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SOCIETY OF PERIODONTISTS &IMPLANTOLOGISTS OF KERALA

SPIK ANNUAL CONFERENCE 2022

Sunday, June 5, 2022, Kottayam

Accredited for 6 CDE Credit Points from Kerala State Dental Council subject to approval

Keynote Speakers

Dr Balaji Manohar
Professor of Periodontics
Kalinga Institute of Dental Sciences
Bhubaneswar

Dr Mihir Kulkarni
Associate Professor of Periodontics
SDM College of Dental Sciences
and Hospital, Dharwad

PerioScope
Clinical Stories of
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For the People
Release of Perio-
awareness video

Kottayam, the "Akshara Nagari (City of Letters) of Kerala" reckons you for the 13th Annual Conference of the Society of Periodontists and Implantologists of Kerala. We have a day filled with inspiring scientific sessions by eminent speakers, a panorama of interesting case presentations by our own members, and the much-awaited release of the awareness video. Let's all make sure to be present for our first offline event after two years of COVID hiatus.

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SOCIETY OF PERIODONTISTS AND IMPLANTOLOGISTS OF KERALA

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President's message

Dear colleagues

We have almost reached the end of this association year. Corona pandemic prevented us from conducting any worthwhile physical programmes during the last two years. During our conference we are bringing out two awareness videos, one in Malayalam for the public and the other is for general practitioners and other branch specialists. Kindly take some time to go through the video and promote them among your professional friends.

Almost 90% of the population belonging to the upper socioeconomic class takes their full body health check up regularly but only half of them visit dental clinics in a regular basis. The other half is blissfully unaware of the need for such a regular checkups. Every practicing dentist would say dentistry is going through a very difficult period, too many dentists, stiff competition, low patient turnout etc. But I believe the problem lays elsewhere "the lack of awareness"

The prevalence of caries, gingivitis & periodontitis in our population is very high but most of them are left untreated because of many reasons like cost involved, the absence of pain, inability to take time from busy work schedule, the lack of awareness about the consequences of not getting proper treatment etc. Caries usually cause pain which motivate the patient to take treatment but periodontal disease are over looked or treated with home remedies / over the counter products which provides effective symptomatic relief.

Imagine if 50% of Kerala population decides to treat their "entire dental problems" the present infra structure will miserably fail to provide treatment for all of them. Unfortunately our health department, dental councils, Colleges or various associations are not effective to create this awareness. Of late many individuals are using the social media to promote their practice through awareness videos but many of them are self promotions and unethical. This can attract actions against them from the dental council, if someone finds the content unethical, as there are no existing guidelines for using social media. I am glad that I was instrumental in formulating the new guidelines for bringing out awareness videos in social media by the dental council; soon it will come into effect.

In a country like India we should focus more on prevention, managing advanced periodontitis and its consequences is a serious financial burden. So I urge all of you to watch the "awareness video for professionals " carefully and try to incorporate the tips provided thereof.

This is my last message as president of SPIK. Let me thank you all for the privilege you provided to me to serve you as the president of SPIK for the last two years. I am very grateful to my secretary Dr Jayan Jacob for the laudable management of the office and Dr Sameera G Nath for meticulously bringing out seven issues in three volumes of our journals during my term. I thank all the executive members for providing total support to me during this entire period.

Thank you

Dr Sabu Kurian
President, SPIK

Secretary's Message

Dear SPIK members,

The extended term of the current SPIK office is coming to its end. The unforeseen COVID scenario restricted the conduct of almost all our regular activities in the last two years and whatever we could carry out was in the digital mode. However, we were able to organize the prestigious SPIK Periodontology Scholarship Exam on March 21, 2022, at Kannur Dental College. There was record participation from various colleges across the state. Ashish Anz of Sri Sankara Dental College, Varkala, was the winner of the exam. SPIK congratulates Dr. Mohammed Feroz, the exam convenor, for the excellent conduct of the exam and expresses our gratitude to the expert panel of examiners.

We are having the culmination of our activities on June 5, 2022, at Kottayam with our Annual Conference. Enriching scientific sessions are planned for our members and our Annual General Body Meeting, and the election of new office bearers shall also take place. The highlight of this conference is the release of the Periodontal Awareness Videos, which is the dream project of our President, Dr. Sabu Kurian. On behalf of SPIK, I extend a warm welcome to you at this event.

On a parting note, I would like to express my sincere gratitude to my two Presidents, Dr.Harikumar Menon and Dr.Sabu Kurian, for the memorable years. I thank all the Office Bearers and Executive Members for their support. A special word of thanks to our dedicated and enthusiastic Editor, Dr. Sameera G. Nath, for her painstaking efforts to bring out excellent issues of JSPIK. Finally, a big thanks to all SPIK members for their cooperation and encouragement in the last three years. We have unfinished business at SPIK, but I am sure that the new Office under the leadership of Dr. Presanthila Janam shall achieve them and take our association to greater heights. I wish the forthcoming Office the very best.

Dr. Jayan Jacob Mathew
Secretary, SPIK

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JSPIK

The Effects of Subgingival Application of Ozonated Olive Oil Gelatin Film in Patients with Chronic Periodontitis – A Clinical Study

CP Vijeyakumar[1], S Thangakumaran[2], Sasikumar[3], Santhosh[3]

ABSTRACT

Introduction : This study evaluates the effects of subgingival application of ozonated olive oil gelatin film as an adjunct to scaling and root planning in patients with chronic periodontitis.

Materials & methods : Thirty patients were randomly selected and equally divided into Group I received Scaling and root planing (SRP) only, Group II received SRP and ozonated olive oil gelatin film. Subgingival application of ozone gel was performed following initial SRP and after 7 and 14 days. Clinical measurements included Probing pocket depth (PPD), plaque index (PI), gingival index (GI), Oral hygiene index-simplified(OHI-S) and clinical attachment level (CAL) were recorded at baseline and after 21 days.

Results : The results showed improvement in all clinical parameters in (Group II) which was maintained up to 21 days ($P<0.001$).

Conclusion : The study concluded that Ozonated olive oil gelatin film could be a promising adjunct to SRP in the treatment of Chronic periodontitis.

Keywords : Ozone, Chronic periodontitis, SRP

Introduction

Periodontal disease has a multifactorial etiology. Elimination of periopathogens containing biofilms remains the primary goal of periodontal treatment. Although mechanical debridement such as scaling and root planning (SRP) reduces the level of sub-gingival bacteria, it does not eliminate all the pathogens which resides deep into the connective tissue.[1] For the shortcomings of systemic administration, local delivery systems containing antibiotic or antiseptic agents were introduced.[2] These systems allow the therapeutic agents to be delivered directly into the diseased site with no appreciable systemic effects.

In the past years, therapeutic effect of ozonated oil was attributed to its antibacterial, antifungal, antiviral, antiparasitic, antihypoxic, analgesic and immunomodulatory effects on biological systems. In dentistry ozone therapy can be used in various specialties. Particularly in periodontics the sub gingival ozonated oil application on human and animal models have been shown to improve cellular function, improve healing of tissue, and scavenge the defective tissue in the biological system, promoting the healthy cells to survive and multiply more rapidly.[3] Apart from Ozonated oil, Ozonated olive oil[4] has a valuable antimicrobial activity against bacteria, fungi, viruses. Extra virgin olive oil[5] (EVOO) contains more than 36 phenolic compounds.

Although all of the phenolic compounds in EVOO have known beneficial effects, one specific compound known as oleocanthal, has been discovered to have powerful natural anti-inflammatory benefits.[6,7] Oleocanthal is unique only to olive oil and it has anti-inflammatory property. This property was homolo-

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gous with the non-steroidal anti-inflammatory drug (NSAID) - Ibuprofen. NSAIDs have many known side effects such as gastric irritation, nausea, vomiting etc. But olive oil is a natural anti-inflammatory agent that can be safely consumed by anyone. Oleocanthal inhibits cyclooxygenase 1 and 2 (COX 1 & 2) enzymes in a dose dependent manner.

Based on the anti-inflammatory, anti-microbial, anti-oxidant properties of ozonated olive oil, the present study is conducted to evaluate the clinical effects of subgingival application of ozonated olive oil gelatin film as an adjunct to scaling and root planing in patients with chronic periodontitis

Materials and Methods

A randomized, controlled, split mouth clinical study was conducted to evaluate the effectiveness of subgingival application of Ozonated olive oil gelatin film as an adjunct to scaling and root planning compared with scaling and root planning alone in patients with chronic periodontitis. The protocol was reviewed and approved by institutional ethical board.

The study related procedures were explained to the patients before they sign an informed consent form. A total of 30 subjects each with bilateral 5-6 mm probing pocket depth (PPD) were recruited from the outpatient in Department of Periodontics, J.K.K. Nattraja Dental College and Hospitals, Kumarapalayam, Tamilnadu.

Criteria for Grouping

Selected sites were randomly divided into control sites and experimental sites as follows. Group I consists of 30 sites, in which scaling and root planning was done (control sites). Group II consists of 30 sites, in which scaling and root planning was followed by the placement of the Ozonated olive oil gelatin film inside the pocket (Test sites).

Clinical parameters

The following variables were measured at baseline and at 21 days. Plaque index (PI) (Silness and Loe 1964) Gingival index (GI) (Loe and Silness 1963) Oral hygiene index-Simplified (OHI-S) (Greeneand Vermillion 1964) Probing Pocket Depth (PPD) according to (Ramfjord, 1967),[8] Clinical attachment level (CAL) according to (Ramfjord, 1967). Non-surgical Periodontal therapy at baseline full mouth ultrasonic scaling was done. Under local anesthesia with 2% lignocaine solution (1:80,000), root planning was done in the test and control sites using area-specific double-ended Gracey curettes (Hu-Friedy).

The test site was isolated with cotton rolls to prevent contamination with saliva. The Ozonated olive oil gel was carried with a tweezer and placed in the periodontal pocket. The pocket opening was covered with Coe-Pak to retain the material in the pocket, as well as to prevent the ingress of oral fluids. Oral hygiene maintenance instructions were given. Subjects were recalled at 7th and 14th day for application of Ozonated olive oil gelatin film. Clinical parameters were repeated after 21 days.

Gel application

The selected teeth were isolated carefully with cotton rolls and thoroughly dried and the gel was applied carefully subgingivally and interproximally until excess gel was observed from the gingival margin.

Excess gel was removed with a cotton roll and patients were instructed not to eat, drink, or rinse for at least 30 min and to refrain from chewing hard or sticky foods, brushing near the gel treated site or using inter-dental aids. Gel application was performed after initial SRP and at 7, 14 days.

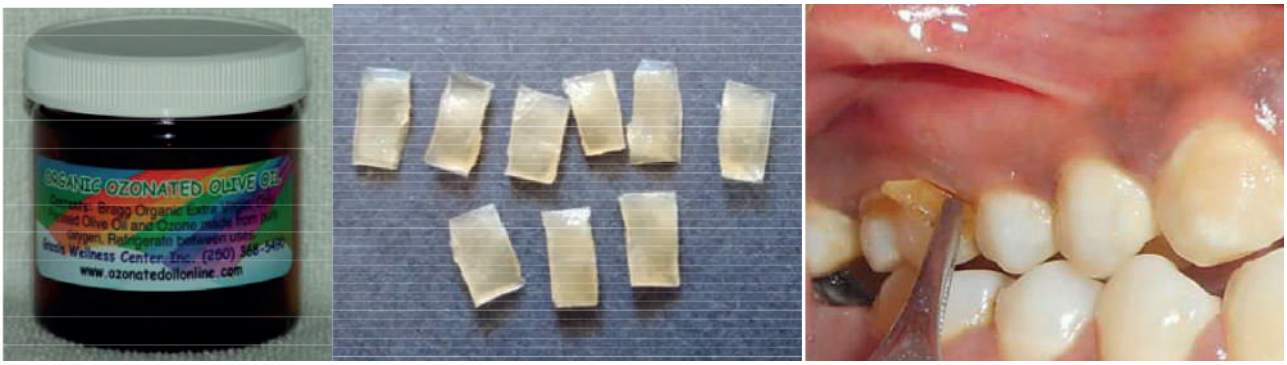

Fig 1: Ozonated olive oil gelatin film & placement

Statistical analysis

All the results were tabulated and statistically analyzed using Statistical package for social science (SPSS version 12). Intragroup comparison was done using paired t-test. The difference between groups was statistically analyzed using independent sample t-test.

Results

In the present study mean plaque index, gingival index, OHI-S scored at baseline were 1.65± 0.91, 1.65±0.09, 3.29±0.32 respectively and after 21 days these were reduced to 0.81± 0.19, 0.79±0.18, 1.52±0.43. There was statistically significant reduction in the plaque index, gingival index, OHI-S were observed 21 days post-treatment (p<0.001).(Table 1)

In our study the mean PPD in Group I, at baseline was 5.03±0.18 mm, and at 21 days reduced to 3.03±0.18 mm. In Group II, at baseline the mean PPD was 5.33±0.47 mm, and at 21 days reduced to 2.83±0.59 mm. There was statistically significant reduction in PPD after 21 days post-therapy in both groups (p<0.001).(Table 2).Group II showed more statistically significant reduction in PPD at 21 days post-therapy, compared to group I (p<0.001). The results was in accordance with Issac AV et al., (2015).[9] In his study the use of ozonated water resulted in probing pocket depth reduction and it was due to the prevention of free radical mediated tissue destruction, anti-inflammatory and immune stimulating effect of ozone.

In the present study the mean CAL at baseline in Group I was 4.06 ± 0.12 mm, and at 21 days reduced to 2.14 ± 0.12 mm (CAL gain~ 2mm). In Group II, at baseline the mean CAL was 4.36 ± 0.28 mm, and at 21 days reduced to 1.43 ± 0.29 mm (CAL gain~ 2.8 mm). There was statistically significant gain in CAL after 21 days post-therapy in both groups (p<0.001). Group II showed more statistically significant gain in CAL at 21 days post-therapy, compared to group I (p<0.001) (Table 2).This was in accordance with the study done by Ramzy MI et al., (2005)[10] where subgingival irrigation with ozonized water improved the clinical attachment level by the formation of thin junctional epithelium on the diseased cementum

Discussion

The present study was designed to evaluate the clinical effects of subgingival application of ozonated olive oil gelatin film as an adjunct to scaling and root planing in patients with chronic periodontitis. In

Table 1: Comparison of mean values of Plaque index, gingival index, Oral hygiene index between groups 1 and 2 at baseline and postoperative stages (21 days)

Index	Time						Paired Samples t-test	
	Baseline			PO - 21 Days				
	Mean	SD	SE	Mean	SD	SE	t-value	P-value
Plaque Index	1.657	.091	.017	.815	.194	.035	27.825	.000(P<0.001)
Gingival Index	1.652	.093	.017	.792	.183	.033	27.729	.000(P<0.001)
OHI Index	3.293	.327	.060	1.528	.433	.079	22.419	.000(P<0.001)

Table 2: Comparison of Mean values of Control (Group 1) and Test (Group 2) Groups between Baseline and Post operation (21 days)

Group		Time						Paired Samples t-test	
		Baseline			PO - 21 Days				
		Mean	SD	SE	Mean	SD	SE	t-value	P- value
Group 1	PPD	5.033	.183	.033	3.033	.183	.033	@	@
	CAL	4.06	.12	.02	2.14	.12	.02	@	@
Group 2	PPD	5.333	.479	.088	2.833	.592	.108	23.924	.000(P < 0.001)
	CAL	4.36	.28	.07	1.43	.29	.09	20.526	.000(P < 0.001)

@ - t-test could not be carried out since the difference between the variances is zero

this study ozonated olive oil gelatin film selected over ozonated water as the retention of ozone molecules were more in olive oil compared to other oils as well as ozonated water. In the present study mean plaque index, gingival index, OHI-S scored at baseline were reduced after 21 days. There was statistically significant reduction in the plaque index, gingival index, OHI-S were observed 21 days post-treatment ($p<0.001$).

In our study the mean PPD were reduced after 21 days post treatment in group 2 compared to group 1. There was statistically significant reduction in PPD after 21 days post-therapy in both groups ($p<0.001$). Group II showed more statistically significant reduction in PPD at 21 days post-therapy, compared to group I ($p<0.001$). The results was in accordance with Issac AV et al.,[9] In his study the use of ozonated water resulted in probing pocket depth reduction and it was due to the prevention of free radical mediated tissue destruction, anti-inflammatory and immunostimulating effect of ozone.

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Hence Ozonated olive oil gelatin film can be used as a potent local drug delivery system in non surgical periodontal therapy.

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Diagnosis and Management of Peri-Implant Disease: A Clinical Update

Nitya Kala[1], Harikrishnan Prasad[2]

ABSTRACT

Introduction/Objectives: Peri-implant disease (PID) is the disease affecting the mucosa and/or the bone surrounding the implant. In the early days periodontitis and PID were considered as very similar entities, with dental biofilm being the common causative factor. Years of observation has changed this view. The pathogenesis and the risk factors associated with PID are being researched extensively. The treatment modalities are ever evolving with the advent of latest technology. It is our aim to provide the latest information in the diagnosis, pathology, associated risk factors and treatment options for peri-implant disease in this review.

Data: Data that is published either as literature review or original research which is relevant to PID are included in the review. Only those articles that are published in English are included.

Sources: A search was conducted in PubMed and Google Scholar databases using the terms peri-implantitis, peri-implant mucositis and peri-implant disease to identify relevant literature.

Study selection: Studies that had did not have any information about peri-implant disease in the abstract were eliminated. Other articles were then studied fully by both the investigators, and any relevant information present was included in this review.

Conclusion: Although the success rate of implant is very high, with increasing rate of implant placement, the number of cases with peri-implant disease is on the rise. The precise pathogenesis and the increased rate of progression is still under research. In this review in addition to giving a clinical update, we are also proposing an algorithm for the management of peri-implant disease based on the available clinical findings.

Key-words: peri-implant disease, peri-implantitis, peri-implant mucositis, implant failure

Introduction

In today's world implant is considered as an ideal replacement for lost tooth. It is the favoured option for both the patient and the clinician. It is even considered as the third set of dentition available for the humans. Gosta Larssson was the first person to receive a dental implant.[1] Her implant survived till her death for about 40 years. The mean survival rate of implant today is as high as 94.6% +/- 6%.2 There is a mean prevalence rate of 43% and 22% for peri-implant mucositis and peri-implantitis in Europe and America.[2] At this rate, Peri-implant disease (PID) may become the iceberg that sinks the use of implant in future. It is therefore in our best interest that the rate of peri-implant disease be minimised by following a proper prevention and early intervention protocol.

A dental implant unites with its surrounding bone by osseointegration. On clinical examination

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the healthy peri-implant mucosa is about 3 to 4 mm in height, appears pale pink to coral pink in colour, with firm and resilient texture. It shows no sign of bleeding on probing, has a pocket depth of not more than 5 mm and exhibits no mobility. It should also not show any bone loss beyond that caused by initial bone remodelling. Microscopically the mucosa shows central connective tissue core that is lined by either keratinized or non-keratinized epithelium on the surface adjacent to the implant. Two types of epithelia are found, one is the sulcular epithelium, which lines the sulcus and the other is the barrier epithelium or junctional epithelium that is in direct contact with the implant. Few inflammatory cells are found in the connective tissue adjacent to barrier membrane. Multiple small blood vessels are also seen. Below this epithelium there is a region of implant, which has direct connective tissue attachment to the implant surface. Just below this, lies the bone with its osseointegrated implant (Fig.1). This interface lacks periodontal ligament and cementum like structure. The collagen fibres run parallel to the direction of implant, in stark contrast to the periodontal ligament.

Classification of Peri-Implant Disease

World workshop 2017[3] classified peri-implant diseases as

1. Peri-implant health
2. Peri-implant mucositis
3. Peri-implantitis
4. Peri-implant soft and hard tissue deficiencies

Pathogenesis

Colonisation of bacteria occurs in the peri-implant sulcus within 30 minutes of implant placement. It has been found that the organisms found at about 10 days is an indicator of the organisms that would be seen after 6 months.[4] This emphasizes the importance of a good periodontal health before implant placement. A healthy sulcus is found to be rich in gram positive, facultative aerobic organisms. The sulcus of patients with peri-implant disease is found to harbour gram-negative anaerobic organism, mainly those from red and orange complex. In the initial days PID resembles gingivitis and periodontitis. The bacterial challenge and inflammatory response appear to be similar in kind and magnitude. It is only in the later stages that the difference seems to occur. It has been found that the progression of peri-implant disease is nonlinear and accelerating.

Quantitatively the host response to the microbial challenge seems to be more pronounced and rate of PID progression is found to be high after 3 months. This could be attributed to lack of periodontal ligament and to the absence of supra-crestal connective tissue fibre compartment to wall of the disease. Qualitatively physical and chemical properties of implant surface have been suggested to play a role in peri-implant disease. Microorganisms that are different from that causing periodontitis are said to play a role in PID. It has been found that Staphylococcus aureus shows affinity to Titanium surface and is associated with therapy-resistant cases of PID. An increase in surface roughness of the implant increases of osseointegration. But it has been found that, in Titanium implants the rough surface increases the risk of PID. This is minimised by using Titanium nitride or Zirconium nitride coated implants.

Hydroxyapatite coated implants show an

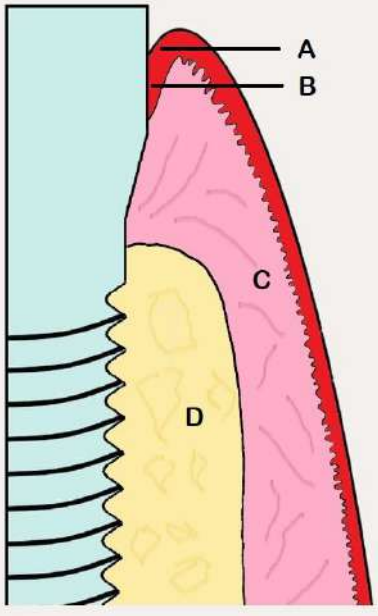

Fig-1: Schematic representation of a healthy implant-tissue interface. A) Sulcular epithelium B) Attachment epithelium C) Connective tissue D) Bone

increased risk in developing PID. This could be attributed to several factors. The endotoxin could have a greater affinity to hydroxyapatite coated surfaces, or the endotoxin may be bound to the implant surface by chlorhexidine and stannous fluoride due to charge interaction. The bonding between hydroxyapatite and bone is also questionable in the long term.

Risk factors for PID

Factors such as history of periodontitis, absence of regular follow-up, improper maintenance by the patient, and poor implant positioning by the surgeon have a strong evidence to be suggested as risk factors for peri-implantitis.[2] Factors like smoking, alcoholism, perioperative use of non-steroidal anti-inflammatory drugs or glucocorticoids, implant surface characteristics, titanium dissolution products, implant platform design, occlusal overload and proximal contact opening have inconclusive or limited evidence. Factors like irradiated bone, altered glycemic level, tissue phenotype,residual endodontic infection, excess cement and over contoured restorations have moderate evidence.[2]

Diagnosis

Implants should be evaluated every 3 to 6 months. Mucosa should be inspected for changes in colour, texture, and size. Probing should be done to assess bleeding and pocket depth. Bone loss and mobility should also be assessed. For an implant to be osseointegrated, it should resist a torque of 10-20 Ncm and produce a ringing sound on percussion.

Probing:

Probing around an implant causes tear in implant soft tissue attachment. This takes 5 days to heal with new epithelial attachment. Probing should be done with a non-metallic probe using 0.2 to 0.3 N force.[3] Audio Probe,titanium plasma-sprayed probes and HAWE-click probes are a few examples for standardized probes. While probing around the implant, the probe sometimes violates the connective tissue and is stopped only by the bone. So, any pocket depth that is less than or equal to 5 mm is considered normal around implant. An increase in pocket depth is a good indicator of the severity of the disease and directly reflects the bone loss in the area.

Radiographs:

Intraoral periapical radiographs (IOPA) should be taken using standardized film holders at the end of first year after implant placement and after every 2 years if the implant is asymptomatic. The baseline radiograph (one taken immediately after prosthesis placement) should reveal a fixed reference point, successive implant threads, and mesial and distal bone levels. The bone around the implant undergoes remodelling immediately after its placement, so bone loss of about 2 mm is considered normal during the first year. Use of digital subtraction radiography is found to be advantageous over conventional radiography.

Mobility:

Presence of mobility is an indicator of failed osseointegration. Periotest is a device that can measure

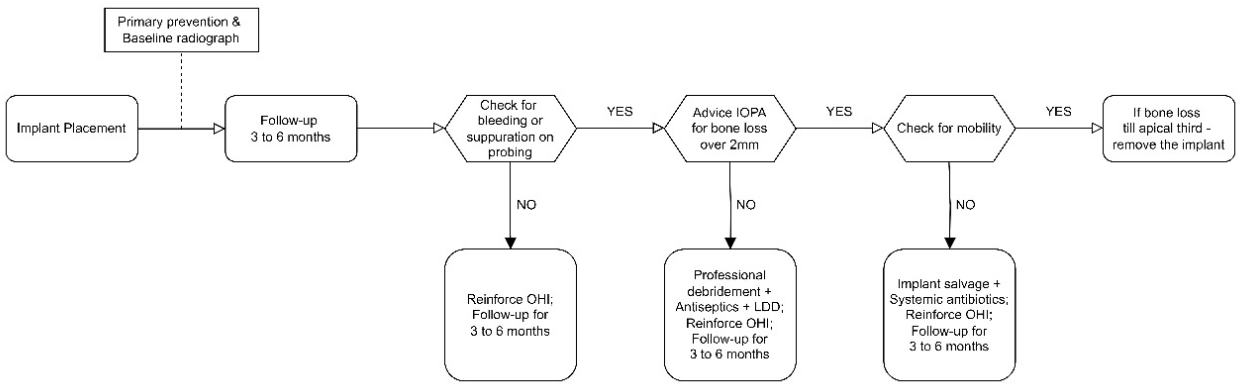

Fig-2: Algorithm depicting suggested management protocol for prevention and treatment of Peri-implant disease

mild difference in mobility by calculating the damping effect of peri-implant tissue. Osstell is a device that analyses the resonance frequency to evaluate mobility.

Peri-Implant Mucositis (PIM)

PIM can be defined as the presence of inflammation in the mucosa at an implant with no signs of loss of supporting bone.[5] The diagnostic criteria for PIM include bleeding and/or suppuration on probing, and the absence of additional bone loss after initial remodelling. This is similar to gingivitis except that there is more frequent suppuration, increased bleeding sites, and it takes more than three months for PIM to reverse. There may be an increase in pocket depth due to enlarged mucosa or decreased resistance to probing.

Peri-Implantitis

This is defined as "a plaque-associated pathological condition occurring in tissues around dental implants, characterized by inflammation in the peri-implant mucosa and subsequent progressive loss of supporting bone".[1] The exact reasons why PIM develops into peri-implantitis is unknown. When data of earlier examinations are available, bleeding or suppuration on probing, increased pocket depth, and presence of additional bone loss after initial remodelling are suggestive of peri-implantitis. When data of trial evaluations are unavailable, bleeding on or suppuration on probing, pocket depth greater than or equal to 6 mm, and bone loss of 3 mm or more can be used as diagnostic criteria. Sometimes hard and soft tissue deficiencies may be associated with peri-implantitis.

Periapical Peri-Implantitis

Few cases with peri apical radiolucencies without overt clinical signs of inflammation have been reported. They have been found to be correlated with retrograde peri-implantitis and endodontic periapical lesions in adjacent teeth. They might occur at any time following implant placement.

Prevention of Peri-Implant Disease

The periodontal condition of the patient should be analysed before placing the implants. If the patient is found to be suffering from periodontitis, it should be treated before implant placement. Once the implant is placed, the patient should follow oral hygiene measures meticulously. Toothbrushes, either handheld or powered, interdental aids like dental floss, interdental brush, and end tufted brushes should be used. Chemical plaque control can be advised as an adjuvant to mechanical plaque control. Oral irrigation devices can be prescribed for use. Irrigation either supra or subgingival will decrease the bacterial load in the area.

Management

The management of peri-implant disease is time sensitive. Early diagnosis and prompt treatment is the key to save the implant. A suggested algorithm for this is presented in Fig-2.

Peri-implant mucositis:

The primary cause of peri-implant disease is the bacterial endotoxin. Its management therefore lies incomplete anti-infective therapy. This includes professionally delivered mechanical debridement, with or without use of antiseptics and local drug delivery. Mechanical debridement can be performed with a curette or ultrasonic scaler. In case of curettes, steel curettes should not be used on titanium implants for the fear of scratching the implant and causing a galvanic corrosion. However, they can be used on titanium zircon oxide or titanium oxynitride implants. Titanium coated curettes, carbon fibre curettes and Teflon curettes can be used on any implant surface. In ultrasonic scaler polyether-ether ketone coated tips are preferred. These tips have a central steel part with high resilient plastic on the surface. This provides good debridement of the sulcus, without causing any damage to the implant. Abrasive systems like sodium bicarbonate- air powder system or rubber cup with polishing paste can also be used. Antiseptics like chlorhexidine can be used as a subgingival irrigant or applied as a gel. Local drug delivery can be used after debridement. Full mouth disinfection, as described by Quirynen, can be done after mechanical debridement around the implant.[6] Tetracycline fibres can be used to provide anti-infective therapy. However, it has been found that the addition of antiseptics and local drug delivery does not add much benefit to mechanical debridement.[7]

Secondary prevention at home following treatment of peri-implant mucositis is essential for

long-term success of the implant. This is similar to primary prevention. It has been found that the use of chemical plaque control is beneficial with mechanical block control in this criterion.[7]

Peri-implantitis:

Management of peri-implantitis, also known as implant salvage, is based on the amount of bone remaining at that site. In the initial stages, non-surgical therapy is indicated. This is similar to the peri-implant mucositis management. If the patient has periodontitis in other regions, it is treated at the same time as peri-implantitis management.

Surgical management of peri-implantitis includes flap reflection, and debridement/decontamination of the implant. Sometimes regenerating or resective procedures are performed. Use of systemic antibiotics is considered beneficial.[8] This is because peri-implantitis is rapidly progressing and this progression is different from periodontitis. Clindamycin (150mgtid), or Doxycycline hyclate (100mg bid), or Amoxycillin without clavulanic acid (500mg qid) and with Metronidazole (400 mgtid) may be prescribed. This can be selected after doing antibiotic sensitivity test. This should be started two days before the surgery and should be continued till 10 days after the procedure. The flap technique for this procedure is the access flap. Sometimes apically displaced flap can be used in areas of less esthetic concern or if the bone defect has a more horizontal component. Once the flap is reflected debridement is done, decontamination of the implant is based on its surface material. If the implant has hydroxyapatite coating, application of anhydrous citric acid at 40% and pH 1 for 30 seconds to 1 minute is done.[8] Any signs of deterioration on its surface should be treated by grinding this layer completely. For titanium implants, air power abrasives, saline, or citric acid application for one minute is done. Use of laser for decontamination is studied widely. Carbon dioxide laser has not shown any superiority over simple decontamination with saline. Er-YAG laser decreases bleeding on probing, better pocket depth reduction and clinical attachment loss. Photodynamic therapy has also been tried for decontamination procedure.

Regeneration can be attempted by guided bone regeneration with the help of expanded poly tetra fluoro ethylene membrane with bone grafts. Topical application of tetracycline 50 mg per ml on decontaminated implant for 3 minutes before attempting regeneration is preferred.[8] The membrane is left undisturbed for at least six weeks before removal. In case there is a premature exposure, the membrane should be removed immediately, to prevent further contamination. Some cases require resective procedures like implantoplasty for better management. If bone loss is upto apical third of implant, it should be removed.

Conclusion

Implant can be used as an ideal replacement to lost tooth if PID is prevented. Appropriate preventive measures, regular follow-up, early diagnosis and prompt treatment can help us manage PID. As the treatment does not guarantee prevention of PID recurrence, it is mandatory that the patient realises the importance of oral hygiene maintenance. Management of PID solely rests on the shoulders of the periodontist. Our community should be aware of the latest information about the disease, and its management. We should implement this in our day-to-day practice and try to eradicate PID.

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Can Socket Shield Technique with Immediate Implant be a Solution for Ridge Preservation in Maxillary Anterior Region? – A One Year Follow Up Case Report

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ABSTRACT

Alveolar bone loss after extraction causes ridge collapse and makes implant placement difficult. To overcome this, there are various ridge preservation techniques. Socket Shield Technique is one of the modalities of Partial Extraction Therapy where a section of root is left in the extraction socket. It helps in preserving the bundle bone apparatus and maintains the hard tissue and soft tissue contours mainly in esthetic areas. This case report directs on replacing maxillary anteriors using Socket Shield Technique with immediate implant placement.

Key-words: Socket Shield Technique, Maxillary Anteriors, Esthetic Zone, Immediate Implants

Introduction

Tooth extraction leads to ridge collapse which hinders ideal implant placement. There are various techniques to preserve alveolar ridge dimensions which helps in minimizing the necessity for alveolar ridge augmentation at a later stage. Socket Shield Technique is a procedure is a ridge preservation technique which involves intentional retention of a section of the remnant root to preserve the buccal/proximal bone from resorption. It has been proved to improves pink esthetics by maintaining the bundle bone-PDL apparatus with minimal marginal bone resorption particularly in esthetic regions. This case report focuses on replacing maxillary anteriors using Socket Shield Technique with immediate implant placement.

Case Report

A 23-year old male patient reported to the Department of Periodontology and Implant Dentistry with the chief complaint of missing tooth in the upper anterior region (Figure 1). He was apparently healthy with no contributing medical history. The patient requested reconstruction of the maxillary anteriors with replacement of maxillary right and left lateral incisors. Clinically, the recorded parameters showed healthy soft tissues with probing pocket depth ranging 1-2mm at all the six sites in the maxillary right central incisor. Corresponding contralateral central showed similar probing pocket depth.

After initial examination, patient was advised for IOPA in relation to 11 which revealed root canal treated teeth with only one third of the crown remaining(Figure 2). An attempt to preserve the maxillary right central incisor with root canal treatment failed. Several treatment options were discussed with the patient regarding best replacement method of the non-restorable tooth; all risks and benefits for each

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treatment option like conventional fixed bridges, resin bonded bridge and implant supported crown were explained. The patient gave consent to perform socket shield technique with immediate implant placement in 11 and conventional implant in 22.

The area to be operated was anaesthetized by infiltration with 2% lignocaine 1:80,000 epinephrine and the crown was hemisected using a coarse bur (Figure 3). Then the root was dissected mesio-distally along the long axis down to the apex using a long shank root resection bur connected to a hydrated high speed hand piece. The root was split into labial and palatal fragments. Using Periotome (HuFriedy Periotome, Anterior PT2), the periodontal attachment was severed in between the palatal fragment and socket wall. The palatal fragment was removed with high caution without disturbing the labial fragment attached to the labial bone. Using a round diamond bur, the remaining labial root fragment was contoured, the height of the labial shield was trimmed to 1mm coronal to the alveolar crest (Figure 4) and the thickness of the shield was 1.5mm mesio-distally following the concave contour (Figure 5).

Curettage was performed in the extraction socket and saline irrigation was done to remove the root remnants. Using a probe, the socket shield was checked for its stability. The osteotomy site was prepared using sequential drills in the socket palatal to the shield. The implant of size 3.3x16mm (NORIS) was placed without touching the shield with an optimum gap of 1.5mm from the shield and the jumping distance was left ungrafted (Figure 6).

Simultaneously, osteotomy site was prepared using conventional drilling technique in relation to 22 and implant of size 3.3x13mm (NORIS) was placed. On inspection, there was a minimal thread exposure at the implant site in relation to 22 (Figure 7) which was counter grafted by alloplast graft material (B-OstIN, Beta tricalcium phosphate). Both the osteotomy site was closed without tension using 3-0 silk sutures with

Figure 1: Pre-Operative image of Fractured 11

Figure 2: Pre-Operative IOPA

Figure 3: Mesio-distal dissection of tooth

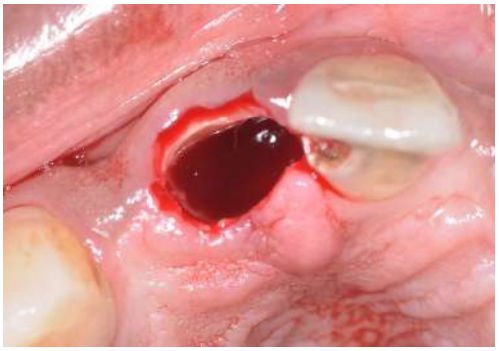

Figure 4: Contoured buccal shield in 11

Figure 5: IOPA showing buccal shield

Figure 6: Implant placement in 11

simple interrupted sutures in 11,22 respectively (Figure 8). The patient was put on antibiotic (Amoxicillin 500mg and Metronidazole 400mg) and analgesic (Zerodol-SP) for 5 days. The patient was provided with a removable prosthesis for the period of healing and was given oral hygiene instructions.

A post-operative examination was performed after one week and follow up visits were scheduled for one month and three months post operatively which showed uneventful healing. The site was allowed to heal for a period of four months. At 4 months after surgery, a periapical radiograph verified radiographic healing (Figure 9) and healing abutment was placed in both maxillary right central incisor and maxillary left lateral incisor (Figure 10).

Root canal treatment and Tooth preparation was

Figure 7: Implant placement in 22

Figure 8: Sutured with 3-0 silk

Figure 9: IOPA showing Radiographic healing

Figure 10: Socket Shield integrated with implant

Figure 11: Healthy soft tissue collar in 11, 22

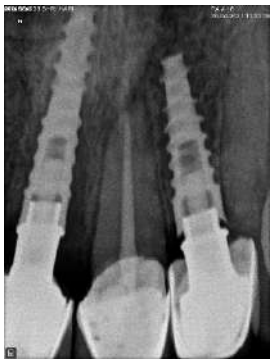

Figure 12: IOPA with Screw Retained PFM in 11, 22

Figure 13: Post operative image

Figure 14: Oblique smile

done in relation to 21. After 2 weeks, at the removal of healing abutment, healthy soft tissue interface was noted (Figure 11). Cement retained porcelain-fused-to-metal crowns (Figure 12) were placed on each implant with cantilever crown in 12 (Figure 13&14). The patient was on periodic recall visits and proper oral hygiene instructions were given accordingly.

Discussion

Healing of extraction socket is a dynamic process where approximately 50% of the alveolar bone width is lost within 12 months after extraction, and around 30% occurs within the first 12 weeks, mainly because of the loss of the buccal plate of the alveolar bone.[1] The buccal bone receives blood supply from the gingiva as well as the periodontium. So, when the tooth is extracted, buccal bone is deprived of the blood supply from the socket wall which leads to buccal bone resorption.[2]

Buccal bone loss seen in the alveolar bone after tooth extraction hinders ideal implant placement. Though osseointegration remains the predominant parameter of success in implant dentistry, recently new parameters like peri-implant soft tissue level, prosthesis level, patient's subjective assessment are also considered to assess implant success. Patients nowadays are more concerned for esthetics and the focus is shifting from implant survival to creation of naturally looking and healthy implant and peri-implant tissue levels.[3]

Alveolar bone loss after extraction can be counteracted by various ridge augmentation and ridge preservation techniques.[4] Many studies have demonstrated the preservation of alveolar bone at the extraction site by retaining decoronated roots, either vital or endodontically treated. Hurzler et al described the socket shield technique (SST), where implants are placed in close proximity to the intentionally retained roots to preserve the buccal bone.[5]

Socket Shield Technique is one of the modalities of Partial Extraction Therapy, where the shield is created by preparation of a tooth indicated for extraction at an immediate implant placement site, most commonly in anterior maxilla. The biological principle behind SST is that it preserves a part of the root in the socket so that periodontium, the bundle bone and the buccal bone remains, intact, which arrests the expected post extraction alveolar socket remodelling.[6]

SST is a minimally invasive surgical procedure, which preserves both hard and soft tissue contours by preserving a part of the root. It also minimizes the need for future hard and soft tissue augmentation thereby saving time and shortening the duration of the treatment.

This technique proves to be valid in cases of esthetically demanding areas with adequate pink and white esthetics in cases of maxillary anteriors by preserving the interdental papillae.[6]

In this case report, the tooth selected for socket shield preparation was periodontally healthy, without mobility or radiological pathology. Periodontally weak teeth might affect the long-term stability of peri-implant tissues, by interfering with implant bed preparation and placement. The present case had failing endodontically treated tooth which was clinically eligible for socket shield technique. As reported by Kumar PR and Kher U the optimum gap between socket shield and implant is 1.5 mm or more and a bone graft is suggested if the gap is more than 3 mm. As the gap was less than 3mm, grafting was not done in the present case.

Also, after 6 months of healing, it was noticed that attached gingiva width was more in socket shield site when compared to the adjacent implant site 22 because of the post extraction bone loss which was counteracted by socket shield in 11.

Various studies showed altering success rate with this technique. A retrospective study by Gluckman et al,[7] showed the 4 years survival rate of 96.1% for 128 immediate implants whereas Sirompas et al,[8] showed 98% survival rate with socket shield technique.

Recently, a systematic review and meta-analysis by Velasco et al[9] showed lower marginal bone loss, low implant failure rate (1.37%) and higher pink esthetic scores in socket shield technique when compared to the conventional technique.

Conclusion

SST is a highly promising technique which may significantly alter future management of the failing dentition and postextraction ridge, through a transformation from extract and augment to salvaging

the patient's own tissues where possible. Though it is technique sensitive, its success greatly depends on the operator's skill. The present case report demonstrates the SST's potential for highly esthetic outcomes, with reduced time and expense.

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Periodontitis as a Risk Factor for Cancer : Review

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ABSTRACT

Periodontitis is a chronic infection caused by inflammatory reactions in response to microorganism in endogenous dental plaque. The condition is characterizedby epithelial proliferation and migration, release of inflammatory cytokines, chemokines, prostaglandins, growth factor and enzymes all of which are closely associated with carcinogens. The link between periodontitis and systemic diseases has been examined since long, and there has been a notable increase in the interest in understanding the possible association with cancer. Aim of this review article is to review the published literature regarding chronic periodontitis as a risk factors for oral cancer. Relevant published studies were selected which states that chronic infection such as periodontitis can play a direct/indirect role in carcinogenesis.

Keywords: carcinoma, inflammation, microorganisms, poor oral hygiene, virus, oral cancer, periodontal disease.

Introduction

Periodontitis is an infectious disease caused by gram-negative anaerobic microorganisms in the dental biofilm, resulting in inflammation within the supporting structures of the tooth, leading to progressive attachment and bone loss, characterized by pocket formation and recession. Periodontitis leads to epithelial proliferation and migration, which results in the chronic release of inflammatory cytokines, prostaglandins, growth factors, and enzymes, all of which are closely associated with the development of cancer.[1]

Ample research evidence has suggested that chronic infections and inflammation are related with increased risk of cancer. The role of bacterial and viral infections in carcinogenesis has also been highlighted.[1]

There has been a notable interest in understanding the role of periodontitis and risk of cancer, due to the advancement of research on human microbiome and the advent of animal models, enabling the assessment of the effect of periodontal pathogens on immune response.[2]

Cancer Ascertainment

Incidence of cancers were ascertained from 1987 to 2012 through linkage with state cancer registries in Minnesota, North Carolina, Maryland, And Mississippi, and supplemented by abstraction of medical records and hospital discharge codes for self-reported cases (supplementary methods, available online). Cancer deaths were obtained from death certificates where cancer was the underlying cause of death. From enrolment through 2012, 4107 first primary cancer cases and 1661 cancer deaths were noted . Out of which , 3748 were first primary cancers and 1332 were cancer deaths that occurred after visit[4]. Of these, 1648 and 547, respectively, occurred among

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those in the study population over a mean of 14.7 years of follow-up

A. Evidence for association

Individuals diagnosed with cancer often refrain from regular brushing of teeth and usage of cleaning aids. This can be attributed to the fact that they suffer from depression, which is often not diagnosed and treated. As the therapy starts , patients may undergo chemotherapy /radiotherapy, which have negative affect on oral hygiene habits. Poor maintenance of oral hygiene along with a depressed state of mind, increases the likelihood of gingival bleeding and burden of periodontal infection.[2-5] The association between poor oral health and cancer has been substantiated in several studies. Compromised oral health was observed in nasopharyngeal carcinoma survivors (1-4 years after radiotherapy).[7]

A study on 73 patients diagnosed with acute myeloid leukemia (AML), pointed that about three-quarters of the patients with acute myeloid leukemia showed poor or fair oral hygiene.[8]

A prospective study, evaluated the link between tooth loss and the risk of developing esophageal squamous cell carcinoma, gastric cardia adenocarcinoma or gastric non-cardia adenocarcinoma in a 28,868-person cohort, and was followed prospectively for 5.25 years. The analytic cohort included 620 esophagus, 431 gastric cardia, and 102 gastric non-cardia cancer cases. In this cohort study, tooth loss increased the risk of developing upper gastrointestinal cancer. It was stated that this may be related to alterations in the oral bacterial flora and subsequent increases in the in-vivo production of carcinogens, such as, nitrosamines.[9]

Recent study conducted JNCI National CANCER INST 2018 on assessing periodontal disease assessed using clinical dental measurement and cancer risk in ARIC study. This study gave additional evidence that cancer risk, especially for lung and colorectal cancer, increases in individuals with periodontitis. Additional research is essential to understand cancer site–specific and racial differences in findings.[10]

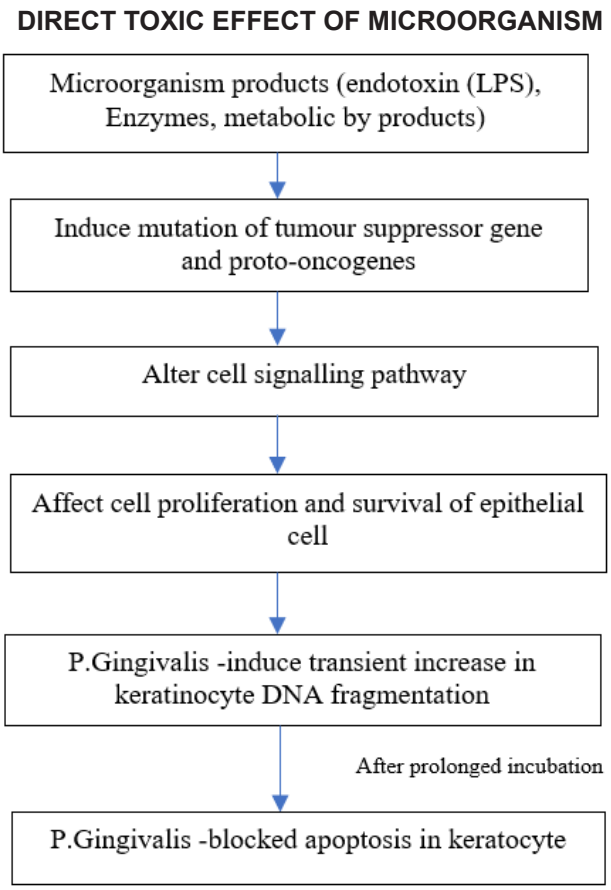

Figure 1A: Direct Toxic Effect of Microorganism
P. Gingivalis-major periodontal Pathogen, capable of invading Epithelial cell

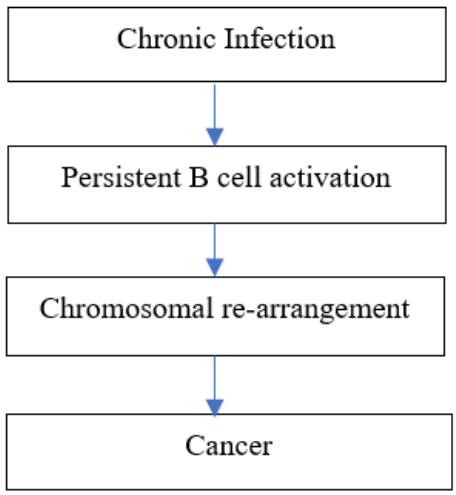

Figure 1B: Example of Direct Toxic Effect of Microorganism.
Direct Bacterial stimuli – in association between Helicobacter pylori and primary B cell gastric lymphoma

The Relationship Between Chronic Periodontitis and Oral Cancer

Genetic predisposition, presence of premalignant lesions, effect of environmental or carcinogenic factors, such as tobacco and alcohol consumption, are some of the risk factors suggested as risk factors for the incidence of oral squamous cell carcinoma. The role of infection and inflammation with human papilloma virus has also been noted in oropharyngeal carcinomas[9] along with the role of chronic periodontitis in the oral cancer development.[10,11]

In a recent study, bone loss, a clinical sign of chronic periodontitis was indicated as an independent risk factor for the development of cancer in the oral cavity. The research concentrated on the different types of cancer , their association with the inflammation and the pathophysiological mechanisms behind malignant cellular transformation.[12] A key factor in inflammation linked with cancer is NF-ϰb transcription factor, a basic regulator of the innate inflammation and immunity through an activated expression of inflammatory biomarkers namely cytokines, chemokines, adhesion molecules, proteolytic enzymes, angiogenic factors and a wide spectrum of toxic metabolic cellular substances. It therefore plays a important role in the inflammatory reaction in chronic periodontitis as well as a potent endogenous tumor promoter.[9]

Furthermore, inflammation, despite the underlying etiology, can predispose to cancer development,[13] thereby explaining the malignant transformation of the oral epithelial cells. This may be a adverse effect of an immune response via the release of activated t-cells substances, such as cytokines IL-1, IL-8 and TNF-α.[14]

The biological plausibility of the association between periodontitis and cancer may be interpreted on the basis of the following mechanisms,[1]

1. Periodontal disease - facilitating enhanced carcinogen penetration (smoking and alcohol), disrupt the mucosal barrier

2. Chronic inflammatory response causes a chronic diffuse hyperplasia of the epithelial cells (increased cell load in the blood vessels and the connective) indicated as a common precursor of the intraepithelial neoplasia

3. Immunosuppression is a common mechanism that leads to periodontal disease and oral cancer

4. Viruses such as human papilloma virus and herpes simplex virus[1] or the fungus Candida albicans have been isolated in both oral cancer and periodontal disease

5. In patients with poor oral hygiene, bacterial overload can lead to an increase in the proportion of metabolites with a potential carcinogenic effect

6. In dizygotic twins, who share common genetic risk factors, periodontal disease is a risk factor for cancer development, whereas in monozygotic twins this association was significantly impaired.[16]

Recent studies reported that some viruses such as human papilloma, cytomegalo virus and Epstein Barr present in periodontal pockets and in dental plaque,[17,18] are implicated in oral cancer etiology.

DIRECT TOXIC EFFECT OF MICROORGANISM

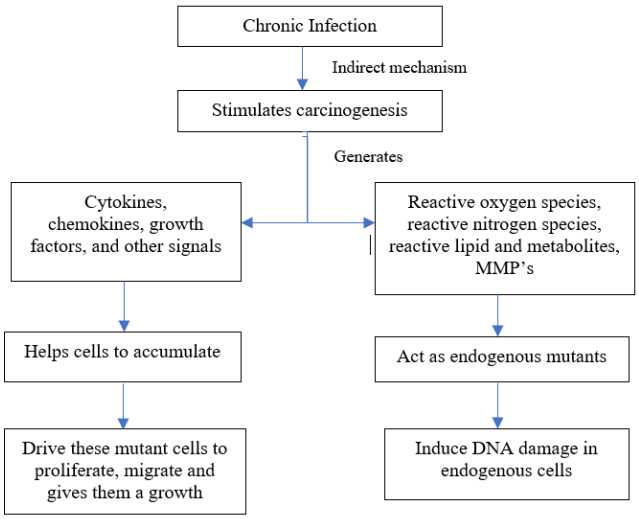

Figure 2: Indirect effect through inflammation. MMP-Matrix metalloproteinases

Bacteria and Viruses in Associated Cancer

Several studies have established the role of helicobacter pylori in development of gastric cancer and chlamydia pneumonia infection with lung cancer. Streptococcus bovis infection was found to be associated with colon cancer while other bacteria such as salmonella typhi was linked with gall bladder cancer

and hepatobiliary carcinoma.[19]

Epstein-Barr virus is believed to be associated with Burkitt lymphoma, Hodgkin lymphoma, nasopharyngeal carcinoma, and stomach cancer. Human herpes 8 is associated with kaposi sarcoma. The hepatitis b virus has its involvement in liver cancer and human t-lymphocytic virus with adult T-cell leukemia. The cytomegalovirus is associated with cancer of the salivary gland and prostate. The human papilloma virus is linked with cancer of the cervix, vulva, penis, and anus. [20]

It has been suggested that herpes viruses might impair local host defense by disrupting the protective epithelial barrier by lysing the epithelial cells, or by altering the expression of cell surface receptor molecules. Human papilloma viruses, cause characteristic cytopathic effects (koilocytosis), a proliferation of epithelial cells. It is known that proliferation and migration of the junctional epithelium are a major hallmark of periodontal breakdown, these known biological effects of HPV might provide a link between viral infection and periodontal disease. Productive infection by HPV is tightly linked to the differentiation program of the host keratinocyte. Following entry into the basal epithelial cells, HPV genomes are established as autonomous replicating extra-chromosomal elements and a low level of HPV expression occurs. Upon differentiation of infected cells, productive replication and expression of capsiden coding genes (L1 and L2) is induced, resulting in the production and assembly of mature viral particles. Theoretically, the junctional epithelium attached to the tooth surface appears to fully serve the cellular functions required by HPV. It has a basal cell-like phenotype and does not differentiate. The basal cells are exfoliated through the gingival crevice before differentiation occurs.

Biological Mechanism

Chronic infections, such as periodontitis, play two roles in carcinogenesis :

- Direct toxic effect of microorganism (Fig. 1A, 1B)
- Indirect effect through inflammation (Fig. 2)

Another link was explained between periodontal disease and risk of pancreatic cancer. The carcinogenic compounds, namely, nitrosamines and bacteria, react with the digestive chemicals in the gut to create conditions that favour the development of pancreatic cancer.[10]

The shared genetic factors may partially explain the association between cancer and periodontal disease, in the case of digestive tract cancer.[19] There are many other risk factors modifying the development of cancer. Smoking and usage of smokeless tobacco have also proved to be the main risk factors associated with cancer development.[22] The strength of association is higher in a past smoker or a 'never smoker' when compared to current smokers.[1] Alcoholism along with smoking gives an additive effect in carcinogenesis.[21] Diet, age, gender, and family history also play an important role in carcinogenesis.

Conclusion

Summarizing the review, the studies done by various authors throw light on the fact that compromised oral health may prove a risk factor for carcinogenesis. The association between periodontal disease and oral and/or oropharyngeal cancer may have implications for public health in preventive measures and for the patients who might experience major improvements in the complications of cancer and its treatment. An association between periodontal disease and cancer may lead us to consider patients with periodontitis as population at risk for developing oral/oropharyngeal cancer. In considering all the studies it is obvious that there is a need for further investigation.

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Periodontal Microsurgery: A Review

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ABSTRACT

Microsurgery is one of the rapidly emerging trends in the field of Periodontology. It is widely accepted by both the patients and clinicians as it improves the ergonomics of the clinicians and also increases the rate of compliance level among the patients. Thus it has slowly shifted the focus of periodontal therapy from macrosurgical to microsurgical level. Magnification systems evolved through a fledgling state from lower magnification systems such as magnification loupes to the more advanced surgical operating microscopes. Periodontal microsurgery not only involves magnification systems but also includes appropriate use of microsurgical instruments with adequate illumination. Magnification systems, microsurgical armamentarium and its role in the field of both non surgical and surgical periodontal therapy are highlighted in this article.

Key words: microsurgery, magnification loupes, surgical microscope, microsurgical instruments.

Introduction

In contemporary surgical practice there are many prodigious evolution in order to accomplish therapeutic objectives and to fulfill the patient's expectations. One such innovation is the introduction of magnification systems in the periodontal surgical practice.

Periodontal microsurgery provides adequate visibility of the surgical field, enhanced precision of the surgical technique, better tissue handling with less tissue trauma and healing by primary intention with less postoperative morbidity.

Introduction of magnification systems in periodontal surgeries have shown a great leap in the surgical intervention and is a boon to periodontology. This review article pivots mainly on the magnification systems, microsurgical armamentarium and their clinical applications in the field of periodontology and its future perspectives.

Definitions of Periodontal Microsurgery

- In 1979, Daniel defined microsurgery in broad terms as surgery performed under magnification by the microscope[1].
- In 1980, Microsurgery was described by Serafin as a methodology – modification and refinement of existing surgical techniques using magnification to improve visualisation with applications to all specialities[1].
- Dennis.A.Shanelac and L.S.Tibetts, defined microsurgery as a refinement in surgical technique by which visual acuity is increased using a microscope at magnification exceeding 10x[2].
- In 1992 Shanelac, defined periodontal microsurgery as refinement in existing basic surgical techniques that are made possible by the use of surgical microscope and subsequent improved visual acuity[3].

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History of Microsurgery

Microsurgical techniques are utilised in different specialities like ophthalmology, orthopaedic, gynaecological surgery, otolaryngology, neurosurgery, plastic surgery and in dentistry as well. Even though it is widely used in different specialities, its use was first reported by a Swedish otolaryngologist Carl-O-Nylen in late 1800s[4].

He introduced monocular Brinell–leitz microscope in the University of Stockholm and used it in the management of labyrinthine fistula in chronic otitis patient in the year 1921[1]. Nylen's monocular microscope was later replaced by a binocular microscope in the year 1922. Because of his pioneering work, he is called as the father of microsurgery[1].

The first surgical operating microscope was introduced in 1950, with coaxial lighting system with stereoscopic view by the Carl zeiss company[5].

In 1950 Barraquer used microscope in corneal surgery[6].

Neurosurgeons Jacobsen and Suarez in 1960 Donaghy and Yesargil in 1967 introduced microsurgical technique in vascular surgery[5].

In 1978, Apothekar and Jako established the concept of microsurgery in dentistry[7].

Microsurgical repair of nerve injuries following trauma was introduced by Leblanc JP and Van Boven RW[8].

In 1992 Carl introduced the use of surgical operating microscope in endodontics[9]. Microsurgery in the surgical practice of periodontics was initiated by Tibetts and Shanelac in the year 1992.

In 1993 Shanelac and Tibett introduced the continuing education course on periodontal microsurgery at the annual meeting of American academy of periodontology[7].

Kim in 2001, proposed the term microsurgical triad that comprise magnification, illumination, and instruments in microsurgery[1].

Principles of Microsurgery

1. Enhancement of motor skills which increase the precision of the surgical technique[4].

2. Passive wound closure with exact opposition of the wound margins in order to eliminate dead spaces at the wound edges[4].

3. Reduced tissue trauma with the proper use of microsurgical instruments and microsurgical suture material[1].

Belcher et al in 2001 confined these three principles such as, magnification, illumination, microsurgical instruments together as a triad called microsurgical triad[10].

Magnification Systems in Microsurgery

Magnification Loupes:

Magnification loupes are basically two monocular microscopes with side by side lenses angled to focus an object. It is based on the keplerian optical system. The magnification of the loupes ranges from 1.5x -10x. Magnifying loupes with magnification less than 2x are often inadequate for the visual acuity in microsurgery. The optimum magnification for the periodontal microsurgical procedures is 4x-5x.

Simple Loupes:

It contains a pair of side by side meniscus lenses each containing two refracting surfaces[3]. The magnification can be increased by increasing the diameter of the lens and the thickness of the lens[3]

Range of magnification: 1.5x[5]

Advantages:

1. It is cost effective.

Disadvantages[3]:

1. It is highly subjected to spherical and chromatic aberrations which distorts the image of the object.

2. The magnification can be increased only by increasing the thickness and diameter of the lens which may eventually lead to increase in weight of the magnification loupe.

3. Because of their limitations in size and weight, it offers no application beyond the magnification range of x1.5.

4. It is highly difficult to maintain the focus.

FIG. 1: Simple loupe

Compound Loupes:

It consists of two glass pieces of an achromatic lens bonded together with clear resin. The lenses are usually mounted on the eyeglass[5]. It is of converging multiple lens type with intervening air spaces to gain additional refracting power, magnification, working distance, and depth of field.[7]

Range of magnification is up to 3x5.

Advantages:

1. Magnification can be increased with significant working distance and field of vision without increasing the thickness and diameter of the lenses[11]
2. Lenses are achromatic – specific density of each lens counteracts the chromatic aberration[5].
3. Optimum optical effect.
4. Improved ergonomics.

Disadvantages:

1. It is optically inefficient at magnifications beyond 3x.
2. Individual light source may be required.
3. Protective coating of anti-reflective material is needed to prevent the loss of light transmitted.

FIG 2. Compound loupe

Prism Loupes (Wide Field Loupes):

It consists of prisms which are fixed at the top called as rooftop or Schmidt prisms and it refracts the light rays. It lengthen the path of light by virtually folding the light through a series of switchback mirrors present between the lenses and hence it contains shortened barrel. The barrel can be mounted on the eyeglasses or head band and thus it is more comfortable and stable than the other magnification loupes[11]

Range of magnification: 1.5x -6x4

Advantages:

1. Provides better magnification.
2. Wider depth of field.
3. Longer working distance.
4. Larger field of view.

FIG 3. PRISM LOUPE

Surgical Operating Microscope:

The surgical operating microscope (OPM) is the higher magnification system of lenses which provides binocular viewing at magnification ranging from 4x-40x12. The operating microscope contain fully coated optic achromatic lens with high resolution and high contrast. The surgical operating microscope follows the gallilean principle of optics[3]. The parts of surgical operating microscope include[3]

Magnification Changer[3]:

Magnification changer also called as galilean changer contains one cylinder with two galilean telescope systems. The magnification can be changed either through the manual system or motor system of magnification changer. The magnification can be changed more quickly with manual magnification

changer than the motorised one.

Objective Lens[3]:

The image is projected by a single objective lens. The commonly used objective lens is 200mm. The focal length of the objective lens usually corresponds to the working distance of the object and thus it determines the distance between the lens and the surgical area.

Lighting Unit[8]:

The lighting unit is a key feature of the surgical operating microscope where incandescent, halogen and fibre optic lamps are used for illumination. It provides a whiter light than the conventional lamps because of its higher temperature[13]. Xenon lamps are also available which is 10 times more effective than the halogen lamps as it produces brighter images with sharp contrast[1]

Binocular Tubes[3]:

Binocular tubes are of two types, straight and inclined type. In straight tubes the direction of the view is almost parallel to the axis of microscope. In inclined type direction of the view is about 45 degrees to the microscopic axis.

Eyepieces[3]:

The eyepieces enhance the image produced in the binocular tube. Eyepiece selection not only determines the magnification, but also the size of the field of view. Eyepieces are available in the powers of 6.5x, 10x, 5.5x, 12.5x, 16x and 20x3. The 10× eyepiece generally provides a sufficient compromise between magnification and field of view. Modern eyepieces allow a facility within −8 to +8 diopters that is a purely spherical correction.

Advantages:

1. Decreased trauma, painless and less anxiety.
2. Enhance the esthetic outcome.
3. Increased outcome predictability
4. Increased acceptance by the patient.
5. It allows wound apposition with minimal gaps or voids at wound edges and thus it facilitates faster wound healing.

Disadvantages[8]:

1. It is more difficult to use.
2. It is more expensive.
3. It is more difficult to master the technique.
4. Longer adjustment period for clinical proficiency.
5. Initially, the clinician may feel longer duration of the surgical procedure.
6. Restricted area of vision and loss of depth of the surgical field as the magnification increases.

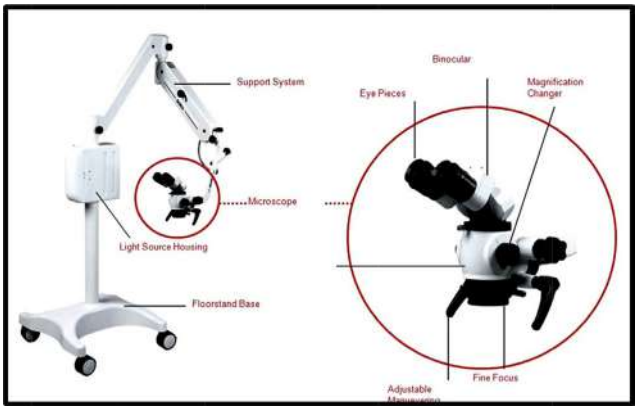

FIG 4. Surgical operating microscope

Microsurgical Armamentarium

The microsurgical instruments are circular in cross section in order to facilitate rotational movement by the operator[14]. The microsurgical instruments are made of titanium which provides increased strength, lightness and it is nonmagnetic in nature. Such instruments are also resistant to distortion from repeated use and sterilization. The working tip are much smaller than the conventional instruments. The instruments are 18cm long and 15-20 gm in weight in order to reduce the muscle fatigue[12].

Basic Microsurgical Kit Incudes:[15]

- ❖ Microsurgical blade - Swann Morton fine range
- ❖ Sharp and fine elevator - Buser papilla elevator
- ❖ Fine tissue pickups - Micro Adson forceps
- ❖ Fine needle holders - Castroveijo pattern
- ❖ Fine scissors - Castroveijo scissors
- ❖ Microsurgical blade - Swann Morton SM 64
- ❖ Fine sutures -6-0 Prolene, Ethicon

Microsurgical Needle Holder:

The length of the microsurgical needle holder vary and the most commonly used are 14cm and 18cm. It can be curved or straight. T. The tip of the needle holder vary depending on the type of suture. A delicate tip of 0.3mm is used for 8-10, 10-0 sutures and 1mm tip is used for 5-0, 6-0 sutures.

Microsurgical Forceps:

The microsurgical forceps are usually 15cm long with round handles and 0.2-0.3mm tips. Types of microsurgical forceps include

1. Straight forceps
2. Curved forceps

As the handles are flat, it restricts the rotational movement by the operator and hence it is less efficient.

Microscissors[12]:

Microscissors are available in 14cm and 19cm long. It is also available in straight and curved type.

Microsurgical Knives[12]:

Ophthalmic knives are used in microsurgery which are minimal in size and provide extreme sharpness. The smaller size of the ophthalmic blades offers precise incision with reduced surgical trauma when compared to the conventional blades. Microsurgical incisions are established at a 90-degree angle to the surface using ophthalmic microsurgical scalpels.

1. Blade –breaker knife:

It has a handle with the affixed ophthalmic blade. It is used in places of conventional 15 no blade.

2. Crescent knife:

It used for intrasulcular incisions and in connective tissue graft procedure. It is also used to obtain donor graft and also to prepare the recipient site.

3. Spoon knife:

It is used to undermine lateral sulcular region for the connective tissue graft placement.

Microsurgical Needle[12,2]:

The parts of the microsurgical needle include swage, body and tip. Finer microsurgical needles are used in microsurgery. The sharp 3/8 circular needles with reverse cutting edge with precision tip are commonly used in periodontal microsurgery[15]

Microsutures[16]:

The most common principle of microsurgery is the passive wound closure which is obtained by the proper apposition of wound edges A suture material must be selected that will retain its strength until the wound heals sufficiently to withstand stress on its own. Ideally, the needle and the suture material should be the same size. In periodontal microsurgery 6-0, 7-0 sutures are commonly used.

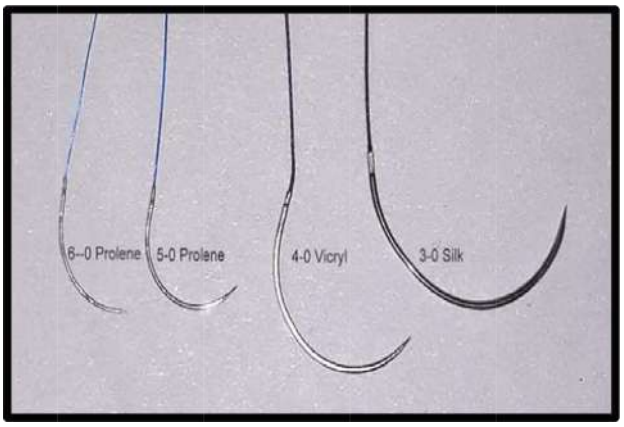

FIG. 5: Microsurgical Needles and suturres

Geometry of Microsurgical Suturing[9]:

1. The angle of entry and exit of the microsurgical needle is always less than 90 degrees.

2. The bite size for the suture is approximately 1.5 times the normal thickness of the tissue.

3. The bite should be symmetrical on either sides of the wound.

4. The passage of the microsurgical needle should be always perpendicular to the surface of the tissue.

Knot Tying[16]:

Higher magnification is used to place the sutures and lower magnification is used for tying the knot. The breaking force of the microsurgical sutures is always less than the human threshold of touch[5].

Applications of Microsurgery in Periodontology

Diagnosis:

Magnification helps in better visualisation of probe markings in the operating field and significant evaluation of the radiographs[17]. It plays a vital role in better visualisation of the soft tissue defects such as Stillman's cleft and an abrerrant frenal attachment[14]. It allows better determination of any residual calculus following periodontal therapy[17].

Scaling and Root Planing:

Lindhe and coworkers (1984) suggested that the success of the periodontal therapy is based on the complete mechanical debridement of the root surface[7].

Micro surgically treated mechanical debridement shows complete removal of plaque and calculus than the conventional method of mechanical debridement. Rashmi hedge et al in 2009 concluded that SRP done at 16x magnification surgical operating microscope showed a clean and smooth root surface[14].

Ranjana et al in 2013 concluded that efficacy of SRP improves with magnification especially under surgical operating microscope than the magnification loupes[18].

Crown Lengthening:

Periodontal microsurgery can also be employed in the crown lengthening procedures to permit precision in the surgical technique with finer incisions without any ragged edges and atraumatic handling of the soft tissues to enhance wound healing. Rashmi hedge et al 2009 used microsurgical technique in crown lengthening of maxillary left premolar using apically positioned flap[14].

Periodontal Flap Surgery:

Microsurgical approach improves tissue preservation and handling of specific flap designs and enhance the mobility of the flaps and provides primary wound closure[18]. It provides proper elevation of the periodontal flap with adequate uniform thickness and scalloped butt joint without the risk of perforation of the flaps[19]. It allows proper apposition of the flaps with no dead spaces and thus it favours complete periodontal regeneration.

Meena Priya et al in 2015 concluded that micro surgically assisted open flap debridement showed significant gain in clinical attachment level with reduction in probing depth and better results in wound healing with less postoperative pain[20].

Shreya Shetty et al in 2018 concluded that micro surgically assisted open flap debridement shows significant results in wound healing with minimal loss of tooth structure[21].

Root Coverage Procedures:

Microsurgical technique has found its way in root coverage procedures in order to meet the esthetic satisfaction of the patient. Microsurgery in the management of gingival recession defects show complete root coverage with improved width and thickness of keratinised tissue and better patient compliance than the conventional root coverage procedures. Microsurgical technique enhances the precision of the surgical technique with decreased haemorrhage and postoperative morbidity[5].

Patricia et al in 2010 concluded that microsurgical technique showed significant root coverage in miller class I and class II recession defects treated by coronally positioned flap with enamel matrix derivative[22].

Uditi Jindal et al in 2015 achieved a significant root coverage in miller class I and II recession defects treated by subepithelial connective tissue graft under microsurgical technique[23].

Omur uack et al in 2016 achieved complete root coverage in miller class III recession defects by microsurgically assisted laterally moved coronally advanced flap[24].

Regenerative Periodontal Surgery:

Microsurgically assisted flap design in the periodontal regenerative procedures result in increased percentage of healing in interproximal areas with significant attachment gain (cortellini and Tonetti 2001)[20].

Fickl et al in 2009 concluded that the use of enamel matrix derivative in the management of intrabony defects by microsurgical approach allows significant regeneration[25].

The advantages of microsurgical technique in the management of intrabony defects show improved illumination, better magnification of the surgical field, improve the access for proper mechanical debridement of the intrabony defects. It results in primary wound closure and enhanced periodontal regeneration[26].

Papillary Reconstruction:

Microsurgical approach produces a significant esthetic results in the management of papillary loss. Microsurgical approach is an atraumatic technique which allows the position of the donor tissue in the limited access area of the interdental papilla with the help of the microsurgical instruments and it facilitates improved visibility, greater precision of the technique, eliminates the releasing incisions[3].

Deepa jain et al in 2018 achieved successful reconstruction of papilla with improved esthetics by beagle technique under microsurgical approach[27].

Microsurgery in Implantology:

All the steps of implant procedure can be done under magnification which produces better benefits than the conventional technique. Surgical microscope allows the visualisation of even last thread of implant for subcrestal placement of implants and during recovery of implants with minimal trauma to the adjacent tissues and in the management of periimplantitis[1]. Microsurgical technique provides better sensation and prevents damage while placing the implants in the region of inferior alveolar nerve[2].

Sinus Lift Procedures:

Magnification employed in the microsurgical technique allows better visualisation of the sinus membrane during the procedure and thereby it reduce the occurrence of perforation. Sinus lift microsurgery shows a success rate of 97%.[28]

Future of Periodontal Microsurgery

Three Dimensional on Screen Microsurgery System (TOMS)[5,29]:

TOMS is a three dimensional system where the surgical field is viewed on the monitor rather than through the microscope in order to reduce the unnecessary strain to the eye.

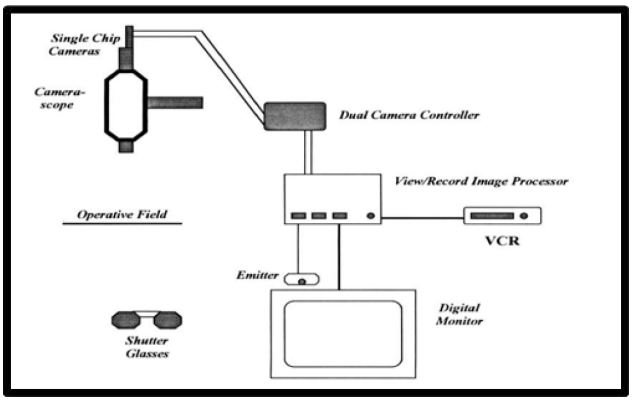

FIG. 6 Three dimensional onscreen microsurgery system

It includes,

1. Two single chip video cameras fixed on to the eyepiece adaptors
2. Dual camera holder
3. Record image processor
4. VCR for recording
5. Digital monitor
6. Signal emitter
7. 120 MHz shutter glass

Disadvantages:

1. Highly technique sensitive
2. Increased cost
3. It is a time consuming procedure
4. The field projected on the monitor is only the restricted view

Robotic Assisted Microsurgery[15,6]:

This system employs a tele robot with the computer controlled mechanical arms usually operated by the micro surgeon. The major disadvantage of the conventional microsurgical techniques is the unwanted physiologic tremor of the micro surgeon's hand during the procedure which ultimately results in decreased precision of the technique. This has been eliminated by the invention of robotic arms for the microsurgical procedure.

FIG. 7 Robotic microsurgery

Advantages of Robotic Microsurgery:

- ✓ Enhanced precision of the procedure
- ✓ Reduced surgical trauma
- ✓ Decreased haemorrhage
- ✓ Less postoperative pain
- ✓ Better patient compliance
- ✓ Decreased postoperative morbidity

Disadvantages of Robotic Microsurgery:

- ✓ More technique sensitive
- ✓ Not cost effective

Conclusion

Microsurgery has a wide range of applications in the field of periodontics. It offers optimum therapeutic benefits both to the dentist and patients.

✓ Recent magnification loupes are fitted with LED system which provides adequate illumination of the surgical field.

✓ Surgical operating microscope are provided with integrated video systems, photographic adapters for cameras, colour printers and LCD screen which helps in documentation and better patient education.

✓ The microsurgical instruments enhances the motor skill of the operator and enhance the precision of the surgical technique.

✓ It enhances the patient compliance with improved esthetic outcome.

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Evaluation of Prevalence of Mucosal thickening of Maxillary Sinus in Patients with Periodontal Bone Loss Using CBCT - A Clinical Retrospective Study

V.R. Balaji[1], M. Allan Jeba Seeli[2], D. Manikandan[3]

ABSTRACT

Background: Maxillary sinuses are covered by a 1 mm thick mucous membrane that when this membrane becomes inflamed, the thickness may increase 10-15 times. The Periodontal disease are the common causes of odontogenic sinusitis. Computed tomography (CT) is considered the gold standard for sinus diagnosis. Recently, cone beam computed tomography (CBCT) has been introduced for dental and maxillofacial imaging, which has several advantages over traditional CT, including lower radiation dose and chairside process. This study aims to find the association between mucosal thickening (MT) of the sinus and periodontal bone loss (PBL)

Aim and Objectives: The aim of the present study is to determine the prevalence , association and severity of mucosal thickening of maxillary sinus and presence of any mucosal cysts in patients with periodontal bone loss using cone – beam computed tomography (CBCT).

Materials and Methods: Patient who reported to the department of periodontology and implant dentistry, CSI college of dental sciences and research for implant surgery with CBCT images were taken for the study. A total of 125 CBCT scans of patients were obtained and evaluated. Institutional Review Board and patient consent were obtained.

Results: 60% of patients with severe periodontal bone loss had mucosal thickening of maxillary sinus. The prevalence: higher on the right side(42.4%) ,more commonly seen in males (64%) , higher in age group more than 49 years (48%), mucosal thickness/ height greater in range of 8.1-12mm (38.4%). Overall there is a significant difference between periodontal severity and sinus findings such as mucosal thickness, mucosal height and mucosal cyst

Conclusion: Severe periodontal bone loss was significantly associated with the mucosal thickening of maxillary sinus.

Key Words: maxillary sinus, periodontal bone loss, CBCT, clinical study

Introduction

Maxillary sinuses are pneumatic cavities in the maxillary bone. They are lined with a thin respiratory mucous membrane referred to as the Schneiderian membrane which adheres firmly to the periosteum and is about 0.8-1 mm thick (Goller – Bulut et al., 2015).[1]

Mucosal thickening of the maxillary sinuses is a routine radiographic finding detected in an otherwise asymptomatic individual with a prevalence ranging from 8% to 29% (Soikkonen and Ainamo, 1995). An infection or allergic process like chronic sinusitis may cause the inflammation of the mucosa, making it visible on the radiograph.[2]

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A relationship between maxillary sinusitis and dental infection has been established by many preliminary investigations (Brook, 2009).[3] Maloney and Doku, (1968) have attributed 10–12% of the cases of maxillary sinusitis to odontogenic infections.[4] Violation of the Schneiderian membrane due to conditions like periapical abscesses, periodontal diseases, dental trauma, tooth extractions or placement of dental implants tend to increase the risk of maxillary sinusitis (Bolger et al., 1991).[5] Periodontitis has been thought to be one of the principal causes of odontogenic maxillary sinusitis and the subsequent thickening of the sinus mucosa associated with it (Goller-Bulut et al., 2015; Ren et al., 2015).[6,7]

Bauer, (1943)[8] explained that the vicinity of the root soft hemaxillary molars and premolars to the maxillary sinus, as well as the multudinous an astomoses between the blood and lymph vessels in the apical region of the teeth and the corresponding blood vessels of the sinus mucosa, are mechanisms that favor the spread of odontogenic infections to the maxillary sinus. The porosity of the maxillary bone permits the diffusion of pathogens and their pathogenic products to the sinusfloor.[9-11]

Panoramic radiography, Water's projection and intraoral radiography have been routinely used in the imaging of the maxillary sinus in dentistry (Burke et al., 1994). However, the complex anatomy of the oral and maxillofacial region causes the superimposition of the important anatomical features and are therefore difficult to visualize (Lofthag-Hansenet al., 2007). Moreover, the literature indicates that conventional radiographic techniques are less accurate in detection and measurement of the mucosal thickening of the maxillary sinus (Soikkonen and Ainamo, 1995; Vallo et al., 2010).

On the other hand, Computed tomography (CT), the gold standard for sinus diagnosis, allows the visualisation of both hard and soft tissues by providing multiple sections through the sinus at different planes.[3] The recent introduction of Cone Beam Computed Tomography (CBCT) in dental and maxillo-facial imaging, facilitates the diagnosis of delicate structures in multiplanar reconstructions. A lower radiation dose and an isotropic volume resolution gives an advantage to CBCT over other radiographic techniques in being extensively used for imaging of paranasal sinuses (Ruprecht and Lam, 2009). Therefore , the aim of the study was to measure the thickness of the mucous membrane lining the maxillary sinus and to correlate this thickening of the Schneiderian membrane with the presence of periodontal bone loss of adjacent teeth, by using CBCT.[9]

Materials and Methods

Study Samples:

The study protocol was approved by the Institutional Ethical Committee. The CBCT images taken at the Department of Radiology, CSI College of Dental sciences and Research from June 2019 to January 2020 were retrospectively examined. Individuals received CBCT scans for dental treatment purposes, mostly for dental implant treatment planning.

The inclusion criteria were:[6]

1. Good quality of the images;

2. Existence of at least one of the first and second molar or second premolar in each left or right sides;

3. No sign of acute non-odontogenic sinusitis, including air-fluid level and thickening of all the sinus walls;

4. No prescription of CBCT due to the developmental problems or trauma.

CBCT images fulfilling the following criteria were included in the study:

1) the image was taken using P-mode (15-cm diameter field of view, 0.29-mm image resolution) and standard parameters (120 kVp, 15 mA, 9.6 second);

2) the occlusal plane of the image was parallel to the floor;

3) there was 1 upper posterior tooth below each sinus; and

4) the sinus had no sign of acute sinusitis, such as an air–fluid level or complete opacification.

Out of two hundred and fifty six CBCT, only one twenty five consecutive CBCT images were examined, and images were included for the analysis. The main reason for exclusion of the CBCT images, was the absence of upper posterior teeth below the sinus.

Analysis of CBCT images:

Commercially available CBCT software was used for the image analysis. The panoramic and cross-sectional views of the maxilla were reconstructed for evaluation and measurement. For calibration and evaluation of intra and inter examiner reproducibility, ten CBCT measurements were evaluated twice by each examiner.

Assessment of PBL: [periodontal bone loss]

All erupted maxillary second premolar and first and second molar teeth were examined. PBL was assessed from the panoramic and tangential views. The normal situation of the alveolar crest was assumed to be 2 mm under the cemento-enamel junction (CEJ). To calculate the amount of PBL, the distance between the point 2 mm under the CEJ and the crest of the alveolar bone was measured at the mesial and distal sides of each tooth (totally 12 points in each patient). Furthermore, we classified the amount of the PBL as follows:

1. Normal;
2. Mild, <25% bone loss;
3. Moderate, 25-50% bone loss and
4. Severe, >50% bone loss.

Assessment of MT: [mucosal thickening]

The presence or absence of MT in the floor of the maxillary sinus was evaluated from the cross sectional, tangential and panoramic views. For each sinus, mucosal thickening was considered present when the thickness of the sinus mucosa was >=1 mm. When thickening of the sinus mucosa was present, the thickness was measured in millimeters from the floor of the sinus to the highest border of the mucosa. The amount of MT was also classified into four groups:

1. 2-4 mm
2. 4.1-8 mm
3. 8.1-12 mm
4. >12 mm

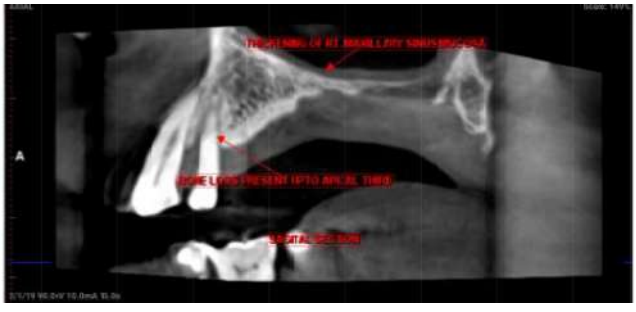


Figure 1: Mucousal Thickening on Right Side of Maxillary Sinus Mucosa

Age groups:

To evaluate the impact of age on the prevalence and severity of maxillary sinus MT, the patients were stratified as follows:

<30 years of age (young adults);

30-49 years of age (adults);

>49 years of age (Giedraitic adults); and

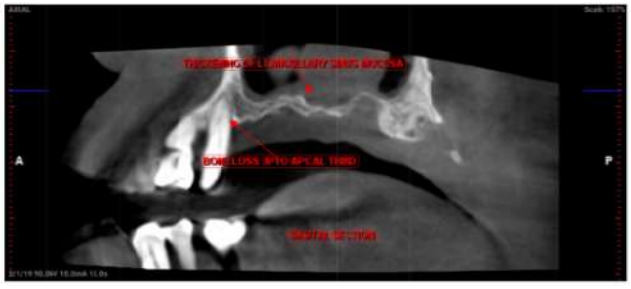


Figure 2: Mucousal Thickening on Left Side of Maxillary Sinus Mucosa

Assessment of Mucosal Cysts:

The presence of a mucosal cyst was assessed from the panoramic and cross-sectional views. The diagnosis was based on the following radiographic characteristics:

1) a homogeneous dome-shaped opacity within the maxillary sinus with sharp demarcation of lateral borders;

2) absence of bony erosion;

3) absence of communication with a tooth root; and

4) a smooth, spherical outline at the free border of the cyst.

The size of mucosal cysts was measured in millimeters from the floor of the sinus to the highest border of the cyst. Only mucosal cysts located on the floor of the maxillary sinus were included in this analysis. For each patient, a mucosal cyst was considered present when sinus had a mucosal cyst.

Results

Table 1: Prevalence of mucosal thickening

No.of. mucosal Thickening Evident in the Maxillary Sinus	N=125	percentage
	Present: 103	82.4
	Absent :22	17.6

A total of 125 sides were evaluated using CBCT of which 82.4%(n = 103) had a prevalence of mucosal thickening.

Table 2: Prevalence of mucosal height

Mucosal Thickness/ Height[MM]	n	percentage
2-4	5	4
4.1-8	30	24
>8	68	66

In order to evaluate the prevalence of mucosal thickness height it was further divided into 3 groups namely 2 – 4mm, 4.1 – 8mm, 8.1 – 12mm and the prevalence was found to be 4%, 24%, 66% respectively.

Table 3: Correlation between Periodontal Severity and Mucosal Height

Severity	NIL	2-4MM	4.1-8MM	8.1-12MM	Total	P Value
Mild	1	0	0	2	3	0.043*
Moderate	9	2	4	2	17	
Severe	12	3	26	64	105	
Total	20	5	30	68	125	

*Student t test, *-statistical significant, P value <0.05- statistical significant*

For comparison of periodontal severity and mucosal height, it was found that severe periodontal bone loss was significantly associated with a mucosal height of 8.1 – 12 mm.

Table 4: Correlation Between age and Mucosal Height

Age	NIL	2-4 MM	4.1-8MM	8.1-12 MM	Total	P VALUE
<30	13	5	4	3	25	0.004*
30-49	5	0	9	26	40	
>49	4	0	17	39	60	
Total	22	5	30	68	125	

*Chi square test, *-statistical significant, P value <0.05- statistical significant*

Table 5: Correlation Between Age and Mucosal Thickness

Age Group (Years)	Mucosal Thickening n (%)		Chi value P VALUE
<30	Present	12(9.6)	156.360a
	Nil	13(10.4)	
30-49	Present	45(20.8)	0.09
	Nil	5(4)	
>49	Present	56 (44.8)	
	Nil	4(3.2)	

** x2 test, *-statistical significant, P value <0.05- statistical significant*

When distribution of mucosal thickness was evaluated in relation to gender it was found to be higher in females.

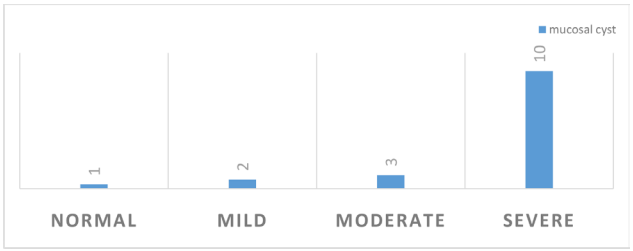
Table 6 : Prevalence of mucosal cyst

mucosal cyst	Frequency	Percent
ABSENT	109	87.2
PRESENT	16	12.8
Total	125	100.0

A total of 125 sides were evaluated using CBCT of which 12.8%(n = 16) had a prevalence of mucosal cyst which is an accidental finding in the study.

When distribution of mucosal cyst was evaluated in relation to gender it was found to be higher in males. (n=13)

Table 7 : Prevalance of Mucosal Cyst in Relation to Periodontal Severity



When the prevalence of mucosal cyst was evaluated in relation to periodontal severity it was to be higher in severe periodontal bone loss group.

Discussion

In the present study we analyzed the association of presence of radiographic evidence of periodontal bone loss on the mucous membrane thickening of maxillary sinus and found a relatively increased mucosal thickening in the patients having presence of radiographic evidence of periodontal bone loss as compared to those without. This indicates a positive role of periodontitis in increasing the thickness of Schneiderian membrane further substantiating the possible role of periodontal disease in causing odontogenic sinusitis. These findings concur with those reported by Phothikhun et al., (2012) and Sheikhi et al., (2014) who have also reported mucosal thickening to be commonly associated with periodontal boneloss.[12-14]

The presence of deep periodontal pockets may evoke a local reaction in the sinus mucosa, such as edema, round cell infiltration, fibrosis or cystic degeneration, which might be a possible mechanism for the thickening of sinus mucosa (Bauer, 1943)

Maxillary sinusitis resulting from periodontal infections can be caused by two mechanisms: either the spread of microorganisms and their products as well as the cytokines via the several anastomoses between the blood and lymph vessels in the apical region of the tooth and the corresponding vessels in the sinus mucous mucosal lining or by a direct spread of infection through the porous maxillary bone and the supporting tissues. The findings of Engstrom et al., (1988) and Falk et al., (1986) further substantiates this relationship.

In the present study, CBCT images have been used to assess the mucosal thickness of maxillary sinus while maximum of the previous studies have used conventional radiographic techniques[1]. (Soikkonen and Ainamo, 1995; Vallo et al., 2010).

We considered the mucosal thickness of >2 mm as an evidence of mucosal thickening. This was consistent with previous reports (Janner et al., 2011; Lu et al., 2012; Maillet et al., 2011).

Soikkonen and Ainamo(1995)considered the presence of diffuse radiopacities along the wall of the sinus as an evidence of mucosal thickening.

Previous reports have shown a positive relation of age and mucosal thickening (Iwabuchi et al., 1997; Vallo et al., 2010). In this study also, the effect of age on the maxillary mucosal thickness was investigated. As the mean age in Periodontal Bone Loss group was significantly higher than Healthy group, and considering its relevance in bone loss, the mucosal thickness at each point was adjusted with age, even though only a weak but yet positive correlation was noticed between age and mucosal thickness.[8]

The results of our study are in consistence with the study of Goller-Bulut et al. (2015) who evaluated CBCT images of 410 maxillary sinuses and found that the prevalence of maxillary sinus mucosal thickening increased with an increase in age.[7] The study done by Phothikhun et al., (2012) also showed results similar to our study; they found a higher prevalence of mucosal thickening among males and older age group (>49-year-old). The weaker association obtained in our study can be attributed to as maller sample size in comparison with other similar studies.

As the age advances, the process of pneumatization increases and favors the spread of infection through the porous maxillary bone to the maxillary sinus consequently causing mucosal thickening.[12] On the contrary, Rege et al., (2012) detected no influence of age on the occurrence of sinus abnormalities.

Mucosal thickening of maxillary sinus and other sinus pathologies may pose problems when a sinus augmentation surgery is planned. A sinus augmentation procedure done in cases of in sufficient height of maxillary residual alveolar ridge may result in the

obstruction of the sinus and also impair its drainage (Carmeli et al., 2011).[10] Thus, periodontal treatment should be performed before sinus augmentation surgery to reduce the inflammation of the sinus mucosa.[5]

Also this, study establishes an association between age and increased mucosal thickening. Most of the patients requiring dental implant therapy and sinus augmentation are edentulous and demonstrate increased pneumatization and the associated increased mucosal thickening.

Limitations

Since this study retrospectively analyzed the CBCT images, a clinical examination and recording a proper history about periodontal and sinus problems was not possible. Thus the presence of sinus pathologies in these patients cannot be completely ruled out although the images showing air fluid levels were excluded. The possibility of a periodontal treatment and regression of mucosal thickening cannot be completely ignored. So a precise strength of association of periodontal infection and mucosal thickening cannot be very well ascertained.[13]

The retrospective design of this study presents with certain limitations and requires the design of further prospective studies to establish an exact causal relationship.

Conclusion

CBCT may provide accurate diagnostic information for better evaluation of the maxillary sinus. Profound knowledge of the sinus and anatomy of the surrounding structures will certainly decrease the risk of complications.

This study shows that mucosal thickening and mucosal cysts of the maxillary sinus were common among dental patients. Severe periodontal bone loss was significantly associated with mucosal thickening of the maxillary sinus. Sinuses with severe periodontal bone loss were three times more likely to have mucosal thickening. Mucosal cysts were not associated with dental findings.[14]

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The flagship event of SPIK – 8th SPIK Periodontology Scholarship Examination 2022 was held on Monday 21st March 2022. It was hosted by the Department of Periodontics, Kannur Dental College, Anjarakandy, Kannur. Dr Arun Narayanan was the Organizing Chairman and Dr Mohammed Feroz TP was the Organizing Convenor of the program. The Exam panelists were Dr Harikumar K (Professor –CAP, Govt. Dental College, Kozhikode), Dr Sanupa Madhavan (Reader, Sree Anjaneya Institute of Dental Sciences, Kozhikode) and Dr Navia George (Reader, KMCT Dental College, Kozhikode) along with Dr Mohammed Feroz TP. Around 20 Undergraduate students from various Dental Colleges across the state participated in the exam. The winner was Mr Ashish Anz of Sree Sankara Dental College, Thiruvananthapuram. He was awarded a cash prize of Rupees Ten Thousand by Dr Harikumar K, Immediate Past President, SPIK.

PROGRAM SCHEDULE

9:00 AM - 9:30 AM Registration

9:30 AM to 11:00 AM

Guest Lecture
"Successful Autogenous Soft Tissue Grafting in Periodontics and Implantology"
Dr. Mihir Kulkarni

11:00 AM Inaugural Function

12:00 Noon Guest Lecture
"Ortho-Perio Synergy"
Dr Balaji Manohar

1:30 PM - 2:00 PM Lunch Break

2:00 PM - 2:30 PM
Screening of the Periodontal Awareness Videos

2:30 PM - 3:30 PM
"PerioScope"
Snippets of challenging periodontal cases

3:30 PM
Annual General Body Meeting and
Election of Office Bearers 2022-23'

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REGISTRATION DETAILS

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Rs 1000/- (Spot registration)
Mode of Payment: Online (Bank transfer or UPI)

Account Details

Name: Society of Periodontists and Implantologists of Kerala
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