.343-70.
9. Chang TL, Garrett N, Roumanas E, Beumer J III. Treatment satisfaction with facial prosthesis. J Prosthet Dent 2005;94:275-80.
10. Pruthi G, Jain V, Sikka S. A novel method for retention of an orbital prosthesis in a case with continuous maxillary and orbital defect. J Indian Prosthodont Soc. 2010; 10(2):132-6.
11. Jain S, Makkar S, Gupta S, Bhargava A. Prosthetic rehabilitation of ocular defect using digital photography: A case report. J Indian Prosthodont Soc. 2010;10(3):190-3.
12. Gupta RK, Padmanabhan TV. Prosthetic rehabilitation of a post evisceration patient with custom made ocular prosthesis: A case report. J Indian Prosthodont Soc. 2012;12(2):108-12.
13. Murphey PJ, Pitton RD, Schlossberg L, Harris LW. The development of acrylic eye prosthesis at the national naval medical center.J Am Dent Assoc 1945; 32:1227-44.

CASE REPORT

ENDODONTIC STABILIZATION USING IMPLANTS WITH AN IMPROVED DESIGN - A REPORT OF 2 CASES

Authors:
Meera Gopalakrishnan[1],
Prasanth PS [2],
Raison Sujai[3],
Mohan B[4]

[1]Reader,
Dept. of Conservative Dentistry and Endodontics,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

[2]Reader,
Dept. of Orthodontics,
Indira Gandhi Institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.

[3]Private Practitioner.
[4]Professor and Head,
Department of Conservative Dentistry and Endodontics,
SRM Kattankulathur Dental College,
SRM University , Kattankulathur,
Kancheepuram Dt.

Address for correspondence:
Dr. Meera Gopalakrishnan,
Reader,
Dept. of Conservative Dentistry and Endodontics,
Indira Gandhi institute of Dental sciences,
Nellikuzhy P. O., Kothamangalam 686 691,
Kerala, India.
E mail: meeragkrish@gmail.com.

ABSTRACT

Endodontic endosseous implants are metallic extensions of the tooth root which extends beyond the root apex and is inserted into a prepared channel in the bone. They have been used in the past for stabilization of teeth with compromised crown root ratio to improve their prognosis. The inability of these implants to obtain an adequate coronal seal and lack of osseointegration have been cited as the cause of their varying amount of success. The improvement of the implant design and the material used for its fabrication is believed to overcome these drawbacks. This article reports two cases in which endodontic implants with a new design and a predictable technique are used to stabilize teeth with compromised crown root ratio.

Key words: Endodontic endosseous implants, osseointegration, endodontic stabilization.

J Odontol Res 2013;1(1):93-8.

INTRODUCTION

Preservation and maintenance of natural tooth is a prime consideration in modern dentistry. Extraction and subsequent replacement should only be considered after all other means of retaining the natural tooth has been fully explored [1]. A tooth with an unfavorable crown root ratio is a situation with a guarded prognosis in which a successful endodontic therapy alone is insufficient in maintaining the tooth in the arch in its physiologic and functional integrity. Teeth with an unfavorable crown root ratio due to root resorption or horizontal root fracture are a few of the major indications for the placement of an endodontic endosseous implant [2].

Endodontic implants are metallic extensions of the tooth root which extends beyond the root apex and is inserted into the previously prepared channel in the bone [3]. The objective of placement of the endodontic implant is to increase the support for the tooth by passing it beyond the confines of the root canal into the periapical bone, thus improving the crown root length ratio and stabilizing the tooth. Endodontic endosseous implants were first reported by Strock and Strock in 1943[4]. During the last 50 years many clinicians have used stabilizers as an adjunct to dental treatment. The endodontic endosseous implant avoided the greatest site of routine implant failure – epithelial down growth along the supracrestal portion of the fixture. This problem was avoided by placing the endodontic endosseous implant directly through the canal of the tooth into the periapical tissues as an extension of the already present root length [5].

This article presents two case reports in which endodontic stabilization has been done in two teeth with impaired crown root ratio using an endodontic endosseous implant with an improved design.

Case Report 1

A 24 year old male patient was referred to the Department of Conservative Dentistry and Endodontics, from the Department of Orthodontics after the completion of a 3 year long orthodontic treatment.

The medical condition of the patient was essentially negative. The right maxillary central incisor 11 was found missing. The intra oral peri apical radiographs made at the initial visit revealed apical root resorption in the left maxillary central incisor 21. The root structure was resorbed to a level that only 7-8mm of the root was found within the bone (Fig 1). 21 tested non vital on electrical and thermal pulp testing (heated gutta percha) and the tooth exhibited Grade I mobility. The gingival status and the periodontal probing depth was found to be within normal limits for 21.

After a detailed discussion with the orthodontist and the patient the following treatment plan was agreed upon. Initiation of the endodontic treatment, stabilization of the tooth using an improved design endodontic implant and restoration of the missing adjacent tooth with a fixed partial denture using the stabilized tooth as an abutment.

The procedure was undertaken in two appointments. The armamentarium (Figure 2) included stainless steel K-files till ISO size 120(Mani Inc, Japan) and the No. 4 self tapping endodontic stabilizer implant with its corresponding bone drill and a millimeter measuring rod (Weiss et. al , Oratronics Inc, New York). In the first appointment, the root canal treatment was initiated, working length determined to be 16 mm and cleaning and shaping done till 2mm beyond the apex(18mm) till the file of ISO size 110 (Mani) using a conventional hand filing technique for cleaning and shaping of the root canals. Normal saline solution was used for copious irrigation during the procedure. An interappointment intracanal dressing was given with apexcal (Ivoclar vivadent, Liechtenstein) and the access was sealed with Glass Ionomer cement (Fuji Corporation, Japan). After three weeks in the second visit, proper cleaning of the canal was done with copious irrigation with saline solution, the bone drill corresponding to the No.4 endodontic stabilizer was mounted on an endodontic handpiece (X Smart, Dentsply, Germany) and used at slow speed (800 rpm) to prepare the apical third of the root canal and a 10mm receptor site in the bone. A millimeter measuring rod

was placed through the canal into the prepared channel and a radiograph exposed to ascertain the length to which the receptor site extends beyond the root apex by counting the number of millimeter serrations on the rod surface as visible in the radiograph.

After the exact working length was determined, the rubber stopper was placed at the marked length on the No.4 endodontic stabilizer and it was tapped into the canal with mild apical pressure and clockwise rotation till the stopper reached the incisal edge. The implant was then retrieved by anticlockwise rotation. This led to the tapping of the apical third of the dentin and the channel in the bone so that there is close adaptation of the implant to the tissues when it is in its final position.

The canal was thoroughly irrigated and dried with paper points, the intra osseous portion of the endodontic implant was marked and only the portion of the implant that would be inside the canal is coated with Type II glass ionomer cement and the implant was tapped back into place. The excess length of the stabilizer extending beyond the incisal edge was sheared off using a diamond disk while maintaining finger pressure to minimize vibration and the access sealed with glass ionomer cement. Figure 3 shows an immediate post operative radiograph.

The patient experienced no post operative pain or swelling and healing was uneventful. He was scheduled for post operative evaluation at 1 month interval for 6 months. The follow up radiographs showed no signs of deterioration. A one year radiographic follow up showed good periapical healing (Fig 4). The restoration of the adjacent tooth was done using a three unit fixed partial denture after six months using the stabilized tooth as an abutment.

Case Report 2

A 24 year old male patient reported to the Department of Conservative Dentistry and Endodontics, complaining of pain and loosening of the upper front tooth following a road traffic accident. Clinical examination revealed that the right maxillary central incisor 11 was extremely mobile in the labio lingual direction with inflamed gingiva and various enamel craze lines. Enamel craze lines were also seen on the left maxillary central incisor 21. 11 was tender on percussion and the probing depth was found to be within normal limits in 21. A periapical radiograph of 11 indicated a horizontal root fracture 1mm below the crest of the alveolar bone (Fig 5). There was no evident periapical changes noted in the radiograph.

A provisional diagnosis of horizontal root fracture in 11 was made. There was no adequate tooth structure for the construction of a post and core. Adequate retention and resistance form could not be achieved without encroaching on the biological width. Hence after reviewing the risks and potential outcomes, a treatment plan consisting of removal of the coronal fragment of the fractured tooth, initiation of endodontic therapy, forced eruption of 11 and reconstruction using a No.5 endodontic stabilizer which is a single unit post core and implant stabilizer was presented to the patient and an informed consent was taken.

The treatment was carried out in multiple sittings In the first appointment, the coronal fragment of the tooth was removed and endodontic therapy was initiated in 11. Working length was established using radiographic technique to be 11mm. Cleaning and shaping of the root canal was done using conventional hand filing technique with stainless steel files (Mani Inc, Japan) till ISO size 90 using normal saline as an irrigant. An interappointment intracanal dressing with calcium hydroxide (Apexcal, Ivoclar Vivadent, Leischenstenin) was given for one week after giving a coronal seal with IRM. In the second appointment the orthodontic extrusion (Fig 6) of the root of 11 was initiated by bonding brackets on adjacent teeth, drawing an arch wire through them , bonding a wire loop into the canal and engaging the loop onto the arch wire by ligation. The extrusion of the tooth was done over a period of three weeks so as to bring the root 2mm supragingival. The tooth was stabilized for four weeks by leaving the orthodontic appliance in place.

This procedure created an unfavourable crown root ratio. In the next appointment the orthodontic appliance was removed, the canal was cleaned thoroughly with copious irrigation with normal saline, cleaning and shaping was done till 2 mm beyond the apex till ISO size 90, the bone drill corresponding to No.5 stabilizer (Oratronics Inc. New York) was used in a slow speed handpiece to create a channel in the bone extending beyond the apex for 16mm. The working length was confirmed by making a periapical radiograph with the corresponding millimeter measuring rod in place and counting the millimeter markings extending beyond the apex. The No.5 Endodontic stabilizer with a core (Oratronics Inc. New York)was used to tap the apical dentin and the channel in the bone till 16mm. The stabilizer was retrieved with counter clockwise rotations. From the horizontal shoulder of the core of the stabilizer the portion apical to 27mm (11+16mm) was cut off. The canal was dried with paper points, the intracanal portion of the stabilizer was coated with type II Glass ionomer cement and the stabilizer was tapped into place.

Recall examinations were conducted at periodic intervals for 8 months with continued normal findings. At the follow up appointment after two weeks a heat cure acrylic temporary crown was cemented. At 8 months the examination indicated that the tooth was well stabilized and asymptomatic, which was verified by a periapical radiograph which showed good periapical healing and a normal periradicular picture (Fig 9).

DISCUSSION

Since the introduction of endodontic implants into dentistry it has been used in clinical practice with varying degrees of success. The added advantage of endodontic implants was that they are completely encased within tooth and bone. Theoretically it eliminates any communication with the oral cavity and its accompanying bacterial flora and reduces the susceptibility to infection.

Despite improvements in materials and metallurgy most implants of early period displayed high percentage of failure. Early endodontic pins were smooth tapered usually made of a chrome cobalt molybdenum alloy. The tapered pins showed inherent problems such as lack of retention and inadequate apical seal [6]. Judy et al and Zmenur reported substantially stronger retention for threaded endodontic implants compared with smooth tapered implant in vitro for single rooted teeth.

In the new design of the stabilizer used (Fig 10) the implant consists of a major diameter and a minor diameter. The drill corresponds to minor diameter so that when the implant is tapped into place the apical dentin and bone get firmly engaged onto the flutes leaving minimal gap and thus obtaining a perfect apical seal.

An additional feature is the groove running on the crest of the serrations which is twofold in function. One is that within the canal it acts as a sluiceway for escape of the endodontic sealing material in an occlusal direction as the implant is tapped apically hence preventing the apical extrusion of any cement. Secondly within the bone it helps in better stress distribution by attaching to the periimplant ligament fibers. The titanium alloy allows better osseointegration as it has been proved to have a good osteogenic effect.

The clinical success of the two presented cases as assessed by the clinical examination and the follow up radiograph suggest that a proper case selection, the improvement of the implant design, the better predictability of the technique employed and the osteogenic potential of the titanium alloy has brought back Endodontic stabilization as a viable treatment option for improving the prognosis of teeth with an impaired crown root ratio. However further clinical evaluation and long term clinical studies are suggested.

Figure 1.
Pre operative radiograph of 21 showing apical root resorption.

Figure 4. 1yr follow-up radiograph.

Figure 7.Immediate post operative radiograph

Figure 2.The armamentarium - 25mm long K-files size 90-140, millimeter measuring rod and the titanium alloy endodontic stabilizer.

Figure 5. Orthodontic extrusion of the root fragment.

Figure 8.Post operative photograph with No.5 Endodontic stabilizer in place.

Figure 9.8-month follow-up radiograph.

Figure 3. Immediate post operative radiograph.

Figure 6. Pre operative radiograph of 11 showing horizontal root fracture.

Figure 10. Serrated design of endodontic endosseous implant.

REFERENCES

1) Girish Parmar, Pramod Kumar AV. Custom-fabricated endodontic implants- Report of two cases. J Endod 2000; Vol 25 (5): 301-3

2) Franklin S Weine, Alfred L Frank. Survival of the endodontic endosseous implant. J Endod 1993; Vol 19(10): 524-8

3) John I. Ingle, Lief K. Bakland. Endodontics 5th ed 2002: B. C. Decker 732-736.

4) Strock AE, Strock MS . Method of reinforcing pulpless anterior teeth – preliminary report. J oral surg 1943 Vol1:252-5,

5) Frank AL. Improvement of the crown-root ratio by endodontic endosseous implants. J Am Dent. Assoc 30: 188-97, 1967

6) Kenneth W. Judy, Charles M. Weiss: Improved technique of endodontic stabilization – Biofunctional considerations: Manual, Oratronics Inc Newyork.

GUIDELINES FOR SUBMISSION OF MANUSCRIPTS

About the Journal

Journal of Odontological Research, an official publication of Indira Gandhi Institute of Dental Sciences, Nellikuzhy P. O., Kothamangalam 686 691, Kerala, is a peer-reviewed journal published bi-annually in print format.

Scope of the journal

The journal will cover studies related to dentistry and applied basic subjects.

Submission of manuscripts

The manuscripts can be submitted under the categories of **Original Research, Review and Case reports. The guidelines and instructions for authors regarding the drafting and submitting the manuscripts are given below.** Kindly submit your valuable contributions as per the guidelines to the e-mail id **jorigids@gmail.com.**

The Editorial Process

A manuscript will be reviewed for possible publication with the understanding that it is being submitted to Journal of Odontological Research alone at that point in time and has not been published anywhere, simultaneously submitted, or already accepted for publication elsewhere. The journal expects that authors would authorize one of them to correspond with the Journal for all matters related to the manuscript. All manuscripts received are duly acknowledged. On submission, editors review all submitted manuscripts initially for suitability for formal review. Manuscripts with insufficient originality, serious scientific or technical flaws, or lack of a significant message are rejected before proceeding for formal peer-review.

Manuscripts that are found suitable for publication in Journal of Odontological Research are sent to two or more expert peer reviewers. The journal follows a double-blind review process, wherein the reviewers and authors are unaware of each other's identity. Every manuscript is also assigned to a member of the editorial team, who based on the comments from the reviewers takes a final decision on the manuscript. The comments and suggestions (acceptance/ rejection/ amendments in manuscript) received from reviewers are conveyed to the corresponding author. If required, the author is requested to provide a point by point response to reviewers' comments and submit a revised version of the manuscript. This process is repeated till reviewers and editors are satisfied with the manuscript.

Manuscripts accepted for publication are copy edited for grammar, punctuation, print style, and format. Page proofs are sent to the corresponding author.

Authorship Criteria

Each contributor should have participated sufficiently in the work to take public responsibility for appropriate portions of the content of the manuscript. The order of naming the contributors should be based on the relative contribution of the contributor towards the study and writing the manuscript. The journal prescribes a maximum number of authors for the manuscripts. **The maximum num-**

ber of authors for original research articles is six and for case reports and reviews is four.

Conflicts of Interest/ Competing Interests

All authors of must disclose any and all conflicts of interest they may have with publication of the manuscript or an institution or product that is mentioned in the manuscript and/or is important to the outcome of the study presented. Authors should also disclose conflict of interest with products that compete with those mentioned in their manuscript.

Submission of Manuscripts

All manuscripts must be submitted on-line to the e-mail jorigids@gmail.com. Authors will have to pay for submission, processing or publication of articles. If you experience any problems, please contact the editorial office by e-mail. The submitted manuscripts that are not as per the "Instructions to Authors" would be returned to the authors for technical correction, before they undergo editorial/ peer-review. Generally, the manuscript should be submitted in the form of two separate files:

[1] **Title Page/First Page File/covering letter**:

This file should provide

1. The type of manuscript (original article, case report, review article, Letter to editor, Images, etc.) title of the manuscript, running title, names of all authors/ contributors (with their highest academic degrees, designation and affiliations) and name(s) of department(s) and/ or institution(s) to which the work should be credited, . All information which can reveal your identity should be here. Use text/rtf/doc files.
2. Source(s) of support in the form of grants, equipment, drugs, or all of these;
3. Acknowledgement, if any..
4. Conflicts of Interest of each author/ contributor. A statement of financial or other relationships that might lead to a conflict of interest, if that information is not included in the manuscript itself or in an authors' form
5. The name, address, e-mail, and telephone number of the corresponding author, who is responsible for communicating with the other authors about revisions and final approval of the proofs, if that information is not included on the manuscript itself.

[2] **Blinded Article file**: The main text of the article, beginning from Abstract till References (including tables) should be in this file. The file must not contain any mention of the authors' names or initials or the institution at which the study was done or acknowledgements. Page headers/running title can include the title but not the authors' names. Manuscripts not in compliance with the Journal's blinding policy will be returned to the corresponding author. Use rtf/doc files. Do not zip the files. **Limit the file size to 1 MB**. Do not incorporate images in the file. The pages should be numbered consecutively, beginning with the first page of the blinded article file.

[3] **Images**: Submit good quality color images. **Each image should be less than 4 MB in size**. Size of the image can be reduced by decreasing the actual height and width of the images (keep up to 1800 x 1200 pixels or 5-6 inches). Images can be submitted as jpeg files. Do not zip the files. Legends for the figures/images should be included at the end of the article file.

[4] **The contributors' / copyright transfer form** (template provided below) has to be submitted in original with the signatures of all the contributors within two weeks of submission via courier, fax or email as a scanned image. High resolution images (up to 5 MB each) can be sent by email.

Preparation of Manuscripts

Manuscripts must be prepared in accordance with "Uniform requirements for Manuscripts submitted to Biomedical Journals" developed by the International Committee of Medical Journal Editors (October 2008). The uniform requirements and specific requirement of Journal of Odontological Research are summarized below. Journal of Odontological Research accepts manuscripts written in English. For further details regarding the guidelines in drafting the manuscript for publication, log on to www.igids.org

www.igmt.org

Indira Gandhi
GROUP OF INSTITUTIONS

Indira Nagar, Nellikuzhi P.O., Kothamangalam,
Ernakulam, Kerala, S. India, Pin 686 691
Ph: 0485 3390001(100 lines), 2824871, 9447749530
Email: chairman@igmt.org

- Indira Gandhi Institute of Dental Sciences
- Indira Gandhi Institute of Engineering and Technology for Women
- KMP College of Engineering
- Indira Gandhi College of Arts and Science
- KMP College of Arts and Science
- Indira Gandhi Training College
- Indira Gandhi Teacher Training Institute
- Indira Gandhi College of Paramedical Science
- S.S.M. L.P.School

Courses Offered

Bachelor of Dental Surgery (B.D.S.)

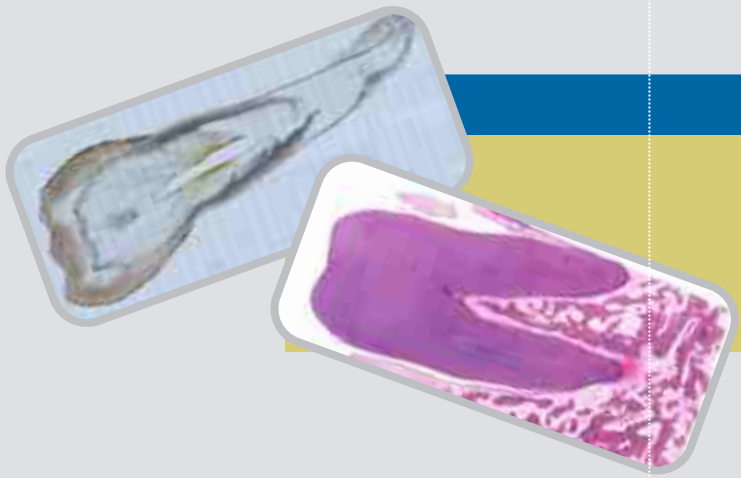
B.Tech. Computer Science & Engg., Electronics & Communication Engg.
Electrical & Electronics Engg., Mechanical Engg., Civil Engg.
M.Tech. VLSI Designs & Embedded Systems (Electronics)
Structural Engineering & Construction Management (Civil)

B.C.A, B.A. Eng., B.A. Economics, B.Com with Computer, B Com Taxation, B.B.A.
B.Sc. Electronics with Computer Hardware, B.Sc. Computer Science,
B.Sc. Biotechnology,B.Sc. Microbiology
M.Sc. Biotechnology, M.Sc. Microbiology,M.Sc. Maths, M.Sc. Physics, M.Sc. Chemistry,
M.Sc. Zoology, M.Com, M.Com Taxation.

B.Ed., TTC, Diploma in Health Inspector Course (DHI)

EDUCATIONAL AIDS

- Oral Histology, Oral Pathology
- Tooth Germ Development, Oral Embryology
- Dental Anatomy - Ground sections of Teeth
- General Histology and General Pathology
- Decalcified section of Teeth
- Rare Lesion of Oral Pathology
- Excellent quality of slides
- Huge Client base in India and Overseas



Contact: Med Aid India

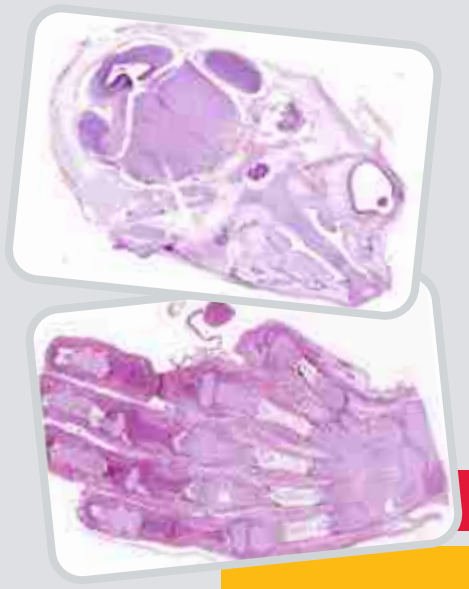
M - 1, Raja House, 30-31,
Nehru Place, New Delhi - 110019
Ph.: 011-26228228, Mobile: 09811502506
www.medaidindia.co.in
Email: medaidindia@yahoo.com

VIRTUAL MICROSCOPY

Digital Whole Slide Images

(Virtual Slides / Digital slides)

- Huge repository of whole slide images
- Oral histology, Oral pathology
- General histology, General pathology
- Ground and Decalcified sections of teeth
- Tooth germ development and oral embryology
- High resolution and clarity of images
- Highly user friendly software
- Excellent tool for theory and practical teaching
- Highly popular for self learning by students
- Teaching modules under preparation
- Demo Whole slide images in the website



Contact: DIGISCAN

611, 6TH Floor, Kasana Tower I,
Alpha I Commercial Belt,
Greater Noida - 201308
Mobile: 09811502506, 08743904252
www.digiscan.in
Email: digiscanvm@gmail.com