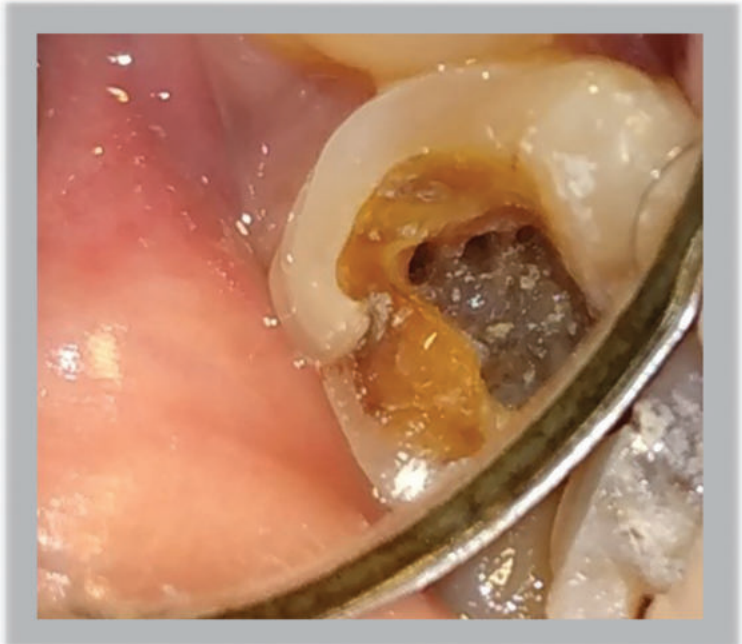


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Phone (0495) 229 0690/229 0693, Fax (0495) 229 4726

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EDITORIAL

Importance of Co-curricular activities for our students

College is a place that lays the foundation to your future and career. It is not only a place where you just gain academic knowledge but also where your overall personality development takes place. Co-Curricular activities play an important role in a student's overall personality development. For example, a student pursuing a degree might also have a passion for music, can easily join the college band to follow his passion for music. Having learnt a foreign language might also be a great addition to his resume as it might aid him in a job abroad. One can take part in team sports like football, cricket or somewhat individual ones like badminton, swimming etc. The team-spirit, ability to work with others and many such soft skills give you a chance to interact with people and make connections.

Co-curricular activities can help students develop skills that will aid them a lot in their professional life later on. Co-curricular activities are not just about developing your technical skills they are also about developing your communication skills. Students who are involved in these activities are often seen to have better time management skills than students who are always buried nose deep in their books. Working out two or three different things in a day helps student learn the art of prioritizing tasks. He needs to take out time for practice and while also being busy with his coursework and project submissions. He knows that he cannot miss out on either of them and thus has to figure out a way to manage them both efficiently. Such a student is more likely to plan a work schedule with work towards following it with sincere dedication. He is less likely to procrastinate or waste his time idling away. Moreover, a few hours of practice might help him relax and return to his studies with a refreshed mind.

KMCT Dental College in the upcoming days plans to incorporate more of co-curricular activities like soft skill development programs, literary club, health club, and language lab in to our campus. Let us hope that our students make good use of these facilities which are helpful in turning them in to better individuals as well as good dentists.

Dr. Manoj Kumar KP
Chief Editor

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MISSING POSTERIOR TEETH AND TEMPOROMANDIBULAR JOINT

Dr. Sheejith M, **Dr. Sruthi Viswanath, ***Dr. Swapna C, *Dr. Abu Nazar**

ABSTRACT

It has been accepted for past many decades that there is an association between missing posterior teeth and temporomandibular joint disorder (TMD). Many systematic review and clinical trials have been conducted by many researchers based on this topic. This paper explores systematic reviews and clinical trial from a time period of 1985 to 2018 year. Many researchers came to a conclusion that TMD and missing posterior teeth have an association between them. But still, there is a question that, whether restoring the missing posterior teeth alone can cure the TMD.

Keywords: Missing posterior teeth, temporomandibular disorder, prosthodontic treatment

Corresponding Author: Dr. Sruthi Viswanath

** Professor & HOD, **Post graduate student, ***Professor, ****Senior Lecturer, Department of Prosthodontics, KMCT Dental College, Manassery 673602, Kozhikode, Kerala*

Introduction

Temporomandibular disorders (TMD) is a collective term that includes a number of clinical problems that which involves the masticatory musculature, TMJ associated structures. To eliminate the pain associated with TMD restoration of posterior teeth has been suggested. Thereby there will be stabilization of occlusion, redistribution of occlusal forces and reduction of joint loading. Prevalence of TMD are not seem to be decreased by the restoration of missing teeth and mixed results have been reported for the effect of restoration on masticatory function. However, missing posterior teeth may accelerate the development of degenerative joint disease.

The association between tooth loss and TMD remains controversial. Loss of posterior teeth

causes drifting and tipping of the remaining teeth. This drifting and tipping causes secondary changes in occlusal contact, called tightly locked occlusion. A single causal factor is not sufficient to cause TMD. In general population TMD is 2 to 5 times more prevalent in women than men.¹ So a literature review was conducted to know the association between missing posterior teeth and TMD.

Aim

To find out the association between missing posterior teeth and temporomandibular joint disorder.

Results

P. Kirveskari and P. Alanen found that maxillary first premolars have shown to

contact prematurely on hinge closure more often than other teeth. So the loss of other teeth seemed independent of the functional state of the stomatognathic system. They suggested that TMJ dysfunction may predispose to the loss of the maxillary first premolar by direct trauma to the tooth.² Later in 1987 in an invivo study conducted by N Barghi et al in which they included 150 patients with minimal 5-year history of bilateral and unilateral missing posterior teeth who were clinically examined for occurrence, types, and location of TM J clicking. A higher incidence of TM J clicking was recorded in these patients when comparing patients with posterior teeth.³ D. J. Witter compared subjects with shortened dental arches characterized by the absence of molar support with a complete dentition with respect to signs and symptoms of mandibular dysfunction and came to a conclusion that in population with SDA the absence of molar support does not appear to provoke signs and symptoms of mandibular dysfunction. The presence of bilateral premolar support seems to provide sufficient mandibular stability.⁴

Later in the 20th century a histological study was conducted by Luder et al in which they stated that rising severity of TMJ degenerative changes appears to be associated primarily with increasing age. In addition, it may also depend on mechanical factors, in particular loss of molar support and, to a minor degree, abnormal disc position.⁵ Tallents R H et al conducted a clinical study in which they included, Eighty-two asymptomatic volunteers and 263 symptomatic TMD patients. Asymptomatic

volunteers completed a subjective questionnaire and underwent clinical examination to document the absence of TMD signs and symptoms. The number of missing mandibular bicuspid and molar teeth (excluding third molars) in each subject was also recorded, and magnetic resonance images were made to document the presence or absence of disk displacement in the temporomandibular joints. They concluded that the replacement of missing posterior teeth does not prevent the development of TMDs. However, missing mandibular posterior teeth may accelerate the development of degenerative joint disease.⁶ In 2003 Sarita et al in their study found that there is no evidence that shortened dental arch provoked signs and symptoms associated with TMD. However, when posteriors are missing unilaterally or bilaterally, the risk of pain and joint sounds are increased.⁷ In 2006 Jabrah O A et al conducted a survey in which A questionnaire and a clinical examination were used to assess 200 patients. One hundred of these were complete denture wearers being treated for the provision of replacement CD. The other 100 patients were partially edentulous patients, who had RPD replacing upper and lower partially missing teeth and their supporting structures. And they concluded that partially edentulous patients wearing upper and lower RPD had a significantly higher prevalence of TMD signs than edentulous patients wearing.⁸

Two recent studies were conducted by Shumailan Y A et al and Felix Bertram et al. Shumailan Y A et al in their study stated that

that loss of posterior teeth is significantly associated with TMD signs and symptoms. In the group studied more women than men reported TMD symptoms and both male and female subjects were found to have TMD signs which were more common in men.⁹ In 2018 Felix Bertram et al in their study used CBCT to find association between missing posterior teeth and occurrence of TMJ disorders and concluded that there is association between them.¹⁰

Discussion

There were in general no clinically significant differences between subjects with SDA of three to five occlusal units and complete dental arches regarding variables such as masticatory ability, signs and symptoms of TMDs, migration of remaining teeth, periodontal support and oral comfort. Recently, experimental findings provided no evidence that shortened dental arch causes overloading of joints and teeth. The tendency for increased severity of TMJ erosion in older individuals has been described by some authors.

In the risk evaluation of developing asymptomatic condylar erosion, local and general factors must be considered. Local factors such as parafunctional habits, occlusion, functional overloading, trauma etc. general factors such as gender, age, sex, hormonal factors.

The variable of missing posterior teeth has been reported to have very limited effect on the incidence on TMD. But loss of posterior teeth may exert secondary osteoarthritic changes that include condylar erosion.

Some authors stated that there is a significant correlation between the extend of TMD based on pain intensity and duration. But some authors reported no correlation between TMD and missing posterior teeth.

Conclusion

In summary, the results of this study indicate that when the variables of the number of missing posterior teeth and the number of dental quadrants with missing posterior teeth function together, their effect on TMD increases. Since the possibility of the formation of occlusal interference is higher with the more teeth remaining, the results support the effect of abnormal occlusion on TMD etiology.

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HOST MODULATION IN PERIODONTOLOGY: A REVIEW

* Dr. S. Hima, **Dr. Harish Kumar V. V.

Abstract

Historically, periodontal disease (gingivitis and periodontitis) has been recognized as being primarily of bacterial origin. But recent evidence indicates that though the bacteria are necessary for disease development they are not sufficient for the clinical manifestation of periodontal disease. It is becoming increasingly apparent that it is the host inflammatory response to the subgingival bacteria that is responsible for the tissue damage and, most likely, progression of the disease. We explore the concept that it is the subgingival microenvironment modified by the inflammatory response that leads to a change from a commensal to pathogenic microbiota. In this review, we examine the evidence for the emerging paradigm supporting the central role of inflammation rather than specific microbiota in the pathogenesis of periodontitis, and that by controlling the inflammation, it is possible to control the infection.

Key words: periodontitis, host modulation, review

Corresponding author: S. Hima

**Post Graduate Student, **Professor & Head, Department of Periodontics, KMCT Dental College, Manassery, Kozhikode 673 602, Kerala*

Introduction

Historically, periodontal disease (gingivitis and periodontitis) has been recognized as being primarily of bacterial origin. However, recent evidence indicates that while bacteria are necessary for disease development but they are not sufficient for the clinical manifestation of the many and varied forms of periodontal disease.¹ So periodontitis can no longer be considered a simple bacterial infection that leads to periodontal destruction.² Rather, it represents a collection of complex diseases involving interactions between the host inflammatory immune systems, sub-gingival microbiota and modifying environmental factors.

It is also recognized that the periodontal microbiota is generally of a commensal

nature and its relationship to the host is usually in a state of homeostasis. However, 'pathologic' shifts in the microbiota may occur by the overgrowth of organisms which are normally present in low numbers, creating 'dysbiosis' of the periodontal subgingival microbiota.³ So there is requirement for a suitable environment and a 'susceptible' host for periodontitis to manifest clinically.

Another way to consider the periodontal diseases is that they are 'eco-genetic diseases' whereby patients are rendered susceptible by genetic or environmental factors such as polymorphisms in the gene for interleukin-1, cigarette smoking, chronic (hyper)-inflammation and diabetes. This will

create chronic (hyper)-inflammation to the subgingival bacteria leading to the development of destructive periodontal disease. Thus, periodontitis is considered to be very much a multifactorial disease in which bacteria are necessary but not sufficient for the disease to develop.⁴

Host response modulation (or host modulation) is a term that has been introduced to the dental profession especially in the periodontal context, and in very simple terms, it means modifying or modulating destructive or damaging aspects of the inflammatory host response that develops in the periodontal tissues as a result of the chronic challenge presented by the subgingival bacterial plaque. Host response modulation is routinely practised by our medical colleagues, who use host modulation strategies in the treatment of disorders such as rheumatoid arthritis and osteoporosis.⁵ And while the term host modulation has only recently started to be widely used in general dentistry, the concept was first introduced to the research community in the late 1980s and early 1990s.⁶

Basic concept in host modulation

Microbial biofilm elicits a host response, with resultant osseous and soft tissue destruction. In response to endotoxins derived from periodontal pathogens, various inflammatory chemical mediators are released by the host cells. The immune-inflammatory response has been described as a double-edged sword, and besides providing specific antibodies and polymorphonuclear

neutrophils (PMNs), that represent the dominant natural factors responsible for control of the bacterial challenge, it initiates the destruction of the connective tissue.⁷

Page et al. (1997), stated that periodontal destruction in periodontitis is the result of connective tissue-degrading mediators such as, matrix metallo proteinases (MMPs) and inflammatory mediators (prostaglandins, interleukins) that occur as a part of the inflammatory response. There is a vital balance between pro and anti-inflammatory chemical mediators in healthy tissue. When the pro-inflammatory mediators increase, tissue destruction results. The purpose of host modulation therapy is to restore this balance.⁵

Host modulation is a therapy that is targeted at the host response. As already stated, the result of the host-bacterial interaction is the release of various inflammatory mediators that cause tissue destruction. Using various therapeutic agents that can downregulate or inhibit the production, activation or biological function of the pro-inflammatory mediators is the basic mechanism of action of host modulation therapy.

Over the last two decades, a variety of pharmacological agents have been studied for a possible role as host modulators in the management of periodontal disease. These include nonsteroidal anti-inflammatory drugs, bisphosphonates and the tetracycline family of compounds (and their chemically modified analogues). Newer agents that have the potential to be of benefit in periodontal treatment include anti-cytokine drugs (which have successfully been used in the treatment

of rheumatoid arthritis), soluble cytokine blockers and lipoxins. To date, only one systemic medication has been licensed specifically as a host response modulator for the treatment of periodontal disease, and that is subantimicrobial dose doxycycline.⁸

Definition of host modulation

The definition of the host from a medical dictionary reads “the organism from which a parasite obtains its nourishment or in the transplantation of tissue, the individual who receives the graft”. The definition for the term modulation is the alteration of function or status of something in response to a stimulus or an altered chemical or physical environment.⁹ The inflammatory response in periodontal disease includes the activation of leukocytes, neutrophils, T-lymphocytes and plasma cells. Subsequently, various chemical

mediators are released by these immune competent cells, which along with providing a defense to the host also degrade the host connective tissue.

Pathogenesis of periodontal disease progression

As already discussed in previous chapters, periodontitis is a complex disease in which disease expression involves intricate interactions of the biofilm with the host immunoinflammatory response and subsequent alterations in bone and connective tissue homeostasis. Periodontal diseases have a well-defined bacterial etiology. Along with that, the present data suggest that environmental factors, acquired risk factors, and genetic risk factors play an important role in the pathogenesis of periodontal diseases.

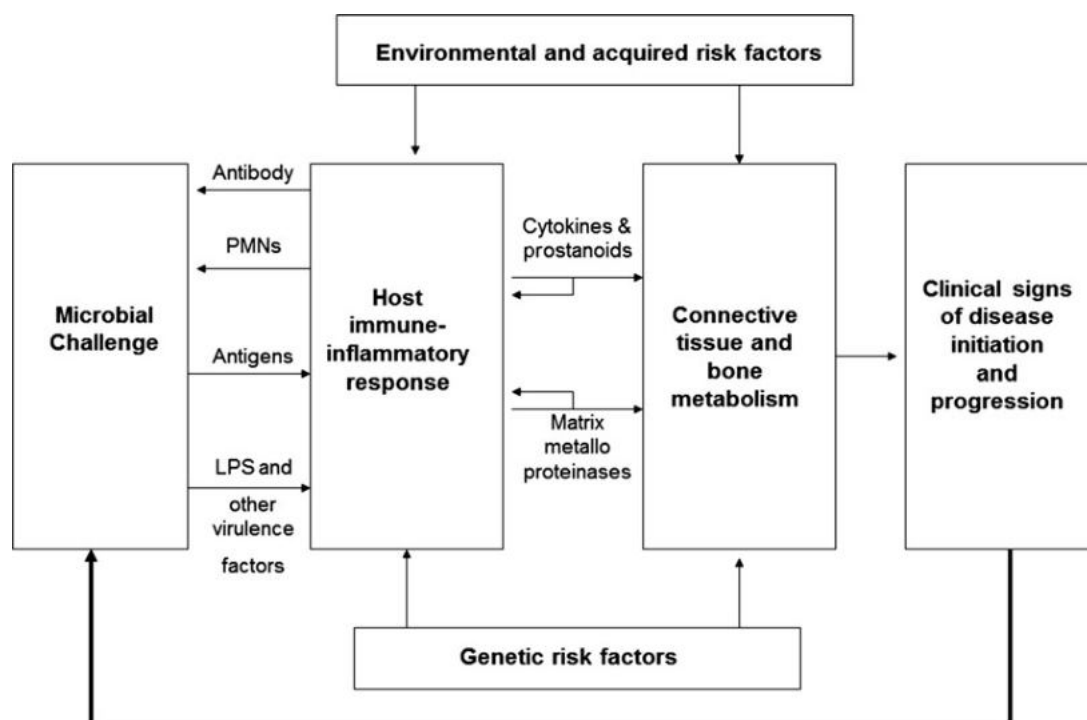


Fig 1: Proposal for the pathogenesis of periodontitis circa year 1999¹⁰

Kornman (1999) proposed a model of periodontal disease pathogenesis that explains how the host-parasite interactions takes place and leads to the disease progression.¹⁰

The host response is characterized by the production of host-derived inflammatory mediators, including cytokines and lipids by neutrophils, monocytes, lymphocytes and fibroblasts. As periodontal diseases are multifactorial diseases, environmental risk factors, such as diabetes mellitus, cigarette smoking, and stress, as well as genetically transmitted traits, such as interleukin-1 (IL-1) gene polymorphisms, may accentuate the host inflammatory response to the bacterial challenge and, eventually, the susceptibility to the disease.¹¹ One of the most important consequences of this host response is the generation of matrix metalloproteinases (MMPs), which seems to be highly related to tissue destruction and remodelling events in periodontal diseases.¹²

A new model for the pathogenesis of periodontitis

Periodontitis is the host inflammatory response that largely drives the pathological process, new concepts for the pathogenesis (and treatment) of peri-odontitis have been proposed.² In these models, the development of gingivitis is a mandatory initiating condition for the subsequent development of periodontitis. It is recognized that gingivitis results from a nonspecific inflammatory reaction in the gingival tissues to supragingival plaque accumulation. The

resulting gingival inflammation alters the subgingival environment as a result of increased concentrations of mediators of inflammation and products of connective tissue breakdown in the gingival crevicular fluid (e.g. collagen peptides).

These conditions provide a suitable environment for the over growth of 'periodontal pathogens' within the subgingival biofilm. If the host inflammatory response is sufficient, and there are favorable genetic and environmental influences, the lesion may be 'contained' as gingivitis and does not progress to periodontitis, although it will mature into a stable inflammatory/immune lesion.¹³ On the other hand, if the host inflammatory immune responses do not stabilize the lesion, and the individual is genetically susceptible as well as influenced by unfavorable environmental factors (e.g. smoking), the condition will progress leading to the clinical manifestation of periodontitis.

Periodontal inflammation – new opportunities for treatment

Opportunities for host modulation

Recognizing the importance of modulating the inflammatory response, new treatment opportunities arise whereby adjuncts to mechanical debridement focusing on controlling or resolving inflammation.¹⁴ One product already commercially available, and approved for clinical use by the US Food and Drug Administration, is Periostat®, which is a low dose of doxycycline. It has been long known that tetracyclines, in addition to their

Another promising area for investigation with regard to host modulation is that of anti-cytokine therapy that has been in use in medicine for the control of many chronic inflammatory conditions.¹⁹ Most of these agents target interleukin-1 and tumor necrosis factor and are based on either monoclonal antibodies or modified receptor proteins. One such agent, Etanercept (a tumor necrosis factor-alpha receptor antagonist) has been found in an animal model of periodontitis to assist in the reduction of inflammation through reduced neutrophil infiltration, reduced nitric oxide levels and reduced apoptosis.²⁰ However, one systematic review reported limited evidence for the use of these agents for periodontitis.²¹

One of the major problems with anti-cytokine therapy is that a functional redundancy of cytokines can enable the host to activate alternative pathways of inflammation if only one specific cytokine is targeted by a therapeutic agent. These agents also have an unacceptable side-effect profile and are extremely expensive, thus precluding their use as an adjunct to periodontal therapy at the present time.

Two areas of considerable interest are the use of diet or specific synthetic and natural resolvins as adjuncts to periodontal therapy. The use of omega-3 polyunsaturated fatty acids (precursors of resolvins) is of interest due to their well documented anti-inflammatory properties. A recent systematic review reported good emerging evidence that

dietary supplementation with fish oil might be of some benefit.²² The benefit is enhanced if combined with aspirin. Dietary supplementation with fish oil could be a cost effective adjunctive therapy to the management of periodontal disease.²³

Interestingly, several studies have reported that combining aspirin with fish oils has a positive clinical benefit in the management of periodontitis.²⁴ The addition of aspirin to the treatment regime is based on its ability significantly to increase the production of more stable resolvins.

An important distinction in this evolving story is the difference between anti-inflammation and proresolution. Anti-inflammation inhibits COX or 5-lipoxygenase enzyme activity and can affect many biological processes other than inflammation. On the other hand, proresolution is a receptor mediated event that drives cellular processes to restore health. The difference between these two processes probably accounts for the presence or absence of unwanted side effects.²⁵

Combination anti-inflammatory and antibacterial therapies

Combining both anti-inflammatory and antibacterial therapies is worthy of consideration. Surprisingly, very few studies have addressed this concept with only one report identified investigating the combined systemic use of an anti-inflammatory and antibacterial agent.²⁶

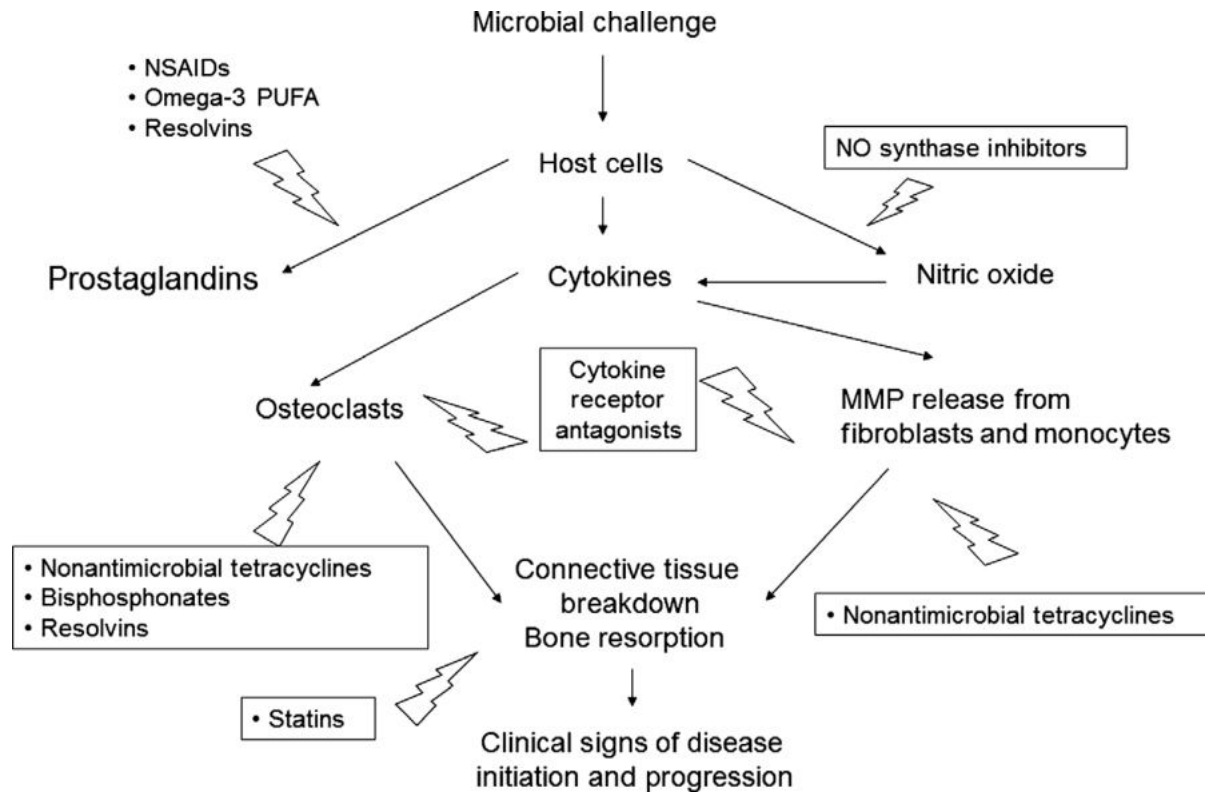


Fig 3: Opportunities for using host modulation to target pathways of inflammation in the development of periodontitis

More recently, interest has focused on the use of the macrolide antibiotic azithromycin in the management of periodontitis. Not only does azithromycin possess antibacterial effects, but it also exerts considerable anti-inflammation properties.²⁷ This provides an opportunity to combine the therapeutic effects of one agent that has both antibiotic and anti-inflammatory capabilities with significantly greater potency than the tetracyclines. A recent meta-analysis concluded that azithromycin used as an adjunct to non-surgical therapy significantly improves the efficacy of non-surgical periodontal therapy on probing depth reduction, bleeding on probing and gain of

attachment, particularly at the initially deep probing depth sites.²⁸

Despite these encouraging findings, caution must be exercised in the use of azithromycin, because of the potential for permanently changing the composition of the host microbiome at other body sites, and particularly in patients with a high baseline risk of cardiovascular disease due to a small risk of increased cardiovascular death.²⁹

Treat to target – a treatment outcome philosophy for periodontitis

In existing models and paradigms for the management of medical chronic inflammatory conditions such as

cardiovascular disease, hyperlipidemia and rheumatoid arthritis, remission of disease activity is a principal aim of treatment and is based on the concept of ‘treat to target’. In rheumatology, ‘treat to target’ is an international initiative to define treatment targets for rheumatoid arthritis and to make recommendations to measure disease severity and encourage earlier diagnosis and optimize treatment.³⁰ It is a therapeutic concept that considers well-defined and specific physiologic targets as aims in controlling the pathophysiology of the disease.

Ten principles for adopting a ‘treat to target’ approach for the management of periodontitis:¹

1. An initial target for treatment of periodontitis should be a state of clinical remission that then allows reconstructive and regenerative procedures to follow if necessary.
2. Clinical remission will be defined as the absence/reduction of signs and symptoms of significant inflammatory disease activity that are responsible for the tissue damage associated with active periodontitis.
3. While remission should be a clear target, based on available evidence low disease activity may be an acceptable alternative therapeutic goal, particularly in long-standing refractory disease.
4. Until the desired treatment target is reached, therapies (mechanical, anti-inflammatory and anti-infective) should be adjusted every 3–4 months.
5. Measures of disease activity must be obtained and documented regularly, as frequently as 3–4 monthly for patients with high/moderate disease activity or less frequently (such as every 6–9 months for patients in sustained low disease activity or remission).
6. The use of validated composite measures of diseases activity, which include periodontal assessments, is needed in routine clinical practice to guide treatment decisions.
7. Structural changes and functional impairment should be considered when making clinical decisions (i.e. predisposing factors).
8. The desired treatment target should be maintained throughout the remaining course of the disease.
9. The choice of the (composite) measure of disease activity and the level of target value will be influenced by consideration of co-morbidities, patient factors, drug-related risks and microbiological profile.
10. The patient has to be appropriately informed about the treatment target and the strategy planned to reach this target under the supervision of the periodontist.

Conclusion

In conclusion, it is evident that current preventive management approaches for the management of the periodontal diseases are only partially effective and have often failed for the most high-risk individuals. This may be due to an overemphasis on the role of plaque and specific bacteria at the expense of considering the host response, genetic and

environmental factors. Accordingly, there is a need for the development of adjunctive agents for the management of periodontitis based on current understanding of the etiology and pathobiology of the periodontal diseases. Host modulation therapy is an important emerging treatment strategy for managing all forms of periodontitis. In light of our current understanding of the central role inflammation plays in the pathogenesis of periodontitis, old concepts focused solely on controlling the infection to control the inflammation should be reworked to consider controlling the inflammation to control the infection.

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DENTAL IMPLANTS IN DIABETIC PATIENTS

*Dr. Swapna C, **Dr. Sheejith M, ***Dr. Eaketha P. Lokesh

Abstract

Dental implants have shown long term success rate in the rehabilitation of missing teeth. Certain adverse conditions like bleeding disorders, cardiovascular diseases, endocrine disorders, and bone diseases, immunologic conditions like cancer therapy, immunosuppressive or antiresorptive medication, smoking, alcoholism can cause marginal bone loss, increase the failure rate and post-operative complication, ultimately affecting the peri-implant health. Diabetes is one such metabolic disorder which delays wound healing and increases the risk of infection due to altered immune response. Therefore the success of implant therapy in diabetics is questionable. This article aims to review whether dental implants is indicated in diabetic patients.

Keywords: dental implants, diabetes mellitus, type 2 diabetes mellitus, osseointegration, glycosylated hemoglobin (HbA1c), peri-implantitis, implant survival, risk factor

Corresponding Author: Dr. Eaketha P. Lokesh

Professor, **Professor & HOD, *Post graduate student, Department of Prosthodontics, KMCT Dental College, Manassery 673602, Kozhikode, Kerala*

Introduction

Rehabilitation of completely or partially edentulous patients with dental implants has become a standardized treatment protocol in the past few years. Various modifications in implant design, surface characteristics and surgical protocols has improved the survival rate to more than 95%. Osseointegration is a presumptuous criterion for survival of dental implants. Any difference in its structural and functional correction can adversely affect the treatment outcomes. However, its use is limited because of various risk factors such as age, smoking, systemic conditions and type of bone. Diabetes mellitus is one such medically compromised condition with multifarious side effects. Hyperglycemia can

indirectly affect osseointegration and bone healing. Even though the number of diabetic patients is increasing dramatically, they are denied of implant therapy depending upon glycemic control. This articles evaluates whether dental implants cause any deleterious effects on diabetic patients.

Diabetes Mellitus

The term “diabetes mellitus” describes a group of metabolic disorders with multiple etiology characterized by chronic hyperglycemia with disturbances of carbohydrate, fat and protein metabolism resulting from defects in insulin secretion, insulin action, or both. The effects of diabetes mellitus include

long-term damage, dysfunction and failure of various organs (WHO 1999).¹ It occurs either as Type I or Type II.

Type I diabetes mellitus occurs when the pancreas does not produce enough insulin.² It is seen in childhood and adolescence and patients require lifelong insulin injections for survival.¹ Type II diabetes mellitus occurs when the body cannot utilize the produced insulin effectively. It is usually seen in adulthood and is associated with obesity, unhealthy diet and lack of exercise. Therefore it can also be called a “lifestyle disorder”. Type II represent 90 % of the diabetic cases worldwide. The WHO estimates that diabetes resulted in 1.5 million deaths in 2012, making it the 8th leading cause of death.³ The World Health Organization (WHO) has recently declared it to be a pandemic with its prevalence increasing dramatically over the past few decades.⁴

Effects of diabetes in oral health

Poor glycemic control is associated with increased incidence of diabetic- related oral complications which includes gingivitis, periodontitis and alveolar bone loss. According to the American Dental Association, the most common oral health problems for patients with diabetes include: cavities, gingivitis (inflammation of the gums), periodontal disease (gum disease), xerostomia (dry mouth), tooth decay, burning mouth syndrome (BMS), taste disorders, rhino cerebral zygomycosis (mucormycosis), aspergillosis, oral lichen planus, geographic tongue and fissured tongue , salivary gland problems, increased chance of bacterial,

viral, and fungal (oral candidiasis) infections.⁵ It alters taste sensation, impairs tooth eruption, fruity breath and cause benign parotid hypertrophy and other neurosensory disorders.⁴

Periodontitis is considered as the sixth most prevalent complication of diabetes. Several enzymatic and immunologic mechanisms can alter the host response, vascularity, subgingival microflora, collagen metabolism and gingival crevicular fluid (GCF) and genetic patterns. These mechanisms vitiate neutrophil function, decreases phagocytosis and leucotaxis, thereby increasing alveolar bone loss in diabetics. It delays wound healing, and increased incidence of infection.

Effect of diabetes on bone and osseointegration

Diabetes mellitus impairs the function of polymorphonuclear leukocytes (leukocyte adhesion, chemotaxis, and phagocytosis), impaired bactericidal activity, altered response to exposure to antigens, and alteration to the function of T lymphocytes.⁵ Hyperglycemia inhibits osteoblastic differentiation and alters the response of parathyroid hormone that regulates the metabolism of calcium and phosphorus.⁶

It decreases collagen formation during callus formation, induces apoptosis in lining cells of bone and increases osteoclastic activity due to persistent inflammatory response. It also induces deleterious effect on bone matrix and diminishes growth and accumulation of extracellular matrix. The consequent result is diminished bone formation during healing, which is observed in number of experimental studies⁷ which is summarized in table 1.

Table 1: Various studies of implant placement in diabetes

Author (year of study)	Type of study	Study design	Summary of the study
Balshi et al (1999) ¹¹	Retrospective study	Placed Branemark dental implants in diabetic patients	99.9% Branemark implants osseointegrated. Screening for diabetes and trying to ensure that implant candidates are in metabolic control are recommended to increase the chances of successful Osseo integration.
Al Jabbari et al (2003) ⁹	Literature review	Parameters related to clinical success of implant treatment in geriatric patients	Diabetic patients with acceptable glucose control demonstrated a success rate of 92.7% one year after first-stage surgery.
Morris et al (2005) ¹⁷	Prospective study	Determine if type 2 diabetes represents a significant risk factor to the long term clinical performance of dental implants	The use of chlorhexidine rinses following implant placement resulted in a slight improvement (2.5%) in survival in non-Type 2 patients and a greater improvement in Type 2 patients (9.1%).
Dowell et al (2007) ¹²	Prospective cohort study	To explore the relationship between implant success and glycemic control in patients with type 2 diabetes mellitus	Patients with HbA1c levels ranging from 7.4 to 8.3% showed minor complications in healing.
Hasegawa et al (2008) ⁸	In-vivo study in rats	To evaluate whether type 2 diabetes impairs implant osseointegration capacity.	Type 2 DM impaired osseointegration capacity disproportionately between the cortical bone and bone marrow areas.
Inbarajan et al (2012)	Retrospective study	Evaluate the efficacy of implant placement diabetics with early non-functional loading protocol control	All implants were well integrated in bone for the duration of the study as evaluated by clinical and radiographic criteria.
Gomez-Moreno (2014) ¹³	Prospective study	Analyse the changes in peri-implant area by monitoring the HbA1c levels	Elevated HbA1c causes more bone loss and significantly higher bleeding on probing.
Oates et al (2014) ¹⁴	Prospective study	To determine whether poor glycemic control is contraindicated for implant therapy in type 2 diabetic patients	The survival rates was 93% in patients without diabetes, 92.6% in patients with well-controlled diabetes and 95% in patients with poorly controlled diabetes.
Aguilar-Salvatierra et al (2015) ¹⁵	Prospective study	Evaluate implant survival and stability in diabetic patients with different HbA1c levels.	Patients with diabetes can receive implant-based treatments with immediate loading safely, providing they present moderate HbA1c values. Peri-implantitis increases with elevated HbA1c.
Eskow and Oates (2017) ¹⁶	Prospective study	Placing 2 or more implants in 24 poorly controlled type 2 diabetic patients with HbA1c levels ranging between 8 to 12 %. ^[16]	Peri-implant mucositis was identified in few participants, but there was no realtion with HbA1c.
Mokeem et al (2018) ¹⁹	Prospective study	To determine the clinical and radiographic bone level (RBL) around short dental implants in type 2 diabetes mellitus, prediabetic and non-diabetic patients.	Clinical and radiographic peri-implant parameters are compromised around short dental implants in type 2 diabetes mellitus patients.

Is HbA1c Critical?

HbA1c is a form of hemoglobin formed by the non-enzymatic glycation pathway by hemoglobin's exposure to plasma glucose. Assessment of HbA1c is used as a diagnostic test to evaluate the glycemic control in diabetics. It provides an overall picture of patient's average blood sugar levels over weeks or months. This can be tested without fasting, any time of the day making the patient more convenient. HbA1c has been shown to be more strongly associated with

the insulin sensitivity in healthy subjects with normal glucose tolerance.²¹ Therefore it is a reliable biomarker and an excellent indicator of insulin resistance for testing individuals for diabetes and prediabetes. The diagnostic standard for HbA1c in diabetes by American Diabetes Association is given in Table 2.²⁰ HbA1c can predict whether the glycemic patient will encounter any microvascular or macrovascular complications. Certain illness associated with blood can affect the HbA1c values.

Table 2: Diagnostic standard for HbA1c

HbA1c	Diagnosis
<5.7%	Normal
5.7-6.4%	Prediabetes
>6.5%	Diabetes

Conclusion

Diabetes has a negative influence on bone remodeling. It indirectly reduces the osseointegration of dental implants. Various experimental studies over the past few decades has succeeded to prove that good glycemic control during the peri-operative period can improve the survival rates of dental implants in diabetics. Pre-operative antibiotic therapy, chlorhexidine mouth rinse, longer duration of post-surgical antibiotic course, bioactive material coated implants and implants with greater length and width have seen to improve the survival of implant in diabetics. More longitudinal studies with greater number of diabetic individuals and non-diabetic control are still

required to analyze the impact of diabetes over the success of dental implant.

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ENDODONTIC MANAGEMENT OF MANDIBULAR FIRST MOLAR WITH MESIAL CANAL – A CASE REPORT

***Dr. Mohammed Ashik, **Dr. Ashir KR, ***Dr. Chandini,
Dr. Sarin K, *Dr. Hamesh Gangadharan**

Abstract

The main objective of root canal treatment is the thorough mechanical and chemical cleansing of the entire pulp space followed by complete obturation with inert filling material. Knowledge of the internal anatomy of tooth is of prime importance, for the success of endodontic treatment. Numerous reason for endodontic failures are missed canals, incomplete instrumentation, inadequate cleaning and shaping of the root canal system, and subsequently defective obturation of root canal system.¹ Anatomical characteristics of the different types of teeth and their possible variations are the challenges routinely faced by practitioners while performing endodontic treatment.

Keywords: Endodontic management, Mandibular molar, Case report

Corresponding Author: Dr. Mohammed Ashik

Reader, *Senior Lecturer, Department of Conservative Dentistry, **Reader, ****Senior Lecturer, Department of Oral Medicine & Radiology, KMCT Dental College, Manassery, Kohikode 673602, Kerala, India*

Introduction

Proper access into the pulp chamber, which allows access to the orifices of the root canals and an optimal view of the chamber floor, is a fundamental step in endodontic therapy. Numerous reason for endodontic failures are missed canals, incomplete instrumentation, inadequate cleaning and shaping of the root canal system, and subsequently defective obturation of root canal system.¹ Proper access into the pulp chamber enables the identification of any variation in the number and position of root canals.²

Vertucci³ and Cunningham⁴ observed that substantial number of treatment failures could be attributed to anatomical variations,

such as the presence of extra canals which are not usually detected.

Currently, magnification is one of the major factor in accurate identification of the canals and therefore the outcome of endodontic treatment.^{3,4} Dental operating microscope, surgical loupes are few of them which help in identifying and locating canals accurately.

The present case describes left mandibular first molar with extra canals, three mesial, and two distal canals. The third canal of the mesial root is called mid-mesial canal. Current case is intended to review related literature, describe the clinical case, and make considerations about how dentists

should perform the examination of pulp chamber to achieve successful results in similar cases.

Case report

A 21-year-old patient reported with a complaint of diffuse pain in the left mandibular first molar. Pulp testing and percussion tests in the region revealed intense and continuous pain and confirmed irreversible acute pulpitis. Radiographs showed deep caries in the tooth and no changes in the apical region. No anatomical abnormality was observed on the radiographs. Endodontic treatment was performed as described below(Fig 1a,b,c).

First visit

Proper anesthesia was obtained, tooth was isolated with rubber dam, and the pulp chamber was accessed. As the negotiation of canals began with a no. 10 k-file (Mani, Inc., Japan) third mesial canal was found in between the mesiolingual and mesiobuccal canals along with two distal canals. An apex locator (Sybron endo) and a no. 10 k-file were used to establish working length that was confirmed radiographically. The instrumentation of the five visible canals was performed using the hybrid crown-down technique described by Garrett M.guess.¹⁰ Distal canals were enlarged using 6% protaper files (Dentsply India Pvt. Ltd.) and all mesial canals were prepared using 4% hero files. Ethylene diamine tetraacetic acid (Prime Dental Products Pvt. Ltd., Thane, India), saline (prime dental products.), and sodium hypochlorite (Prime Dental Products Pvt. Ltd., Thane, India) were used during

cleaning and shaping procedure. Following cleaning, shaping, and final irrigation with saline and sodium hypochlorite, the canals were dried with paper points and an intracanal dressing with calcium hydroxide (RC CAL, Prime Dental Products Pvt. Ltd., Thane, India), was given for 7 days.

Second visit

During the second visit, the calcium hydroxide intracanal dressing was removed, the master cone fit was assessed, and the root canals were dried with absorbing paper points. Root canals were obturated using the lateral condensation, temporary sealing done.

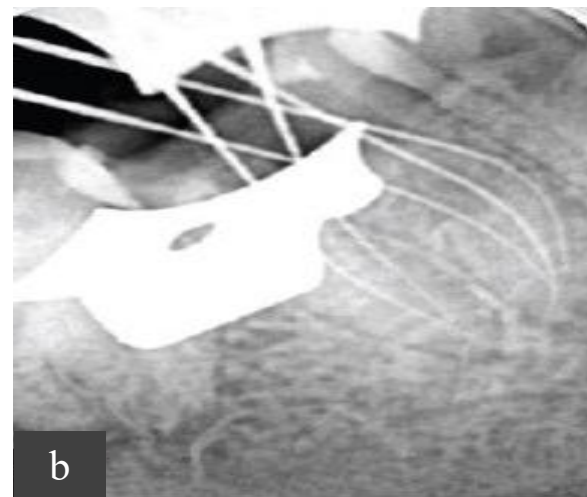




Fig 1a, b, c: Endodontic Treatment of Mandibular Canal

Discussion

Studies conducted by Riccucci. D *et al.*,¹¹ Baughd¹² and Fabra-Campos⁵ emphasized the importance of an accurate clinical evaluation of a possible fourth or fifth root canal to ensure the success of endodontic treatment. Martinez-Berna and Badanelli⁶ highlighted the importance of investigating the presence of a fourth and even a fifth root canal.

Several studies investigated the anatomy of root canal systems and the anatomical variations found in the different types of teeth to provide information that might improve the outcome of endodontic treatment. However, few studies discussed the occurrence of a third mesial canal in the mandibular first molar. New technologies, such as the dental operating microscope, offer great magnification and illumination of the operating field and substantially improve the visualization of root canal orifices. de

Carvalho and Zuolo¹³ described the importance of microscopes for accurately locating of root canal orifices, which may substantially improve treatment outcomes.

Clinical evaluations have shown a small but significant number of mandibular molars with five canals.^{6,13} The region between the mesiolingual and mesiobuccal canals should be carefully examined in case of the possible occurrence of a mid-mesial canal.

Conclusion

Successful endodontic treatment require, knowledge of root canal anatomy with correct diagnosis and careful inspection of morphological variations in the pulpal anatomy. The case reported shows mandibular first molar with mesial 3 canals and distal two canals. Even though the frequencies are rare, each case should be evaluated carefully both clinically and correlated radiographically.

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EDENTULOUS AREA- LATERAL PEDICAL GRAFT: A CASE REPORT

***Dr. Navia George, **Dr. Harish Kumar V.V., ***Dr. Santhosh V.C.**

Abstract

Gingival recession is a multifaceted problem, for which several treatment options are available. Lateral sliding flap offer most predictable solution for the treatment of gingival recession due to disrupted blood supply to the graft tissue. The case report involves a 45 year old female with Miller's class II recession on 17 along with mesio-occlusal caries indicated for root canal treatment. There were missing 16 and 15. After phase I therapy patient had undergone root canal treatment and lateral sliding flap from adjacent edentulous area and crown placement.

Key words: Lateral sliding flap, edentulous area, gingival recession

Corresponding author: Navia George

Senior Lecturer, **Professor & Head, *Professor, Department of Periodontics, KMCT Dental College, Manassery, Kozhikode, Kerala*

Introduction

Gingival recession is defined as the location of the gingival margin apical to cemento-enamel junction.¹ Clinical findings of the gingival recession includes increased root sensitivity, cervical abrasion and cervical caries.² It also causes aesthetic concern to the patient; usually in the anterior regions of the mouth.³ The periodontal literature is replete with the techniques for the correction of the gingival recession.⁴ Various factors to be considered before root coverage are the extent of the recession [Miller's classification], the width of the attached gingival at the site, the position of the tooth in the arch and some patient characteristics like smoking and oral hygiene.⁵

The aim of this case report is to demonstrate lateral sliding flap taken from an adjacent edentulous site to obtain root coverage on a posterior tooth.

Case report

A 45- year old female reported to the department with a chief complaint of decayed tooth in her upper right back tooth region. Patient desires to save that tooth since the teeth adjacent to it were missing. She was systemically healthy.

Examination of the area of chief complaint revealed that 16 had deep dental mesio-occlusal caries which was indicated for root canal treatment. A Miller's class II recession was noted on buccal surface which need correction prior to the crown placement due to inadequate width of attached gingival. [Figure 1]

Clinical procedures consist of phase I therapy followed by a maintenance period, root canal treatment and root coverage procedures.

Phase I therapy was initiated four weeks prior to the root coverage procedure. It consists of

scaling and root planning. Oral hygiene instructions were given to eliminate the faulty brushing technique.

After root canal treatment, patient was posted for root coverage procedure. The patient was asked to rinse with 10 ml of 0.2% chlorhexidine for 60 seconds, followed which, local anaesthesia was administered. The root was planed to reduce its convexity.

The root coverage procedure selected for this case was edentulous-area pedicle grafts introduced by Corn and Robinson.⁶ The steps include:

Preparation of recipient site: Recipient site was de-epithelized using no.15 blade.[Figure 2] Horizontal incision and vertical incision: Prepare a full-thickness vertical incision from the end of a horizontal incision on the edentulous alveolar ridge area to the alveolar mucosa.[Figure 3] Preparation of full-thickness pedicle flap: Make a vertical incision with a no.15 blade and turn the blade tip coronally.



Fig 1: Deep dental caries and Miller's class II recession on 16



Fig 2: De-epithelization of recipient site

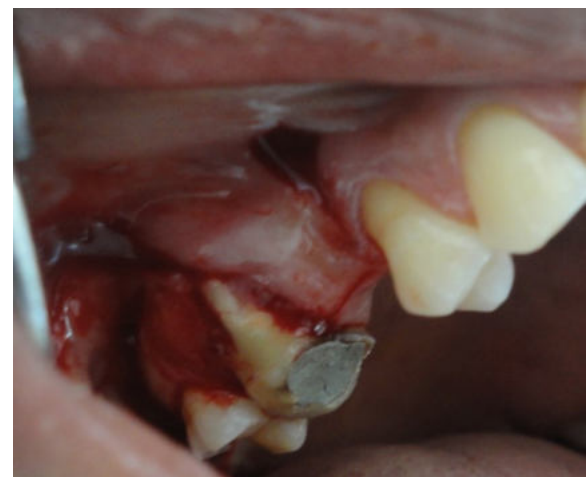


Fig 3: Flap design with both horizontal and vertical incisions

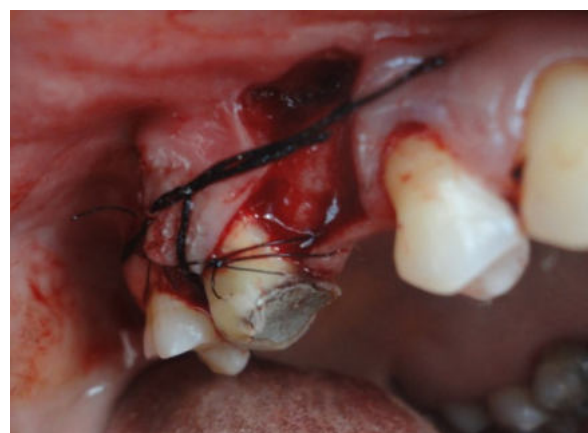


Fig 4: Suturing



Fig 5: Suture removal after 1 week



Fig 6: After 6 weeks

Prepare the full-thickness pedicle flap while positioning the blade from the alveolar mucosa towards the crown of the tooth. Reflection of full-thickness pedicle flap: Prepare a releasing incision of the periosteum at the base of the flap so the flap can be moved freely. Rotate the pedicle flap distally and displace it laterally. Place the flap on prepared recipient site and give interdental suture on the distal part of the flap using 5-0 braided silk suture. Holding suture is given to prevent displacement of the flap during healing. [Figure 4] Periodontal pack is given. Following surgery, the patient was placed on

the soft diet. He was instructed to avoid contact or trauma to the grafted site.

Post-operative medications include Cap Amoxicillin 500mg TID for 5 days and Tab Mefenamic acid 500mg + Paracetamol 325 mg TID for 3 days were given. The patient was also asked to rinse twice daily with a 0.2% CHX for 2 weeks.

Healing following the surgery was uneventful. After 1 week sutures were removed. [Figure 5] After 4 weeks complete remodelling of gingival occurred and there were around 75% root coverage obtained. Porcelain fused metal crown were placed on the root canal treated tooth. [Figure 6]

Discussion

Various techniques either single or in combination have been proposed for the management of root coverage procedure. These techniques offer different rates of success and predictability. The reported success rate of rotational flaps is 41-74%, coronally advanced flap is 70-99%, guided tissue regeneration is 54-68%, connective tissue grafts is 52-98% and that of free gingival graft is 11-87%.³

The different treatment options for this case were:

- A free gingival graft
- Coronally advanced flap
- Sub-epithelial connective tissue graft
- Lateral sliding flap
- Guided tissue regeneration utilizing a barrier membrane in combination with a coronally advanced flap
- Acellular dermal matrix graft

A free gingival graft has been for increasing the width of the keratinized and attached gingival, but complete root coverage is not achieved and this is often a limitation of this procedure.³ The idea of coronally advanced flap alone or with free gingival graft was rejected because of lack in the width of the attached gingival. Using a GTR membrane would have placed the flap under tension, predisposing the site to recession once again.⁴ Excess flap tension would have to be placed in order to fully cover the membrane, and the risk of membrane exposure could risk an unsuccessful result, which could possibly worse than the initial presentation. The success with an acellular dermal matrix was unpredictable and also this allograft was expensive.⁷ As a result, it was not used in this case. Sub-epithelial connective tissue graft would not have helped in gaining sufficient width of the attached gingival and thus the option was discarded. Therefore, in this case we decided to use an edentulous-area lateral sliding flap. In this procedure, the edentulous area is used as a donor site.

Advantages of this procedure are:⁶

- There is no danger of exposing thin marginal bone, which prevents the problems of bone loss and gingival recession at the donor site.
- There is greater likelihood that the exposed root surface will be covered because the thick full-thickness flap can be used as the pedicle graft.
- Since in this case there was an edentulous area mesial to gingival recession, we

preferred this technique to cover exposed root as well as to increase the width of the attached gingiva.

- There is very limited literature available on the use of edentulous-area pedicle grafts. Also, this technique addresses the three main criteria for root coverage as described by Gray- inadequate gingival width, impaired aesthetics, and root hypersensitivity,⁷ and it helps in the correction of all the three.

Conclusion

The results obtained in this case suggest that this surgical procedure is highly predictable for root coverage in the case of deep recession and lack of attached gingival in the tooth with adjacent edentulous area. The procedure holds promise for the successful management of complex marginal tissue recessions, although further studies are warranted.

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KMCT
DENTAL COLLEGE

Manassery P.O., Mukkom, Kozhikode 673 602 , Kerala
Phone (0495) 229 0690/229 0693, Fax(0495) 229 4726