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Ragas Dental College and Hospital,
Chennai, Tamil Nadu

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

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ROLE OF SPIRULINA IN MANAGEMENT OF BURNING SENSATION IN ORAL SUBMUCOUS FIBROSIS - RANDOMIZED CLINICAL TRIAL

Authors:
Prathima Shetty[1]
Prashanth Shenai[2]
Laxmikanth Chatra[3]
Rajesh Shetty[4]
Prasanna Kumar Rao[5]

[1]Reader,
Dept of Oral Medicine and Radiology
Yenepoya Dental College
Deralakatte, Mangaluru, Karnataka

[2]Professor
Dept of Oral Medicine and Radiology
Yenepoya Dental College
Deralakatte, Mangaluru, Karnataka

[3]Professor and HOD
Dept of Oral Medicine and Radiology
Yenepoya Dental College
Deralakatte, Mangaluru, Karnataka

[4]Professor
Dept of Prosthodontics
Yenepoya Dental College
Deralakatte, Mangaluru, Karnataka

[5]Professor
Dept of Oral Medicine and Radiology
AJ Institute of Dental Science
Kuntikana, Mangaluru, Karnataka.

Address for correspondence:

Dr. Prathima Shetty
Reader
Dept of Oral Medicine and Radiology
Yenepoya Dental College
Deralakatte, Mangaluru, Karnataka
Email : dr_prathimashetty@yahoo.co.in

ABSTRACT

OSMF is a potentially malignant disorder which debilitates a patient's quality of life. Various number of medications have been tried but not all are very effective. Antioxidants have shown to reduce the symptoms and therefore a study was conducted using Spirulina, a natural algae that is incredibly high in protein, vitamins and antioxidants, to see if this new group of antioxidant helped in improving the symptoms.

Key words : Oral Submucous fibrosis, Spirulina, Antioxidants.

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INTRODUCTION

Oral submucous fibrosis (OSMF) is a potentially malignant disorder affecting oral cavity.[1,2] Burning sensation is one of the clinical symptoms of OSMF due to atrophy of the oral mucosa. It is believed that the areca nut alkaloids and tannin play an important role in etiology of OSMF. [3] Number of studies has proven that the management of premalignant diseases should include antioxidants. Antioxidants such as lycopene, ALA100 etc. have been tried with various degree of success.[4,5] Spirulina is an antioxidant that has been used for treatment of several oral mucosal lesion with successful results, however its effects on OSMF are not well documented. Therefore, a study was carried out to evaluate role of spirulina as an antioxidant adjuvant to corticosteroid injections in treating burning sensation in OSMF.

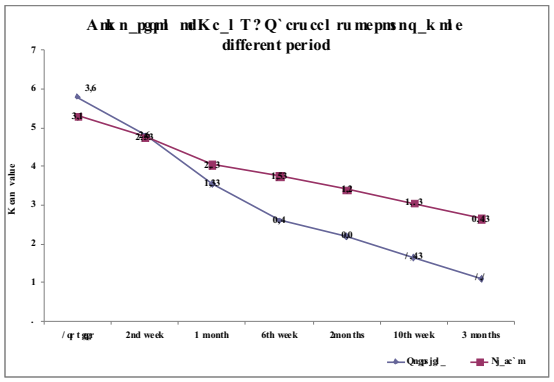
STUDY

This study consisted of 40 clinically and histopathologically confirmed stage 1 and stage 2 OSMF patients. Patients with any systemic disorders and on any other medications or therapy were excluded from the study. 40 patients were divided into two groups, group A (spirulina) and group B (placebo). Both the groups were advised to discontinue the use of areca nut in all preparations before starting treatment. Both the groups were treated with steroid injection (Betamethasone 4 mg/ml) twice a week. Group A was given antioxidants (Spirulina 500 mg) orally twice daily for 3 months and group B was given placebo capsules daily two times for 3 months. On every visit, burning sensation was evaluated by using the Visual Analog Scale (VAS), weekly over a period of 3 months. The result thus obtained was subjected to statistical analysis. Student's paired and unpaired "t" tests and Chi square test were used for statistical evaluation.

RESULTS

The data collected was statistically analyzed and the results showed the following observations. Maximum numbers of patients were in the third decade of life. The mean age of the individuals in group A was 28.05 yrs, whereas in group B the mean age was 30.75 yrs. The mean value of initial VAS of group A was 5.8 and group B was 5.3; statistically no differences were found. There was statistical significance in favor of group A found when values of both groups were compared every 15 days. In group A, VAS by the end of 2nd week compared with that of initial VAS values, the mean difference was 1.00 ± 0.79472. In group B, VAS by the end of 2nd week compared with that of initial VAS value, the mean difference was. 5500 ± 0.82558. When inter-comparison between two groups was done it was found to be statistically non-significant. VAS in group A at the end of 6 week compared with that of initial VAS mean difference value was 3.200 ± 1.19649. In group B, VAS at the end of 6 week compared with that of initial VAS mean difference value was 1.5500 ± 1.19097. When two groups were compared it was found to be statistically very highly significant in favor of group A and these values remained very highly significant even after 3 months period (post-treatment).

In group A, the mean paired VAS difference was 4.6, and ingroup B the paired VAS difference was 2.65. When both the groups were compared, VAS values were less in group A than in group B [Figure 1].



DISCUSSION

To date OSMF is poorly understood and unsatisfactorily treated. Based on clinical, epidemiological, and in vitro studies, areca nut chewing is considered an important predisposing factor. Various methods have been tried to bring relief from burning sensation, these include intralesional injections of corticosteroids, placental extracts, or hyaluronidase either alone or in combination, micronutrient

supplementation, physiotherapy, and surgery.[6] Spirulina is a microalgae, used in daily diet of natives in Africa and America. It contains phenolic acid, tocopherols, and beta-carotene which are known to exhibit antioxidant properties.[7] Spirulina has been used for the treatment of several oral mucosal lesions with successful results. However, its effects on OSMF are not well documented.

In this study, though the reduction in burning sensation was sustained in both the groups, group A seemed to be more effective when compared to group B. The relief from burning sensation in patients treated with spirulina is probably due to the contents of spirulina which contains beta-carotene, phenolic acid, tocopherols, and various micronutrients.[7] A similar finding was reported in 1997, which stated that, multiple micronutrient supplements produced relief from burning sensation in 85% patients.[8] Administration of beta-carotene systemically and topically would improve the integrity of the epithelium as well as induce redifferentiation of dysplastic epithelium.[9]

CONCLUSION

The present study clearly emphasizes that along with habit counseling spirulina has a definitive protective role in reducing clinical signs and symptoms of OSMF. Oral antioxidant spirulina can be used as an adjuvant therapy in management of burning sensation in OSMF patients. Spirulina was well tolerated by these patients and no side effects were reported as well. More patient sample, with longer period of follow-up, is required to get further conclusion on the role of spirulina in the management of burning sensation in OSMF.

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TOOTH BLEACHING
A CRITICAL REVIEW

ABSTRACT

Present tooth-bleaching techniques are based upon hydrogen peroxide as the active agent. It is applied directly, or produced in a chemical reaction from sodium perborate or carbamide peroxide. More than 90% immediate success has been reported for intracoronal bleaching of non-vital teeth. Cervical root resorption is a possible consequence of internal bleaching and is more frequently observed in teeth treated with the thermo-catalytic procedure. When the external tooth-bleaching technique is used, the first subjective change in tooth color may be observed after 2-4 nights of tooth bleaching, and more than 90% satisfactory results have been reported. Tooth sensitivity is a common side-effect of external tooth bleaching observed in patients, but clinical studies addressing the risk of other adverse effects are lacking. This review article will help clinicians improve their understanding of the history of bleaching procedures, bleaching types, components, mechanisms, and their effects on soft tissue, tooth structures, resin composite, and bonding.

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Authors:
John Paul[1]
Meera Gopalakrishnan[2]
Dinesh Kamath[2]
Ajay Joseph[1]

[1]Senior Lecturer
Dept. of Conservative Dentistry & Endodontics
Indira Gandhi Institute of Dental Sciences,
Kothamangalam, Ernakulam Dt., Kerala

[2]Professor,Dept. of Conservative Dentistry & Endodontics
Indira Gandhi Institute of Dental Sciences,
Kothamangalam, Ernakulam Dt., Kerala

Address for correspondence

Dr. John Paul
Sr. Lecturer
Indira Gandhi Institute of Dental Sciences,
Dept. of Conservative Dentistry & Endodontics
Indira Gandhi Institute of Dental Sciences,
Kothamangalam, Ernakulam Dt., Kerala
Email : john_paul_0077@yahoo.com

INTRODUCTION

The history of dentistry is comprised of many efforts undertaken to achieve an effective tooth-whitening method. Tooth discoloration varies in etiology, appearance, localization, severity, and adherence to tooth structure. It may be classified as intrinsic, extrinsic, and a combination of both. Intrinsic discoloration is caused by incorporation of chromatogenic material into dentin and enamel during odontogenesis or after eruption. Exposure to high levels of fluoride, tetracycline administration, inherited developmental disorders, and trauma to the developing tooth may result in pre-eruptive discoloration.[1] After eruption of the tooth, aging, pulp necrosis, and iatrogenesis are the main causes of intrinsic discoloration. Coffee, tea, red wine, carrots, oranges, and tobacco give rise to extrinsic stain.[2] Wear of the tooth structure, deposition of secondary dentin due to aging or as a consequence of pulp inflammation, and dentin sclerosis affect the light-transmitting properties of teeth, [3] resulting in a gradual darkening of the teeth. Scaling and polishing of the teeth remove many extrinsic stains. For more stubborn extrinsic discoloration and intrinsic stain, various bleaching techniques may be attempted. Tooth bleaching can be performed externally, termed night guard vital bleaching or vital tooth bleaching, or intracoronallyin root-filled teeth, called non-vital tooth bleaching. The aims of the present paper are to review critically the literature on the biological aspects of tooth bleaching, including efficacy and side-effects of such treatments.

(II) History of Bleaching

Bleaching of discolored, pulpless teeth was first described in 1864 by Truman, and a variety of medicaments such as chloride, sodium hypochlorite, sodium perborate, and hydrogen peroxide has been used, alone, in combination, and with and without heat activation.[4] The "walking bleach" technique that was introduced in 1961 involved placement of a mixture of sodium perborate and water into the pulp chamber that was sealed off between the patient's visits to the clinician. The method was later modified and water replaced by 30-35% hydrogen peroxide, to improve the whitening effect. [5] The observation that carbamideperoxide caused lightening of the teeth was made in the late 1960s by an orthodontist who had prescribed an antiseptic containing 10% carbamide peroxide to be used in a tray for the treatment of gingivitis. The observation was communicated to other colleagues and must be regarded as the beginning of the night guard bleaching era.

The ''over-the-counter'' (OTC) bleaching agents were first launched in the United States in the 1990s, containing lower concentrations of hydrogen peroxide or carbamide peroxide and sold directly to consumers for home use.[6] Finally, the current in-office bleaching technique typically uses different concentrations of hydrogen peroxide, between 15% and 40%, with or without light and in the presence of rubber dam isolation.

3. Composition of commercial bleaching agents

Current bleaching agents contain both active and inactive ingredients. The active ingredients include hydrogen peroxide or carbamide peroxide compounds. However, the major inactive ingredients may include thickening agents, carrier, surfactant and pigment dispersant, preservative, and flavoring.

(a) Thickening agents:

Carbopol (carboxypolymethylene) is the most commonly used thickening agent in bleaching materials. Its concentration is usually between 0.5% and 1.5%. This high-molecular-weight polyacrylic acid polymer offers two main advantages. First, it increases the viscosity of the bleaching materials, which allows for better retention of the bleaching gel in the tray. Second, it increases the active oxygen-releasing time of the bleaching material by up to 4 times[7].

(b) Carrier:

Glycerin and propylene glycol are the most commonly used carriers in commercial bleaching agents. The carrier can maintain moisture and help to dissolve other ingredients.

c) Surfactant and pigment dispersant:

Gels with surfactant or pigment dispersants may be

more effective than those without them.[8] The surfactant acts as a surface-wetting agent which permits the active bleaching ingredient to diffuse. Moreover, a pigment dispersant keeps pigments in suspension.

(d) Preservative:

Methyl, propylparaben, and sodium benzoate are commonly used as preservative substances. They have the ability to prevent bacterial growth in bleaching materials. In addition, these agents can accelerate the breakdown of hydrogen peroxide by releasing transitional metals such as iron, copper, and magnesium.

(e) Flavoring:

Flavorings are substances used to improve the taste and the consumer acceptance of bleaching products. Examples include peppermint, spearmint, wintergreen, sassafras, anise, and a sweetener such as saccharin.

4. Mechanism of tooth bleaching

The mechanism of bleaching by hydrogen peroxide is not well understood. In-office and home bleaching gels contain hydrogen peroxide or its precursor, carbamide peroxide, as the active ingredient in concentrations ranging from 3% to 40% of hydrogen peroxide equivalent. Hydrogen peroxide bleaching generally proceeds via the perhydroxyl anion(HO_2^-). Other conditions can give rise to free radical formation, for example, by homolytic cleavage of either an O-H bond or the O-O bond in hydrogen peroxide to give H^- + OOH^- and $2OH^-$ (hydroxyl radical), respectively. [9] Under photochemical reactions initiated by light or lasers, the formation of hydroxyl radicals from hydrogen peroxide has been shown to increase. Hydrogen peroxide is an oxidizing agent that, as it diffuses into the tooth, dissociates to produce unstable free radicals which are hydroxyl radicals (HO^-), perhydroxyl radicals (HOO^-), perhydroxyl anions (HOO^-), and superoxide anions (OO^-), which will attack organic pigmented molecules in the spaces between the inorganic salts in tooth enamel by attacking double bonds of chromophore molecules within tooth tissues.[10,11] The change in double-bond conjugation results in smaller, less heavily pigmented constituents, and there will be a shift in the absorption spectrum of chromophore molecules; thus, bleaching of tooth tissues occurs. In the case of tetracycline-stained teeth, the cause of discoloration is derived from photo-oxidation of tetracycline molecules available within the tooth structures.[12] The bleaching mechanism in this case takes place by chemical degradation of the unsaturated quinone-type structures found in tetracycline, leading to fewer colored molecules. Vital bleaching via a long-term night guard can sometimes improve the color of tetracycline-stained teeth.[13] More recently, amorphous calcium phosphate (ACP) has been added to some of the tooth whitening products, to reduce sensitivity, reduce the demineralization of enamel through a remineralization process after whitening treatments, and add a lustrous shine to teeth. A study proved that the bleaching treatments promoted increased sound enamel demineralization, while the addition of Ca ions or ACP did not prevent/reverse the effects caused by the bleaching treatment in both conditions of the enamel. Early artificial caries induced by pH cycling model were not affected by the bleaching treatment, regardless of the type of bleaching agent.[14]

Types of dental bleaching procedures

Vital tooth bleaching

There are three fundamental approaches for bleaching vital teeth: in-office or power bleaching, at-home or dentist supervised night-guard bleaching, and bleaching with over-the-counter (OTC) products.[15] First, in-office bleaching utilizes a high concentration of tooth-whitening agents (25–40% hydrogen peroxide). Here, the dentist has complete control throughout the procedure and has the ability to stop it when the desired shade/effect is achieved. In this procedure, the whitening gel is applied to the teeth after protection of the soft tissues by rubber dam or alternatives [16] and the peroxide will further be activated (or not) by heat or light for around one hour in the dental office [17]. Different types of curing lights including; halogen curing lights, Plasma arc lamp, Xe–halogen light (Luma Arch), Diode lasers (both 830 and 980 nm wavelength diode lasers), or

Metal halide (Zoom) light can be used to activate the bleaching gel or accelerate the whitening effect. The in-office treatment can result in significant whitening after only one treatment, but many more may be needed to achieve an optimum result. Second, at-home or dentist-supervised night-guard bleaching basically involves the use of a low concentration of whitening agent (10–20% carbamide peroxide, which equals 3.5–6.5% hydrogen peroxide). In general, it is recommended that the 10% carbamide peroxide be used 8 h per day, and the 15-20% carbamide peroxide 3–4 h per day. This treatment is carried out by the patients themselves, but it should be supervised by dentists during recall visits. The bleaching gel is applied to the teeth through a custom-fabricated mouth guard worn at night for at least 2 weeks. This technique has been used for many decades and is probably the most widely used. The at-home technique offers many advantages: self administration by the patient, less chair-side time, high degree of safety, fewer adverse effects, and low cost. Despite the fact that patients are able to bleach at their own pace, this at-home bleaching technique, with its various concentrations of bleaching materials and regimens, has become the gold standard by which other techniques are judged. However, it is by no means without disadvantages, since active patient compliance is mandatory and the technique suffers from high dropout rates In addition, color change is dependent on diligence of use, and the results are sometimes less than ideal, since some patients do not remember to wear the trays every day. In contrast, excessive use by overzealous patients is also possible, which frequently causes thermal sensitivity, reported to be as high as 67% . A 35% concentration of hydrogen peroxide is recommended by some clinicians for in-office dental bleaching, followed by at-home bleaching with gels containing 10%, 15%, or 20% carbamide peroxide.[18]

Bailey and Swift showed that higher-concentration bleaching agents can produce more peroxide radicals for bleaching, resulting in a faster whitening process. However, this rapid process of bleaching may increase the side-effects of tooth sensitivity, gingival irritation, throat irritation, and nausea.

Finally, over-the-counter (OTC) bleaching products have increased in popularity in recent years. These products are composed of a low concentration of whitening agent (3–6% hydrogen peroxide) and are self-applied to the teeth via gum shields, strips, or paint-on product formats. They are also available as whitening dentifrices, pre-fabricated trays, whitening strips, and toothpastes.[19] They should be applied twice per day for up to 2 weeks. OTC products are considered to be the fastest growing sector of the dental market. However, these bleaching agents may be of highly questionable safety, because some are not regulated by the Food and Drug Administration.

Non-vital tooth bleaching

There are numerous non-vital bleaching techniques used today, for example, walking bleach and modified walking bleach, non-vital power bleaching, and inside/outside bleaching. The walking bleach technique involves sealing a mixture of sodium perborate with water into the pulp chamber of the affected tooth, a procedure that is repeated at intervals until the desired bleaching result is achieved. This technique is modified with a combination of 30% hydrogen peroxide and sodiumperborate sealed into the pulp chamber for one week; this is known as modified walking bleach. In internal non-vital power bleaching, hydrogen peroxide gel (30–35%) is placed in the pulp chamber and activated either by light or heat, and the temperature is usually between 50° and 60°C maintained for five minutes before the tooth is allowed to cool for a further 5 min. Then, the gel is removed, the tooth is dried, and the 'walking bleach technique' is used between visits until the tooth is reviewed 2 weeks later to assess if further treatment is needed. Finally, the inside/outside bleaching technique is a combination of internal bleaching of non-vital teeth with the home bleaching technique.[20]

Effects of the Bleaching process

Local side-effects

Tooth sensitivity

Tooth sensitivity is a common side-effect of external tooth bleaching. Data from various studies of 10%

carbamide peroxide indicate that from 15 to 65% of the patients reported increased incidence of tooth sensitivity (from 67 to 78%) was reported after in-office bleaching with hydrogen peroxide in combination with heat. Tooth sensitivity normally persists for up to 4 days after the cessation of bleaching but a longer duration of up to 39 days has been reported . In a clinical study that compared two different brands of 10% carbamide peroxide bleaching agent, 55% of the patients reported tooth sensitivity and/or gingival irritation, and 20% of those who experienced side-effects terminated the treatment due to discomfort. The mechanisms that would account for the tooth sensitivity after external tooth bleaching have not yet been fully established.

In vitro experiments have shown that peroxide penetrated enamel and dentin and entered the pulp chamber [21] and that the penetration of restored teeth was higher than that of intact teeth. The amount of peroxide detected in the pulp chamber was related to the concentration of hydrogen peroxide in the preparations applied, [22] and also varied among different brands of bleaching agents with the same declared concentration of carbamide peroxide.

Effects on soft tissues

The more powerful in-office bleaching (30–35% hydrogen peroxide) can easily produce soft-tissue burns, turning the tissue white. In general, these tissue burns are reversible with no long-term consequences if the exposure to the bleaching material is limited in time and quantity.

Rehydration and application of an antiseptic ointment quickly return the color to the tissue. Therefore, it is very important to protect soft tissues with a rubber dam or other measures to prevent tissue burns. In addition, soft-tissue irritation has been reported with at-home bleaching. This irritation is most likely due to an ill-fitting tray rather than to the bleaching agent itself.

Systemic effects

There is more concern about the possible adverse effects of home-bleaching agents, although their concentrations are far below those of in-office bleaching agents, because the latter are controlled by the dentist. Occasionally, patients report gastrointestinal mucosal irritation, e.g., a burning palate and throat, and minor upsets in the stomach or intestines. However, most reports in the literature have concluded that the use of low concentrations of hydrogen peroxide in tooth bleaching is still safe.[23,24]

Effects on Enamel surface morphology and texture

Many studies in the literature have investigated the effects of bleaching on enamel morphology and the surface texture morphological alteration of the enamel surface - increased porosity of the superficial enamel structure, demineralization and decreased protein concentration, organic matrix degradation, modification in the calcium: phosphate ratio, and calcium loss - thereby supporting the hypothesis that bleaching agents are chemically active components potentially able to induce substantial structural alterations in human dental enamel.

Effects on enamel chemical composition

Regarding the effect of dental bleaching on enamel chemical composition, many studies examined it by measuring the changes in constituent enamel elements Al-Salehi et al [25] found that tooth-bleaching agents might adversely affect tooth structure by demonstrating that, with increasing hydrogen peroxide concentrations, ion release from both enamel and dentin increased, and that microhardness of enamel decreased significantly with bleaching. Moreover, Efeoglu et al used micro-computerized tomography to evaluate the effect of 10% carbamide peroxide applied to enamel. Results indicated that this was found to cause demineralization of the enamel extending to a depth of 50μm below the enamel surface.[26] Therefore, they recommended that the application of bleaching agents should be carefully considered in patients susceptible to caries and tooth wear.

Effects of dental bleaching on composite resin restorations.

Surface properties and microhardness

Most studies addressing the effects of bleaching agents on the surface properties of composite showed that the effect of bleaching on the surface texture is material- and time-dependent [28]. In some SEM studies and profilometric analyses, it was shown that 10-16% carbamide peroxide bleaching gels may lead to a slight, but statistically significant, increase in surface roughness and numbers of porosities of microfilled and hybrid composite resins [28].

However, in another SEM study, it was concluded that the application of 6% hydrogen peroxide gel to a hybrid composite in a cycling protocol, with intermittent storage in saliva, could modify or weaken the impact of the hydrogen peroxide by formation of a surface-protective salivary layer on the restorative material.[29]

Genotoxicity and Carcinogenicity of Bleaching Agents

The genotoxicity of hydrogen peroxide and of tooth whiteners containing carbamide peroxide has been evaluated. The consensus arising from these evaluations was that direct contact with hydrogen peroxide induced genotoxic effects in bacteria and cultured cells.

When hydrogen peroxide was administered to bacteria or cultured cells in the presence of catalase or other metabolizing enzymes, the effect was reduced or abolished. Testing of hydrogen peroxide for systemic genotoxic effects in animals revealed no evidence of in vivo mutagenicity. Since hydroxy radicals, perhydroxyl ions, and superoxide anions formed from hydrogen peroxide are capable of attacking DNA, the genotoxic potential of hydrogen peroxide is dependent on the accessibility of free radicals to target DNA.[30] This may explain why hydrogen peroxide induces geno toxicity in the presence of metabolizing enzymes neither in vitro nor in vivo. Tooth whiteners containing carbamide peroxide were mutagenic in certain bacterial strains and non mutagenic in the presence of additional activating enzymes.[31] Several in vivo studies addressing the formation of micronuclei in bone marrow cells and sister chromatide exchange after exposure to carbamide-peroxide containing products revealed no genotoxic effects.[32]

SUMMARY

The increasing demand for tooth bleaching has driven many manufacturers and researchers to develop bleaching products to be used either in the dental office or at home. However, as with any dental procedure, bleaching involves risks. For that reason, this review article is provided to help clinicians improve their information about the bleaching process and their understanding of the controversial issues regarding the effects of bleaching on teeth, resin composite, and bonding, to help reduce the risks to patients. To minimize the risks, the involvement of dental professionals, the prevention of using of OTC bleaching products and the reduction of overused of bleaching products are necessary.

In addition to that interval of 2 weeks post-bleaching procedure is found to be adequate to avoid adverse effects on the polymerization. The need for bleaching solely to achieve a "perfect" smile and a youthful look is thus questioned. We urge the dental profession to maintain high ethical standards and not to recommend performing cosmetic adjustment of tooth color just to comply with the demand of the patient. We advocate a more selective use of tooth bleaching and a limitation on its use to patients for whom such treatment could be professionally justified.

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GROWTH FACTORS IN PERIODONTAL REGENERATION

Authors:

Sasikumar Karuppanan[1]
Sugumari Elavarasu[2]
Thangakumaran[3]
Mohammed Shereef[4]

[1]Reader,
Dept of Periodontics,
JKKN Dental College and Hospitals
Kumarapalayam, Tamil Nadu

[2]Professor & HOD,
Dept of Periodontics,
JKKN Dental College and Hospital,
Kumarapalayam, Tamil Nadu

[3]Reader,
Dept of Periodontics,
J.K.K.N Dental College.
Kumarapalayam, Tamil Nadu

[4]Reader,
Dept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam.

Corresponding author:
Dr. Sasikumar Karuppanan,
Reader,
Dept of Periodontics,
JKKN Dental College and Hospitals,
Kumarapalayam, Namakkal,
Tamil Nadu-638183,
Email: varunisasi@rediffmail.com

ABSTRACT

The term growth factor refers to a naturally occurring protein capable of stimulating cellular growth, proliferation and cellular differentiation. Polypeptide growth factors represent a class of biological mediators that regulate critical cellular activities, including migration, proliferation, differentiation, and matrix synthesis. Since the late 1980s, there has been a concerted effort to increase the knowledge of how polypeptide growth factors influence the repair and regeneration of tissues. The various types of growth factors were classified and studied to regenerate the lost periodontal tissues. The aim of this review is to study the growth factors (GFs) used in the periodontal regeneration and also to compare the various studies to understand the probable mode of action of growth factors for regenerating the lost periodontal tissues.

Key Words: Growth Factors, Periodontitis, Periodontal Regeneration, Signalling Molecules.

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INTRODUCTION:

Periodontitis, evoked by the bacterial biofilm (dental plaque) that forms around teeth, progressively destroys the periodontal tissue supporting the teeth, including the periodontal ligament, cementum, alveolar bone and gingiva.[1] Ultimately, this chronic inflammatory disease can lead to loss of the affected teeth. All over the world this disease remains highly prevalent and is considered to threaten quality of life (QOL) of middle-aged and older populations as far as ''oral'' functions are concerned. Some success has been achieved in suppressing progression of periodontitis by mechanically removing bacterial biofilm.[2]

However, removal of the cause, bacterial plaque, with conventional periodontal and/or surgical treatments can, at best, reduce pocket depth and diminish inflammation in the affected region.[3] No such treatment can ever regenerate lost periodontal tissue or normal structure and functionality. Considering that the ''mouth'' and ''teeth'' have various aesthetic and functional roles to play, establishing a brand-new treatment that enables the regeneration and rebuilding of periodontal tissue once destroyed by periodontal disease represents a task of tremendous importance.[4]

Growth Factors:

The term growth factor refers to a naturally occurring protein capable of stimulating cellular growth, proliferation and cellular differentiation. Growth factors are important for regulating a variety of cellular processes. Growth factors typically act as signalling molecules between cells. Examples are cytokines and hormones that bind to specific receptors on the surface of their target cells. Polypeptide growth factors represent a class of biological mediators that regulate critical cellular activities, including migration, proliferation, differentiation, and matrix synthesis.[5]

Since the late 1980s, there has been a concerted effort to increase the knowledge of how polypeptide growth factors influence the repair and regeneration of tissues. These naturally occurring ligands have been shown to have pleiotropic effects; they support regeneration in several settings and accelerate healing processes. They exert their effects by binding to specific cell membrane receptors to initiate complex cascades that eventually reach a nuclear target gene to generate signals for specific phenotype expression.[6]

Examples of polypeptide growth factors in bone, cementum, and healing tissues include platelet derived growth factor, vascular endothelial growth factor etc. They often promote cell differentiation and maturation, which varies between growth factors.[7] For example, bone morphogenetic proteins stimulate bone cell differentiation, while fibroblast growth factors and vascular endothelial growth factors stimulate blood vessel differentiation (angiogenesis).[8]

Growth factors vs cytokines:

The term growth factor is sometimes used interchangeably among scientists with the term cytokine. Historically, cytokines were associated with hematopoietic (blood forming) cells and immune system cells (e.g., lymphocytes and tissue cells from spleen, thymus, and lymph nodes). For the circulatory system and bone marrow in which cells can occur in a liquid suspension and not bound up in solid tissue, it makes sense for them to communicate by soluble, circulating protein molecules.[9]

While growth factor implies a positive effect on cell division, cytokine is a neutral term with respect to whether a molecule affects proliferation. While some cytokines can be growth factors, such as G-CSF and GM-CSF, others have an inhibitory effect on cell growth or proliferation. Some cytokines, such as Fas ligand are used as "death" signals; they cause target cells to undergo programmed cell death or apoptosis.

History of Growth Factors:

In 1917, Neuhof was the first to describe heterotopic osteogenesis, followed by Huggins in 1930. Growth factors were first described when substances such as blood fluids (for example, foetal calf serum) and tissue extracts were added to cells in tissue culture. Their effect was to alter, usually stimulate, cell proliferation, differentiation or migration.[8]

The study of growth factors is still largely a tissue culture-based (in vitro) science; however, currently there is a great deal of interest in the identification of growth factors in living animal and human systems (in vivo) and in their clinical applications. In the early days of growth factor/cytokine discovery they were being described with great rapidity and, in many cases, the same molecules were given different names by different investigators.[11] Urist et al. (1965)[12] reported that in rats, demineralized bone tissue intramuscular implantation led to formation of new bone.

Functions of Growth Factors

Growth factors regulate cell activity by a number of mechanisms. Importantly, all these may occur simultaneously, and in different tissues where the effects may be different, depending upon the conditions.[13]

1. Mitogenic activity

This was one of the earliest discovered mechanisms of growth factor action because in certain concentrations growth factors substantially increase the rate of cell turnover. It has been proposed that mitosis is stimulated by many growth factors through secondary intracellular messengers. Platelet derived growth factor (PDGF) and epithelial growth factor (EGF) are typical growth factors which stimulate mitosis through the regulation of intracellular calcium and pH.

2. Cell differentiation

The growth factors stimulate cell differentiation and inhibit mitosis. A classic example of this is TGF-ß. It is considered that at least one of the TGF-ß families of growth factors can initiate cellular differentiation.

3. Cell migration

Cell migration is an extremely complex cellular activity. The precise interaction between extracellular substance, growth factors and intracellular events is important in the overall process of migration. Many growth factors influence cell migration.

4. Gene regulation

Growth factors that regulate mitosis and differentiation obviously regulate gene activity. It is now well understood that growth factors operate through highly complex intracellular pathways to regulate intracellular pH and calcium, thereby influencing genetic activity. Insulin-like growth factor 2 (IGF-2), EGF and PDGF are examples of factors which regulate genes.

Mechanism of Action:[14]

Growth factors are among the most potent of biological substances being biologically active at concentrations of picograms per millimetre. The specificity and magnitude of the response is believed to be mainly due to the type and abundance of membrane bound receptors rather than availability of the growth factors. Growth factors are not typical cell nutrients which act within cells, but rather are secreted by cells and interact with specific, membrane-bound glycoprotein receptors, which are crucial in the conversion of the first message into second message or intracellular message which can act within the cell and initiate a series of biochemical reactions that eventually result in cell division.

Biological mediators for growth factors activation:[15]

1)Growth factor receptor 2)Second messengers

3)Transcription factors

Growth factor receptor (biochemistry):

In biochemistry, a receptor is a protein molecule, embedded in either the plasma membrane or cytoplasm of a cell, to which a mobile signalling (or "signal") molecule may attach.

Types of receptors :[16]

Receptors can be roughly divided into two major classes:

1. Cell-surface receptors and

2. Intracellular receptors

Ligand-gated ion channel receptors are a class of receptor that may occur both at the cell-surface and intracellularly. There are many different classes of transmembrane receptor that recognize different extracellular signalling molecules. The examples of receptors are:

1. G-protein coupled receptors, e.g., Chemokine receptors.

2. Receptor tyrosine kinases, e.g., Growth factor receptors.

3. Integrins

4. Toll-like receptors

Individual growth factor proteins tend to occur as members of larger families of structurally and evolutionarily related proteins. There are many families which are listed below:

All this growth factors Stimulates cell cycle from G0 phase to G1 phase.

E P MU RF D? ARMP	MP CE CL Q	CDDCA RQ
Cngbcpk _j epmu rf d_armp	èActivated macrophages •Salivary glands •Keratinocytes	èI cp_rgl mawrc _l b dg`pm`j_qr mitogen •Keratinocyte migration •Granulation tissue formation
Rp_l qdmpk gl e epmu rf d_armp-α	è? arg t _rcb macrophages •T-lymphocytes •Keratinocytes	èF cn_rma wrc _l b cngrhelial cell proliferation •Expression of antimicrobial peptides
F cn_rma wrc epmu rf d_armp	èK cqc l af wk _j cells	èCngrf cjg j _l b cl bmrf cjg_j cell proliferation •Hepatocyte motility
T _qa sj_p cl bmrf cjg_j epmu rf d_armp	èK cqc l af wk _j cells	èT _qa sj_p permeability •Endothelial cell proliferation
Nj_rcjcr bcpg t cb epmu rf d_armp	èNj_rcjcrq •Macrophages •Endothelial cells •Smooth muscle cells •Keratinocytes	èE p_l sjma wrc*k _apmmf_ec* fibroblast and smooth muscle cell chemotaxis •Granulocyte, macrophage and fibroblast activation •Fibroblast, endothelial cell and smooth muscle cell proliferation •Matrix metalloproteinase, fibronectin and hyaluronan production •Angiogenesis •Wound remodelling •Integrin expression regulation
Dg`pm`j_qr epmu rf d_armp/ _l b 0	èK _arophages •Mast cells •T-lymphocytes •Endothelial cells •Fibroblasts	èDg`pm`j_qr af ck mr_vgq •Fibroblast and keratinocyte proliferation •Keratinocyte migration •Angiogenesis •Wound contraction •matrix deposition
Rp_l qdorming growth factor-β	èNj_rcjcrq •T-lymphocytes •Macrophages •Endothelial cells •Keratinocytes •Smooth muscle cells •Fibroblasts	èE p_l sjma wrc*k _apmmf_ec* lymphocyte,fibroblast and smooth muscle cell chemotaxis •TIMP synthesis •Angiogenesis •Fibroplasia •Matrix metalloproteinase production inhibition •Keratinocyte proliferation
I cp_rgl mawrc epmu rf d_armp	èDg`pm`j_qrq	èI cp_rgl mawrc k gep_rgml* proliferation and differentiation

Application of Growth Factors in Periodontal Regeneration

Bone Morphogenetic Proteins (BMPs):

The studies indicated that the primary actions of BMPs are to differentiate mesenchymal precursor cells into cartilage- and bone-forming cells. TGF-β, IGFs, and FGFs all affect the already differentiated or committed bone-forming cells present in the bone, causing them to divide and/or increase secretion of extracellular matrix molecules. By affecting the cells of the bone itself, they have somewhat limited capacity for regeneration.[17] On the other hand, rhBMP-2 will affect the precursor cells, presumably cells from the marrow environment and the soft tissue surrounding the defect site, and stimulate them to infiltrate the defect area and differentiate into cartilage and bone cells.[18]

Wikesjo¨ et al. evaluated hBMP-12 for periodontal tissue regeneration, particularly periodontal ligament formation. hBMP-12 and hBMP-2 were implanted on absorbable collagen sponges in periodontal defects and the results were compared after 60 days of healing. Greater bone regeneration was observed in implants treated with hBMP-2, but ankylosis was noted. Defects treated with hBMP-12 showed less bone regeneration, but exhibited a functionally oriented periodontal ligament system inserting into newly formed cementum.[19]

Platelet Derived Growth Factor:

Its primary effect is as a potent mitogen, initiating cell division, and as a chemotactic factor for cells of mesenchyme origin, including osteoblasts. Several subtypes of PDGFs exist; they consist of homodimers or heterodimers of the PDGF-A and PDGF-B gene products. The most intense area of PDGF research involves the use of platelet-rich-plasma (PRP).

Only recently it has been found that a platelet-derived growth factor receptor is present in osteoblast.[20] Although little is known about the regulation of platelet-derived growth factor AA synthesis, some evidence suggested that platelet derived growth factor AA activity is regulated at the level of receptor binding. Platelet-derived growth factor BB might be critical in wound healing or fracture repair, since it is released after platelet aggregation. It may also play an entirely different role in bone cell physiology than that of platelet derived growth factor AA.[21]

The use of recombinant human PDGF for Periodontal Regeneration

Histological evidence of periodontal regeneration was first reported in defects in beagle dogs. During the development of platelet-derived growth factor for clinical use, recombinant human platelet derived growth factor was used in conjunction with allogenic bone to correct class II furcations and interproximal intrabony defects on teeth with poor prognosis which are to be extracted.[22] Histological evidence of periodontal regeneration was present with excellent furcation fill in this study as well as other studies also.[23]

Subsequently, the effectiveness of 0.3 mg / ml of recombinant human platelet-derived growth factor and tricalcium phosphate (GEM-21) in the improvement in clinical attachment level and bone level were studied. The tricalcium phosphate demonstrated a significant amount of regeneration after 6 months. Another case series suggested that recombinant human platelet derived growth factor with freeze-dried bone allograft can be combined to achieve excellent results in severe periodontal intrabony defects.

Transforming Growth Factor-β

Transforming growth factor-β belongs to a large superfamily of related proteins that also includes BMPs, growth and differentiation factors, activins, inhibins and anti-Mullerian hormone. All members play important roles in regulating cell proliferation and differentiation and the production of extracellular matrix. There are five isoforms of transforming growth factor-β (transforming growth factor-β1 to transforming growth factor-β5).[24] Most cells synthesize and respond to transforming growth factor-β, but high levels were found in bone, platelets and cartilage.

Transforming growth factor β is synthesized by osteoblasts in inactive (latent) form. Transforming growth factor β has been shown to stimulate pre-osteoblast cell replication, osteoblastic collagen synthesis, bone matrix apposition and alkaline phosphatase activity. However, transforming growth factor β also appears to retard terminal differentiation of osteoblasts.[25]

Fibroblast Growth Factor

The fibroblast growth factors are a family of structurally related polypeptides that are known to play a critical role in angiogenesis and mesenchymal cell mitogenesis. To mediate their range of effects, fibroblast growth factor proteins signal via membrane-spanning tyrosine kinases and there are a wide variety of mechanisms for receptor regulation and availability. In normal adult tissues, the most abundant proteins are fibroblast growth factor-1 and fibroblast growth factor-2.[26] Fibroblast growth factor-2 is expressed by osteoblasts and is generally more potent than fibroblast growth factor-1, although the expressions of other fibroblast growth factors are not nearly as ubiquitous.[27]

The use of recombinant human FGF-2 for Periodontal Regeneration:

Several growth factors have recently received attention because of their ability to actively regulate various cellular functions of periodontal ligament (PDL) cells and the effects of topical application of such factor(s) on periodontal tissue regeneration. A recent randomized controlled double-masked Phase II clinical trial at 13 Japanese dental facilities compared the therapeutic response to varying doses of fibroblast growth factor-2 vs. the control.[28] The finding of the study suggested that topical application of fibroblast growth factor-2 can be efficacious in regenerating periodontal tissue of patients with two-walled or three-walled intrabony defects.

Keratinocyte Growth Factor

Regional and temporal changes in KGF expression played important roles in the development and maintenance of epithelial structures and in epithelial wound healing. A lack of KGF expression by periodontal fibroblasts in vivo was expected to hinder apical epithelial migration and thus stabilize the epithelial attachment. The effects of retinoic acid (RA) on KGF expression in vitro provided an indirect mechanism by which KGF may regulate the growth and differentiation of gingival epithelia.[29]

Insulin-Like Growth Factors

There are two types of insulin like growth factors-IGF-I and IGF-II- that function similarly but are independently regulated. As their name indicates, IGFs are biochemically and functionally similar to insulin. They are primarily produced by the liver and circulate in the vascular system.[30]

IGF-I has been shown to be chemotactic for cells derived from the PDL.53 IGF-I also has strong effects on periodontal ligament fibroblasts (PLF) mitogenesis and protein synthesis in vitro. IGF-I receptors have also been localized on the surface of PLFs.38 The role of IGF-II on parameters of PLF and gingival fibroblast metabolism have not been reported to date.[30]

Vascular Endothelial Growth Factor

It has been demonstrated that human cultured epithelial cell sheets prepared by tissue engineering techniques provides useful graft material for wound healing and tissue regeneration.[31] This study indicated that meaningful amounts of VEGF and TGF-α and -βl were released from human cultured gingival epithelial sheets (HCGES), which suggested potential for promoting wound healing and tissue regeneration. However, limited information was available with regard to biological effects such as release of growth factors from human cultured gingival epithelial sheets (HCGES).

Epidermal Growth Factor

High levels of bound EGF were noted on periodontal ligament fibroblasts, preosteoblasts, and prechondrocytes. The EGF receptors were expressed in high amounts by the cells of the epithelial rests, and these cells were responsive to the various actions of EGF. It can be speculated that activation of the epithelial rest cells in various pathologic conditions is associated with a local rise in the tissue level of EGF. The mitogenic, chemotactic, and synthetic responses of rat periodontal ligament (PDL) fibroblastic cells to epidermal growth factor (EGF), TGF-β, IGF-I,II were examined in vitro using PDL cells obtained from the coagulum of healing tooth sockets.[32] They concluded that all this growth factors may be useful for clinical application in periodontal regenerative procedures.

CONCLUSION

The explosion of knowledge and the understanding of the role of growth factors, their mechanisms of actions and molecular signalling pathways, which have been reviewed in this topic, suggest the potential for many novel therapeutic targets, not only for applying growth factors but also for potential use of growth factors inhibitors or agents that target specific part of the intracellular signalling pathways.

There remains an enormous challenge to convert some of the knowledge from basic studies of the bone cell physiology to therapeutically useful techniques for the future. Such novel approaches may result in real qualitative improvements in clinical outcomes over currently available therapeutic modalities.

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DRUGS AND PERIODONTIUM A REVIEW

Authors:
Sai Megha Menon[1]
Jayachandran[2]

[1]Assistant Professor,
Dept of Periodontics,
Amirtha School of Dentistry,
Edapally, Ernakulam.

[2]Professor and HOD
Dept of Periodontics,
Amirtha School of Dentistry,
Edapally, Ernakulam.

Address for correspondence

Dr. Sai Megha Menon
Assistant Professor,
Dept of Periodontics,
Amirtha School of Dentistry,
Edapally, Ernakulam,
Email: saimegham07@gmail.com

ABSTRACT

Effective periodontal therapy involves a comprehensive mechanical therapy and specific antimicrobial coverage. Use of various chemotherapeutic agents in conjunction with mechanical instrumentation provides an additional beneficial effect, offering an increased opportunity to control disease. Host modulation is an emerging avenue in the use of chemotherapeutic agents.

Medications such as the anticonvulsants, antihypertensives, NSAIDs, immunosuppressants etc which have become quite common in the treatment of the multitude of lifestyle associated diseases have a profound influence on the periodontium and this influence should be understood and appropriate care has to be taken while administering these drugs. Finally, the recognition of the beneficial activity of several groups of commensal species such as probiotics might open new strategies for periodontal therapy.

This article gives an overview of the various drugs that are used in our day today lives and their influence on periodontium.

Key words: Drugs, Periodontium, antimicrobial.

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INTRODUCTION

The adult population is living longer and retaining their teeth in old age. A major part of this increase in life expectancy is attributed to an expansion in our understanding of disease processes and the subsequent explosion in drug treatments. Some of these drugs will have an impact on the periodontium and its response to bacterial plaque.

Effective periodontal prevention and therapy includes professional debridement, supragingival and subgingival antiseptics, adequate oral hygiene, specific antimicrobial coverage. However, it seems necessary to employ additional or alternative antimicrobial strategies or surgical access to effectively debride deep periodontal lesions. Incorporation of an appropriate chemotherapeutic agent in conjunction with mechanical instrumentation provides an additional antimicrobial effect offering increased opportunity to control disease. This paper reviews the various possible interactions between a patient's medication and their periodontium in both health and disease.[1]

Common Drugs Influencing the Periodontium

The effect of systemic drug therapy on the periodontium can cause an adverse effect on the periodontal tissues or afford some degree of protection against periodontal breakdown or can lead to an increased risk of periodontal breakdown. The different types of drugs that affect the periodontium include Antihypertensives, Immunosuppressants, Anticonvulsants, Non Steroidal Anti Inflammatory Drugs, Antibiotics, Corticosteroids and Hormonal replacement therapy.

Anticonvulsants, Antihypertensives, Immunosuppressants

A number of medications may cause gingival enlargement. In addition, fibrotic gingival enlargement has been reported and is believed to be the result of a genetic predisposition. Of the predisposing factors associated with gingival overgrowth, selected anticonvulsant drugs, calcium channel blockers and a potent immunosuppressant (cyclosporine A) have generated the most investigative attention in the scientific community.

These agents may reduce cytosolic calcium levels in gingival fibroblasts and T cells, thus interfering with T cell proliferation or activation and collagen synthesis by gingival fibroblasts. The gingival overgrowth results from overproduction of extracellular ground substance characterized by increased presence of sulphated-mucopolysaccharides and collagen, and abundant active fibroblast[2].

Dill et al.[3] have proposed that phenytoin increases the production of platelet-derived growth factor, and that excessive platelet-derived growth factor production would mediate gingival overgrowth. Also it may interfere with folic acid absorption and metabolism. As a result of its role in DNA synthesis, tissues with higher turnover rates are often affected first. In summary, evidence suggests a direct effect on specific subpopulations of fibroblasts, genetic predisposition, intracellular calcium metabolism exchange, molecular mechanisms (cytokines such as epidermal growth factor, platelet-derived growth factor- P), inactivation of collagenase and inflammation induced by bacterial plaque.

Phenytoin-induced gingival overgrowth is characterized by initial enlargement of the interdental papillae, and is less frequently accompanied by increased thickening of the marginal tissue. Affected tissues typically present a granular or pebbly surface, with the enlarged papillae extending facially and or lingually, obscuring the adjacent tissue and tooth surfaces.

Affected papillae may become enlarged to the point that they contact, resulting in the clinical presence of pseudoclefts. Gingival enlargement has also been associated with a number of calcium channel blockers, including nifedipine, verapamil, diltiazem, amlodipine and, to a lesser extent, isradipine. This research supports the concept that alteration of the intracellular calcium level in gingival cells by nifedipine, in combination with appropriate local inflammatory factors, is important in eliciting gingival enlargement.

It also has been shown that in patients unable to discontinue nifedipine use, gingival enlargement did not recur after gingivectomy when thorough plaque control was carried out – again supporting earlier

findings of the role of inflammation and plaque. The interdental papillae are initially affected, becoming enlarged and resulting in a lobulated or nodular morphology. These effects are limited to the attached and marginal gingiva, and are more frequently observed anteriorly, especially on the facial surfaces. The enlarged gingival tissues are often accompanied by inflammatory changes associated with poor plaque control. The gingival overgrowth results from overproduction of extracellular ground substance characterized by increased presence of sulphated-mucopolysaccharides and collagen, and abundant active fibroblasts [2].

Gingival enlargement also has been reported with cyclosporine, with an incidence of approximately 25%. In reviewing cases of patients with gingival enlargement from the above medications, it has been noted that enlargement is most severe in plaque retention areas and in tissue around periodontal pockets. The enlarged gingival tissues were soft, red or bluish-red, extremely fragile and bleed easily upon probing.

These enlarged tissues are generally more hyperaemic than the gingival tissues associated with phenytoin-induced overgrowth. Synergistic effects have been reported when cyclosporine A is administered concurrently with calcium channel blockers of the dihydropyridine derivatives (such as nifedipine)[2] . Pernu et al [4] found that patients who expressed HLA-DR1 appeared to have a protective role against gingival overgrowth from cyclosporine A, whereas those expressing HLA-DR2 showed an increased risk for overgrowth. However, many aspects of connective tissue homeostasis may serve as targets for drug-induced gingival overgrowth, and the response of the connective tissue may be the main cause of this diseased condition. [5]

Non Steroidal Anti Inflammatory Drugs

Non steroidal anti-inflammatory drugs are drugs with analgesic and antipyretic effects and anti inflammatory effects. NSAIDS inhibit the formation of prostaglandins, including PGE2, which is produced by neutrophils, macrophages, fibroblasts & gingival epithelial cells in response to the presence of lipopolysaccharide. Research shows that the periodontal benefits of taking long term NSAIDs are lost when patients stop taking the drugs, with a return to, or even an acceleration of, the rate of bone loss seen before NSAID therapy which is known as the "Rebound Effect"[6] .

The new COX-2 inhibitors have all the attributes of NSAIDs with a reduced risk of unwanted effects. These drugs have been evaluated as an adjunct to root surface instrumentation in patients with chronic periodontitis [7]. The results showed little clinical benefit of COX-2 inhibitors in the management of such patients, but significant reductions in gingival tissue levels of PGE2 and PGF2 [8]. Since bleeding on probing is considered to be indicative of periodontal disease, a false-positive may occur in patients taking oral anti-coagulants, antithrombic agents, non-steroidal anti-inflammatory drugs, or those taking aspirin daily in doses as low as 81mg [9].

ANTIBIOTICS

Selection of an appropriate antibiotic follows a diagnosis and clinician's decision to incorporate chemotherapeutics into treatment. Studies enrolling patients characterized as having refractory or recurrent periodontitis found systemic tetracycline and doxycycline, in conjunction with scaling and root planing, significantly reduced probing pocket depth and resulted in increased attachment gain relative to scaling and root planing and placebo[10]. Such improvements were likely due to the elimination or severe repression of A. actinomycetemcomitans in the infected site.

Hence, systemic administration of the tetracyclines may yield benefits in patients with localized aggressive periodontitis and in some patients refractory to previous mechanical therapy. However, there currently seem to be better choices of an antibiotic for systemic use. Amoxicillin, a semisynthetic penicillin is absorbed well following oral administration, and penetrates into the gingival crevicular fluid. Unfortunately, amoxicillin is highly susceptible to bacterial b-lactamases which hydrolyzes the b-lactam ring, thus destroying all antimicrobial activity of the penicillin.

As a result, amoxicillin's use as an adjunct to periodontal therapy has been limited. Augmentin, combines the antibiotic amoxicillin with a b-lactamase inhibitor, clavulanic acid which can be used as an adjunct to periodontal therapy. Azithromycin demonstrates good in vitro activity against Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis[11]. The fact that metronidazole specifically targets obligate anaerobic bacteria makes it an attractive antibiotic for use as an adjunct to periodontal therapy. Metronidazole readily penetrates into the gingival crevicular fluid[12] and achieves concentrations in excess of the minimum inhibitory concentrations established in vitro for most putative periodontal pathogens [13].

The adjunctive use of metronidazole/ciprofloxacin in periodontal therapy may be useful under certain circumstances. It is highly unlikely that metronidazole/ciprofloxacin would provide significant additive benefit in the treatment of periodontitis due to classical gram-negative anaerobic pathogens. The combination of metronidazole with amoxicillin /clavulanate does not offer any real advantage over metronidazole /amoxicillin in the vast majority of periodontal cases. [14]

Oral contraceptives and Hormonal replacement therapy

Hormones are specific regulatory molecules that modulate reproduction, growth and development and the maintenance of internal environments as well as energy production, utilization and storage[15].

Researchers have shown that changes in periodontal conditions may be associated with variations in sex hormones. Under the broad category of dental plaque induced gingival diseases that are modified by systemic factors, those associated with the endocrine system are classified as puberty, menstrual cycle and pregnancy associated gingivitis.

Estrogen receptors are found on periosteal fibroblasts, scattered fibroblasts of the lamina propria, and also periodontal ligament fibroblasts and osteoblasts.[16] They decrease keratinization while increasing epithelial glycogen that results in the diminution in the effectiveness of the epithelial barrier, increase cellular proliferation in blood vessels and increases the amount of gingival inflammation with no increase of plaque. Menopause usually begins between 45 and 55 years of age unless accelerated by hysterectomy and/or ovariectomy.

The levels of estrogen that have inhibitory effects on osteoclastic function begin to drop mainly during the late follicular and luteal phase of the menstrual cycle when women approach menopause. The effects of reduced estrogen levels on epithelial keratinisation along with decreased salivary gland flow may have other significant effects on the periodontium. Women may demonstrate menopausal gingivostomatitis and the clinical signs of this disease are drying of the oral tissues, abnormal paleness of the gingival tissues, redness and bleeding on probing and brushing. Oral discomfort is also commonly reported by postmenopausal women with burning sensation, xerostomia and bad taste.

The postmenopausal period is associated with an increased risk of osteoporotic fractures, myocardial infarction, menstrual cycle disorders, hot flushes, night sweats, vaginal dryness. The most significant problem that develops during menopause is osteoporosis[7]. The incidence of periodontitis also correlates with signs of generalized osteoporosis. It is also reported that skeletal bone mineral density is related to interproximal alveolar bone loss and, to a lesser extent, to clinical attachment loss.

CORTICOSTEROIDS

Long-term use of corticosteroids, such as methyl prednisolone and prednisone, may result in osteoporosis, which is seen mainly in long bones but also can occur in alveolar bone. Prolonged therapy with corticosteroids may favor osteoporosis, which is now regarded as a risk factor for periodontal disease. When steroids are injected directly into the gingival tissue, they cause a histological reduction in capillary permeability, a reduction in plasma cells and granulation tissue, an inhibition of collagen synthesis and a clinical improvement in hemorrhagic and hypoplastic gingivitis. When steroids are injected directly into the gingival tissue, they cause a

histological reduction in capillary permeability, a reduction in plasma cells and granulation tissue, an inhibition of collagen synthesis and a clinical improvement in hemorrhagic and hypoplastic gingivitis.[1]

Drugs affecting oral hygiene

As salivary flow decreases, these patients accumulate excessive amounts of plaque biofilm on their teeth which contain not only periodontal pathogens, but also caries pathogens[13]. Some of the more common groups of medications that cause xerostomia are cardiovascular medications (antihypertensives, diuretics, angiotensin-converting enzyme inhibitors, calcium channel blockers); antidepressants; sedatives; centrally acting analgesics; anti-Parkinson medications; and anti-allergy medications.

Xerostomia is a concern because saliva plays a major antimicrobial role in protecting both the soft and hard oral tissues. Because of their tendency to accumulate excess plaque biofilm, patients with xerostomia require not only frequent recall intervals but also careful evaluation of the occurrence of root surface and recurrent caries. Detection of these forms of caries can easily be incorporated into the scaling and root planning phase of periodontal therapy as well as periodic recall visits. [17]

CONCLUSION

Mechanical debridement with scaling and root planning can reduce total supragingival and subgingival bacterial masses but the major periodontal pathogens may persist. Systemic antibiotic therapy is administered to reinforce the mechanical periodontal treatment and support the host defense system in overcoming the infection by killing subgingival pathogens that remain after conventional mechanical therapy.

In reviewing cases of patients with gingival enlargement, the author has noted that enlargement is more severe in plaque retention areas and in tissue around periodontal pockets. Therefore, elimination of plaque retention areas is essential in these patients. The pathogenesis of periodontal disease involves a complex interplay between bacterial pathogens and the host tissues. Adjunctive host modulation, although only an emerging area of interest may prove to be promising in the treatment of patients with aggressive periodontitis as well as periodontitis that is refractory to treatment. Finally the recognition of the beneficial activity of several groups of commensal species such as probiotics, might open new strategies for periodontal therapy.[18]

Clinical Significance

It is evident that periodontal tissue is susceptible to a range of systemic medications. Such drug therapy can produce unwanted effects (e.g. gingival overgrowth), and reduce or increase the expression of periodontal disease. As far as antibiotics are concerned the increasing resistance of the periodontal pocket microbiota must remind clinicians to restrict the use of antibiotics to exceptional cases.

Local antibiotics may be of significance when relapse of periodontitis occurs and/or when surgical reintervention conflicts with the patient's emotions. The periodontium may also be the target of adverse reactions. This emphasizes the importance of regular medical and drug histories and thorough oral and periodontal screening for all patients.

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DENTIGEROUS CYST ASSOCIATED WITH BILATERAL IMPACTED SUPERNUMERARY TEETH- A CASE REPORT

Authors:
P.G. Antony [1]
Vimalraj R[2]
Bobby John[3]
Shiney Dominic[4]

[1] Assossiate Professor,
Dept. of Oral & Maxillofacial Surgery
Govt. Dental College, Kottayam.

[2]Senior Resident,
Dept. of Oral & Maxillofacial Surgery
Govt. Dental College, Kottayam.

[3]Assisstant Professor,
Dept. of Oral & Maxillofacial Surgery
Govt. Dental College, Kottayam.

[4]Assossiate Professor,
Dept. of Oral & Maxillofacial Surgery
Govt. Dental College, Kottayam
Email: vimal1286@gmail.com

ABSTRACT

Dentigerous cysts are thought to be caused by a developmental abnormality derived from the remnants of the tooth forming organ. Most common site of dentigerous cysts are those associated with impacted third molar teeth of the mandible, but rarely involve impacted supernumerary teeth in the anterior maxilla which account for 5% of all dentigerous cysts. Dentigerous cyst associated with bilaterally impacted supernumerary teeth is still a very rare entity. Swelling or pain may be the major complaints of the patient. In this case of discussion, radiographic examination reveals bilaterally impacted supernumerary surrounded by a large corticated radiolucency. A provisional diagnosis of infected odontogenic cyst was made. The cyst was enucleated along with the removal of the bilateral supernumerary teeth. Histopathological examination confirmed the diagnosis of infected dentigerous cyst .The patient remained asymptomatic, and no complications were noted.

Key words: Dentigerous cyst, supernumerary teeth.

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INTRODUCTION:

Dentigerous cyst, also known as follicular cyst, is an odontogenic cyst caused by fluid accumulation between the reduced enamel epithelium and the enamel surface of a formed tooth[1]. It is thought to be a developmental abnormality derived from the reduced enamel epithelium of the tooth forming organ and most frequently found in individuals in the age group between 20 and 40 years. Most common site of the dentigerous cysts are those associated with the third molar teeth of the mandible, followed by maxillary third molars, maxillary canines and premolars of both maxilla and mandible. They are occasionally associated with supernumerary teeth. Stafne first described dentigerous cysts associated with supernumerary teeth and found an incidence of 5.5% among 200 supernumerary teeth . Most supernumerary teeth are noted in the anterior maxillary region.

This case report describes a rare entity of dentigerous cyst associated with bilaterally impacted supernumerary teeth.

CASE REPORT

A 22 year old male reported to our Department with missing and mal alingned upper front teeth. On clinical examination upper left lateral incisor was palatally erupted, rotated right lateral incisor, mesially angulated upper left central incisor (fig.2).There was a small bulge in upper labial vestibule. On palpation it was non tender, soft in consistency and thick chocolate colour fluid obtained on aspiration. There was no mobility of anterior teeth.

Radiographic examination with occlusal radiograph and OPG showed a unilocular radiolucent area mesial to upper central incisors extending to palatal area with size 1x1cm, and with smooth sclerotic margins (fig 3). Provisional diagnosis was dentigerous cyst since the histopathology report of the aspirated fuid had more protein content. Then we planned for enucleation and curettage under local anaesthesia.

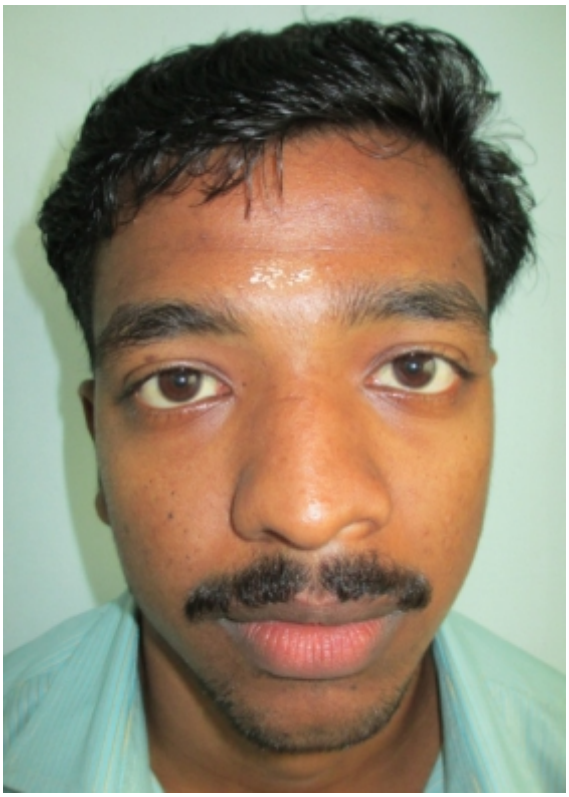

Fig.1 Pre operative exra oral photograph

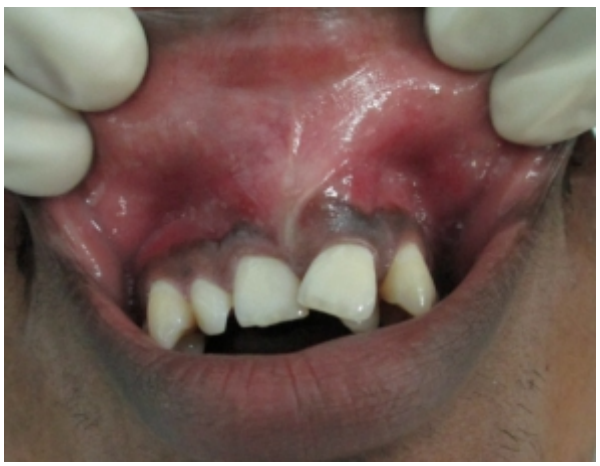

Fig 2 Pre operative intra oral photograph showing labial swelling in vestibular region

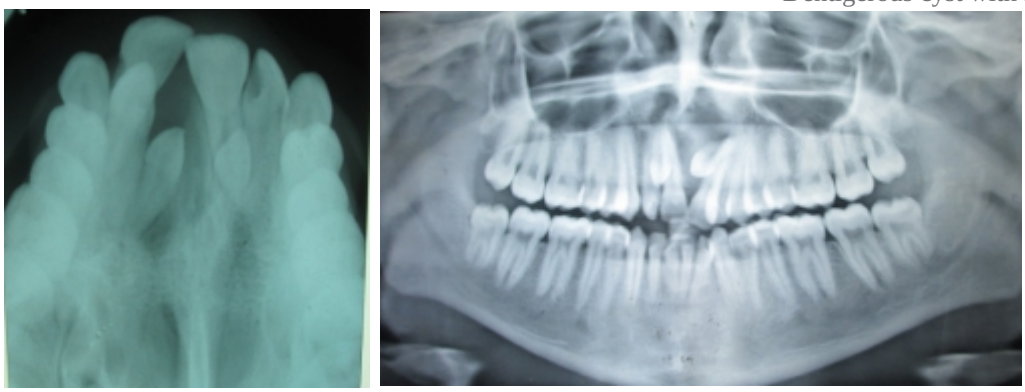

Fig 3. Maxillary occlusal and OPG showing a well corticated radiolucency in relation to upper central incisor and bilateral impacted supernumerary.

PROCEDURE

Patient positioned supine,site painted with betadine and infiltrated with local anesthetic solution. Intra oral crevicular with vertical releasing incision placed over upper left lateral incisor region extending to opposite side lateral incisor. Mucoperiosteum elevated and cyst identified.The supernumerary on left side was identified first which was on the labial side (fig.4)and the other one was found in the palatal side between the right central and lateral incisor (fig.5).Cyst was removed completely with lining and the two supernumerary teeth which was found attached with the cystic lining (fig.7).Specimen was send for histopathological examination and report came as dentigerous cyst.

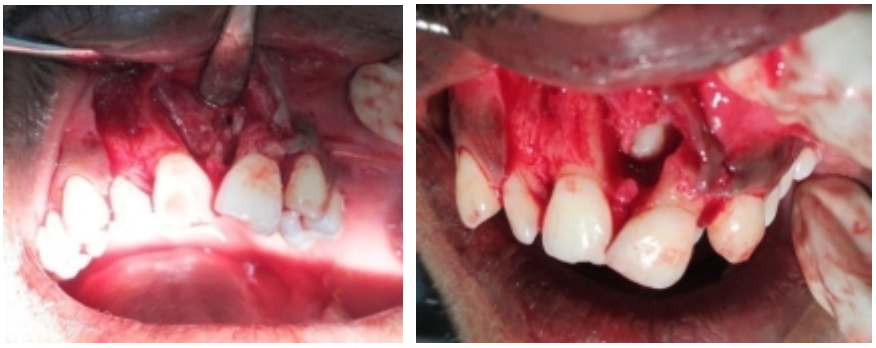

Fig 4 Cyst and one of the supernumerary on the labial side

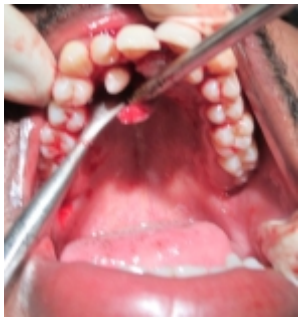

Fig 5. supernumeraryin palatal region

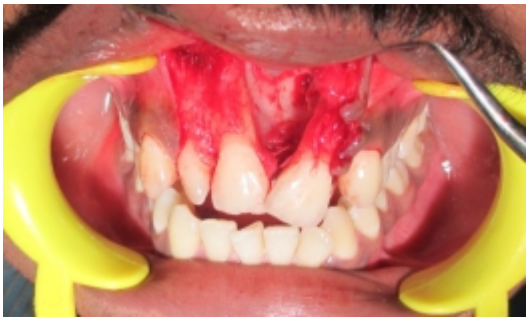

Fig 6. Cystic cavity after enucleation and curettage

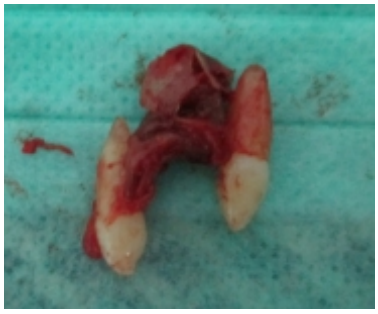

Fig 7. Enucleated cyst along with supernumerary attached to lining

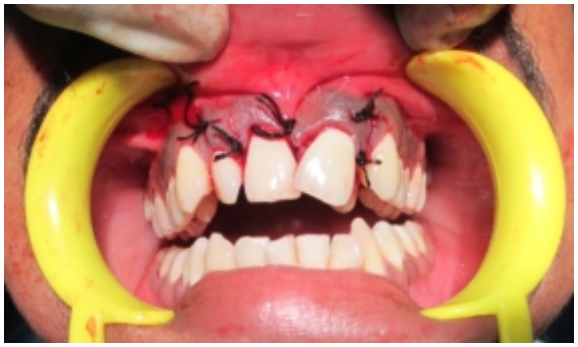

Fig 8. Closure with 3-0 silk

DISCUSSION

Dentigerous cyst is the second most common type of odontogenic cyst and is always associated with the crown of an impacted, embedded, or otherwise unerupted tooth. Dentigerous cysts are typically asymptomatic and may be large, destructive, expansile lesions of bone[1]. The highest incidence of dentigerous cysts occurs during the second and third decades. However, in our case it was diagnosed at a much earlier age. It usually occurs in the mandible and is known to be both unilocular and multilocular and causes apical resorption of the adjacent teeth. The diagnostic feature is by the presence of the unerupted tooth in its cavity, which in this case was bilateral supernumerary teeth.

Dentigerous cyst formation is another problem that may be associated with supernumerary teeth. Primosch reported an enlarged follicular sac in 30% of his cases, but the histological evidence of cyst formation was found in only 4-9% of the cases[1].According to Asaumi et al, dentigerous cyst formation arising from the supernumerary teeth comprises 11% of cases. A further study found that 6% of supernumerary teeth have dentigerous cyst development, and Hurlen etal suggested that dentigerous cysts associated with the supernumerary teeth occur in 7% of cases[8,9].

Radiographically, the dentigerous cyst typically appears as a well-circumscribed, unilocular, usually symmetric radiolucency around the crown of an impacted tooth. An important diagnostic point is that this cyst attaches at the cementoenamel junction. The internal aspect of the cyst is completely radiolucent except for the crown of the involved tooth. One of the most difficult conditions to distinguish in the differential diagnosis is hyperplastic follicle. Other conditions that must be excluded in the diagnosis are odontogenic keratocyst, ameloblastic fibroma, and cystic ameloblastoma[7].

Intra oral periapical radiograph, Water's view and panoramic view are simple and inexpensive methods that can be used in daily practice. The structure of a tooth can be clearly detected on panoramic radiographs. Therefore, panoramic radiographs are preferred over CT. Although the structure of a tooth can be clearly detected on panoramic radiographs, they are inadequate for localizing maxillary ectopic teeth due to their inherently less sharp image and ghost image. CT scan provides superior bony detail, allowing for the visualization of the size and extent of the lesion with determination of orbital or nasal invasion or involvement[1]. Therefore, CT may be more valuable than plain film radiographs, not only for definitive diagnosis, but also for evaluation of the associated pathology, exact localization of the ectopic tooth, and proper treatment planning.

A broad range of conditions may lead to a clinical presentation of painless swelling along the lingual surface of the palate or on the upper lip. Differential diagnosis of a median palatine cyst, nasopalatine duct cyst, radicular cyst, odontogenic keratocyst (OKC) or adenomatoid odontogenic tumor (AOT) were considered in our case. Median palatine cysts and nasopalatine duct cysts are not associated with non-vital teeth as they are non- odontogenic cysts of the hard palate[13, 14]. Most radicular cysts appear as round or pear-shaped, unilocular, radio lucent lesions in the periapical region, and the associated tooth usually has a deep restoration or large carious lesion radiographically[14]. Approximately 40% of OKCs contain an impacted tooth, and the lumen of the cyst often contains 'cheesy' material and has a parakeratinized epithelium lining. They are more likely to show aggressive growth than other odontogenic cysts and may have undulating borders and a multilocular appearance on radiograph[15]. Approximately 75% of cases are associated with an unerupted tooth, and the most common location is in the anterior maxilla. AOTs are more common in young people, affect females more than males and, most importantly, the radiolucency in cases of AOTs extends apically beyond the cementoenamel junction[16].

The standard treatment for a dentigerous cyst is enucleation and extraction of associated impacted or unerupted tooth. However, large lesions can be marsupialised. Histologically, dentigerous cysts are lined by a layer of nonkeratinized stratified squamous epithelium, with a surrounding wall of thin connective tissue containing odontogenic epithelial rests.

CONCLUSION

In summary, although a dentigerous cyst associated with impacted permanent teeth is not uncommon, such development as a result of an impacted supernumerary tooth might be rare. Dentigerous cysts arising from impacted supernumerary teeth in the anterior maxilla should be considered in the differential diagnosis for painless swelling along the lingual surface of the palate or on the upper lip. To prevent the development of a dentigerous cyst and to avoid unwanted effects on adjacent teeth, early detection consisting of a thorough clinical and radiographical examination is necessary for accurate diagnosis and proper treatment planning.

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TREATMENT OF A CLASS 2 FURCATION DEFECT USING XENOGRAFT AND RESORBABLE GTR MEMBRANE

Authors:
Ashitha Mohandas[1]
Angel Jacob[2]
Jayachandran P[3]
Mohammed Shareef[4]

[1] PG Student,
Dept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam.

[2]Professor,
Dept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam.

[3]Prof & HOD,
Dept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam.

[3]Reader,
Dept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam.

Address for correspondence:

Dr. Ashitha Mohandas
PG StudentDept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam,
Email : ashitham@gmail.com

ABSTRACT

Periodontal regeneration in furcation defects is one of the most challenging goals of a periodontist. Here we describe a case report of a successful surgical management of a class 2 furcation defect in a mandibular molar of a 21 year old man. This case report evaluates the efficacy of using xenograft along with resorbable membrane for guided tissue regeneration in furcation defects. The patient reported with a chief complaint of pain and swelling in his right lower back tooth. On clinical and radiographic examination a class II furcation defect(Glickman's classification) was observed. After phase 1 therapy ,the flap was reflected and furcation area was debrided. Xenograft was placed into the defect and covered with resorbable collagen membrane and sutured. At the end of 4 months a significant bone fill was seen in the furcation defect thus improving the prognosis of the tooth.

Key words: Furcation defect, xenograft, GTR.

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Authors:
Ashitha Mohandas[1]
Angel Jacob[2]
Jayachandran P[3]
Mohammed Shareef[4]

[1] PG Student,
Dept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam.

[2]Professor,
Dept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam.

[3]Prof & HOD,
Dept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam.

[3]Reader,
Dept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam.

Address for correspondence:

Dr. Ashitha Mohandas
PG StudentDept of Periodontics,
Amirtha School of Dental College,
Edappally, Ernakulam,
Email : ashitham@gmail.com

ABSTRACT

Periodontal regeneration in furcation defects is one of the most challenging goals of a periodontist. Here we describe a case report of a successful surgical management of a class 2 furcation defect in a mandibular molar of a 21 year old man. This case report evaluates the efficacy of using xenograft along with resorbable membrane for guided tissue regeneration in furcation defects. The patient reported with a chief complaint of pain and swelling in his right lower back tooth. On clinical and radiographic examination a class II furcation defect(Glickman's classification) was observed. After phase 1 therapy ,the flap was reflected and furcation area was debrided. Xenograft was placed into the defect and covered with resorbable collagen membrane and sutured. At the end of 4 months a significant bone fill was seen in the furcation defect thus improving the prognosis of the tooth.

Key words: Furcation defect, xenograft, GTR.

J Odontol Res 2016;4(2):36-40.

INTRODUCTION

Regeneration of the tissues lost due to periodontal disease, is the ultimate as well as the most challenging goal of periodontal therapy. Regeneration is defined as the type of healing which completely replicates the original architecture and function of a part. It involves the formation of a new cementum, periodontal ligament, and alveolar bone. Repair, on the other hand, is merely a replacement of loss apparatus with scar tissue which does not completely restore the architecture or the function of the part replaced. The end product of repair is the establishment of long junctional epithelium attachment at the tooth-tissue interface. Traditional therapeutic procedures used in periodontal therapy have often failed to achieve regeneration of the tissues lost due to the disease. In 1976 Melcher stated that the type of cell which repopulates the root surface after periodontal surgery determines the nature of the attachment that will form. Under physiological conditions, only cells from periodontal ligament can synthesise and secrete cementum to attach newly-synthesised collagen fibres of periodontal ligament or lamina propria of gingiva to tooth. Based on this concept there are several techniques used alone or in combination, which are considered to achieve periodontal regeneration, including bone grafts or substitutes, guided tissue regeneration, root surface modification, and biological mediators.[1] One of the most important indications for the guided tissue regeneration (GTR) treatment is the class II furcation defect. In GTR, a barrier is inserted between the root surface and the gingival tissues, to inhibit the apical migration of the epithelium and gingival connective tissue of the flap, allowing the granulation tissue derived from the periodontal ligament and osseous tissues to repopulate the space adjacent to the denuded root surface.[2] Several clinical studies have shown that GTR can improve the prognosis of class II furcation defects by means of pocket reduction, gain in clinical attachment levels, and bone defect fill. This case report describes the successful use of GTR and bone graft in a classII furcation defect which showed significant bone fill at the follow up period.

CASE REPORT

A 21-year-old male patient reported to the outpatient Department of Periodontics with a chief complaint of pain and swelling with respect to his right lower back tooth region. He did not have any relevant medical history. The patient had a history of RCT done 2 years back on the same tooth. Clinical examination revealed signs of inflammation along with a deep pocket of 9mm on the buccal aspect and class II furcation involvement (Glickman's classification, Subclass B according to Tarnow and Fletcher based on the vertical component) on the buccal aspect of mandibular right first molar (46). On radiographic examination of the region, extensive bone loss was noted with respect to the distal root of 46.A radiolucency of around 7 mm (vertical dimension) was noted. Routine hematological examinations were done before surgical intervention, which were found to be within normal limits. The treatment was divided into Phase I and Phase II therapy. Phase I therapy included scaling and root planing and general oral hygiene instructions were given. After 7 days, the surgical procedure was carried out. After adequate local anesthesia, an intrasulcular incision was made around the involved tooth, extending to the adjacent tooth on both sides for adequate access. A full thickness mucoperiosteal flap was reflected to access the underlying bone morphology in the furcation area. The furcation area was assessed . The assessed defect was 6 mm vertically. The area was properly debrided using Gracey curettes. After debridement, bovine derived bone graft, was placed in the furcation area. Following which resorbable collagen GTR membrane was placed onto the furcation area. Immediately after placing the membrane, the reflected flap was repositioned over the GTR membrane and secured with interrupted direct loop 3-0 nonresorbable silk sutures. The patient was put on Amoxicillin 500 mg and Ibuprofen +Paracetamol combination for 5 days. The patient was given proper oral hygiene and post operative instructions. The patient was advised to use chlorhexidine gluconate mouthwash twice a day to maintain plaque control. After 1 week healing was found to be satisfactory and sutures were removed. At 4 months of follow-up, there was substantial bone fill in the furcation area, representing a significant percentage of bone deposition.

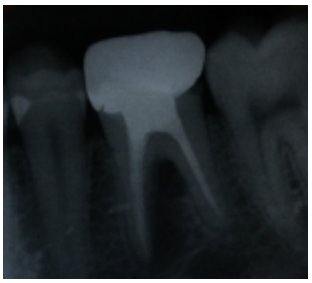

Figure 1
Pre operative radiographic view of 46

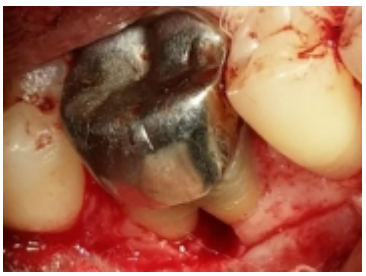

Figure 2
Flap reflected and debridement done

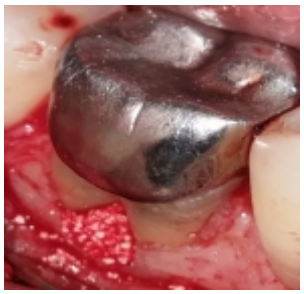

Figure 3
Bone graft placed

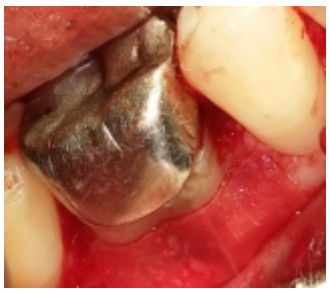

Figure 4
GTR membrane placed

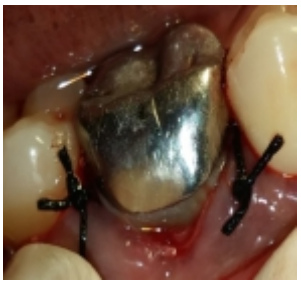

Figure 5
Sutures placed

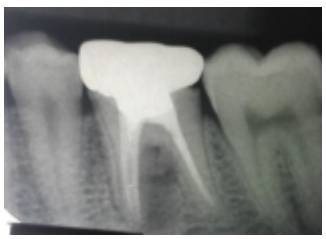

Figure 6
3 months post operative radiograph

DISCUSSION

This case report demonstrates the use of Bone graft and GTR membrane for regeneration of bone lost due to periodontal disease in a class Π furcation defect. Regeneration of lost periodontal tissues is a challenging goal which most of the traditional periodontal surgical procedures fail to achieve. Newer regenerative techniques like the use of GTR can be used for periodontal tissue regeneration in intrabony defects and furcation-involvement areas caused by periodontal disease. The rationale of GTR is to impede apical migration of the epithelium by placing a membrane between the flap and root surface (preventing contact of the connective tissue with the root surface). Cells derived from the periodontal ligament are induced on the root surface selectively and periodontal tissue regenerated.

Treatment of the first human tooth with GTR was reported by Nyman et al. in 1982.Human Case Series of Clinical Attachment Gain and with Histological evidence was presented by Gottlow and Nyman in 1986.

It must be noted that there are many factors acting collectively that influence the final outcome of GTR in class II furcations. Selecting a defect that is amenable to regeneration is also critical for achieving success.[3] Wang and Boyapati[4] have suggested four factors, the so-called PASS principles, that are critical for predictable bone regeneration: Primary wound closure, angiogenesis as a blood supply and source of undifferentiated mesenchymal cells, space maintenance, and stability of the wound. GTR has offered better results than open-flap debridement or bone replacement grafts alone, in class II furcation.[5]

Jepsen S, 2002 in a systematic review concluded that GTR was consistently more effective than open flap debridement in reducing open horizontal furcation depths, horizontal and vertical attachment levels and pocket depths for mandibular or maxillary class II furcation defects[6]. Needleman IG, et al, 2006, concluded that GTR has a greater effect on probing measures of periodontal treatment than open flap debridement, including improved attachment gain, reduced pocket depth, less increase in gingival recession and more gain in hard tissue prob-

ing at re-entry surgery than OFD.Lecovic et al had used GTR in 24 cases and reported significant bone fill and attachment gain at reentry.[7] Lecovic and Scallhorn et al had however reported that the combination of GTR and Various bone substitutes in molar classII Furcations resulted in more pronounced bone fill and attachment gain than GTR alone[8,9].

In the present case we have used a resorbable collagen membrane as GTR. Even though the non resorbable membranes made of expanded polytetraflourethylene (e-PTFE) are considered " Gold standard" for regeneration[10] several clinical studies comparing GTR therapy in class II furcation lesions with non resorbable and resorbable membrane have demonstrated similar results in the bone fill seen[11,12]. Resorbable membranes have a lesser chance of membrane exposure that non resorbable membranes[13] and the need for a second surgery is avoided. Thus making it more comfortable and economical for the patient.

We decided to use a xenograft along with GTR membrane for this case. The function of grafting material along with GTR would be to act as a scaffold to ensure clot stabilization and to provide and maintain space whenever the membrane may have the possibility to collapse, therfore reducing the space for regeneration[14]. Simonpietri C et al demonstrated in a clinical study that use of bovine derived anorganic bone with GTR improved the bone defect resolution in class 2 mandibular molars[15]. The xenograft which we used was demineralized bovine collagen particles and hence it had the additional osteoinductive property along with osteoconduction which would improve the possibility of regeneration. At the end of 4 months we were able to see a significant bone fill thus successfully improving the prognosis of the tooth which was otherwise heading for a hemisection. Radiographic evaluation does not provide as conclusive an evidence as surgical re entry. Nevertheless, the improvement in the radioopacity of the treated furcation site, and improvement in the clinical parameters like gain in clinical attachment level helps us to appreciate the regenerative potential of GTR and xenograft.

CONCLUSION

Treatment and management of furcation involvement is one of the most challenging problems faced by a periodontist because of the inadequate access to these areas thus rendering it impossible to maintain good oral hygiene in that area. GTR is one technique that can deliver periodontal ligament cells and wound stability thereby ensuring a stable clot that allows the activity of signaling molecules and growth factors ensuring periodontal regeneration even in the inaccessible regions.

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GINGIVAL ENLARGEMENT

Authors:
Swaroop Chandy[1]
Kiran Joseph[2]
Annie V Issac[3]
Rino Roopak Soman[4]

[1] Senior Lecturer,
Department of Periodontology.
St Gregorios Dental College, Chelad, India.

[2]HOD & Prof.
Department of Periodontics
St Gregorios Dental College, Chelad, India.

[3]Senior Lecturer,
Department of Periodontology
St Gregorios Dental College, Chelad, India .

[4]Reader,
Department of Periodontology
Pushpagiri Dental College, Thiruvalla.

Address for Correspondence:

Dr Swaroop Chandy.
Senior Lecturer,
Department of Periodontology.
St Gregorios Dental College. Chelad,
Email: swarooprini@gmail.com

ABSTRACT

Gingival enlargement is a common pathologic condition that can develop in response to various stimuli. It may be inflammatory, non inflammatory or a combination of both. It can be due to various factors such as poor OH, mouth breathing or food impaction. This case report discusses a case of gingival hyperplasia in an 18 year old male undergoing orthodontic treatment in relation to lower anteriors for the past 8 months The oral hygiene of the patient was found to be poor. Based on the clinical and histopathological findings, a diagnosis of chronic inflammatory gingival enlargement was made. Along with oral prophylaxis, gingivectomy using scalpel was carried out and results were found to be satisfactory. A proper diagnosis and treatment plan along with proper patient compliance is very essential for successful management of this condition.

Key words : Gingival hyperplasia, epulides, orthodontic therapy, gingivectomy.

J Odontol Res 2016;4(2):41-4.

INTRODUCTION

Gingival enlargement otherwise known as hyperplasia or increase in the size of the gingiva, is a multifactorial condition that can develop as a result of interactions between the host and the environment. It may be mostly plaque-induced or associated with certain drugs, systemic diseases or hereditary conditions like hereditary gingival fibromatosis[1]. It also occurs as a manifestation associated with several blood dyscrasias, such as leukemia, thrombocytopenia or granulomatous diseases[2].

Inflammatory enlargements usually occur as a result of either acute or chronic inflammatory changes involving the gingiva. It orginates as a slight ballooning of interdental papilla & marginal gingiva and usually progresses slowly & painlessly. They can either be localized or generalized[3]. Localized gingival enlargements have been termed epulis[4], describing pedunculated or sessile swellings of the gingiva. It is occasionally accompanied by painful ulceration and initially a life preserver shaped bulge can be seen around the gingiva of involved teeth. It may undergo spontaneous reduction in size, followed by exacerbation & continued enlargement [5]. It is caused by prolonged exposure to dental plaque including factors like poor OH, caries lesions, anatomic abnormalities like malalignment of teeth, improper restorative therapies like overhanging margins of restorations, improperly designed prosthesis & improper orthodontic treatment which can favour plaque accumulation[2]. Gingival enlargement may be influenced by certain hormonal conditions such as pregnancy & puberty and complicated by certain systemic medications such as antiepileptics & calcium channel blockers [6]. The exact mechanism for the development of gingival enlargement has not yet been completely understood, but various studies show that it might be due to increased production by fibroblasts of amorphous ground substance with a high level of glycosaminoglycans [7].

Gingival enlargement may lead to functional disturbances like altered speech, phonetics, difficulty in mastication, esthetics as well as psychological problems [2]. Therefore a proper treatment plan should be formulated in order to treat these adverse effects associated with gingival hyperplasia. Timely professional oral prophylaxis, good patient compliance including proper oral hygiene procedures are essential for effective management of gingival hyperplasia.

This case report presents a case of chronic inflammatory gingival enlargement associated with orthodontic therapy.

Case Report

An 18 yea r old male was referred to the Department of Periodontics from Department of Orthodontics, St.Gregorios Dental College, Kothamangalam, Kerala. The patient complained of swelling in lower gums in the front tooth region. The patient noticed this swelling 2 months back which was asymptomatic and there was no noticeable increase in size afterwards. The patient was under active orthodontic treatment for the past 8 months. The patient also experienced bleeding from the gums in relation to the lower anteriors while brushing his teeth.

The patient was not under any active medication and did not have any relevant medical history. Other than extraction of all first premolars for orthodontic treatment, the patient did not have any significant dental or family history.

Intraoral examination revealed orthodontic molar bands & brackets on all teeth except the mandibular right central incisor. Clinical examination revealed diffuse type of enlargement involving the marginal & interdental papilla extending from 33 to 43. The tissues were red in colour, had rolled out gingival margins & was soft in consistency. On probing, pseudopockets having a depth of 4-5mm were found in relation to 33 to 43 facially and generalized BOP was evident. Spacing of approximately 2mm was evident between 31 & 41 & no mobility was found. Calculus deposits were found in relation to lower anteriors facially and lingually. OHI was taken and was found to be poor.

At this stage, OPG was taken which revealed mild crestal bone loss. No periapical changes were seen in relation to lower anteriors. Complete blood count investigations were taken which was found to be within normal limits.

A treatment plan was formulated consisting of initial periodontal therapy followed by conventional gingivectomy procedure using scalpel. Initial periodontal therapy comprising of supragingival & subgingival scaling was done inorder to improve the oral hygiene status. Oral hygiene instructions consisting of proper brushing techniques, use of orthodontic brushes & 0.2% chlorhexidine mouthwash was recommended for the patient.

Following Phase 1 therapy, gingivectomy procedure using scalpel was carried out after a week inorder to improve the function & esthetics. All asceptic measures were taken for clinical preparation before surgical procedure in relation to lower anteriors, pockets were measured with Williams probe and bleeding points were marked on the outer gingiva on the labial aspect using the probe.

Under local infilteration of gingiva in relation to lower anterior region, a No:15 BP blade was used to give a discontinuous external bevel incision on labial aspect from distofacial line angle of 33 to distofacial line angle of 44 at approximately 45 degrees to the tooth surface. The incision was given approximately 1mm apical to the bleeding points and directed coronally to a point between the base of the pocket & crest of the bone. Excised tissue was sent for histopathological examination. The tissue tags were removed & surgical site was thoroughly irrigated with betadine-saline. The site was then covered with Coe-pack. Analgesics (Tab Combiflam tid for 3days) & Antibiotic (Cap. Amoxcyllin 500mg tid for 3 days) and Chlorhexidine gluconate mouthwash 0.2% was prescribed. Post operative instructions included not to brush over the surgical site and was adviced not to have anything hot for the next 24 hours. Patient was recalled after a week for review and healing was found to be uneventful. The patient was then referred to Department of Orthodontics for further management.

Histopathological examination

Histopathologically, an increase in number and thickening of mature collagen bundles was found in the connective tissue stroma. Edema, vascular engorgement & inflammatory cell infiltration was evident. Microscopic appearance of fibroblasts in the connective tissue stroma and increase in chronic inflammatory cell component was evident suggestive of non-specific gingival enlargement.

DISCUSSION

Clinical examination of the above individual revealed generalized plaque and calculus deposits with poor oral hygiene status. He was also not using an orthodontic brush and did not brush during night. He had bleeding and discomfort during brushing for the past 2-3 months. During his initial visit, subgingival scaling was done and in subsequent visit, a reduction in inflammation was seen eventhough the enlargement persisted. Pubertal age group of the patient may also be one of the factors which favoured gingival hyperplasia [6].

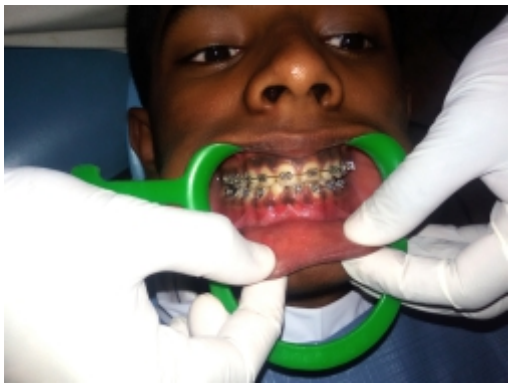
Hypertrophy or enlargement of gingiva is found to be one of the adverse periodontal effects associated with orthodontic treatment. Orthodontically induced gingival enlargement has a specific fibrous & thickened appearance of the gingiva. Various studies have shown that proximal anterior bleeding and excess resin or cements around the brackets were associated with higher levels of anterior gingival enlargement. Presence of orthodontic brackets were found to increase the level of difficulty associated with brushing which contributed to poor oral hygiene. Surface roughness of cements used is another factor which enhances bacterial adhesion and thereby favour plaque accumulation [7].

Contact with certain dental metals were also found to induce gingival hyperplasia. Eliadas et al stated that gingival enlargement associated with orthodontic appliance usually occurs as a result of inflammatory response to metals like nickel. According to Holmstrup, this type of gingival enlargement is usually manifested as nickel allergic contact dermatitis [8].

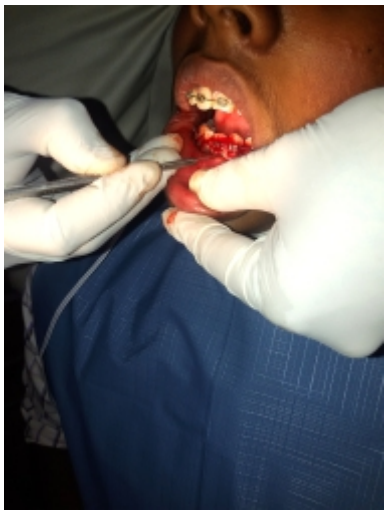
Fixed orthodontic appliances causes an increase in all bacterial counts around the bracket and band's ecosystem. A decrease in concentration of facultative microorganisms & an increase in proportion of anaerobic rods & spirochetes including prevotella melaninogenica, prevotella intermedia & actinomyces odontolyticus were found in gingival sulcus in subjects wearing orthodontic appliances [9]. A study by sallum et al showed a marked influence of orthodontic appliance removal & professional oral prophylaxis on periodontal health [2].

An increase in bulk of the tissue can lead to displacement of tooth in the arch resulting in spacing as well as tooth migration which is most commonly associated with drug induced gingival hyperplasia. A thickening of the parakeratinized epithelium can result in nodular enlargement of gingiva [3]. In case the hyperplastic gingival tissue is edematous, removal of the local factors by scaling & root planning will result in resolution of the condition. If the tissues are fibrotic, which persists even after SRP with involment of either esthetic or functional componant, it might need surgical intervention [2].

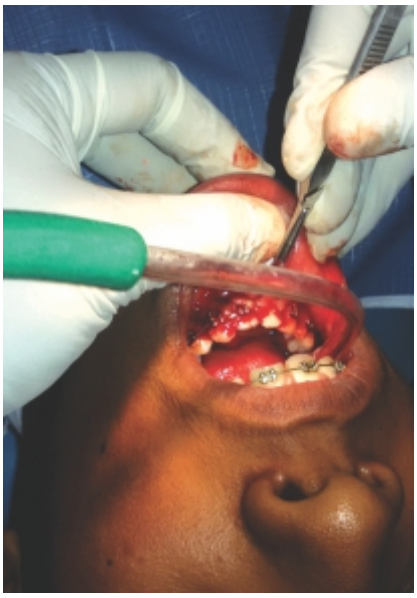
Various methods have been employed for gingivectomy including gingivectomy using scalpel



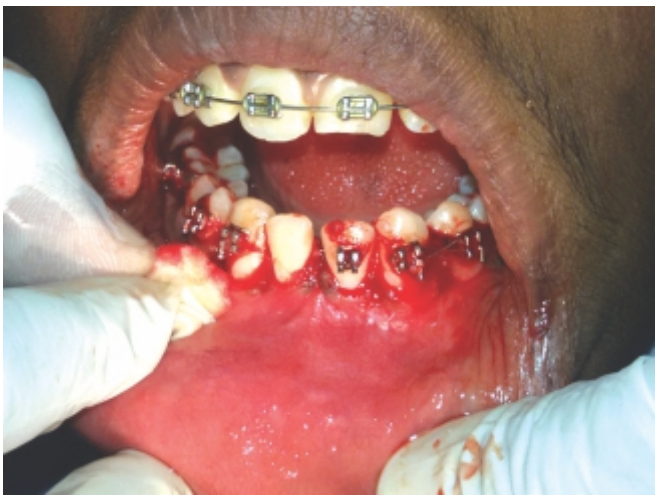
Pic-1- Inflammatory enlargement extending from 33 to 43 facially.



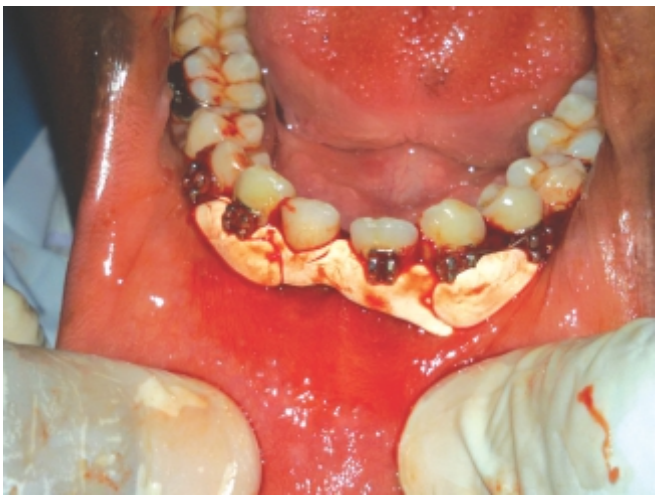
Pic -2- External bevel incision given facially extending from 33 to 43.



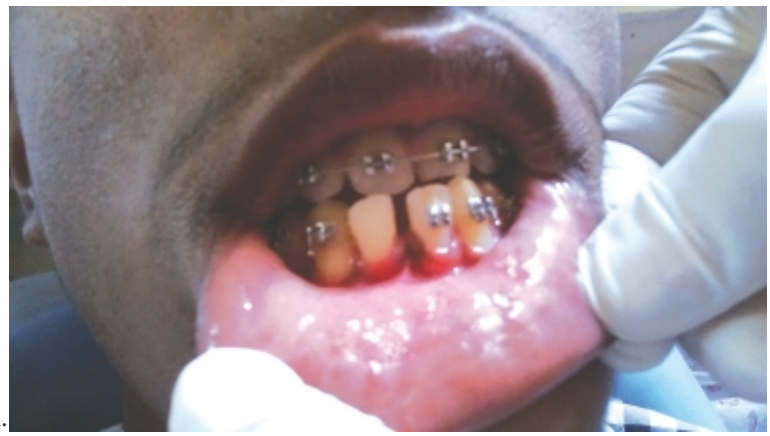
Pic- 3-Interdental incision given facially extending from 33 to 43.



Pic-4- Removal of excised tissue done



Pic-5- Periodontal pack placed.



Pic-6- Healing after 1 month.

and by means of electrosurgery, laser and chemosurgery. Studies have shown that sustained ability of diode laser gingivectomy to maintain adequate gingival health is advantageous in orthodontic patients since treatment can go in for longer periods especially in older patients[10]. Eventhough each of these techniques has it own advantages and disadvantages, factors such as conservation of keratinized tissue, minimal gingival tissue loss & minimal post surgical discomfort should be taken into consideration when opting for different techniques[9].

Recurrence of the lesion after treatment is one of the most commonly encountered complication in the management of gingival enlargement. Chronic inflammatory enlargement occurs mainly due to incomplete removal of local irritation factors mainly plaque & calculus or as a result of repeated food impaction or iatrogenic factors like overhanging margins of restorations[6]. This is usually manifested by a red bead like granulomatous tissue that will bleed on slight provocation[9]. Chronic inflammatory enlargement caused by local factors is self perpetuating since it is often impossible to properly clean the "pseudopockets" which are formed by hyperplastic tissue. This condition can be corrected by removing granulation tissue along with scaling & root planning & by instituting proper oral hygiene methods.

CONCLUSION

In the above case report, anterior gingival enlargement has been found to be associated with plaque induced gingival inflammation and lack of proper oral hygiene methods. Patient motivation & patient compliance are important aspects of treatment planning during management of chronic inflammatory enlargements. Therefore successful management of this condition requires combined effort of both patient and dentist. A proper follow up should be carried out to detect any early recurrence as well as to maintain the gingival contour in its normal functional & physiologic state. Further studies are needed inorder to obtain more data and information regarding the etiology and management of chronic inflammatory gingival enlargement.

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