Index Copernicus ID 6818
ISSN 2231- 1823

Journal of the Society of Periodontists and Implantologists of Kerala

JSPIK
Volume 12 • Issue 3 • December 2020
www.spik.in

SOCIETY OF PERIODONTISTS AND IMPLANTOLOGISTS OF KERALA

Vol. 12 | Issue 3 | December 2020

Index Copernicus ID 6818

ISSN 2231-1823

Contents

President's message

Dear colleagues,

Warm regards to you.

First let me congratulate Dr Jayan Jacob our secretary for conducting the executive meetings and Dr Sameera G Nath our editor for meticulously bringing out the third issue of SPIK journal.

The central council of Indian medicine (CCIM), Ayush ministry issued a gazette notification permitting the Ayurveda postgraduates to practice 58 types of surgical procedures including RCT and dental extractions. When contacted the ministry underplayed the seriousness of the issue and came out with an explanation that "they already have those procedures covered under the curriculum from 2016 and this is just a clarification".

This order has far reaching ramifications, with this they have legalized the mixing up of modern medicine and ayurveda, obviously other systems will follow. Now any ayurveda physician can perform any of these surgeries even though they are not trained for that. Moreover they can prescribe all modern medicines even though they never studied the pharmacopeia.

Merely by completing the prescribed course nobody becomes a competent surgeon, you need years of dedication and practice to polish your skills to become a good surgeon. With this an ayurveda "surgeon" who doesn't have proper/ required knowledge can perform surgeries on unsuspecting public.

The Government gives the excuse that in rural areas we don't have enough allopathic doctors and they expect the ayurvedic "surgeons" to serve the rural area. But in reality they will also end up in urban areas. Unless the living conditions in our villages improve no one will willingly work there. This obstacle may be overcome by doubling the salary or by offering post graduate seats for serving in rural areas and definitely not by creating half baked doctors, who will become a menace to the society.

The ayurveda spoke persons publically claims that they use 'ayurvedic antibiotic' and yoga therapy for anesthesia and in private they say for time being service from anesthetists will be used and in case of complications patients will be shifted to modern medicine ICU's. Hope we can instill some sense into the mind of these "reformists" to correct this blunder.

Thank you.

Dr Sabu Kurian
President, SPIK

Dr. Sameera G. Nath
Editor, JSPIK
(2019-2021)

Editorial

Greetings to all from the Office of JSPIK. This two year tenure as Editor was very rewarding; thanks to all the authors, co-authors, and my fellow members for all the support you showered upon us during this period. Editorial office owes its gratitude to Dr Harikumar Menon (Past President, SPIK-2019), Dr Sabu Kurien (President, SPIK-2020), Dr Jayan Jacob (Secretary, SPIK 2019-2021), all our office bearers, academicians and members of our Society.

This being the first and the last editorial penned by me during this tenure, I take this opportunity to thank the Editorial board members Drs. K Nandakumar, Presanthila Janam, Rosamma Joseph, Harish Kumar V V, Biju Thomas, Bindhu R Nair, the review panel members Drs. Seema Jayakrishnan, Anuradha Bhaskar, Sajith Abraham, Anoop V, Roshni Ramesh and Assistant Editor Dr Shahana C Mohammed for trusting me and supporting me as always. It was Dr Harikumar Menon's vision to bring out in every issue a Guest Editorial by a senior member; which was readily accepted by all. For a new member, these Guest Editorials served as a good platform for knowing the Stalwarts of Periodontics of our State. We also had an array of articles ranging from original research, case reports, and literature review which helped to keep our readers abreast with the recent trends in our Specialty.

Academics and clinical practice are two sides of the coin for any Clinical Specialty-one sowing the seed for the other. Under the clutches of this COVID 19 pandemic several promising and novel research ideas with clinical significance which was proposed earlier across several educational institutions had to be contained and set aside for future. Interestingly, in vitro studies are now the trendsetters of 2020. This proves that under any given circumstance evidence based research will not fall short; provided it helped convert the research findings for improved practice of Clinical Periodontics. Recently a news report was the talk of the town since its release. "Their teeth fell out. Was it another COVID 19 consequence?"- New York Times, Nov 26th 2020. It can be possible that inflammatory and dysbiotic factors as well as comorbidities affect systemic health, so that periodontal status indicates the risk of complication of COVID-19, seeding a pathway for viral co -infection.

Let today's clinical findings serve as stepping stones for tomorrow's research. Let, research flourish scientifically and systematically.

Yours in SPIK,

Dr. Sameera G. Nath
Editor, JSPIK

Secretary's Message

Dear SPIK members,

"Adversity introduces a man to himself"

-Einstein

As the calendar year is fast approaching to an end, all of us are perhaps experiencing one of the most challenging periods of our lifetime and many have successfully adapted to it. From a professional point of view also, we are striving to keep ourselves relevant.

As informed earlier, we are almost done with updating the membership details of the society. I thank all life members for their cooperation in this venture.

Keeping in tune with the changing times, SPIK is planning to use the digital platform for the extension of its activities. The second executive committee meeting has come up with a suggestion to bring out an awareness video highlighting the significance of maintaining periodontal health that can be widely circulated in various social media platforms, a project envisaged by our President, Dr. Sabu Kurian.

The SPIK periodontology scholarship exam has been one of the flagship events of our society, which is well-received by the undergraduate students. Considering the current scenario, we are pondering the possibility of conducting the same online.

As the current editorial board is preparing to release the final issue of JSPIK of their term, I wholeheartedly thank and congratulate our Editor, Dr Sameera G. Nath for her relentless efforts in the past two years in ensuring the regular publication of our journal. I once again solicit the valuable contributions of our members to enrich its pages.

As we wait to welcome 2021, promising signs of effective COVID -19 vaccines are in near sight. Let us hope that it materializes soon and our routines are restored. Until then, continue to stay safe…

Regards

Dr. Jayan Jacob Mathew
Secretary, SPIK

JSPIK

An Invitro Evaluation of Salvadora Persica against Porphyromonas Gingivalis and Herpes Simplex Virus-1 for the Treatment of Periodontitis.

Santhosh Sekar[1], Siji Jacob[2], Thangakumaran Suthanthiran[3], Sasikumar Karupannan[4], Syed Dhasthaheer[5], Swathigan Vikraman[5]

ABSTRACT

Background: Miswak is a chewing stick and also identified scientifically as Salvadorapersica. Miswak due to its antibacterial and antiviral properties can be used to treat periodontal problems by using it along a local drug delivery agent. **Aim:** To identify the anti-bacterial and anti-viral actions of miswak against Porphyromonas gingivalis and herpes simplex virus-1 respectively. **Methods and Materials:** The minimal inhibitory concentration of Salvadorapersica was calculated against Porphyromonas gingivalis and MTT antiviral assay against Herpes simplex virus-1. **Results:** Salvadorapersica has antibacterial action against Porphyromonas gingivalis (62.5µg/ml) and antiviral action against HSV-1(50µg/ml). **Conclusions:** Miswak exhibits anti-bacterial and anti-viral actions against microbes involved in periodontitis. So miswak can be manipulated to treat periodontal infections.

KEY WORDS: Miswak, porphyromonas gingivalis, herpes simplex virus-1, periodontitits

Introduction

Periodontitis is a localized infection by microbes involving gingival and the other supporting structures of the teeth. It causes periodontal pocket and loss of attachment.[1] Microbes include fungi, protozoa, virus, bacteria etc. But the role of bacteria is most accepted.[2] The various periodontal pathogenic bacterias are Porphyromanas gingivalis, Treponema denticola, Tannerella forsythia, Prevotella intermedia and Actinobacilus actinomycetem comitans.[3] Porphyromonas gingivalis is one among the most pathogenic and extensively studied. It is commonly found in subgingival flora of adult patients. Pathogenicity of P.gingivalis is due to various virulence factors like fimbriae, hemagglutinins, gingipains and lipopolysaccharide which enable it to enter host tissue.[4]

But the bacterial etiology cannot explain disease remission, reactivation,site specificity and periodontal destruction in a few patients and not present in others. It is recently believed that periodontitis is complex interaction between bacteria, virus, host and different environmental factors. Recently Herpes viruses are found in gingival crevicular fluid and periodontal structures in periodontitis patients. Some the viruses of herpes virus are Ebstain bar virus, Human cytomegalovirus and Herpes simplex virus. Herpes simplex virus-1 is the most common of all the above viruses.[5]

Subgingival bacteria are normally removed by mechanical debridement and systemic administration of antibiotics. Mechanical debridement fails to remove the bacteria completely because of their location in deep and inaccessible areas within gingival tissues.[1] Consequently adjunctive use of systemic antibiotics are recommended for compensating the short coming of mechanical debridement. But these systemic antibiotics also have disadvantages, of causing super infection, toxic side effects and these systemic antibiotics attain low concentration in crevicular fluid.[4,5] To overcome

[1]Senior Lecturer, JKKN Dental College and Hospital, Komarapalyam; [2]Professor, Rajah Muthiah Dental College and Hospital, Chidambaram, [3]Professor and HOD, [4]Reader, [5]PG student, JKKN Dental College and Hospital, Komarapalyam. Corresponding Author: Dr. Santhosh Sekar, E-mail: dr.santhoshsmds@gmail.com

these shortcomings of conventional method recent advances advocate the use of herbs.[6]

In history many chewing sticks have been used to maintain oral hygiene. Miswak is a chewing stick (Fig. 1) used in the Arab world. Babylonians used them more than 7000 years ago. Miswak also identified scientifically as Salvadorapersica Linn belongs to the salvadoraceae family. It is a small tree 3m tall and has small leaves. It is also known as toothbrush tree. The branches of this tree are used as chewing sticks traditionally.[7]

Miswak contains many compounds like Benzyl isothiocyanate, Alkaloids, Silica, Calcium fluoride, sodium bicarbonate, tannic acid, resins, essential oils and vitamin C. These compounds areknown to have various properties like antiplaque, anticaries, antibacterial, antiviral, antimycotic, antidiabetic, antihypertensive, antioxidant, anti hyperlipidemic anticonvulsant, sedative and antiulcer activity.[8]

Periodontal pocket is a suitable site for placement of local drug delivery agents like dental flims these agents can deliver antibacterial agents for a longer a period.[6] Miswak due to its antibacterial and antiviral properties can be used to treat periodontal problems by using it along a local drug delivery agent.

The aim of the present study is to identify the Minimum inhibitory concentration of miswak against Porphyromonas gingivalis and MTT- (3-(4, 5-dimethylthiazol-2-yl)-2, 5-diphenyl tetrazolium bromide) antiviral assay against Herpes simplex virus- 1 to be used along with anylocal drug delivery agent for the treatment of periodontal infection.

Materials and methods

The study was conducted in Department of Pharmaceutical Biotechnology, JSS College of Pharmacy, Ooty. The bacteria Porphyromonas gingivalis and Herpes Simplex virus – 1 wereobtained from bacterial and viral stock preserved in the college. Certified powdered form of Salvadorapersica extract was purchased from Vilumin Herbals (Vilumin traders, Omkar herbals), Omkar house, Indore (Fig 2). The minimal inhibitory concentration of Salvadorapersica was calculated against Porphyromonas gingivalis and MTT antiviral assay against Herpes simplex virus-1.

MIC against Porphyromonas gingivalis

Preparation of standard bacterial suspension

The average number of viable, gram negative porphyromonas gingivalis organisms per ml of stock suspension was determined by means of the surface viable counting technique (Miles and Misra, 1938).[9] About (10^5-10^6) colony forming units per ml was used. Each time, a fresh stock suspension was prepared. The experimental conditions were maintained constant so that suspensions with very close viable counts would be obtained.

Determination of minimal inhibitory concentrations (MIC)

MIC was determined with 96 well plate microdilution method. Briefly the bacterial strain was grown for 24h anaerobically and inoculated into volume of 100µl of new half strength BHI (Brain heart infusion) broth containing 2 fold serial dilutions of samples. The final optical density of the bacterial cells was adjusted to 0.1 at 600 nm in 100 µl of mixture. The mixture was cultured anaerobically at 37^0 C for 48 h and the bacterial growth was evaluated via measurement of the optical density at 600 nm.10 The lowest concentration of the sample used for MIC were between 1000- 15.625 µg/ml.

Results

Determination of MIC by micro – dilution method:

The sample exhibited the strongest antibacterial effect on the tested strain and MIC was determined to be 62.5 µg/ ml for the P.gingivalis.

MTT antiviral assay against – HSV-1

Cell line: Vero (African Green Monkey, Kidney)

Virus: HSV-1

Assay: MTT antiviral assay

Concentrations used: 50, 25, 12.5, and 6.25µg/ml

MTT- antiviral assay

MTT- A rapid and sensitive procedure to evaluate antiviral compounds invitro is used on spectrophotometrical assessment for viability of virus-infected and mock infected cells via in situ reduction of tetrazolium dye MTT. Mitochondrial enzymes of viable cells con-

vert yellow water soluble dye MTT to a soluble, purple coloured insoluble formazon. The quantitation of the amount of the formazon product present in each well of microtitre plate is then determined spectrophotometrically at 490/650 nm. While the toxicity of the test compounds to host cells is measured in the same microtitre plate.[11]

Procedure

Cells ($1*10^5$ cells/ml) were seeded on 96-well tissue culture plates. After a 24 h period of incubation, the medium was removed and the HSV-1 was added at the dose of 100 $TCID_{50}$ (Median tissue culture infectious dose) for 2 hrs to ensure the attachment of virus to the cell and after 2 hrs the cells were washed with PBS(Phosphate buffer saline) and replenished with 100 µl of medium containing increasing concentrations of compounds (serially diluted twofold).As cell control,100 µl of medium only is added and as virus control 100 µl of 100TCID50 does was added. After three days of incubation, the medium was removed and 50ml of MTT solution (2mg/ml)was added to each well for 4h at 37^0C. Then, 100µl of DMSO (Dimethyl sulfoxide) was added to each well in order to dissolve the formazan crystals. After shaking gently the plates for 10min to dissolve the crystals, the colour reaction was measured in an automated microplate reader at 490nm.11The untreated control was arbitrarily set as 100%for each compound, the percentage of cell protection/virus inhibition can be calculated as follows:

$$\frac{\text{(Mean OD (Optical density) of control group – Mean OD of treated group)} \times 100}{\text{Mean OD of control group}}$$

Result

Table 1: Antiviral activity of extracts against HSV -1 virus at 100 TCID50 (50% Tissue culture infectivity dose).

Sample description	Concentration µg/ml	100 TCID50
		% virus Growth
Salvadorapersica	50	44.4
	25	59.4
	12.5	88.95
	6.25	93.95

Table 2: Antiviral –HSV-1 activity in vero cells

Treatment	IC50 µg/ml	CC50 µg/ml	TI µg/ml
Salvodorapersica	18.60 ± 3.05	210.31± 7.02	11.03

IC50- Half minimal inhibitory concentration

CC50- Cytotoxicity concentration 50.

TI- Therapuetic index

Discussion

Salvadorapersica or Miswak is commonly called by various names like Siwak in Arabic, Ugaai in Tamil and Peelu in Urdu. Miswak in Arabic means "Tooth cleaning stick". It is used traditionally as a tooth brushing stick to maintain oral hygiene.[8] The effect of this traditional miswak is comparable with modern tooth brushes. It was demonstrated by a study conducted by Al-Otaibiet al in 2004 on Saudi Arabian individuals who showed that miswak chewing sticks reduced significant amount of subgingival plaque microflora when compared with modern toothbrush usage.[12] Miswak can also use as mouthwash to maintain oral hygiene. A study conducted by Shetti NA et al 2016 showed that miswak mouth wash had significant reduction in mean gingival and plaque score when compared with use of distilled water.[13] These studies show that miswak acts as a physical plaque control agent by acting as a tooth brush and also as a chemical plaque control agent by acting as a mouthwash.

Recent studies by Jorgen slots has shown a close association between periodontitis and herpes virus group. He states that there is a synergistic interaction between perio pathogenic bacteria and herpes virus group. So he suggests controlling both pathogenic bacteria as well as herpes virus for effective treatment of periodontitis.[3]

Miswak is an ideal drug to show both antibacterial and antiviral action. Various studies have been conducted to support this claim. Miswak contains a wide range of chemical compounds like trimethylamine, salvadorine,α – tocopherol, β- sitosterol, benzylisothio- cyanate etc.[14]

According to a study published by Dhalkari Chandulalet al 2016 miswak showed antibacterial action against periodontal pathogenic bacteria. He stated

that benzylisothiocyanate is a major component in salvadorapersica and it has bactericidal effect aganist periodontal pathogens.[15]

According to a study published by Adran Sukkarwallaet al 2013, Salvodorapersica contains alkaloids like nitrogenous organic compounds as well as the sulphur components that gives it its pungent taste and smell possess bactericidal property. Essential oil like benzyl nitrate, eugenol and thymol have antibacterial property.[16]

According to a review article published Mohammed M.Hagueet al 2015, the crude extract of Salvodorapersica had pronounced effect on Porphyromonas gingivalis, Aggregatibacter actinomycetocomitans and Haemophilus influenza. Benzyl isothiocyarate was the main antibacterial component.[17]

In a review published by Mohammad M.Haque in 2015 noted that benzyl isothyocyanate in miswak has virucidal activity against Herpes simplex virus type 1. Benzyl isothiocyanate exhibited antiviral property at 133µg/ml.[17]

A study conducted by Mahmoud YM Taha in 2008 on mice infected with HSV-1 showed that topical application of Salvadorapersica reduced the cutaneous lesion on skin and reduced the viral titre in the ganglia.[18]

From the above studies it is shown that Salvadorapersica has both antibacterial and also antiviral action against periodonto pathogenic bacteria and virus. So this property of miswak can be used along with a local drug delivery agent in periodontal pockets to treat periodontitis. For miswak to be used as a drug, invitro tests have to be conducted to confirm its action and to find the dosage at which it exerts its antibacterial and antiviral action. In our study we decided to check the concentration of miswak against one pathogenic bacteria, Porphyromonas gingivalis and one pathogenic virus,Herpes simplex virus -1 for treatment of chronic periodontitis.

We tested the MIC of crude extract of Salvador-

Figure 1 – Miswak stick

Figure 2 – Powdered form of Salvadorapersica extract

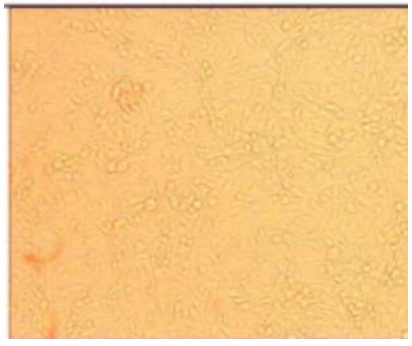


Figure 3 –Plain Vero cells

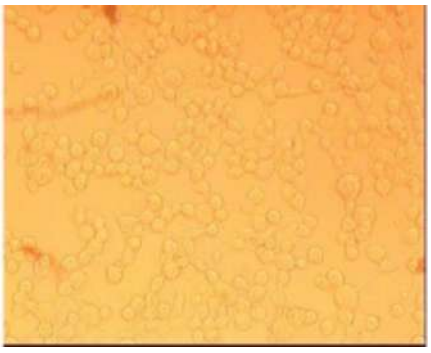


Figure 4 –Vero cells infected with HSV-1

Figure 5 –Vero cells are protected at 50µg/ml.

apersica on the periodontal pathogen Porphyromonasgingivalis by 96 well plate microdilution method. The various concentrations used were 1000-15.625 µg/ml. The result according to showed that at 62.5 µg/ml Salvadorapersica inhibited the growth of Porphyromonasgingivalis.

We also tested the MT Tantiviral assay of crude extract of Salvadorapersica on periodontal pathogen Herpes simplex virus-1 by 96 well plate. The various concentrations used were from 6.25µg/ml to 50µg/ml. The result according to (Table.1) showed that at 50µg/ml property the HSV-1 growth was reduced to 44.4% from 100%.

In our study we conducted anti-HSV-1 activity in Vero cells. Vero cells obtained from monkey's kidney. HSV-1 grows in invitro conditions in these Vero cells. The IC50 or half maximal inhibitory concentration against HSV-1 is 18.60± 3.05. The CC50 or cytotoxic concentration 50 was found to be 210± 7.02 (Table 2). TI or Therapuetic index is calculated to be 11.30 µg/ml.Figure 3 shows plain Vero cells, figure 4 shows Vero cells infected 100% growth in Vero cells and figure 5 shows Vero cells are protected at 50µg/ml.

From the above results it can be seen that Salvadorapersica has antibacterial action against Porphyromonas gingivalis and antiviral action against HSV-1. Since IC50 is 18.60 ±3.05, it is the 1/10th dose that can inhibit 50% HSV-1 growth. Since CC50 is 210.31±7.02, it is the concentration at which 50% cell death is expected. TI is calculated to be 11.3 µg/ml (TI=IC50/CC50). TI tells us the minimum dose of Salvadorapersica that can give anti HSV-1 action. The cells line used here is Vero (African Green monkey's kidney). According to Masahiko Kurokawaet al 2016 these results obtained in Vero cells can be compared to human cells also.[11] With the help of these results we can formulate the dosage of Salvadorapersica to be used along with any local drug delivery agent for placement in periodontal defects.

A review by Verma Rajesh et al 2009 mentions the use of miswak already in India in the form of decoction to treat fever, amenorrhoea, and antidote to all types of poison, scurvy, gonorrhoea, asthma, cough and rheumatism. So miswak can be used safely without much complications[12]. As our invitro study of Salvadora Persica shows both antibacterial action and antiviral action, it can be effectively used for the treatment of periodontitis when used along with a local delivery system.

Conclusion

Salvadorapersica has many properties which helps to maintain good oral hygiene. Our study has demonstrated its antibacterial and antiviral properties. The reason for these properties have to be investigated thoroughly. But these properties can be used for the better treatment of periodontal disease. Salvodorapersica can be used successfully in the form of local drug delivery system either in the form of mouthwash, gel or film to control and treat periodontitis.

References

1. Nitesh Kumar Sharma, Anushree Prasad. Evaluation of efficacy of tetracycline as a local drug delivery system in the treatment of chronic periodontitis as an adjunct to scaling and root planing –A Clinical and microbiological study. International journal of contemporary medical research. May 2017; 4(5):998-1003.
2. Thomas George V, SaumyaJohn,Annu Elizabeth Josep,BindiyaBalram,Sreejith C K, Merry Mariyam Varghese. Detection of herpes simplex virus in chronic generalized periodontitis polymerase chain reaction. A piolet study international journal of contemporary medical research.May 2017; 4(3):729-32.
3. Slots J. Herpesviruses in periodontal diseases. Periodontology 2000. 2005 Jun;38(1):33-62.
4. Maggie M Fawzi, Mounir M Fawzi, Hany M. El – Amin, Mahmoud H. Elafaudy. Detection and quantification of Porphyromonasgingivalis from saliva of Schizophrenia patients by culture and Taqman real – time PCR: A Pilot study. Life science journal, 2011:8(2) 65 – 74.
5. SanjaMaticPetrovic, KsenijaZelic, JelenaMilasin, BrankaPopovic, Ana Pucar. Detection of herpes simplex virus 1 in gingival crevicular fluid of gingival sulcus/periodontal pocket using polymerase chain reaction. SrpArhCelokLek. 2014 May-June;142(5-6):296-300.
6. HymansBhusanSamal, ItishreeJogamayaDas, Ch.NiranjanPatra, S.A. Sreenivas. Design and invitro evaluation of curcumin dental films to the treatment of periodontitis. Asian journal of pharmaceutics. Jul – Sep 2017 (suppl). 11 (3)\s 579-87.
7. MohammedAbhay, Abdul – Aziz Al hazmi. Antibacterial activity of miswak (salvodorapersica) extracts on oral hygiene. Journal of taibah university for science 2016;(10): 513-20.
8. Dr.JaishreeTukaramKshirsagar and Dr. AJ Jareen. The miracle twig – Miswak. International journal of applied dental sciences 2017; 3(2):66-70.
9. Miles AA, Misra SS, Irwin JO. The estimation of the bactericidal power of the blood. Epidemiology & Infection. 1938 Nov;38(6):732-49.
10. Bauer AW. Antibiotic susceptibility testing by a standardized single disc method. Am J clinpathol. 1966;45:149-58.
11. Masahiko Kurokawa, Ashish Wadhwani, Hisahiro Kai, Muneaki Hidaka, Hiroki Yoshida, Chihiro Sugita, Wataru Watanabe, Koji Matsuno and Akinori Hagiwara. Activation of cellular immunity in herpes simplex virus type 1- infected mice by the oral administration of aqueous extract of MorigaoliferaLam.Leaves short title: Activation of cellular immunity by MoringaOlifera extract.

Phytotherapy Research, 2016; 30(5):797-804.

12. Al-Otaibi M, Al-Harthy M, Gustafsson A, Johansson A,ClaessonR,Angmar- Mansson B. Subgingival plaque microbiota in Saudi Arabians after use of miswak chewing stick and both brush. J clic periodontal 2004; 31:1048-53.
13. Shetti NA, Metgud R, Pattar V, Hugar SS. Salvodorapersica (miswak) mouthwash: A promising home core agent. Annals of dental speciality 2016; 4(1);6-9.
14. Wasimuzzamma K, Mujib A, Tausif S, Rahmi T, Katekar S, RukhsanaA. Phytochemical and pharmacological profile of miswak (salvodorapersicalinnsalvodoracae): An overview. Pharmacology online 2010; 2(2):534 – 48.
15. Dhalkari C, Wagatkar J and Ingle K. Herbal remedies in periodontics. International journal of information research and review2016; 03(02):1884-86.
16. Adnan Sukkarwala, SalimaMehboob Ali, Pranee Lundberg, FarzeenTanwir. Efficacy of miswak on oral pathogens. Dental research journal 2013; 10(3): 314-20.
17. Mohammad M.Haque, Saeed A.Alsareii. A Review of the therapeutic effect of using miswak(Salvodorapersica) on oral health, Saudi Med J 2015; 36 (5); 530-43. .
18. Mahmoud YM Taha. Antiviral effect of ethanolic extract of Salvadorapersica (Siwak) on Herpes simplex virus infection. Al-Rafidin Dent J.2008; 8(1):50-1.
19. Verma Rajesh, Purohit Suresh, Bhandari Anil, Kumar Brijesh, P.Priyanka. SalvadoraPersica L (Tooth brush tree): A review. Journal of pharmacy research 2009; 2(12):1809- 12.

JSPIK

Clinical Evaluation of Fenugreek Toothpaste and Regular Toothpaste in Control of Gingivitis – A Comparative Study

Neethu Varghese[1], Amitha Ramesh[2], Raksha Potdar[3]

ABSTRACT

Introduction: Gingivitis is the inflammation of gingiva and it occurs mainly in response to bacterial biofilms/plaque accumulation on tooth surfaces. There are non-plaque induced gingivitis but plaque induced gingivitis being the commonest, is considered in this study. Mechanical aids like toothbrush, tooth paste, dental floss can be used to remove plaque. Recently, consumers have switched from regular to herbal toothpastes to avoid synthetic constituents. One of the medicinal herbs being considered recently in the industry is Fenugreek. There are limited studies available on its efficacy.

Objective: To compare the effectiveness of a commercial fenugreek toothpaste with a regular toothpaste in controlling moderate gingivitis.

Methodology: 30 subjects randomised into 2 groups of toothpaste usage – Regular (control) and Fenugreek toothpaste (test). Gingival index, Simplified Oral hygiene index (OHIS) and Sulcus bleeding index were measured at baseline and 14 days after usage of toothpaste. Gingival bleeding being an early indication of gingivitis, Sulcus bleeding index was determined mainly for comparison.

Results: Fenugreek toothpaste was found to be effective in the treatment of gingivitis with reduction in the clinical parameters of gingivitis. The results obtained showed that there is no statistically significant difference between the control group and the test group in relation to sulcus bleeding index and OHIS index. The Gingival index before and after, in control and in test group, calculated using Wilcoxon signed ranks test showed a p value of 0.001, which is statistically significant. This suggests significance of fenugreek toothpaste in reducing gingival inflammation. There was significant improvement of gingival health on usage of fenugreek toothpaste by patients with chronic gingivitis. However, there is no significant difference between the effects of regular toothpaste (control) and fenugreek toothpaste(test) results.

Conclusion: The use of fenugreek toothpaste as an alternative to conventional toothpaste can be explored.

KEY WORDS: gingivitis, fenugreek, bleeding, index

Introduction

Genco, in 1990 stated that "gingivitis is inflammation of gingiva when the junctional epithelium remains attached to the tooth at the original level".[1] Gingival bleeding is the early indication of gingivitis. In 1971, Muhlemann demonstrated that bleeding from the gingival sulcus was the earliest clinical sign of gingivitis and it precedes discolouration and swelling of the gingival units.[2] Lennox and Kopezyk noted that bleeding points do not correlate directly with clinical inflammation but rather precede apparent inflammation.[3]

Recent investigations have stated that gingivitis develop within 2 weeks without proper oral hygiene

[1]Intern, [2]Professor, [3]Post graduate student, Department of Periodontics, AB Shetty Memorial Institute of Dental Sciences, NITTE (Deemed to be University), Deralakatte, Mangalore, Karnataka. Corresponding Author: Neethu Varghese, Email: neethu.nazareth@gmail.com

and early carious lesions may be detected after about four weeks, when the plaque is allowed to accumulate.[4] Effective plaque removal daily is one of the key basis for prevention of these two oral diseases – gingivitis and carious lesions, as the presence of plaque could be one of the main factors for dental caries, gingivitis, periodontal problems and halitosis.

The mechanical aids that can be used to control plaque include – toothbrushes, dental floss, mouth rinses and dentifrices. Numerous toothpastes are available in the market. Toothpaste is a paste or gel dentifrice used with a toothbrush to clean and maintain the aesthetics and health of gingiva and teeth. It serves as an abrasive that aids in removing dental plaque and food from the teeth. In the last decade, many consumers have switched over to herbal toothpastes in order to avoid synthetic and artificial flavours commonly found in regular toothpastes.

Various medicinal herbs are used in the formulation of Herbal toothpastes. One among the very recent herbs being used is -Fenugreek. Fenugreek is used as an herb (dried or fresh leaves) spice (seeds), and vegetable (fresh leaves, sprouts, and micro greens).[5]

Various ayurvedic uses of fenugreek for oral diseases have been identified. Infusion of fenugreek leaves is used as a gargle for recurrent mouth ulcers. In chronic infection of gingiva, the tooth powder prepared with fenugreek seeds powder, cardamom, and triphala are used. Fenugreek in the form of tea is used to cure the infection in an abscessed tooth.

Rationale for study

One of the recently used beneficial herbs in toothpastes for oral health in the market is - Fenugreek. Studies are limited regarding the efficacy of fenugreek extract in herbal toothpastes towards oral health. Fenugreek being an anti inflammatory herb and containing mucilagins which help in soothing inflamed tissues,[5] the oral health aspect undertaken in the current study to record the efficacy of fenugreek extract in herbal toothpastes is - Gingivitis.

Gingivitis is inflammation of the gingiva in response to bacterial biofilms / plaque that is accumulated on tooth surfaces. Though there are non plaque induced gingivitis like- of bacterial, viral, fungal, genetic, traumatic, systemic origin, plaque induced gingivitis is the most common. In the current study, Plaque induced gingivitis is considered.

Hence the present study is undertaken to compare the effectiveness of a commercially available "fenugreek" toothpaste with a regular toothpaste in controlling moderate gingivitis.

Aim of the study

To compare the effectiveness of a commercially available "fenugreek" toothpaste with a regular toothpaste in controlling moderate gingivitis.

Subjects and methods

The double blinded study comprised of 30 subjects in the age group 18-45 years, selected from the Department of Periodontics, A B Shetty Memorial Institute of Dental Sciences, Mangalore.

The subjects were randomised using coin toss method into 2 groups. Group A- 15 subjects were given regular toothpaste, Group B- 15 subjects were given commercially available fenugreek toothpaste.

Inclusion criteria

1. Subjects with a minimum complement of 20 teeth.
2. Subjects considered with Gingival index score of 1.1 – 2 (moderate gingivitis).[6]
3. Subjects considered with Simplified Oral Hygiene Index score of 1.3 – 3.0 (fair).[7]
4. Patients who are not allergic to fenugreek or any kind of toothpaste component.

Exclusion criteria

1. Presence of any other systemic disease.
2. History of periodontal treatment in the past 6 months.
3. Use of medications like NSAIDS, antibiotics or any drug known to influence the periodontal tissues and smokers
4. Pregnant or lactating women.

Screening examination

A detailed dental and medical history was recorded and gingival index, simplified oral hygiene index (OHIS) and sulcus bleeding index scores were measured at baseline and after 14 days.

Methods

Gingival Index, Simplified Oral Hygiene Index and Sulcus Bleeding Index was measured at baseline followed by oral prophylaxis, and follow up conducted 14 days after the use of toothpaste. Toothpaste is asked to be used in pea size amount, daily, two times – morning and at night, for 14 days.

Results

Continuous variables were expressed in terms of mean and standard deviation. Data was statistically analysed by a software using Independent sample t-test/ Mann– Whitney U test and Wilcoxon Signed ranks test. p value < 0.005 will be considered as significant. Fenugreek toothpaste is effective in patients with gingivitis, as the p value obtained from statistical analysis is significant.

The sulcus bleeding index between control and test group after toothpaste usage calculated according to unpaired t test showed a mean difference of 0.085 and p value of 0.43, which is not statistically significant. The OHIS after toothpaste usage between control and test group according to unpaired t test showed a mean difference of 0.083 with a p value of 0.65, which is not statistically significant.

The sulcus bleeding index before and after in control group calculated according to paired t test showed a mean difference of 1.29 with p value of 0.00, which is statistically significant.

The OHIS before and after in control group calculated according to paired t test showed a mean difference of 1.03 with a p value of 0.00, which is statistically significant.

The sulcus bleeding index before and after in test group was calculated using paired t test showed a mean difference of 1.98 and p value of 0.00 which is statistically significant.

The OHIS before and after in test group was calculated using paired t test showed a mean difference of 1.44 with a p value of 0.00, which is statistically significant.

The gingival index before and after usage of regular toothpaste in control group was calculated using Wilcoxon signed ranks test showed a p value of 0.001, which is statistically significant.

The gingival index before and after use of fenugreek toothpaste in test group, calculated using Wilcoxon signed ranks test showed a p value of 0.001, which is statistically significant.

Table 1: Unpaired t test of sulcus bleeding index and OHIS

group	N	Mean	Std. Deviation
control	15	0.4593	0.31601
Sulcus bleeding After			
Test	15	0.3740	0.27495
control	15	0.8867	0.39797
OHIS After			
Test	15	0.9700	0.59094

(OHIS: Simplified Oral Hygiene Index)

Table 2: Sulcus bleeding index and OHIS paired t test for control group

	Mean	Std. Deviation
Sulcus bleeding Before	1.7567	0.62944
Pair 1		
Sulcus bleeding After	0.4593	0.31601
OHIS Before	1.9200	0.99657
Pair 2		
OHIS After	0.8867	0.39797

(a: group = control)

Table 3: Sulcus bleeding index and OHIS paired t test for test group

	Mean	Std. Deviation
Sulcus bleeding Before	2.3627	0.90754
Pair 1		
Sulcus bleeding After	0.3740	0.27495
OHIS Before	2.4133	0.84673
Pair 2		
OHIS After	0.9700	0.59094

(b: group = test)

Discussion

Various studies have shown beneficial effects of fenugreek on general and oral health. Fenugreek has the properties which can be useful in controlling the growth of micro-organisms, and reducing inflammation of the gingiva - antibacterial, anti inflammatory, and anti oxidant properties. Fenugreek is an excellent source of selenium, an anti-oxidant. Fenugreek has mucilagins which helps in soothing and relaxing inflamed tissues.[5]

Gingival bleeding is the earliest clinical sign of gingivitis, and it precedes discolouration and swelling of the gingival units.[2] Therefore, one of the main parameters of this study involves the determination of sulcus bleeding.

In our study we used gingival index, simplified oral hygiene index and sulcus bleeding index which are indicators of gingival health.

The study focused on the effects of fenugreek toothpaste on gingival inflammation, bleeding and plaque formation. The results obtained showed that there is no statistically significant difference between the control group and the fenugreek toothpaste test group in relation to sulcus bleeding index and OHIS (Table 1). This result is similar to the study conducted by Rooplai Gupta et al,[9] which concluded that the herbal toothpaste was as effective as the conventionally formulated fluoride dentifrice in controlling plaque and gingivitis.

The Sulcus bleeding index before and after, in test and control group, calculated using paired t test showed a p value of 0.00 which is statistically significant.

The Oral hygiene index – Simplified before and after, in test and control group, calculated using paired t test showed a p value of 0.00 which is statistically significant. (Table 2, 3)

The Gingival index before and after, in control and test group, calculated using Wilcoxon signed ranks test showed a p value of 0.001 which is statistically significant.

This suggests that the use of fenugreek toothpaste reduces gingival inflammation. The result is similar to a study conducted by George J et al, which demonstrated statistically significant reduction in gingival inflammation by herbal toothpastes.[10]

Diosgenin, a steroid sapogenin found in fenugreek is the starting compound for over 60% of the total steroid production by the pharmaceutical industry. Externally, the seeds may be applied as a paste to treat abscesses, boils, ulcers and burns.

At present, there is a considerable commercial interest in growing fenugreek for its high sapogenin content.[5]

This study shows there was significant improvement of gingival health on use of fenugreek toothpaste by patients with chronic gingivitis. However, there is no significant difference between the effects of regular toothpaste (control) and fenugreek toothpaste(test) results.

Summary and conclusion

Fenugreek toothpaste is found to be effective in the treatment of gingivitis with reduction in the clinical parameters of gingival inflammation like, bleeding, oedema, and change in colour. Maintaining gingival health is essential for oral and systemic health of the patient.

Bleeding being one of the active parameters of gingival inflammation, determination of bleeding in control and test group, before and after usage of toothpaste, have been made using Sulcus bleeding index in this study. The Sulcus bleeding index before and after, in control and test group, calculated using paired t test showed statistical significance.

In our study, the use of fenugreek toothpaste reduced gingival inflammation and bleeding. Hence, the use of fenugreek toothpaste as an alternative to conventional toothpaste can be explored. It can be considered as a treatment modality as an adjunct to oral prophylaxis. However, not many studies have been conducted on fenugreek toothpaste and its effect on periodontal health. To further evaluate the medicinal properties of fenugreek, more expanded longitudinal studies are required.

Grant support

This research work was supported by Grant from NITTE University [NITTE UNIVERSITY SHORT TERM UNDERGRADUATE FELLOWSHIP AWARD Application Id:NUSR2/2018/10/07]

References

1. Genco RJ, Goldman HM, Cohen DW: Contemporary periodontics. St. Louis, Mosby. 1990; 113-8.
2. Muhlemann, H. R. and Son, S.: Gingival Sulcus Bleeding: A Leading Symptom in Initial Gingivitis. Helv. Odontol. Acta., 15:107, 1971.
3. Lenox, J. A. and Kopczyk, R. A.: A Clinical System for Scoring a Patient's Oral Hygiene Performance. J. Am. Dent. Assoc., 86:849, 1973.
4. Newman MG, Takei HH, Klokkevold PR. Carranza's Clinical Periodontology. 11th ed. Elsevier, 2006.
5. Toshiyuki MURAKAMI, Akinobu KISHI, Hisashi MATSUDA, and Masayuki YOSHIKAWA Medicinal Foodstuffs. XVII.1) Fenugreek Seed. Chem Pharm Bull 2000; 48(7): 994-1000.
6. Essentials of Preventive and Community Dentistry (Public Health Dentistry); 4th edition, page- 325,326,342.
7. Soben Peter, Essentials of Public Health Dentistry, 6th edition, Chp 22, Pg 485-87.
8. Gupta R, Ingle NA, Kaur N, Yadav P, Ingle E, Charania Z. Effectiveness of herbal and nonherbal fluoridated toothpaste on plaque and gingivitis: A randomized controlled trial. Journal of Indian Association of Public Health Dentistry. 2015 Jul 1;13(3): 218 – 21.
9. George J,Hegde S, Rajesh K S, Kumar A. The efficacy of a herbal – based toothpaste in the control of plaque and gingivitis:Aclinico-biochemical study. Indian J Dent Res 2009;20:480-2.

JSPIK

Comparative Evaluation of Diode Laser with other different types of Desensitizing Agents for the Treatment of Dentinal Hypersensitivity– A Clinical Study

Swetha Chacko[1], Deepak Baby[2], Sreedevi P.V[3], Plato Palathingal[4], Drisya L[1]

ABSTRACT

Background & Aim: To compare and assess the efficiency of diode laser alone and in combination with sodium fluoride, Bioglass containing toothpaste, Duraphat varnish and Gluma desensitizer for the management of hypersensitivity.

Materials and method: Total of 10 patients contributing 25 teeth complaining of dentin hypersensitivity with visual analog scale (VAS) score being >2 were recruited and were divided into 5 groups. Group I: treated with bioglass containing toothpaste, Group II: Gluma desensitizer, Group III: duraphat varnish, Group IV: diode laser in continuous, non contact mode, Group V: diode laser in continuous, non contact mode with sodium fluoride application. Dentine hypersensitivity was evaluated using air blast and VAS score noted at baseline, immediately after treatment, at two weeks and four weeks intervals.

Statistical Analysis: Krusckal wallis test. **Results:** A reduction of Dentinal hypersensitivity (DH) was recorded at the end of 1 month. Group I showed better result than group II. Group III showed the least reduction in dentinal sensitivity, whereas group IV and V showed the best results.

Conclusion: Diode laser with sodium fluoride application showed superior result and appears to be a promising treatment alternative in alleviating sensitivity.

Key words: Bioactive glass, Dentinal hypersensitivity, Diode laser, Duraphat varnish, Gluma desensitiser.

Introduction

Dentinal hypersensitivity (DH), or cervical dentinal sensitivity, is a frequent clinical disorder with an incidence ranging from 4 to 74%. "Dentine hypersensitivity is characterized by short, sharp pain arising from exposed dentine in response to stimuli, typically thermal, evaporative, tactile, osmotic or chemical and which cannot be ascribed to any other dental defect or pathology".[1]

It can affect the patient of any age, most affected patients are in the age group of 20–50 years, with a peak between 30 and 40 years of age.[2] Regarding the type of teeth involved, canines and premolars of both the arches are the most affected teeth. Buccal aspect of cervical area is the commonly affected site.[3]

The most common factors involved in DH are abrasion, caused by inadequate intensity tooth brushing; abfraction, caused by teeth flexion due to abnormal occlusal forces; parafunctions or occlusal disequilibrium; erosion, secondary to the presence of acids in the oral cavity, as in bulimia nervosa or gas-

[1]Post graduate student, [2] Professor & HOD, [3] Professor, Department of Conservative Dentistry & Endodontics, [4]Professor, Department of Periodontics, P.S.M. College of Dental Science & Research, Akkikavu, Thrissur, Kerala 680519. Corresponding Author: Dr Swetha Chacko, E-mail: swethachacko@gmail.com

troesophageal reflux; anatomic predisposition due to structural deficiency of the enamel-cement junction; cavity preparations in vital teeth that expose dentine or badly controlled dentinal acid conditioning.[4]

The aim of this prospective clinical study was to evaluate and compare the clinical efficacy of diode laser alone and in combination with sodium fluoride gel with bioactive glass containing tooth paste and gluma desensitiser in the management of dentin hypersensitivity.

Materials and method

This clinical study was carried out in the Department of Conservative Dentistry & Endodontics and Department of Periodontics, P.S.M Dental college, Thrissur. The patients recruited for this study were selected from the outpatient department. Ten systemically healthy patients, previously untreated for DH aged between 30-40 years with a chief complaint of dentin hypersensitivity, contributing 25 teeth were recruited for the study. Written informed consent was obtained from patients who were willing to participate voluntarily.

Inclusion criteria:

Patients in good systemic health with clinically elicitable dentin hypersensitivity and who were not treated earlier for DH were included in the study.

Exclusion criteria:

Patients with any systemic conditions, those who had used any desensitizing paste or mouthwash during the last 6 months, those who were on any analgesics/ anti-inflammatory drugs at the time of the study and pregnant patients were excluded from the study. Cracked teeth, large carious lesions, or restored teeth were also excluded from the study.

Twenty five teeth from ten patients were randomly divided into five groups of five teeth each. (Figure:1). Before treatment, all the patients underwent scaling and root planning. The degree of sensitivity to stimulus before and after treatment was determined with a cold air stimulus. To check the air stimulus, the selected tooth was isolated, dried, and a jet of cold air was applied from a distance of 1 cm for 1 second and response to air stimuli was recorded according to Visual Analog Scale (VAS). Recordings were assessed at baseline, immediately after treatment and at two weeks and four weeks intervals.

Treatment procedure

Group I: Bioactive glass containing tooth paste (Shy NM) to be used twice daily was given.

Group II: The tooth was conditioned with Gluma Etch 20 Gel for 20 seconds. Then it was rinsed, air dried and the tooth surface was moistened by pellets damped with distilled water. Gluma Desensitizer was applied using a disposable brush applicator. An additional coat of the Desensitizer was applied and light cured for 15 seconds.

Group III: Each sensitive tooth was cleaned with a polishing paste, rinsed with water and air dried. Duraphat was applied using a disposable brush applicator. An additional coat was applied after 5 minutes.

Group IV: Selected teeth were isolated with cotton rolls and diode laser having a wavelength of 810 nm was irradiated in noncontact, continuous mode with a power of 0.7 W for 1 min. Each site received three applications of 1 minute each.(Figure:2)

Group V: Selected teeth were isolated with cotton rolls. 1.25% NaF gel was left on tooth surface for 60 seconds before the irradiation. Diode laser having a wavelength of 810 nm was irradiated in noncontact, continuous mode with a power of 0.7 W for 1 min. Each site received three applications of 1 min. (Figure:3)

Figure 1: Pre operative photograph

Figure 2: NaF application

Figure 3: LASER application

Each group is divided into four subgroups:

Sub group 1: VAS at baseline

Sub group 2: VAS immediately after treatment

Sub group 3: VAS after two weeks

Sub group 4: VAS after 4 weeks

Statistical Analysis

The data analysis was carried out using SPSS (version 23). Descriptive statistics were calculated. Mean & standard deviation for VAS score was calculated. Statistical analysis was done using Krusckal Wallis test.

Results

All the groups registered showed a significant reduction of Dentinal Hypersensitivity during the treatment sessions. Comparing the five regimens, a higher decrease of Dentinal Hypersensitivity was registered in Group V followed by Group IV, Group I, Group II & Group III. The VAS was valued at baseline, immediately after treatment, at two weeks and four weeks intervals. Results are demonstrated in table 1 and graph 1.

Discussion

According to Miglani.S et al dentinal sensitivity can affect the patient of any age, most affected patients are in the age group of 20–50 years, with a peak between 30 and 40 years of age. So in this study patients under the age group of 30-40 years of age are included.[2] The degree of dentine hypersensitivity was evaluated by visual analogue scale (VAS) in accordance with Gojkov-Vukelic. M et al.[5]

In the present study, it was noted that after 1 month of clinical follow up, all desensitizing agents

TABLE 1: Comparison between each groups

		Minimum	Maximum	Median	Mean	Std. Deviation	P value (Krusckal wallis test)
Group 1	Sub group 1	2.00	4.00	3	2.8000	.83666	0.115
	Sub group 2	2.00	4.00	3	2.8000	.83666	
	Sub group 3	1.00	3.00	2	1.8000	.83666	
	Sub group 4	1.00	2.00	2	1.4000	.54772	
Group 2	Sub group 1	3.00	5.00	4	3.8000	.83666	0.005
	Sub group 2	1.00	2.00	2	1.8000	.44721	
	Sub group 3	1.00	3.00	2	1.8000	.83666	
	Sub group 4	1.00	3.00	2	1.8000	.83666	
Group 3	Sub group 1	2.00	4.00	3	3.0000	1.00000	0.243
	Sub group 2	.00	2.00	1	1.2000	.83666	
	Sub group 3	1.00	3.00	2	2.2000	.83666	
	Sub group 4	1.00	3.00	2	2.2000	.83666	
Group 4	Sub group 1	3.00	4.00	3	3.4000	.54772	0.008
	Sub group 2	1.00	2.00	2	1.6000	.54772	
	Sub group 3	.00	2.00	1	.8000	.83666	
	Sub group 4	.00	1.00	1	.6000	.54772	
Group 5	Sub group 1	2.00	5.00	3	3.0000	1.22474	0.005
	Sub group 2	1.00	2.00	1	1.2000	.44721	
	Sub group 3	.00	2.00	0	.8000	.83666	
	Sub group 4	.00	1.00	0	.4000	.54772	

were capable of reducing dentin hypersensitivity. This was in accordance with a study conducted by Bilichodmath. R et al.[3] Sgolastra et al found that lasers are efficient in reducing DH without damage to the pulp and without adverse effect.[5] Lasers in association with desensitizing agents shows a synergistic action. In this study better result was obtained from Group V than Group IV. This may be because the laser system can favour the permanence of the sodium fluoride for longer time than when they are used alone.[4] The higher effectiveness of the use of the diode laser associated to NaF may be related to the action of diode laser that could allow a deeper penetration into the dentin tubules of NaF facilitating the precipitation of CaF2 which in combination with laser-induced occlusion for superficial melting of protein denaturation into dentin fluid, maintaining the occlusion of tubules longer, lessening the discomfort from dentin hypersensitivity.[6]

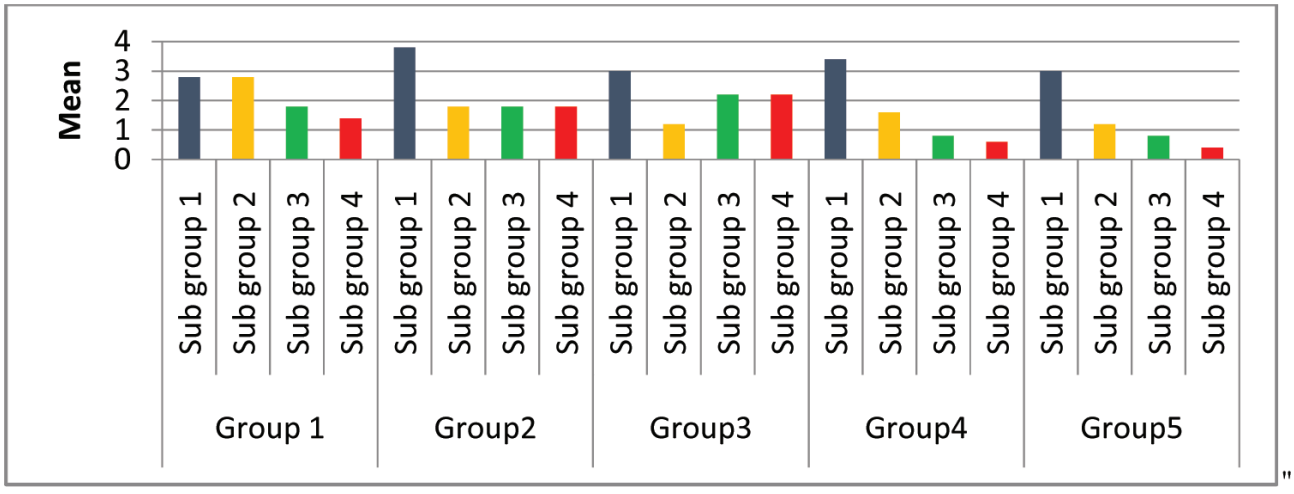
It is postulated that a low intensity laser mediates an analgesic effect related to the depolarization of C-fiber afferents. This interference in the polarity of cell membranes by increasing the amplitude of the action potential of cell membranes can block the transmission of pain stimuli in hypersensitive dentin.[7]

Histological studies have reported that hard tissue formation is enhanced as a reaction of dental pulp to laser light. According to the reports published by Asnaashari et al.and Aranha et al, Low-power laser therapy for DH is an appropriate treatment strategy to promote biomodulatory effects, minimizes pain, and reduce inflammatory processes.[8] According to Femiano. F et al higher efficacy with clear improvements of discomfort was recorded using the laser diode in association with the NaF compared to NaF and diode laser used alone.[6]

Bioactive glass containing tooth paste showed the next better result. In the case of products with NovaMin, the active ingredient is a calcium sodium phosphosilicate (CSP).[9] NovaMin (CSP) is a bioactive glass in the class of highly biocompatible materials that were originally developed as bone-regenerative materials. In saliva, sodium ions (Na+) in CSP particles immediately begin to exchange with hydrogen cations (H+ or H3O+). This rapid exchange of ions allows calcium (Ca2+) and phosphate (PO43−) species to be released from the particle structure. A modest, localized, transient increase in pH occurs that facilitates the precipitation of calcium and phosphate from the particles and from saliva to form a calcium phosphate (Ca-P) layer on tooth surfaces. As the reactions and the deposition of Ca-P complexes continue, this layer crystallizes into hydroxycarbonate apatite, which is chemically and structurally similar to biological apatite.[10]

According to Gupta. A.K, Gluma desensitizer produced a greater number of partially occluded tubules and fewer completely occluded tubules, while in the case of specimens treated with NovaMin, a greater number of completely occluded tubules and fewer partially occluded tubules were observed. This might be due to the mechanism of Gluma desensitizer,

GRAPH 1: Mean Value Comparison between Groups



which reportedly is based on total or partial closure of the tubules by protein coagulation and precipitation upon reaction with glutaraldehyde and hydroxyethyl methacrylate. DuMin et al found that NovaMin to be a more effective desensitizer than Gluma.[9]

Gluma desensitizer reduced sensitivity more than Duraphat but is inferior to NovaMin containing toothpaste at all measurement intervals. The Gluma Desensitizer product contains 5% glutaraldehyde and 35% hydroxyethyl methacrylate (HEMA). The immediate occlusion of the dentin tubules is due to the effect of glutaraldehyde on the proteins of the dentinal fluid.[9] Glutaraldehyde seals the dentin tubules by coagulation of the dentin fluid proteins within the tubules, thereby counteracting the hydrodynamic mechanism of DH. HEMA, being water soluble, might promote deep penetration of glutaraldehyde into the tubules up to 200 μm leading to formation of a peripheral intrinsic barrier consisting of multiple thin septa within its lumen.[11]

In a study conducted by Mehmood. Z et al showed that Gluma Desensitizer as a better agent in relieving dentinal hypersensitivity than Duraphat in non-carious cervical lesions.[12] According to Brahmbhatt et al, Gluma showed better immediate effect as compared to topical 2% sodium fluoride.[13]

Duraphat varnish showed least reduction of DH (Group III). This is likely to be due to the intradentinal sealing observed with sodium fluoride takes time to form calcium fluoride crystals.[14] The fluoridated substances react chemically with the calcium and phosphate ions providing a precipitation of CaF2 crystals within the dentinal tubules without adhesion.[6,7] Because it is an unstable compound, CaF2 rapidly dissociates after application.[7] These precipitates are removed by the mechanical means and saliva.[14] Corona et al reported that the efficacy of fluoride varnish decreases from 15- 60 days during tooth brushing.[8]

Conclusion

According to these results, the Diode laser showed a very high capability to improve immediately the Dentinal hypersensitivity-related pain, both alone and even better in combination with NaF gel.

References

1. Biagi.R, Cossellu.G, Farronato.G. Laser-assisted treatment of dentinal hypersensitivity: a literature review. Annali di Stomatologia.2015;6(3-4):75-80.
2. Miglani.S, Aggarwal.V, Ahuja.A. Dentin hypersensitivity: Recent trends in management. J Cons Dent.2013;13(4):218-24.
3. Bilichodmath.R, Kumar.R.V, Bilichodmath.S, Sameera.U. Diode Laser in the Treatment of Dentinal Hypersensitivity: A Reliable Approach. J Dent Lasers.2018;12:56-62.
4. Umberto.R, Claudia.R, Alessandro.D.V. Treatment of Dentine Hypersensitivity by Diode Laser: A Clinical Study. Inter J Dent.2012:1-8.
5. 5. Gojkov-Vukelic.M, Hadzic.S, Pavlic.V. Application of Diode Laser in the Treatment of Dentine Hypersensitivity. Med Arch.2016;70(6):466-9.
6. Femiano.F, Femiano.R, Lanza.A, Festa.M.V, Rullo.R, Perillo.L. Efficacy of diode laser in association to sodium fluoride vs Gluma desensitizer on treatment of cervical dentin hypersensitivity. A double blind controlled trial. Am J Dent.2013;26:214-8.
7. Aranha.A.C.C, Pimenta.L.A.F, Marchi.G.M. Clinical evaluation of desensitizing treatments for cervical dentin hypersensitivity. Braz Oral Res.2009;23(3):333-9.
8. Aghanashini S, Puvvalla B, Nadiger S, Mundinamanae DB, Bhat D, Andavarapu S. Comparative evaluation of diode laser and fluoride varnish for treatment of dentin hypersensitivity: A clinical study. J Interdiscip Dentistry.2018;8:110-7.
9. Gupta.A.K, Sharma.N, Bramta.M. Dentin tubular occlusion with bioactive glass containing dentrifice and gluma desensitizer– a comparative sem evaluation. Dent J Adv Stud.2014;2(1):16-21.
10. Majji P, Murthy KR. Clinical efficacy of four interventions in the reduction of dentinal hypersensitivity: A 2-month study. Indian J Dent Res.2016;27:477-82.
11. Pandey R, Koppolu P, Kalakonda B, Lakshmi BV, Mishra A, Reddy PK, et al. Treatment of dentinal hypersensitivity using low-level laser therapy and 5% potassium nitrate: A randomized, controlled, three arm parallel clinical study. Int J App Basic Med Res.2017;7:63-6.
12. Mehmood.Z, Jawad.A.S, Javed.M.U, Manzoor.M, Asghar.I, Bin Saeed.M.H. Efficacy of gluma desensitizertm and duraphatt m in relieving dentinal hypersensitivity in non-carious cervical lesions. Pak Oral Dent J.2011;31(1):183-6.
13. Verma.S, Boppana.N, Valleri.U, George.A, Shetty.M.S. Comparison of the Effi cacy of Two Different Desensitizing Agents: A Randomized Controlled Trial. J Int Acad Periodontol.2017;19(1):22-7.
14. Shakoor.M.M, Tayab.R, Riaz.M.A. Management of Dentinal Hypersensitivity Using Fluoride Application and Dentine Bonding Agent: a Clinical Study to Explore the Best Outcome in Getting Relief from Pain Associated with Hypersensitivity. Pak J Med Health Sci.2017;11(1):453-5.

JSPIK

Periodontal Treatment and Maintenance – Implication in the Covid Scenario

Punnyamol P.G[1], Mahesh Narayanan[2], Shabeer Ahamed[3], Haris Muhammed[4], Ramya Ganesh[5]

ABSTRACT

The outbreak of coronavirus disease 2019 (COVID-19) rapidly escalated into a worldwide pandemic, creating a global health and economic crisis. Signs and symptoms include dry cough, fatigue, coughing sputum, and shortness of breath. Transmission mainly occurs by spreading droplets or touch routes. Due to the continuous production of aerosol in periodontal clinics and because of the fact that the virus remains alive for 12 h, periodontists are highly susceptible and prone for infection. This article offers a brief description of the virus structure, transmission modes and clinical characteristics of COVID-19 disease. The aim of this article is to recommend infection control strategies and patient management protocols to provide optimum periodontal care and simultaneously prevent nosocomial infection in periodontal settings.

Key Words: Coronavirus, Covid -19, periodontal practice, periodontal maintenance

Introduction

The outbreak of novel coronavirus (2019-nCoV) pneumonia emerged last December in one of China's largest cities, Wuhan, in Hubei Province, and has become a major challenge to public health not only for China but also for countries around the world.[1] On January 30, 2020, the World Health Organization (WHO) announced that this outbreak had constituted a public health emergency of international concern.[2] The two main unique features of this virus are its low pathogenicity and high transmissibility that distinguish it from other members of the coronavirus family such as SARS-CoV (Severe Acute Respiratory Syndrome) and MERS-CoV (Middle Eastern Respiratory Syndrome).[3] It is a highly infectious disease and its clinical symptoms include fever, dry cough, myalgia and fatigue and severe cases progressing to acute respiratory distress syndrome leading to bleeding and coagulation dysfunction.[4]

The risk of increased severity is noticed in the elderly and individuals with underlying chronic diseases. Human-to - human transmission of the virus occurs from an infected person or from a contaminated surface through airborne droplets, contact, or touch.

Health care workers are amongst the most vulnerable group who have the greatest risk of getting infected. There have been reports of medical staff acquiring the disease while taking care and treating infected individuals[5]. According to Occupational Safety and Health Administration (OSHA), dental health care personnel (DHCP) are placed in very high exposure risk category as dentists work in close proximity to the

[1]Post graduate student, [2]Professor and Head, [3]Professor, [4]Reader, [5]Senior Lecturer, Department of Periodontics, Malabar Dental College & Research Centre, Edappal, Malappuram - 679 578, Kerala. Corresponding Author: Dr Punnyamol P.G E mail: drmaheshn@gmail.com

patient's oral cavity.[6] Also, dental procedures involve the use of rotary instruments such as handpieces and scalers, which generate aerosols. Thus, a greater understanding of the structure of the virus, modes of transmission, clinical features, and testing methods is needed that can help to form protocols for dental practices to identify cases and prevent further spread of infection to the patients and DHCP.

In the mouth the fluids are contaminated with bacteria and viruses. A significant source of such species is the dental plaque, both supragingival and subgingival in the periodontal pocket. However, it should not be overlooked that the mouth is also a member of the oronasal pharynx. As part of this complex, the mouth harbors bacteria and viruses from the nose,throat and respiratory tract. These can involve numerous pathogenic viruses and bacteria found in the oral fluids and the saliva.[7]

Most periodontal procedures that use mechanical instrumentation will produce airborne particles from the site where the instrument is used. Dental hand pieces, ultrasonic scalers, air polishers and air abrasion units produce the most visible aerosols. Each of these instruments removes material from the operative site that becomes aerosolized by the action of the rotary instrument, ultrasonic vibrations or the combined action of water sprays and compressed air. The water spray usually is the portion of the aerosol that is most visible to the naked eye and is noticed by the patient and dental personnel.[8]

Due to the characteristics of periodontal settings, the risk of cross infection may be high between periodontists and patients. In spite of relentless research over pharmaceutical treatment being done every minute, there is currently no vaccine or specific antiviral treatment, which makes this infection even more scary and serious. However, efforts aiming toward managing symptoms and supportive therapy have been widely recommended.[9]

Periodontal settings invariably carry the risk of 2019-nCoV infection due to the specificity of its procedures, which involve face-to-face communication, generation of aerosol, frequent exposure to body fluids such as saliva, blood, and the handling of sharp instruments. Hence, it becomes imperative that periodontists should be familiar with how 2019-nCoV spreads, how to identify patients with 2019-nCoV infection, and what extra protective measures should be adopted during the practice to prevent the transmission of 2019-nCoV.

This article is based on the relevant guidelines given by various reputed organizations which deals with introduction of the essential knowledge about COVID-19 and nosocomial infection in periodontal settings, providing recommended treatment and maintenance protocol for periodontists in (potentially) affected areas.

Structure

SARS-CoV-2 is the seventh member of the family of coronaviruses that infect humans. Although similar to some beta coronaviruses, it is distinct from SARS-CoV and MERS CoV. It is a novel virus belonging to the subgenus sarbecovirus, Orthocorona virinae subfamily, with Chinese horseshoe bats (Rhinolophus sinicus) being the most probable orgin.[10] It is an enveloped positive-stranded RNA virus, 60–140 nm in diameter, spherical or elliptical in form, and pleomorphic, exhibiting a crown-like appearance under an electron microscope (coronam is the Latin word for the crown).[11, 12] (Figure 1)

Incubation Period

The average duration of incubation for COVID-19 is 5 to 6 days, although there is evidence that it may be as long as 14 days, which is now the widely accepted duration for medical monitoring and quarantine of (potentially) exposed persons.[13]

Clinical manifestations

Common symptoms at onset of illness include fever, non-productive cough, myalgia, or fatigue; less common symptoms are sputum production, headache, haemoptysis, and diarrhoea.[14]

Another common symptom is pneumonia which can be seen on chest X-ray or chest CT as multiple small patchy shadows and interstitial changes, remarkable in the lung periphery. Organ dysfunctions such as acute respiratory distress syndrome (ARDS), acute

cardiac injury (shock and arrhythmia), acute kidney injury, and death can occur in severe cases.[14,15] Age and comorbidity have been found to be risk factors for poor outcome.[15]

Transmission

The three most common transmission routes[16] of novel CoV include:

1. Direct transmission (through cough, sneeze, or droplet inhalation)

2. Contact transmission (through oro nasal ocular route) and

3. Aerosol transmission.

Asymptomatic carriers of the infection are equally capable of transmitting the virus as symptomatic patients.[13] The SARS CoV 2 virus can be detected in aerosols up to 3 h postoperatively, and can persist on surfaces for extended periods. The nature of the surface alters the persistence of the virus. On copper surfaces, the virus can persist for up to 4 h, on cardboard up to 24 h, and on plastic and stainless steel up to 2–3 days.[17] The droplet and aerosol transmission of SARS CoV 2 is the most important concern in dental clinics and hospitals[18] because it is hard to avoid the generation of large amounts of aerosol and droplet mixed with patient's saliva and even blood during dental procedures.[19]

Diagnosis

For COVID-19 research, different approaches are available, and the decision to carry out a test on suspicious individuals should be focused on clinical symptoms and epidemiological factors. It would be helpful if rapid COVID19 identification kits were supplied to dental practises to screen high-risk and suspect patients. In this way, they can take appropriate measures to reduce the spread of the virus, as well as help the national healthcare system monitor potential infected cases and gather data about the number of infected cases in the country.[20]

Rapid diagnostic tests based on antigen detection

One form of rapid diagnostic test (RDT) detects the presence in a sample from a person's respiratory tract of viral proteins (antigens) expressed by the COVID-19 virus. If the target antigen is present in appropriate concentrations in the sample, it will bind to the particular antibodies attached to the paper strip found in the plastic casing and, usually within 30 minutes, produce a visually detectable signal. The identified antigen(s) are only expressed while the virus is actively replicating, so it is safer to use these tests to recognise acute or early infection. The performance of the tests depends on a variety of factors, including the period since the onset of the disease, the concentration of the virus in the sample, the quality of the sample obtained from the individual and how it is processed, and the exact composition of the reagents in the test kits. The sensitivity of these tests may be expected to range from 34 percent to 80 percent based on experience with antigen-based RDTs for other respiratory diseases such as influenza, in which infected patients

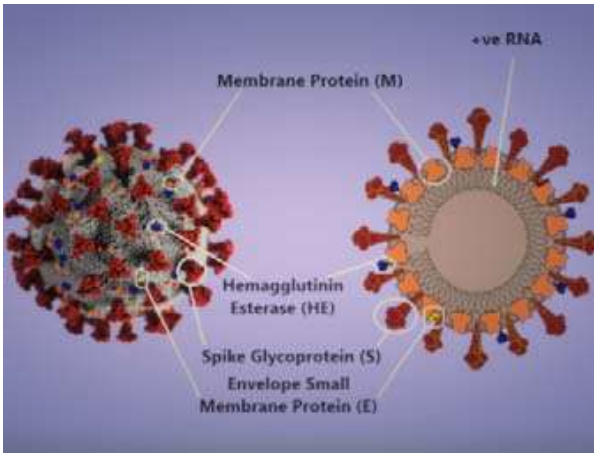


Figure 1

Figure 2

have comparable influenza virus concentrations in respiratory samples as seen in COVID-19.[21]

RT-PCR

In asymptomatic or mild symptomatic patients and those who have had interaction with COVID-19 positive cases, the RT-PCR test should be done according to WHO guidelines. It is important to follow and comply with screening protocols and local guidelines for patient assessment. Rapid collection of samples and testing of suspected cases using nucleic acid amplification test (NAAT) such as RT-PCR are a key step in outbreak management.[22]

In this process the RNA test is used to detect the genetic signature of SARD-CoV-2. A swab is usually used to obtain specimens from the inside of the nose or posterior part of throat. The outcome of PCR testing of nucleic acid which can detect even a small amount of RNA can be communicated within hours. The test is highly sensitive and specific but its accuracy depends on sample quality. The limitation of PCR testing is that it can only identify patients with active infection, and those in recovery do not have detectable viruses, hence, the test could be negative.[23]

Serological or Antibody Testing

Serological COVID-19 tests measure the presence of immune system-generated antibodies in the blood when the SARS-CoV-2 virus is detected. Most of the commercially available tests measure immunoglobulin M (IgM) and immunoglobulin G (IgG) antibodies in either serum, plasma, venous whole blood, or, preferably in a dental office, finger-stick whole blood. Most of the commercially available tests measure antibodies to immunoglobulin M (IgM) and immunoglobulin G (IgG) in either serum, plasma, whole venous blood, or finger-stick whole blood, preferably in a dental clinic. In response to a novel antigen, IgM antibodies are the first antibodies to appear which may indicate a recently initiated infection. In the course of infection, IgG antibodies have a greater affinity for the target antigen and are produced later. These serology tests are called lateral flow immunoassays and use cassettes (figure 2). containing a membrane-based immunoassay with a similar principle and look to that of an hCG pregnancy test. Ease of use and expediency are the advantage of the serology test as it can be ready in 10 minutes, making it particularly useful in a dental office.[24]

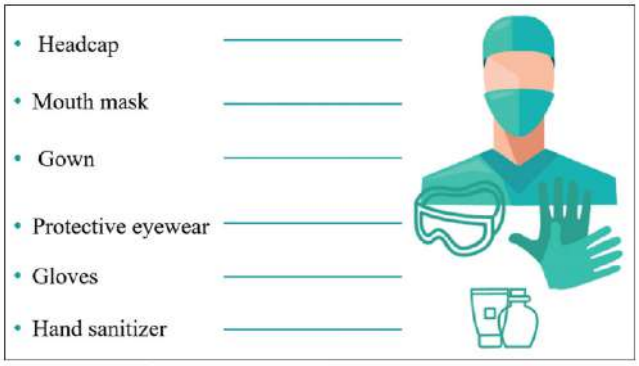


Figure 3 Protective wear for dental professionals as personnel in periodontal setup.

According to a statement from the US Food and Drug Administration (FDA):"Serological tests can play a critical role in the fight against COVID-19 by helping healthcare professionals to identify individuals who have overcome an infection in the past and have developed an immune response. In the future, this may potentially be used to help determine, together with other clinical data, that such individuals are no longer susceptible to infection and can return to work. In addition, these test results can aid in determining who may donate a part of their blood called convalescent plasma, which may serve as a possible treatment for those who are seriously ill from COVID-19."[25]

Most of the serology tests claim to have an IgM sensitivity of 62%–97% and an IgG sensitivity in the range of 86%–100% (with a 95% confidence interval).[26]

The problem with these tests is that many will falsely test negative in the early incubation phase of the disease, thus lowering the sensitivity of the test.

The majority of the serology tests claim to have an IgM specificity of 86%–100% and an IgG specificity of 90%–100% (with a 95% confidence interval), indicating that the number of people who falsely test positive is very low.

Medical Imaging

A review of the relevant medical imaging litera-

ture shows that chest radiographs do not have any diagnostic value in the early stages of infection. However, even before the onset of symptoms, CT scanning could present some findings.[27] The typical characteristic of CT in COVID-19 positive cases is bilateral multilobar ground-glass opacity associated with a peripheral asymmetric and posterior distribution. As the infection develops subpleural dominance, crazy paving and consolidation can be observed in most cases. A comparative study conducted in Wuhan, the origin of COVID-19, suggests that CT is significantly more sensitive than PCR test; however, it is less specific as many of its imaging characteristics overlap with other types of pneumonia.[28] American College of Radiology also recommends that CT imaging should not be used as a first-line test to screen potential COVID-19 cases.[29]

Treatment/Management

Pharmacological management

Clinical treatment for COVID-19 is predominantly symptomatic. In intensive care, extreme cases need respiratory intervention and organs support. Multiple pharmacotherapies were experimentally evaluated on COVID-19 patients throughout the globe. Some studies have shown faster clearance of the patient's virus, and some have shown decreased mortality. No large randomized scale-controlled trial has settled the argument of the true effectiveness of any medicine against COVID-19.[30]

Hydroxychloroquine/chloroquine and azithromycin

The most widely used dosage of hydroxychloroquine is 400 mg orally for two doses twice daily, followed by 400 mg orally for a total of 5 days daily. The dosage of chloroquine recommended by the FDA is 1 g on day 1, then 500 mg daily for a total of 4 to 7 days. Azithromycin is an antibacterial macrolide and worksBy binding to 50s ribosomal subunit and inhibiting polypeptide synthesis directed by messenger RNA.[31]

Remdesivir

The agent was first discovered during the finding of antimicrobials with action against RNA viruses in 2015. It was first used for treating Ebola.It showed promising results in studies of animals with MERS and SARS caused by a coronavirus.[30] The existing dose under investigation is a single loading dose of 200 mg, followed by a normal infusion of 100 mg.[32]

Favipiravir

By targeting RNA polymerase, Favipiravir inhibits influenza viral replication, and this mechanism is also being extended to the novel coronavirus, a single-stranded RNA virus that requires RNA polymerase for replication.[32]

Recommended dosage is 2.400 to 3.000 mg loading dose per 12 h for two doses, followed as maintenance dose by 1.200 to 1.800 mg twice day.[32]

Interleukin (IL)-6 pathway inhibitor

In the blood of COVID-19 patients, elevated levels of inflammatory markers, including IL-6, have been identified and poor prognosis has been documented in patients.IL-6 is a proinflammatory cytokine and binds to both the soluble receptor IL-6 (sIL-6R) and the membrane-bound receptor IL-6R (mIL-6R).The resulting complex activates an inflammatory response by interacting with the transducing component glycoprotein 130 (gp130), which can cause a cytokine storm. Sarilumab and tocilizumab are the two commonly available IL-6 inhibitors on the market, binding explicitly to sIL-6R and mIL-6R, and Block transduction signal.[33]

Lopinavir/ritonavir

The dosage of lopinavir / ritonavir commonly studied in COVID-19 patients is 400mg/100 mg twice a day for up to 14 days.

Histamine 2 receptor antagonist (H2RA)

While H2RA is a medication which is very widely used,No definitive evidence, even available over the counter, support the way H2RA helps against COVID-19. This thought originated from Michael Callahan, an infectious disease doctor at Massachusetts General Hospital. He observed that many of the COVID-19 survivors had chronic heartburn and took famotidine rather than omeprazole, which is more expensive

when he was working in Wuhan during the coronavirus epidemic began. It was later investigated in more than 6,000 patients who recovered and found a slightly higher number in the famotidine group, but was not high enough to be statistically significant.

Interferon (IFN) beta

IFN-beta is the most relevant IFN-I that should be administered as early as possible to optimise antiviral therapy and prevent viral complications.[34] No specific dose has been validated, especially for COVID-19. The general dosing guideline is being followed.

Convalescent plasma (cp)

CP has been in use for over 100 years to treat a variety illness starting with measles, polio, chickenpox to recent epidemics as SARS-CoV-1 epidemic (SARS) in 2003, H1N1 influenza pandemic (H1N1) in 2009 - 2010, avian influenza A (H5N1), Ebola and MERS-CoV epidemic in 2012. For over 100 years, CP has been used to treat a number of diseases, from measles, polio, chickenpox to recent epidemics such as the SARS-CoV-1 (SARS) outbreak in 2003, the H1N1 (H1N1) influenza pandemic in 2009-2010, the avian influenza A (H5N1), the Ebola and MERS-CoV outbreak in 2012.[35]

Shen et al[36] have published a preliminary study of five patients with COVID-19 who were severely ill and treated with CP from China. All five patients were mechanically ventilated, and one needed ECMO. The donor CP, an apheresis product, had demonstrable immunoglobulin G (IgG) and IgM anti-SARSCoV-19 antibodies and in vitro virus-neutralizing properties. The authors concluded that the CP might have contributed to the recovery, although the patients were also on lopinavir/ritonavir antiviral therapy and IFN.[37]

Plasma adsorption and exchange

Emerging evidence indicates that the control of cytokine storm by steroid or IL-6R blocking antibodies in critically ill COVID-19 patients may be beneficial.[33,38] Patients may develop sepsis, ARDS and multi-organ failure in cases of fulminant systemic infection that are not specific to coronavirus.

In the setting of sepsis, the use of blood purification therapy in the form of plasma adsorption or therapeutic plasma exchange (TPE) has been confirmed, but the same has not been adequately demonstrated in critically ill patients with COVID-19.[39,40] Knowledge of optimum management of critically ill COVID-19 patients in the late phase is quite limited. The presence of cytokine storms or pathogenic antibodies in patients with critically ill COVID-19 has a strong correlation with the intensity of the disease. Inflammation and antibody monitoring is critical, particularly in patients with persistent fever or abnormal coagulopathy who are infected with the virus. Expeditious cytokine storm management using plasma adsorption or TPE may be useful for limited COVID-19 patients.[41] If done with the near supervision of clinicians, these therapies are well tolerated, and chances of infection or bleeding are reduced. To date, in COVID-19, randomised trial results are not available on this, but such clinical trials should be conducted to allow use of blood purification therapy for suitable patients.

Periodontal Aerosolization

Periodontists, who treat patients using aerosolization are at an extremely dangerous risk of inoculation of themselves, their dental assistants, other office staff members, and reinoculation of the patients. Most risk occurs from splatter and droplet transmission to the midface of the operator and assistant, as well as the nasal area of the patient.[42,43] Periodontal treatment has a much higher incidence of droplet transmission than prosthetic treatment.[6] Ultrasonic and sonic transmission during nonsurgical procedures had the highest incidence of particle transmission, followed by air polishing, air/water syringe, and high-speed hand piece aerosolization.[44] One study found that ultrasonic instrumentation can transmit 100,000 microbes per cubic foot (~28 L) with aerosolization of up to six feet (1.8m), and, if improper air current is present, microbes can last anywhere from 35 minutes to 17 hours.[45]

Dental professionals should be familiar with how COVID-19 is transmitted, how to evaluate and identify patients with COVID-19, and what extra-protective measures should be adopted during the practice, in order to prevent the transmission of COVID-19.[45] The

body temperature of the patient should be measured in the first place. A contact-free forehead thermometer is strongly recommended for the screening.[46] A questionnaire should be used to screen patients with potential infection by SARS-CoV-2 before they could be led to the dental chair-side

Masks; Ordinary surgical masks have pores of about 2-10 microns, while the N95 has pores of about 0.3 microns, gloves, gowns and goggles or face shields are recommended to protect skin and mucosa from infected blood or saliva. As respiratory droplets are the main routes of virus transmission, particulate respirators.

Risk of Nosocomial Infection in Periodontal Settings

Dental OPD or clinics often receive a whole bunch of patients belonging to different age groups, habits, and strata of society. Some of them often belonging to vulnerable groups. The patients who cough, sneeze, or receive dental treatment including the use of a high-speed handpiece or ultrasonic instruments make their secretions, saliva, or blood aerosolize to the surroundings, contaminating dental apparatus with various pathogenic microorganisms or become exposed to a contaminated clinic environment. Thereafter, infections can occur to clinicians, their support staff, and other patients through the puncture of sharp instruments or direct contact between mucous membranes and contaminated hands.[6]

Due to the unique characteristics of dental procedures where a large number of droplets and aerosols could be generated, the standard protective measures in daily clinical work are not effective enough to prevent the spread of COVID-19, especially when patients are in the incubation period, are unaware they are infected, or choose to conceal their infection.

Diagrammatic representation of personal protective measure to be worn by both dental professional and personnel while performing any dental procedure has been shown in Figure- 3

Recommendations for Periodontal (Dental) Practice

Interim guidance on infection prevention and control during health care is recommended when COVID-19 infection is suspected.[16] Up to now, there has been no consensus on theprovision of dental services during the epidemic of COVID-19. On the basis of my experience and relevant guidelines given by the Dental Council of India, periodontists and other clinicians performing a periodontal procedure should take strict personal protection measures and avoid or minimize operations that can produce droplets or aerosols.

The following guidelines are suggested:[47,48]

1. All clinicians should be well versed and updated with all information on coronavirus infection and should ensure that the same is known to their support staff too.
2. Patient Evaluation: During COVID-19 outbreak, it is recommended that dental clinics establish pre-check triage to measure and record the temperature of each staff and patient as a routine procedure, and ensure that patients do not mask their temperature by taking counter drugs such as paracetamol before visiting the dental clinic.
3. Precheck staffs should ask patients questions about the health status and history of contact or travel, and should inform the police and other concerned administrative authorities immediately if they find themselves suspicious of the same.
4. Fever patients should be registered and taken to designated hospitals. If a patient has been to outbreak regions within the last 14 days, quarantine is recommended for at least 14 days after the police and other administrative authorities concerned are told.
5. Every patient and accompanying patient who enters and exits dental clinic should be provided with mouth masks and their hand should be sterilized using hand sanitizers.
6. The waiting room should have hand sanitizers and pictorial information in the form of charts or digital information in the form of bulletin boards regarding COVID-19 should be displayed
7. Each seat in the waiting area should have a minimum distance of 1 meter (3 feet) as a preventive social distancing measure. All patients including

their attendants should be counseled by the clinicans regarding COVID-19 and the need to stay home for quarantined.

8. The waiting room/clinic including handles and doors as well as dental chairs and other surfaces should be wiped several times in a day with alcohol-based disinfectant.
9. Oral examination: Preoperative antimicrobial mouth rinses with chlorhexidine gluconate (CHX) 0.12%[49], cetylpyridinium chloride (CPC) 0.05%,[50] povidone-iodine (PVP-I) 0.23%[51,52], and hydrogen peroxide (H2O2)1%[53,54] have been recommended to reduce the number of microorganisms in aerosols and drops during oral procedures.[55] Procedures that are likely to induce coughing should be avoided (if possible) or performed cautiously. Aerosol-generating procedures, such as the use of a 3-way syringe, should be minimized as much as possible. Intraoral X-ray examination is the most common radiographic technique in dental imaging; however, it can stimulate saliva secretion and coughing. Therefore, extraoral dental radiographies, such as panoramic radiography and cone-beam CT, are appropriate alternatives during the outbreak of COVID-19.
10. Thorough medical and travel history of each patient should be recorded before any clinical procedure.
11. Patients should be scrubbed with isopropyl alcohol extraorally.
12. All dentists and support staff should wash their hands thoroughly with soap and water and follow up with alcohol-based sanitizers before and after every patient.
13. Surgical scrubbing of hands is recommended
14. Staff and doctors should avoid touching their face, especially ear, nose, and mouth.
15. Wearing of N95 or at least 3-Ply masks and suitable head caps, protective eye wear, and/or face shield is recommended.
16. Treatment of a periodontal patient:
    a. Phase 0: This phase called as the "emergency phase" involves procedures that require immediate attention by the clinician. Thus, patients having pain due to exposure of pulp, abscess, or extraction cases should be treated (in case of periodontal abscess) or referred to other departments (for root canal treatment or extraction accordingly).
    b. Phase I: This phase is known as "nonsurgical phase" and involves scaling and/or root planning which involves usage of ultrasonic machines which produce aerosol. Since there has been enough evidence supporting the spread of COVID-19 infection through aerosol production, patients having mild and moderate form of gingivitis and periodontitis should be avoided or minimized for ultrasonic scaling. Scaling should be done in cases of severe form of gingivitis or periodontitis with the use of saliva ejectors with low or high volume reducing the production of droplets and aerosols.
    c. Phase II: This phase is also known as "surgical phase" and deals with all the surgeries of periodontium (gingiva and alveolar bone). Periodontal emergencies such as pericoronitis or in some cases pregnancy tumor can occur and exacerbate in a short period and therefore need immediate treatment with continuous use of high-volume saliva ejectors. The other surgical procedures such as flap surgeries, all mucogingival surgeries, perio plastic surgeries, and dental implant surgeries should be postponed for further visits.
    d. Phase III: This phase is known as "restorative phase" and deals with interdisciplinary approach of restoring carious and missing tooth in patients after undergoing oral prophylaxis. Patients falling under this category need not to be treated immediately and can be given appointments for the same.
    e. Phase IV – Supportive phase: Oral hygiene instructions and diet modifications should be given to patients explaining their im-

portance and the same should be recalled for subsequent visits accordingly. Patients belonging to vulnerability group, i.e., old age patients, smokers, and diabetic patients, should be counseled more about this disease and educated to lay more emphasis of improving their periodontal as well as overall health. Smokers should be motivated more for smoking cessation programmers.

18. All masks, gloves, head caps to be disposed as per waste management protocol.
19. Fumigation of clinicals periodically is recommended.
20. Patients could be treated in an isolated and well-ventilated room or negatively pressured rooms if available for suspected cases with COVID-19.
21. In areas where COVID-19 spreads, nonemergency dental practices should be postponed.[56]

It was reported that dental practice should be postponed at least 1 month for convalescing patients with SARS. It is unknown yet whether the same suggestion should be recommended for patients with COVID-19. Since its outbreak, in most cities of the mainland of China, only dental emergency cases have been treated when strict implementation of infection prevention and control measures is recommended. Routine dental practices have been suspended until further notification according to the situation of epidemics.[57]

Conclusion

The virus responsible for coronavirus disease 2019 (COVID-19) was recently identified in saliva of infected patients. Saliva may play an important role in the transfer from human to human. Periodontists and other healthcare professionals who conduct aerosol-generating procedures can, unknowingly, provide direct treatment for patients with COVID-19 contaminated but not yet diagnosed, or those considered to be surveillance suspects. Inhalation of airborne particles and aerosols produced during periodontal procedures on patients with COVID-19 can be a high-risk procedure in which periodontists and the dental assistants are directly and closely exposed to this virus. Therefore, it is crucial for practitioner to refine preventive strategies to avoid COVID-19 by focusing on patient placement, hand hygiene and all personal protective equipment. Since there is currently no vaccine or specific antiviral treatment, ongoing rigorous research is giving us a ray of hope and paving the way for development of a vaccine in near future. Until we receive any good news in the form of drug or vaccine discovery, efforts should be made to manage symptoms and supportive therapy, including hand washing; using an alcohol-based hand sanitizer with at least 60 percent volume alcohol when soap and water are not readily available when appropriate; avoiding touching of the nose, eyes or mouth without washing hands; coughing/sneezing into a tissue and throwing the tissue directly into a dustbin; and (for those who may already have the infection) wearing a surgical mask in public. The guidelines when treating a patient in periodontal setup are highly recommended, so safety of clinician as well as patient is maintained and the infection is not spread further.

References

1. Phelan [1] AL, Katz R, Gostin LO. The novel coronavirus originating in Wuhan, China: challenges for global health governance. JAMA 2020; 323(8): 709-10. [http://dx.doi.org/10.1001/jama.2020.1097] [PMID: 31999307].
2. Mahase E. China coronavirus: WHO declares international emergency as death toll exceeds 200. BMJ 2020; 368: m408. [http://dx.doi.org/10.1136/bmj.m408] [PMID: 32005727].
3. Nemati M., Ebrahimi B., Nemati F.: Assessment of Iranian Nurses' Knowledge and Anxiety Toward COVID-19 During the Current Outbreak in Iran. Arch Clin Infect Dis 2020. In Press (In Press):e102848.
4. Chen N., Zhou M., Dong X., Qu J., Gong F., Han Y., Qiu Y., Wang J., Liu Y., Wei Y., Xia J., Yu T., Zhang X., Zhang.: Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. Lancet. 2020;395:507-13.
5. Modi P.D., Nair G., Uppe A., Modi J., Tuppekar B., Gharpure A.S, Langade G.: Awareness AmongHealthcare Students and Professionals in Mumbai Metropolitan Region: A Questionnaire-Based Survey. Cureus. 2020;12: e7514. DOI 10.7759/cureus.7514.
6. Centers for Disease Control and Prevention, Interim Infection Prevention and Control Guidance for Dental Settings during the COVID-19 Response, Centers for Disease Control and Prevention, Atlanta, GA, USA, 2019, https://www.cdc.gov/coronavirus/2019-ncov/hcp/dental-settings.html.
7. AL-Bayaty FH. Impact of COVID-19 on periodontal therapy: An overview. Journal of Oral Research. 2020 Jul 17:10-3.
8. Singh M. Novel coronavirus infection and periodontal clinic: Are we prepared?. International Journal of Academic Medicine. 2020 Apr 1;6(2):56.
9. Micik RE, Miller RL, Leong AC. Studies on dental

aerobiology:III. Efficacy of surgical masks in protecting dental personnel from airborne bacterial particles. J Dent Res.1971;50:626-30.

10. L. Meng, F. Hua, and Z. Bian, "Coronavirus disease 2019 (COVID-19): emerging and future challenges for dental and oral Medicine," Journal of Dental Research, vol. 99, no. 5, pp. 481–487, 2020.
11. Z. Chen, J. Fu, Q. Shu et al., "Diagnosis and treatment recommendations for pediatric respiratory infection caused by the 2019 novel coronavirus," World Journal of Pediatrics, pp. 1–7, 2020.
12. N. Zhu, D. Zhang, W. Wang et al., "A novel coronavirus from patients with pneumonia in China, 2019," New England Journal of Medicine, vol. 382, no. 8, pp. 727–733, 2020.
13. Backer JA, Klinkenberg D, Wallinga J. Incubation period of 2019 novel coronavirus (2019-nCoV) infections among travellers from Wuhan, China, 20-28 January 2020. Euro Surveill 2020; 25(5)2000062.
14. C. Huang, Y. Wang, X. Li et al., "Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China," &e Lancet, vol. 395, no. 10223, pp. 497–506, 2020.
15. J. F.-W. Chan, S. Yuan, K.-H. Kok et al., "A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster," &e Lancet, vol. 395, no. 10223, pp. 514–523, 2020.
16. F. Bennardo, C. Buffone, and A. Giudice, "New therapeutic opportunities for COVID-19 patients with Tocilizumab: possible correlation of interleukin-6 receptor inhibitors with osteonecrosis of the jaws," Oral Oncology, Article ID 104659,2020.
17. Lu CW, Liu XF, Jia ZF. 2019 nCoV transmission through the ocular surface must not be ignored. Lancet 2020;395:e39.
18. van Doremalen N, Bushmaker T, Morris DH, Holbrook MG, Gamble A, Williamson BN, et al. Aerosol and surface stability of SARS CoV 2 as compared with SARS CoV 1. N Engl J Med 2020;382:1564 7.
19. Peng X, Xu X, Li Y, Cheng L, Zhou X, Ren B. Transmission routes of 2019 nCoV and controls in dental practice. Int J Oral Sci 2020;12:9.
20. Barabari P, Moharamzadeh K. Novel Coronavirus (COVID-19) and Dentistry–A Comprehensive Review of Literature. Dentistry Journal. 2020 Jun;8(2):53.
21. Bruning AHL, Leeflang MMG, Vos JMBW, Spijker R, de Jong MD, Wolthers KC, et al. Rapid Tests for Influenza, Respiratory Syncytial Virus, and Other Respiratory Viruses: A Systematic Review and Meta-analysis. Clin Infect Dis [Internet]. 2017 Sep 15 [cited 2020 Apr 1];65(6):1026–32. Available from: http://academic.oup.com/cid/article/65/6/1026/3829590/Rapid-Tests-for-Influenza-Respiratory-Syncytial.
22. WHO | Laboratory Biosafety Manual—Third Edition. Available online: https://www.who.int/csr/resources/publications/biosafety/WHO_CDS_CSR_LYO_2004_11/en/ (accessed on 9 April 2020).
23. Briefing: Coronavirus (COVID-19) Testing | ABPI. Available online: https://www.abpi.org.uk/medicinediscovery/covid-19/briefing-coronavirus-covid-19-testing/ (accessed on 9 April 2020).
24. COVID-19 serology tests and the role of the dental office, available online: https://www.perioimplantadvisory.com/clinical-tips/best practice/article/14174339/covid19-serology-tests-and-the-role-of-the-dental-office
25. Hahn SM. Coronavirus (COVID-19) update: serological tests. US Food and Drug Administration. April 7, 2020. https://www.fda.gov/news-events/press announcements/coronavirus-covid-19-update-serological-tests
26. Rapid COVID-19 antibody detection tests: principles and methods. Antibody Genie. https://www.assaygenie.com/rapid-covid19-antibody-detection-tests-principles-and-methods
27. Lee, E.Y.P.; Ng, M.Y.; Khong, P.L. COVID-19 pneumonia: What has CT taught us? Lancet Infect. Dis. 2020, 20,384–385.
28. Ai, T.; Yang, Z.; Hou, H.; Zhan, C.; Chen, C.; Lv, W.; Tao, Q.; Sun, Z.; Xia, L. Correlation of chest CT and RT-PCR Testing in Coronavirus Disease 2019 (COVID-19) in China: A report of 1014 cases. Radiology 2020,200642.
29. ACR Recommendations for the use of Chest Radiography and Computed Tomography (CT) for Suspected COVID-19 Infection. Available online: https://www.acr.org/Advocacy-and-Economics/ACR-Position Statements/Recommendations-for-Chest-Radiography-and-CT-for Suspected - COVID19-Infection (accessed on 5 May 2020).
30. Bose S, Adapa S, Aeddula NR, Roy S, Nandikanti D, Vupadhyayula PM, Naramala S, Gayam V, Muppidi V, Konala VM. Medical Management of COVID-19: Evidence and Experience. Journal of Clinical Medicine Research. 2020 Jun 4;12(6):329-43.
31. Asada M, Yoshida M, Suzuki T, Hatachi Y, Sasaki T, Yasuda H, Nakayama K. et al. Macrolide antibiotics inhibit respiratory syncytial virus infection in human airway epithelial cells. Antiviral Res. 2009;83(2):191–200. doi: 10.1016/j.antiviral.2009.05.003.
32. Sanders JM, Monogue ML, Jodlowski TZ, Cutrell JB. Pharmacologic treatments for coronavirus disease 2019 (COVID-19): A Review. JAMA. 2020 doi: 10.1001/jama.2020.6019.
33. Tanaka T, Narazaki M, Kishimoto T. Immunotherapeutic implications of IL-6 blockade for cytokine storm. Immunotherapy. 2016;8(8):959–970. doi: 10.2217/imt-2016-0020.
34. Channappanavar R, Fehr AR, Zheng J, Wohlford-Lenane C, Abrahante JE, Mack M, Sompallae R. et al. IFN-I response timing relative to virus replication determines MERS coronavirus infection outcomes. J Clin Invest. 2019;130:3625–3639. doi: 10.1172/JCI126363.
35. Luke TC, Kilbane EM, Jackson JL, Hoffman SL. Meta-analysis: convalescent blood products for Spanish influenza pneumonia: a future H5N1 treatment? Ann Intern Med. 2006;145(8):599–609. doi: 10.7326/0003-4819-145-8-200610170-00139.
36. Shen C, Wang Z, Zhao F, Yang Y, Li J, Yuan J, Wang F. et al. Treatment of 5 critically ill patients with COVID-19 with convalescent plasma. JAMA. 2020 doi: 10.1001/jama.2020.4783.
37. Chen L, Xiong J, Bao L, Shi Y. Convalescent plasma as a potential therapy for COVID-19. Lancet Infect Dis. 2020;20(4):398–400. doi: 10.1016/S1473-3099(20)30141-9.
38. Xu X, Han M, Li T, Sun W, Wang D, Fu B, Zhou Y. et al. Effective treatment of severe COVID-19 patients with tocilizumab. Proc Natl Acad Sci U S A. 2020;117(20):10970–10975. doi: 10.1073/pnas.2005615117.
39. Busund R, Koukline V, Utrobin U, Nedashkovsky E. Plasmapheresis in severe sepsis and septic shock: a prospective, randomised, controlled trial. Intensive Care Med. 2002;28(10):1434–1439. doi: 10.1007/s00134-002-1410-7.
40. Monard C, Rimmele T, Ronco C. Extracorporeal blood purification therapies for sepsis. Blood Purif. 2019;47(Suppl 3):1–14. doi: 10.1159/000499520.
41. Ma J, Xia P, Zhou Y, Liu Z, Zhou X, Wang J, Li T. et al. Potential effect of blood purification therapy in reducing cytokine storm as a late complication of critically ill COVID-19. Clin Immunol. 2020;214:108408. doi: 10.1016/j.clim.2020.108408.
42. Lu DP, Zambito RF. Aerosols and cross infection in dental practice-a historic view. Gen Dent.1981;29:136-43.
43. Harrel S, Barnes J, Rivera-Hidalgo F. Aerosol and spatter contamination from the operative site during ultrasonic scaling. J Am Dent Assoc.1998;129:1241-9.

44. ECDC - European Centre for Disease Prevention and Control. European surveillance for human infection with novel coronavirus (COVID-19). Available at: https://www.ecdc. europa.eu/en/european-surveillance-humaninfection-novelcoronavirus- COVID-19.
45. World Health Organization-WHO. Global surveillance for human infection with novel coronavirus (COVID-19) Interim guidance. Available at: https://www.who.int/docs/defaultsource/ coronaviruse/2020 0121-globalsur veillance-for- COVID-19.pdf
46. Xie X, Li Y, Sun H, Liu L. Exhaled droplets due to talking and coughing. J R Soc Interface 2009;6(Suppl 6):S703–S714.
47. World Health Organization. Clinical Management of Severe Acute Respiratory Infection when Novel Coronavirus (2019-nCoV) Infection is Suspected: Interim guidance 2020. Available from: https://www.who.int/publications-det ail/clinical-management-of-severe-acute-respira tory-infection-when-novel coron avirus-(ncov)-infection-is-suspected. [Last retrieved on 2020 Apr 05].
48. Dental Council of India. Precautionary and Preventive Measures to Prevent Spreading of Novel Coronavirus (Covid-19) 2020. Available from: http://www.dci.gov.in. [Last retrieved on 2020 Apr 01].
49. Yoon JG, Yoon J, Song JY, et al. Clinical significance of a high SARS-CoV-2 viral load in the saliva. J Korean Med Sci 2020;35:e195.7
50. Baker N, Williams AJ, Tropsha A, et al. Repurposing quaternary ammo-nium compounds as potential treatments for COVID-19. Pharm Res2020;37:104.
51. Eggers M, Koburger-Janssen T, Eickmann M, et al. In vitro bacterici-dal and virucidal efficacy of povidone-iodine gargle/mouthwash againstrespiratory and oral tract pathogens. Infect Dis Ther 2018;7:249–59.
52. Mady LJ, Kubik MW, Baddour K, et al. Consideration of povidone-iodineas a public health intervention for COVID-19: utilization as "PersonalProtective Equipment" for frontline providers exposed in high-risk headand neck and skull base oncology care. Oral Oncol 2020;105:104724.
53. Peng X, Xu X, Li Y, et al. Transmission routes of 2019-nCoV and controlsin dental practice. Int J Oral Sci 2020;12:9.
54. American Dental Association. ADA interim guidance for minimiz-ing risk of COVID-19 transmission; 2020. Available from URL:https://www.kavo.com/en-us/resource-center/ada-interim-guidance-minimizing-risk-covid-19-transmission [last accessed 13.08.20].
55. A.Vergara-Buenaventura, C. Castro-Ruiz.Use of mouthwashes against COVID-19 in dentistry:British Journal of Oral and Maxillofacial Surgery 58 (2020) 924–927.
56. Samaranayake LP, Peiris M. Severe acute respiratory syndrome and dentistry: A retrospective view. J Am Dent Assoc 2004;135:1292-302.
57. Li ZY, Meng LY. The prevention and control of a new coronavirus infection in department of stomatology. Zhonghua Kou Qiang Yi Xue Za Zhi 2020;55:E001.

JSPIK

Indices for Assessment of Calculus

Devi Balakrishnan[1], Rahul Bhandary[2], Amitha Ramesh[3], Aparna C Murali[4]

ABSTRACT

Dental indices are used to determine an individual's and oral health, as well as disease status. Several scholars have proposed various indices for the evaluation of plaque and debris, for calculus, gingival inflammation, gingival bleeding, for the evaluation and treatment of periodontitis and for the measurement of bone level. This article includes brief discussion about some of the indices used for assessment of calculus such as calculus surface index and its modification, calculus component in Oral Hygiene Index and Periodontal Disease Index, Marginal line calculus index, Standardized coil technique and Probe method which can be practiced by dentists.

Keywords: Calculus Surface Index (CSI), Calculus Surface Severity Index (CSSI), Periodontal Disease Index (PDI), Oral Hygiene Index-Simplified (OHI-S).

Introduction

Dental indices are used to determine an individual's systemic and oral health, as well as the disease status. This additionally motivates the person to monitor and prevent the disease in preventive and clinical treatment. According to Russell A.L, an Index is defined as a numerical value describing the relative status of a population on a graduated scale with definite upper and lower limits, which is designed to permit and facilitate comparison with other populations classified by the same criteria and methods.[1] As far as periodontics is concerned, several scholars have proposed various indices for the evaluation of plaque and debris, for calculus, gingival inflammation, gingival bleeding, for the evaluation and treatment of periodontitis and for bone level calculation.[2] Dental calculus is a common factor in the development of periodontitis and requires careful assessment in most cases. There are several indices proposed for calculus measurement which can be used for patient monitoring and recording of the hygiene status. This article discusses some of the indices which were introduced in dentistry, for the evaluation of calculus.

Calculus Surface Index

Calculus Surface Index (CSI) is used to calculate the presence of the calculus on the four mandibular incisors (supra and subgingival).[3] The examined surfaces are the labial, lingual, mesial, and distal, according to the number of surfaces on which calculus is registered, each tooth is assigned number from 0 to 4.

The CSI is the total number of surfaces covered by definition (max. 16).

The CSI score is the total number of surfaces covered by calculus.

There is an extension of this index known as the Calculus Surface Severeity Index (CSSI), where the CSSI is a measure from 0 to 3, where 0 is no calculus present to 3, where the calculus thickness and width

[1,4] Post graduate student, [2,3] Professor, Department of Periodontics, A B Shetty Memorial Institute of Dental Sciences, NITTE Deemed to be University, Mangaluru - 575018, Karnataka Corresponding author: Prof (Dr). Rahul Bhandary, Email: bhandaryrahul@gmail.com

are quantified as shown below.

0 = no calculus present.

1 = calculus observable but less than 0.5 mm in width and/or thickness.

2 = calculus not exceeding 1 mm in width and/or thickness.

3 = calculus exceeding 1 mm in width and or thickness.

Calculus Component of Periodontal Disease Index

The Periodontal Disease Index was Introduced by Sigurd Ramfjord which is a tooth- and site-specific measurement tool. In contrast to other indices, the Periodontal Disease Index used a periodontal probe with marks at 3, 6 and 8 mm, to measure the distance from the cemento–enamel junction to the bottom of the pocket (i.e. attachment loss).[4,5]

Ramfjord recommended the assessment of six 'index teeth' that soon became known as the 'Ramfjord teeth' These teeth were:

Maxillary right first molar (tooth 16),

Maxillary left central incisor (tooth 11),

Maxillary left first bicuspid (tooth 24),

Mandibular left first molar (tooth 36),

Mandibular right central incisor (tooth 41) and

Mandibular right first bicuspid (tooth 44).

Originally, the Periodontal Disease Index required measurements to be made on the mesial, buccal, distal and lingual sides, but later it was modified to include just two sites, mesial and mid-buccal. Validation research on the 'Ramfjord teeth' as representing the entire mouth showed mixed results.

Fig 1: calculus formation

Marginal Line Calculus Index

The index is similar to the Volpe and Manhold series. Solelysupragingival calculus is performed on the lingual surfaces on the marginal gingiva of the lingual surfaces of the lower incisors.

An abstract, axial plane bisects each tooth vertically into a mesial and distal section. The percentage of the enamel surface covered by the deposits of calculus is then reported using only 0, 12.5, percentages. The calculus index value for the marginal line per tooth is calculated by comparing the two half units per tooth. For each subject the MLC score is extracted from a sum of the ratings, separated by the number of teeth examined.[6]

Calculus Component of OHI-S

The Oral Hygiene Index consists of the combined "Debris Index" and "Calculus Index." Each of these indexes, in turn, relies on 12 numerical determi-

Table 1. Different Indices used for the Assessment of Calculus[2]

SL NO	INDEX	AUTHOR	YEAR
1.	Calculus surface index	Ennever. J, Sturzenberger Radike A.W	1961
2.	Calculus surface severity index	Ennever. J	1961
3.	Calculus component of periodontal disease index	Sigurd PRamjford	1959
4.	Standardized coil technique	Marthaler, Shroeder Muhlemann	1957
5.	Calculus component of OHI-S	John. C. Green Jack. R. Vermillion	1964
6.	Marginal line calculus index	Muhlemann H.R, Villa. P	1967
7.	Probe method of calculus assessment	Volpe	1967

nations representing the amounts of debris or calculus found on the buccal and lingual surfaces of every three segments of each dental arch:

(1) the segment distal to the right cuspid,
(2) the segment distal to the left cuspid and
(3) the segment mesial to the right and left first bicuspids.

In deliberation of the differences in the individual indices, different measurements made for the buccal and lingual surfaces are extracted from grades dependent on the fraction of the tooth surface covered by debris or calculus.

Just because there are enough disparities in the height of permanent and deciduous teeth clinical crowns, only fully erupted permanent teeth are taken for scoring. A tooth is considered to have erupted completely when the occlusal or incisal surface reaches the plane of occlusion.

Third molars and incompletely erupted teeth are not graded because of the large differences in oral hygiene status of the clinical crown heights that can occur between the surfaces.[7]

0	No calculus present
1	Supra gingival calculus covering not more than third of the exposed tooth surface.
2	Supra gingival calculus covering more than one third but not more than two thirds of the exposed tooth surface or the presence of individual flecks of sub gingival calculus around the cervical portion of the tooth or both.
3	Supra gingival calculus covering more than two third of the exposed tooth surface or a continuos heavy band of sub gingival calculus around the cervical portion of the tooth or both.

Standardized Foil Technique (Marthaler, Schroederan, D Muhlemann)

This technique used thin, rounded, triangular foils which are punched from sandblasted polyester sheets. The foils are perforated and a nylon thread is attached to the lingual surfaces of the lower central incisors. The quantity of deposits on the contoured strips can be measured by calculating the strip carefully before insertion and after removal from mouth after insertion.[8]

Probe Method of Calculus Assessment

In 1962, Volpe proposed a simplified approach for calculus scoring, using a periodontal probe graduated in millimeters and colored for precision on one end. The technique is used for the lower anterior lingual surface calculus calculation by inserting the probe into the inferior most region of the calculus bisecting with tooth surfaces and records the calculus heights in millimetres or diagonally through the point of greatest height of calculus formation.[9]

Discussion

Dental indices or indexes can be regarded as the key instrument in dental disease epidemiological studies to assess the occurrence, prevalence and seriousness of diseases on the basis of which preventive measures are implemented to monitor and prevent them. Epidemiological indices are the attempt to measure health problems on a graded scale, making comparisons between populations easier. Any system of classification or ranking is executed so that the information can be easily and accurately recorded and can be effectively shared among professionals around the world. In addition to being cost effective, an index should meet most of the ideal criteria such as transparency, objectivity, sensitivity, acceptability and aptitude for statistical evaluation.

Many authors have used Simplified Oral hygiene Index for the evaluation of calculus and debris in their studies because of the simplicity and less time consumption and it is considered as the most reliable index and can be used in different circumstances.[10,11] Rams et al in their study concluded that partial-mouth exams with sufficient adjustment of Ramfjord index teeth data may be useful in assessing the development of periodontal disease in longitudinal population analysis of human periodontitis.[12] Different indices have been used by authors for the measurement of calculus in their studies according to their convenience and criteria.

Conclusion

There are many indexes which are introduced, for the evaluation of calculus, out of this some have been discussed in this article very shortly, and can be

applied in dental practice by practitioners. The index will continue to be a resource that is relevant and important to the dentist. As per Periodontics considered, Calculus is one of the etiologic agent for periodontal inflammation, so these indices help physicians for the better understanding of the disease and treatment plan.

References

1. Park's Textbook of Preventive and Social Medicine. - K. Park - 16th ed. Banarsidas Bhanot Publishers.
2. Prasad Kulkarni, Dhirendra Kumar Singh, M D Jalaluddin, IpsitaJayanti. Indices in Dentistry: Recitation of Oral Diseases at Numerical Value.J Res Adv Dent 2016;5:2:261-8.
3. J. Ennever, O.P Sturzenberger, A.W Radhike. The Calculus Surface Index Method for scoring clinical calculus studies. Journal of periodontology 32 (1) 54,1961.
4. Ramfjord, S. P.: Indices for prevalence and incidence of periodontal disease, JPedodontol, 1959; 30:51-9.
5. Ramfjord, S. P.: The Periodontal Disease Index (PDI), J Periodental, 1967;38:602-10.
6. MuhlemannH.R, et al. The marginal line calculus index. HelvOdontolActa. 1967; Oct;11(2):175-9.
7. Greene, J. C., Vermillion, J. R. The simplified oral hygiene index. Journal of American Dental Association 1964;68:25–31.
8. Schroeder HE, Marthaler TM, Muhlemann HR. Effects of some potential inhibitors on early calculus formation. HelvOdontActa. 1962 Apr; 6:6-9.
9. Volpe, Manhold J.H, HazenSP.In vivo calculus assessment. i. a method and its examiner reproducibility.J PeriodontolJul-Aug 1965;36:292-8.
10. Bakhtiar K, Gharouni K, Gharouni B, Bastami F, Almasian M, Hosseintalai M. DMFT and OHIS Indexs in the Pregnant Mothers: An Explanation Based on the Health Belief Model. JCHR. 2018; 7 (1):1-10.
11. Ammajan RR, Joseph R, Rajeev R, Choudhary K, Vidhyadharan K. Assessment of periodontal changes in patients undergoing radiotherapy for head and neck malignancy: a hospital-based study. Journal of Cancer Research and Therapeutics. 2013 Oct 1;9(4):630.
12. Rams TE, Oler J, Listgarten MA and Slots J: Utility of Ramfjord index teeth to assess periodontal disease progression in longitudinal studies. J ClinPeriodontol 1993; 20: 147-50.
13. Marthaler, T.M, Schroeder, H.E and Muhlemann,H.R. A method for the quantitative assessment of plaque and calculus formation. HelvOdont Acta5:39,1962.

JSPIK

Piezosurgery and its Role in Periodontics - Review

Najia Nazir[1], Anil Melath[2], Mohammed Feroz[3], Subair K[3], Varun Murali[4]

ABSTRACT

Ultrasonic instruments for periodontal surgery represent a novel alternative technique to traditional instruments used for hard tissue surgery. Piezosurgery, based on peizo-electric effect, produces ultrasonic microvibrations to selectively resect bone without injuring adjacent soft tissues. The piezosurgery device consists of ultrasonic transducer and a wide range of inserts for various periodontal surgical procedures. The exceptional level of precision and minimal trauma has made this technique highly acceptable in the field of medicine as well as dentistry. This review article gives an insight on the mechanism of action and biologic effects of the piezosurgery technique, additionally emphasizing on its advantages and limitations over conventional hard tissue surgical techniques.

Keywords: piezosurgery, periodontal surgical techniques, implantology

Introduction

Ultrasonic instruments were solely used for scaling procedures. Initial attempts to use ultrasonic vibrations for tissue cutting resulted good results but were not effective enough to resect mineralized bone. Moreover, repeated cuts of bone caused an increase in temperature leading to necrosis of bone.

Piezosurgery is a technique which has garnered popularity in the recent past as a novel power driven instrument to perform periodontal surgery. 'Piezo' is a Greek word meaning 'pressure, to press tight' and piezosurgery is based on 'piezo-effect' which is the effect of changing of electrical field on crystalline structure resulting in ultrasonic vibrations.[1] Piezosurgery has received a wide acceptance in the medical and dental profession in a short span of time due to its wide application and myriad benefits.

History

The French physicists Jaques and Pierre Curie, in 1880s, discovered that a measurable electric loading is induced by the application of mechanical pressure and power to crystalline body surfaces. Gabriel Lippmann, in 1881, found the converse piezoelectric effect forming the basis of piezosurgery. Catuna, in 1953, developed an ultrasonic drill for cavity preparation on human teeth and an article on the effects of ultrasound on dental hard tissues was published.Richman, in 1957, was the first to report the surgical use of an ultrasonic chisel to remove bone and resect roots in apicoectomies. In 1960, evidence of an ultrasonic scalpel-like blade to directly resect osseous tissue was reported by Mazzarow. Mcfall et al., in 1961, found slow healing with no severe complication by use of oscillating scalpel blades. In 1980, Horton et al. stated that bone regeneration was better using ultrasonic device and observed easy and efficientmineralized tissue removal along with good acceptance by patients without any complications.[2]

In 1999, Dr. Thomas Vercelloti invented piezo-

[1]Postgraduate student, [2]Professor & HOD, [3]Professor, [4]Senior Lecturer, Department of Periodontics, Mahe Institute of Dental Sciences and Hospital,Chalakkara, Palloor, Mahe - 673310. Corresponding Author: Dr Najia Nazir, Email: najiajishin@gmail.com

electric bone surgery in collaboration with Mectron Spa and published the first article introducing piezoelectric bone surgery in the International Journal of Periodontics and Restorative Dentistry. This publication reported a case of ridge expansion, in which, due to the extreme thinness and mineralization of the edentulous crest, osteotomy would not have been possible with any other surgical instrument.[3] In 2005, the US Food and Drug Administration approved the use of ultrasonics in dentistry, including bone surgery.

Mechanism of action

Oscillations are produced by passage of electric current across certain ceramics and crystals.[4] A polarized piezoceramic expands in the direction of and contract perpendicular to polarity on application of current across it. This sets up microvibrations in the transducer, creating an ultrasound output, which is transmitted to the handpiece insert. A longitudinal movement occurs in the insert which can cut osseous tissues by microscopic shattering.

Cavitation is a phenomenon that occurs in all acoustic transitions occurring in liquid or solid-liquid interface vibrating at a certain intermediate frequency. It is as a result of rapid rupture of molecular cohesion in liquid along with appearance of vapour filled depressions until it is ready to implode. This creates a cooling effect on tissues, as well as an effective debridement and increased visibility of operating field is achieved.

The active element of most acoustic transducers used today is a piezoelectric ceramic,like lead zirconate titanate compositions, which can be cut in various ways to produce different wave modes. Piezo-polymers and composites are some of the newer materials being used in some applications.

Figure 1: Piezosurgery unit

Parts of the Piezosurgery Device

The Piezosurgery® unit is consists of the main body, activated with a pedal, a hand-piece, andfeatures a number of inserts with different shapes depending on the surgical need.

Main Body

The main body consist of a display, an electronic touchpad, a peristaltic pump, one stand for the handle and another to carry the bag containing irrigation fluid. The interactive touchpad has four keys that enable to select the feature mode, the specific program, and the flow of cooling fluid.

There are two primary operating modes: BONE mode and ROOT mode.

ROOT Mode

The following characteristics are seen in ROOT Mode:

- Average ultrasonic power without frequency over-modulation
- Two different programs
    - ENDO Program: A limited level of power creates oscillation of a few microns, optimal for washing out the apical part of the root canal in endodontic surgery.
    - PERIO Program: An intermediate level of power between the ENDO program and the BONE program. The ultrasonic wave is transmitted through the transducer incontinuous sinusoidal manner characterized by a frequency equal to the resonance frequency of the insert used.

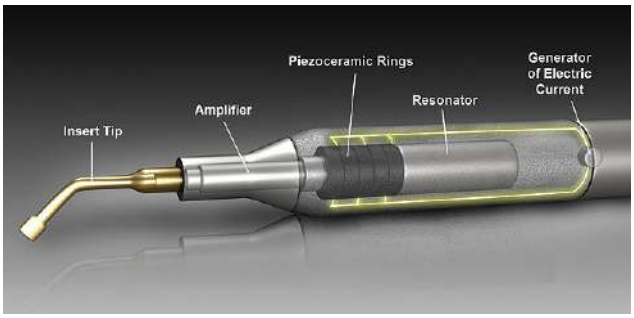


Figure 2: Parts of Piezosurgery Handpiece

BONE Mode

The following characteristics are seen in BONE Mode:

- Extremely high ultrasonic power compared to Root mode.
- Frequency over-modulation provides unique cutting of different kinds of bone. The recommended selection is:
  - Quality 1: for cutting the cortical bone or for high-density spongy bone.
  - Quality 2: for cutting low-density spongy bone
- SPECIAL Program was designed with a standard power level slightly lower than the BONE programs and it is characterized by the same frequency over-modulation. The SPECIAL program is dedicated to effective thin and delicate cut.

Handle

Ceramic plates are subjected to an electrical field produced by an external generator to generate ultrasonic vibrations. These are channelled into the amplifier, which transmits them to the sharp end of the handle. The insert is tightened with a special keyin this manner; the highest degree of efficiency is obtained for the cut and duration of the insert.

Inserts

The inserts have been organized according to a dual classification system, taking into consideration morphological-functional and clinical factors.

- Morphological-functional classification: The morphological description defines the structural properties of the insert, while the functional description outlines its cutting characteristics:
- Sharp - Cutting
- Diamond-coated - Abrasive
- Rounded - Smoothing

Clinical classification: The clinical classification sorts the inserts (sharp, abrasive, smoothing) according to surgical technique:

- Osteotomy (OT): OT1, OT2, OT3, OT4, OT5, OT6, OT7, OT7S4, OT7S3, OT8R/L
- Osteoplasty (OP): OP1, OP2, OP3, OP4, OP5, OP6, OP7
- Extraction (EX): EX1, EX2, EX3
- Implant site preparation (IM): IM1 (OP5), IM2A, IM2P, OT 4, IM3A, IM3P
- Periodontal Surgery: PS2, OP5, OP3, OP3A, PP1
- Endodontic Surgery: OP3, PS2, EN1, EN2, OP7
- Sinus Lift: OP3, OT1 (OP5), EL1, EL2, EL3
- Ridge Expansion: OT7, OT7S4, OP5 (IM1), IM2, OT 4, IM3
- Bone Grafting: OT7, OT7S4, OP1, OP5
- Orthodontic Microsurgery: OT7S4, OT7S3

Biological Effects on Bone Cut

It is imperative to understand the effect of mechanical instruments on bone and the viability of cell for effective regeneration of the periodontium. Several studies have been conducted for the same.

A study conducted by Happe[7] has shown that bone grafts with a clean, precise and smooth cut with good healing property could be harvested using piezosurgery.Sohn et al[8] reported an effective osteotomy with minimal trauma by using piezosurgery system, in contrast to conventional surgical burs or saws.

Immunohistochemical staining and polymerase chain reaction analysis revealed that the cell viability and differentiation of autogenous bone chips from intro-oral sites using piezosurgery was as good as that obtained from conventional rotating devices.[9]

Histomorphological analyses of implant sites drilled using piezosurgery demonstrated more inflammatory cells compared to traditional drills. Besides, neo-osteogenesis was steadily more active in bone samples from the implant sites that were prepared using piezoelectric bone surgery. Additionally, the peri-implant hard tissue treated with the piezoelectric surgery technique exhibited an earlier increase in BMP-4 and TGF-beta2 proteins limiting the pro-inflammatory cytokines.[10]

Applications of Piezosurgery

Dental plaque and strongly bound calculus can be effectively removed from tooth surfaces by cavitational activity of the ultrasonic oscillating tip.[11] The piezosurgery device can be used for soft-tissue debridement to remove the secondary flap after incision through

retained periosteum by changing to a thin, tapered tip and altering the power setting.[12]

Crown lengthening procedure by lifting a full-thickness flap, pericoronal ostectomy, and making placement more apical to the gingival margin with OP_3 inserts is effective with no risk of damaging the root surface. High degree of precision and speed is ensured in removal of bony spikes and root smoothening after ostectomy using piezosurgery.[3]

Piezosurgery has added advantages in implant dentistry for obtaining bone grafts, splitting of crestal bone and sinus lift procedures.

Assessment of size and quality of bone grafts, complications at the donor and recipient sites, integration of the grafts, bone quality, resorption of the grafts, and ability to place implants revealed significant positive results. A precise, clean, and smooth cutting with excellent visibility and a mean graft size of 1.15 cm3, with a maximum of 2.4 cm3 was obtained. 71% of the obtained grafts were cortical and the donor sites healed uneventfully.[7]

Figure 3: Various inserts used for Piezosurgery

The alveolar ridge is split to avoid autologous bone transplants in bone with satisfactory density. Blus and Szmukler-Moncler[13] did split-crest procedures using piezoelectrical surgery on 57 patients to place 230 implants over a period of three and a half years. A survival rate of 96% was achieved after 2 months of loading. Compared to classic procedures, it was less time-consuming and the technique was easy to learn.

Elevation of the sinus floor is usually most effective to treat bone loss in the posterior maxilla. Through a crestal or lateral approach, the Schneiderian membrane is raised without perforation, and the space between the bone and membrane filled by the new graft. A more conservative and controlled osseous incision is achieved using piezosurgery compared to conventional ostectomy using diamond drills, as it reduces the risk of perforating the Schneiderian membrane and improves vision and hygiene of the operating area.[14]

Apart from these, piezosurgery has also been used extensively in endodontic procedures like root-end resection and root-end preparation.[15] It has also been proven to be effective in the field or oral and maxillofacial surgery for procedures like Le Fort I osteotomies, rhinoplasty, osteogenic distraction, ridge expansion, inferior alveolar nerve decompression, cyst removal, and impacted tooth extraction.[16]

Applications of piezosurgery in medicine include cranial osteoplasty, ENT surgery, neurosurgery, paediatric surgery, orthopaedics, and otologic surgeries due to rapid and easy intraoperative management, and precise cutting, particularly in critical anatomical areas.[1]

Advantages

- Highly precise and safe cutting of hard tissue is achieved without harming the adjacent soft tissue and nerve.
- A constant and optimal vibration frequency avoids excessive temperature.[1]
- Cavitation creates a clean surgical field increasing visibility with a cooling effect on the tissue.[11]
- Minimal intra-operative bleeding seen and rapid post-operative healing has been reported.[7]
- Possibility to harvest bone of different quality as

well modification of the bone to fit recipient site can be achieved.[4,7,9,10]

- Microvibrations produces less noise level as compared to a surgical saw or bar minimizing patient's psychological stress.[8]
- The risk of subcutaneous emphysema is reduced as compared to the effect of air-water spray generated by osteotomy with rotary instruments.[16]

Table 1 indicates the clinical advantages of using piezosurgery as compared to conventional techniques.

Limitations

- Adequate dexterity and gentle touch is required since an increase working pressure increases the heat.1
- Increased operative time compared to traditional cutting instruments.
- Rapid wearing of inserts may cause breakage leading to inadvertent tissue damage.
- Expensive compared to traditional instruments.

Table 1: The clinical advantages of using piezosurgery as compared to conventional techniques.[3]

Surgical techniques	Limits of traditional instruments	Advantages using piezosurgery
Periodontal flap surgery and debridement	• Manual skill required • Considerable bleeding present	• Easy removal of inflamed tissue • Cavitation provides haemostasis and maximum visibility
Ostectomy	• Using a bur can be traumatic to the bony tissue • Difficult to use in interproximal areas as it may damage root surfaces	• Minimal risk to root surfaces
Osteoplasty	• Highly imprecise and traumatic for bone and root. •Bone spikes are not less effectively removed	• Precise remodelling of the cortical bone without bleeding • Unwanted bone spikes can be removed effectively in close proximity of root
Ridge expansion procedure	• The width of the thinnest bur being 1.5 mm, there is a limited depth which is achieved. • Limitation in performing vertical osteotomy without damaging crestal bone	• Deep osteotomy cuts achieved by inserts of 0.06 mm and 0.40 mm OT7 and OT7S burs • Minimum degree of invasion is achieved
Bone grafting procedure	• Imprecise and complex bone fragment gathering • Macrovibrations by burs may result in losing about 1.5 mm of graft width • Cutting action is limited to the width of cortical bone • Cut of the internal spongy surfaces are uneven, torn using a chisel • Additional remodelling will be needed before placing it at recipient site which results in additional loss of graft thickness.	• Quick and precise collection of bone fragments • Deep cut on the internal surface achieved • Flat surface of spongy bone can be obtained • Osseous tissues are conserved • Fast postoperative recovery rate
Maxillary sinus lift procedure	• Burs and manual elevators may increase risk of damaging Schneiderian membrane	• Mineralized tissue can be cut without damaging the membrane and allows easy separation

Conclusion

Piezosurgery has proven to be a major breakthrough in the field of periodontics owing to its numerous benefits and wide range of application. Its physical and mechanical properties, including production of microvibrations, optimal frequency, precise cutting and efficient post-operative healing increases the rate of success of various surgical procedures in the field of periodontology, implantology and other oral surgical procedures.

Piezosurgery has the potential to redefine the concept of minimally invasive surgical technique with an increased focus on tissue engineering and bone regeneration.

References

1. Labanca, M., Azzola, F., Vinci, R., & Rodella, L. F. (2008). Piezoelectric surgery: Twenty years of use. British Journal of Oral and Maxillofacial Surgery, 46(4), 265–69.
2. Ballato A. Piezoelectricity:History anti new thrusts. IEEE Ultrasonics Symp 1996;1:575-83.
3. Varcellotti T. Essentials in Piezosurgery, Clinical advantages in Dentistry. 2009. Quitessenza Edizioni.
4. Leclercq, P., Zenati, C., Amr, S., & Dohan, D. M. (2008). Ultrasonic Bone Cut Part 1: State-of-the-Art Technologies and Common Applications. Journal of Oral and Maxillofacial Surgery, 66(1), 177–82.
5. Thomas M, Akula U, Ealla KK, Gajjada N. Piezosurgery: A Boon for Modern Periodontics. J Int Soc Prev Community Dent. 2017;7(1):1-7.
6. Nalbandian S. Piezosurgery techniques in implant dentistry. Aust Dent Pract. 2011:116–26.
7. Happe A. Use of a piezoelectric surgical device to harvest bone grafts from the mandibular ramus: report of 40 cases. Int J Periodontics Restorative Dent 2007;27:241–9.
8. Sohn DS, Ahn MR, Lee WH, Yeo DS, Lim SY. Piezoelectric osteotomy for intraoral harvesting of bone blocks. Int J Periodontics Restorative Dent 2007;27:127–31.
9. Chiriac G, Herten M, Schwarz F, Rothamel D, Becker J. Autogenous bone chips: influence of a new piezoelectric device (Piezosurgery) on chip morphology, cell viability and differentiation. J Clin Periodontol 2005;32:994–9.
10. Preti G, Martinasso G, Peirone B, et al. Cytokines and growth factors involved in the osseointegration of oral titanium implants positioned using piezoelectric bone surgery versus a drill technique: a pilot study in minipigs. J Periodontol 2007;78:716–22.
11. Walmsley AD, Laird WR, Williams AR. Dental plaque removal by cavitational activity during ultrasonic scaling. J Clin Periodontol. 1988; 15:539-43.
12. Seshan H, Konuganti K, Zope S. Piezosurgery in periodontology and oral implantology. J Indian Soc Periodontol. 2009;13(3):155-6.
13. Blus C, Szmukler-Moncler S. Split-crest and immediate implant placement with ultra-sonic bone surgery: a 3-year life-table analysis with 230 treated sites. Clin Oral Impl Res 2006;17:700–7.
14. Torella F, Pitarch J, Cabanes G, Anitua E. Ultrasonic osteotomy for the surgical approach of the maxillary sinus: a technical note. Int J Oral Maxillofac Implants 1998;13:697–700.
15. Abella F, de Ribot J, Doria G, Duran-Sindreu F, Roig M. Applications of Piezoelectric Surgery in Endodontic Surgery: A Literature Review. J Endod 2014;40:325-32.
16. Rahnama M, Czupkałło L, Czajkowski L, Grasza J, Wallner J. The use of piezosurgery as an alternative method of minimally invasive surgery in the authors' experience. Wideochir Inne Tech Maloinwazyjne. 2013;8(4):321-6.

JSPIK

Role of Tactile Sensitivity in Dentistry

Shaheena K P[1], Sanjeev R[2], Shyamaladevi M P[3], Plato Palathingal[3], Megha Varghese[4], Najeer Musthafa[5], Rasha P Razak[5]

ABSTRACT

Tactile sensitivity is the ability to distinguish relative degrees of tooth surface roughness or smoothness through the sense of touch and proprioception. Tactile sensitivity helps to determine subgingival calculus, deposits, tooth surface irregularities defective margin on restoration, decalcified areas, and carious lesions and for effective root debridement non- surgically, thereby render the root surface acceptable to the tissues so that healing occurs. Endodontic technique requires the operator to develop a sense of tactile feedback from the file as root dentine and supporting structures of the tooth do not allow direct visualization of the canal preparation. Acquiring abilities and skills to perform dental procedures is essential for dental students and clinicians. it needs a practical experience of the tactile information to gain clinical expertise

Keywords: tactile sensitivity, instrument grasp, strokes, dentinal caries, fixed partial denture

Introduction

The profound reliance of the human species on tools for its survival and adaptation is unique in the animal world. Prehistoric evidence for tool use as an adaptive strategy in human evolution extends back at least 3.3 million years, when stone tools began to be found at prehistoric sites in Africa in regions containing fossils of early bipedal ancestors. Recognition and treatment of periodontal disease can be traced back to antiquity. Descriptions of treatment are found in ancient Egyptian and Chinese writings and would suggest that periodontal diseases were recognized possibly 500 ears ago. Albucasis (936-1013), who was born and lived in Moorish Spain. The contributions of Albucasis to dentistry and periodontology were outstanding achievements. He had a clear understanding of the major etiologic role of calculus deposits, and he described the techniques of scaling the teeth with the use of a set of instruments that he developed.

For the treatment of periodontitis, Eustachius recommended both the scaling of calculus and the curettage of granulation tissue so that actual reattachment of the gingival and periodontal tissues could take place. Thorough root debridement is a demanding task, and it is necessary to achieve a biologically-acceptable root surface. To assess subgingival deposits and effectiveness of scaling and root planing one of the reliable method is tactile sensitivity. In dentistry Tactile sensitivity felt with dental explorers, curetes, endodontic files or other dental instruments used to detect the texture and character of tooth surface before, during and after treatment, to assess the progress and thoroughness of instrumentation.

Tactile sensitivity is the ability to distinguish relative degrees of tooth surface roughness or smoothness through the sense of touch and proprioception.[1]

[1]Post graduate student, [2]Professor and Head, [3]Professor, [4]Reader, [5]Lecturer, Department of Periodontics, PSM College of Dental Science and Research, Thrissur, Kerala, India. Correspondence Author: Dr. Shaheena K P, Email: dr.shaheenakp@gmail.com

Tactile sensitivity can be defined as the ability to feel vibrations transferred from the instrument working end, shank, and handle to the clinician's finger(4). Tactile sensitivity helps to determine subgingival calculus, deposits, tooth surface irregularities defective margin on restoration, decalcified areas, and carious lesions and for effective root debridement non- surgically, thereby render the root surface acceptable to the tissues so that healing occurs. Endodontic technique requires the operator to develop a sense of tactile feedback from the file as root dentine and supporting structures of the tooth do not allow direct visualization of the canal preparation. Poor tactile sensitivity can lead to over instrumentation or under instrumentation, both are unacceptable.

Physiology of Tactile Sensitivity

Tactile sensitivity is reliant on several structures in the hand, including the median nerve, encapsulated nerve endings, and specialized capsules of connective tissues.[2] The median nerve runs through and innervates the thumb, index, middle, and median aspect of the ring finger. The median nerve houses large nerve fibres, such as A-beta fibres, which are characterized by vibratory, proprioceptive, and tactile discriminatory sensation. According to Vinik et al., "tactile discriminatory sensation is mediated primarily via the large, but thinly myelinated, fast-conducting sensory afferents (A-beta fibres) innervating skin and underlying soft tissues. Due to difficulties in quantitatively detecting specific sensory deficits, little definitive data exists addressing the issue of nerve fibre involvement."

Microscopic mechanoreceptors involved in sensation lie within the Pacinian corpuscle, which is most sensitive to skin displacements. The Pacinian corpuscle is a rapidly adapting receptor that lies on a nerve ending and consists of a multi-layered connective tissue sheath that is approximately 1mm in diameter and 3mm in length.[2] Its purpose is to aid in vibration detection.

Factors Affecting Tactile Sensitivity

1) Instrument Design
2) Instrument Grasp
3) Stroke Character
4) Angulation of Instrumentations
5) Gloves

Modifications to Improve Tactile Sensitivity

1. WEIGHT—light weighted hollow handles improve tactile sensitivity
2. STEEL INTERNAL CORE – Helps to transfer tactile perception to finger tips
3. DIAMETER—it ranges from 3/8 inch to 3/16 inch
   - Large diameters are easier to grip and reduce muscle fatigue
4. SURFACE TEXTURE—Serrations helps to increases control and reduces hand fatigue
5. BALANCE—blades of working ends are aligned with the long axis of the handle
   - Balancing allows the finger pressure to be transferred more effectively to the working end

Figure 1 Neutral positioning for forearms, parallel to the floor or 900 to the floor

Figure 2 Neutral positioning of the hand

- Assists in maintaining adaptation and rotating between fingers with control
- Reducing muscle stress on the hands and arms

6. ROUND OR HEXAGONAL CROSS-SECTION
7. SHARP INSTRUMENTS -Clinician need sharp instruments to deliver quality care as sharp instruments provide the clinician with greater tactile sensitivity. Clinician using dull curettes subgingivally will feel much less, as the rounded cutting edge slides over calculus deposits. A sharp cutting edge will catch on the deposit, and the clinician will feel it more readily. Dull instruments are horrible and require a tighter pinch grip, which creates fatigue and reduces tactile sensitivity, and increases the force and pressure a clinician has to use to try to remove deposits.

Instrument Grasp

(a) Neutral wrist position

Maintaining a neutral position with the modified pen grasp requires modification based on wrist and forearm positioning. A neutral position eliminates strain from flexion and extension. The goal of the neutral forearm position is to keep the forearms parallel to the floor and to not stray beyond 60° in an upward direction from parallel, or beyond 100° in a downward direction (Figure 1).

Neutral positioning of the hand includes maintaining the pinkie finger slightly lower than the thumb and keeping the wrist aligned with the forearm (Figure 2). The clinician should avoid rotating the thumb side of the hand down so that the thumb is lower than the pinkie finger; the hand should not be bent in an upward or downward direction.

(b)Modified pen grasp

The recommended grasp for holding periodontal instrument is the modified pen grasp, which allows precise control of the working end. The modified pen grasp is a variation of the pen grasp. Whereas a pen grasp varies from person to person, the modified pen grasp requires all clinicians to hold the instrument in a similar manner. The advantages of the modified pen grasp are better control of the instrument, prevention of finger fatigue, and an increase in tactile sensitivity.

Clinicians should notice placement of the thumb, index, and middle fingers as well as the relationship of the instrument handle to the second and third knuckles of the index finger. In a modified pen grasp, the instrument is held in the dominant hand with the pads of the index finger and thumb opposite each other on the handle closest to the working end. The thumb and index finger are not touching, thereby creating a tripod effect with the middle finger that is placed along the shank of the instrument (Figure 3A). This tripod effect balances the instrument in the clinician's hand to provide stability and control. By keeping the index finger and thumb separated, the clinician can roll the instrument between these digits with ease and control.

The thumb is slightly curved in a c-shape, whereas the index finger is straight from the fingertip to the second knuckle. The index finger bends at the second knuckle, and the instrument handle rests somewhere

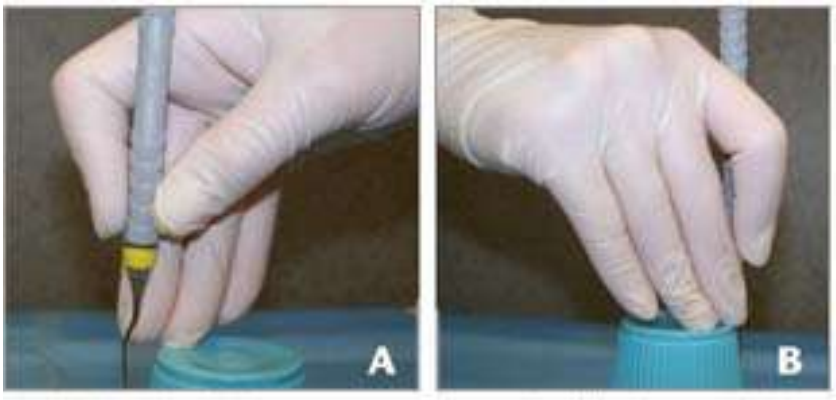


Figure 3. Modified pen grasp. A shows the side view while B depicts the front view

Figure 4 Handle position beyond third knuckle of index finger

between the second and third knuckle (Figure 3A). If the index finger bends at the first knuckle, it may result in loss of wrist motion and strength for scaling.

Precise placement of fingers in the modified pen grasp is important for successful instrumentation technique. light grasp is needed for increased tactile sensitivity during assessment procedure, and also proper fit of sterile glove is important in avoiding muscle strain and to increase tactile perception.

The practice of dental hygiene with its repetitive movements and static postures often leads to a variety of musculoskeletal problems for clinicians. More than 80% of dental hygienists experience some sort of upper body and back pain. Common disorders related directly to hand and forearm pain include carpel tunnel syndrome, tendonitis, tenosynovitis, and extensor wad strain. These disorders occur over time as a result of repetitive movements—related to improper grasp and hand movements—for long periods of time on a daily basis.

Stroke Character

For subgingival assessment a relaxed grasp and light lateral pressure are used to enhance tactile sensitivity. For assessment fluid strokes of moderate length are used also many, overlapping stokes are used to thoroughly cover the entire root surface with a combination of hand fore-arm and digital activation. The grasp pressure is light so that tactile sensitivity is enhanced. Assessment stroke is also called exploratory stroke. The purpose of the assessment stroke is to do the following:

- Assess the surface texture of the root
- Confirm positioning of the back of the curate below the apical margin of the calculus at the soft tissue attachment.
- Rehearse the movement of the curette before activating a working stroke to confirm correct adaptation.

Probe is used to locate the attachment at the bottom of periodontal pocket and to estimate the amount of deposits present on the root surface while probing. Explorer is used to evaluate surface smoothness following treatment. Historically, the use of tactile examination of a tooth surface, with firm application of a sharp tip of an explorer into a suspected site of caries, was a common method of caries detection, but this approach is no longer recommended.

Angulation of Instrumentations

A factor closely related to and directly influencing instrument adaptation is angulation. Angulation refers to the angle formed at the cutting edge of an instrument between the tooth surface and the face of the instrument. Each instrument is applied to a surface in a specific manner for optimum adaptation and angulation. The curette is angulated at 0 degree with tooth surface when used in an exploratory or insertion stroke. The usual adaptation of probe is to maintain the side of the working tip on the tooth, with the long axis of the working end nearly parallel to the tooth surface. The side of the explorer tip is kept on the tooth at all times to feel for changes in the surface roughness, the angle is 5 degree or less.

Gloves

The routine wearing of gloves by all dental clinical staff is now considered essential for prevention of cross infection (British Dental Association 1986). Glove use was also shown to have no effect on the acquisition of psychomotor skills by a group of first year dental undergraduates performing simple restorations (Brantley et al. 1986). The use of gloves resulted in a statistically significant reduction in tactile sensitivity (N. P. Chandler 1990).[1] Reduced sensitivity was reported when using small hand instruments, and improvements in instrument design were suggested to overcome the lack of tactile sensitivity, and also to reduce the incidence of punctured gloves. Chemical resistant gloves offer excellent dexterity and tactile sensitivity and work in a variety of applications.

Tactile Sensitivity in Periodontics

Calculus detection and removal

The primary goal of periodontal therapy is to remove biofilm and calculus deposits to promote a healthy periodontium. Non-surgical periodontal therapy, involving the removal of readily detectable calculus, helps to achieve and maintain the health of the adjacent periodontal tissues.[7,8] Removal of bacteria-

harbouring calculus and contaminated cementum reduces the levels of subgingival plaque and promotes the healing process. Although the complete removal of calculus may not always be achievable, periodic re-evaluation of the periodontal tissues for signs of inflammation, aids in the detection of residual calculus. Supragingival calculus which is easily seen can be removed effectively using scalers, but the problem arises in completely removing the sub-gingival calculus, which mainly depends on tactile sensation.

For subgingival assessment of calculus, a relaxed grasp (modified pen grasp) and light pressure are used to enhance tactile sensitivity; pressure with middle finger against the instrument shank should be avoided, as this reduces tactile sensitivity. A combination of hand forearm activation, made by rotating the hand and fore arm as a unit to provide the power for an instrumentation stroke; similar to the action of turning a doorknob, and digital activation, created by flexing the thumb, index and middle fingers to move the instrument, with many overlapping fluid strokes are used to thoroughly cover the entire root surface.

Tactile Sensitivity in Conservative and Endodontics

a) Root canal treatment

The technical quality of root canal treatment (RCT) may impact on the outcome. Tactile perception is how accurate we can be in determining what the tip of the negotiating endodontic instrument is encountering. Superior tactile perception is a direct result of the instruments design and how it is used. A reasonable analytic task is to determine what endodontic instrument designs and techniques enhance tactile perception. The information conveyed from the tip of the instrument will become increasingly clear as the engagement along length is reduced.

In that light the typical K-file design consisting of 30 horizontally oriented flutes along length will engage the walls of the canal significantly more than a reamer with 16 more vertically oriented flutes. If both the reamer and the file are made from a square wire, the reamer with 16 flutes will have a total of 64 contact points while the file with 30 flutes will have 120 contact points. The greater the number of contact points the greater the engagement and the increase in resistance to apical negotiation. Increasing resistance along length reduces the tactile perception at the tip.

Ideal tactile perception tells the dentist when a solid wall is hit. The dentist differentiates this type of engagement from being in a tight canal by the degree of tug back. No immediate tug back means the dentist is hitting a solid wall. Immediate tug back means the dentist is most likely in a tight canal that will allow him to progress to greater depths using either a tight watch-winding motion or via the instrument's use in the 30-degree reciprocating handpiecce.

Fewer more vertically oriented flutes increase tactile perception. Fewer flutes also make the instrument less work-hardened, which in turn makes the instrument more flexible, another feature that enhances tactile perception. Placing a flat along the entire working length further improves tactile perception by further reducing engagement along length while making the instrument even more flexible. Those 64 contact points are now reduced to 48.

A cutting tip is an additional feature that improves tactile perception. Unlike a non-cutting tip that has the potential to impact pulp tissue, a cutting tip tends to pierce it. There is no concern about a cutting tip creating its own pathway because the degree of motion is limited to either a tight watch winding stroke or the 30-degree arc generated by the reciprocating headpiece.

Compare this approach to the use of K-files and the subsequent use of rotary NiTi. The K-files are poorly designed to enhance tactile perception because they engage excessive amounts of tooth structure along length. Their horizontally oriented flutes are designed to engage, not cut, and the great number of flutes resulting from twisting the wire more times produces a stiffer instrument incompatible with superior tactile perception.

b) Early caries detection

Identification of early caries lesions, as early as possible, have greatly aided in minimally invasive dentistry or "micro dentistry". Dental caries has a multifactorial aetiology and as extensive advances have been made in understanding the relationship between oral environment and caries, it is now clear that the early stages of caries which involve demineralization can be

reversed/arrested or its progression be delayed. The traditional methods of caries detection have included direct visual-tactile inspection followed by radiographs.

Dental caries detection methods:

Visual-tactile examination: The basic first step in caries detection is visual inspection (VI) of teeth aided by the use of compressed air. In the VI the dental practitioner should be looking for: White chalky lesions, changes in colour and translucency of enamel, any discoloration (grey/black), and break in the continuity in the enamel surface. This is usually followed by exploring the surface of teeth with an explorer. This tactile examination is no longer recommended as its scratching tends to prevent any chances of remineralization of any early lesion. If it has to be done either a slightly blunt probe should be used, that to with very little pressure.

For caries detection, apply light pressure technique with the point of the explorer against the region of the suspected caries; firm pressure should not be used to force the explorer tip to penetrate the root surface. When exploring a suspicious area,the walls of the lesion will feel soft, tacky or leathery consistency. Subgingival, smooth surface lesion is detected as a rough, concave area on the surface of root. "Tackiness" on the surfaces of the tooth. The probe sticks to surfaces of the tooth when a little pressure is applied, and a definite pull is required to remove the probe (resistance in removing probe known as a tug back). This method is no longer used since it may result in the cavitation of initial lesions which may be reversed.

Irregular overhanging or deficient restorations can be detected while passing explores along the margins of restorations.

Tactile Sensitivity in Prosthodontics

Tactile perception in marginal opening identification

In fixed partial dentures, an opening of approximately 50 micrometre may be considered clinically satisfactory. Larger marginal gaps have been linked to fracture and dissolution of the cement, with retention of bacterial plaque. Walton et al reported that defective margins are the cause of approximately 10% of failures after a mean period of 7.4 years. Thus, the margins of fixed partial dentures should be intimately adapted to tooth structure. To evaluate restorations before cementation, marginal adaptation is usually verified by radiography" and/or by running a sharp-tipped explorer over the prosthesis margins, commonly in an apex-crown and crown-apex direction. Resistance in both directions indicates a defect of the open margin type; a unidirectional obstacle indicates either a ledge or an overhanging restoration

Two phases can be distinguished when using explorers for tactile evaluation. First the surface defect is discovered, and then the resistance it causes on explorer advancement is evaluated. Only in the second phase may a conclusion be drawn about the shape and size of the defect, but the defect on the prosthesis surface must be recognized first.

Paolo Baldissara, Silvia Baldissara, Roberto Scottlto (1998) done a study to establish the reliability of different instruments for discovering marginal openings. Result was

1. The tactile perception method, using a sharp-tipped explorer, is reliable in detecting grooves simulating open margin defects on a smooth metal surface. This instrument allowed easy identification (greater than 95%) of grooves ranging from 165 to 36 micrometre in width.
2. The dull-pointed stylet was markedly less sensitive ($P < 0.001$) than the sharp-tipped explorer. Worn, large, radius-tipped instruments are not suitable for marginal defect detection.
3. Whereas the tactile perception with a sharp explorer is reliable in discovering open margin defects.

Conclusion

Manual dexterity is a crucial skill in mastering dentistry. Operative dental procedures involve drilling into a tooth with precise hand–eye coordination and depth perception. While holding the rotating hand piece between thumb and fingers, the operator registers the continuous flow of both tactile input and visual feedback to the brain.

To bring better tactile sensitivity Modifications in instrument types, instrument grasp, stroke character, angulation of instrumentations are made. Tactile sen-

sitivity felt with dental explorers, curetes, endodontic files or other dental instruments used to detect the texture and character of tooth surface before, during and after treatment, to assess the progress and thoroughness of instrumentation. Acquiring abilities and skills to perform dental procedures is essential for dental students and clinicians, it needs a practical experience of the tactile information to gain clinical expertise.

References

1. Tsutsui P. Periodontal instruments and instrumentation theory. In: Darby ML, Walsh MM., editors. Dental hygiene theory and practice. 2nded. Philadelphia, PA: WB Saunders Co.; 2003. 385- 431.
2. Hunter JM, Schneider LH, Macking EJ. Tendon and nerve surgery in the hand. 3rded. St. Louis, MO: Mosby; 1997
3. N. P. Chandler, G. P. Bloxham: Effect of gloves on tactile discrimination using an endodontic model. Department of Restorative Dentistry, School of Dentistry, University ofOtago. Dunedin, New Zealan, International Endodontic Journal,1990;23(2):97-9.
4. Daniel SJ, SA Harfst, RS Wilder. Mosby's Dental Hygiene: Concepts, Cases and Competencies. 2nd ed. Philadelphia: Elsevier; 2008.
5. PaoloBaldissara, Silvia Baldissara, DDS" Roberto Scottl, MD, DDS'; Reliability of Tactile Perception Using Sharp and Dull Explorers in Marginal Opening Identification. Intl J Prosthodont 1998: 11:591-4.
6. Ryan DL, Darby M, Bauman D, Tolle SL, Naik D. Effects of ultrasonic scaling and hand-activated scaling on tactile sensitivity in dental hygiene students. J Dent Hyg. 2005 Winter;79(1):9.
7. Cobb CM. Clinical significance of non-surgical periodontal therapy: an evidence-based perspective of scaling and root planing. J Clin-Periodontol. 2002 May;29Suppl 2:6-16.
8. Corbet EF, Vaughan AJ, Kieser JB. The periodontallyinvolved root surface. J ClinPeriodontol. 1993 Jul; 20(6):402-10.

JSPIK

Altered Passive Eruption and its Treatment - A Review

Shilpa Lakshmi M[1], Mahesh Narayanan[2], Shabeer Ahamed[3], Jeethu John[4], Vidya Vishnu[5]

ABSTRACT

A smile is the most common facial expression to express happiness. A pleasant smile is not only influenced by the teeth alone, but also by the gingiva and the lips. All these components in an esthetic harmony create a beautiful smile. Excessive gingival display while smiling is one of the significant esthetic concern for many of the patients. One of the aetiology for this is altered passive eruption (APE). Altered passive eruption is characterized as a dento gingival relationship in which the gingival margin is coronally placed at the cemento enamel junction due to the disruption of the teeth 's development and eruptive pattern. There are many techniques available to correct the gummy smile problem caused by altered passive eruption. A clinician must have sound knowledge about the classification and treatment options of altered passive eruption to treat patients accurately and to provide the desired aesthetics and functional outcome. The aim of this article is to present a narrative review of the aetiology, classification and management of altered passive eruption.

Keywords: Passive eruption, gummy smile,smile esthetics, gingivectomy

Introduction

An esthetic smile is considered as an important aspect of an individual's beauty. A pleasant smile is not only influenced by the teeth alone, but also by the gingiva and the lips. The periodontal tissues are considered as important factors to achieve this esthetic smile. Excessive gingival display or gummy smile is a common condition that impairs smile esthetics. A smile may be considered as pleasant when the upper teeth are completely exposed, and approximately 1mm of buccal gingival tissue is visible. According to Allen et al[1] in 1988, A gum exposure not exceeding 2-3 mm is often considered good, whereas an extreme exposure (> 3 mm) is usually deemed unattractive by many patients.

Excessive gingival tissue, frequently called Gummy Smile, can be associated to vertical maxillary growth, dentoalveolar extrusion, short upper lip, upper lip hyperactivity, Altered Passive Eruption (APE) or a combination of these factors.[2] The prevalence of APE is reported to be approximately 12% considering more than 1,000 adult patients with mean age of 24 years.[3]

Review of Eruptive Processes

Tooth eruption includes two phases. One is an active eruption phase in which the tooth emerges into the oral cavity and a passive eruption phase that is characterized by apical migration of the soft tissue covering the crown of the tooth.

[1]Post Graduate Student, [2]Professor and HOD, [3]Professor, [4] Reader, [5]Senior Lecturer, Department of Periodontics, Malabar Dental College & Research Centre, Edappal, Malappuram - 679 578, Kerala

Active eruption phase

According to Steedle & Proffit [4] the active eruption phase can be subdivided into six distinct phases

- Pre-functional phases (follicular growth, pre-emergence and post-emergence eruptive outbreak)

- Post-functional phases (juvenile occlusal equilibrium, pubertal eruptive outbreak and adult occlusal equilibrium).

Passive Eruption Phase

Passive eruption, a term coined by Gottlieb & Orban in 1933, is a gradual process by which the epithelial attachment of the gingival tissues migrates apically from the coronal enamel to a stable position just coronal to the cemento–enamel junction with a fibrous connective tissue attachment forming at the base of the gingival sulcus. This phase can be subdivided into four phases. In stage I, base of the gingival sulcus and junctional epithelium is on the enamel. In stage II, base of the sulcus is on the enamel and part of junctional epithelium on the root. In stage III, the base of the sulcus is at the cementoenamel line,and the entire junctional epithelium is on the root. In stage IV, base of the gingival sulcus and junctional epithelium are on the root. The first phase of passive eruption is considered to be physiological, while the remaining three phases are considered as consequences of pathological periodontal destruction.[5]

Concept of Altered Passive Eruption

Goldman and Cohen (1968)[6] termed the failure of the tissue to adequately recede to a level apical to the cervical convexity of the crown as "altered passive eruption." Volcansky and Cleaton-Jones (1974)[7] described the tissue's failure to reach the CEJ junction as "delayed passive eruption".

Classification of Altered Passive Eruption (APE)[Coslet et al][8]

APE TYPE 1- This is determined by an exclusive failure of the passive eruption resulting in excessive gingival overlap on the anatomical crown of the tooth, while the distance from the bone crest to the cemento enamel junction should be usual in comparison.

APE TYPE 2 - It is determined by primary failure of active eruption phase, as a result of which the tooth will not emerge sufficiently from the alveolar bone, thereby leaving cementoenamel junction very close to bone crest. This would prevent apical migration of gums during passive eruption phase.

Both types are subtyped in turn into subtypes A and B.

In subtype A the distance between the bone crest and cementoenamel junction is 1.5-2 mm (which enables the root cement to have a regular dimension of the connective tissue fibre attachment).

In subtype B the bone crest lies very close to, or even at the same level as the cement enamel line.

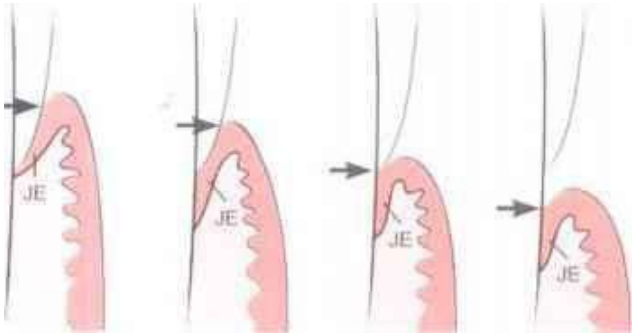


Figure 1: Stages of passive eruption as described by Gottlieb and Orban5

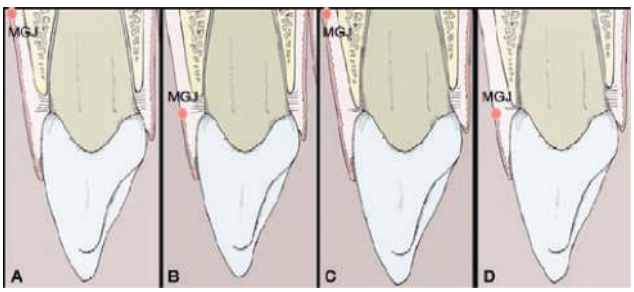


Figure 2: Diagramatic representation of altered passive eruption types 1 &2 Subtypes A and B

TYPES AND SUBGROUPS OF ALTERED PASSIVE ERUPTION[9]

Fig A	Type 1 subgroup A	Mucogingival junction is located apically with respect to the cemento–enamel junction and the buccal bone crest. The distance between the cemento–enamel junction and the bone crest is physiological for connective tissue fiber attachment.
Fig B	Type 2 subgroup A	Mucogingival junction is located at the level of, or coronal to, the cemento–enamel junction. The distance between the cemento–enamel junction and the bonecrest is physiological for connective tissue fiber attachment.
Fig C	Type 1 subgroup B	The mucogingival Junction is located apically with respect to the cemento–Enamel junction and buccal bone crest. The bone crest is located at the level of, or coronal to, the cemento–enamel Junction and there is no physiological space for connective tissue fiber attachment.
Fig D	Type 2 subgroup B	Mucogingival junction is located at the level of, or coronal to, the cemento–enamel junction. The bone crest is located at the level of, or coronal to, the cemento–enamel junction and there is no physiological space for connective tissue fiber attachment.

Etiology of Altered Passive Eruption

Failure of the passive eruption phase, gives rise to Type 1 altered passive eruption. There is excessive overlap of gingiva and the anatomical crown of the tooth. There is no change in the distance from the bone crest to the cemento–enamel junction. Failure of the active eruption phase can lead to type 2 altered passive eruption and the tooth does not emerge sufficiently from the alveolar bone, leaving the cemento–enamel junction positioned in proximity to the alveolar bone crest. The apical migration of gingiva during the passive eruption phase is thus prevented.

The presence of thick bone that might prevent the apical migration of soft tissue, thick and fibrotic gums that tend to migrate more slowly during the passive phase, occlusal interference by soft tissues during the eruptive phase, orthodontic trauma endocrine conditions and genetic causes are the possible causes of altered passive eruption.[10]

The prevalence of altered passive eruption following orthodontic treatment using maxillary and mandibular fixed appliances, compared with patients who had never received orthodontic treatment was determined by Nart et al.[11] Certain endocrine alterations, such as hypopituitarism and hypogonadism, are also related to a delay in tooth eruption. Barberi et al[12], reported that children who had a deficiency of growth hormone also presented a delay in dental and bone age. The association between hypothyroidism and the presence of altered passive eruption was not infrequent, determined by Goldman & Cohen.[6]

The presence of thick buccal bone was a common observation in surgically managed cases of altered passive eruption, proposed by Zucchelli.[13] The genetic component of altered passive eruption was studied by Rossi et al. The study investigated whether patients with altered passive eruption have siblings or parents with similar dental characteristics.[14]

Piattelli et al[15] used the term "primary failure of eruption" in for those cases with no apparent underlying cause, suggesting as etiological mechanisms an alteration of the metabolism or of the blood flow in the periodontal ligament. This mechanism was suggested by Proffit to explain certain open posterior bites. Pri-

mary failure of eruption in turn could be associated to other general alterations such as osteoporosis, Gardner syndrome, chondroectodermaldysplasia, sternocleidal dysplasia, achondroplasia, and Down's syndrome.

Influence of Altered Passive Eruption on Periodontium

According to various Studies altered passive eruption is a potential risk factor for development of periodontal disease. In altered passive eruption, there is a risk from repeated trauma as the gingiva does not recede to its normal position and remains on the surface of the crown. Sometimes, excess gingival tissue may also interfere with adequate oral hygiene and the accumulation of plaque may give rise to marginal gingival inflammation. Orthodontic appliances restorations and placed in close proximity to the gingival margin may cause an inflammation resulting in gingivitis and attachment loss in periodontally susceptible patients.

According to Prichard[16] when the gingival margins are incisally placed there is more chance of trauma from oral function and is more susceptible to periodontal pathoses. This can contribute to chronic inflammation of a bulbous marginal gingiva.

Relationship between the presence of altered passive eruption and acute necrotizing ulcerative gingivitis was reported by Volchanky & Cleaton-Jones.[17] The presence of a deep gingival sulcus favours necessary anaerobic conditions for the development of infection. Weinberg & Eskow[18] suggested that in the presence of pseudopockets which develop as a result of excessive keratinized mucosa,increased plaque accumulation and an inflammatory response may not be esthetically pleasing to the patients.

Altered Passive Eruption and Restorations

Restoration of tooth with altered passive eruption is challenging to the dentist both in the functional and esthetic aspect. Difficulty may arise in resistance and retention form if the clinical length of the crown is not increased. A dilemma exist in the placement of the crown margins. Although it is preferable, where possible, to place restorative margins supragingivally, for esthetic reasons it may sometimes be necessary to place subgingival restorative margins. Though the subgingival margins provide esthetics, there are increased chance for plaque retention and periodontal breakdown. A common error made when patients with altered passive eruption are treated is the placement of margins at what would ordinarily be normal anatomic level. Such marginal placement may invade the biological width because of increased alveolar bone height, resulting in inflammation.

Evian et al[19] proposed that, in multirooted teeth, excess soft tissue can interfere with the interproximal placement of restorations. Eliminating the excess tissue allows improved access and makes plaque control easier for the patient. Anteriorly, altered passive eruption can result in short-looking teeth, and crown and bridgework can be used to improve esthetics. However, incorrect placement of crown margins can cause an inflammatory response, worsening the appearance of the teeth in the long term.

Altered Passive Eruption and Smile Esthetics

There are many factors influencing the smile esthetics. In fact, maxillary anterior teeth, along with the position of the gingival zenith and balance of the gingival levels, are considered to be the key elements of a pleasant smile.[20]

Figure 3: High smile line or gummy smile (pre- and postsurgical correction). (A) Gummy smile as a result of altered passiveeruption. (B) Surgical treatment of altered passive eruption modifies the Peck classification of this smile from a high smileline to a normal smile line.[9]

According to Peck et al[21] there are three classes of smile line:

A 'normal' smile line - the upper anterior teeth are completely visible and the lower border of the upper lip reveals 1–2 mm of gingiva.

A 'low' smile line - the lower border of the upper lip covers 25% of the upper anteriorteeth. A 'high' smile line- known as a 'gummy smile', is one having more than 2 mm of maxillary gingival display.

The prevalence of excessive gingival display is more common in women (14%) than in men (7%)[22]. Excessive display of gingiva can have an adverse effect on the patient's self-confidence. If the origin of the excessive gingival display is a skeletal abnormality, then orthodontic treatment and orthognathic surgery is preferred. If there is a dental reason for the excessive gingival display, then the gingival and osseous architecture is corrected.

Diagnosis of Altered Passive Eruption

The literature identifies various techniques for diagnosing altered passive eruption, including clinical evaluation, cemento-enamel junction examination, alveolar bone crest, and radiographic study.

Clinical Examination

The extra-oral examination includes an evaluation of facial symmetry and height, lip or smile line, lip length and mobility. The first step in diagnosis is to observe the patients smile. If there is excessive display of gingival then further data is required. The length and activity of the maxillary lip is measured. This is usually 20–22 mm in female patients and 22–24 mm in male patients. If the excessive gingival display is caused solely by a shorter lip or lip hyperactivity, no treatment is indicated.[23]

The next is to locate the cementoenamal junction. If the CEJ is located in the normal position within the sulcus, the patient dosent have an altered passive eruption. This may be due to short teeth as a result of incisal wear or due to abnormal dental anatomy. The average length of central incisor is 10.5mm[24]. So inorder to find the missing incisal edge the dentist should measure from the CEJ to the incisal edge and subtract this from 10.5mm. In such cases crown lengthening can be performed. When CEJ is not detectable in the sulcus then it can be diagnosed as altered passive eruption. So bone sounding can be performed.

'Bone sounding', under anesthesia, is a technique used to differentiate between altered passive eruption subtypes A and B. If transgingival probing reveals the cemento–enamel junction subgingivally, it is altered passive eruption subtype A. If the alveolar bone crest is palpated by the probe without the cemento–enamel junction being detected, then it is subtype B. The suggested 'sounding' of Zucchelli[13] is only useful in extreme cases because only one disruption is heard during subgingival sampling in the vast majority of cases and it is difficult to differentiate between the cemento-enamel junction and the bone crest. Moreover, even if two interruptions of subgingival are observed, it can be very difficult to decide if the distance between them is physiological (1–2 mm) or not.

Radiographic Diagnosis

Dental x-ray provides a valuable contribution to the treatment of altered passive eruption. Parallel profile radiography, as defined by Alpiste- Illeuca[25], was used to determine the dimensions of the components of the dentogingival unit, allowing the measurement of differences and the degree of overlap characterizing the altered passive eruption. This radiograph was obtained using a radio-opaque gutta-percha inserted into the base of the sulcus and a self-adhesive lead plate placed over the base of the sulcus and a self-sticking lead plate positioned over the keratinized gingival surface. Thus the clinical and radiographic diagnoses were correlated.

Levine & McGuire[2] proposed using periapical radiography with the parallel long-cone technique to gain details on the cemento – enamel junction and bone crest. Some writers use radiographic measures to obtain clinical measurements to guide them during surgery.

Batista et al[26] suggested the use of cone beam computed tomography to diagnose and characterize the anatomical hard and soft tissue characteristics of altered passive eruption-affected teeth and to present a novel, combined surgical approach to their correction based on biometric information obtained using cone beam computed tomography.

Treatment of Altered Passive Eruption

1. Presurgical Phase

Treatments of altered passive eruption described in the literature are primarily surgical; there are no data on altered passive eruption non-surgical treatment. The initial therapy consists of oral hygiene instruction, scaling and 'root' planning. Pseudopockets cannot be reduced by 'root' planing because the tissue beneath it is enamel and not cementum or root dentine.

At the other hand, the accidental curettage of the pseudopocket's soft-tissue wall may cause some mild soft-tissue shrinkage, which the patient may perceive as an unesthetic 'gingival recession' as a result of the tooth's elongated appearance with a pseudopocket as compared to the adjacent teeth. Debridement reduced inflammation in the absence of pseudopockets, allowing accurate assessment of the extent of the altered passive eruption. The process of preoperative care will precede any surgical procedure.

2. Surgery

The treatment approach depends upon a number of factors which include the position of the marginal gingiva, the width of keratinized gingiva, the location of the alveolar buccal crest, the location of the mucogingival junction and the probability of a concomitant restorative therapy. According to Garber & Salama[27] there are only two treatment options for cases of altered passive eruption.

- A simple gingivectomy to expose the hidden anatomy in cases of altered passive eruption type 1A

- An apically repositioned full-thickness flap, with or without osseousresective surgery, in other cases of altered passive eruption

a) Gingivectomy/Gingivoplasty

Gingivectomy is indicated when sufficient amount of attached gingival remain after surgery and greater than 3mm of tissue exists from bone to the gingival crest. The initial incision is placed on the gingiva at the level of the CEJ. This reflects the normal gingival architecture, the highest point of the gingival margin is slightly distal to the centre of the tooth. This incision should be symmetric and precise,hence should be accomplished while the operator is in front of the patient. A stent can be used as a guide for initial incision.[28]

Dolt & Robbins[23] say that, when sitting behind the patient, it is difficult to make the initial scoring incision correctly and recommended sitting in front of the patient and using an acrylic resin or resin composite stent as an operative guide. Papillary tissue is left undisturbed except for minor blending with gingivectomy incision and the tissue is only removed from the facial aspect. A second incision is made in the sulcus of each tooth, which leaves a gingival collar that is excised with a periodontal curette. Kurtzman & Silverstein[29] are suggesting the use of a black permanent marker guide line for the initial incision along with an operating template. Different instruments can be used to expose the cemento–enamel junction and to obtain a more physiologic gingival contour, including scalpel, electrosurgery or carbon dioxide laser.

A bipolar electrosurgery pen can be used for gingivectomy under copious irrigation; A gingivoplastic pen can then be used to plane the bulky tissue back on the papilla and provide a natural contour. Eventually, a coagulation ball pen can be used in the coagulation mode of the bipolar device to seal any bleeding on the surface.[29]

b) Apically Positioned Flap

The width of keratinised gingival is normal in type 2A, hence conventional gingivectomy may eliminate too much of keratinised gingival. In such cases apical positioning of the band of attached gingival at or near to the CEJ is performed.

Zucchelli[13] proposed that, apically positioned flap is the treatment of choice in majority of the patients affected by altered passive eruption with an increased thickness of the buccal bone, necessitating osteoplasty.

c) Apically Positioned Flap with Osseous Resective Surgery

The ostectomy is signaled when the diagnostic procedures show osseous levels approximating the cemento – enamel junction point. This technique is often connected to an apically located flap, although some scholars propose a flapless esthetic crown lengthening technique. The initial incision either can be made as that of gingivectomy procedure, with or without a

surgical template, or can be as a sulcular incision.

The incision will be made as submarginal as possible, preferably as scalloped. This enables the incision to replicate the scalloped shape of the cemento – enamel junction and permits wound healing at the level of interdental space by primary intention. A variable-thickness flap is elevated, split thickness at the surgical papillae and full thickness at the buccal aspect of the incision, with the aim of providing the surgical flap with a uniform thickness. The incisions slash through the buccal surface of each papilla, leaving the papilla interproximally totally intact.

This flap configuration retains the height of the papillae and provides fixed tissue during suturing for flap stabilisation. The buccal interdental papilla are not reflected with the flap. It is thus advisable to lift a buccal flap, keeping the interproximal papillae and palatal tissue intact so as not to impair the flow of blood to such tissues, thus reducing the possibility of tissue shrinkage. Beyond the mucogingival junction, a full-thickness flap is reflected, and the positions of the cemento–enamel junction and bone crest are visually verified.

Osteoplasty is performed to minimize bone thickness, while ostectomy is performed to assess the proper distance between the cemento-enamel junction and bone crest. The reflection of the buccal flap ends 3–5 mm apical of the buccal bone crest and is controlled, for example, by the degree of osteoplasty, if the buccal bone is especially thick, more osteoplasty is suggested and the edges of the full-thickness flap become more apical. The osteoplasty is carried out using a high-speed rotary instrument. According to Zucchelli[13], most osteoplasty is done in interradicular regions, where concave surfaces are produced for the eventual repositioning of thinned surgical papillae to reduce the interdental soft tissue rebound. The amount of octectomy needed to establish the correct distance between the cemento-enamel junction and the buccal bone crest exists as a discrepancy among authors.

Camargo et al[30] proposed that the biological width element be measured on the teeth not influenced by altered passive eruptions. He also supports further bone removal in the thick biotype to reduce the growth of soft tissue and ensure a healthy long-term outcome. Authors suggest different extent of ostectomies. Despite differences in the extent of ostectomies, all authors claim that the ostectomy's purpose is to allow space to accommodate the 'biological width.' Cairo et al.[31] and Zucchelli[13] suggested shaping the osseous crest parallel to the cemento–enamel junction. Ostectomy may be carried out with hand instruments, such as Oschenbein or Weidelstadt chisels.

Most of the authors do not advise scaling or root planing of the denuded root surfaces after ostectomy. This is because scaling or root planing could produce additional, unpredictable attachment and bone loss with potential impact on the esthetic outcome ofr patients. But according to Ribeiro et al.[32] and Cairo et al.[31], the exposed root surfaces are carefully planned using curettes. Likewise, with respect to the flap placement at the end of the surgery, some writers recommend suturing the flap at the cemento – enamel junction[23,32] and others suggest sutures to be placed slightly coronal to the cemento–enamel junction.[31]

d) Flapless Procedure with Osseous Resective Surgery

According to Ribeiro et al.[32] esthetic crown-lengthening procedure can be performed for the treatment of altered passive eruption. Without flap elevation, the alveolar bone is removed and recontoured using microchisels, via incision. Esthetic crown lengthening, with or without flap elevation, was found to have similar and stable clinical results. It was suggested that flapless procedure is more feasible, predictable and less time-cosuming for treatment of gummy smile caused by altered passive eruption compared with flap elevation. But this was not applicable in cases of type 2B altered passive eruption.

Conclusion

The demand for improvement of esthetics is part of the current periodontal practice. Esthetic treatment of a smile line is often a multidisciplinary approach where teeth, lips and periodontal tissues are involved. The treatment of APE is often challenging for the clinician. Correct diagnosis and procedure is important in the case of APE, it was possible to obtain harmony in the smile by means of crown-lengthening surgery with gingivectomy and flap surgery with osteotomy &

osteoplasty, as this procedure is less invasive and makes an esthetic smile and reduction in the case of gummy smile. The treatment may become complicated because of the difficulty in distinguishing between subgroup A and B, the presence of different subgroups of altered passive eruption among teeth in the same surgical area. Although the Surgical treatment of altered passive eruption improves patient's appearance and smile, there are still lack of studies evaluating how often patients request treatment of altered passive eruption for esthetic reasons, and evaluating patient's satisfaction with the surgical treatment outcome.

References

1. Allen EP. Use of mucogingival surgical procedure to enhance esthetics. Dent Clinic North Am 1988;32:307- 30.
2. Levine RA, McGuire M (1997) The diagnosis and treatment of the gummy smile. Compend Contin Educ Dent 18: 757-64.
3. Volchansky A, Cleaton-Jones PE (1974) Delayed passive eruption. A predisposing factor to Vincent's infection? Journal of Dental Association of South Africa 29: 291-4.
4. Steedle JR, Proffit WR. The pattern and control of eruptive tooth movements. Am J Orthod 1985: 87: 56–66.
5. Gottlieb B, Orban B. Active and passive continuous eruption teeth. J Dent Res 1933: 13: 214.
6. Goldman HM, Cohen DW. Periodontal Therapy, de 4 St. Louis, C.V. Mosby Company 1968.
7. Volchansky A, Cleaton-Jones PE. Delayed passive eruption. A predisposing factor to Vincent´s infection? J Dent Asso SAfrica 1974;29:291-4.
8. Coslet GJ, Vanarsdall R, Weisgold A. Diagnosis and classification of delayed passive eruption of the dentogingival junction in the adult. Alpha Omegan. 1977; 10:24-8.
9. Monica mele, pietro felice, praveen sharma, claudio mazzotti,Pietro bellone & giovanni zucchelli esthetic treatment of altered passive eruption periodontology 2000, vol. 77, 2018, 65–83.
10. Alpiste-Illueca F. Altered passive eruption (APE): a littleknown clinical situation. Med Oral Patol Oral Cir Buccal 2011: 16: e100–4.
11. Nart J, Carrio N, Valles C, Solis-Moreno C, Nart M, Rene R,Esquinas C, Puigdollers A. Prevalence of altered passiveeruption in orthodontically treated and untreated patients.J Periodontol 2014: 85: e348–53.
12. Barber_ıa Leache E, Mara~nes Pallardo JP, Mourelle Mart_ınez MR, Moreno Gonz_alez JP. Tooth eruption in children withgrowth deficit. J Int Assoc Dent Child 1988: 19: 29–35.
13. Zucchelli G. Altered passive eruption In: Mucogingival esthetic surgery, vol. 29. Berlin: Quintessence Publishing and Co. Inc., 2013: 749–93.
14. Rossi R, Brunelli G, Piras V, Pilloni A. Altered passive eruptionand familial trait: a preliminary investigation. Int J Dent2014: 2014: 874092.
15. Piattelli A, Eleuterio A. Primary failure of eruption. Acta Stomatol-Belg. 1991;88:127-30.
16. Prichard JF. Advanced periodontal disease, 2nd edn. Philadelphia, PA: Saunders, 1979: 420.
17. Volchansky A, Cleaton-Jones PE. Delayed passive eruption.A predisposing factor to Vincent's infection? J Dent Assoc S Afr 1974: 29: 291–4.
18. Weinberg MA, Eskow RN. An overview of delayed passive eruption. Compendium 2000: 21: 511–20.
19. Evian CI, Cutler SA, Rosenberg ES, Shah RK. Altered passive eruption: the undiagnosed entity. J Am Dent Assoc 1993:124: 107–10.
20. Magne P, Belser U. Bonded porcelain restorations in theanterior dentition: a biomimetic approach. Chicago, IL:Quintessence, 2002.
21. Peck S, Peck L, Kataja M. The gingival smile line. Angle Orthod 1992: 62: 91–100.
22. Tjan AH, Miller GD, The JG. Some esthetic factors in asmile. J Prosthet Dent 1984: 51: 24–8.
23. Dolt AH, Robbins W. Altered passive eruption: an etiology of short clinical crowns. Quintessence Int 1997: 28: 363–71.
24. Ash M.Wheeler's Dental Anatomy,Physiology and Occlusion,ed 6. Philadelphia:Saunders,1984:120
25. Alpiste-Illueca F. Dimension of the dentogingival unit inmaxillary anterior teeth: a new exploration technique(Parallel Profile Radiograph). Int J Periodontics Restorative Dent 2004: 24: 387–96.
26. Batista EL Jr, Moreira CC, Batista FC, de Oliveira RR, PereireKKY. Altered passive eruption diagnosis and treatment: acone beam computed tomography-based reappraisal of thecondition. J Clin Periodontol 2012: 39: 1089–96.
27. Garber DA, Salama MA. The esthetic smile: diagnosis and treatment. Periodontol 2000 1996: 11: 18–28.
28. Townsend C Resective surgery:An esthetic application.Quintessence Int 1993:24:535-42.
29. Kurtzman GM, Silverstein LH. Diagnosis and treatment planning for predictable gingival correction of passive eruption. Pract Proced Aesthet Dent 2008: 20: 103–8.
30. Camargo PM, Melnick PR, Camargo LM. Clinical crown lengthening in the esthetic zone. J Calif Dent Assoc 2007: 35:487–98.
31. Cairo F, Graziani F, Franchi L, Defraia E, Pini Prato GP.Periodontal plastic surgery to improve aesthetics in patientswith altered passive eruption/gummy smile: a case seriesstudy. Int J Dent 2012: 2012: 837658.
32. Ribeiro FV, Hirata DY, Reis AF, Santos VR, Miranda TS,Faveri M, Duarte PM. Open-flap versus flapless estheticcrown lengthening: 12-month clinical outcomes of a randomizedcontrolled clinical trial. J Periodontol 2014: 85:536–44.

JSPIK

Unraveling the Fungal Link in Periodontal Disease

Raji A R[1], Rosamma Joseph Vadakkekuttical[2]

ABSTRACT

Recent paradigm of periodontal diseases emphasize on a more comprehensive dysbiotic – synergistic community to initiate periodontal destruction. Evidences heads up to the importance of cross-kingdom interactions in the progression of periodontitis. Association between Candida albicans and periodontitis has been reported in the literature with varying evidences. Ability of candida species to adapt to adverse host environment, morphological switching, epithelial adhesion and tissue invasion, production of hydrolytic enzymes such as phospholipases and proteinases, synergistic coaggregation with bacteria may contribute to candida colonization and biofilm formation. Aim of this review is to give an insight to the various virulence factors of candida and plausibility of relationship between oral candida carriage and periodontitis by understanding the current literature.

Keywords: Periodontitis, Candida albicans, Morphological switching, virulence factors

Introduction

Oral cavity provides complex, rich and distinct ecologic niches to billions of microbes comprising of bacteria, fungi, viruses and protozoans.[1] Microbial colonization are seen within the teeth, gingival sulcus, tongue, tonsils, hard and soft palate and are differentiated based on the nature of the surface, varying nutrient availability and dynamic occurrence of oxygen limitation. Microbes resides within 3-dimensional communities termed 'biofilms', a syntrophic consortia and results in an oral ecology that is highly diverse in species composition.[2]

This may alter overtime under the influence of environmental factors and leads to transition from high diversity homeostasis to low diversity dysbiosis. Synergistic and antagonistic actions takes place through physical, chemical or metabolic interactions, host or environmental modulations, competition for nutrient or adhesion sites and formation of mixed species biofilms. This signifies that individual bacterias are no longer perceived as 'causative pathogens' of inflammatory diseases but rather an entire microbial community under the influence of specific organisms and/ or conditions tips the balance from homeostasis to destructive inflammation. This underpins the concept of polymicrobial diseases and periodontitis may be one of the most classically defined polymicrobial biofilm mediated disease.[3]

Periodontal diseases are chronic multifactorial inflammatory diseases associated with dysbiotic plaque biofilms and characterized by progressive destruction of tooth-supporting apparatus. It varies in severity from gingivitis to advanced periodontitis with formation of deepened periodontal pockets, and the resorption of alveolar bone.[4] Periodontal disease has a well understood bacterial etiology, but the recent paradigm of periodontitis progression challenges the traditional concept of it being induced by few periopathogens

[1]Junior resident, [2]Professor and Head, Department of Periodontics, Government Dental College, Kozhikode, Kerala, India
Corresponding Author: Dr. Rosamma Joseph Vadakkekuttical, E-mail: drrosammajoseph@gmail.com

such as 'Red complex' bacterias to a more comprehensive dysbiotic- synergistic community. 'Community' is now considered the unit of pathogenicity to approach the polymicrobial etiology of periodontitis.[5] Synergism between pathogenic bacteria and viruses and between bacteria and fungi such as candida albicans have been hypothesized which suggest that bacterial-viral and bacterial-fungal co-infections may contribute to periodontitis.

Concept of Nososymbiocity & Interkingdom interactions

'Nososymbiocity' refers to the potential of an indigenous community to cause disease and is a more accurate term than 'pathogenicity'. Multi-level physical and chemical communication systems are formed among constituent organisms and this functional specialization of community participants, originating from their metabolic co-dependance has shifted our perception from a commensal- pathogen duality to a 'commensal-to-pathogen spectrum'.[6] A commensal which by itself do not contribute to disease but may do so in an interactive multi-species community. Commensal or pathogenic properties are not intrinsic features of an organism but are to be considered within the context of both the microbial community in which they reside and the host immune status. So a microbe act as an accessory pathogen when it is inherently commensal in a particular microenvironment and synergistically enhances the colonization or metabolic activity of other pathogens or as a pathobiont when they exploit the disrupted host homeostasis to flourish and promote inflammatory diseases.

Although around 100 fungal species have been reported to colonize and co-exist within complex biofilm populations,[7] they are often neglected due to their low quantitative contribution within diseased sites. Comparing the enormous size of the fungus (70μm^3) to a bacterium (0.5μm3), they occupy an adequate space of relevant oral surfaces suggesting it to be a significant part of healthy oral ecology and should not be disregarded.[8] Moreover, these fungal scaffolds may provide a shield for the interacting bacteria and protects it from the host immune system.

Candida albicans are the most common commensal and opportunistic fungal pathogen associated with oral lesions with a prevalence of 3 to 48%.[9] Other candida species isolated from the oral cavity includes C. glabrata, C tropicalis, C.parapsilosis, C. krusei and C. dubliniensis.[10] Candida albicans have been isolated from the periodontal pockets in numerous studies and is also correlated with the severity of periodontitis. In dysbiotic microbial communities, virulence refers to any microbial trait that elevates the pathogenicity of community and Candida species possess various virulence factors that includes adhesion to epithelial cells, morphological switching, production of hydrolytic enzymes etc.

a. Epithelial adherence:

Both non-specific (hydrophobic and electrostatic) and specific (protein –protein) interactions plays a role in the adhesion of bacteria to fungi. These are mediated through non-specific hydrophobic surface proteins and specific hyphal wall proteins such as Als3p. Candida albicans can attach to epithelial cells using these proteins through host cell receptors such as EphA2, E-cadherin and via host cell transglutaminases. Electrostatic forces also contribute to affinity between fungi and epithelial cells. Most candida strains showed a significantly higher adherence to gingival epithelial cells compared to gingival fibroblasts and pulmonary fibroblasts in an invitro evaluation[11] but environmental factors such as diet, composition of body fluids may modulate candida adhesion. Such adhesion and biofilm formation on C. albicans protects bacteria from antibiotic challenges and the surviving biofilms are the source of recolonization.

b. Dimorphic switching

C. albicans is a pleiomorphic fungus that exists in three distinct morphological states: yeast cells, pseudohyphae, and filamentous hyphae. The ability of pathogenic fungi to switch between a multicellular hyphal and unicellular yeast growth form is known as dimorphic switching.[12] Changes in environmental condition influenced by local or systemic factors can trigger the yeast form to differentiate into hyphal form. Hyphae have the ability to penetrate and invade host tissues resulting in disease pathogenesis. Studies shows an increased transition to hyphal forms and candidal colonization in periodontal pockets of diabetic subjects.[13] This may be attributed to presence of

inflammatory cytokines and altered immune response in both periodontitis and diabetes.

Hyphal forms exhibits induced endocytosis in which host cell is induced to produce pseudopods that then progressively surround the fungus and finally pull it into the host cell. C. albicans expresses invasins (Als3 and Ssa1), which mediate binding to host epithelial surface proteins (such as cadherins and EGFR). This stimulates recruitment of clathrin, dynamin and cortactin in host cells to the sites where hypha enters the epithelial cells, sequentially inducing the actin cytoskeleton reorganization and provides the force required for fungal internalization.[14]

c. Production of hydrolytic enzymes

Phospholipids and proteins represent the major chemical constituents of the host cell envelope. Invasion of host cells entails penetration and damage of host cell. Candida species secretes hydrolytic enzymes extracellularly such as phospholipases and secreted aspartyl proteinases (SAP). 7 phospholipase genes (PLA, PLB1, PLB2, PLC1, PLC2, PLC3, PLD1) and 10 SAP genes have been identified in C. albicans.[15] Phospholipases cleaves phospholipids, destabilizes host cell membrane resulting in cell lysis.[16] SAPs digest the surface of epithelial cells and provides an entry to cells, helps in nutrient acquisition by hydrolyzing complex proteins to provide nitrogen to cells and are necessary to survive and escape from macrophages by hydrolyzing proteins of immune system.[17]

d. Other factors

Other virulence factors proposed includes its ability to adapt to an adverse host environment by altering pH, oxygen concentration and nutrient availability, hemolysin secretion and synergistic coaggregation or competition with other bacteria.

Mounting evidences suggest that candida has the capacity to interact and influence the behavior of periodontal pathogens. They are facultative anaerobe and can survive in both aerobic and anaerobic conditions. They are able to rapidly deplete oxygen within mixed species environments and may explains why obligate anaerobes and yeasts are observed together. Anaerobes such as F.nucleatum and P.gingivalis are co-isolated with C.albicans in periodontal pockets. P.gingivalis can increase hyphal formation of C.albicans and conversly C.albicans can enhance invasion of gingival epithelial cells by P.gingivalis.

Different morphological states of candida albicans are associated with colonization and growth among which, hyphal form enables biofilm formation. Candida posess several virulence properties that adds to its pathogenic potential. Expressed cell wall adhesins, including the members of the agglutinin-like sequence family (Als) and hyphal wall protein (Hwp1), are crucial for C. albicans attachment to host tissue and for multispecies biofilm formation.[18,19] C. albicans secretes a number of hydrolytic enzymes, such aslipases, esterases, and secreted aspartyl proteinases (SAPs), that affects biofilm formation, tissue invasion, and immune evasion.[17,20] A newly reported candidalysin, a cytolytic peptide toxin secreted by C. albicans hyphae, causes damage to oral epithelial cells by intercalation, permeabilization, and calcium influx, triggers a proinflammatory signaling pathway response; and activates epithelial immunity.[21]

Discussion

Numerous studies in the literature reports the presence of Candida albicans in the subgingival sites of chronic periodontitis patients.[22–24] Urzua et al in 2008[25] observed that C. albicans and C. dubliniensis were capable of colonizing the periodontal pockets in patients with chronic periodontitis, while only C. albicans was identified in the subgingiva of healthy individuals and patients with aggressive periodontitis. Machado et al in 2011[26] conducted a study in chronic periodontitis patients and healthy subjects and observed a higher Candida adherence to epithelial cells in samples isolated from patients with chronic periodontitis. Cannabarro et al in 2013[27] conducted a study in chronic periodontitis patients and healthy subjects and concluded that the presence of severe chronic periodontitis increases the odds of having high densities of subgingival yeast but no difference was observed between moderate periodontitis and healthy groups. Brandilyn et al in 2017[28] compared oral mycobiome of subjects with and without periodontal disease and observed trends of higher abundance of candida in periodontal disease and in those with greater tooth loss.

Jaziya et al in 2019[29] observed a higher candidal

carriage with increased occurrence of periodontal disease among subjects with metabolic syndrome and healthy controls without metabolic syndrome. AS Unniachan et al in 2020[30] in a systematic review of 23 articles assessed the prevalence of Candida species in periodontal diseases and the results were indicative of a positive association between Candida species and periodontal diseases.

While the evidences points to the possible role of C.albicans in periodontal disease, there have been conflicting findings as well. Popova et al in 2014[31] found no correlation between candida species presence and periodontal disease as they observed only low levels of candida strains in deep periodontal pockets. De-La-Torre et al in 2019[32] analyzed oral candida carriage among healthy subjects, moderate and severe chronic periodontitis patients and found that although severe chronic periodontitis patients had higher percentage of candida carriage, there was no statistically significant relation between candida colonization and severity of periodontitis.

Eventhough candida species isolation and hyphal invasion in periodontal tissues are reported, the role of candida in periodontal disease is still controversial. Without doubt, different properties and virulence factors of candida albicans allows them to colonize periodontal pockets. Epithelial adhesion and tissue invasion as well as coaggregation with other microbial species makes it an active participant of inflammatory and destructive process of periodontal diseases. This coaggregation with other bacterias may act as a barrier and hinders access to the anti-microbial molecules or immunoglobulins present in the oral cavity. This might reduce or block the therapeutic action of antibiotics or other chemotherapeutic agents and further clinical studies are required to demonstrate these therapeutic implications.

Conclusion

Fungi and bacteria can interactin physical, chemical and metabolical ways to influence microbial survival, colonization and biofilm formation. Deep pockets results in a change in balance of subgingival microflora and predisposes such sites to greater periodontal destruction. Candida may be more abundant in the oral cavity of individuals with periodontal disease. Detection of candida species in subgingival biofilm is only a first step for connecting fungi and periodontal disease. Long term studies are needed to elucidate whether candidal carriage is a cause or consequence of periodontal disease and thereby unravel the fungal link in periodontal diseases.

References

1. Simon-Soro A, Tomás Carmona I, Cabrera-Rubio R, Catalan M, Nyvad B, Mira A. Microbial Geography of the Oral Cavity. J Dent Res. 2013; 92(7):616-21.
2. Chandki R, Banthia P, Banthia R. Biofilms: A microbial home. J Indian Soc Periodontol. 2011;15(2):111–4.
3. Darveau RP. Periodontitis: a polymicrobial disruption of host homeostasis. Nat Rev Microbiol. 2010;8(7):481–90.
4. Nanci A, Bosshardt DD. Structure of periodontal tissues in health and disease*. Periodontol 2000. 2006;40(1):11–28.
5. Siqueira J, Rôças I. Community as the unit of pathogenicity: An emerging concept as to the microbial pathogenesis of apical periodontitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod. 2009;107(6):870-8.
6. Hajishengallis G, Lamont RJ. Dancing with the Stars: How Choreographed Bacterial Interactions Dictate Nososymbiocity and Give Rise to Keystone Pathogens, Accessory Pathogens, and Pathobionts. Trends Microbiol. 2016;24(6):477–89.
7. Ghannoum MA, Jurevic RJ, Mukherjee PK, Cui F, Sikaroodi M, Naqvi A, et al. Characterization of the Oral Fungal Microbiome (Mycobiome) in Healthy Individuals. PLOS Pathog. 2010;6(1)::e1000713. doi: 10.1371/journal.ppat.1000713.
8. Janus MM, Willems HM, Krom BP. Candida albicans in Multispecies Oral Communities; A Keystone Commensal? In: Imbert C. (eds) Fungal Biofilms and related infections. Advances in Experimental Medicine and Biology, 2016;931:13-20.
9. Arendorf TM, Walker DM. The prevalence and intra-oral distribution of Candida albicans in man. Arch Oral Biol. 1980;25(1):1–10.
10. Byadarahally Raju S, Rajappa S. Isolation and Identification of Candida from the Oral Cavity. ISRN Dent.2011:487921. doi: 10.5402/2011/487921.
11. Nikawa H, Egusa H, Makihira S, Okamoto T, Kurihara H, Shiba H, et al. An in vitro evaluation of the adhesion of Candida species to oral and lung tissue cells. Mycoses. 2006;49(1):14–7.
12. Boyce KJ, Andrianopoulos A. Fungal dimorphism: the switch from hyphae to yeast is a specialized morphogenetic adaptation allowing colonization of a host. FEMS Microbiol Rev. 2015;39(6):797–811.
13. Venkatesan G, Uppoor A, Naik D, Kadkampally D, Maddi A. Oral Candida Carriage and Morphotype Differentiation in Chronic Periodontitis Patients with and without Diabetes in the Indian Sub-Continent. Dent J. 2015;3(4):123–31.
14. Moreno-Ruiz E, Galán-Díez M, Zhu W, Fernández-Ruiz E, d'Enfert C, Filler SG, Cossart P, Veiga E. Candida albicans internalization by host cells is mediated by a clathrin-dependent mechanism. Cell Microbiol. 2009;11(8):1179-89.
15. Samaranayake YH, Dassanayake RS, Cheung BPK, Jayatilake JAMS, Yeung KWS, Yau JYY, et al. Differential phospholipase gene expression by Candida albicans in artificial media and cultured human oral epithelium. APMIS. 2006;114(12):857–66.
16. Ghannoum MA. Potential Role of Phospholipases in Virulence and Fungal Pathogenesis. Clin Microbiol Rev. 2000;13(1):122–43.
17. Naglik J, Albrecht A, Bader O, Hube B. Candida albicans proteinases and host/pathogen interactions. Cell Microbiol. 2004;6(10):915–26.

18. Hoyer LL, Cota E. Candida albicans Agglutinin-Like Sequence (Als) Family Vignettes: A Review of Als Protein Structure and Function. Front Microbiol. 2016;7:280. doi: 10.3389/fmicb.2016.00280.
19. Nobile CJ, Nett JE, Andes DR, Mitchell AP. Function of Candida albicans Adhesin Hwp1 in Biofilm Formation. Eukaryot Cell. 2006;5(10):1604–10.
20. Lindsay C. Dutton, Howard F. Jenkinson, Richard J. Lamont, Angela H. Nobbs, Role of Candida albicans secreted aspartyl protease Sap9 in interkingdom biofilm formation.Pathogens and Disease.2016;74(3), https://doi.org/10.1093/femspd/ftw005
21. Sztukowska MN, Dutton LC, Delaney C, Ramsdale M, Ramage G, Jenkinson HF, Nobbs AH, Lamont RJ. Community Development between Porphyromonas gingivalis and Candida albicans Mediated by InlJ and Als3. mBio. 2018;9(2):e00202-18.
22. Järvensivu A, Hietanen J, Rautemaa R, Sorsa T, Richardson M. Candida yeasts in chronic periodontitis tissues and subgingival microbial biofilms in vivo. Oral Dis. 2004;10(2):106–12.
23. Reynaud AH, Nygaard-Østby B, Bøygard GK, Eribe ER, Olsen I, Gjermo P. Yeasts in periodontal pockets. J Clin Periodontol. 2001;28(9):860–4.
24. Slots J, Rams TE, Listgarten MA. Yeasts, enteric rods and pseudomonads in the subgingival flora of severe adult periodontitis. Oral Microbiol Immunol. 1988;3(2):47–52.
25. Urzúa B, Hermosilla G, Gamonal J, Morales-Bozo I, Canals M, Barahona S, et al. Yeast diversity in the oral microbiota of subjects with periodontitis: Candida albicans and Candida dubliniensis colonize the periodontal pockets. Med Mycol. 2008;46(8):783–93.
26. Machado AG, Komiyama EY, Dos Santos SSF, Jorge AOC, Brighenti FL, Koga-Ito CY. In vitro adherence of Candida albicans isolated from patients with chronic periodontitis. J Appl Oral Sci. 2011;19(4):384–7.
27. Canabarro A, Valle C, Farias MR, Santos FB, Lazera M, Wanke B. Association of subgingival colonization of Candida albicans and other yeasts with severity of chronic periodontitis. J Periodontal Res. 2013;48(4):428-32.
28. Peters BA, Wu J, Hayes RB, Ahn J. The oral fungal mycobiome: characteristics and relation to periodontitis in a pilot study. BMC Microbiol. 2017;17(1):157. doi: 10.1186/s12866-017-1064-9.
29. Jaziya Banu, Rosamma Joseph Vadakkekuttical and Chandni Radhakrishnan. Association of Periodontal Disease, Oral Candidal Carriage, and Salivary pH in Patients with MetabolicSyndrom. EC Dental Science. 2019;18(8): 1893-1901.
30. Suresh A, Nisha K, Sethuraman S. Association between Candida species and periodontal disease: A systematic review. Curr Med Mycol. 2020;6(2):63–8.
31. Popova C, Dosseva-Panova V, Kisselova-Yaneva A, Panov VE. Subgingival microbiota in severe chronic periodontitis. J IMAB - Annu Proceeding Sci Pap. 2014;20(3):554–7.
32. De-La-Torre J, Quindós G, Marcos-Arias C, Marichalar-Mendia X, Gainza ML, Eraso E, et al. Oral Candida colonization in patients with chronic periodontitis. Is there any relationship? Rev Iberoam Micol. 2018;35(3):134–9.

JSPIK

Management of Gingival Enlargement and Periodontitis in a Patient with Hypertension

Beena M.P[1], Mahesh Narayanan[2], Shabeer Ahamed[3], Haris Muhammed[4], Ramya Ganesh[5], Sisira Ramachandran[6]

ABSTRACT

Gingival enlargement or gingival overgrowth is the preferred term for all medication-related gingival lesions previously termed gingival hyperplasia or gingival hypertrophy. Anticonvulsants, antihypertensive calcium channel blockers and immunosuppressants are the three main classes of drugs known to cause drug-induced gingival hypertrophy or hyperplasia. Patients with gingival enlargement are concerned with the aesthetic appearance but clinicians frequently encounter periodontitis also in such individuals which can be due to poor oral hygiene or other confounding factors. The management requires a proper understanding of the case and a multidisciplinary approach including medical, surgical and supportive care. A case of amlodipine-induced gingival hyperplasia in a 46-year-old hypertensive presented which was managed with periodontal flap surgery and gingivectomy.

Key words: Gingival Hyperplasia, Chronic periodontitis, Internal Bevel Gingivectomy

Introduction

Gingival enlargement is one of the side effects associated with the administration of several drugs. Currently, more than 20 drugs are associated with gingival enlargement. Drugs having side effect of gingival enlargement can be broadly divided into three categories: anticonvulsants, calcium channel blockers and immunosuppressants.[1] The prevalence of drug induced gingival overgrowth (DIGO) substantially varies for different medications and among studies. Three drugs associated with DIGO are phenytoin, nifedipine, and cyclosporine. About 20 other medications have been linked to DIGO. An estimated 30% to 80% of the patients using these medications are at risk for overgrowth.[2,3] Calcium channel blockers have been reported to be associated with gingival hyperplasia since 1984.[4] Many of the calcium channel blockers used as antihypertensive drugs have been implicated in causing gingival enlargement. Amlodipineis a comparatively new dihydropyridine calcium channel blocker that is used in the management of both hypertension and angina.[5]

Amlodipine is dihydropyridine derivative used as antihypertensive drug having longer action and comparatively lesser side effect than Nifedipine but even this drug results in gingival enlargement but the reported incidence is limited.[1] Jorgensen 1997 reported a prevalence of 3.3%; Ellis et al. 1999 reported 1.7% for Amlodipine induced gingival overgrowth (GO).[5,6] Gingival enlargement produces aesthetic changes and clinical symptoms including pain, tenderness, bleeding, speech disturbances, abnormal tooth movement, dental occlusion problems, enhancement of caries

[1]Post Graduate student, [2]Professor and Head, [3]Professor, [4]Reader, [5]Senior Lecturer, [6]Post Graduate Student, Department of Periodontics, Malabar Dental College & Research Centre, Edappal, Malappuram, 679 578, Kerala

development and periodontal disorders.[7]

This report presents a case of severe periodontitis with generalized gingival enlargement in a 46-year-old male who was a known case of hypertension and was managed by gingivectomy and flap surgery

Case Report

A 45-year-old male patient was referred to the Department of Periodontics, Malabar Dental College & Research Centre, with the chief complaint of loose tooth in the lower right back region for 6 months. He was referred from a general dentist after completion of oral prophylaxis. The patient also complained of swollen gums which bled on brushing and bad smell in the mouth. Medical history revealed that patient was diagnosed with hypertension since the age of 35 and was on medication since then. He was also having hypothyroidism for past 2 years. He was taking T Amlodac 5 mg BD and T-Tazloc 40 mg BD, T -Aquazide 12.5 mg OD and T- Thyronorm 50 mg OD for hypertension and hypothyroidism respectively.

Intraoral examination

The oral hygiene status was not satisfactory. Clinical examination revealed generalized Grade III gingival enlargement involving the interdental papillae and the marginal and attached gingiva. Gingiva was reddish pink in colour, rounded, and bulbous contour, smooth surface texture covering most of the clinical crowns of teeth. The enlargement was more marked in the upper right quadrant. According to the patient, the swelling began in the upper back jaw region and gradually became diffuse involving all the gums [Fig- 1] with bleeding on brushing and pain. On palpation, it had a firm-fibrotic consistency. There were deep pockets of 8–10 mm in several teeth and tooth 47 shows grade 3 mobility. Russel's periodontal index score was 5, suggestive of terminal periodontal disease.

Investigations

Panoramic radiograph revealed generalized bone loss that was severe around molars [Figure 2]. Orthopantomogram (OPG) revealed vertical bone loss in relation to 16, 17, 26, 27, 36, 37, 46 and 47. Blood tests included complete hemogram and bleeding time, clotting time, and also TSH levels which was within normal limits and blood pressure was also monitored.

Diagnosis

A diagnosis of Generalised Chronic Periodontitis and drug-induced gingival enlargement was made, and the patient was referred back to his Physician to substitute the antihypertensive drug Amlodipine that is a known cause for gingival enlargement. The orthopantomogram shows bilaterally impacted supernumerary teeth in relation to 34 and 44.

Treatment plan

Complete periodontal management was planned for the patient that included Phase 1 therapy, maintenance therapy, surgical therapy, restorative therapy followed by supportive periodontal therapy. The treatment planned was permanent restoration of maxillary teeth 11 and 21 and prosthetic rehabilitation of lost 47.

Phase I therapy

Patient education and motivation about maintaining proper oral hygiene, demonstration of professional tooth brushing was done. Multiple scaling and root planing sessions were undertaken along with subgingival irrigation of 0.2% chlorhexidine for the inflammatory component to subside. Extraction of 47 and

Figure 1 – PreOperative Pictures

Figure -2 Orthopantomograph showing generalised alveolar bone loss.

restorative treatment of abraded teeth, along with temporary crown of 11 and 21 was done. Although we had requested for a change of drug, the physician asked to continue the T-Amlodac and T- Tazloc because of fluctuating blood pressure levels but the frequency of intake was changed for T -Amlodac 5 mg OD and T-Tazloc 40 mg OD. Once the patient's blood pressure stabilised, surgical therapy was planned since the enlargement persisted along with periodontitis.

Surgical phase

Internal bevel gingivectomy was done to remove the enlarged gingiva [Fig -4] and periodontal flap was raised to remove granulation tissue and subgingival calculus, and root planing was done and the flap was sutured. Splinting was also done in relation to 12 and 13 to prevent anticipated mobility. Conventional flap surgery was performed in relation to 34-38 region and bone substitutes, osseograft (DMBM- XENOGRAFT) was placed into vertical bone defects (35,36 region) before suturing.

He was advised to take analgesics, antibiotics (tablet Paracetamol 500 mg BD, capsule Amoxicillin 500 mg TDS × 5 days) and 0.12% Chlorhexidine mouthwash. The patient was recalled after 1 week for suture removal [Fig 6].

Follow-up

The patient was kept on regular weekly recall. Healing was uneventful, and the patient was able to maintain meticulous plaque control after 6 weeks using toothbrush and dentifrice, interdental brushes, 0.12% chlorhexidine mouthwash, and warm saline rinses.

Histo pathological Examination

Surgically excised gingival specimens were sent for histopathology. H&E stained section shows, stratified, squamous hyperplastic epithelium with long Rete ridges. Connective tissue is moderately collagenous, with chronic inflammatory cell infiltration. This was suggestive of gingival hyperplasia [Fig -8].

Discussion

The American Academy of Periodontology defined drug-influenced gingival overgrowth (DIGO) as "an increase in size of the gingiva resulting in whole or in part from systemic drug use.[8]

Three classes of drugs are primarily implicated as causative of gingival hyperplasia, these are anticonvulsants, the immunosuppressant cyclosporine and

Figure 3 - 1 Month After SRP

Figure- 4 Internal Bevel Incision

Figure – 5 Post-Operative Clinical picture

Figure 6 - After suture removal

calcium channel blockers (CCB). Amongst the CCB, the commonest agent associated with gingival enlargement is nifedipine, though other agents implicated are Verapamil, Felodipine, Nitrendipine, Diltiazem, and Amlodipine.[9] Calcium channel blockers have been reported to be associated with gingival hyperplasia since 1984. The vast majority of published reports deal with the drug Nifedipine. Amlodipine, a newer calcium channel blocker, has also been associated with gingival hyperplasia.[4]

Amlodipine is a third generation dihydropyridine calcium antagonist, which has a mode of action pharmacodynamically comparable to Nifedipine. However, Amlodipine has a unique physiochemical profile, which is characterized by near complete absorption, late-peak plasma concentrations, high bioavailability and slow hepatic biodegradation.[8] Nifedipine and Amlodipine also have different pharmacokinetic profiles. Amlodipine has a long half-life (T1/2 = 34 hours) and a high volume of distribution (21 L/kg). The respective figures for nifedipine are 7.5 hours and 0.78 L/kg respectively. These differences imply that at any one time, the majority of Amlodipine will be tissue bound (and hence "inactive") rather than circulating freely in the blood. It has been suggested that a plasma threshold may exist above which drug-induced gingival changes are initiated.[10]

The common mechanism of action at the cellular level of all these three categories of dissimilar drugs appears to be inhibition of cation influx, particularly sodium and calcium ions. Clinicians believe that gingival overgrowth is multifactorial. Bacterial plaque appears to be a contributory factor and the severity of gingival overgrowth is believed to be directly proportional to the degree of plaque buildup and plaque-induced inflammation. Decreased cation dependant folic acid (FA) active transport within gingival fibroblasts causes reduced FA uptake by the cells, causing changes in the metabolism of matrix metalloproteinases and inability to activate collagenase. This results in accumulation of connective tissue and collagen due to lack of collagenase causing DIGO.[11]

The most effective treatment of drug-related gingival enlargement is withdrawal or substitution of medication. When this treatment approach is taken, it may take from 1 to 8 weeks for resolution of gingival lesions. Unfortunately, not all patients respond to this mode of treatment, especially those with longstanding gingival lesions.[13] The treatment options for drug-induced gingival enlargement should be based on the medication being used and the clinical presentation of each particular case. Firstly, consideration should be given to the possibility of discontinuing the drug or of changing medication. Either one of those scenarios should be examined in conjunction with the patient's physician. In this case although an attempt was made to change the drug it could not be possible because of the elevated blood pressure. The clinician should also emphasize plaque control as the first step in the treatment of drug-induced gingival enlargement.

Gingival enlargement may persist, despite dose adjustments and good plaque control. In this case we chose internal bevel gingivectomy and flap elevation because of the need to debride intrabony defects along with excision of hyperplastic gingival tissues.

The clinician's decision to choose gingivectomy alone (either external or internal bevel) or gingivectomy combined with flap surgery must be made on a case-by-case basis and should take into consideration the extent, area to be surgically treated, the presence

Figure - 7 2 -weeks after suture removal

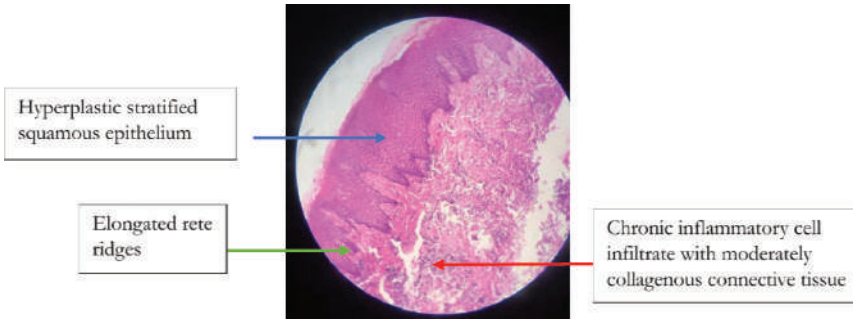


Figure - 8 Histopathologic view

of periodontitis, the presence of osseous defects combined with the gingival enlargement lesions and the position of the bases of the pockets in relation to the existing mucogingival junction.[12]

The classical surgical approach has been the external bevel gingivectomy. However, a total or partial internal gingivectomy approach has been suggested as an alternative. This more technically demanding approach has the benefit of limiting the large denuded connective tissue wound that results from the external gingivectomy, thereby minimizing postoperative pain and bleeding.[13]

Conclusion

Stringent maintenance of oral hygiene, switchover to alternative drugs and surgical therapy if required, remains the mainstay of available treatment modalities. Better results were obtained where drug substitution along with oral prophylaxis were followed. In the susceptible patient, drug-associated gingival enlargement may be ameliorated, but not prevented, by elimination of local factors, meticulous plaque control, and regular periodontal maintenance therapy.

The periodontal flap should be used in areas in which the alveolar bone needs to be accessed for osseous recontouring purposes and in areas with limited keratinized tissue. Despite being technically more demanding, healing following the periodontal flap less uncomfortable for the patient and there is less chance of postoperative haemorrhage.

A 3-month interval for periodontal maintenance therapy has been recommended for patients taking drugs associated with gingival enlargement. Each recall appointment should include detailed oral hygiene instructions and complete periodontal prophylaxis. So, cooperative teamwork between the patient, his physician, and the dental health care professional is mandatory to minimize and successfully treat such unwanted side effects of drugs. Newer molecular approaches are needed to clearly establish the pathogenesis of gingival overgrowth and to provide novel information for the design of future preventive and therapeutic modalities.

References

1. Amit Kumar Srivastava, DebabrataKundu, PrasantaBandyopadhyay, and Asit Kumar Pal. Management of amlodipine-induced gingival enlargement: Series of three cases. J Indian SocPeriodontol 2010; 14(4): 279–81.
2. Seymour RA, Ellis JS, Thomason JM. Risk factors for drug induced gingival overgrowth. JClin Periodontol.2000;27:217–23.
3. Trackman PC, Kantarci A. Molecular and clinical aspects of drug-induced gingival overgrowth. J Dent Res. 2015; 94:540–6.
4. M.G Jorgensen, Prevalence of amlodipine-related gingival hyperplasia. J Periodontol.1997;68(7):676-8.
5. Seymour RA, Ellis JS, Thomason JM, Monkman S and Idle JR: Amlodipine induced gingival overgrowth. J Clin Periodontol 1994; 21: 281-3.
6. SaumiyaGopal, Rosamma Joseph, VediyeraChandroth Santhosh, Vadakkedath Venugopal Harish Kumar, Shiny Joseph, Abhijeet-RajendraShete. Hospital-based study. J Periodontol 1997; 68:676-8.
7. Lluis Brunet, l Jaume Miranda, Magi Farre, Leonardo Berini and CarlesMendietal, Gingival Enlargement Induced by Drugs. Drug Safety 1996; 15 (3): 219-31.
8. SuhaniGoel, ShivjotChhina, Sachit A. Arora Drug-Induced Gingival Enlargement: A Case Report. jdentlasers.org, September 28, 2020, IP: 27.62.4.153.
9. Vineet Bhatia, Ajay Mittal, Ashok K Parida, Rajesh Talwar, Upendra Kaul. Amlodipine induced gingival hyperplasia: A rare entity International Journal of Cardiology 2007; 122: e23–4.
10. Ellis JS, Seymour RA, Steele JG, Robertson P, Butler TJ, Thomason JM. Prevalence of gingival overgrowth induced by calcium channel blockers: a community□ based study. Journal of Periodontology. 1999;70(1):63-7.
11. SujataTungare; Arati G. Paranjpe. Drug Induced Gingival Overgrowth (DIGO). Treasure Island (FL): Stat Pearls Publishing; 2020 Jan.
12. Paulo M. Camargo, Philip R. Melnick, Flavia Q. M. Pirih, Rodrigo Lagos & Henry H. Takei. Treatment of drug-induced gingival enlargement: aesthetic and functional considerations Periodontology 2000, 2001;27: 131–8.
13. Informational Paper Drug-Associated Gingival Enlargement J Periodontol 2004; 75:1424-31.

JSPIK

Management of Palatogingival Groove- A Case Report

Lakshmy M.[1], Mahesh Narayanan[2], Shabeer Ahamed[3], Jeethu John Jerry[4], Vidhya Vishnu[5]

ABSTRACT

Palatogingival grooves are developmental malformations quite notorious for precipitating endodontic - periodontal lesions. Owing to their inconspicuous occurrence, funnel-shaped morphology and variable extent on tooth root, they promote adherence of plaque and bacteria to levels significant for the development of pathology. This article presents a case report of a maxillary lateral incisor with a palatogingival groove extending up to the middle third of the root. Despite the poor prognosis, management of the defect was successfully carried out with combined endodontic and periodontal therapy

Key words: Palatogingival groove, classification, endodontic lesion, periodontitis, case report.

Introduction

Palatogingival groove(PGG) or radicular lingual groove (RLG) is a developmental anomaly that occurs as developmental infoldings of the inner enamel epithelium and Hertwig's epithelial root sheath (HERS), involving primarily maxillary lateral incisors. RLG formation presumably represents an aborted attempt to represent an additional root.[1] According to Kogon's investigations, the groove can be found in cingulum, followed by lateral fossa, cementoenamel junction, and root in decreasing order. He also found that most common location is in the midpalatal area of lingual surface followed by distal and then mesial.[2] The tube-like channel serves as an ideal locus for plaque and calculus accumulation, thus acting as a secondary local etiologic factor, encouraging the development of periodontal disease. In rare instances, these grooves present a diagnostic and treatment dilemma. The few references in dental literature dealing with this anomaly trace the relationship of these defects to severe, localized and often hopeless periodontal disease.[3]

The presence of RLG does not always indicate the development of pathology. In most cases the epithelial attachment remains intact across the groove. Once the attachment is breached, a self-containing pocket forms along the length of the groove or by gingival irritation secondary to microbial plaque retention. The attachment may be breached due to endodontic involvement. Inflammation can progress from an apical lesion coronally along the groove, causing a primary endodontic/secondary periodontic lesion. If inflammation spreads to the pulp through defects in the groove or involvement of apex, a primary periodontic/secondary endodontic lesion develops.[4]

Lee et al (1968) was the first to report an association between palatoradicular grooves and localized periodontitis. They described eleven cases where unilateral and bilateral defects in lateral incisors were associated with moderate / severe periodontal disease.[3]

Two classifications were independently put for-

[1]Post graduate student, [2]Professor and Head, [3]Professor, [4]Reader, [5]Senior Lecturer, Department of Periodontics, Malabar Dental College & Research Centre, Edappal, Malappuram - 679 578, Kerala, Corresponding author; Dr Mahesh Narayanan
E mail : drmaheshn@gmail.com

ward by Goon et al. and Gu YG. depending on the type and radicular extent of the groove.[5,6]

Palatogingival groove classification

1. Classification by Goon et al

Type I	Groove not beyond coronal third of root Short Groove
Type II	Groove long extends beyond coronal third Shallow Groove with normal canal
Type III	Groove long extends beyond coronal third Deep Groove with complex canal system

2. Classification by GU YU

Mild	Groove terminating at CEJ or immediately after Gentle depressions
Moderate	Extending apically for some distance along root Shallow or fissured
Complex	Extends entire root length or separates a accessory root Deep invaginated defect

Although these grooves are of some curiosity to morphologists, they have an immediate concern to the dentist and his patient, as food debris, plaque and calculus may collect in the depths of groove, initiating gingivitis and eventually periodontitis.[3] Here is a case report to present the detrimental effects of the presence of a palatogingival groove in lateral incisor and its clinical management effectively.

Case Report

A 32-year-old male patient reported to the Department of Periodontology and Implantology of Malabar Dental College and Research Center, Edappal, Kerala, India, with a chief complaint of pus discharge and pain in the right upper lateral incisors. On clinical examination, a localized gingival inflammation was present with soft edematous tissue with the accumulation of plaque and calculus.

Clinical examination revealed mobility (Grade II), probing depths of > 7 mm circumscribing the involved teeth with associated clinical attachment loss. Palatal examination revealed the presence of a palatogingival groove in relation to the tooth#12 (figure 1).

Radiographic examination revealed the presence of radiolucency in the periapical region which necessitated primary endodontic treatment, followed by open flap debridement (figure 2).

Splinting was done for the upper anteriors for tooth stability in #11and #12. After completion of root canal (figure 3), periodontal flap surgery was planned.

Under local anesthesia, a papilla preservation flap was elevated from the buccal aspect to gain access to the PGG (figure 4).

On elevating the flap, thicksubgingival calculus was seen (figure 5), Scaling and root planning was performed over the groove to eliminate calculus and debris. The granulation tissue was curetted out using Gracey curette numbers 1, 2 [Hu-Friedy Manufacturing Co., Chicago, USA] (figure 5).

After thorough scaling and root planning, the PGG was sealed with glass ionomer cement. Sealed PGG with GIC (figure 6).

The defect was filled with bone graft (osseograft), the flap was replaced and sutures were given (figure 7,8). Post operative follow up was also done (figure 9-11).

Discussion

Numerous authors have reported and discussed the clinical implications of palatogingival groove or radicular lingual groove which is a developmental

Figure 1

Figure 2

Figure 3

Figure 4

anomaly of maxillary anterior teeth that occurs due to infolding of inner enamel epithelium and Hertwig's epithelial root sheath. This groove is more common in maxillary lateral incisors, and the most common location is midpalatal area of lingual surface followed by distal and then mesial. The presence of this groove in maxillary incisor teeth has been implicated as an initiating factor in localized gingivitis and periodontitis eventually leading to loss of periodontal attachment. Whenever these grooves extend to apical third of root, it results in poor prognosis for retention of the tooth. Displacement of cementoenamel junction, extension of enamel in the groove has been suggested as a possible factor in the progression of localized periodontal disease.[2,7]

Treatment depends on the extent, depth and direction of these grooves. Closed debridement coupled with odontoplasty should generally be successful for shallow defects that do not extend down the length of the root for any great distance. Jeng et al[8] in 1992 treated an osseous lesion associated with severe palatoradicular groove. However, open flap debridement becomes mandatory for deeper and more tortuous grooves. Deep grooves that terminate further down the root or on the lateral surface may indicate a poor or hopeless prognosis. Anderegget al[9] have treated 10 cases of palatogingival groove involved periodontitis with open flap debridement and ePTFE membrane and has showed improved results.

Similar to other previous reports our case report also shows successful treatment of palatogingival grooves using endodontic treatment,open flap debridement and management of osseous defect with demineralised bone grafts. It is thus obvious that the practising dentist should be aware of such palatogingival/palatoradicular grooves and when detected, treat them adequately with an interdisciplinary approach so that they do not pave the way for the future tissue destruction and ultimately tooth loss.

Conclusion

A palatogingival groove is a hazard for periodontal as well as endodontal problems. Thorough clinical examination of the lingual surface of incisors should be done as a part of daily routine check up. If their presence is suspected, they have to be restored to prevent from subsequent complications; or subjected to regular prophylaxis and the concerned tooth kept under constant reevaluation.

Figure 5

Figure 6

Figure 7

Figure 8

Figure 9
Post Operative Picture after 1 week

Figure 10 & 11 Post Operative Picture After 1 Year

References

1. Simon JH, Glick DH, Frank AL. Predictable endodontic and periodontic failures as a result of radicular anomalies. Oral Surgery, Oral Medicine, Oral Pathology. 1971 Jun 1;31(6):823-6.
2. Kogon SL. The prevalence, location and conformation of palato-radicular grooves in maxillary incisors. Journal of periodontology. 1986 Apr; 57(4):231-4.
3. Bose BB, Sudarsan S. Palatogingival groove-An added dimension in the etiology of localised periodontitis. Int J Dent Sci Res. 2013;1(1):5-7.
4. Gupta KK, Srivastava A, Srivastava S, Gupta J. Palatogingival groove - a silent killer: Treatment of an osseous defect due to it. J Indian SocPeriodontol 2011;15:16972.
5. Goon WW, Carpenter WM, Brace NM, Ahlfeld RJ. Complex facial radicular groove in a maxillary lateral incisor. Journal of endodontics. 1991 May 1;17(5):244-8.
6. Gu YC. A micro-computed tomographic analysis of maxillary lateral incisors with radicular grooves. J Endod 2011;37:789-92.
7. Estrela C, Lopes HP, Pecora JD. Radicular grooves in maxillary lateral incisor: case report.Brazilian Dental Journal.1995 6(2): 143-6.
8. Jeng JH, Jackson Lu HK, Hou LT. Treatment of an osseous lesion associated with a severe palato-radicular groove: a case report. Journal of periodontology. 1992 Aug;63(8):708-12.
9. Anderegg CR, Metzler DG. Treatment of the palato-gingival groove with guided tissue regeneration. Report of 10 cases. Journal of periodontology. 1993 Jan;64(1):72-4.

JSPIK

Healing Outcomes after Gingival Depigmentation With Scalpel and Diode Laser - A Split Mouth Study

Karishma Ashok[1], Amitha Ramesh[2], Biju Thomas[3], Rahul Bhandary[2]

ABSTRACT

Background: Pigmentation of gingiva is a common concern for patients, especially when it is associated with excessive gingival display or a high smile line. Although hyperpigmentation of gingiva does not pose as a medical concern but it can lead to psychological concerns in some people, particularly if the gingiva is visible during speech and smiling (high lip line). Various surgical techniques have been suggested for depigmentation but healing outcomes vary with each procedure.

Materials and Methods: In the current case series, 5 cases of hyperpigmentation are reported which were treated using 2 techniques ie scalpel and diode LASER, in a split mouth design.

Results: The postoperative bleeding, pain, as well as discomfort was lesser in case of LASER treatment as compared to scalpel method; however, healing was better in the latter.

Conclusion: Majority of the patients in this case report showed better compliance to LASER therapy.

Keywords: Gingiva, Depigmentation, LASER, Scalpel

Introduction

Esthetic dentistry has been gaining importance over the time with a marked increase in the awareness about dental treatments. Clinicians are therefore, faced with the challenge of achieving gingival aesthetics along with function. Gingival hyperpigmentation is one such aesthetic concern especially in patients with high smile line.[1] The normal gingival colour is considered to be coral pink, with varied melanin deposition. The patterns of distribution of oral pigmentation vary significantly between different individuals and even within the same individual's oral mucosa.[2] Though melanin pigmentation may not be medically significant,[3] it may have psychological effects on patients and therefore require to be treated. Several procedures have been developed for depigmentation of gingiva, for example epithelial abrasion, free gingival graft, gingivectomy, cryosurgery, acellular dermal matrix allograft, electrosurgery, LASER surgery, etc. 4 Thus, the aim of this case series was to clinically evaluate the effectiveness of two different treatment modalities (Scalpel method, LASER surgery) for gingival depigmentation in five patients in a split mouth study design.

CASE 1

A female patient aged 22 years, was referred to the Department of Periodontics, with the chief complaint of dark gums. On clinical examination, the gingival mucosa revealed heavy pigmentation and a high smile line from canine to canine in the form of a band extending from marginal gingiva to mucogingival junction. Medical history was non-contributory.

[1]Post graduate student, [2]Professor, [3]Professor and Head, Department of Periodontics, NITTE (Deemed to be University), A B Shetty Memorial Institute of Dental Sciences, Mangaluru. Corresponding author: Prof (Dr) Amitha Ramesh, E-mail: amitharamesh71@yahoo.in

A split mouth technique, with LASER on one side and scalpel on the other was planned. The patient was explained the procedure and consent was taken prior to the procedure.

Following local anaesthesia (Lignocaine Hydrochloride Gel- Lox 2% jelly for 5 minutes) maxillary anterior gingiva from 13 to 11 was depigmented using a scraping technique with surgical blade no 15. The pigmented epithelium was removed with scalpel along with a layer of connective tissue. Digital pressure was applied with sterile gauze, soaked in local anesthetic agent. Saline irrigation was done followed by placement of a periodontal dressing on the exposed area.

The maxillary anterior gingiva from 21 to 24 was depigmented by using AMD PICASSO DIODELASER at 2.5-Watt, pulse mode. To fulfill the FDA LASER safety rules, protective eye glasses were worn by the patient and the staff. Following the application of topical anesthetic gel to the surgical field, tip of the LASER unit was used with small brush like strokes back and forth with gradual progression to remove the tissue. After LASER ablation, any left-out tissue tags were removed using sterile gauze soaked in saline. Periodontal dressing was not given on these sites.

At the 1-week recall, wound healing was complete on the scalpel side whereas LASER side took 10-12 days to heal completely. Healing was uneventful in both cases. However, patient experienced mild discomfort and pain on the scalpel treated site for 2-3 days postoperatively. Currently, the patient is under regular follow up and recurrence was not noted on either side..

CASE 2

A female patient, 19 years of age, reported to the department, with the chief complaint of dark gums. On clinical examination, heavily pigmented gingiva with brownish black pigmentation in both maxillary and mandibular arches was noted. Treatment protocol was maintained similar as case 1 with LASER on 1st quadrant (11-14) and scalpel on 2nd quadrant (21-24). 1 month post-operatively, recurrence of pigmentation was not observed.

CASE 3

A 25 year old female patient, reported with the chief complaint of dark gums that are visible on smiling. On clinical examination, the gingival mucosa revealed heavy pigmentation and a high smile line. Treatment protocol was maintained similar as case 1 was undertaken with scalpel on 1st quadrant (11-14) and LASER on 2nd quadrant (21-24). 1- month post-operative assessment of these patients showed no recurrence of gingival pigmentation.

Figure 1a Figure 1b Figure 1c

Figure 2a Figure 2b Figure 2c

CASE 4

A female patient, 23 years old, reported with the chief complaint of dark gums to the Department of Periodontics. On clinical examination, the gingival mucosa revealed diffuse pigmentation. Treatment protocol was maintained similar as case 1 was undertaken with LASER on 1st quadrant (11-14) and scalpel on 2nd quadrant (21-24). 1- month post-operative assessment of these patients showed no recurrence of gingival pigmentation.

CASE 5

A 22 year old, male patient, reported with the chief complaint of dark coloured gums that are visible on smiling. On clinical examination, the gingival mucosa revealed heavy pigmentation and a high smile line. Treatment protocol was maintained similar as case 1 was undertaken with scalpel on 1st quadrant (11-14) and LASER on 2nd quadrant (21-24). 1- month post-operative assessment of these patients showed no recurrence of gingival pigmentation.

Patients were asked about level of discomfort on a Verbal Pain Intensity Scale as no pain,mild, moderate, severe, very severe and worst possible pain. Patients reported mild discomfort on sites treated with laser as compared to moderate discomfort on scalpel sites.

Discussion

Two types of clinical LASERs are currently in use in dentistry: soft tissue LASERs and hard tissue LASERs. Soft tissue LASER applications include frenectomies, incisional and excisional biopsies, gingivectomies, gingivoplasty, de-epithelization, soft tissue tuberosity reductions, operculectomy, coagulation of graft donor sites, and crown lengthening procedures[5].

Wigdor et al in their study in 1995, stated that LASER has the following advantages over surgical technique[5]

Figure 3a

Figure 3b

Figure 3c

Figure 4a

Figure 4b

Figure 4c

Figure 5a

Figure 5b

Figure 5c

1. Dry, bloodless surgical field
2. Sterile surgical site
3. Reduced mechanical trauma
4. Minimal or no post-op swelling and pain

Melanin is produced by specialized pigment cells in the gingiva called melanocytes. They are located primarily in the basal layer of the epithelium. Melanocytes have a reproductive self-maintaining system of cells. When locally depleted, they repopulate.

The wound healing takes place by proliferation of cells present along the periphery of the wound. The mechanism of repigmentation is not understood, but according to the migration theory, active melanocytes from the adjacent pigmented tissues migrate to treated areas, causingre-pigmentation. Hence, it is necessary to remove the entire epithelium.[6]

Re-pigmentation was reported with nearly all methods. Bur abrasion has the highest rate of re-pigmentation by 8.89%, followed by laser 1.16%, then electrosurgery 0.74%,then cryosurgery 0.32%, and then diode laser 0.19%, while re-pigmentation rate following scalpel technique may occur after 7 years.[7]

In the scalpel technique,gingival epithelium is removed along with a layer of the underlying connective tissue. The denuded tissue heals by secondary intention. The observations by Procaccini et al that structures containing melanin are affected whereas non-melanised melanosomes are spared, demonstrates that diode laser irradiation is absorbed by melanin.[8] Since diode lasers are absorbed by dark substances such as haemoglobin, their in-depth propagation into tissue is related to the wave length employed and the coefficient of absorption of the same tissue.[9]

The disadvantage of laser systems is represented by histologically evident thermal destruction around the laser beam incision and thermal damage that may range from a transient heating to protein denaturation, water evaporation, carbonization, or burning. Wavelength of the laser, power setting (watts), continuous/pulsed mode, pulse duration, pulse frequency, and exposure time are important laser parameters governing the extent of thermal injury to the tissues[9].

Scalpel wounds, in contrast, do not cause any thermal damage but allow extravasation of blood and lymph, causing a more marked inflammatory response with resultant swelling and formation of a scab.[10]

On clinical examination, at low power settings and using pulse mode in minor surgical procedures, laser therapy showed indisputable advantages in comparison to traditional scalpel technique. This reduced the pronounced changes that occur due to thermal damage and also favoured healing. However, rate of healing was almost the same in both the cases. Patients were recalled at 1 week and 1 month following depigmentation procedure for assessing re-pigmentation at which there were no signs of recurrence. Further follow up is required to assess difference in recurrence of pigmentation between the two treatment modalities

The current case series was in accordance with the aforementioned findings. Majority of the patients in this case report showed better compliance to LASER therapy and also reported of post-operative pain and discomfort on the sites treated with scalpel.

To conclude, with the use of LASER, there was minimal bleeding, post-operative pain and discomfort unlike surgical scraping or partial thickness flap. LASERs were beneficial for patients with anxiety as it requires only topical anaesthesia. It also significantly reduced chairside time in comparison to its traditional counterparts, therefore increasing patient co-operation and efficiency.

Conclusion

The traditional use of scalpel for gingival depigmentation is a widely used technique employed for correction of gingival hyperpigmentation. Gingival depigmentation using scalpel method has an advantage of being effective and requires minimum time and effort with the lowest rate of re-pigmentation compared to LASER and abrasion methods.[11]

In this modern era of dentistry, LASER technology, has promising use as a mode of treatment for intra-oral procedures such as gingival depigmentation. There is however, scarce literature regarding the recurrence of pigmentation post-surgical treatment. Therefore, research should focus on finding its permanent solution, till then, repeated depigmentation and regular follow-up is the only available option currently.

References:

1. Sagar G, Rajesh N, Kumar TS, Reddy KK, Shankar BS, Sandeep V. Comparative evaluation of two surgical techniques using conventional scalpel method and diode laser for treatment outcome of depigmentation: 6 months follow-up study. Journal of Dental Lasers. 2016;10(1):2-9
2. Abdelmagyd HA, Al-Ahmari MM, Shetty SR. Treatment of gingival hyperpigmentation using different techniques. Journal of DattaMeghe Institute of Medical Sciences University. 2019 Jan 1;14(1):50-5.
3. Patil KP, Joshi V, Waghmode V, Kanakdande V. Gingival depigmentation: A split mouth comparative study between scalpel and cryosurgery. Contemporary clinical dentistry. 2015 Mar;6(Suppl 1):S97-9.
4. Lee KM, Lee DY, Shin SI, Kwon YH, Chung JH, Herr Y. A comparison of different gingival depigmentation techniques: ablation by erbium: yttrium-aluminum-garnet laser and abrasion by rotary instruments. Journal of periodontal & implant science. 2011 Aug 1;41(4):201-7.
5. Janam P, Parate N, Suchitra A, Mohan R, Vadakkekuttical RJ, Nath AR, Jose M, Ramesh A, Thomas B, Anand A, Kumari S. Gingival depigmentation by lasers—a case series. J SocPeriodont Implant Kerala. 2010;4:5-7.
6. Prasad SS, Neeraj A, Reddy NR. Gingival depigmentation: A case-report. Peoples J Sci Res 2010;3:27-9.
7. Dumett CO, Bolden TE. Post surgical clinical repigmentation of the gingiva. Oral Surg Oral Med Oral Pathol 1963;16:353-7.
8. Procaccini EM, Riccio G, Bellocci M, Di Martino C, Monfrecola G. The effects of a diode laser (810 nm) on pigmented guinea-pig skin. Lasers Med Sci. 2001;16(3):171-5.
9. Pogrel MA, Yen C-K, Hansen A. A comparison of carbon dioxide laser, liquid nitrogen cryosurgery, and scalpel wounds in healing. Oral Surg Oral Med Oral Pathol 1990;69:269-73.
10. Schaffer CJ, Reinisch L, Polis SL, Stricklin GP, NanneyLB.Comparisons of wound healing among excisional laser-created, and standard thermal burns in porcine wounds of equal depth. Wound Rep Reg 1997;5:52-61.
11. Kathariya R, Pradeep AR. Split mouth de-epithelization techniques for gingival depigmentation: A case series and review of literature. Journal of Indian Society of Periodontology. 2011 Apr;15(2):161-4.
12. D'Arcangelo C, Di Maio FD, Prosperi GD, Conte E, Baldi M, Caputi S. A preliminary study of healing of diode LASER versus scalpel incisions in rat oral tissue: a comparison of clinical, histological, and immunohistochemical results. Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology. 2007 Jun 1;103(6):764-73.
13. Murthy MB, Kaur J, Das R. Treatment of gingival hyperpigmentation with rotary abrasive, scalpel, and laser techniques: A case series. Journal of Indian Society of Periodontology. 2012 Oct;16(4):614-7.

JSPIK

Desquamative Gingivitis: A Challenging Diagnosis – A Case Report

V.R. Balaji[1], D. Manikandan[2], A. Gunasundari[3]

ABSTRACT

Gingival desquamation is a clinical sign in which the gingiva appears reddish, friable with desquamation of epithelium. Desquamative Gingivitis (DG) is an elucidating term used to demonstrate epithelial desquamation, erythema, erosions, and/or vesiculobullous lesions of the gingiva. It may be the result of various disease process such as, Mucous Membrane Pemphigoid (MMP), Oral Lichen Planus, and Pemphigus Vulgaris which accounts for a major cause of desquamation. Of all the diseases Lichen Planus was considered to be the most common skin and mucous membrane lesion that also involves the oral mucosa. Smooth erythema, desquamation, and erosion of the gingiva are common a sign of Desquamative Gingivitis, irrespective of the etiopathogenesis. This is a unique case report of Desquamative gingivitis diagnosed and managed conservatively based on the symptoms, clinical findings and histological analysis.

KEYWORDS: Mucous Membrane Pemphigoid, Desquamative Gingivitis, hyperkeratosis, subepithelial blisters, steroids.

Introduction

Desquamative Gingivitis is considered as a clinical disorder that can be recognized by an experienced clinician. More than 100 years ago, this condition was described by Tomes et al. Later, Prinz[1] in 1932 first coined the term "Chronic Diffuse Desquamative Gingivitis" for cases characterized by severe epithelial desquamation.

The management of chronic Desquamative Gingivitis has been a major problem, largely because the etiology of the disease has been elusive with early investigators believing that it was due to a single cause. Etiologic factors that have been considered includes estrogen deficiency, hypothyroidism and nutritional deficiencies.[2]

Widespread desquamation and/or erosion of the buccal side of attached gingiva of anterior teeth is considered as the chief characteristic feature of Desquamative Gingivitis. Nevertheless, DG can be confined to a limited multiple area and these lesions can be more extensive gingival lesions with oral/and or extra-oral involvement, in the primary phases or in disease recurrence. This disease is not a specific disease, but is a gingival response manifested by a variety of clinical disease entities.[3]

Desquamative Gingivitishas been most commonly caused by Lichen Planus and Pemphigoid. Unlike Lichen Planus and Pemphigoid, Pemphigus rarely seen as a cause of Desquamative Gingivitis. In general, conditions associated with DG have the highest incidence between the 4th and 6th decade of life.[4] Local hypersensitivity responses to various substances such as mouthwashes, dental materials, drugs, cosmetics, chewing gum, cinnamon, sodium lauryl (a usual ingredient of toothpaste) may also play a role as

[1] Professor & HOD, [2] Reader, [3]Post Graduate Resident, Department of Periodontics and Implant Dentistry, CSI College of Dental Sciences and Research, Madurai. Corresponding Author: Dr A. Gunasundari, E Mail: gunasundarialappan1995@gmail.com

causative agents in some patients.[5]

Identification of etiology of Desquamative gingivitis depends on the clinical and histologic criteria. Certain techniques like Immuno-Fluorescence (IF) assay would be confirmatory diagnosis. Direct IF consists of examining cryostat cut frozen sections on which fluorescence labeled agents to human antibodies IgG, IgA, IgM and the compliment components are being incubated. Michel's development of special holding solution for the transport of biopsies have enhanced the use of direct IF test. Incase of indirect IF, the antibodies in the serum would bind to the specific tissue components which can be identified by fluorescein labeled antibody to the bound immunoglobulin.[6,7]

This report highlights on a case of Desquamative Gingivitis, which was diagnosed and managed conservatively based on the clinical and histological features along with brief review of literature.

Case Report

A 62 years old female patient reported to the Department of Periodontology and Implant Dentistry, with the complaint of pain, burning sensation in gums and for routine dental checkup. On general physical examination patient was co-operative and well nourished. No signs of pallor, icterus, cyanosis and edema. Extra oral examination revealed apparently symmetrical face. Lymph nodes were not palpable and not tender. Oral examination revealed bright red lesion with diffused area of desquamation and erythema involving buccal aspect of marginal and attached gingiva resembling Desquamativelesion (figure 1 &2). Desquamation was also found on palatal aspect (figure 3). Her medical history showed no relevant significance. Etiologic factor might be related to stress, as she missed her children who lived abroad. Periodontal Treatment included full mouth oral prophylaxis along with oral hygiene instruc-

FIG 1:- Intraoral examination showing severe desquamation of gingiva

FIG 2:- Redness seen in buccal mucosa in relation to 1st quadrant

FIG 3:- Desquamation and redness in 2nd quadrant

FIG 4:- Palatal aspect showing redness and desquamation

tions. Complete blood investigation was performed which showed hematologic parameters within normal range.

After obtaining an informed consent from the patient, Smear was collected in relation to buccal aspect of maxillary anterior teeth and excisional biopsy was performed which included a free gingival graft from palate in relation to 24-26 and a punch biopsy in relation to 14,15. The tissue was stored in 10% formalin and sent for histopathological examination. Based on the clinical findings the case was diagnosed as Desquamative Gingivitis as provisional diagnosis. The differential diagnosis included Cicatricial Pemphigoid and Pemphigus Vulgaris. The histopathological examination revealed hyper parakeratosis of epithelium, saw tooth appearance of retepegs and infiltration of lymphocytes and plasma cells (figure 7). Thus, the diagnosis of Desquamative Gingivitis was established based on the clinical and histological features.

Before instituting our treatment, drug therapy and underlying disease was ruled out. The treatment included topical steroid 0.1% Triamnicolone Acetonoide (Kenacort) for two weeks (figure 4,5). For ease in application of topical steroids, a soft splint (similar to bleaching tray) with extension into the gingiva was made and steroids were applied in the splint and patient was asked to wear it full time except while eating foods. Later, oral prophylaxis was carried out. As the patient had burning sensation, she did not brush her teeth for past one month, so the patient was advised to do meticulous oral hygiene measures during subsequent visits. Consultation was also made with the confluence of Department of Oral Medicine and Department of Oral Pathology.

After the first visit, the patient was asked to consult a Dermatologist to confirm if she had any skin lesions and an Ophthalmologist for any conjunctival lesions. Both the specialists reported that there were no involvement of the eyes and skin. Patient was also counselled with a help of a counselor to break her blues out. As the patient followed the instructions meticulously, her oral hygiene improved on subsequent follow up visits. Recurrence of the lesion was not observed till a follow up period of 4-6 months (figure 6).

FIG 5:- Healing after 1 week harvesting of FGG for biopsy

FIG 6:- Review after 3 weeks

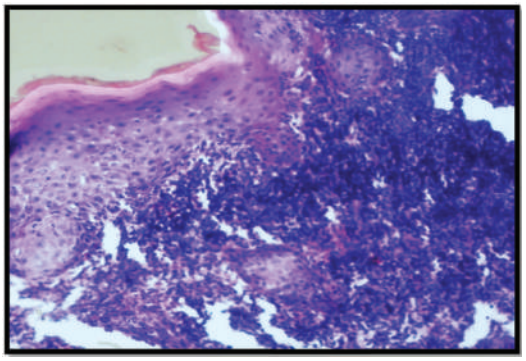


FIG 7:- Histopathological picture

Discussion

Desquamative Gingivitis is considered as descriptive term, in 1932 Prinz was the first to compose a definition, which included the occurrence of erythema, desquamation, erosion, and blistering of the attached and marginal gingiva with the possibility that marginal gingiva to be unharmed.

Almost all of the disorders associated with Desquamative Gingivitis can affect various sites in the oral cavity and also have involvement of extraoral regions. Skin, scalp, nails, and mucosae with squamous differentiated epithelium such as laryngeal, esophageal, nasal, genital, and conjunctival, represent the most possible locations. There are variations in gingival features from erythema to erosive and/or visibly ulcerated areas. Intact vesicles/bullae may occur, but they often rupture quickly in the oral cavity.[8]

Desquamative Gingivitis can mimic plaque-related gingival inflammation and may cause a delay in diagnosis in all cases in which the gingiva is the only site of involvement. Desquamative Gingivitis had also been linked with a small list of nonimmune-mediated disorders that involve endocrine imbalance disorders.

In the present case, the patient presented with bright red desquamative lesion which showed involvement of oral mucosa without any cutaneous involvement. There was generalized erythema and inflammation of labial gingiva and on palatal aspect.[9] Limitation of oral function and speech difficulties due to pain was also be observed.

Desquamative Gingivitis may occur due to aging, abnormal response to bacterial plaque, allergy, idiopathic, chronic infections, and autoimmune diseases or idiopathic. Sometimes contact allergic reactions to various oral hygiene products have also been reported to present as Desquamative Gingivitis. Initially, it was suggested that gingival desquamations were related to the hormonal changes due to menopause on the basis that gingival desquamations were more common in the middle-aged and in women. Another etiological factor reported in literature for Desquamative Gingivitis is stress. This period of life is associated with emotional disturbances and stress due to changing pattern of life. In this case, patient had stress as the common precipitating factor in the pathogenesis which was elucidated in the history.

Mucous Membrane Pemphigoid consists of a group of subepithelial blistering diseases primarily involving the mucosal surfaces. Since in this patient the characteristic lesions were restricted only to the mucosa it was diagnosed as MucousMembrane Pemphigoid.[10] Similar results were observed in the study done by Rogers et al.,[11] Laskaris et al.,[12] Gallagher and Shklar.[13]

However, reports by Agbo et al[14] were not in accordance with the present case report since both gingival and conjunctival lesion was observed. Pathogenesis of Mucous Membranous Pemphigoid probably includes an autoantibody induced, complement mediated sequestration of leukocytes with resultant cytokine and leukocyte enzyme release and detachment of the basal cells from the Basement Membrane Zone (BMZ) but there may also be complement mediated cell lysis.[15,16]

In this case report, the patient was advised to use the soft splint with topical steroids instead of manual application as this method shall deliver the drug comparatively more than manually with less patient compliance. Moreover, this method had more contact time of the drug on to the lesion. This unique way of using steroids similar to local drug delivery on to the lesion might have improved the healing potential of the lesion and paved the way for an excellent healing which was noticed in the subsequent visits.

Treatment of Mucous Membrane Pemphigoid is based on the severity of symptoms and the site involved. Patients with these types of lesion may be benefited with the use of steroids. Treatment of Mucous Membrane Pemphigoid is considered to be a compromised condition, due to its auto-immune nature.[17] The lesions may occur intermittently and affect different parts of the oral mucosa at different times.[18]

Since this disease has an irregular pattern of recurrence, dentists should be in close contact with an ophthalmologist and dermatologist in cases with possible conjunctival or skin involvement.[19]

Moreover, the patient was counselled for her anxiety and stress which might also would have contributed for the quick healing of the lesion. In our present report, consultation was done with Dermatologist, Ophthalmologist, Department of Oral medicine, Department of Oral pathology and a counsellor to rule out the involvement of skin, conjunctiva and to

eliminate her stressful situation. The lesion healed without any recurrence with the follow up period of 6 months. Thus, a Multispecialty treatment approach should be considered and it could be a valuable treatment modality in such cases.

This case report is unique because of its challenging diagnosis and treatment approach with follow up of 6 months as there are many Mucous Membranous conditions that mimic Desquamative Gingivitis.

Conclusion

Desquamative gingivitis is a complex term that needs a clear definition. However, it is essential to remember that Desquamative Gingivitis is not a diagnosis, but a gingival response associated with a variety of clinical conditions. A significant number of cases of Erosive Lichen Planus and Pemphigus present with a picture of Desquamative Gingivitis. A multispecialty treatment approach could bring a more insight and valuable treatment protocols which shall imbibe more values to patient care.

References

1. Prinz H. Chronic diffuse desquamative gingivitis. Dent Cosm1932;74:332-3.
2. Mariotti A. Desquamative gingivitis: Revisited. Today's FDA 1991;3(1):1C-2.
3. Lo Russo L, Fedele S, Guiglia R, Ciavarella D, Lo Muzio L, Gallo P, et al. Diagnostic pathways and clinical significance of desquamative gingivitis. J Periodontol 2008;79(1):4-24.
4. Robinson NA, Wray D. Desquamative gingivitis: A sign of mucocutaneous disorders – A review. Aust Dent J 2003;48(4):206-11.
5. Scully C, Porter SR. The clinical spectrum of desquamative gingivitis. SeminCutan Med Surg 1997;16(4):308-13.
6. Glickman I, Smulow J. Chronic desquamative gingivitis: Its nature and treatment. J Periodontol1964;35:397-405.
7. Mariotti A. Desquamative gingivitis: Revisited. Todays FDA 1991;3(1):1C-2.
8. Position paper: Oral features of mucocutaneous disorders. J Periodontol 2003;74(10):1545-56.
9. Aguirre A, Vasquez JL, Nisengard RL. Desquamative gingivitis. In: Carranza FA, Editor. Clinical Periodontology, 10th ed. New Delhi: Elsevier publishers; 2007. p. 411-33.
10. Ziskin DE, Zegarelli EV. Chronic desquamative gingivitis – A report of twelve cases. Am J Orthod Oral Surg 1945;31:1-33.
11. Rogers RS 3rd, Seehafer JR, Perry HO. Treatment of cicatricial (benign mucous membrane) pemphigoid with dapsone. J Am Acad Dermatol 1982;6:215-23.
12. Laskaris G, Sklavounou A, Stratigos J. Bullous pemphigoid, cicatricial pemphigoid, and pemphigus vulgaris. A comparative clinical survey of 278 cases. Oral Surg Oral Med Oral Pathol 1982;54:656-62.
13. Gallagher G, Shklar G. Oral involvement in mucous membrane pemphigoid. Clin Dermatol 1987;5:18-27.
14. Agbo-Godeau S, de Lima Soares P, Szpirglas H. Cicatricial pemphigoid: Management in stomatology. Rev StomatolChirMaxillofac2004;105:206-10.
15. McCarthy FP, McCarthy PL, Shklar G. Chronic desquamative gingivitis: A reconsideration. Oral Surg 1960;13:1300-13.
16. Abbayya K, Kadashetti V, Baad R, Lohana M. Desquamative gingivitis: A clinical sign in mucous membrane pemphigoid. Eur J Gen Dent 2015;4:136-9.
17. Vijayakar HN, Shah PP, Desai AB, Ghonasgi SR, Gawankar RJ. Chronic desquamative gingivitis in siblings: A report of two cases. J Indian Soc Periodontol2014;18:385-9.
18. Rajendran R. Diseases of the skin. In: Shafer WG, Editor. Textbook of Oral Pathology 5th ed. Noida: Elsevier publishers, 2007. p. 1099-1162.
19. Merritt PL. Chronic desquamative gingivitis. J Periodontol1933;4:30-4.

INTERVIEW

COFFEE WITH THE LEGENDS

The editorial team of JSPIK is happy to share with our readers, a short interview session with the Senior members. We interacted with them to understand how far our Specialty has made its mark among the public. Few questions asked were:

1. What was the scenario in your region (Malabar/Central Kerala/South Kerala) when you started your career as Periodontist? How many specialists were there practising Periodontics then? Were fellow Dentists motivated enough to refer cases to a Periodontist? How often were patients referred for expert periodontal care from other Dentists?

2. What was the level of awareness of about periodontal diseases and treatment options among the general public? Over the years many procedures evolved in our specialty. How has these changed affected our clinical practice and academic preaching?

3. As you are one among the Senior specialists' of Kerala, how do you evaluate the metamorphosis of our speciality till date? Words of wisdom to the young specialists of SPIK?

DR. SHAMSUDDIN HASSAN BAVA

Retired Principal and Professor, Government Dental College
Former Member, Dental Council of India
Former Dean, Faculty of Dentistry, Calicut University

Sir graduated in 1974 from Govt. Dental College, Thiruvananthapuram. He joined as Tutor in GDC Trivandrum, and later completed his post graduation in 1980 from the same institution after which he was promoted as Assistant Professor in Periodontics and posted at GDC, Kozhikode in 1980. In 1988 Sir was the Professor at the department. In 1989, he headed the Dental wing, Govt. Medical College, Kottayam. From 1990, till 2003 Sir remained department head at GDC, Kozhikode. In 2004, he served as Principal, GDC Thiruvananthapuram and in 2005 as Principal, GDC Kozhikode. He was also the Dean, Faculty of Dentistry, Calicut University and has been nominated to DCI as member. After retirement from Govt. service Sir has worked in many private Dental Colleges as Principal.

"My career as a Periodontist started in North Malabar in the year 1976 in the new Dental wing of Calicut Medical College. I was appointed as Assistant Professor just after completing my post-graduation in 1980. At that time, there were only five functioning dental specialties in Calicut Medical College – Oral Medicine, Orthodontics, Prosthodontics, Oral Surgery and Periodontics.

For many years in the beginning, I was the only Periodontist in North Malabar region and the leading Dental surgeons of that time like Dr Radhagopi of Kottaparamba, Kozhikode, Dr Harris from Kannur etc used to refer their perio patients to me. I also used to treat patients referred by physicians and those coming from different areas like Trivandrum, Bangalore, Malappuram, Kasargod and Palghat. Those days there was very low awareness among the general population regarding dental hygiene and I used to conduct awareness programs and try to advocate good oral hygiene methods to the patients approaching me for treatment.

Nowadays I see that science and technology in Dentistry is advancing at such a fast pace that there are many new techniques and procedures that simplifies the treatment protocols. Multiple new specialties and sub-specialties have also come up which has made the treatments better targeted and more patient centered than before. I hope that the new generation can contribute a lot to bring even further advancements to the field of dentistry".

DR KUNJAMMA SEBASTIAN

Date of Birth: 30th January 1948

Madam joined as a tutor in the Department of Periodontitis at Government Dental College Trivandrum on 19th June 1980. She worked as an Associate Professor for 23 years and retired on 30th January 2003. After retirement from Govt. service, Madam has been Visiting Professor in Meenakshi Amal Dental Collage Chennai, Principal of Mookambika Institute of Dental Science, Kulashekaram, Tamilnandu, Principal of MES Dental College Perithalmanna, Principal of Kannur Dental college. Married to late Dr. N.B Abraham, she has two children, Dr Lekshmi Minu Abraham and Dr Nimi Biju Abraham. Both of them are Dentists in New Zealand. She has 6 grandchildren and she loves spending retired years in Trivandrum and visiting her children in New Zealand.

" I had never heard of professional cleaning of the teeth before I joined the BDS dental program. As to my knowledge there were very few dental surgeons at that time and they mainly did extractions, fillings and dentures. In 1973 Periodontics as a specialty was commenced in Trivandrum Govt. Dental College. I remember in the beginning hand scaling was done for the removal of calculus and plaque.Although later there was the introduction of ultrasonic scalers and non-surgical treatment. This was followed by surgical treatment for periodontal diseases like gingivectomy, mucogingival surgery, free gingival graft ,lateral sliding flap procedures etc.

I started my career as a general dentist in 1976. Back in those days, dentist used to take care of all the treatment need of the patients for example, cleaning, filling, extractions, removal dentures, scaling and polishing and even minor orthodontic corrections. Amalgam was used for restorations of posterior teeth. Anterior restorations were done with silicate cements as we didn't have the luxury of the now available composite cements. Commonly used temporary cements were Zinc oxide eugenol and Zinc phosphate cements. After a few years of practicing as general dentist I decided to take up my master's degree. I was fascinated with Periodontology, so I joined as a Tutor in the Department of Periodontics in Government Dental College, Trivandrum. As far as I can remember there were only a few Periodontiststhen; Dr BRR Varma(my post graduate Teacher) , Dr Thomas Thelly, Dr Mehrunisa Bhai, Dr Nandakumar, Dr Shosha and Dr Shamshuddin. During the early stages of my career, there were not many referring general practitioners. It may be due to the lack of knowledge of the various periodontal surgical treatments modalities available or the general practitioners usually did all the treatment themselves. So referral for advanced periodontal treatment wasn't as common as it is today.

Countless advancement have taken place in the field of Periodontics over the years. Patient evaluation and education was carried out by using disclosing solution and tablets.The introduction of bone grafts for the management of the infra-bony pockets in periodontal procedures has certainly revolutionized treatment outcomes and all this improved our clinical practice. The usage of local drug delivery system for the treatment of periodontal procedures as well as guided tissue regeneration has been significant in successful treatment outcomes. The use of blood products for the regeneration of periodontium has had a positive response. As periodontology became popular as a specialty there was a wide usage of C-T scan and laser. The use of laser in periodontal surgery has been very advantageous. A laser is beneficial in clinical periodontal practice as there is less bleeding and it is less painful, hence patient acceptance is high.

Personally I would suggest to avoid surgical treatment as much as possible. The first option would be to correct by non-surgical procedures and see how the tissues are responding, then to proceed accordingly. Never do unethical practice and it is very important to communicate effectively with the patient at all stages of periodontal therapy as it is a lasting relationship builder. Always think as a patient and be kind and considerate to the patient and treat them as a whole individual and not just the gums, or teeth. It is important to understand the psychology of the patient as at the end of the day proper home care and motivation brings lasting results .Never underestimate the power of team work and patient education that brings a positive outcome".

DR PRESANTHILA JANAM

Former Principal, Govt. Dental College, Trivandrum

Director of CDE, Associate Dean Research and Professor,

Dept. of Periodontics PMS College of Dental science &Research

Trivandrum, Kerala

Date of Birth: 01-06-1956

Madam completed BDS and MDS from Govt. Dental College, Thiruvananthapuram. Madam joined Medical Education service in 1983. She is the recipient of the State Best Doctor Award in Modern Medicine 2010 from Government of Kerala. She is the PhD guide of Kerala University 2010 onwards, and PhD guide of KUHS 2014 onwards. In 2016 she entered Private Dental college service. She received Best Dentist Award 2017 from Kerala Dental Council. With several other accolades to her credit she has more than 140 publications to her credit; and has authored a textbook in Periodontics- "Essentials of Comprehensive Periodontology" published by GOVEN in 2019. Her goal is to maintain up to date clinical and theoretical knowledge about the science and art of dentistry especially in the field of Periodontics and contribute towards research.

"With the grace of Lord almighty, teachers and the support of my family, I started my career as a Periodontist in 1986 in Govt. Dental College, Calicut. Later I joined GDC, Trivandrum; back then, there were hardly 5 practicing Periodontists' in South Kerala. We used to get a good number of referral cases on daily basis from dentists across South Kerala which is very contradictory to today's situation where Periodontists are fighting for their survival. Also the awareness about periodontal diseases were much less among the general public in those times which is also contradictory to today's situation.

Periodontitis can only be reduced by prevention and interception. For this, awareness about the disease is the primary need. So in earlier times we had to arrange camps, write articles, conduct talks in various print and audio visual media. These efforts made the public more closer to the Periodontists. Still individual approaches were lagging behind because of the high cost of dental treatment which increased beyond the purchasing capabilities of common people. Non-Surgical therapy was done mostly and also cosmetic procedures were done sparingly those times.

Periodontics has undergone drastic changes over the years. Earlier people approached us mainly for Periodontal diseases; over the years, awareness has increased so much that now they are approaching us more for aesthetics. Non-Surgical therapy if done properly can give equivalent results to surgical treatment and a proper SPT schedule need to be maintained. In the recent years, newer instruments, advanced diagnostic aids, newer and more effective bio materials, concepts of tissue engineering, stem cell therapy, LASERs, Photodynamic therapy, piezo surgery etc. are making periodontology more interesting.

On a personal note the recent developments have played an important role in my academic profile too. Especially for doctors like me in the Medical Education Department it is important to keep updated and research on these. Many of these works have done great accolades for me. Sophisticated procedures like LASERs, piezosurgery, implant surgery etc. were done in Periodontics Dept in GDC Trivandrum. We also had patients approaching us for the new procedures. Academically it was so challenging that it changed the profile of the young Periodontists trained under me.

Periodontology has grown tremendously much more than I could have imagined in my post graduation days. These developments are a blessing for the patient and a big asset for the doctor. For the patient, there is better treatment, less morbidity, less time consuming and lesser complications. For the doctors they help to make the diagnosis and treatment more precise, better infrastructure and comfortable working condition. So the developments are a relief for the patient and the doctor.

The recent developments in travel and communication has been a great boon for the patients especially in remote areas. Dental health awareness has also increased due to this. Since the number of Periodontists is high nowadays, I recommend that the younger specialists to work more in the peripheral parts of Kerala which will increase their clinical expertise and provide more monetary benefits. Also it is important to maintain a cordial doctor-patient relationship in this era of social media. Be truthful to the patient and empathise with them. But always be cautious and prudent. Never criticize your fellow doctors in front of them. Get the opinions of seniors if in doubt. Remember it's always-Give respect, take respect".

INFORMATION TO AUTHORS

About the Journal

JSPIK accepts articles from dentists, dental specialists (any speciality) and students. The articles submitted must have relevance to the speciality of Periodontics. Authors are encouraged to submit research papers, interdisciplinary case reports, interesting case discussions, letters to editor review articles or short communications.

Manuscripts

Articles should be type written on one side of A4 size (21x28cm) white paper in double spacing with a sufficient margin. Use a clear and concise reporting style. SPIK reserves the right to edit, manuscript, to accommodate space and style requirements. A soft copy of the article also has to be send to the editor's email: **editorspik@gmail.com**

- **Title Page:** Title page should include the title of the article and the name, degrees, positions, professional affiliations of each author. The corresponding authors, telephone, e-mail address, fax and complete mailing address must be given.
- **Abstract:** An abstract of the article not exceeding 200 words should be included with abbreviated title for the page head use. Abstract should state the purpose of the study, investigations, basic procedures and the main findings. 4 key words of article should be mentioned below the abstract.
- **Tables:** Tables should be self explanatory, numbered in roman numbers, according to the order in the text and type on separate sheets of paper.
- **Illustrations:** Illustrations should be clearly numbered and legends should be typed on a separate sheet of paper, while each figure should be referred to the text(figure 1,2 etc.)
- **Reference:** Reference should be selective and keyed in numerical order to the text in Vancouver Style (not alphabetical). Type them double spaced on a separate sheet of paper. Journal references must include author's names, article title, journal name, volume number, page number and year. Book reference must include author's or editor's names, chapter title, book title, edition number, publisher, year and page numbers.

Copy right

Submission of manuscripts implied that the work described has and not been published before (except in the form of on abstract or as part of published lectures, review or thesis) and it is not under consideration for publication else where, and if accepted, it will not be published else where in the same form, in either the same or another language without the comment of copyright holders. The copyright covers the exclusive rights of reproduction and distribution, photographic reprints, video cassettes and such other similar things. The views/opinions expressed by the authors are their own. The journal bears no responsibility what so ever. The editors and publishers can accept no legal responsibility for any errors, omissions or opinions expressed by authors. The publisher makes no warranty, for expression implied with respect to the material contained therein. The journal is edited and published under the directions of the editorial board/review panel who reserve the right to reject any material without giving explanations. All communications should be addressed to the Editor. No responsibility will be taken for undelivered issues due to circumstances beyond the control of the publishers.

Books for review

Books and monographs will be reviewed based on their relevance to SPIK readers. Books should be sent to the Editor and will become property of SPIK.

Article publication charges

There are no article publication charges for JSPIK. If the author wishes for print, processing fee shall be levied for hard copies of the journal depending upon the number of reprints asked for.

Subscription Rates

Free distribution for all the members of the SPIK.

Inland Subscription for non members and institutions : Rs. 1000 per issue, Rs. 3000 for 1 Year, Rs. 6000 for 2 years, Rs. 7500 for 3 years

Address for communication

Dr. Sameera G Nath, Editor JSPIK, Reader, Department of Periodontics, KMCT Dental College, Manassery, Kozhikode 673602, Kerala, India **e-mail: editorspik@gmail.com**