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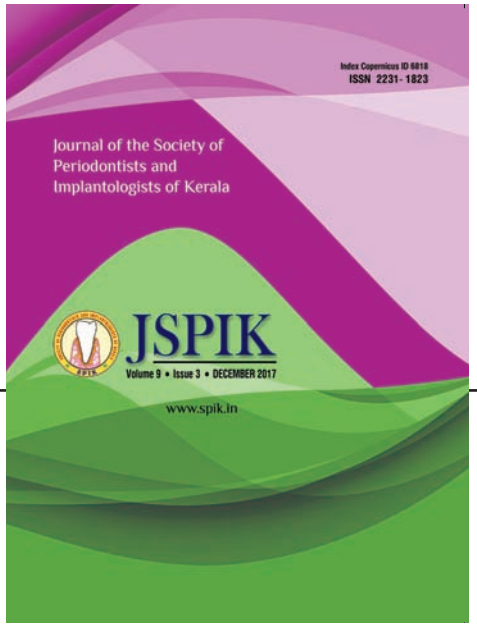

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President's message

Seasons Greetings.... and a very happy & prosperous New Year to all... have wonderful, cheerful and exhilarating experiences in 2018 throughout...

....after the successful CDE at Cochin, SPIK office were on the lookout for the platforms for its performance...

..Team SPIK observe and appreciate the amazing zeal and energy of the members in the activities related to our speciality, though many not on the SPIK platforms. SPIK is proud of its members, for being the forefronter, which paved to be a nidus, behind them. SPIK also congratulate the SPIKans who are being adjudged and elevated to the higher posts of ISP and other fraternity Associations.. Owing to such activities, the Team SPIK struggle really hard to adhere to its calender schedules and routines

....and,..now.. our Mid Term Conference is on the air... Dr. Mahesh and team with the guidance of SPIK head office is all set to welcome you all to Thrissur, for the most important program of SPIK for the year... Let us come together and unfold the latest in the Periodontics and Implantolgy....

...come.....share...update...and practise....

...together let's grow...together let's flourish...

Yours in SPIK,

Dr Anto Joseph Puthanangady
President SPIK

Editorial

Warm Greetings to All of You.

Welcome to the 2nd issue of JSPIK (December). As now the curtains are down for the 42nd ISP National Conference at Kolkata. I hope this JSPIK issue will reach your hands before New Year 2018. SPIK Mid Term Conference in Jan at Thrissur with Scientific lectures and Clinical workshop will be very helpful for the postgraduates. SPIK helps to update our knowledge through these scientific sessions. We are also waiting to attend PG convention in Mar 16-18 at Chennai. JSPIK gives good opportunity to the post graduates as well as specialists in periodontics to write down and publish on their good clinical cases and original research articles. Please don't hesitate to contact me with your suggestions on JSPIK.

Happy New Year

Dr Plato Palathingal
Editor
editorspik@gmail.com

Secretary's Message

Dear Members,

Greetings from JSPIK.

This issue comes to you as we gear up for this year's edition of the SPIK Mid term conference, the exclusive two days scientific extravaganza for the young minds of Periodontology in the state- the post graduate students. SPIK has been in the forefront of providing suitable platforms for aspiring young professionals in periodontology to showcase their clinical and academic skills right from its inception more than a decade ago.. This year two new sections have been planned as part of the conference namely the *table clinic* and a mentor guided *clinical workshop on bone grafting*. We all are excited about these two new events and are eagerly waiting to see how it turns out in reality. The organizing team under Dr Mahesh Narayanan, an experienced campaigner, has left no stone unturned in making it resourceful and enjoyable. The JSPIK has been on time this year thanks to the brilliant efforts of the young and talented editor Dr Plato Palathingal whose has been spot on right from day one of his assuming the office. He has been working hard to speed up the review process and enjoys a good rapport with the contributors as well as the reviewers in making the entire process of publishing as trouble free as possible. As we move towards the fag end of the association year I would like to thank our committed President Dr Anto Joseph for his support and understanding which has made my life easy as secretary. Once again I wish all success for the Mid term conference and sincerely hope it would be beneficial to the participants and the presenters alike.

Perio is thrilling

Dr. Baiju R.M.
Secretary, SPIK

JSPIK

Recurrent pyogenic granuloma by diode laser – a case report

G. Kokila[1], Renuka Devi[2], Esther Nalini[3]

ABSTRACT

Pyogenic granuloma is one of the inflammatory hyperplasia seen in the oral cavity. This arises in response to various stimuli such as local irritation, traumatic injury or hormonal factors. It most commonly occurs in females than males possibly due to vascular effect of sex hormones. Clinically pyogenic granuloma appears as smooth or lobulated exophytic lesion manifesting as small red erythematous papules on pedunculated or sometimes sessile base. Its colour may varies from pink to red to purple, depending on age of the lesion. Excisional surgery is the only treatment mode. There are varies advanced protocols such as the use of laser, cryosurgery, intralesional injection of ethanol or corticosteroid and sodium tetradecyl sulfate sclerotherapy which have been proposed. In this report, we seek to highlight the therapeutic success achieved with diode laser in the treatment of pyogenic granuloma.

Key words: Diode laser, Pyogenic granuloma, Pregnancy tumour, Recurrent.

Introduction:

Pyogenic granuloma is one of the inflammatory gingival hyperplasia seen in the oral cavity. This term is a misnomer as the lesion neither produces pus nor it is granulomatous.[1] Pyogenic granuloma is also known as "Pregnancy tumour," "Granuloma gravidarum," "Vascular epulis," and "Hemangiomatous granuloma."[2]

Hullihens, in 1884, first reported pyogenic granuloma in English literature.[3] Pyogenic granuloma is a vascularised mass originally described in 1897 by Poncet and Dor, who named this lesion as human botryomycosis.[4] The term pyogenic granuloma was introduced by Harzell in 1904.[5] The pyogenic granuloma arises in response to various stimuli like local irritation, traumatic injury, hormonal imbalance, arterio-venous malformations, drugs, production of angiogenic growth factors and cytogenetic abnormalities.[6]

In 75% of cases, pyogenic granuloma appears on the gingiva. More often it is seen in maxilla than mandible. Extragingivally it can occur on the lips, tongue, buccal mucosa and palate.[1] Females are most commonly affected than males, probably due to vascular effects of female hormones.[7]

Clinically Pyogenic granuloma is a smooth or lobulated exophytic lesion which manifests as small, red erythematous papules on pedunculated or sometimes sessile base. The colour may varies from red/pink to purple. Generally lesions are asymptomatic and tend to bleed on minor trauma.[8]

An excisional therapy is the one of the choice for treatment of pyogenic granuloma. But some alternative approaches are also available such as laser, electrocautery, cryosurgery, intralesional injection of ethanol or cortiocosteriods and sodium tetradecyl sulfate sclerotherapy.[1] In this report we try to highlight the therapeutic success achieved with diode laser in the treatment of pyogenic granuloma.

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Case report:

A 50 year old female patient reported to Department of Periodontics with a chief complaint of growth in relation to upper left back region of jaw for past 6 months. The growth which started as small sessile painless mass, progressively increased in size. It was associated with profuse bleeding on provocation and there was hindrance in mastication.

Patient complained that a similar growth was noticed in the same region 2 years before and she had reported to a private dental clinic and surgical excision had been carried out. After surgical excision, patient had noticed the recurrence of the growth in the same region for 6 times and each time surgical excision had been done without any histopathological investigation. Following each surgical excision and curettage, the lesion reoccurred within 2 weeks, returning to the original size. Because of the recurrence of growth in the same region for 6 times, the patient had been advised to go for an extraction of tooth in that region. Even after the extraction of tooth, the lesion again recurred within 2 weeks and so the patient reported to the Department of Periodontics for further management. Patient had no relevant medical history and brushed her teeth once a day using a tooth brush and tooth paste using horizontal stroke and consumed a mixed diet.

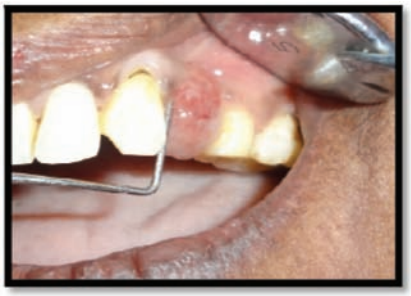

Fig 1: Clinical view of lesion.

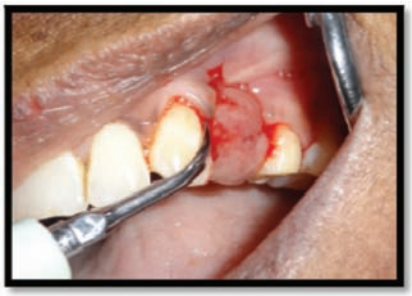

Fig 2: Oral prophylaxis.

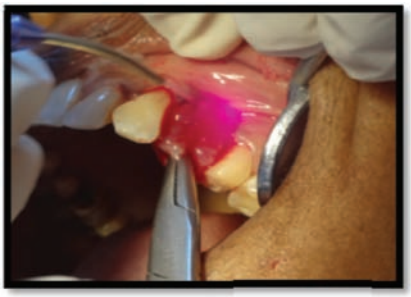

Fig 3: Growth excisied by laser.

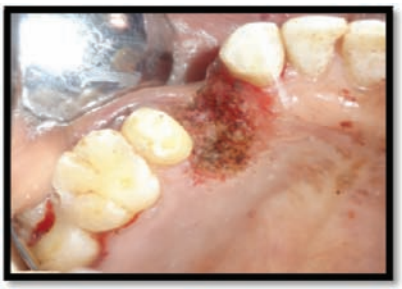

Fig 4: Immediate post operative.

Fig 5: Excised tissue.

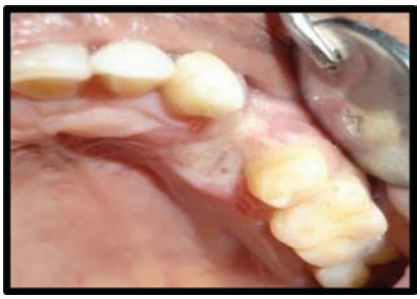

Fig 6: Post operative after 1 week.

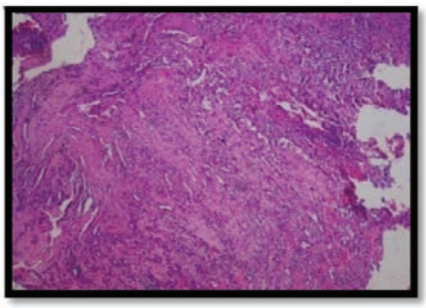

Fig 7: Histological appearance of the lesion.

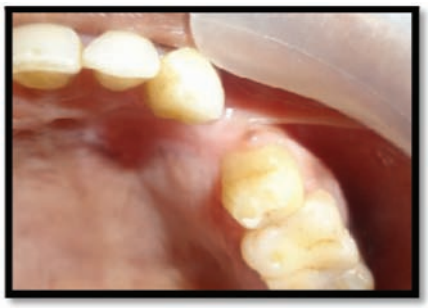

Fig 8: Post operative after 6 months.

On intraoral examination, a growth was seen in the gingiva in the relation to maxillary left first premolar region (buccual and palatal aspect of gingiva). It revealed an oval shaped pinkish, exophytic growth with a smooth surface. The growth was measured about 2x11/2cm in size. On palpation, the growth was soft to firm in consistency, sessile, non tender and bleed frequently.

The oral hygiene status of the patient was fair. Routine blood investigation (Haemoglobin, RBC, WBC, DC, BT, CT, and Random Blood Sugar) was carried out and that were within normal limits. Based on the clinical findings, a provisional diagnosis of inflammatory gingival hyperplasia or peripheral giant cell granuloma or pyogenic granuloma was considered.

The differential diagnosis included peripheral ossifying fibroma, koposi's sarcoma, maliganant tumours, hemangioma and granulation tissue.

Phase I therapy (SRP) was completed; patient was recalled after 3 days. The lesion was excised by soft tissue diode laser without local anaesthesia. We used it at a wavelength of 810nm and 7 W powers in continuous mode and the tip was kept at a distance of 1mm from the soft tissue throughout the procedure. The process took 4-5 min to completely excise the mass. The lesion totally excised along with 2-3 mm of normal surrounding tissue. The diode laser provided an optimum combination of clean cutting of the tissue and haemostasis. Patient was discharged with all necessary post operative instructions. She was not prescribed any antibiotics, analgesic or anti-inflammatory medication. She was recalled after 5 days for follow-up. The healing was uneventful.

Wound healing was observed to be highly advanced 5 days after the intervention. The patient was reviewed after 15 days, 3 months and 6 months. There has been no recurrence for 1 year after the intervention.

The excised tissue was sent for histopathological examination which showed stratified squamous parakeratinized epithelium with underlying fibro vascular stroma with numerous proliferating capillary buds which correlated with the clinical diagnosis. A final diagnosis of pyogenic granuloma was rendered.

Discussion:

Pyogenic granuloma is a common tumor like growth of oral cavity or skin and is considered to be non-neoplastic in nature.[1] Pyogenic granuloma occurs at any age, but it commonly affects young adults. Females are more susceptible than males because of hormonal changes that occur in women during puberty, pregnancy and menopause.[7] The oral sites of pyogenic granuloma include gingiva, lip, tongue, buccal mucosa and palate. The lesions are slightly more common on maxillary gingiva than mandible.[1] The size varies in diameter from few millimetres to several centimetres, rarely exceeding 2.5cm. Lesions may grow rapidly and reach its maximum size and remain static. It is rarely associated with bone loss.[7]

Pyogenic granuloma is similar to benign neoplasm which is usually considered to be a reactive tumour like lesion that arises in response to various stimuli like as chronic low grade irritation, traumatic injury, infection, Angiopoitin-1, 2 and ephrin B2 agents,[9] human herpus virus 8, hormonal imbalance, arteriovenous malformation along with inclusion bodies and gene depression in fibroblast has a role in recurrent pyogenic granuloma.[10]

Differential diagnosis of pyogenic granuloma includes peripheral ossifying fibroma, peripheral giant cell granuloma, koposi's sarcoma, hemangioma, pregnancy tumor, maliganant tumor, inflammatory gingival hyperplasia, angiosarcoma, non-hodgkins lymphoma and granulation tissue.[1]

Peripheral giant cell granuloma is clinically similar to pyogenic granuloma, although the PGCG is often more bluish-purple as compared with the bright red colour of a typical pyogenic granuloma. Histologically, PGCG is composed of nodules of multinucleated giant cells in a background of mononuclear stromal cells, extravasated red blood cells and deposits of haemosiderin.[11] Peripheral ossifying fibroma appears as pale pink in colour with lobulated surface. On palpation it was firm to hard in consistency. In comparison with pyogenic granuloma, hemangioma has more plump, histiocytiod, endothelial cell proliferation without an acute inflammatory cell infiltrate. Diagnosis of pregnancy tumour is valid clinically in describing a pyogenic granuloma occurring in pregnancy with no clinical and histological differences. Kaposi's sarcoma of AIDS shows proliferation of dysplastic spindle

cells, vascular clefts, extravasated erythrocytes and intracellular hyaline globules, which are none of feature of pyogenic granuloma.[1]

Many treatment techniques have been described for pyogenic granuloma which is a benign lesion; therefore surgical excision is the treatment of choice. There is a recurrence rate of 16% after surgical excision.[12] Recurrence is believed to result from incomplete excision, failure to remove etiologic factors or re-injury of the area. To avoid the possibility of recurrence, the lesion must be excised down to the underlying periosteum and predisposing irritants should be removed.[13] Other conventional surgical modalities for the treatment of pyogenic granuloma such as the use of lasers (Nd: YAG and CO_2), cryosurgery, intralesional injection of ethanol or corticosteroids and sodium tetradecyl sulfate have been effective.[1,10]

The Nd: YAG and CO_2 lasers are successful in treatment of pyogenic granuloma, because of low risk of bleeding and superior coagulation.[14] White et al. proposed that laser excision with no side effects is well tolerated by patients.[15] However there is minimal convincing proof of its efficacy in intraoral pyogenic granuloma. By the way, we achieved complete resolution of this lesion located in upper gingiva with diode laser without producing any complication. There was no scarring or recurrence. Hence diode laser can be a good therapeutic option for pyogenic granuloma.

After surgical removal, occasionally the lesion recurs and re-excision is necessary like in the present case. In rare instances, multiple recurrences are noted.

Conclusion:

Though pyogenic granuloma is a non neoplastic growth in the oral cavity, proper diagnosis, prevention, management and treatment of lesion are very important. The use of diode laser offers a new tool that can enhance the level of treatment of the existing treatments. Modern medicine needs to explore and take advantage of current trends to derive maximum benefits.

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Nano warriors in Periodontics- Probiotics (A Review)

Sugumari Elavarasu[1], Thangakumaran Suthanthiran[2], Arthiie Thangavelu[3], Shiva Shangkharii Kanagaraj[4]

ABSTRACT

As addressed as a delicate microscopic specialty, Periodontics has entered the saga of metamorphosis that explores and understands human body mechanisms at biomolecular levels. There has been a major shifts in treatment paradigm from nonspecific to specific approach. Periodontal disease is a host modulated, multifactorial, infectious disease resulting in inflammation within supporting tissues of the teeth, progressive attachment loss which eventually leads to bone loss and hence the loss of the tooth. The primary etiological factors responsible for causation of periodontal disease are bacteria in gingival biofilm, and hence effort for disease prevention and treatment are mainly focused on pathogen reduction. Due to the emergence of antibiotic resistance and frequent recolonization of treated sites with pathogenic bacteria, there was a need for new treatment paradigm to be introduced and the need was fulfilled by probiotics and bacterial replacement therapy. Probiotics are living organisms administrated in adequate amount with beneficial health effects on the host. In the arena of periodontics, the probiotic pose a great potential of plaque modification, halitosis management, altering anaerobic bacteria colonization, improvement of pocket depth and clinical attachment gain.

Introduction

Periodontal disease is a host modulated, locally centered, multifactorial, infectious disease resulting in inflammation within supporting tissues of the teeth, progressive attachment loss which eventually leads to loss of the tooth. Dental plaque, a biofilm consisting of microorganisms is considered as a primary etiological factor and the progression and build- up of dental plaque can give rise to various inflammatory process.[1] In spite of the best efforts by dental professionals to disinfect the oral cavity, it is inevitably repopulated with the pathogenic bacteria eventually resulting in destruction of soft and hard tissues. As disease progresses there is a shift in the microflora from gram positive to gram negative and there is increase in heterogeneity of the microbial species.

The main pathogens associated with periodontitis are Porphyromonas gingivalis, Treponema denticola, Tannerela forsythia and Aggregatibacter actinomycetemcomitans.[2] These pathogens have a wide variety of virulent factors with which they invade, colonize the subgingival sites, escapes host defense and cause damage to the host tissues.[3] Scaling and root planing accompanied by oral hygiene procedures have served as a gold standard of periodontal therapy.[4] In recent years, there have been drastic changes with regard to the effectiveness of conventional antibiotic therapy.

With the development of antibiotic resistance, many antibiotics have proved to be useless against infectious diseases. In order to overcome the emergence of antibiotic resistance and frequent recolonization of treated sites with pathogenic bacteria, there was a need for a new paradigm for treating periodontal diseases.[5]

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The need was fulfilled by introduction of probiotics and bacterial replacement therapy. Bacteriotherapy or the use of harmless bacteria to displace pathogenic organisms is a promising way of eradicating infections.

History

Traditional Egyptian fermented milk products Laban rayeb and Laban khad were consumed as early as 7000 BC. The Vedic hymns of India, written before 2000 BC indicated that Hindu people used fermented milk products in their diet since prehistoric times. The Bible dated to the 13th century BC reports that "Abraham offered to God, showed in an oak wood, fermented milk". It was in late 19th century, great French chemist Louis Pasteur who concluded that lactic acid fermentation was initiated by microorganisms and he defined fermentation as "Respiration without air".[6]

In early 20 th century, Ilya Ilyich Metchnikoff, a Russian scientist linked the health and longevity of Bulgarian peasants with their heavy intake of yoghurt which contained Lactobacillus species. Later in 1907, Metchnikoff wrote his famous text "The prolongation of Life", which first described the potential to improve human health through eating substances. The term probiotic is derived from Latin (pro) and Greek (bios) which means "for life". In 1962 Lily and Stillwell expanded the definition of probiotics to include "the anaerobic bacteria that are able to produce lactic acid and stimulate growth of other organisms".[7]

Probiotics

The term prebiotic was introduced by Gibson and Roberfroid, and it is defined as "non digestible food ingredients that are beneficially affect the host by selectively stimulating the growth and /or activity of one or a limited number of bacteria in the colon".[8] Our current definition of probiotics was formulated by FAO/WHO in 2001 as "living microorganisms which when administered in adequate amount, confers health benefit to the host".[9] With the advent in research, it is found that the most commonly used strains of probiotics belong to genera Lactobacillus and Bifidobacterium. The first probiotic species identified was Lactobacillus acidophilus in the year 1984 and Bifidobacterium bifidum was identified in 1991. Various other probiotic strains were identified such as Lactobacillus reuteri, Lactobacillus brevis, Bifidobacterium infantis, Streptococcus faecalis, Streptococcus thermophiles, Streptococcus sanguis and hence they benefit in pathogen interference, immunomodulation, immunostimulation, anticariogenic, antimutagenic, decreased incidence and duration of diarrhea, reduction in blood pressure.[10]

Different forms of probiotics

Most probiotics are bacterial species consisting of single bacterial strain or it may be a consortium. The main advantage of multiple strains is that they are active against a wide range of conditions and in wide range of animal species. Probiotic products are administered as culture concentrate, inoculated into prebiotic fibers, and milk based foods and also as concentrated dried cells. The different vehicles for delivery of Probiotics are lozenges, capsules, tablets, Mouth rinses, chewing gums, tooth paste and syrup.[11]

Ideal properties of probiotics

The probiotic strain must possess the following characteristics[12]

1. High cell viability
2. Ability to adhere and persist in gut
3. Interaction with immune cells
4. Should be of human origin and non-pathogenic
5. Resistant to processing
6. Capacity to influence local metabolic activity
7. Clinically validated and documented health effects.

Probiotics and periodontics

Prevention of plaque formation

Since microbial plaque initiates periodontal disease, probiotics have proved to inhibit plaque formation by lowering the salivary PH. It also prevents plaque formation by the production of antioxidants which in turn prevents plaque formation by neutralizing the free electrons which are needed for the mineralization of plaque. Probiotics competes with pathogenic organisms for attachment and it forms a biofilm inhibiting the adhesion of pathogenic organisms.[13]

Farzeen T and Umair A (2013)[14] stated that the probiotic strains such as S. sanguis and S.uberis

inhibited the growth of periodontopathogens and basis for their inhibition lay in the production of hydrogen peroxide. The strong negative association between A. actinomycetemcomitans and S. sanguis, laid a strong foundation for S.sanguis as an effector strain for inhibition of periopathogens.

In a study by Amer et al., (2012)[15] illustrated that probiotics incorporated mouth rinses showed a proven advantage over the normal antimicrobial mouth rinses. Probiotic mouth rinses were found to be effective in reducing plaque accumulation and gingival inflammation by utilizing natural beneficial bacteria to promote healthy balance of microorganisms in oral cavity.

Gingivitis

In a study by Vivekananda et al., (2010)[16] L.reuteri Prodentis lozenges was found to reduce the plaque and gingival bleeding and the periopathogens such as Porphyromonas gingivalis, Aggregibacter actinomycetemcomitans and Prevotella intermedia. Three plausible possibilities that are stated for L.reuteri in preventing the disease progression. First, secretion of bacteriocins (reuterin and reutericyclin), that inhibit the growth of a wide variety of pathogens; second, adherence of L. reuteri to host tissues, thereby competing with pathogenic bacteria; and third, the recognized anti-inflammatory effects of L. reuteri, leading to inhibition of secretion of pro-inflammatory cytokines, that worsens the inflammatory conditions.

Periodontitis

Lactobacillus reuteri and Lactobacillus brevis have positive effect in inhibiting plaque formation. Lactobacillus brevis has a capacity to prevent the production of nitric oxide and release of Prostaglandin E2. The probiotic Lactobacillus helveticus demonstrated release of short peptide which stimulates osteoblast to promote bone formation, thus proposing important role in repair of periodontal bone destruction.[17]

The results of the clinical study by Gizem et al., (2015)[18] demonstrated the beneficial effect of Lactobacillus reutri in moderately deep pockets and also found that MMP 8 levels were decreased and hence a significant improvement in clinical parameters such as probing pocket depth and CAL were noticed in subjects administered with probiotics.

In a study by Litty et al., (2015)[19] the efficacy of probiotic strain Streptococcus salivarius M18 against the periodontal diseases was analyzed and the results suggested that the test group had significant reduction in clinical parameters such as plaque index, Gingival index, Modified sulcular bleeding index. It is also proposed that probiotic bacteria act through Pattern Recognition Molecules like TLR-2 and TLR-4, possibly on epithelial cells and they induce the production of protective cytokines that enhances epithelial cell regeneration and hence inhibits epithelial cell apoptosis. They have distinct immunomodulatory effects on epithelial cells, dendritic cells, monocytes, lymphocytes and Natural Killer cells.[17]

Teughels et al., (2013)[20] in his study examined the clinical and microbiological effect of Lactobacillus reutri probiotic in chronic periodontitis patients and found that there was a marked reduction in probing pocket depth and clinical attachment loss and also concluded that Porphromonas gingivalis count was much reduced in the subgingival samples.

Halitosis

Halitosis (malodor) is a common problem with multiple local and systemic etiological factors including periodontitis, poor oral hygiene, tongue coating. It has recently been proved that Lactobacillus and Weissella cibaria have shown a definite inhibitory effect on the production of volatile sulfur compounds (VSC) by F. nucleatum and was also able to reduce the count of the oral black pigmented bacteroides, the bacteria that is strongly associated with production of the volatile Sulphur compounds.[21]

Conclusion

Probiotics play an essential role in combating issues with overuse of antibiotics and antimicrobial resistance. Today's newer technologies have changed the concept of bacteria s, not only as pathogens but also as beneficial organisms. The oral cavity with a well maintained balance serves as a potential source for health-promoting probiotic bacteria and daily intake of probiotic supplements have potential to control common oral and dental infections. There are lot of clinical evidence proving the beneficial effect of probiotics.[22] Further studies have to be conducted to

understand the ability of probiotic bacteria to survive, grow, and have a therapeutic effect. Hence, systematic studies and various randomized controlled trials are needed to find out the best possible probiotic strains and means of their administration in different oral health conditions. Finally, possibilities to genetically modify or engineer potential probiotic strains may offer all new visions. Better scientific understanding and extended research of these tiny forms of life and their effect on humans in the treatment of periodontal diseases might further broaden the field of potential applications.

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Decision making in oral reconstructive and corrective considerations in periodontal therapy

Tintu Madona Joy[1], Seema. G[2]

ABSTRACT

Objective- The purpose of this practical application is to illustrate the management of Gingival Recession defects with a primary goal of complete root coverage.

Treatment of gingival recession has become an important therapeutic issue due to the increasing number of cosmetic requests from patients. The dual goals of mucogingival treatment include complete root coverage, up to the cemento-enamel junction, and blending of tissue color between the treated area and non-treated adjacent tissues. Even though the connective tissue graft is commonly considered the "gold standard" for treatment of recession defects, it may not always be the best surgical option for every case. Careful analysis of patient- and defect-related factors,are key considerations prior to selecting an appropriate surgical technique.

Key words: Decision making,root coverage procedures

Introduction

Periodontal reconstructive surgery consists of a variety of mucogingival procedures including root coverage, tooth exposure, crown exposure, vestibular deepening, papilla reconstruction, ridge augmentation, and ridge preservation. While the primary goal of these procedures is to benefit periodontal health through the reconstruction of lost hard and soft tissues or by preventing additional loss, they also enhance the patient's appearance[1].

According to the evidence, patients with <15% of sites presenting with plaque and residual infection, non-smokers, with a high degree of compliance, and systemically healthy are the best candidates for root coverage procedures.[2] The aim of this article is to describe various root coverage procedures.

The ultimate goal of root coverage procedures should be complete coverage of the recession defect with a pleasing color and tissue blend between the treated area and adjacent tissues, thereby achieving both biologic and esthetic success and thus minimize gingival recession associated complications.

Gingival Recession (GR) is defined as the migration of the marginal soft tissue apical to the cemento-enamel junction (CEJ), is accompanied by alveolar bone dehiscence and a potential reduction in the gingival tissue surrounding the tooth. GR is encountered commonly in adults aged >30 years.[3]

The exposure of the tooth root and the loss of hard and soft tissue supporting structures ultimately increases the likelihood that the patient will experience: 1) dentinal hypersensitivity; 2) soft tissue discomfort; 3) root surface caries; 4) esthetic concerns; 5) interference with the performance of adequate mechanical plaque control; and 6) greater susceptibility to inflammatory insult

Clinicians can rely only on evidence obtained from earlier case reports and systematic reviews

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regarding evidence to make decisions in manging gingival recessions.

Factors Affecting Complete Root Coverage (CRC)

1. Miller Class

Surgical therapeutic approaches are highly predictable for Miller ClassI and II single-tooth defects. Challenges for the clinician arise when patients present with Miller Class III and IV defects, as well as multiple-tooth GR defects and lingual/palatal mucogingival concerns.[3]

2. Post-Surgical Position of Gingival Margin (GM)

The more coronal the GM after suturing, the greater the probability of achieving CRC.[4]

3. Flap Tension

Pini Prato et al showed that the greater the flap tension (suggested flap tension should not exceed 4 g), the less successful the recession improvement. Periosteal incisions including careful dissection of the muscle insertions from the flap 2 should be used to eliminate tension from the flap.

4. Flap Thickness

Survival of the flap, depends on the residual vascularity after surgical incisions.The thicker the flap (full thickness), the greater the vascularization of the marginal gingiva and the probability of CRC (suggested flap thickness >0.8 mm5). Must avoid interrupting the supra periosteal vessels that enhance the survival of the flap on the avascular root surface.

4. Interdental Papilla Height

According to Saletta etal CRC is more likely to be achieved in sites with a lower height of inter dental papilla. Thicker gingiva of the flat-thick biotype allows a thicker flap, which may result in a greater success rate of CRC.

5. Cemento-Enamel Junction

CEJ is the most widely used reference parameter to evaluate root coverage results. Conditions such as 1) cervical abrasion, 2) traumatic loss of the tip of the interdental papilla, 3) tooth rotation, and 4) tooth extrusion with or without occlusal abrasion may lead to diagnostic mistakes preventing CRC. Thus, in such clinical conditions, the line of root coverage may be considered the clinical CEJ.

Preparation of Exposed Root Surface

Before root coverage is attempted the exposed root should be rendered free from bacterial plaque, this is achieved by the use of rubber cup and polishing paste.

The use of root surface demineralisation and conditioning agents (tetracycline, sodium hypochlorite, or EDTA) helps removal of smear layer and also facilitate the formation of new fibrous attachment through exposure of collagen fibrils of dentine matrix which allows subsequent interdigitation of these fibrils with those in the covering connective tissue. Based on clinical experience we suggest using simple root preparation procedures such as scaling and root planning with sonic devices and curets. The need to flatten prominent roots may represent a clinical indication for the use of rotary instruments.

Extensive root planning may only be performed in situations where a reduced root prominence would be considered beneficial for the graft survival or tissue regeneration.

Restorative Approach in Mucogingival Therapy

In conditions like dental abrasion due to tooth brushing or cervical caries, lack of a definable anatomic CEJ may present clinicians with difficulties during the diagnostic phase. In cases where there is an identifiable CEJ, we suggest predetermining the line of root coverage as described by Zucchelli etal.[6] and treating the portion of the tooth coronal to the CEJ using a restorative approach prior to the surgical phase or during the surgery.

Decision making in treatment strategy

Treatment of gingival recession includes multifactorial treatment approach comprising careful selection of patients and defects, different surgical techniques, many suturing approaches, and various types of adjunctive materials.

Surgical procedures in recession defects may be classified as

- Pedicle soft tissue graft procedures

- Free soft tissue graft procedures

Pedicle graft procedures ,depending on the direction of transfer as:

➢ Rotational flap procedures(eg.lateral sliding flap, double papilla flap, oblique rotated flap)

➢ Advanced flap procedures(eg. coronally repositioned flap, semilunar coronally repositioned flap)

Free soft tissue graft may be performed as:

➢ An epithelised graft

➢ A sub epithelised connective tissue graft

Different surical techniques for root coverage

Pedicle soft tissue graft procedures

1. Laterally Advanced Flap (LAF)

Grupe and Warren[10] introduced this method for the treatment of localized gingival recession.

An alternative keratinized tissue donor site must be represented by adjacent teeth. It was originally described as a "sliding flap" that started as full thickness then became split thickness at the mucogingival junction. Periodontal biotype should be classified as thick and flat. This surgical technique is not affected by vestibule depth due to the small coronal displacement required to cover the recession defect.

Bone loss and gingival recession on the donor site are the most frequent adverse events related to this surgical procedure. Zucchelli et al[11] proposed a modified approach that appears to be more reliable and safe.

Clinical consideration

LAF should be preferably performed when the donor site is localized mesial to the gingival recession defect. As the flap is moved in the distal-mesial direction, another short horizontal incision ('cut back' incision) should be performed at the most apical extension of the distal vertical releasing incision in order to facilitate mesial mobilization of the flap. Collagen sponges, stabilized with criss-cross sutures are used to promote wound healing of the keratinized tissue donor site adjacent to the recession defect.

Laterally Advanced Flap + Connective Tissue Graft (LAF+CTG)

Similar to LAF technique, an alternative keratinized tissue donor-site must be available at adjacent teeth to perform LAF in conjunction with CTG. Periodontal biotype should be classified as thick and flat. This surgical technique is not affected by vestibule depth due to the small coronal displacement required to cover the recession defect.

2. Double Papillae Flap (DPF)

Cohen and Ross introduced the method in which bilateral interdental papilla is used as donor tissue for localized root coverage. An alternative keratinized tissue donor-site must be represented by adjacent interdental papillae. Periodontal biotype should be classified as thick and flat. This surgical technique is not affected by vestibular depth due to the small coronal displacement required to cover the recession defect.

Clinical consideration

Releasing incisions across the MGJ during the initial phase of the surgical procedure is avoided to reduce postoperative swelling and pain.

Once the interdental papillae have been dissected, join them using interrupted sutures before proceeding with the next steps of the surgical procedure; this will make flap manipulation simpler

Double Papillae Flap Connective Tissue Graft (DPF+ CTG)

As described for the DPF technique, an alternative keratinized tissue donor site must be represented by adjacent inter dental papillae to perform DPF in conjunction with CTG.

Periodontal biotype should be classified as thin and scalloped and this surgery is not affected by vestibular depth.

3. Coronally Advanced Flap (CAF)

The surgical procedure was originally described by Allen and Miller[7] in 1989.

A CAF procedure alone should be performed when a thick and flat periodontal biotype with a moderate or deep vestibule is present that will allow coronal displacement of the flap without tension. A shallow vestibule does not prevent the use of a CAF technique but requires an extensive partial-thickness dissection apically to the MGJ to make the flap tension free. A distance from GM to mucogingival junction

(MGJ) of at least 2 mm is needed to enhance the stability of the surgical flap after suturing.

CAF in esthetic areas

Suggest using an envelope flap technique, avoiding vertical releasing incisions to reduce the probability of scar tissue formation. For coronal repositioning of the flap, make a horizontal incision that extends mesiodistally to include three teeth. The horizontal incision of this modified envelope technique consists of oblique sub marginal incisions in the interdental areas, which continue the intrasulcular incision at the recession defect. The starting point of oblique incisions at a distance from the tip of the anatomic papilla equal to the recession depth plus 1/2 mm of this surgical technique

Disadvantages Need to involve healthy adjacent teeth in the procedure and smaller dimension of the flap.

Coronally Advanced Flap + Connective Tissue Graft (CAF+CTG)

A CAF procedure in conjunction with a CTG is the technique of choice when a thin and scalloped periodontal biotype is present, so that both the amount and quality of marginal soft tissue may be appropriately transformed.

A moderate or deep vestibule will allow coronal displacement of the flap without tension. When placing CTG in a shallow vestibule ,it requires an extensive partial thickness dissection apical to the MGJ to make the flap tension free. (CAF+CTG) cannot be used on a thick biotype, the placement of CTG can create an impaired esthetic due to irregular gingival profile or scar tissue.[8]

4. Semilunar Coronally Repositioned Flap.

This technique was first described by Tarnow 1986. This is a simple technique which provides sufficient satisfactory results for treating class I and class II labial and buccal recession defects especially in anterior esthetic zone. A prerequisite for using this technique is the presence of a thick-tissue biotype, with adequate tissue thickness apical to the recession defect, to prevent tooth root or alveolar bone fenestration. Vascularity to the mobilized pedicle must be maintained. This offered a 100% root coverage with minimal postoperative discomfort

Free soft tisssue graft procedures

5. Thin Free Gingival Graft Technique

This technique was first introduced by Sullivan and Atkins in 1968 and later modified by Miller in 1982. The thin free gingival graft (FGG) technique, like the LPF, was one of the earliest techniques used for root coverage. This technique is used in single tooth as well as in multiple tooth,when the is no acceptable donor present in the adjacent area of recession.

The technique worked best on shallow, narrow defects, the technique was unreliable, particularly for larger defects. Preparation of a recipient bed is crucial for the success of free graft procedure. Studies attributed most of the defect coverage achieved, by "bridging," not to immediate surgical results but instead to creeping attachment that occurred within 1 year.[13]

Thick Free Gingival Graft Technique

A thick free gingival graft was more likely to revascularize and survive on an avascular root surface than a thin graft.[14] A recession classification based on interproximal bone and soft tissue loss was devised to help identify indications for the thick FGG. A disadvantage of this technique is that the free graft may heal as scar tissue and then be difficult to elevate by blunt dissection. This complication requires sharp dissection that can lead to excessive thinning or flap perforation.

6. Connective Tissue Graft Techniques

Langer and Langer introduced the use of subepithelial CTGs for root coverage.[9]

The subepithelial connective tissue graft (CTG) is a highly predictable procedure that lacks the esthetic disadvantages of the thick free gingival graft.[15] Successful defect coverage can be achieved with less donor tissue since revascularization occurs from both the periosteal or osseous base and the overlying flap. This dual blood supply is responsible for the increased predictability of CTG procedures.

The overlying flap ensures an excellent color match when the graft is completely covered. It has recently been demonstrated that smaller, thinner connective tissue grafts work as well as larger, thicker grafts when the graft is completely covered by a coronally positioned flap.[16]

Modifications of this technique "Envelope technique was introduced by Ratezke 1985 later modified by Allen in 1994 in which the base of the connective tissue graft is within an envelope prepared by undermining partial thickness from the soft tissue margin.

For the treatment of multiple adjacent recessions, a multi envelope recipient bed ("tunnel") may be prepared and this technique was first introduced by Zabaleugi etal 1999 Connective tissue graft procedures have clearly been established as a highly effective means of covering recession defects.

7. Multiple Gingival Recessions

Zucchelli and De Sanctis[12] proposed a new surgical approach for the treatment of multiple recession defects. To minimize the number of surgeries and to optimize the esthetic result, all of the contiguous recessions should be treated simultaneously. CRC on these recessions are achieved by using the soft tissue adjacent to the defects and consists of an oblique submarginal incision in the interdental area, which continues with an intra sulcular incision at the recession defects.

Clinical consideration

1. When performing an envelope-type flap, avoid vertical releasing incisions to help maintain adequate blood flow to the flap and reduce the formation of visible white scars.

2. Use "split-full-split" flap elevation, with full thickness for that portion of the flap residing over the previously exposed root surface, to increase the potential to achieve CRC.

3. The absence of a wide zone of keratinized tissue apical to the defects is not considered a limitation; a CTG may be used at one single specific recession defect if necessary.

4. Suture the flap using a sling suture technique passing through the connective tissue of the anatomic papilla.

Guided Tissue Regeneration Technique

Numerous studies of recession defect coverage utilized the principles of guided tissue regeneration (GTR) and employed either bioabsorbable[17] or non-resorbable membranes. The membrane is sutured into place, then covered with a coronally positioned flap. Complete coverage of the membrane is preferred at the time of surgery and throughout the healing period since membrane exposure can compromise the result. An advantage of this technique is that it is theoretically possible to regenerate bone and periodontal ligament rather than just gain soft tissue coverage alone. Another advantage is that a secondary surgical site to obtain donor tissue is not needed.

Acellular Dermal Matrix Technique

The use of acellular dermal matrix (ADM) as a substitute for connective tissue when covered by a coronally positioned flap is a relatively new approach that allows coverage of multiple sites and does not require autogenous donor tissue.[18] The ability to cover an unlimited number of sites without the need for a second surgical site to obtain donor tissue is a significant advantage for this material. ADM is obtained from human dermis harvested and treated to remove all cells while preserving the intact structure of the extracellular matrix, including an intact vascular network. Studies show mean defect coverage ranging from 66% to 99% with a mean final root exposure ranged from 0.2 to 1.1 mm relative to a mean initial recession of 3.7 mm.[1]

Enamel Matrix Derivative

Enamel matrix derivative applied to a coronally positioned flap may enhance root coverage, although some studies show no advantage to its use. Studies show mean defect coverage ranging from 72% to 94% with a mean for all studies of 86%. Mean final root exposure ranged from 0.2 to 1.2 mm relative to a mean initial recession of 3.9 mm[1]

Conclusion

Due to an increasing public demand for cosmetic dentistry, the treatment of gingival recession has become an important therapeutic and esthetic issue for the contemporary periodontal practice. Root-coverage procedures can provide significant reduction in GR depth for most defects. SCTG is considered as the Gold standard which provide the best outcomes for mean root coverage as well as an increase in KT, less invasive approach, such as a CAF, may yield an equally acceptable result. Additionally, biomaterials, such as ADMG and EMD, in conjunction with CAFs may

be used as an alternative to autogenous donor tissue when necessary or desired.

Each clinical situation must be evaluated to determine the most appropriate surgical approach to achieve the esthetics expected by the patient. Therefore, to achieve the best clinical and esthetic success, a careful assessment of existing anatomic parameters, such as the amount of keratinized tissue, the periodontal biotype, and vestibule depth, is a vital part of the surgical decision-making process.

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Plasma cell gingival enlargement with associated periodontitis – A case report

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ABSTRACT

Coexisting gingival enlargement and periodontitis pose diagnostic, therapeutic and maintenance challenges. Concomitant occurrence of plasma cell gingival enlargement, a condition of allergic origin, and periodontitis is rarely reported. A 28 year old systemically healthy female reported with complaint of bleeding swollen gums with respect to the lower front teeth. Examination showed atypical erythematous enlargement of mandibular anterior labial gingiva along with generalized periodontal pockets and generalized moderate horizontal radiographic bone loss. The case was surgically managed by internal bevel gingivectomy. Histopathologic examination of the excised tissue showed a preponderant infiltration by plasma cells. A possible cause of allergy could not be elucidated from the history or habits. However, the patient is asymptomatic and the surgical results are well maintained to date.

Key words: gingival enlargement, plasma cells, periodontitis, gingivectomy

Introduction

Plasma cell gingival enlargement is an allergic enlargement of the gingiva, sometimes involving the attached gingiva. It is marked by a dense infiltration of the connective tissue by plasma cells which are separated into aggregates by strands of collagen.[1] It may be due to an allergic reaction to an antigen, generally to flavouring agents found in chewing gums, toothpastes and lozenges.[2] A coexisting periodontitis can complicate the clinical picture of the enlargement and the patient may be at an increased risk of progressive attachment loss

Case report

A 28-year-old female reported to the Department of Periodontics and Implantology, Mar Baselios Dental College, Kothamangalam with a chief complaint of bleeding swollen mass in her lower front teeth region. Patient first noticed the swelling six years ago in her upper and lower jaw, which was slowly increasing in size. She visited a periodontist for the same and underwent flap surgery in the maxillary arch. She was recalled for the surgery of the lower arch but did not follow up since she felt the problem in the lower jaw as mild. She was systemically healthy and did not report a positive drug history. The oral hygiene was good. The gingiva had a reddish pink appearance with soft consistency and bleeding on probing was present [Figure 1]. The mandibular anterior gingiva showed grade 2 enlargement extending from canine to canine with a predominant papillary component[3]. Probing depth varied from 7 mm to 9 mm in the mandibular anterior region. Radiographs showed a moderate amount of bone loss in the mandibular anterior region [Figure 2]. Blood specimen was obtained in order to rule out blood dyscrasias. A provisional diagnosis of chronic generalized periodontitis with inflammatory

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gingival enlargement was made.

Treatment and follow-up-initial periodontal therapy involving scaling and root planing was done and directions regarding oral hygiene were emphasized. Internal bevel gingivectomy was done in relation to 43, 42, 41, 31, 32, 33 under local anaesthesia and the excised tissue was sent for biopsy. Postoperative follow-up at two weeks showed firm and resilient gingiva with no enlargement [Figure 3].

Histopathologic examination

Microscopic examination revealed parakeratinized stratified squamous epithelium with underlying connective tissue stroma. The CT showed sheets of monomorphic plasma cells with eccentric nucleus in most of the areas. Areas of vascular channels and extravasated RBCs were present. Few areas showed eosinophilic secretory material [Figure 4]. Suggestive diagnosis indicated plasma cell gingivitis.

Discussion

plasma cell gingival enlargement is an infrequent condition, characterized by dispersed and immense infiltration of the plasma cells into the connective tissue. Kerr et al. in 1981 reported the first case when they detected gingival enlargement in gum chewers, which evanished following the cessation of the chewing tendency.[4] Clinically, this condition presents as a diffuse reddening, conjointly with edematous enlargement of the gingiva and a sharp delineation along the mucogingival line. It may cause severe gingival inflammation, discomfort, and has a predisposition to bleed thereby mimicking more serious conditions.

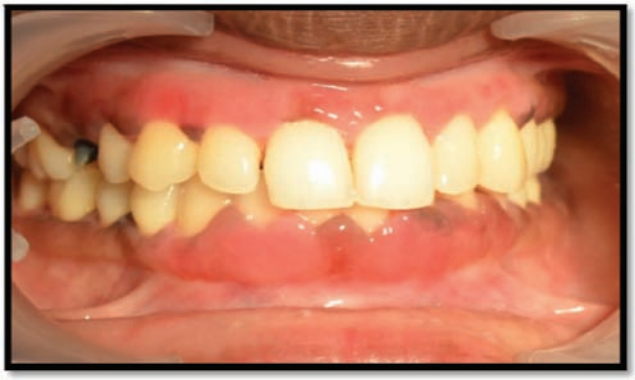

Fig 1: Atypical gingival enlargement in relation to mandibular anterior labial gingiva

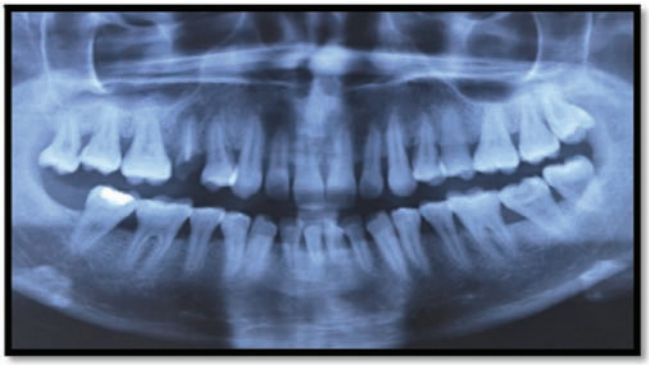

Fig 2: OPG showing generalized bone loss

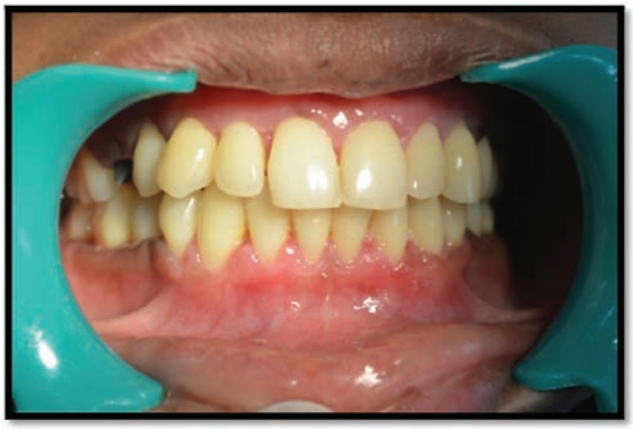

Fig 3: Normal tissue contours observed two weeks post-operatively

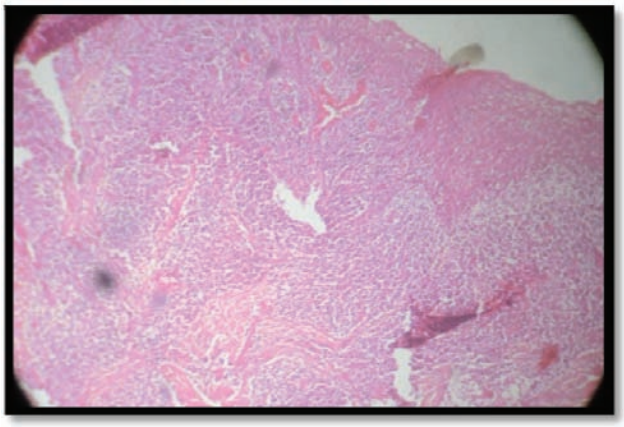

Fig.4 Histopathological examination revealing infiltration of the plasma cells

Gingival enlargements can invariably result in the creation of pseudopockets. It enables subgingival periodontopathic bacteria to colonize and proliferate, leading to the development of attachment and bone loss at some point of time. In the current case, the patient had bleeding, enlarged gingiva and a loss of periodontal attachment. It is however impossible to confirm whether the involvement of the supporting structures in this case occurred secondary to the enlargement.

Plasma cell gingivitis/enlargement is essentially a histologic diagnosis. In the present case, histopathological picture revealed infiltration of underlying connective tissue by sheets of plasma cells suggestive of plasma cell gingivitis. However, enlargement due to chronic inflammation is nonspecific and may have a mixed infiltrate of inflammatory cells.[6]

Identification of the allergen is the key to the definitive management of plasma cell gingivitis. Unfortunately, it is quite difficult to isolate the exact cause in majority of the patients. However, periodontal therapy can prevent further loss of the alveolar bone, despite the recurrence of the gingival enlargement.[5] In the present case, according to the patient's history, there was a similar enlargement in the maxillary anterior gingiva that resolved following surgical treatment.

Management of plasma cell gingival enlargement includes various medical modalities such as topical and systemic steroids that provide good results.[6] The patient was not prescribed any medications in our case since symptoms such as pain or burning sensation were not reported.

Conclusion

Dietary and environmental allergens can cause significant effects on the periodontal tissues manifesting as plasma cell gingivitis or gingival enlargement. Any case of long standing enlargement stands a risk of subsequent progress to periodontitis. It is imperative upon the periodontist to effectively manage both the conditions for ensuring long term prognosis and patient well being.

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Platelet Rich Fibrin - An enigma to explore?

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ABSTRACT

Platelet rich fibrin (PRF), an autogenous biomaterial consist of growth factors and cytokines entrapped in a fibrin matrix. It combines the fibrant sealant properties along with growth factors providing an ideal environment for wound healing and tissue regeneration. In recent times, it has been successfully used in various disciplines in dentistry for a wide range of treatment modalities. The following review attempts to summarize the relevant literature regarding the preparation and technique of using PRF, the types of PRF, the merits and demerits of using it in clinical applications including the recent advances. PRF alone or in combination with other biomaterials seems to have several advantages for its applications both for medicine and dentistry. It is also a minimally invasive technique with low risks and satisfactory clinical results.

Keywords: platelet rich fibrin, platelets, cytokines, blood, biomaterial

Platelet rich fibrin (PRF) was initially developed in France by Choukroun et al[1] for its application in oral and maxillofacial surgery. It is a WBC and platelet rich fibrin biomaterial with a specific composition and three dimensional architecture. PRF which is classified as a second generation platelet concentrate is prepared as a natural concentrate without the addition of any anticoagulants. PRF is usually called as Choukroun's PRF, as there are other platelet concentrates with similar names (Vivostat PRF, considered a pure platelet-rich plasma) or (Fibrinet PRF - without leukocytes). PRF has a dense fibrin network with leukocytes, cytokines, structural glycoproteins along with growth factors such as transforming growth factor 1, platelet-derived growth factor, vascular endothelial growth factor and glycoproteins.

Leukocytes that are concentrated in PRF scaffold play a pivotal role in release of growth factors, immune regulation, anti-infectious activities, and matrix remodeling during the wound healing period. The slow polymerization mode of PRF and cicatricial capacity creates a physiologic architecture conducive for wound healing. It is a fibrin matrix in which platelet cytokines, cells and growth factors may be released after a certain period of time and that can serve as a resorbable membrane. It exhibits several merits over traditional Platelet Rich Plasma(PRP).

Here the biochemical handling of blood is avoided, also it is a simplified and cost-effective process. There is no use of bovine thrombin and anticoagulants and it imparts favourable healing due to slow polymerization, more efficient cell migration and proliferation. PRF has a supportive and beneficial effect on immune system and also plays an important role in haemostasis.[2]

Preparation of PRF:

The classical technique for PRF preparation was invented by Dr. Joseph Choukroun in 2000. It is the current PRF technique authorized by the French Health Ministry. Here the PRF preparation is done without using an anticoagulant during blood harvesting or bovine thrombin during gelling.[3]

For the PRF preparation, blood sample is collected from the patient without anticoagulant using a butterfly needle. 10 ml blood collection tubes are

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used here. After collection of blood, it is immediately centrifuged on a table-top centrifuge at a rate of 3000 rpm for 10 minutes. After centrifugation, three layers are obtained in the test tube (Figure 1). The uppermost layer consisting of acellular PPP (platelet poor plasma), PRF clot in the middle and RBCs at the bottom of the test tube. The middle layer of PRF clot is then removed with sterile tweezers and is separated from the underlying RBC layer using scissors. Then it is transferred on a sterile dish and stored in a refrigerator. It is believed that the junction of PRF to the RBC layer is rich in growth factors and therefore this region is preserved for the procedures.[4]

Here, PRF results from a natural and progressive polymerization which occurs during centrifugation.5 Blood starts to coagulate as soon as it comes in contact with the glass surface because of the absence of an anticoagulant. Therefore, for successful preparation of PRF, faster blood collection and immediate centrifugation, before the initiation of the clotting cascade is absolutely essential.[6] Slow handling of blood to centrifugation process may result in diffuse polymerization of fibrin leading to the formation of a small blood clot having irregular consistency.[7]

PRF membrane can also be obtained by squeezing out the liquids present in the fibrin clot. Liquid removal from the PRF fraction can be done by applying mechanical pressure between gauze layers resulting in a fairly solid and gel-like material that can be used in various clinical applications as a filling material or as a suturing membrane.[8] PRF membrane can also be prepared by compressing PRF clot in special tools like "PRF Box" . It results in standardized membranes of constant thickness and size along with PRF exudate. PRF exudate contains adequate amount of growth factors (TGF-b1, PDGF-AB), matrix glycoproteins (fibronectin, vitronectin etc.) and proteins specialized in increasing cell attachment to biomaterials and titanium. Therefore it can be used for biomaterial impregnation, rinsing surgical sites, hydration of graft materials and for storage of autologous grafts.[7,9,10]

Although PRF belongs to a new generation of platelet concentrates, it is in the first place, a fibrin technology. This is because of the gelling mode. PRP uses bovine thrombin and calcium chloride for coagulation. So there is sudden fibrin polymerization leading to tetramolecular or bilateral junction type of organization of fibrin network which is a rigid fibrin network not conducive for cytokine enmeshment and cellular migration. On the other hand, PRF has a characteristic property of polymerizing naturally and slowly during centrifugation. So, formation of a tripod arrangement of molecules and organization of fibrin network with same distance on all three sides occurs. This is elastic and flexible and supports cytokine enmeshment and cellular migration.[2]

General classification of platelet concentrates

The first classification[11] was proposed in 2009. It separated the products following at least 2 key parameters: the presence of a cell content (mostly leukocytes) and the fibrin architecture. This separation allowed to define four main families to regroup the products.

1. Pure Platelet-Rich Plasma (P-PRP) – or Leukocyte-Poor Platelet-Rich Plasma – products are preparations devoid of leukocytes and with a low fibrin density network after activation. The products of this family can be used as liquid solutions or in an activated gel form. It can therefore be injectable (for application in sports medicine) or placed during gelling on a skin wound or suture (similar to the use of fibrin glues).

Many methods of preparation exist, particularly using cell separators (continuous flow plasmapheresis) from hematology laboratory. This method is much too heavy to be used frequently and easily in daily practice. One largely advertised method of P-PRP is known under the commercial name PRGF26 [Plasma Rich in Growth Factors or Preparations Rich in Growth Factors or EndoRet, Biotechnology Institute BTI (dental implant company), Vitoria, Spain] and was tested in many clinical situations, particularly in sports medicine. Significant drawbacks of the technique include the lack of ergonomics and the need for approximate pipetting steps during the preparation.[11,12]

2. Leukocyte-and Platelet-Rich Plasma (L-PRP) products are preparations with leukocytes and with a low-density fibrin network after activation. All the products of this family can be used as liquid solutions or in an activated gel form.[13] It can therefore be injected (for example in sports medicine) or placed during gelling on a skin wound or suture (similar to the use of fibrin glues).

It is in this family that the largest number of commercial systems exists with many interesting results in general surgery, orthopaedics and sports medicine.[14,15] Particularly many automated protocols have been developed in the last years, requiring the use of specific kits that allow minimum handling of the blood samples and maximum standardization of the preparations, for example Harvest Smart-PreP (Harvest Technologies, Plymouth, MA, USA) and Biomet GPS III (Biomet Inc., Warsaw, IN, USA). Other kits with more handling also exist, such as Plateltex (Prague, Czech Republic) or Regen PRP (RegenLab, Le Mont-sur-Lausanne, Switzerland).

3. Pure Platelet-Rich Fibrin (P-PRF) – or Leukocyte- Poor Platelet-Rich Fibrin – are preparations without leukocytes and with a high-density fibrin network.

Per definition, these products only exist in a strongly activated gel form. This cannot be injected or used like traditional fibrin glues. Because of their strong fibrin matrix, they can be handled like a real solid material for other applications.[11]

There is only one product in this family, commercially known as Fibrinet PRFM (Platelet- Rich Fibrin Matrix, Cascade Medical, Wayne, NJ, USA, also marketed for orthopedic applications by Vertical Spine, Marconi Road Wall, NJ, USA). The main inconvenience of this technique remains its cost and relative complexity in comparison to the other forms of PRF available, the L-PRF (Leukocyte- and Platelet-Rich Fibrin).[11]

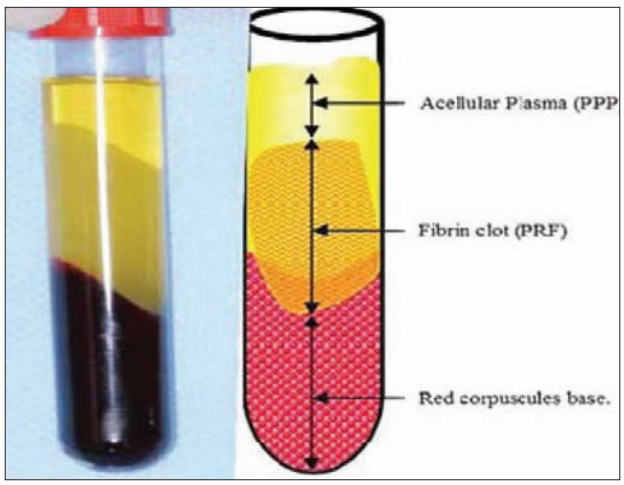

Figure 1

4. Leukocyte- and Platelet-Rich Fibrin (L-PRF) products are preparations with leukocytes and with a high-density fibrin network[16]. As per definition, these products only exist in a strongly activated gel form, and cannot be used in injectable form or used like traditional fibrin glues. They have a strong fibrin matrix which help them to be handled like a real solid material for other applications.

Mishra et al.[17] proposed a classification only for sports medicine applications and taking into consideration the platelets and leukocytes concentrations.

This classification creates 4 types of PRP, depending on the presence or absence of leukocytes and on the activation or not of the PRP. Following this proposal:

- Type 1 PRP is a L-PRP solution
- Type 2 PRP is a L-PRP gel
- Type 3 PRP is PPRP solution
- Type 4 PRP is a P-PRP gel.

This classification follows the same idea than the general classification published in 2009, but is more limited (PRP only) and less intuitive.

Another system called PAW (Platelets, Activation, White cells)[18] was proposed to organize and compare results in the literature, and it insists on the platelet quantity (absolute number), the activation mode of the platelets and the presence of white cells. This system has the limitation that it also covers only PRP and is in fact very similar to the proposal of Mishra et al.[17]

Fibrin – a natural guide for angiogenesis

Fibrin, the activated form of a plasma molecule called fibrinogen[19] is a soluble fibrillary molecule and is massively present both in the plasma and also in the platelet alpha granules. It plays an important role in platelet aggregation during homeostasis and the fibrin matrix also has the property of angiogenesis.[20,21] Infact, fibrinogen is the final substrate of all coagulation reactions. Being a soluble protein, it is transformed into an insoluble fibrin by thrombin, while the polymerized fibrin gel constitutes the primary cicatrical matrix of the injured site.[22-24]

Platelet rich fibrin- a natural fibrin matrix

PRF is an immune and platelet concentrate collecting on a single fibrin membrane, containing all the constituents of a blood sample that are favourable to healing and immunity. This new biomaterial looks like an autologous cicatrical matrix, which is neither like fibrin glue nor like a classical platelet concentrate. It is simply centrifuged blood without any addition. PRF consists of a fibrin matrix polymerized in a tetra molecular structure, incorporating platelets, leucocytes, cytokines, and circulating stem cells. Studies reveal that this biomaterial would be a favourable matrix for the development of a coherent healing,[20,25] without any inflammatory excess. PRF in the form of a platelet gel can be used along with bone grafts. It has several advantages, such as promoting wound healing, bone growth and maturation, wound sealing and haemostasis, and imparting better handling properties to graft materials. It can also be used as a membrane. Many clinical trials suggest the combination of bone grafts and PRF help to enhance the bone density.[26,27]

PRF-advantages

The advantages include

- Simple preparation, with a single step centrifugation, free and openly accessible for all clinicians.
- Addition of external thrombin is not required because polymerization is a completely natural process, excluding the risk of suffering from an immunological reaction .
- .● The manipulation of blood is minimised.
- It is obtained by autologous blood sample.
- It has a natural fibrin framework with growth factors within that may keep their activity for a relatively longer period which, in turn , may stimulate the tissue regeneration effectively.
- It can be used alone or in combination with bone grafts, depending on the purpose.
- Increases the healing rate of the grafted bone .
- It is an economical and quicker option compared with recombinant growth factors when used in conjunction with bone grafts.
- Used as a membrane, it avoids a donor site surgical procedure and results in a reduction in patient discomfort during the early wound-healing period.
- The studies comparing both PRF and PRP shows that PRF is more efficient with less controversies on its final clinical results.

PRF-disadvantages

The disadvantages include :

- The final amount available is low because it is autologous blood.
- Glass-coated tube may be needed to achieve clot polymerization.
- Possible refusal of treatment by the puncture is required for blood sample collection.
- The success of the PRF protocol depends directly on the handling related to blood collection time and its transfer for the centrifugation process.
- Clinical experience and knowledge of the clinician for PRF manipulation plays an important role in the success. [28,29]

Discussion

The periodontal ligament cells play a dominant role in the regeneration of periodontal tissues. Human periodontal ligament fibroblasts (PDLFs) play an active role in alveolar bone formation and resorption in the development of periodontitis, forming a heterogenous population, with some cells having osteoblast-like characteristics and the potential to differentiate into osteoblasts. PDLFs exhibit strong alkaline phosphatase activity, which appears to be very important for the apposition of acellular cementum.[30,31]

Recently, PRF was found to stimulate the proliferation of human PDLFs.[32] However, the underlying mechanisms are yet to be explored. The extracellular signal-regulated protein kinase (ERK) signalling pathway is one of the mitogen-activated protein kinase cascade which plays an important role in the regulation of cell growth and differentiation.

Osteoprotegerin (OPG), a naturally occurring inhibitor of osteoclast differentiation, binds to the receptor activator of nuclear factor-B ligand (RANKL) and blocks RANKL from its interaction with RANK. Alkaline phosphatase (ALP) remain one of the markers of osteoblastic differentiation.

Alkaline phosphatase (ALP), a membrane-bound glycoprotein, is a marker for osteogenic differentiation. It is considered to indicate the presence of osteoblast

and the formation of new bone. The expression of ALP has indicated osteoblast-like characteristics in human PDLFs. ALP activity was seen to be elevated by PRF in a time-dependent manner. Similar results have reported that PRF could increase ALP activity in osteoblasts and human pulp cells in vitro. These results indicated that PRF may contribute to the differentiation of human PDLFs into osteoblasts. PRF increased the secretion of osteoprotegerin (OPG), suggesting that the enhancement of OPG secretion could inhibit osteolytic activity.[30,33-35]

Role of PRF in wound healing:

- Prolonged release of growth factors at the wound site.
- Proliferation of fibroblasts and osteoblasts.
- Mechanical adhesion by fibrin.
- Trapping of circulating stem cells.
- Immunity regulation
- Promotes angiogenesis.
- Induces collagen synthesis.
- Guides in wound coverage.

Wound healing consists of three phases

1. Inflammatory phase (1-4 days) (substrate-preparation phase)
2. The proliferation phase (2-22 days) (collagen-building phase)
   - Epithelialation
   - Angiogenesis
   - Granulation tissue formation
   - Collagen deposition
3. Maturation (remodeling phase) (6-12 months)
   - Collagen maturation and contraction[36]

Current Applications of PRF in Dentistry:

In recent times, a lot of research has been done on PRF and numerous cases have been reported regarding the usage of PRF clot and PRF membranes.

The use of PRF in oral surgery for bone augmentation, sinus lifts, avulsion sockets etc and in periodontics to correct intra-bony defects, gingival recession, guided bone regeneration, periapical lesions etc are being widely researched. In Endodontics, it has also been used for regeneration in open apex, regenerative pulpotomies, periapical surgeries etc.[10]

Recently, the use of PRF has also been proposed in the management of bisphosphonate-related osteonecrosis of the jaw (BRONJ) and those who were treated with both L-PRF (leukocyte rich PRF) and BMP-2 showed favorable outcomes with complete resolution of the lesions.[37]

In Periodontics:

In periodontics, PRF has been successfully used to treat gingival recession, intra-bony defects and periapical lesions. Some case reports show the combined usage of PRF gel, hydroxyapatite graft and guided tissue regeneration (GTR) membrane to treat intrabony defects.[6] Some studies show the combined usage of PRF gel and PRF membrane along with with a bone graft for treating a tooth with a combined periodontic- endodontic lesion.[38] The use of two layers of PRF membrane to cover the defect has also been appreciated. The membranes are very thin and inhomogeneous and leucocytes and platelet aggregates are believed to be highly concentrated in end of the membrane. Therefore, two layers of membrane in opposite sense can be used to prevent the resorption of the thin membrane and also to allow the entire surgical area to be exposed to same components (leucocytes and platelet aggregates).[38] Platelet rich fibrin as a potential novel root coverage approach has been reported by Anil kumar et al. for covering localised gingival recession in mandibular anterior teeth using combined laterally positioned flap technique along with PRF membrane.[39]

PRF can promote the healing of osseous defects by the following mechanisms. PRF promotes the expression of phosphorylated extracellular signal-regulated protein kinase (p-ERK) and stimulates the production of osteoprotegerin (OPG) which in turn causes proliferation of osteoblasts.[3,40] PRF stimulates the osteogenic differentiation of the human dental pulp cells by up regulating osteoprotegerin and alkaline phosphatase expression. PRF also releases growth factors such as platelet-derived growth factor and transforming growth factor which promote periodontal regeneration.[36]

The use of PRF as a tissue engineering scaffold is also being widely researched. Gassling et al. reported

that PRF appears to be superior to collagen as a scaffold for human periosteal cell proliferation. It was seen that PRF membranes can be used for in vitro cultivation of periosteal cells for bone tissue engineering. Thus it was considered that PRF is a potential tool in tissue engineering but clinical aspects of PRF in this field needs to be investigated.[41]

Recent advancements

I-PRF

Mourao et al postulated a study in which represent an alternative to platelet concentrates by using platelet rich fibrin in liquid form(injectable) and its use with particulate bone graft materials. Here, 9 ml tubes are used for blood collection. After blood sample collection, three tubes were placed in a horizontal centrifuge with a tube filled with water in order to maintain balance during centrifuging for two minutes at 3300 rpm.[42]

i-PRF demonstrated the ability to release higher concentrations of various growth factors and induced higher fibroblast migration and expression of PDGF, TGF-β, and collagen.[43]

Advanced PRF(A-PRF)

It is a fourth generation PRF. It is obtained at 1500 rpm for 14 minutes centrifugation time. It has greater concentration and more homogeneous distribution of monocytes, which play a key role in bone formation and clot formation. A-PRF might influence bone and soft tissue regeneration, especially through the presence of monocytes/macrophages and their growth factors. The relevance and feasibility of this tissue-engineering concept have to be proven through in vivo studies.

Other cells that can be observed in these advanced fibrin clots are B- and T-lymphocytes.

Lymphocytes are responsible for specific and nonspecific intervention in tissue response for injury, although they are not prominent in the first phase of tissue repair. It was revealed that CD8+ T-lymphocytes decreased wound healing, whereas B-lymphocytes were associated with an increased healing. Platelets are distributed more evenly throughout the entire clot. It appears that a decrease in centrifugation speed and an increase in centrifugation time results in higher platelet concentrations in the distal part of clot.

Platelets, providing the name for these fibrin-rich scaffolds (ie, platelet-rich fibrin) have a vast regenerative potential by releasing a broad spectrum of cytokines, chemokines, growth factors, and other mediators. Platelets are able to release molecules such as von Willebrand factor, P-selectin, fibronectin, VEGF, platelet-derived endothelial growth factor (PDEGF), vitronectin, and fibrinogen. With these different growth factors, adhesion molecules, and other mediators, platelets have the ability to initiate and modulate host immune responsiveness through influencing neutrophils, monocytes, and endothelial cells, as well as lymphocytes. Upon stimulation, platelets actively participate in pathogen detection, capturing, and sequestration. They can even induce the death of infected cellular targets. Monocytes are also essential for tissue healing. They migrate to the inflamed area after the neutrophil influx where they become macrophages. Macrophages are multifunctional cells that represent distinct phenotypes. They have substantial roles in foreign body response, osteogenesis, and angiogenesis as they respond to inserted biomaterials. Macrophages support cell proliferation and tissue restoration following injury. They are seen throughout all the processes of tissue repair from early inflammation through tissue-remodeling and scar formation.

Neutrophilic granulocytes are most commonly considered to be early inflammatory cells due to their phagocytotic capacity, degranulation, and neutrophilic extracellular traps. However, it is shown that neutrophilic granulocytes have tissue regeneration properties as well. Neutrophils also facilitate trafficking of monocytes into the wound to phagocytose inflammatory remnants (necrotic and apoptotic cells). Moreover, they also participate in the process of wound debridement by secreting several proteases, including matrix metalloproteinase 9 (MMP9), an extracellular matrix digesting enzyme.

Furthermore, neutrophilic granulocytes expressing MMP9 play a part in the process of revascularization of the tissue defect by being recruited. Neutrophilic granulocytes and monocytes/macrophages are in mutual communication, and their interplay contributes to further differentiation towards a pro- or anti-inflammatory state of the macrophages.

These cells modulate the innate as well as adaptive

immune response in a direct and indirect manner by crosstalk with B- and T lymphocytes. Thus, the distribution of neutrophilic granulocytes within the A-PRF clot might be the basis for a better functionality of the transplanted (but also resident) monocytes/ macrophages and lymphocytes and their deployment to support tissue regeneration.[44]

Titanium PRF(T-PRF)

This is based on the concept that titanium plays an important role in the platelet activation when compared to silica activators. Titanium has the highest strength to weight ratios and is also corrosion resistant among the metals. Due to its non corrosive property, it maintains excellent biocompatibility. The material passivates itself invivo by forming an adhesive oxide layer. It also plays an important role in osseointegration, connecting structurally and functionally with the underlying bone.[45]

The T-PRF membrane exhibited positive effects on palatal mucosal wound healing. T-PRF, which is a promising autogenous matrix for histoconduction, may also be preferred as an autogenous alternative to connective tissue grafts in the treatment of gingival recessions and peri-implant mucosal recessions.[46]

The fibrin structure of T-PRF seemed to have been woven more tightly and thicker than classic L-PRF. It was established that the fibrin carpet formed with titanium had a firmer network structure. Strong fibrin structure is important to extend the time for resorption of fibrin and increase the release time of growth factors. T-PRF is also used to avoid any short- and/or long-term negative effects of dry glass or glass-coated plastic tubes and to eliminate the concerns regarding silica. Tunali et al in his study showed that modified T-PRF is a new autogenous product with superior fibrin network. He showed that fibrin formation was made more organised and denser with 2-way direction centrifugation.[47]

Conclusion

The clinical experience confirms that PRF can be considered a healing biomaterial, as it features all the necessary parameters permitting optimal wound healing. The use of PRF as an adjunct in wound healing and periodontal regeneration has shown promising results Currently, platelet-rich fibrin seems to be an accepted minimally invasive technique with low risks and satisfactory clinical results. However, most studies with PRF have shown short term results only. More controlled clinical trials with long term results are required to acquire deeper knowledge about the efficacy and credibility of this biomaterial on a long term basis and to optimize its use in day to day procedures. In addition to clinical trials, more histopathological studies need to be initiated to study the nature of the newly formed tissues in the defect and to understand the biology, efficacy and its mode of action of PRF more efficiently and effectively.

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Lip re-positioning surgery for managing excessive gingival display in a patient with chronic periodontitis - A case report

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ABSTRACT

Excessive gingival display is an aesthetic concern for many and its coexistence with periodontitis pose a unique challenge in management. Lip repositioning surgery has received a renewed interest in the past few years as a clinically feasible method in the management of gummy smile. This case report portrays the management of excessive gingival display in a female patient with chronic generalized periodontitis using modified lip repositioning surgery along with surgical periodontal treatment

Key words- Chronic Periodontitis, modified lip repositioning, gummy smile, aesthetics.

Introduction

A gingival display of more than 3 mm while smiling is often considered unaesthetic and has been termed 'gummy smile'.[1,2,3] The reasons for gummy smile are manifold including muscular hyperactivity, vertical maxillary excess, anterior dentoalveolar extrusion, altered passive eruption, short or hyperactive upper lip or their combination.[3,4] Based on the aetiology, various treatment modalities are indicated such as aesthetic crown lengthening with or without osseous resection[4,5], orthodontic therapy[6], and orthognathic surgery.[7] However, orthognathic surgery is perceived by many as an invasive procedure with associated morbidity and the periodontal status of the patient often precludes orthodontic therapy and osseous resection. Lip repositioning surgery offers a compromised albeit effective way of managing gummy smile in such situations.

Case report

A 38-year-old, female patient reported to the department of Periodontics and Implantology, Mar Baselios Dental College, Kothamangalam with a chief complaint of bleeding gums observed while brushing. The patient did not have any relevant medical history or any history of drug allergy.

Extra-oral examination showed moderate gingival display during smiling which extended from the maxillary right canine to the maxillary left canine [Figure 1]. On intra-oral examination, she had fair oral hygiene. Periodontal findings revealed generalized bleeding on probing and probing pocket depth of 7-8 mm. The maxillary anteriors showed 1-2 mm recession also. There was attrition in relation to both maxillary central incisors with incisal notches and spacing between lateral incisor and canine. The premaxilla was prominent and an associated vertical maxillary excess with a gingival display of around 5 mm was present [Figure 1]. There was generalized moderate horizontal bone loss in panoramic radiographic examination [Figure 2]. The case was diagnosed as chronic generalised periodontitis along with a degree 2[8] gummy smile. After phase I therapy and re-evaluation, the patient underwent full mouth access flap surgery, completed in four sittings. Subsequently, the patient was put under regular maintenance.

At three months follow up, the patient was suggested treatment options for the correction of

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the gummy smile. Aesthetic crown lengthening was not possible because of the pre-existing gingival recession and the patient did not want to undergo an extensive procedure such as orthognathic surgery. After discussing the advantages and limitations with the patient, it was decided to perform lip repositioning surgery with her written informed consent.

After obtaining adequate anaesthesia, the apical, coronal and lateral boundaries of the planned incision were outlined using a marker pen. Pre suturing was performed accordingly to have an approximate idea of the post-surgical result [Figure 3].

Sequentially, incisions were made at the level of mesial line angle of left maxillary first premolar which was carried along the mucogingival junction by the frenum without interrupting the frenal attachment. A second incision 10 mm above the first incision was made in the left labial mucosa. The two incisions were joined on either side and a strip of partial thickness flap was raised and removed, exposing the underlying connective tissue. The procedure was repeated on the right side in a similar manner. The incisions margins were then approximated using continuous interlocking sutures, keeping the frenal attachment as a reference for midline [Figure 4]. Suitable antibiotics and analgesic anti-inflammatory agents were prescribed for five days.

Post-operatively, the patient was advised soft diet, limited facial movements, avoidance of brushing around the surgical site for two weeks and use of 0.2% Chlorhexidine gluconate mouth rinse. No untoward complications were reported by the patient in the immediate post-operative period other than tension on the lip while talking or smiling, which lasted for a week. Sutures were removed after two weeks. Surgical site demonstrated normal pattern of healing and the patient expressed satisfaction at the surgical result. Since the incisal edges of the maxillary anteriors showed attrition, re-shaping was done to achieve a more even appearance. At three months review post lip re-positioning, the patient's appearance had improved to her satisfaction and the gingival display showed a reduction of 2-3 mm [Figure 5]. The periodontal status also showed improvement in terms of reduction in probing depths.

Fig.1- Pre-operative view of excessive gingival display while smiling

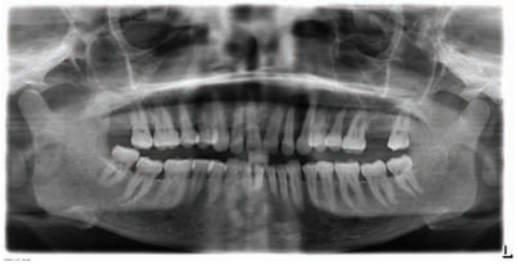

Fig.2- Panoramic radiograph of generalized moderate horizontal bone loss

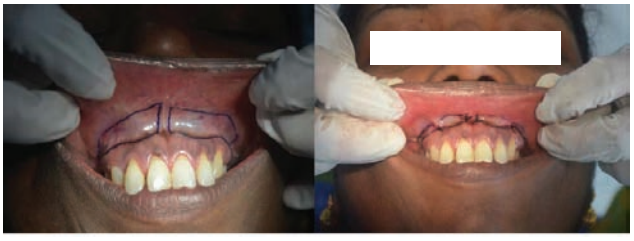

Fig.3- Surgical extent outlined and trial suturing done

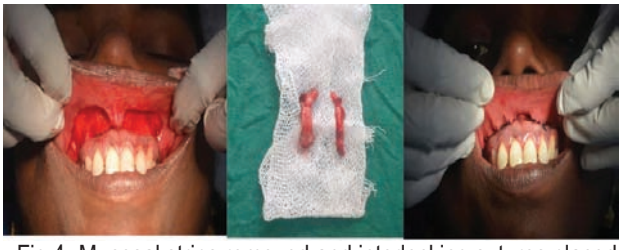

Fig.4- Mucosal strips removed and interlocking sutures placed

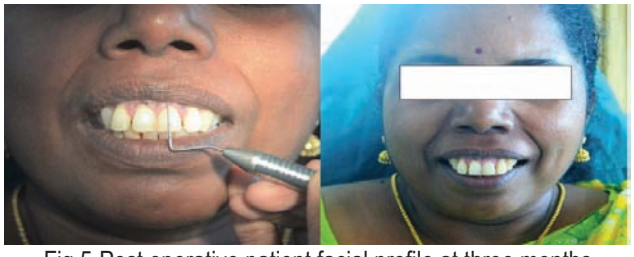

Fig.5-Post operative patient facial profile at three months showing reduction in gingival display

Discussion

Excessive gingival display while smiling affects both the patient facial profile as well as the periodontal status. The persistent exposure results in dryness of the gingiva leading to reduced flushing action of saliva that favours plaque accumulation. Many treatment

options available for managing gummy smile may not be possible in patients with co-existent periodontitis. Lip repositioning is one of modalities that may be undertaken in such a scenario. Lip repositioning technique was introduced in the field of plastic surgery in 1973.[9] The major indications for this technique include patients with bony maxillary excess having excessive gingival display (EGD) of Degree 1(1-2mm), Degree 2 (4-8mm) and cases with excessive mobility of maxillary lip with sub class 1(1-3mm), sub class 2(4-6 mm) and sub class 3(>7mm).[8] This technique is considered as an excellent alternative to more costly procedures with high morbidity rates.[10] The procedure has an advantage of improving facial aesthetics with addressing unilateral EGD with the additional option of reversibility if necessary via a vestibular extension procedure,[8] and is a less invasive surgery with low morbidity rate[10] and minimal complications. During surgery care must be taken to avoid damage to minor salivary glands in the sub mucosa. In some cases, rare complications have been reported in the literature such as paresthesia[11] and transient paralysis.[12]

The most frequent complication encountered in the surgical procedure is the lip asymmetry created during the surgical procedure. To overcome this, Silva et al introduced the modified lip repositioning technique. Treatment consisted of the removal of two strips of mucosa, bilaterally to the maxillary labial frenum and coronal repositioning of the new mucosal margin. The baseline gingival display of 5.8 ± 2.1 mm significantly decreased to 1.4 ± 1.0 mm at 3 months and was maintained until 6 months (1.3 ± 1.6 mm). Subjects were satisfied with their smile after surgery and would likely choose to undergo the procedure again (92%).[13]

One of the main periodontal contraindications for lip repositioning surgery is patients who exhibit EGD of Degree 3 (>8 mm),[8] inadequate width of attached gingiva in the maxillary anterior sextant. In those cases, difficulties are encountered during flap design, approximation and stabilization at the time of suturing.[14, 9] The disadvantage of this technique is the possible chance of relapse within six months to one year.[15-16]

Patient's gingival biotype is the main factor responsible for the relapse. Patients with thin gingival biotype are more susceptible to relapse than those compared with thick biotype.[14,7]

Conclusion

Excessive gingival display in patients with periodontitis has aesthetic as well as periodontal concerns. Lip repositioning offers a reasonable alternative to the more traditional approaches in such situations. Sustenance of results in the long term is unclear; yet short term outcomes are considered good, especially patient satisfaction.

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Consequences of smoking on periodontal treatment

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ABSTRACT

Deleterious effects of smoking on general health as well as periodontal tissues have been studied and established. Smoking is a proven risk factor in the progression of periodontitis, affecting prevalence, extent, and severity of disease. This review emphasizes on studies related to the influence of smoking on different periodontal treatment modalities like scaling and root planing, surgical therapies, regenerative or grafting procedures, implant placement and their outcomes.

Key words: Smoking; non-surgical periodontal treatment; surgical periodontal treatment; Dental implant therapy.

Introduction

Smoking is the inhalation of smoke of burning tobacco encased in cigarettes, pipes, and cigars. The deleterious effect of smoking has impact on most of the parts of body and results in multiple diseases leading to reduced life expectancy and quality of life.

Cigarette smoke contains over 4,000 chemicals, including carcinogenic compounds and toxins. It includes nicotine, tar, carbon monoxide, formaldehyde, ammonia, hydrogen cyanide, arsenic, and dichloro diphenyl trichloroethane (DDT). Nicotine is highly addictive. Cigarette smoking is a strong risk factor for oral cancer, oral mucosal lesions, and periodontal diseases.[1] Smokers may harbour a higher prevalence of potential periodontal pathogens. Smoking impairs various aspects of the innate and adaptive immune responses, including neutrophil function, antibody production, fibroblast activities, vascular factors and inflammatory mediator production.[2] In addition to being positively linked with cardiovascular disease, lung disease, poor pregnancy outcomes and cancer; tobacco use has been reported to negatively affect the treatment outcomes of all periodontal procedures starting from mechanical debridement, local and systemic antimicrobial therapy, periodontal surgery and regenerative procedures.[3] In a prospective study, 26-year-old individuals of European descent who had smoked between the ages of 15 and 26 were almost three times as likely as non-smokers to have at least one site with 4 mm or more attachment loss, after controlling for gender, plaque levels, and professional dental care.[4] This article focuses on the impact of smoking on various periodontal and implant therapy.

1. Nonsurgical therapy

With non-surgical therapy as the main treatment modality, most studies shows greater reductions in probing depth in non-smokers compared with smokers (Preber & Bergstrom 1985, Preber et al. 1995, Grossi et al. 1997, Renvert et al. 1998, Preshaw et al. 1999). A meta-analysis by Labriola in 2005, on the impact of smoking on nonsurgical therapy, found that probing depth reduction in sites where probing depth was initially ≥5 mm was significantly greater (0.433 mm) in non-smokers than in smokers in eight studies.[5] In a separate study where treatment included 4 - 5 hours of scaling and root planing, oral hygiene instructions, and a session of scaling at 3 months , non-smokers had 0.9

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mm more probing depth reduction and 0.6 mm more clinical attachment gain at periodontitissites (probing depth ≥5 mm, clinical attachment level ≥3 mm) compared to smokers at 6 months(6). Darby reported 0.7 mm less improvement in probing depth and 0.4 mm less attachment gain in smokers compared to non-smokers at the 6- to 8-week re-evaluation following scaling and root planing[7]. A trend has been noted for heavy smokers (≥20 cigarettes per day) to respond less favourably to treatment than light smokers (<20 cigarettes per day)[8]. A study conducted by Grossi et al in 1997 on response to nonsurgical therapy reported mean reduction of plaque index by 0.54%, 0.4%, 0.69 % and mean reduction of pocket depth by 0.33mm, 0.49mm, 0.49mm among smokers,non-smokers and ex-smokers respectively. Zuabi et al. (1999) reported significantly more plaque in smokers compared with the non-smokers and the smokers had significantly greater probing depths at baseline compared with the non-smokers. Papantonopoulos (1999) noted that between 6 and 8 weeks following non-surgical therapy, significantly more smokers (42.8%) than non-smokers (11.5%) required further treatment and the smokers may have benefitted from a surgical approach in the first instance (Papantonopoulos 1999). In radiographic study, Meinberg et al. (2001) reported significantly more bone loss after 12 months follow-up in smokers compared with non-smokers and concluded that more longer-term studies are essential to identify the association between smoking status and outcome variables.[9]

2. Soft- and Hard-Tissue Grafting

Harris consecutively treated recession sites using a connective tissue with a partial thickness pedicle graft and found no difference between the percentage of root coverage among light smokers (n=11, 97%), heavy smokers (n=21, 99%) or non-smokers (n= 68, 98%). (10) Sub epithelial connective-tissue grafts were less successful in smokers than non-smokers. Following a coronally positioned flap, root coverage was significantly less for smokers compared to non-smokers (69.3% vs. 91.3%).[11] When expanded polytetrafluoroethylene membranes were utilized in guided tissue regeneration procedures at recession sites, smokers had significantly less root coverage (57%) than non-smokers (78%). Smoking has also been reported to negatively impact regenerative procedures in interproximal and furcation defects, including osseous grafts, membranes or membranes in combination with osseous grafts[8]. Tonetti et al. (1995) reported clinical attachment gain in non-smokers (5.2 mm) compared with smokers (2.1 mm) following guided tissue regeneration of infrabony defects. It also concluded that higher plaque levels are seen in smokers compared with non-smokers.[9]

3. Implant Therapy

Many of the studies on implant outcomes in smokers are retrospective in nature or are case series. In majority of these studies, smokers had at least twice the failure rate of implants compared to non-smokers. A study initiated in 1991 showed that 8.9% of implants had failed in smokers compared to 6% in never or former smokers. The use of pre-operative antibiotics reduced failures in smokers by more than 10%, compared to a 3% reduction in the non-smoker/ quit group. Implants are susceptible to the same bacterial and host influences as the natural dentition. Factors that interact with smoking to impact implant outcomes include an interleukin-1 genotype, implant location (maxillary vs. mandibular) and the presence of periodontal disease.[12] The dose effect of smoking is another important consideration. The implant failure rate in grafted maxillary sinuses in smokers was 12.7% compared to 4.8% in non-smokers.[8] Smoking has been shown to be a risk factor for periimplantitis, with most of studies showing significant increase of periimplant bone loss compared to non-smokers.[13]

4. Effect of smoking on host modulation therapy

Adverse effects of smoking affect the host tissue by means of the increased levels and/or activity of proteolytic enzymes directed against the structural elements of the periodontium, the elevation of destructive inflammatory cytokines, and/or suppression of the regenerative/reparative functions of the periodontium. Because of the less favourable treatment response in smokers, clinicians may be more likely to utilize adjunctive antimicrobial therapy in these patients. Another study comparing adjunctive systemic antibiotic therapy to scaling and root planing alone, smokers receiving amoxicillin and metronidazole showed significantly more improvement in bleeding scores, probing depth and attachment levels than smokers receiving only scaling and root planing.[14]

Response of various periodontal treatment among smokers and non-smokers.

Table 1- Review of studies that have compared the effectiveness of treatment of chronic periodontitis in smokers and non-smokers.[9]

Study	Treatment	Interval of follow up	Smokers	Non-smokers	Conclusion
Preber and Bergstrom, 1985	Nonsurgical therapy	1 month	Mean PD reduction of 1.1 mm	Mean PD reduction of 1.2 mm	Non-surgical therapy can reduce PD in smokers and non-smokers. Compared with non-smokers, smokers have less reduction.
Preber and Bergstrom, 1990	Periodontal surgery	1 year	Mean PD reduction of 0.76 mm	Mean PD reduction of 1.27 mm	Statistically significant PD reduction. Smoking interfere with therapeutic outcomes because of interference with healing events
Tonetti et al, 1995	Guided tissue regeneration (GTR)	1 year	2.1 mm mean attachment gain	5.2mm mean attachment gain	Smoking associated with reduction healing response to GTR treatment.
Ryder et al ,1999	Non-surgical treatment or subgingival doxycycline	9 months	Nonsurgical group-attachment level gain of 0. 76mm.PD reduction of 1.02mm in pocket Doxycycline group—Attachment level gain-0.83mm; PD reduction-1.21mm	Nonsurgical group-attachment level gain-1mm. PD reduction of 1.43mm in pocket Doxycycline group—Attachment level gain-0.69mm; PD reduction-1.12mm	Significantly greater clinical attachment gain seen in non-smoking non-surgical treatment modality. Local doxycycline and Non-surgical treatment act in synergy if used together in periodontal treatment.
Trombelli et al 2003	Flap surgery at furcation defect	6 months	27.6% of classII furcation showed improvement. After 6 month- 3.4% of presurgery classI furcation defect showed complete closure.	38.5% of class II furcation showed improvement. After 6 month- 27.8 % of presurgery class I furcation defect showed complete closure	Flap surgery provided both clinically and statistically significant PD reduction and clinical attachment gain in class I/II molar furcation defect. Smokers exhibited less favourable healing outcome.
Stavropoulos et al.,2004	Surgical treatment of vertical defects with bio resorbable membranes	12 months	Mean PD reduction of 4.5mm.Mean CAL gain of 3.2mm	Mean PD reduction of 5.5mm.Mean CAL gain of 4.3mm	Smoking impairs healing of GTR-treated infrabony defect
M Feres, et al,2015(16)	Scaling and root planing	180 days	Significant increase in the host-compatible species (24.1% to 35. 3%).No significant reduction was observed in the proportion of pathogenic species (69.0% to 55.3%	Significant increase in the proportion of host-compatible species (20.1% to 50.8%). Decrease in the proportion of pathogenic species (72.6% to 45.0%)	Re-establishment of a more pathogenic subgingival biofilm than non-smokers
Silva et al, 2010(17)	Mucogingival surgery—using Free gingival graft (FGG)	90 days	FGG width, length, and area were respectively reduced by 44%, 25%, and 58%	FGG width, length, and area Were respectively reduced by 31%, 22%, and 44%.	Smoking alters FGG donor-site wound healing by delaying epithelialization.
Stramazzotti et al, 2015(18)	Guided tissue regeneration.	12 months	PD reduction-3.6 ±1.9 mm); CAL gain----2.8±2.2. Recession---0.8±0.9mm	PD reduction (6.3 ± 2.1 mm. CALgain (4.4± 1.1mmn Recession (1.8 ±1.4mm	Smoking impairs the healing outcome of GTR treatment of Intrabonydefects
Amri et al, 2016(19)	Implant-immediate and delayed implant	5 years	Mean PI (range) of ID implants --- 47.1 (36.4 to 60.1) Mean PI range of DL implants --45.5 (39.5 to 62.4)	Mean PI (range) of ID implants--24.3(15.6 to 30.6) Mean PI of DL-- 21.8 (8.6 to 39.3)	smoking enhances peri-implantitis and CBL around IL and DL implants.

PD, probing depth; PI, plaque index; BOP, bleeding on probing; SPT, supportive periodontal therapy; GCF, gingival crevicular fluid; GI, gingival index; CAL, clinical attachment level- immediate loaded implant, DL- delayed loaded implant.

Impact of smoking during maintenance

Detrimental effect of smoking on treatment outcomes appears to be long-lasting and independent of the frequency of maintenance therapy. In a study, four different modalities of surgeries including scaling, scaling and root planing, modified Widman flap surgery, osseous surgery was carried out. Maintenance therapy was performed by a hygienist every 3 months to 7 years.[15] Smokers consistently had deeper pockets than non-smokers and less gain in attachment then evaluated each year for the 7-year period. Even with more intensive maintenance therapy given every month for 6 months after flap surgery, smokers had deeper and more residual pockets than non-smokers, although no significant differences in plaque or bleeding on probing scores were found. These data suggest that the effects of smoking on the quality of subgingival plaque, the host response, and the healing characteristics of the periodontal tissues may have a long-term effect on pocket resolution in smokers that may not be managed by conventional periodontal therapy.[8]

Conclusion

As a conclusion, it is evident that smoking affects the healing phase of various periodontal treatment and hence the outcome. When comparing current smokers with former smokers and non-smokers, the former and non-smoking subjects appear to respond equally well to non-surgical care. (20) Effects of smoking tip the balance towards periodontal destruction and impairment of regenerative responses, results in terms of improvements in clinical parameters show that smokers responses are around 50% that of non-smokers. Therefore, smoking cessation is important and should be considered as an integral section of periodontal therapy in smokers with periodontitis.

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Microbiology of periodontal diseases: a review

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ABSTRACT

Periodontal diseases are infections that are caused by microorganisms that colonize the tooth surface at or below the gingival margin. Periodontal diseases lead to destruction of the periodontal tissues supporting the teeth. Periodontitis have multifactorial etiology,evidence suggests that certain Specific Gram-negative microorganisms in the subgingival plaque biofilm play a major role in the initiation and progression of periodontitis. Porphyromonasgingivalis, Treponemadenticola and Tannerella forsythia are considered as the principal periodontopathogenic bacteria. Other predominant species in the disease process are: Aggregatibacteractinomycetemcomitans, Fusobacteriumnucleatum, Prevotellaintermedia, Campylobacter rectus, Peptostreptococcus micros, Eikenellacorrodens. The initial colonization of the tissues by these pathogenic species followed bacterial invasion by pathogenic products into the periodontal tissues along with interactions of bacteria with host cells directly or indirectly causes degradation of the periodontal structures which ultimately results in tissue destruction.

Keywords: Keywords: gingivitis, periodontal disease, periodontal pathogens, microbial diagnosis of periodontitis, oral microbiome.

Introduction:

Periodontal disease is characterized by persistent inflammation, connective tissue breakdown and alveolar bone destruction due to bacterial infection. Destructive periodontal diseases are infections caused by bacteria that colonize the tooth surface, gingival margin and subgingival environment. It is well known that periodontitis is not associated with a single microorganism, but is a consortium of bacteria in the initiation and progression of the destructive periodontal lesion which is related to the lack or minimal proportions of the beneficial microorganisms in a susceptible host. Risk factors for periodontal disease includes smoking, systemic diseases, medications such as steroids, antiepileptics, drugs for cancer therapy, poor placement of dental bridges, dental crowding, lack of teeth, pregnancy and contraceptive use. This may cause periodontal inflammation and destruction with attachment loss and bone loss[1].

The periodontal pathogens currently known represent a small part of all of the 600 bacterial species that can colonize dental surfaces over and below the gingival margin and oral mucous membranes. Most of the periodontal pathogens are anaerobes but the biofilm can also harbour facultative aerobes, capnophiles and microaerophiles whose number depends on the environment in the developed biofilm and periodontal pocket. Most periodontal pathogens represent the true periodontal infection. Some bacterial species in the periodontal environment that are part of the commensal flora (Actinomyces,

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certain Streptococcus and Staphylococcus species.) can provoke opportunistic infections in case of ecosystem disturbance.

The complexity of the periodontal micriobiota causes difficulty in isolating the microorganisms in the laboratory. Advances in molecular biology have improved the ability to detect specific bacteria and their products, which may serve as markers of ongoing disease or predictors of future destruction.

This review attempts to highlight the role of microbes in periodontal infection with an over view on the laboratory diagnosis of the pathogens for early detection of periodontal disease to ensure better quality treatment.

Microbiology of periodontal disease

The composition of the oral microbiota is influenced by temperature, pH and atmosphere, as well as by the host defences and host genetics. Recent research identified 800-1,000 species that colonize the oral cavity. Among these species only 50 species are strongly related to periodontal disease,predominantly Actinobacillusactinomycetemcomitans (Aa), Porphyromonasgingivalis (Pg) and Tannerellaforsythensis (Tf). Bacteria are responsible for stimulating the host response, which define tissue changes causing periodontal lesions. Such bacteria are within a glycocalyx forming a biofilm, which allows microorganisms to join and multiply on different surfaces[1].

The biofilm protects the microorganisms from toxic substances in the environment and also facilitates the intake of nutrients, cross-feed, elimination of metabolic products and development of an appropriate environment with suitable physicochemical condition for the growth of microorganisms. Biofilms that colonize the oral cavity are among the most complex in nature. There are 4 different niches: masticatory mucosa, tongue dorsum, saliva and hard surfaces such as tooth surfaces and restorative materials[2].

Many different bacterial species live in the healthy gingival sulcus and are present in different periodontal diseases. The bacterial flora associated with healthy periodontal tissue contains mainly Gram-positive microorganisms with a dominance of Actinomyces and Streptococcus species. Gram-negative species and spirochetes may be also present in healthy patients, although in low concentration.

Microbiota in different periodontal diseases[4]

The current concept about the pathogenesis of periodontitis considers three groups of factors that determine whether active periodontitis will occur in a patient. They are:

1) A susceptible host

2) Presence of pathogenic species

3) Absence or a small proportion of "beneficial" species.

Evidence suggest that in the subgingival flora there is balance between "beneficial" and "pathogenic" species in inactive sites. This part of the protection is probably due to the fact that certain species control others through antagonistic bacterial interactions.

These bacteria can act in different ways:

- by passively occupying the niches;
- by limiting the ability of a periodontal pathogen to adhere to appropriate tissue surfaces;
- by enhancing the vitality and growth of a pathogen;
- by enhancing the ability of a pathogen to produce virulence factors;
- by producing anti-periodontal pathogen .

Plaque-induced gingivitis

Plaque-induced gingivitis is a more prevalent and generalized, and is more severe in individuals with poor oral hygiene. Experimental gingivitis found that inflammatory features are related to emergence and growth of Gram-negative rods and filaments in the dental biofilm, and subsequently, of spirochetes and motile microorganisms. The identified organisms are mainly Gram-negative, anaerobic bacteria and include Streptococcus sanguis, S. mitis, Fusobacterium spp., Actinomycesviscosus, and Veillonellaparvula.

There is evidence that gingivitis-associated microflora is composed of predominantly actinomyces and Streptococcus species, and there is a very small number of Gram-negative bacilli, obligate anaerobes such as P. gingivalis and P. intermedia.

Periodontitis as a manifestation of Systemic disease

Many systemic disorders have been implicated

as risk factors for adverse periodontal conditions such as coronary heart disease, diabetes, preterm labour, low birth weight and respiratory disease. Pregnancy-associated gingivitis is accompanied by increase in steroid hormones in the crevicular fluid and demonstrates modification in the bacterial composition with a higher level of P. intermedia.

Chronic periodontitis

Heterogenic subgingival flora has been found in chronic periodontitis but the bacteria most cultivated in elevated levels are P. gingivalis, T. forsythia, P. intermedia, C. rectus, Eikenellacorrodens, F. nucleatum, A. actinomycetemcomitans, P. micros, Treponemadenticola, and Eubacterium spp. Gramnegative anaerobes and capnophiles are dominant; spirochetes may be present.

Aggressive periodontitis

The specificity of periodontal microflora is strongly supported by the identification of A. actinomycetemcomitans as a pathogen associated with localized aggressive periodontitis. A. actinomycetemcomitans, Tannerella forsythia and Porphyromonas gingivalis are strongly related with the initiation of periodontal disease, disease progression and unsuccessful periodontal therapy. Moderately strong evidence has been accumulated for other bacteria isolated from subgingivalmicrobiota, including Prevotellaintermedia, Prevotellanigrescens, Campylobacter rectus, Peptostreptococcus micros, Fusobacteriumnucleatum, Eubacteriumnodatum and various spirochetes, such as Treponemadenticola, although their etiologic role is less evident.

Refractory periodontitis

Refractory periodontitis microflora is associated with the presence of F. nucleatum, P. intermedia, A. actinomycetemcomitans, P. micros, and spirochetes, rather than with P. gingivalis, T. forsythia and Candida spp. in the dental biofilm.

Acute periodontal diseases

Studies on the microflora of acute periodontal diseases have secured the most solid evidence about the periodontal infection specifics. A strong relation has been found between necrotic periodontal diseases and some bacterial species. The microflora associated with necroticulcerative gingivitis and periodontitis contains Gram-negative bacilli, obligate anaerobes such as F. nucleatum and P. intermedia and spirochetes.

Peri-implant diseases

Investigations in humans demonstrate that the microbiota associated with peri-implant diseases is similar to that of periodontitis: with a high proportion of anaerobic Gram-negative rods, motile microorganisms and spirochetes.

Periodontal disease and viruses

Recent data demonstrate some herpes viruses present in the periodontal pockets, e.g. Epstein-Barr virus-1 and human cytomegalovirus. A study showed the association between virus and periodontal disease and demonstrated the presence of some virus like Epstein-Barr type 1, cytomegalovirus and human herpes in crevicular fluid of Nigerian children with necrotizing ulcerative gingivitis. This pathogenicity is attributed to the degradation of the host defense mechanisms due to viral infection of the gingiva, favouring the bacteria colonization.

The oral and periodontal conditions most closely associated with HIV infection include oral candidiasis, oral hairy leukoplakia, Kaposi's sarcoma, salivary gland diseases, linear gingival erythema and necrotizing gingival and periodontal diseases. Immunosuppressant drugs influences the response of gingival and periodontal tissues to bacterial plaque. They do not abolish the reaction of the tissues to plaque, but appear to dampen down inflammatoryreactions.[8]

Criteria for identification of bacterial species as periodontopathogens:

According to the criteria proposed by Socransky and Haffajee[3], a periodontal microorganism has to meet some conditions to be considered a potential pathogen:

- to be associated with the disease by means of increased number in diseased patients and sites;
- to be reduced or eliminated after treatment and, with the healing, to be capable of provoking the destructive host response;
- to possess the capacity to cause the disease in experimental animal models;
- to demonstrate production of virulence

factors known to cause periodontal destruction.

- to realize their pathogenic potential, bacterial species should be able to colonize the subgingival area,
- to produce virulence factors that directly (enzymes and toxins) or indirectly (antigens and activators) lead to initiation of a destructive inflammatory reaction in the individual and injury of periodontal tissues.

The virulence capacities of bacteria depend on the production of certain factors for adhesion such as adhesins, lectins, fimbriae and vesicles. Agents that directly damage the periodontal tissues are proteases, alkali and acid phosphatases produced by microorganisms, fatty and organic acids, IgG- and IgA-proteases, chondroitinsulfatase and toxic products.

Aggregatibacteractinomycetemcomitans and Porphyromonas gingivalis are the two species classified as periodontal pathogens that fulfill all of the criteria listed above. Other suspected periodontal pathogens are: Treponemadenticola, T. forsythia, Fusobacteriumnucleatum, Prevotellaintermedia, Campylobacter rectus, Eikenellacorrodens, Peptostreptococcus micros, Selenomonas sp. Based on scientific evidence, Socransky and Haffajee classified the microorganisms in the bacterial complexes depending on their presence in the biofilm and subgingival area and their involvement in the pathogenesis of periodontal diseases.

Table 1: Subgingival bacterial classification in Socransky complexes[2]

Bacterial Species	Complex
Actinomyces Veilonella	Purple
Streptococcus: gordonii, intermedius, mitis, sanguis	Yellow
Capnocytophaga E.corrodens	Green
Campilobacter rectus Fusobacterium nucleatum	Orange
P.micros P.intermedia	
T.forsythia P.gingivalis T.denticola	Red
A.actinomycetemcomitans Selenomonas	Not grouped

Bacteria of the purple, yellow and green complexes correspond to periodontal health, but bacteria of the orange and red complexes and other not grouped ones are suspected periodontopathogens.

Periodontal microbiome[5]

Aggregatibacteractinomycetemcomitans is a small, short, straight or curved rod with rounded ends that is nonmotile and gram-negative and is strongly associated with destructive periodontal lesions. It has five serotypes and many biotypes based on differences in polysaccharide composition and can be isolated in aggressive periodontitis and from active lesions of the chronic periodontitis. This microorganism possesses a great number of virulence factors, including leukotoxin (forms pores in neutrophil granulocytes, monocytes, some lymphocytes, which die due to the osmotic pressure), collagenase (destruction of connective tissue), protease (able to cleave IgG), endotoxin (lipopolysaccharide), fibroblastinhibition factor, factor inducing the bone resorption.

Porphyromonas gingivalis is a Gram-negative, nonmotile, asaccarolytic rod-like obligate anaerobe which is related to the initiation and progression of the periodontal destruction. it possesses a capsule which protects against phagocytosis, fimbriae (for adhesion) and vesicles, and produces a number of virulence factors: proteases (for destruction of immunoglobulins and the complement factors, and degradation of host-cell collagenase inhibitors), collagenase, hemolysin (heme-sequestering proteins), endotoxin, fatty acids, h2S, nh4.

Tannerella forsythia (previously Bacteroidesforsythus) is a Gram-negative nonmotile rod-like obligate anaerobe which is found in periodontal sites with active destruction as well as with disease recurrence. This species produces some proteolytic enzymes that can destroy immunoglobulins and complement factors, and induces apoptotic cell death.

Prevotellaintermedia is a short, round-ended nonmotile gram-negative anaerobic pathogenic rod which is less virulent and less proteolytic than P. gingivalis. Treponemadenticola is a Gram-negative motile anaerobe related to periodontal lesions. it is

MICROBIOLOGICAL DIAGNOSIS[1].

Table2: Microbiological diagnostic methods and their advantages

Method	Description	Advantages
Bacterial Culture	Currently the gold standard. Determines the presence of the different species	Evaluates the antibiotic susceptibility Estimates the number of isolated bacteria
Immunological diagnostic methods	Direct immunofluorescence -Indirect immunofluorescence - Flow cytometry -Latex agglutination -Enzyme-linked immunoabsorbentassay (ELISA)	- Calculates the percentage of bacteria -Can recognize the nature of bacteria in the biofilm - Greater sensitivity and specificity
Enzymatic detection methods	- Doesn´t detect bacterias directly, determines the presence of enzymes that bacterias produce - BANA (benzoil arginine naftilamida)	Enables detection of certain bacterial species capable of producing tripsyn enzymes as a virulence factor
Molecular biology techniques of DNA and RNA	Polymerase chain reaction (PCR): amplifies DNA strands	- High specificity - Diagnosis of major periodontal pathogenic bacteria - Does not need living microorganisms

-

found in patients with severe periodontitis, rather than in patients with healthy periodontium or gingivitis. thismicroorganism produces proteolytic enzymes that can destroy immunoglobulins and complement factors.

Campylobacter rectus is a motile gram-negative anaerobic short rod that produces leukotoxin.

Conclusion

The key to successful periodontal therapy and maintenance is elimination or reduction of pathogenic bacteria from periodontal pockets and establishment of microbiota compatible with periodontal health. Identification of subgingival pathogenic strains in gingivitis and periodontitis could aid in the better differentiation of the different periodontal diseases. Advanced microbiological methods will offer a clearer understanding of the biofilm functioning as an ecosystem and help identify new periodontal pathogens. The knowledge of host response and modifiying factors can help in the future treatment of periodontal diseases and development of new preventive measures. Further developments of biological approaches in the periodontal therapy are expected.

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Quorum sensizing- 'The Language of Bacterial Community'

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ABSTRACT

Microorganisms communicate and coordinate with each other; for this some chemical molecules secreted by them. These molecules are useful in both intra and inter-species communication and for coordinating bacterial behavior. Bacteria prefer living in complex surface associated communities which were later named biofilms[1].

Biofilm can be defined as an aggregation of one or more groups of different microorganisms, embedded in a self-produced matrix and adhering to a firm surface. Quorum sensing is a way of communication within a bacterial species, whereas competitive or cooperative signaling can occur between groups of bacteria or between bacteria and the host[1]. These bacterial systems are often integrated into complex, multilayered signal transduction networks which can control numerous multicellular behaviors, including biofilm formation and other virulence traits etc. Bacteria that may be either as an individual single-celled organisms or as multicellular populations. Bacteria exhibit these behaviors by secreting some of the chemical molecules i.e., chemically"talking" to one another through a process called quorum sensing. The deeper understanding of microbial cell communication shed light on the complexities of the host-microbe relationship and this may lead to novel therapeutic applications[8]

Key words:- Biofilm, Bacteria, Quorum sensing, Periodontitis, Periodontal pathogens

Introduction

Microorganisms communicate and coordinate with each other; for this some chemical molecules secreted by them. These molecules are useful in both intra and inter-species communication and for coordinating bacterial behavior. Quorum sensing is the process by which microorganisms monitor and regulate their population density through chemical signaling.[1]

History

Several decades ago bacteria were believed to be independently functioning single-cell organisms, living in a self-lifestyle. But in the mid-20th century several pioneer studies conducted on marine bacterias and it showed that bacterial cells actually favor in living in close proximity to such an extent that the number of the organisms adhering to a firm surface greatly surpasses the number of the free swimming, planktonic organisms in the surrounding liquid media[7]. First Identified in late 1960s by Nealson et al, discovered in Vibrio fischeri and Vibrio harveyii, V. fischeri isa symbiotic microbiota of Howaiian bobtail squid light organ[7]. Hawaiian bobtail squid is a nocturnal hunter living in, as its name suggests, clear Hawaiian shallow coastal waters. Its light organ houses bioluminescent bacteria, Aliivibrio scheri, producing the right amount of light during the night, squid's active hours thus camouflaging their host[1].(Figure- 1)

V. Harveyi is a free living marine bacterium.

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Bioluminescence in Vibrio is control via Quorum sensing (QS). Lux operon was discovered as QS regulatory coding region.

It was further confirmed that bacteria prefer living in complex surface-associated communities which were later named biofilms.

Biofilm can be defined as an aggregation of one or more groups of different microorganisms, embedded in a self-produced matrix and adhering to a firm surface. Bacteria can form them on the greatest variety of surface conditions like living or non-living, in humid natural conditions, on medical equipment and living tissue, but also in the most extreme living conditions. Some of the recent studies have stated that bacterial biofilms even found in the extreme subzero temperatures of the Antarctic seawaters, thermal waters ranging from 35 to 50 degrees Celsius andin conditions of extreme acidity, high metal content and lack of nutrients etc. 15-20% of bacterial colonies are of biofilm volume while the rest is an extra cellular polymeric substance, (EPS matrix). In this EPS matrix the colonies are embedded, it is a mixture of different natural polymers, primarily polysaccharides, but also a variety of proteins, glycoproteins, glycolipids, and also nucleic acids. e surrounding matrix has many important roles in the lives of its inhabitants[1]. It can be representing as a place of a safe haven for the bacterial colonies. Quorum sensing is a way of communication within a bacterial species, whereas competitive or cooperative signaling can occur between groups of bacteria or between bacteria and the host. These bacterial systems are often integrated into complex, multilayered signal transduction networks which can control numerous multicellular behaviors, including biofilm formation and other virulence traits etc. The deeper understanding of microbial cell communication shed light on the complexities of the host-microbe relationship and this may lead to novel therapeutic applications[1].

Biofilms can be formed from a single-species bacterial community or, which is more typical, represent a community derived from several different microbial species living an interdependent lifestyle. Dental plaque biofilm and biofilm in periodontal diseases are best described multi-species biofilms. Their formation on non-shedding surfaces like teeth, prosthodontic appliances subsequently enables the formation of stable and complex bacterial communities, characterizes these biofilms.

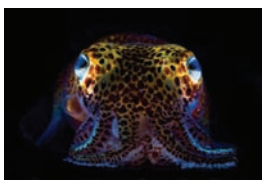

Figure 1: The Hawaiian Bobtail Squid

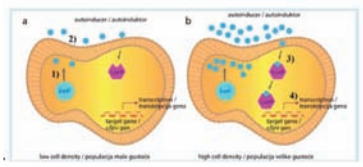

Figure 2; Quorum sensing mechanism – from signaling molecule production to gene transcription

Quorum sensing

It means that minimum number of members who were assembled to make a decision. Bacteria that may be either as an individual single-celled organisms or as multicellular populations. Bacteria exhibit these behaviors by secreting some of the chemical molecules i.e., chemically "talking" to one another through a process called quorum sensing. Quorum sensing involves the production, release, and community-wide sensing of chemical molecules called auto inducers(AI) which modulate gene expression, and ultimately controls bacterial behavior, coordination etc.

Quorum sensing can be divided into 4 main steps:

1) Intracellular synthesis of the signal molecules,

2) Molecules secretion, either actively or passively,

3) Detection of the signaling molecule and its binding to an inducer

4) Activation of gene transcription[8] (Figure 2)

Quorum sensing in Gram-positive bacteria

Unlike Gram-negative bacteria, Gram-positive bacteria use modified oligopeptides in the role of auto inducers which are detected by two-component membrane bound histidine kinase receptors[8]

Autoinducer-2 (AI-2) quorum sensing

While both Gram-negative and Gram-positive bacteria possess a certain quorum sensing system which enables them to communicate within their own species, to live in a complex biofilm, in a heterogenic community and it requires communication and behavior co-ordination in a common language. Autoinducer-2 represents a universal auto inducer molecule that enables inter-species communication and was first discovered as the auto inducer of the second quorum sensing system in the marine bacteria Vibrio harveyi[8]

Types of Auto inducers

In bacterial quorum sensing microbial derived

signaling molecules act as auto inducers. The Gram-negative bacteria use fatty acid derivatives called Homoserine Lactones (HSLs) whose synthesis is dependent on LuxI homolog or LuxR homolog encoding a transcriptional activator protein responsible for detection of the cognate HSL and the resulting gene expression which results in phenotypic changes. More than 30 species of Gram-negative bacteria use HSL derivatives for the control of the cell density and hence the quorum sensing phenomenon occurs and they communicate each other[1].

The Gram-positive bacteria use amino acids and short peptide derivatives for quorum sensing.

(1) Acyl Homoserine Lactone molecules

The AHL signal molecules from different bacteria are related in structure, but differ in the nature of the acyl side chain moieties attached to them. The acyl group can vary from 4 to 14 carbons depending on the auto inducer. It also possesses one hydroxyl group, a carbonyl group, it is either fully saturated or itcontains a single carbon-carbon double bond. A significant number of microbial acyl HSLs have even number of carbons in their acyl side chains whichare synthesized by different bacterial genera. Many of these bacterial species can produce more than one type of Acyl Homoserine Lactone and the type of acyl HSL produced by a particular species can be strain dependent[1].

(2) Synthesis of Auto inducers

The Homoserines found in bacteria are the intermediates of the methionine-lysine-threonine bio synthetic pathway. S-adenosylmethionine (SAM) is one of the intermediates of methionine/homocysteine pathway

Quorum sensing and the periodontal pathogens

Although several studies conducted to characterize quorum sensing systems and quorum sensing coordinated gene expression in periodontal pathogens have mostly been limited to Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis, several reports also identified luxS homologous genes in other pathogens, Fusobacterium nucleatum, Prevotel la intermedia and Eikenella corrodens[8]

Acyl homoserine lactone (AHL)-mediated quorum sensing system, similar to the one of Aliivibrio scheri, has not been found in periodontal pathogens so far[8].

Periodontitis disease onset and progression is now associated with population shift within the microbial bio film community. Periodontal pathogens are overpresented in samples taken from diseased places, whereas they represent a significantly small portion of the total species in healthy sites. The increased complexity and shifts in the microbial community are the result of species association and intra and inter-species communication[2].

A study done by Kochl et al and Sam et al[3] stated that various plants, algae and fungi which produces molecules that can have the capability to inhibit quorum sensing in bacteria, some of them are turmeric, garlic, citrus flavonoids, red marine algae, grape fruit extract, nut meg, Sweet basil, clove extractetc.

Conclusion

Many oral bacterias like Porphyromonas gingivalis, Actinomycetum comitansetc. communicate and co-ordinate their pattern and behavior through this quorum sensing. So, to disrupt bacterial biofilm we need to break this quorum sensing. These methods along with mechanical plaque removal measures, daily oral hygiene practices etc. may help to reduce periodontal disease severity. Many of the plants have quorum quenching potential, use of such plants,i.e., phytomedicine which may be beneficial. This offers an effective alternative method and reduces risk of antibiotic resistance[8]

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