Index Copernicus ID 6818
ISSN 2231- 1823

Journal of the Society of Periodontists and Implantologists of Kerala

JSPIK

Volume 9 • Issue 1 • MARCH 2017

www.spik.in

Vol. 9 | Issue 1 | March 2017
Index Copernicus ID 6818
ISSN 2231-1823

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President's message

Dentistry has been evolving rapidly, developing new concepts, and expanding fresh avenue for research. So it is very important to show an increase emphasis on evidence based research. Every practitioner/Post Graduate Students should have a sound knowledge and technique, good skill and critical thinking about the current advances, research and the importance of Medical emergency in Dental practice.

JSPIK has always been the mirror of knowledge to the Periodontists. As the Post Graduate students of Periodontics in and around Kerala is increasing in large number, SPIK always have given the Post Graduate Students, a platform for gaining scientific knowledge, as well as presenting papers and posters as a part of their Post Graduate Programme.

We have successfully completed midterm conference in Mahe Institute of Dental Sciences & Hospital with active participation of life and associate members. The conference was well appreciated and highlights of this conference was that the sessions were handled by our own SPIK life members in an exemplary manner.

Being the decennial year of Society of Periodontists and Implantologists of Kerala, the head office had decided to make the registration fee, free for the 10th annual conference for the benefit of life and associate members.

All preparations are going in full swing at Calicut and I request all the life and associate members to participate for the same and make the conference a memorable one.

Dr.Anil Melath
President SPIK

Secretary's Message

Dear Members,

Greetings from JSPIK.

It is heartening to see yet another issue of JSPIK. Although there had been few pressing problems regarding the journal which thankfully have been sorted out. Though we have converted the publication into an ejournal, the invitation for articles received a tremendous response. All the contributions were subjected to a blind scrutiny by peer reviewers before acceptance for publication. Indeed, the variety and completeness of the scientific content is commendable. Hope it will be a valuable read.

Dr. Vivek Narayan
Secretary , SPIK

Interdisciplinary Management of Grade 2 Mobile Anterior Tooth With Class I Gingival Recession- A case report

Anahita Punj[1], Amitha Ramesh[2], Santhosh Shenoy B[3], Biju Thomas[4]

Introduction

Tooth mobility is defined as the displacement of a tooth away from its normal position when a light force is applied[1]. It is one of the primary concerns of a patient with periodontal disease as it interferes with function, aesthetics and the potential risk of losing the tooth if not treated in time. There are a number of causative factors for mobility of tooth, which range from trauma, inflammatory periodontal disease, trauma from occlusion, parafunctional habits etc. Tooth mobility if detected early can be managed. To quantify the degree of tooth mobility, a number of classifications have been used. The most commonly used is the Miller's classification with a scoring of 0 to 3.[2]

Tooth mobility arising as a result of periodontal disease can lead to occlusal instability, impaired oral hygiene which in turn can exacerbate periodontitis due to plaque accumulation, disturbed mastication and secondary occlusal trauma.[3]

Occlusal trauma is defined as an injury to the attachment apparatus or the tooth itself as a result of excessive occlusal forces. It can be classified as primary, secondary or combined. Primary occlusal trauma occurs as a result of excessive occlusal forces, secondary occlusal trauma results due to the application of normal occlusal forces on teeth with inadequate periodontal support and combined occlusal trauma is a result of both abnormal occlusal forces and inadequate periodontal support.[4]

Gingival recession is the apical displacement of the gingival margin with respect to the cemento-enamel junction. The various etiologic factors which can result in gingival recession are: inflammatory periodontal disease, trauma from faulty tooth brushing, occlusal trauma, high frenal attachment, tooth malposition or root prominence leading to the thinning of bony plate, orthodontic tooth movement in unusual direction, underlying alveolar dehiscence and thin gingival biotype. Miller's classification is used most commonly to classify gingival recession as class I, II, III, IV.[5-7]

Historically, it has been suggested that excessive occlusal force might be a causative factor in causing gingival recession. Occlusal forces were, at one time, implicated in the progression of periodontal disease. However, there is very little conclusive evidence to support this statement. At present, there is a consensus that trauma from occlusion may be a co–destructive factor in periodontal destruction, especially affecting the supporting alveolar bone.[8-9]

Thus, a case report of a patient with grade 2 mobile lower anterior tooth due to trauma from occlusion and class I gingival recession and its management is presented.

Case Report

A 35-year old female patient reported to the department of Periodontics, with the chief complaint of loosening of the lower left front tooth (31) since 1 month which aggravated while chewing food. The patient reported no history of trauma. The patient reported restoration of the same tooth one week back. The patient was systemically healthy, non-smoker and gave negative history of traumatizing habit like clenching/grinding, had no masticatory muscle tenderness, no biting habits (lip/tongue/cheek).

Intraoral examination revealed class I gingival recession, inadequate width of attached gingiva, grade II mobility in relation to 31 and grade I mobility in relation to 41, 32 (Fig 1).

There were open contacts present in relation to 11, 12; 21, 22; 31, 32; 41, 42 and crossbite in relation to

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12 and 22. Crowding was present in relation to upper anterior teeth and wear facets were present in relation to 31, 32, 41 and 42. There was trauma from occlusion (fremitus test ++) present in relation to 21 and 31, 32. Pulp vitality testing revealed 31 as non-vital.

Based on the above mentioned findings the provisional diagnosis was given as chronic localised periodontitis in relation to 31,32,41,42, chronic irreversible pulpitis in relation to 31 and primary trauma from occlusion. The overall prognosis and the individual prognosis in relation to 31 was good.

The treatment plan included phase I therapy, followed by splinting of the mobile tooth, occlusal equilibration to relieve trauma from occlusion, root canal treatment, lateral pedicle flap for coverage of recession defect. Phase I therapy – etiotropic phase, included patient education and motivation, scaling and root planning and use of chlorhexidine 0.2% mouthwash twice daily. Next, root canal treatment for 31 was carried out (Fig 2).

Occlusal equilibration was done to relieve trauma and this was followed by labial splinting (Fig 3) of the mobile tooth to the adjacent teeth using a fiber-reinforced composite splint.

Evaluation of response to phase I therapy was carried out. After 17 days of splinting, lateral pedicle flap surgery (Fig 4) was carried out for root coverage of 31. Frenotomy was done for the mandibular labial frenum to avoid tension on the flap for lateral pedicle graft. Procedure for lateral pedicle flap included anesthetizing the mandibular anterior left region using 2% lignocaine, giving a v-shaped incision using number 15 B.P blade to prepare the recipient site at 31. Then a full thickness flap was raised from the donor site (32 to 33 region). This was followed by a vertical releasing incision at the mesial line angle of 33. A cut back incision was also given and the flap was slided to cover the recipient site. The flap was then secured over the recipient site using 3-0 non-resorbable suture. The suturing was done using the composite stent as an anchor to maintain the coronal position of the displaced flap in relation to 31, and interrupted sutures were given to approximate the releasing incision. The area was covered with tin foil and periodontal dressing was given. The suture removal was done after 10 days and healing was uneventful (Fig 5). The patient was kept on recall and maintenance (Fig 6).

After 5 months splinting was removed, the tooth mobility decreased from grade 2 to 0, complete root coverage of 31 was obtained, recession depth decreased from 3 mm to 0 mm, successful root canal treatment was achieved in relation to 31 (Fig 7).

Discussion

Presence of traumatic occlusion can result in the

Figure 1. Preoperative View

Figure 2. Completion of root canal treatment of 31

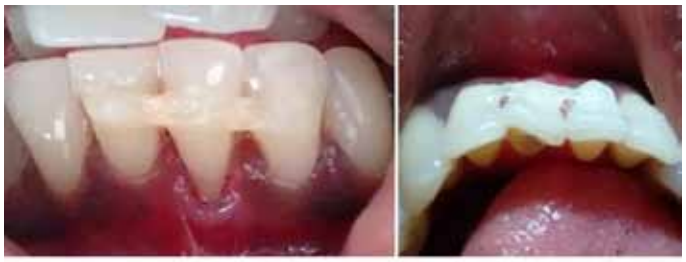

Figure 3. Splinting of mobile tooth

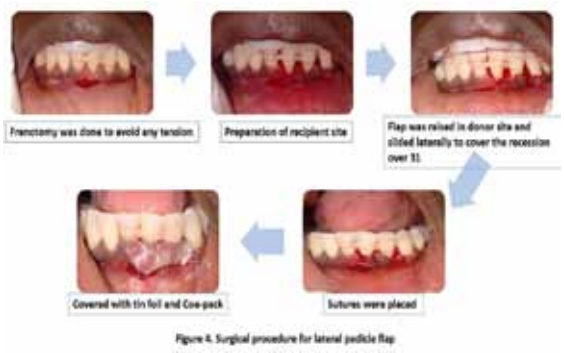

Figure 4. Surgical procedure for lateral pedicle flap

jiggling forces which can lead to further periodontal destruction. Malocclusion and abnormal tooth position are now recognized as potential contributors to the disease process when they cause occlusal traumatism.[10]

As per this case report, malocclusion over a period of time has led to loss of buccal bony plate and gingival recession. Andrade RN et al[11] 2014, highlighted the influence of malocclusion on gingival recession. Kundapur et al[12] 2009, found no conclusive relationship between fremitus and tooth wear with gingival recession. However, the sign of tooth mobility, which is a feature of trauma from occlusion, appeared to be a predictor of positive association with gingival recession.

Although, correction of the malocclusion should be the mainstay of treatment, the patient was unwilling for orthodontic treatment initially and wanted an alternative treatment plan.

Phase I therapy was carried to prevent plaque associated periodontal attachment loss. As per Lindhe and Nyman[13] 1977, tooth mobility can be reduced by occlusal adjustment/equilibration and/or splinting. Borges et al[14] 2011, reported that occlusal adjustment is recommended for the treatment of periodontal injuries caused by traumatic occlusion.

It is an accepted practice to splint mobile teeth, particularly lower incisors, to maintain the patient's natural dentition as long as possible.[15] In this case 31 was splinted with 32,41,42 to reduce jiggling forces on 31 and redistribute forces, to stabilize 31, to prevent movement during endodontic treatment and allow periapical healing to occur, to facilitate uneventful healing of root coverage procedure planned for 31.[16,17]

Extracoronal Splinting was done labially to allow access opening of 31 and fiber reinforced composite splint was used for its esthetics, good adaptability and strength. Azodo et al[3] 2016, highlighted splinting is a well-accepted integral part of holistic periodontal treatment which results in morale boost, improved patient comfort, and oral functions.

The etiology of non-vitality was not clear and could be attributed to trauma from occlusion. Jafari et al[18] 2016, reported the persistence of periapical lesion after appropriate endodontic treatment and surgery due to trauma from occlusion. Indramohan et al[19] 2011, reported that traumatic occlusion was identified as the etiology of a radicular cyst. After completion of endodontic treatment, sufficient time was given to allow periapical healing.

Coverage of class I recession in 31 was planned using lateral pedicle graft procedure as adequate amount of attached gingiva was present in the adjacent sites. Moreover, 21 was rotated and the patient was not willing for autogenous graft procedures which required a second surgical donor site.

The treatment plan incorporated is based on the findings of various authors, such as Ustun et al [20] 2008, who presented a multidisciplinary approach for severe gingival recession caused by traumatic occlusion and mucogingival stress and highlighted the importance of early diagnosis of trauma from occlusion and its treatment.

Limitations of this case report include a short follow up period and the inability to correct malocclusion.

Conclusion

Interdisciplinary approach of splinting, occlusal equilibration, endodontic treatment and lateral pedicle graft procedure led to management of tooth mobility and gingival recession arising as a result from trauma from occlusion.

The results suggest successful management of a lower anterior mobile tooth with recession,

Figure 5. Postoperative picture after suture removal

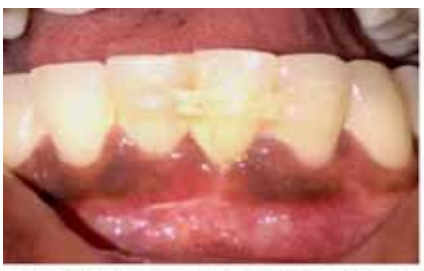

Figure 6. Postoperative view after 2 months

Figure 7. Postoperative view after 5 months

maintaining oral hygiene and esthetics. The possibility of dentin hypersensitivity and future loss of tooth was also eliminated.

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Bullous Pemphioid

Bhat Ramesh Amitha[1], Khandelwal Ankita[2], Shetty Pushparaj[3], Thomas Biju[4]

ABSTRACT

Bullous Pemphigoid (BP) is an autoimmune blistering disease and occurs very rarely. It is a sub epithelial vesiculobullous disorder and generally targets the older population. It usually occurs on flexural areas of the skin but in few cases the lesions might occur in the oral cavity before the skin. These are fluid filled blisters which are usually tense and don't rupture easily. They might be associated with desquamation and erythematous area. When they occur on the skin they might also be pruritic in nature. Diagnosis is based on the histopathological examination. Treatment is based on the extent and severity of the disease. The most common treatment is topical and systemic corticosteroids but in severe cases immunosuppressive drugs are also given.

Key-words: Bullous Pemphigoid, Oral cavity, Sub epithelial blister formation, Steroids, Vesiculobullous lesion

Key Messages : Since few lesions precede the oral cavity before skin a dentist should be vigilant enough to identify the disorder at an early stage and provide a suitable treatment in integration with other departments. This will help in improving the prognosis of the disease and enhance the quality of the life.

Introduction

Pemphigoid is the terminology applied to a group of immune-mediated blistering diseases. These diseases are characterized by the production of autoantibodies to the attachment apparatus between the epithelium and the underlying connective tissue. The destruction of these structures results in the formation of sub epithelial vesicles.[1] It occurs at a rate of 10 cases per million population per year and is associated with significant morbidity.[2]

There are different types of pemphigoid which vary in terms of where on the body the blistering occurs. The most common types of pemphigoid are bullous pemphigoid and cicatricial or mucous membrane pemphigoid.[2]

Case report

A 33 year old female patient reported to the Department of Periodontics with a chief complaint of fluid filled blisters in her mouth which would rupture and form ulcers. The patient reported that the blisters after rupturing would recur in a different location. She also gave a history of burning sensation in her mouth on consumption of spicy food but there was no associated pain. Medical history of the patient revealed hypothyroidism since 7 years and she was under medication for the same.

On clinical examination there was presence of a vesicle in relation to attached gingiva of 36 measuring around 2 *2. The surrounding area was erythematous and rubbing with the gauze showed desquamation of the gingiva (Fig. 1). On palpation the lesion was slightly tender. The submandibular and sub mental lymph

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nodes were not palpable. There were no associated skin lesions. Nikolksy's sign was noted to be positive.

Based on the clinical features a provisional diagnosis of chronic desquamative gingivitis was made. An incisional biopsy was performed in the same region and given for histopathological examination with the patient's consent (Fig. 2).

The histopathological examination was done under a magnification of 40X and it revealed a stratified squamous keratinised epithelium. A split was seen between the surface epithelium and underlying connective tissue (sub-epithelial split). Also there was presence of mild chronic inflammatory cell infiltrate and extravasated RBC (Fig. 3). Overall histopathological features were suggestive of Bullous Pemphigoid.

Discussion

Bullous pemphigoid represents the most common autoimmune sub epidermal blistering disease and is seen generally in elderly; among the 5th– 7th decade of life. There is no known ethnic or racial predilection. Bullous Pemphigoid has been related with diseases like diabetes mellitus, pernicious anaemia, chronic inflammatory skin diseases such as lichen planus.[2]

Oral manifestations in Pemphigoid are relatively common (40%) and they follow cutaneous eruptions. Vesicles and bullae occur on the skin and oral cavity and might be preceded by erythematous papular eruption. These bullae are tense and fluid filled and rupture leaving erosions which usually heal without scarring. These lesions can affect the buccal mucosa, gingiva, palate, tongue and lower lip. On gingiva they can also occur as desquamative gingivitis. The bullae formed in Pemphigoid are more stable and don't rupture as easily as in pemphigus. Skin lesions are characterised by a trunk and limb distribution.[2-3]

Patients with BP have circulating IgG auto antibodies against majorly two hemidesmosome proteins, BP (Bullous Pemphigoid) 230 (also known as BPAG1) and BP180. BP230 promotes the association of hemi desmosomes with keratin intermediate filaments whereas BP180 is a type II, transmembrane collagen that is associated with hemidesmosome–anchoring filament complexes. BP 180 is thought to be the key antigen because the antibodies attach to this protein. Subsequent to binding of circulating autoantibodies to tissue antigens, a series of events occurs, one of which is complement activation. This attracts neutrophils and eosinophils to the basement membrane zone. These cells then release lysosomal proteases, which in turn participate in degradation of the basement membrane attachment complex. The final event is tissue separation at the epithelium-connective tissue interface.[3]

The diagnosis of bullous pemphigoid is confirmed by histologic and immunopathologic investigations. Usually, an incisional biopsy is taken and sent for histopathological examination. Histopathological examination reveals separation of the epithelium from connective tissue at basement membrane zone. Presence of both acute and chronic inflammatory cells are seen and the presence of eosinophils is considered characteristic[4]. In the present case report the histopathological examination revealed similar features.

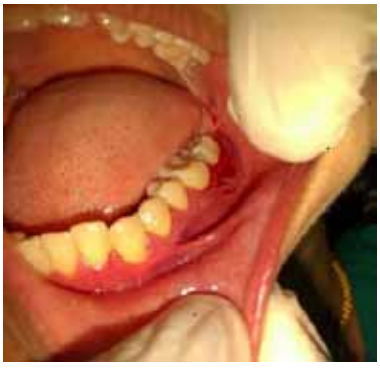

Figure 1-- Desquamation of the gingiva on rubbing with a gauze wrt 36, 37

Figure 2-Tissue specimen after performing incisional biopsy

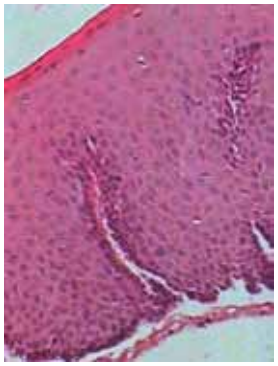

Figure 3-Histopathological picture revealing a sub-epithelial split

Bullous pemphigoid can be treated successfully with topical corticosteroids alone. As the disease becomes extensive systemic anti-inflammatory and immunosuppressive agents are required for complete resolution of the disease. Systemic prednisone is usually the choice of drug but some patients don't respond to it. Alternative drug choices include Azathioprine, Dapsone and Cyclophosphamide.Most of the patients' exhibit remission within 2 to 3 years but individual patient factors has to be kept in mind while deciding the prognosis of the disease.[5]

In this case the patient was kept on a kept on a symptomatic therapy of antimicrobial and anaesthetic (Dyclonine 1%) rinse, topical anaesthetic (Choline salicylate and Lignocaine) and topical corticosteroid gel (Triamcinolone Acetonide). The patient was asked to consume vitamin B and C supplements along with antioxidant Lycopene (OS- Fibro) .The patient was kept on a follow up of 3 months and the recurrence of the lesions was noticed every 10 days. Post 3 months the patient was referred to a dermatologist for systemic therapy.

Conclusion

In the present case study, considering the clinical and histopathological features, a diagnosis of Bullous Pemphigoid was made. It is extremely critical to achieve a confirmative diagnosis in vesiculobullous lesions because these diseases may present with high morbidity and mortality. Most of the times, these diseases require management by interdisciplinary approach with the help of a dentist, physician and a dermatologist.[6] Dentists can play a major role in identifying these diseases as in some cases oral lesions might precede skin lesions and hence diagnosing at an early stage might help to improve the overall prognosis of the disease and aid in improving quality of life.

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History of Periodontology –An overview

The longer you can look back, the farther you can look forward. *-Winston Churchill,1944*

Priyada C. [1], Bindu R. Nayar[2]

ABSTRACT

Studies in paleopathology have indicated that destructive periodontal diseases has affected early humans of diverse culture for which treatment was been provided. However methodic, carefully reasoned therapeutic discussion did not exist until the Arabic surgical treatises of the middle ages. The period of Renaissance owns many literary works on dentistry such as *Zene Artzney* and *Libellus de Dentibus*. Eighteenth century is credited with the contribution from Pierre Fauchard, the father of dentistry. Nineteenth century is embarked by the discovery of anesthetia, X-rays, proposal of germ theory of disease and contributions from John W.Riggs -first specialist in periodontology. During the first third of the twentieth century, periodontics flourished in central Europe, with two major centers of excellence: Vienna and Berlin. After World War II, United States and Scandinavia took leading roles in basic and clinical periodontal research with major advances made in the fields of experimental pathology, microbiology, immunology, and therapy that led to growth of this speciality as to what is seen today .

Keywords: History of Periodontology, Riggs Disease

Introduction

Gingival and periodontal diseases in their various forms have affected man since the dawn of history. The earliest historical records of ancient Egypt and pre Columbian America reveals an awareness of periodontal diseases and the need of treatment. However methodic, carefully reasoned therapeutic discussion did not exist until the Arabic surgical treatises of the middle ages, and modern treatment with illustrated text and sophisticated instrumentation, did not develop until the era of Pierre Fauchard, in eighteenth century. Dental medicine, especially periodontics, has changed dramatically since then due to extensive research works. Periodontics was recognized as a specialty of dentistry by the American Dental Association in 1947. From then on scope and development of periodontics as an individual speciality in dentistry has expanded .

The Prehistoric Era and Early Civilizations

Paleostomatology studies show evidences of dental caries,enamel hypoplasia,chronic periodontal diseases, temperomandibular joint dysfunctions,jaw cyst, tumours and implants in skeletal remains (Fig 1). Tablets of early Mesopotamian civilizations, starting with Sumerians in 3200 BC and ending with, Babylonians and Assyrians showed evidences of using various herbal medications as remedy for many diseases. The most significant work of Babylonia-Assyria is the Code of Laws of Hammurabi that established penalities for unsuccessful treatment from the surgeons[1].

Periodontal diseases was the most common of all diseases found in the embalmed bodies of the ancient Egyptians of 3000 BC. Two important Egyptian medicine papyri worthy of mention are Edwin Smith papyrus (surgical treatise) and Ebers papyrus (medical

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treatise 1600 BC). Hebrew medicine records evident from Talmudic writings describes usage of fine salt as dentifrices and qesem (tooth picks)[1].

The Classical and Medieval Ages

Among the ancient Greeks –Hippocrates of Cos (460-377 BC) the father of Modern Medicine, believed that the "*inflammation of gums is caused by accumulation of pituita or calculus"*Among Romans,Cornelius Celsus (25 BC -50 AD) authored *De Re Medicina* and offered treatment for a number of periodontal conditions. Paulof Aegina (625-690 AD) differentiated between "*epulis*"(fleshy growth on gums) and "*parulis*"(abscess of gums) [1].

The decline and fall of the Roman empire that plunged Europe into darkness was accompanied by the rise of Islam and the Golden age of Arabic science and medicine. Abulcasis (936-1013) in his medical encyclopedia: *al –Tasrif* (The method) describes etiologic role of calculus, detail scaling technique using a set of instruments he developed and,performed splinting of loose teeth with gold wire. Avicenna (980-1037) wrote a comprehensive medical text *Canon treatise*, that described the use of material medica for oral and periodontal diseases and rarely resorted to surgery[2].

The Modern Era

The period of Renaissance spanned three centuries, starting from fourteenth century in Italy and continuing into seventeenth century. Andreas Vesalius-Father of Modern Anatomy (1514-1564), wrote a magnificent book on anatomy (*On the Fabric of the Human Body)* that included many excellent illustrations including gross anatomy of teeth.

The first book in the common language of German devoted to dental practice, was "*Artzney Buchlein* or *Zene Artzney*"("Medicine of the Teeth"), by Michael Blum in 1530 .The Frenchman Ambroise Paré (1509-1590) developed wound dressings, gingivectomy techniques and a set of scalers. Paracelsus (1493-1541) developed the: *doctrine of tartar* and authored Grosse

Fig. 1. Mayan mandible 6th century with implanted pieces of shell in place of mandibular incisors

Fig. 2. Libellus de Dentibus ("A Little Treatise on theTeeth")

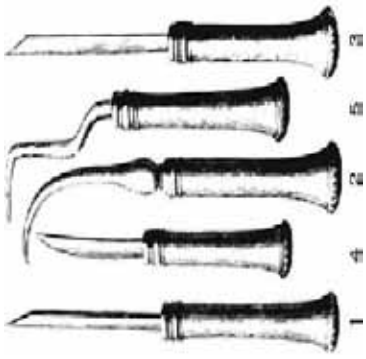


Fig. 3.The five types of instruments used by Fauchard for detaching tartar from the teeth: 1, chisel; 2, parrot beak; 3, graver; 4, convex blade; 5, Z-shaped hook

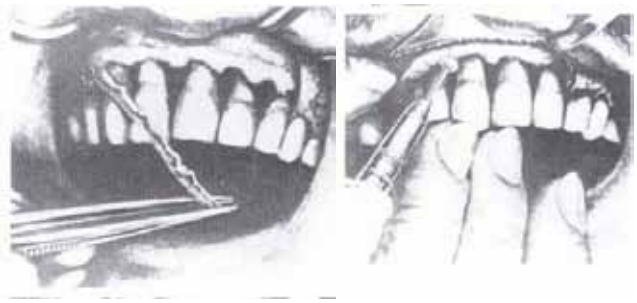


Fig. 4. Neumann 's flap technique

Fig. 5.Grace Rogers Spalding & Gillette Hayden

Fig. 6.Oldest mouthwash 115 years old -Listerine

Wundartzney (Large Book of Treatment of Wounds).[1]

Bartholomeus Eustachius (1520-1574) of Rome wrote *Libellus de Dentibus* ("A Little Treatise on the Teeth"), the first original book about the teeth that included a description of the periodontal diseases and their treatment (Fig 2).

Seventeenth century embarks the introduction of microscopy, Anton van Leeuwenhoek (1632-1723) observed oral spirochetes and bacilli and performed antiplaque experiments using vinegar in his own mouth[2].

The Eighteenth Century

Modern dentistry essentially developed in eighteenth century Europe, particularly France and England. Pierre Fauchard, of Brittany (1678), is regarded as the father of the dental profession. His book, *Le Chirurgien Dentiste* (The Surgeon Dentist or Treatise on the Teeth -1728) covered all aspects of dental practice, including periodontics (Fig 3). John Hunter (1728-1793) of England,authored *The Natural History of the Human Teeth* in 1771 that offered remarkably clear illustrations of teeth and supporting structures, and described features of periodontal diseases[1].

Nineteenth Century

In the second half of nineteenth century three major developments in medical sciences had particular impact on periodontics- discovery of anesthesia, germ theory of disease and discovery of X-rays[2].

There is a disagreement on who discovered anesthetia, Crawford Long (1815-1878) or Horace Wells (1813-1848) and William Morton (1819-1868) whom discovered general anesthetic effects of nitrous oxide and ether, respectively. The discovery of Local anesthesia using cocaine was developed by the Vienna ophthalmologist Carl Köller (1857-1944). Later with addition of adrenaline, which was discovered separately by Jokichi Takamine and Thomas Bell Aldrich, local anesthesia was born[2].

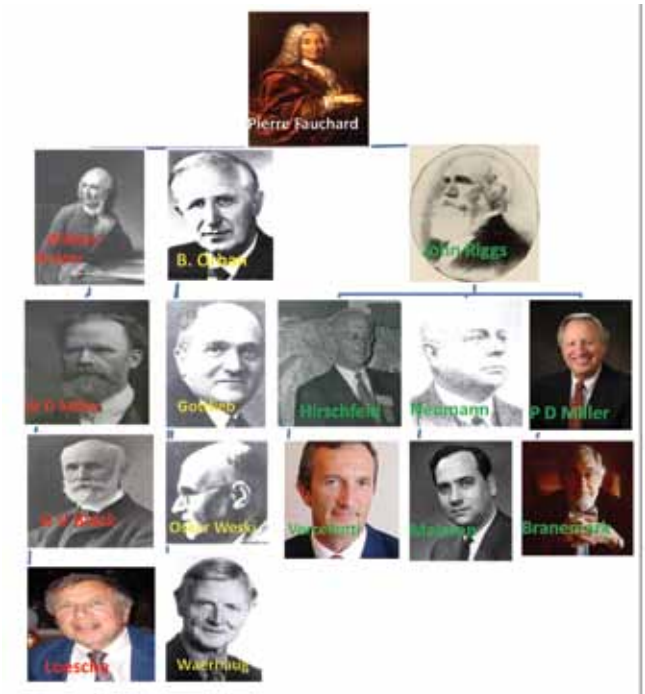
The second scientific breakthrough was made by the French chemist Louis Pasteur (1822-1895), who established the *germ theory* of disease. The concepts of Pasteur were transferred to clinical and surgical practice by Joseph Lister (1827-1912) of England, and thus the era of antisepsis—and later, asepsis—in surgery was born[1].

The first true oral microbiologist was an American, Willoughby D. Miller (1853-1907) in his classic book, *The Microorganisms of the Human Mouth*-1890, described the features of periodontal disease and proposed that it is caused by a complex array of bacteria rather than a single bacterium. James Leon Williams (1897) recognized the existence of dental plaque, and in 1899 G.V. Black (1836-1915) coined the term *gelatinous microbial plaque*.

A third scientific finding that changed the practice of dentistry in general and of periodontics was the discovery of radiographs in 1895, by the German physicist Wilhelm Conrad Röntgen (1845-1923).

Leonard Koecker (1785-1850) in 1821,described the inflammatory changes on the gingiva and associated presence of calculus and mentioned the need for oral hygiene. Koecker was an early advocate of the "odontogenic focal infection" theory, and recommended extraction of all severely involved teeth to prevent systemic infections.

Levi Spear Parmly (1790-1859) from New Orleans is considered the father of oral hygiene and invented dental floss. During the mid-nineteenth century, John W. Riggs (1811-1885) was the leading authority on periodontal disease and its treatment in the United States; at the time, periodontitis was known



as "*Riggs' disease*". Riggs seems to have been the first individual to limit his practice to periodontics and can be considered the first specialist in this field. Riggs was a strong proponent of conservative approach to periodontal therapy and he developed the concept of oral prophylaxis and opposed surgery.

The Russian N.N. Znamensky described complex interaction of local and systemic factors in the etiology of periodontal disease and described its histopathology for the first time in 1902[3]. In 1778, Hunter described clinical features of necrotizing ulcerative gingivitis and Hyacinthe Jean Vincent (1862-1950),described the associated spirillum and fusiform bacilli,which later became known as Vincent's angina.

The Twentieth Century

During the first third of the twentieth century, periodontics flourished in central Europe, with two major centers of excellence: Vienna and Berlin[2].

The Vienna school developed the basic histopathologic concepts on which modern periodontics was built. The major representatives from this group was Bernhard Gottlieb (introduced concept of epithelial attachment) (1885-1950), Rudolph Kronfeld (1901-1940), Joseph P. Weinmann (1889-1960), and Harry Sicher (1889-1974).

The Berlin group consisted mostly of clinical scientists who developed and refined the surgical approach to periodontal therapy. Prominent in this group were Oskar Weski, Robert Neumann, Leonard Widman of Swedan and Cieszinski of Poland (Fig 4).

Localist vs Generalists

Those who hypothesized on the causes of periodontal diseases can be grouped as the Localists and Generalists. The Localists believed that primary causes of periodontal disease are intra-oral and intraoral interventions can cure them. Fauchard, Harald Loe, Riggs were among them. The Generalists believed that primary causes of periodontal disease are remote from the oral cavity and that periodontal disease is only amenable if the remote causes are intervened upon. Miller, Maurice Roy, (senility), Bergstrom (smoking), Cheraskin (vit C) are among them. The localists were dominant in the second half of the 20th century& the remote causes became minimized in importance and periodontics centered on plaque control[4].

In the United States, before World War II, important contributions to periodontal surgery were made by A. Zentler, J. Zemsky, G.V. Black, O. Kirkland, A.W. Ward, A.B. Crane, H. Kaplan, and others. In 1913, Alfred Fones (1869-1938) opened the first school for dental hygienists in Bridgeport, Connecticut.

After World War II

The United States and Scandinavia took leading roles in basic and clinical periodontal research during and after the 1950s, led by Irving Glickman (1914-1972), Henry M. Goldman (1911-1991), Balint J. Orban (1899-1960), Sigurd P. Ramfjord (1911-1997), and Helmut A. Zander (1912-1991).The leading figure of the Scandinavian group was Jens Waerhaug (1907-1980) of Oslo, Norway, whose dissertation entitled *The Gingival Pocket* (1952) opened a new era in the understanding of the biology of the periodontium and the management of periodontal problems.

Organised Periodontics

American Academy of Periodontology was founded in 1914 by two female periodontists, Grace Rogers Spalding & Gillette Hayden (Fig 5). It was initially named the American Academy of Oral Prophylaxis and Periodontology and later name was changed to the American Academy of Periodontology in 1919. The first president is Austin F Jamesin1915[9]. The German ARPA (1922) and European Federation of Periodontology in (1989) also contributed to development of periodontlogy[1].

Periodontology in India

Indian Medicine is divided into period of Vedas-Vedic period and Brahman period. The famous writings in medicine during the *vedic* period (1500 -800 BC) includes *Rig veda* & *Atharva veda* that mentions about diseases of teeth & herbal remedies. Brahman period includes famous writings on medicine- Susruta samhita and Charaka samhita. The pioneer periodontists in India include; Dr Tehmi M.S. Ginwalla, Dr Govind B Shankwalkar, Dr Lalit M Guglani. Indian Society of Periodontolgy (ISP) was founded in 1975.

Dr Tehmi M.S. Ginwalla (1976) was the first president of Indian Society of Periodontology. Dr Govind B Shankwalkar was the first post-graduate teacher in India to start M.D.S. course. Oral Hygeine

day is celebrated on August 1st to commemorate his birthday.

Future of Periodontology

The first step towards future of periodontology must be to rename it as the speciality of 'periodontal surgery and medicine'. We must begin to explore on the medical management of periodontal diseases and understand the association of periodontal diseases to systemic conditions. Periodontal surgery track will greatly advance implant dentistry .Computer assisted imaging, robotics, tissue engineering advances etc will contribute for the further development of this speciality .

Conclusion

Periodontology represents one of the younger specialties in dentistry and can be said to have had its birth as a specialty in 1914 with the organization of the American Academy of Periodontology. The goal of a study on the history of periodontology is to bring knowledge into possibilities to reinforce what one may already have learned in the field of periodontology, to examine one's current level of understanding in light of today's expanded knowledge base, and to look to the future with confidence that periodontics will play a major role in one's dental practice and will help address many of the problems cited recently.

MILESTONES IN DEVELOPMENT OF PERIODONTOLOGY**

First specialist in periodontology [1]	John Mankey Riggs (1811-1885)
Historical review on periodontal pathologies[2,3,4]	
1.Pyorrhea Alveolaris term introduced by	Alphonse Toirac 1823
2.Classification of clinical gingival conditions	Kinane &Lindhe 1997
3.Chronic diffuse desquamative gingivitis term	Prinz 1932
4."Gingivosis" term coined	Massler & Schour in 1947
5.Term "diffuse atrophy of alveolar bone" for aggressive periodontitis	Gottlieb 1923
6. "Periodontosis" term coined by	Massler & Schour in 1947
7.Trauma from Occlusion first described	Morris Karolyi 1901
8.Drug induced gingival enlargement first reported for phenytoin / nifedipine	Kimball 1939 / Lederman & Ramon 1980.
9.Classification of endo- perio lesions	Simon et al 1972
Historical review on periodontal pathogenesis[2,3,4]	
1. Non Specific & Specific plaque hypothesis	Walter Loesche 1976
2. Ecological plaque hypothesis	P D Marsh 1997
3. "Biofilm" term coined	Bill Costerton 1978
4.Six microbial complexes in periodontal diseases.	Socransky 1998
5.Quorumsensing in biofilms	Prosser 1999
6.The Keystone-Pathogen Hypothesis	Hajishengallis et al 2012
6.Theories of calculus formation Mineralisation theory Colloidal precipitation theory Salivary ph theory	Magitot 1878 Prinz 1921
7. GCF studies conducted	Hodge& Leung 1950
8.VSC s as cause of oral malodour	Waerhaug, Krasse and Brill 1950
9. Organoleptic rating	Tonzetich 1970
10.Concept of "radius of action"in osseous defects	Rosenberg & Mc Culloch 1992
11.Bone factor concept given by	Garant &Cho 1979 Irving Glickman 1951

Historical review of Non Surgical Periodontal Therapy[7,8]	
1.First toothbrush with bone handles &hog bristles designed.	William Addis 1780
2.Concept of oral prophylaxis	John W Riggs 1876
3.First powered toothbrush / Broxodent	Dr Philippe Guy Woog 1954
4. Manufacture of toothpaste in a collapsible tube	Dr Washington Shefûeld 1892
5. Listerine mouth wash	Joseph Lawrence & Jordan Wheat Lambert 1884
6.Chlorhexidine antiplaque properties	Loe & Schiott 1970
7.Fluoride mouthrinses	Bibby in 1946
8.One Stage Full Mouth Disinfection.	Quirynen et al 1995
9.Use of ultrasonics in periodontal therapy	Zinner 1955
10.Host Modulation Therapy	Williams 1990 & Golub 1992
11. Management of dentinal hypersensitivity	Trowbridge and Silver in 1995
12. Use of zinc chloride rinses for malodour	Schmidt & Tarbet 1978
Historical review of Surgical Periodontal therapy[3,5,6]	
1.Straight incision for gingivectomy	Robicsekof Vienna 1862
2.Scalloped incisions for gingivectomy	Zentler 1916
3. "Gingivectomy" term used firstly	Pickerell 1912
3.Use of electrocautery for gingivectomy	Coolidge 1838
4.Gingivoplasty	Goldman 1950
5.Subgingival curettage	Isadore Hirschfeld 1952
6.ENAP	Yukna et al 1976
7.Suturing & Suturing techniques	Ethicon 1985
8.Principles of flap surgery	Neumann 1912
9.Unrepositioned gingival flap	Cieszynski 1914
10.Widman flap technique	Leonard Widman 1916
11.Wards wondr Pack	Abraham Wesley Ward 1923
12.Semi /modified flap technique	Kirkland 1931
13.Apically repositioned flap	Friedman 1962
14.Distal wedge procedure	Robinson 1969
15.Modified Widman flap	Ramfjordand Nisslein 1974
16.Papilla preservation flap	Takei et al 1985, Cortellini 1995(modified)
17.Mucogingival surgery	Friedman 1957
18.Father of 'Periodontal plastic surgery'/introduced term	Preston D Miller 1993

19. Curtain procedure for anterior maxilla	Frisch in 1967
20.Described procedures frenectomy &frenotomy	Gottesgen 1953
20.Tissue barrier concept	Goldman & Cohen 1979
20.Free Soft tissue Autografts	Bjorn 1963
21.Laterally /Horizontally displaced flap	Grupe &Warren 1956
22.Double Papilla flap	Cohen & Ross 1968
23.Semilunar coronally repositioned flap	Tarnow 1986
24.Laterally moved coronally advanced flap	Zuchelli et al 2004
25. Sub epithelial Connective Tissue Graft	Langer & Langer 1985
26.Ostectomy & Osteoplasty technique	Friedman 1955
27. Grooving or Festooning	Ochsenbein 1958
28.Radicular blending.	Carranza 1984
29.Biologic width quantified	Garguilo et al 1961
30.Guided Tissue Regeneration concept	Gottlow 1986
31.Firstly performed GTR in human tooth.	Nyman 1982
32.Root Bio modification using Citric acid	Burdick 1975
33.Enamel Matrix Derived proteins for regeneration	Hammarstrom & Heijl 1997
34.Tissue engineering concept in periodontal therapy	Langer 1993
Evidence based practice in periodontology [10,11]	Alexia Antczak Bouckoms 1980
Father of microsurgery Periodontal microsurgery (Magnifying loupes)	Carl Nylen 1921Shanelec &Tibbets 1994
Father of piezosurgery	Tomaso Vercellotti 1997
First invented Ruby LaserFirst application of a laser to dental tissue	Theodore Maiman 1960Stern &Sognnaes in 1964
Father of implant dentistry/osseointegration concept[12]Fibroosseous integration[12]	Dr.P.Branemark 1978Charles Weiss 1980
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Guided Bone Regeneration – A review

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ABSTRACT

Guided bone regeneration, the most commonly used ridge augmentation procedure, is a surgical procedure that uses barrier membranes with or without particulate bone grafts or/and bone substitutes. There are two approaches of guided bone regeneration in implant therapy: Guided bone regeneration at implant placement (simultaneous approach) and before implant placement to increase the alveolar ridge or improve ridge morphology (staged approach). It has allowed for placement of restorations at a more ideal location in the oral cavity, thus improving esthetics and function. This paper reviews basic principles and materials used for guided bone regeneration.

Keywords: Ridge augmentation, regeneration, guided bone regeneration, barrier membranes, bone grafts.

Introduction

The use of titanium dental implants is considered a successful and largely predictable treatment option for partial and full edentulism.[1] Sufficient amount of bone at the implant recipient site to allow osseointegration of the endosseous implant surface is a key prerequisite. However, patients often require augmentation procedures to reconstruct lost bone and place implants in a prosthetically driven position. Any procedure designed to enlarge or increase the size, extent or quality of deformed residual ridge is known as ridge augmentation. Numerous surgical graft and implant procedures attempting to reconstruct a partially edentulous ridge or ridge defect have been described in the literature over the years. There are four methods to increase the rate of bone formation and to augment the bone volume[2]:

- Osteoinduction by the use of appropriate growth factors (Reddi AH,1981,1987; Urist MR.,1965)
- Osteoconduction where a grafting material serves as a scaffold for new bone growth (Buch F,1986; Reddi AH,1987)
- Distraction osteogenesis by which a fracture is surgically induced and the two fragments are then slowly pulled apart (Ilizarov GA. 1989)
- Guided bone regeneration (GBR) (Dahlin C, 1990) which allows spaces maintained by barrier membranes to be filled with new bone.

The procedures may be grouped according to the methods used for ridge augmentation as soft tissue augmentation procedures and hard tissue augmentation procedures.[2]

Classification of ridge augmentation:

I. Soft tissue augmentation

a. Pedicle graft procedure: Roll flap procedure

b. Free graft procedures:

c. Combined onlay interpositional graft

II. Hard tissue augmentation

a. Guided bone regeneration (GBR)

b. Distraction osteogenesis

c. Ridge splitting/ expansion technique

d. Tissue engineering

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Among these techniques, the best documented and most widely used method to augment bone in localized alveolar defects is GBR. Indications for GBR include ridge augmentation, socket preservation, treatment of peri-implantitis and treatment of dehiscence around implants.[3]

Principles of GBR

In 1988, Dahlin et al put forward the concept of GBR based on Melcher's guided tissue regeneration (GTR) principle. GBR is a surgical procedure involving the placement of a cell – occlusive physical barrier between connective tissue and the alveolar bone defect. GTR deals with regeneration of the supporting periodontal apparatus including cementum, periodontal ligament and alveolar bone whereas GBR refers to the promotion of bone formation alone. In the mid-1980s, the GTR principle was applied in periodontal regeneration, based on the early studies of Melcher (1976), who developed the concept of using barrier membranes to "guide" the biologic process of wound healing. This was supported by the classic studies of Nyman, Lindhe, Karring and Gottlow and is based on the assumption that only the periodontal ligament cells have the potential for regeneration of the attachment apparatus of the tooth.[5] This is based on criteria that reflect the biological behavior of different tissues during wound healing.[6] The progenitor cells reside in the periodontal ligament / alveolar bone both of which remain around tooth or the bone defect. Placement of physical barrier between the gingival flap and defect before flap repositioning and suturing prevents gingival epithelium and connective tissue from contacting space created by barrier.[7,8,9,10] This can be achieved either with the use of bone grafts or without grafts.[11] The use of the barrier membrane in GBR enhances complete osteogenesis by preventing the rapid ingrowth of fibroblasts into a bony defect and promoting the migration of osteogenic cells from the adjacent bony edges or bone marrow into the defect i.e., osteopromotion.

To achieve better clinical outcomes, the GBR barrier should possess the following properties: [12]

Cell exclusion: In GBR, the barrier membrane is used to prevent gingival fibroblasts and/or epithelial cells from gaining access to the wound site and forming fibrous connective tissue.

Tenting: The membrane is carefully fitted and applied in such a manner that a space is created beneath the membrane, completely isolating the defect to be regenerated from the overlying soft tissue. It is important that the membrane be trimmed so that it extends 2 to 3 mm beyond the margins of the defect in all directions. The corners of the membrane should be also rounded to prevent inadvertent flap perforation.

Scaffolding: This tented space initially becomes occupied by a fibrin clot, which serves as a scaffold for the in-growth of progenitor cells. In GBR, the cells will come from adjacent bone or bone marrow.

Stabilization: The membrane must also protect the clot from being disturbed by movement of the overlying flap during healing. It is therefore often, but not always, fixed into position with sutures, mini bone screws, or bone tacks. Sometimes, the edges of the membrane are simply tucked beneath the margins of the flaps at the time of closure, providing stabilization.

Framework: where necessary, as in non-space maintaining defects such as dehiscences or fenestrations, the membrane must be supported to prevent collapse. Bone-replacement grafts are often used for this purpose. They serve as a sort of internal framework to provide a measure of support to the graft. Stiffer membranes such as titanium-reinforced membranes have also been used for this purpose.

Barrier membranes for GBR

Barrier membranes are those which are used to achieve guided tissue / bone regeneration which is a method for the prevention of epithelial migration along the cemental wall of the pocket. Barrier membranes are biologically inert materials that serve to protect blood clot and prevent soft tissue cells from migrating into bone defects.

A. First generation material: Non-Resorbable:

Expanded polytetrafluoroethylene (ePTFE), GORE-TEX membrane, dense polytetrafluoroethylene (dPTFE), Nucleopore, Millipore filters, Ethyl cellulose and Semipermeable silicone barrier.

B. Second generation material: Resorbable membranes:

a. Collagen – Biomend, Biomend – extend, Periogen, Paroguide, Biostite, Bioguide, Tissue Guide and Biobar

b. Polylactide and Polyglycolide – Guidor, Vicryl, Atrisorb, Resolut, Epiguide and Biofix

c. Others – Periosteum, Connective tissue graft, Alloderm, Emdogain, Surgicel, Gelform, Gengiflex, Capset, Hapset and Cargile membrane.

C. Third generation material - Resorbable bioactive barrier membranes with added growth factors.

Polytetrafluoroethylene (PTFE)

Expanded PTFE (e-PTFE) is a first generation of clinically well-documented barrier membranes used for GBR. It is one of the most inert materials known which has an extremely long carbon chain protected by a dense sheath of fluorine atoms and because the body cannot react with it chemically, tissue "accepts" it, while exhibiting a healthy tissue reaction. Advantages are that it does not induce immunologic reactions, resists enzymatic degradation by host tissues and microbes. Integration of titanium reinforcement within e-PTFE membranes increases their mechanical stability and allows the membranes to be individually shaped. Disadvantages include an increased rate of soft-tissue complications after premature membrane exposure. Once exposed to the oral cavity, the porous surface of e-PTFE membranes is rapidly colonized by oral microbes. This often leads to infections of the adjacent tissues and to the need for early membrane removal, resulting in impaired bone regeneration. There is a need for re-entry surgery and membrane removal with increased patient morbidity and the risk of tissue damage.[13] To overcome this, high density PTFE membrane (d-PTFE) with a nominal pore size of less than 0.3 mm was developed in 1993. Even when the membrane is exposed to the oral cavity, bacteria is excluded by the membrane while oxygen diffusion and transfusion of small molecules across the membrane is still possible. Thus, the d-PTFE membrane scan result in good bone regeneration even after exposure. Because the larger pore size of e-PTFE membranes allows tight soft tissue attachment, it usually requires sharp dissection at membrane removal. On the contrary, removal of d-PTFE is simplified due to lack of tissue in growth into the surface structure.[14]

Titanium mesh

The use of titanium mesh which can maintain the space can be a predictable and reliable treatment modality for GBR. The main advantages of the titanium mesh are that it maintains and preserves the space to be regenerated without collapsing and it is flexible and can be bent. It can be shaped and adapted so it can assist bone regeneration in non-space maintaining defects. Due to the presence of holes within the mesh, it does not interfere with the blood supply directly from the periosteum to the underlying tissues and bone grafting material. It is also completely biocompatible to oral tissues.[14]

Resorbable membranes

Advantages include no need for membrane-removal surgery and thus elimination of the need to expose the regenerated bone, better cost-effectiveness and decreased patient morbidity. Disadvantages include difficulty of maintaining the barrier function for an appropriate length of time and resorption process of the membrane may interfere with wound healing and bone formation. Lack of stability of the material makes the use of membrane-supporting materials mandatory.

Collagen membrane

Currently, native collagen membranes are the standard treatment for the majority of GBR. Advantages are that it exhibits good tissue integration, fast vascularization, biodegradation without a foreign-body reaction and render good results and low complication rates in both animals and human. In contrast to non-resorbable membranes, epithelialization of the exposed collagen achieving secondary wound closure is spontaneous.[15]

Synthetic resorbable membranes

Synthetic resorbable membranes made out of aliphatic polyesters such as polylactic acid, polyglycolic acid, trimethylcarbonate and their copolymers are effective for GBR procedures in experimental as well as in clinical studies. Drawbacks such as inflammatory foreign-body reactions associated with their degradation products, reduced defect fill when applying polylactic acid and polyglycolic acid membranes as opposed to e-PTFE membranes.[16]

In 1994 Sascha Jovanovic, Hubertus Spiekermann & Jürgen Richter did a clinical study to evaluate bone regeneration potential at dehisced dental implant sites. Nineteen titanium dental implants with exposed threads were studied and concluded that surgical

application of an e-PTFE membrane suggested a viable clinical method for enhancing bone formation around dental implants.[13] In 1998 Paul Fugazotto reported three hundred and two consecutive ridge augmentation procedures (289 buccolingual and 13 apico-occlusal) in 284 patients. Gore-Tex membranes of various configurations were used in conjunction with various non-autogenous particulate materials. The results show an overall "success" rate of 96%, 97% for horizontally augmented ridges and 92% for vertically augmented ridges.[15] In 2004 Stavropoulos et al has made the prototype of long-term study of using a new bioabsorbable, synthetic polyglycolic acid/ trimethylene carbonate membrane was compared to a collagen membrane in a GBR study in dogs. Sites covered with a PGA/ TMC membrane showed a significantly higher percentage of bone regeneration and less soft tissue relative to sites covered with collagen membranes.[16] In 2014, Hsun-Liang Chan and Hom-Lay Wang demonstrated that the newly designed Ti-mesh applied in combination with allografts, is effective in increasing ridge height in the posterior mandible. The amount of vertical bone gain is comparable with the published data in the literature using other types of barrier membranes, including titanium-reinforced membrane.[17] In 2014, Dennis P. Tarnow and Paul Fletcher demonstrated histologically acellular dermal matrix (ADM) as a barrier for guided bone regeneration procedures. The histologic results confirmed that formation of new bone was evident, indicating that ADM had likely functioned as a barrier and had allowed for guided bone regeneration during the healing process. As dermal matrix is also successfully used for soft tissue ridge augmentation, practitioners now have an allograft material that can serve a dual function and is available in unlimited supply.[18]

Bone grafts

Bone grafts are those which provide scaffolding for bone regeneration and augmentation of bone defects which result from trauma, pathology or surgery. They can be used to restore bone loss resulting from dental disease to fill extraction sites and to preserve height and width of alveolar ridge through augmentation and reconstruction. Some grafts are more dependent on host bed for successful incorporation such as freeze dried allograft, while vascularised autografts capable of incorporating into host bed under adverse physiological conditions. The different mechanism by which it supports the use of graft materials are osteoconduction, osteoinduction and osteogenesis. At a minimum, bone graft materials should be osteoconductive. Bone graft materials that are osteoinductive are believed to be more advantageous than those that are only osteoconductive. Bone graft materials help maintain space under a barrier membrane to facilitate the formation of bone within a confined space. Perhaps a more important requirement of bone graft materials is that they should facilitate the in growth of neovascularization and migration of osteoprogenitors. The typical size of bone graft particles ranges from 100 to 1000 micrometers, which is conducive to the in growth of bone. Bone forms in cones called osteons with a central blood supply. The dimension of these cones (100A radius) is determined by the distance that the central vasculature can supply nutrients to cells. The mechanism by which graft material works depend on origin and composition of the material.[19, 20]

Classification of bone grafts

Autograft: A graft transplanted from one site to another within the same individual.

Allograft: Tissues transferred from two genetically different individuals of same species.

Xenograft: A graft transplanted from one species to a member of different species.

Isograft: A graft which is transferred from on monozygotic thin to other.

Alloplast: Synthetic or inorganic implant materials which are used as substitutes for bone graft, functions primarily as defect fillers.

Autografts:

Autogenous bone is highly osteogenic and fulfils dental defect requirement by providing scaffold for bone regeneration.[21] They can be placed at either from extra oral sites such as iliac crest /tibia[22, 23, 24] or intraoral sites such as mandibular symphysis, ramus, exostoses, and maxillary tuberosity. Factors for successful incorporation are (1.) Embryonic origin of graft (2.) Rate and extent of vascularization (3.) Structural and biomechanical features (4.) Rigid fixation of graft to recipient site (5.) Graft mentation (6.) Availability of

local growth factors (7.) Soft tissue pressure from periosteum and from flap covering graft. Auto grafts can be either[25] cortical bone, cancellous bone or cortico -cancellous bone.

Cortical bone: This lack vascular and cellular pool on endosteal and periosteal surfaces may not be able to sustain cellular liability.

Cancellous bone: They powers cell survival because of diffusion of nutrients and revascularization from the recipient bed.

Cortico - cancellous bone: Placed with cancellous bone against the recipient site and cortical part act as the barrier and space keeper against pressure from flap.

Amount of bone – extra-oral: According to the site of harvesting the following amount of bone will be obtained from each site are posterior iliac crest -140 ml, anterior Iliac crest - 70 ml, tibial plateau - 20-40 ml

Amount of bone – intra-oral: Intra orally the amount of bone which will be obtained are ascending ramus - 5 to 10 ml, anterior mandible- upto 5 ml, maxillary tuberosity - 2 ml

Harvesting autogenous bone [26, 27, 28]

Mandibular symphysis

This is a source of cortico cancellous bone. They can provide bone to augment up to two to six teeth. First incision is made in the mucosa atleast 5 mm below gingival attachment. Incision made with lips drawn anteriorly thus placing soft tissues under tension and perpetrate mucosal and muscle layers. Second incision is carried through the periosteum to bone. A periosteal elevation done will expose the symphysis and mental foramen. A ½ round, high speed bur under copious irrigation is used to outline the periphery of template and cut should be 5 mm - away from inferior border, 5 mm - from mental foramen, 5 mm - from midline spine and 5 mm - from apices of root. A 701 - surgical length bur is used to connect this outline. After this the osteotome is deepened in to marrow spaces. Then osteotome and mallet are used to elevate the graft. This is employed at variety of sites until block is mobilized. The graft is stored in mixture of saline and patient blood with the addition of 50mg gentamicin. Complications are bleeding, soft tissue injury, mental nerve injury, block graft fracture, infection, bicortical block harvest and neurosensory defects.

Mandibular tori

Mandibular tori is the hyperostosis or enlargement of lingual aspect of the mandible. Removal of tori can be considered when there is traumatic ulcers from mastication, prosthodontics considerations, tongue function interference and speech interference. A small fissure bur in a high speed handpiece is used under copious irrigation to score superior aspect of the cortical bone. If in case of small tori it is possible to use fissure bur to cut through entire bony exostosis. In cases with large tori combination of depth cuts followed by final chisel fracture is used. Moist gauze is packed against the lingual aspect of donor site to minimize swelling and hematoma formation. The graft obtained can be used either in enbloc for inlay and onlay bone grafting procedures. Alternatively they can be particulate using a bone well to repair the defect. Advantages are tori has no structural benefits, minimal trauma and low postoperative morbidity and excellent choice as onlay grafting procedure.

In 1992, Eugene E. Keller et al has used mandibular onlay composite grafts which include autogenous iliac bone and titanium cylindrical threaded endosseous dental implants placed in seven patients with advanced bone resorption. All patients reported substantial improvement in prosthesis function and comfort; they had no complaints concerning the donor or recipient sites.[29] In 2001 Antoun et al has used two techniques of ridge augmentation using onlay bone graft alone in one group and with a non-resorbable membrane in other. Six months following surgery, the membrane group experienced significantly less bone resorption than the graft alone group.[30] In 2006 Arx & Daniel Buser analyzed the clinical outcome of horizontal ridge augmentation using autogenous block grafts (harvested from the symphysis or from the retromolar area) covered with anorganic bovine bone mineral and a bioabsorbable collagen membrane. Fifty-eight sites were augmented in 42 patients, including 41 sites located in the anterior maxilla. The mean initial crest width measured 3.06 mm. At re-entry, the mean width of the ridge was 7.66 mm, with a calculated mean gain of horizontal bone thickness of 4.6 mm. Only minor surface resorption of 0.36 mm was observed from augmentation to re-entry.[31]

In 2008 Smukler, Capri & Landi et al has

introduced a technique which is based on the principles for guided bone regeneration, in which a created space is kept isolated from the surrounding soft tissues by a resorbable membrane with an excellent extended resorption profile, thus permitting the accrual of bone-formative elements into the graft site. The absorbable membrane is propped up by an autogenous mixture of native cortico-cancellous bone cores taken in the graft site and reduced to smaller particle sizes and osseous coagulum obtained from the lingual/palatal surfaces of the recipient areas using specially designed Osseous Coagulum Bone Collectors / Scrapers. The major advantage of this technique is that all the autogenous bone graft material is obtained from the actual graft site, avoiding second remote intra- or extraoral surgical sites and attendant morbidities. The successes attained with this method have prompted the initiation of a clinical research study designed to more definitively quantify the amount of horizontal and vertical augmentation that can be predictably achieved in this way.[32] In 2009 Contar et al with 15 patients who had atrophic maxillary ridge necessitating bone block grafts prior to implant placement were submitted to maxillary reconstructions performed with human block grafts of tibia fresh-frozen chips. Thirty-four blocks were placed, and the number of blocks each patient received ranged from 1 to 4. During the re-entry procedures, all of the grafts were found to be firm in consistency, well-incorporated, and vascularized. A total of 51 implants were placed over the grafts with a minimum of 40-Newton torque in all cases. None of the implants were lost. The follow-up period ranged from 24 to 35 months.[33]

In 2010 Luigi Canullo & Angelo Sisti et al has evaluated the survival of implants loaded 14 weeks after vertical ridge augmentation (VRA). Nano-structured Mg-enriched hydroxyapatite was used as the only augmentation filler material. It was covered with a titanium reinforced e- PTFE membrane. This clinical study suggests that VRA around rough-surface implants using e-PTFE membrane and nano-structured Mg-e hydroxyapatite can be successful[34] In 2011 Ludovichetti, Stefano & Enricho et al has designed a study to evaluate the effectiveness of an alternative material, a flexible equine bone sheet, for vertical ridge augmentation. Forty-nine implants were placed in 18 patients. After 4 months, the ridge volume for all patients was completely restored, all implants had successfully osseointegrated, and definitive prostheses were placed.[35]

Allografts

Bone allografts are commercially available from tissue banks. They are obtained from cortical bone within 12 hours of death of the donor, disinfected, cut in pieces, washed in alcohol and deep frozen. They undergo strict screening and processing by tissue banks. Bones are cut into standard shape or size. The tissues are soaked in sterile solutions to remove unwanted compounds. Once the lipids and cells are removed, the pieces are crushed into powders. They are then freeze dried with liquid nitrogen or with chemical solvents to eliminate the moisture. The freeze dried bone is packaged in air tight container. De-mineralised freeze dried bone is obtained by placing a freeze dried bone in strong hydrochloric acid to remove its mineral matrix.[36, 37, 38]

Freeze dried bone allograft

This is an osteoconductive material which is available in variety of sizes and can be selected according to the needs. FDBA can be used in the following situations repair and restoration of fenestrations, minor ridge augmentation, fresh extraction sites, sinus lift cases and repair of dehiscence and in implant failures.

Decalcified freeze dried bone allograft

Demineralization will expose the collagen fibrils termed as bone morphogenic protein studies shown that DFDBA results in significant probing depth reduction, attachment gain and osseous regeneration. DFDBA is osteoinductive due to presence of BMP and other non-collagenous proteins in the exposed matrix. They can be combined with osteogenin a bone inductive protein isolated from the long bones. Advantages are commercially available which is ready to use, elimination of secondary surgical procedure, reduced time and minimal blood loss.[39] Disadvantages are antigenicity of the tissues which may cause resection due to induction of host immune response, not osteogenic and hence take long time and only less volume.

Xenografts

Xenografts constitute the fourth category of commonly used bone-grafting materials, with porous bovine derived material—the most popular xenograft variety. Numerous researchers have reported a high degree of osteoconductivity and bovine bone particles are well incorporated within newly regenerated grafted bone. According to histological findings,it has been argued that the slow resorption profile of bovine derived bone may contribute to increased stability of the regenerated bone

1. Calf bone [boplant]:This is treated by detergent retraction, sterilized and freeze dried and has been used for treatment of osseous defects.

2. Kiel bone: This is a calf or ox bone which is denatured with 20 % hydrogen per oxide dried with acetone and then sterilized with ethylene oxide.

3. An organic bovine bone: This is chemically treated to remove its organic component sterilized and used as a graft without causing a host immune response. Pepgen p-15 is an enhanced form of bovine derived hydroxyapatite which contains a short chain peptide P 15. P 15 mimics the cell binding domain of type I collagen which is responsible for cell - migration, differentiation and cell proliferation.[40]

Alloplasts

Alloplasts work through osteoconduction, providing a scaffold for bone growth. Alloplasts can be divided in to bioinert and bioactive. Bioinert materials (aluminum or titanium oxides) do not bond directly to the underlying natural bone and need to be mechanically fixated in place. Bioactive materials (calcium carbonate, calcium phosphates, calcium sulfate) readily bond to the underlying bone. Both, bioinactive and bioactive materials are available in a resorbable and a non resorbable variant.

There are many studies which compared xenografts and alloplastic material for augmenting bone. In 2001 Zitzmann et al, conducted a study to investigate the healing of alveolar ridge defects augmented with cancellous bovine bone mineral. In six partially edentulous patients, the defect sites, all located in the maxilla, were filled with Bio-Oss and covered with the resorbable collagen membrane Bio-Gide. The histologic analysis revealed an intimate contact between woven bone and Bio-Oss along 37% of the particle surfaces. A mixed type of bone was found; it contained woven bone and parallel-fibered bone, which demonstrates features of remodeling activity. Within the limits of study it is suggested that Bio-Oss may be a very suitable material for staged localized ridge augmentation in humans.[41] In 2006 Masago et al have done alveolar ridge augmentation with β-tricalcium phosphate granules, calcium phosphate cement powder and web form of titanium fibers added to Platelet-Rich-Plasma and histologically observed for 5 months by the experiments using the maxilla of rabbits. After five months the β-tricalcium phosphate granules were completely absorbed and replaced with the new viable bone in almost all of the treated areas. In case of Calcium Phosphate Cement powder, most of the crystallized substances still remained up until 5 months after surgery. With titanium fibers, five months later new viable bone was observed throughout the entire space.[42]

In 2007 Strietzel et al has evaluated the tissue composition of augmented sites after the use of a nano-crystalline hydroxyapatite (ncHA) bone substitution material by clinical and histological examinations. The alveolar ridge width gain was found to be significant after lateral augmentation utilizing ncHA, providing a quantitatively and qualitatively sufficient site for primary stable implant placement.[43] In 2009 Rothamel et al, histologically compared the healing following vertical ridge augmentation using three different therapies: (1) xenogenous block alone; (2) xenogenous block, covered with a chemically cross-linked collagen membrane; & (3) autologous blocks, harvested during defect preparation. In general, histologic analysis revealed that xenogenous blocks, used alone or combined with a collagen membrane, exhibited osteoconductive properties on a level equivalent to that of autologous blocks, resulting in means of 50% to 60% of ossification of the blocks. Within the limits of the present study, it was concluded that screwable xenogenous bone block might be a useful scaffold for ridge augmentation procedures. However combination of xenogenous blocks with cross-linked collagen membrane did not appear to improve outcome.[44] In 2010 Betilium et al has evaluated the outcome of bone augmentation in 50 patients. In 27 patients particulate mineralized Freeze Dried Bone Allograft was covered and in another 23

patients FDBA with addition of autogeneous bone chips applied in bilayered technique. Both the grafts is covered by ribose cross linked collagen membrane. Both groups has showed enhanced horizontal and vertical height of bone and hence remain one of the treatment of choice for ridge defects. But addition of autogeneous bone chips does not showed any added benefit to this procedure.[45] In 2012 Steigmann et al, described a buccal periosteal pocket flap procedure proposed to overcome the challenges in cases of severe or localized horizontal bone deficiencies where sufficient soft tissue mobilization to ensure primary wound closure over the augmented area is difficult. The flap design is a periosteal pocket, which allows filling of bone-grafting material while facilitating primary, tension-free soft tissue closure by splitting of the mucosa. The flap gives stability to the augmented volume within the pocket. Data obtained from this study suggest that the periosteal pocket flap design could be a predictable alternative flap approach for correction of severe or localized horizontal bone deficiencies.[46]

In 2012 Wallowy et al evaluated the effectiveness of lateral ridge augmentation in 36 patients with severely atrophic alveolar ridge using allogeneic bone grafts in a framework technique. A full-thickness mucoperiosteal flap was displaced to gain access to the bone. This was then followed by contouring and thinning of the cortical graft by means of rotating instruments to a thickness of approximately 0.3-0.5 mm to fit the alveolar ridge. A thinned allogeneic cortical graft was screwed to the alveolar ridge, leaving a hollow space that was filled with particulated allogeneic cortical bone. Frame be fixed in such a way that it is rendered absolutely immobile. Out of 36 patients, in 33 patients dental implants were successfully installed and continued to be well maintained at the last follow-up. In three patients, dental implants could not be installed as the graft was lost because of wound dehiscence; however, repeat surgery was done successfully.[47] In 2013 Hannes Wachtel et al selected four systemically healthy patients with inadequate dental alveolar ridge width. All ridge defects were augmented using a xenogeneic cortical bone shield in combination with particulated bone substitutes and a thin collagen barrier. This case series indicates that the bone lamina technique has the biologic and mechanical properties to successfully achieve hard tissue augmentation of deficient ridges.[48]

Conclusion

There is ample evidence demonstrating the successful use of guided bone regeneration to regenerate missing bone at implant sites. But the influence of guided bone regeneration on implant survival and success rates, and the long-term stability of the augmented bone, remain unknown. Many of the materials and techniques currently available for bone regeneration of alveolar ridge defects were developed many years ago. New materials should allow optimal cell in growth and present adequate mechanical properties sufficient to maintain space for bone regeneration. Future research should focus on understanding the regulation of gene expression and the molecular features of the bone regeneration process. Cell-based tissue engineering and gene-delivery therapy represent new therapeutic strategies that have the potential to overcome several shortcomings associated with the existing bone regeneration techniques.[2]

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JSPIK

Platelet concentrates: A promising innovation in Periodontal regeneration

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ABSTRACT

The ultimate aim of periodontal regenerative surgery is to achieve complete wound healing and regeneration of the periodontal unit. Regenerative techniques like grafts and barrier membranes are widely used in today's clinical practices. Platelets contain high quantities of key growth factors which are able to stimulate cell proliferation, matrix remodelling and angiogenesis. A recent innovation in dentistry is the preparation and use of platelet concentarates like platelet-rich plasma (PRP) and platelet rich fibrin (PRF). These are autologous preparations made from patient's own blood that result in local release of growth factors when applied to the defect site. Evidence from literature have proved that this may promote the bone regeneration in intrabony defects, furcations and cyst cavities. The main aim of this review article is to briefly describe platelet concentrates including their classification, role in regeneration and preparation techniques.

Key words: Regeneration, Platelets, Platelet rich plasma (PRP), Platelet rich fibrin (PRF)

Introduction

Periodontitis is defined as "an inflammatory disease of the supporting tissues of teeth caused by specific microorganisms or groups of specific microorganisms,resulting in progressive destruction of the periodontal ligament and alveolar bone with pocket formation, recession or both."[1] The aim of periodontal therapy should be to protect and maintain patient's natural dentition for optimal comfort, function and esthetic appearance.

Healing after periodontal therapy includes repair and regeneration. Repair is the healing of a wound by tissue that does not fully restore the architecture or function of the affected unit, whereas regeneration is reproduction or reconstitution of a lost or injured part.[2] In open periodontal flap surgery healing occurs usually by long epithelial attachment that results in reduction in pocket depth. Regenerative surgical techniques uses graft materials and barrier membranes and results in reduction of pocket depth by supporting formation of new functional periodontal ligament.

Many cell types and growth factors are involved in wound healing process. Several factors, including cell–matrix interactions and soluble ligands such as cytokines and growth factors affect cell behavior and function during healing. After injury, platelets begin to form a stable blood clot, that releases a variety of growth factors that promote healing process. Platelet-derived growth factors, vascular endothelial growth factor,insulin like growth factor, transforming growth factor beta, parathyroid hormone, fibroblast growth factors, epidermal growth factor and bone morphogenetic proteins have been extensively studied to understand their role in periodontal wound healing, both in vitro and in vivo. Administration of these growth factors may be combined with tissue regeneration techniques in the repair of intrabony defects, furcations and cyst cavities. Application of

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platelet concentrate is a novel method for obtaining these polypeptide growth factors.

Tissue healing and role of platelets

The wound healing process involves coagulation,inflammation, matrix synthesis, angiogenesis, and remodeling. Formation of platelet plug arrests initial bleeding. The activation of the coagulation cascade and modeling of the blood clot is the first step. Following that acute inflammatory response occurs resulting in platelet secretion of tissue growth factors and cytokines. New granulation tissue forms and serves as a temporary tissue scaffold. Then angiogenesis, extracellular matrix modeling and maturation of full strength tissue occurs. Large number of locally released molecules, including inflammatory cytokines, chemokines, and growth factors initiate these complex process. These mediators work in harmony and play a critical role in initiating tissue-renewal processes. Growth factors that play a primary role in tissue-repair processes include platelet-derived growth factor AA and BB (PDGF-AA and PDGF-BB), transforming growth factor α and β (TGF-a and TGF-b), fibroblast growth factor-2 (FGF-2), vascular endothelial growth factor (VEGF), and granulocyte macrophage colony stimulating factor (GM-CSF).[3]

Growth factors that come under PDGF family recruit neutrophills, macrophages and osteoblasts. They promote chemotaxis of mesenchymal cells and production of vascular endothelial growth factor. Transforming growth factor beta induce extracellular matrix production and periodontal ligament cell proliferation. It also inhibit formation of osteoclast precursors and reduces scar formation. Vascular endothelial growth factor induce vasculature formation, forms mature vascular tissue and recruit bone marrow cells. GM-CSF enhance wound healing process, improve wound healing quality and inhibit bone resorption. Fibroblast growth factor family (FGF 1 and FGF 2) recruit endothelial cells, stimulate periosteum derived cells, induce bone formation and plays a role in homeostatic regulation.[3]

Platelet concentrate

Platelet concentrates for topical and infiltrative use are first of all blood extracts obtained after various processing of a whole blood sample, mostly through centrifugation[4]. The objective of the processing is to separate the blood components in order to discard elements considered as not usable (mostly the red blood cells, heavy and easily separated) and to gather and concentrate the elements that may be use for therapeutic applications (fibrinogen/fibrin, platelets, growth factors, leukocytes and other forms of circulating cells, in solution in liquid plasma). The development of platelet concentrates for surgical use, often termed under the general acronyms PRP (Platelet-Rich Plasma) or PRF (Platelet-Rich Fibrin), is an important current transversal field of research across many fundamental and clinical disciplines.[5] The biocompatibility and versatility of platelet growth factors makes them useful in a variety of applications, including tissue engineering, orthopedics, sports medicine, periodontics, and dental implantology, to name a few.

Classification of platelet concentrate

First classification was proposed in 2009[6] and it is widely cited as it provide clarification of the terminology. This classification separates the products following 2 key parameters: the presence of a cell content (mostly leukocytes) and the fibrin architecture. This separation define 4 main families to regroup the products.

1. Pure Platelet-Rich Plasma (P-PRP) – or Leukocyte- Poor Platelet-Rich Plasma

Products are preparations without leukocytes and with a low density fibrin network after activation. Per definition, products of this family can be used as liquid solutions or in an activated gel form. Many methods of preparation exist, particularly using cell separators (continuous flow plasmapheresis). One largely advertised method of P-PRP is known under the commercial name PRGF [Plasma Rich in Growth Factors or Preparations Rich in Growth Factors or EndoRet, used particularly in sports medicine. Another technique of P-PRP was widely promoted for skin ulcers and is known under the commercial name Vivostat PRF

2. Leukocyte-and Platelet-Rich Plasma (L-PRP)

Products are preparations with leukocytes and with a low-density fibrin network after activation. Per definition, like the P-PRP, all the products of this family can be used as liquid solutions or in an activated

gel form. 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).

3.Pure Platelet-Rich Fibrin (P-PRF) – or Leukocyte- Poor Platelet-Rich Fibrin

These are preparations without leukocytes and with a high-density fibrin network. Per definition, these products only exist in a strongly activated gel form, and cannot be injected. However, because of their strong fibrin matrix, they can be handled like a real solid material for other applications.

4. Leukocyte- and Platelet-Rich Fibrin (L-PRF)

These products are preparations with leukocytes and with a high-density fibrin network. The technique was initially developed and evaluated as an open-access technique, based on the concept of one-step centrifugation of blood without anticoagulant and without blood activator. The preparation is completely natural, and this remains a key difference with all other families of products. Nowadays, the only FDA-approved CE-marked system of L-PRF with certified materials is marketed under the name Intra-Spin L-PRF (Intra-Lock Inc., Boca Raton, FL, USA). The technique is very simple, quick, inexpensive and allows to produce large quantities of fibrin clots and membranes in a very short time, particularly using the Xpression preparation box.[5]

L-PRF clots and membranes present a volume and shape easy to combine with most surgical techniques, as filling and interposition healing biomaterial or as protection healing membrane. These membranes are also strong and offer a slow release of many growth factors during long periods. Finally, it is easy to prepare in large quantity and inexpensive, what makes it particularly adapted for daily clinical practice.

This classification system was largely cited, advocated, and validated by a multi- disciplinary consensus conference published in 2012[7]. The POSEIDO (Periodontology, Oral Surgery, Esthetic and Implant Dentistry Organization) hold it as its guidelines for all publications on the topic in 2013[8].

There is a very large debate particularly in sports medicine on the selection of the adequate

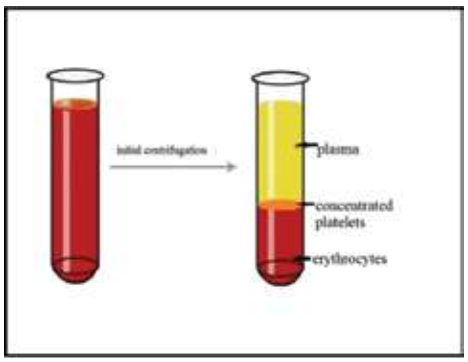

Fig 1

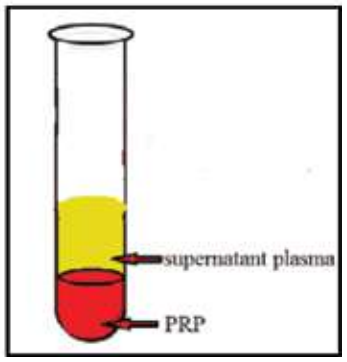

Fig 2 (after final centrifugation)

Fig (3): PRP added to graft

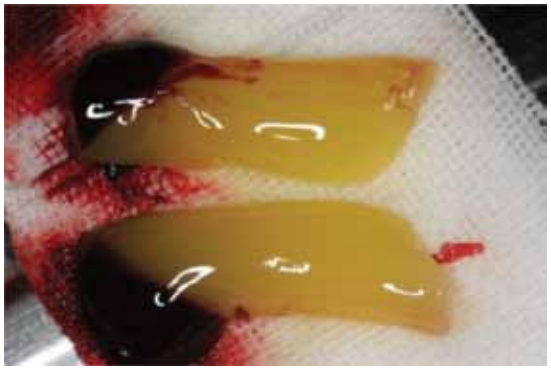

Fig (4) : Platelet rich fibrin

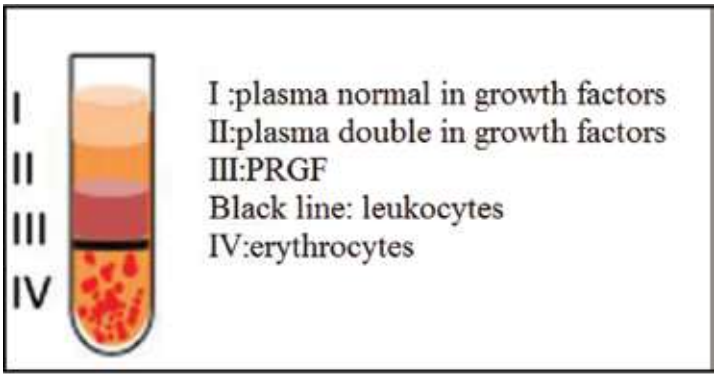

Fig: (5)

technique, concerning the exact cell content of the platelet suspensions. There are two schools of thought. One group advocated that the presence of leukocytes may be negative for the therapeutic outcome, due to a potential risk of stimulation of the inflammatory process after administration of the product. On the contrary, other groups insisted on the need of some leukocyte population in the platelet concentrate, in order to increase the growth factors production, the release of anti-pain mediators and the natural anti infectious activity. Leukocytes are not only inflammatory cells, as they also present anti-nociceptive effects through different chemokines, anti-inflammatory cytokines (IL-4, IL-10 and IL-13) and opioid peptides (b-endorphin, metenkephalin, and dynorphin-A), and can therefore promote a clinically relevant inhibition of pathological pain. During inflammation, these cytokines counteract the effects of the pro-inflammatory mediators generated naturally in the early stages of inflammation.[5]

Platelet rich plasma(PRP)

The platelet count in PRP exceed 2 million platelets per micro liter. A natural blood clot contains 95% RBCs, 5% platelets, less than 1% WBCs, and numerous amounts of fibrin strands. While a PRP blood clot contains 4% RBCs, 95% platelets, and 1% WBCs. Compared to using a single recombinant growth factor, PRP provides the advantage of multiple growth factors that work together synergistically. In the early 1970s, autologous PRP was developed, and its clinical use was first reported in the 1980s.[9] In the 1990s, dental uses were reported. A major strength of PRP is that it mimics the natural process of the tissue renewal, and it can be obtained using a reproducible, safe, and effective preparation technique.

Preparation technique

There are various techniques available including discontinuous plasmapheresis used in blood banks. However, some techniques for the preparation of small amounts of autologous PRP for dental use can be completed in minutes and involve less stress. Two systems are now available commercially for office use by dental practitioners. The Platelet Concentrate Collection System [PCCS] and the Curasan PRP kit. The PCCS and Curasan systems use different protocols, but the end product is suitable for the same oral surgical applications. In addition to these there is Smart PReP system produces PRP gel as well as fibrin glue, but it is used for preparation in large quantity. The absolute gain in platelets was higher with the PCCS system, but the highest concentration of platelets per microlitre was obtained with the Curasan system.[10]

PRP prepared from 8 to 10 mL of whole blood is usually sufficient for periodontal regenerative therapies. The technique described here uses a general purpose laboratory tabletop centrifuge. It is simple and cost-effective method for producing PRP in an in-office environment. Patients are selected based on the absence of any blood abnormalities or use of anti-coagulants. 10 ml blood is withdrawn from the anticubital region with a 10ml syringe and transferred to a container containing 1.4ml anticoagulant (Citrate phosphate dextrose solution). It is then centrifuged for 10 mins at 1300 rpm. The result is a separation of whole blood into a lower red blood cell (RBC) region and upper straw-colored plasma region. There is relatively high concentration of platelets found in the boundary layer between these two regions. (fig 1) The upper straw colored plasma layer (platelet poor plasma; PPP) and 1-2 mm of the top part of the RBC layer is aspirated and transferred into another container and again centrifuged for 10 mins at 2000 rpm. This results in an upper portion of clear yellow supernatant serum and the bottom red tinged layer consisting of highly concentrated PRP (fig 2). The upper clear layer is aspirated until 1.5ml of serum is left. The contents of the tube is mixed well and transferred into a sterile container. At the time of the application, the PRP is combined with an equal volume of a sterile saline solution containing 10% calcium chloride (a citrate inhibitor that allows the plasma tocoagulate) and 100 U/ml of sterile bovine thrombin (an activator that allows polymerization of the fibrin into an insoluble gel, which causes the platelets to degranulate and release the indicated mediators and cytokines). This results in formation of a sticky gel that is relatively easy to apply to the surgical defects.[11]

The product is immediately used onto the graft site or added to particulate graft and sutured in place. PRP can also be gelled into a membrane that can be placed into a surgical site.

Clinical applications of PRP

- Enhanced healing of intrabony defect in periodontitis
- Ridge augmentation
- Sinus augmentation
- Improved osseointegration around dental implant
- Closure of cleft lip and palate defects
- Repair of bone defects created by removal of teeth or small cysts
- Repair of fistulas between the sinus cavity and mouth.
- Pain reduction in chronic ulcers
- Anti-inflammatory effect that reduces echymosis and edema

Platelet rich fibrin (PRF)

Platelet-rich fibrin (PRF) commonly known as Choukroun's PRF is a second-generation platelet concentrate which contains platelets and growth factors in the form of fibrin membranes prepared from the patient's own blood free of any anticoagulant or other artificial biochemical modifications. PRF (platelet rich fibrin) was first developed in France for use in the field of oral and maxillofacial surgery.[12] PRF is a potent autologous regenerative material with many clinical applications in the field of periodontics as it accelerates both soft tissue and hard tissue healing. The PRF clot forms a strong natural fibrin matrix, which concentrates almost all the platelets and growth factors of the blood harvest and shows a complex architecture as a healing matrix with unique mechanical properties which makes it distinct from other platelet concentrates.

Preparation technique

The classical technique for PRF preparation was invented by Dr.Choukroun in 2000. A sample of blood is collected from patient without anticoagulant in 10 ml tubes which are immediately centrifuged at a rate of 3000 rpm for 10 min using a PC-02 table centrifuge. Coagulation cascade is initiated by the activation of platelet when the blood comes in contact with the test tube walls. After centrifugation, the resultant product consists of three layers. The topmost layer consisting of a cellular PPP (platelet poor plasma), PRF clot in the middle and RBCs at the bottom of the test tube. The fibrin clot obtained is removed from the tube and the attached red blood cells scraped off from it. PRF can also be prepared in the form of a membrane by squeezing out the fluids present in the fibrin clot.

Advantages of PRF over PRP

- The preparation of PRF is simpler and faster because it does not require additional anticoagulants and chemical activators.
- Compared with PRP, PRF exhibits a greater expression and concentration of growth factors and matrix proteins, which are released more slowly because of the three dimensional architecture of the adhesive glycoproteins in the fibrin and results in significantly better performance.
- It results in a dense fibrin-rich membrane matrix that has a consistency (gel form) that is better suited for manipulation and suturing.

Plasma rich in growth factor (PRGF):

A new technique for the preparation of a platelet-rich biologic known as PRGFs was described by Anitua in 199113. PRGF, in contrast to PRP, does not contain leukocytes and the associated inflammatory by products. Plasma-derived adhesive molecules, such as fibrinogen, fibronectin, vitronectin, and thrombospondin-1, serve as a matrix or scaffold that attracts progenitor cells and platelets. This is a 100% autologous preparation rich in biologic mediators to promote hard- and soft-tissue regeneration. PRGF is a promising complement to treatment that favors improved hard- and soft-tissue healing by using the patient's own biologically active proteins, growth factors, and biomaterial scaffolds for therapeutic purposes.

Preparation of PRGF

Ten to 20mL of peripheral blood is drawn from the patient and placed directly into blood-collecting tubes that contain 10% trisodium citrate as an anticoagulant. Liquid PRGF is prepared by centrifugation at 160 x g for 6 minutes. The blood is then separated into four layers (Fig:5): red blood cells at the bottom or fourth layer; PRGF is the third layer; plasma double in growth factors is the second layer; and plasma-poor growth factors (PPGFs) at the top layer. The red blood cell layer is separated from the plasma layers by a thin layer of white blood cells known

as the buffy coat. The PPGF layer is discarded, and then the PRGF layer is transferred into another tube containing 10% calcium chloride. This yields liquid PRGF. Another preparation, the PRGF scaffold, is prepared the same way,but there is an additional 5 to 8 minute wait until the final clot or scaffold is formed.[13]

Limitations in use of platelet concentrates

PRP may be most advantageous in cases where compromised healing is expected and in cases in which more rapid soft-tissue healing is important. Both case selection and site selection are important. An optimal case may heal well with or without PRP, and the same is true of an optimal indication or site. Thus, compromised cases or sites may benefit more. Another limitation is selection of the PRP preparation technique. The method must yield an adequate amount of platelets and growth factors or the preparation will be ineffective.

Massimo Del Fabbro et al[14] conducted a meta analysis to systematically evaluate the effects of autogenous platelet concentrates on clinical outcomes of the surgical treatment of periodontal diseases. All the included studies used PRP. They found out that PRP may exert a positive adjunctive effect when used in combination with graft materials, but not with GTR, for the treatment of intrabony defects. No significant benefit of platelet concentrates was found for the treatment of gingival recession. Another systematic review conducted by Vittorio Moraschini et al[15] done in 2016 suggest that the use of PRF membranes did not improve the root coverage, keratinized mucosa width, or clinical attachment level of Miller Class I and II gingival recessions compared with the other treatment modalities.

Conclusion

The use of autologous growth factors or other plasma products to enhance wound healing is an exciting prospect for surgical treatments especially for regenerative therapies. Currently, some studies support the use of these products,but there are also conflicts in the literature concerning their effectiveness. Additional studies are needed to ascertain the reasons for the lack of predictability. These adjuncts to treatment are simple and inexpensive to implement in daily clinical practice. Their use will be welcomed by clinicians, as long as they provide a consistent benefit in treatment.

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JSPIK

Suture Materials – A Review

Anu Mathew[1], Raseena Beevi[2]

ABSTRACT

Surgical sutures are used to approximate wound margins and enhance tissue healing. A suture is a strand of material used to ligate blood vessels and to approximate tissues together .There is a wide range of suture materials and the main types include absorbable and non-absorbable. They are also available in the form of monofilament, multifilament/braided and pseudomonofilament. The emerging trends in suture technology includes antimicrobial sutures, bio-active sutures and smart sutures like electronic sutures. These newer strategies expand the versatility of sutures as biologically active component enabling delivery of drugs and cells to the desired site with immense application potential in therapeutics.

Key words: sutures, staples, surgical tapes, tissue adhesives

Introduction

The primary purpose of sutures is to hold apposing tissues together to facilitate and hasten healing process with minimal or no scar formation following an injury or surgical procedure.[1] Suture literally means to "join". In surgery suturing is the act of sewing or bringing tissue together and holding them in apposition until healing has taken place. There are several possibilities to close a wound, for example staples, skin tapes, tissue adhesives. By far the most common technique is the use of sutures. Nowadays there is a wide variety of materials that are being used for surgical sutures.

Requirements of an ideal suture material[2]

- Adequate tensile strength .
- Low capillarity:
- Tissue biocompatibility:
- Good handling & knotting properties:
- Sterilization without deterioration of properties:
- Its use should be possible in any operation.
- Low cost
- It should not fray
- Should be readily visualized
- On break down, should not release toxic agents.

Preparation of Sutures:

Modern surgical sutures are packaged with minimal handling and sterilized with either ethylene oxide or ionizing radiation, often cobalt 60. Each suture is placed in an inner foil suture packet that in turn is placed in an exterior half plastic, half foil packet, called the overwrap, to help ensure sterility[3]

Classification

Absorbable sutures

Natural absorbable sutures are digested by body enzymes. Synthetic absorbable sutures are hydrolyzed by which water gradually penetrates the suture filaments, causing the breakdown of the suture. Compared to enzymatic degradation, hydrolysis results in lesser tissue reaction. Hence synthetic sutures cause less inflammation

Surgical gut sutures

These sutures are derived from naturally occurring purified connective tissue (mostly collagen) of the small intestine of sheep or cattle. Surgical gut

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is absorbed by proteolytic enzymatic degradation.[4] It is monofilament and is available in the plain form as well as "tanned" in chromic acid. Catgut is packaged in a preservative solution (isopropyl alcohol) serves to condition or soften it. It will swell due to absorption of alcohol and its tensile strength will be reduced and also cause irritation to the tissues.

Chromic catgut

Chromic catgut sutures are plain gut sutures coated with thin layer of chromium salt solution to minimize the tissue reaction. It increases the tensile strength of suture materials and slow the absorption rate. These materials have better knot security, and good handling properties. Allergy to suture material, particularly catgut, has been reported. Chromic salts added to catgut may also provoke an allergic reaction in those who are chromate-sensitive.

Polyglycolic acid (dexon)

This is an absorbable braided synthetic homopolymer of glycolic acid. It is supplied as an uncoated or coated form. Coated polyglycolic acid suture is undyed or dyed green, violet or bicolored with greater knot pull, tensile strength and minimal tissue reactivity.[5] This materials does not tolerate wound infection so should not be placed at an infected site. It is not recommended as a percutaneous suture, but effective in deeper tissue layers.

Polyglyconate(maxon)

Synthetic, absorbable, monofilament type sutures. It is composed of Polyglycolic acid and trimethylene carbonate. Polyglyconate[6] has some advantages over other sutures, such as improved handling properties, lacks memory, passes easily through tissues and demonstrates superior strength. The material does not slide easily on itself, making tying difficult. The manufacturer recommends wetting the material with saline solution to facilitate tying.

Polyglactin 910 (vicryl®, polysorb®)

Synthetic suture composed of a mixture of lactide and glycolide acids and calcium stearate produced in a braided configuration that improves the handling properties. Lactide component has hydrophobic qualities that delay the loss of tensile strength. It is made of polyglycolic acid and polylactide in a mixing

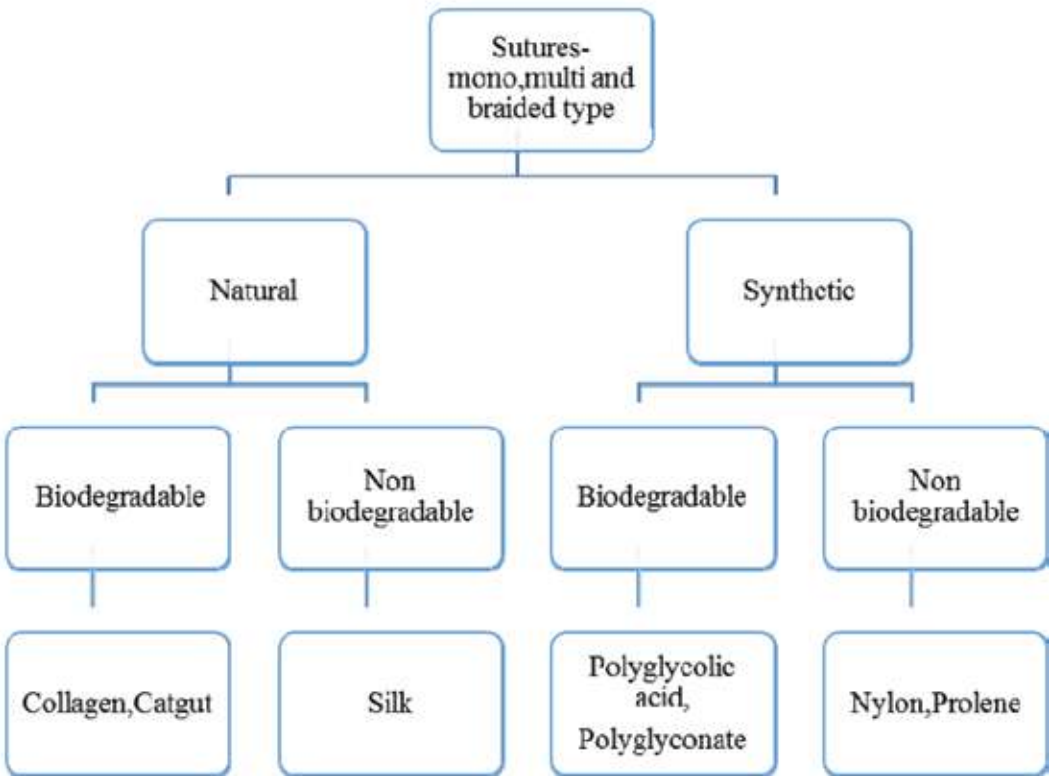

Fig. 1 Classification of Suture Materials

ratio of 9:1, so known as polyglactin 910. Advantages include better handling, minimal tissue reactivity, and does not tear tissue. Occasionally, the suture is extruded without inflammation, resulting in a small nodule the suture line ('spitting'). It can be used in infected tissues. (Figure 2)

Polydioxanone (PDS II)

Synthetic, Monofilament polymer with prolonged tensile strength and may persist for more than 6 months. Good for high-tension areas or contaminated wounds. Polydioxanone is stiff and difficult to handle. It is a low reactivity suture that maintains its integrity in the presence of bacterial infection.[7]

Disadvantages of absorbable sutures

- In fever, infection, or protein deficiency, the suture absorption process may accelerate, causing too rapid a decline in tensile strength, the absorption may begin prematurely. Such situations predispose to postoperative complications, as the suture strand will not maintain adequate strength.
- Highly reactive compared to non- resorbable sutures
- Evoke intense inflammatory reaction
- May delay wound healing: eg: gut sutures

Non absorbable sutures

Silk (Dysilk®)

Braided natural proteinaceous thread of silkworm larval cocoons. Each silk filament is processed to remove the natural waxes and sericin gum. After braiding,the strands are put in a dye, stretched, and impregnated with a mixture of waxes and silicone. Dry silk suture is stronger than wet silk suture. It provides better handling, knot security, and pliability (making it ideal for mucosal surfaces) and is made non capillary in order to withstand the action of body fluids& moisture (wax or silicon coated). Disadvantages are low tensile strength, and high coefficient of friction, and tissue reactivity. 8(Figure 3)

Contraindication: Silk suture materials should not be used in the presence of infection.

Nylon (Ethilon®, Dermalon®, Surgilon®)

Synthetic, monofilament with good tensile strength, and minimal tissue reactivity and knot security, but it remains one of the most popular non-absorbable sutures in dermatological surgery. Surgilon® and Nurilon® handle better but are more expensive

Polyester (Dacron®, Dersilene®, Ethibond®)

Braided multifilament fibers of polyester or polyethylene terephthalate. This suture has excellent tensile strength, good handling, and low tissue reactivity.

Polypropylene (Prolene®, Surgilene®, Surgipro®)

A monofilament polymer with a very low coefficient of friction making it the suture of choice for running subcuticular stitches. It has good plasticity but limited elasticity, and it is relatively expensive. Polypropylene sutures are favoured for facial repairs. It is a polymer of propylene, inert and maintains tensile strength for 2 years. Holds knots better than other synthetic sutures. Advantages includes minimal suture reaction and so used in infected and contaminated wounds. It do not adhere to tissues and is flexible. So used for 'pull-out' type of sutures.[9]

Recent and emerging trends

Fig. 2 Vicryl Suture

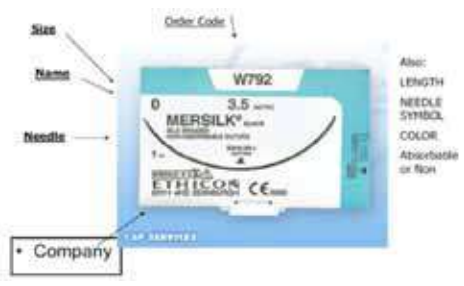

Fig. 3 Silk suture

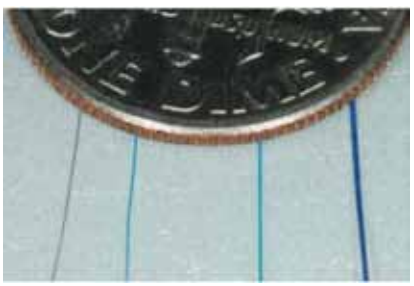
Fig. 4 8-0 nylon, 7-0 polypropylene, 6-0 polypropylene, 4-0 vicryl

Recent trends includes the development of sutures with additional properties such as those modified with antimicrobial agents, bioactive molecules like drugs, antibodies, proteins, growth factors. Sutures with bioactive substance can be therapeutically useful for wide range of procedures in a site specific manner and hasten the healing process.

Antimicrobial sutures[10]

Suture material	Bioactive modification	Clinical significance
Polyglycolic acid (braided)	Chlorhexidine diacetate	Antimicrobial efficacy upto 5 days with good biocompatibility
Chromium gut, Nylon, Silk	Quaternary ammonium compound	Bacterial growth inhibition of Porphyromonas gingivalis (periodontal) and Enterococcus faecalis (endodontic) virulent pathogens

Drug-eluting sutures[11]

Depending on the type of therapeutic agent used, drug eluting sutures can alleviate postoperative complications such as surgical site infections and expedite wound healing. It also can reduce the need for supplemental drugs which can have decreased potency or availability at the site of the procedure following a systemic administration.

Suture material	Bioactive modification	Clinical significance
Silk	Tetracycline hydrochloride	Antimicrobial efficacy
Polyglycolic acid	Ibuprofen	Relief from post operative pain

Stem cells seeded sutures[12]

Biodegradable scaffolds are widely used in tissue engineering and regenerative medicine as a carrier to transplant and differentiate stem cells to various tissues. Recent studies revealed that sutures coated with growth factors or stem cells could be used as a mode of delivery for these biological components to the desired site. The primary objective of stem cells seeded suture is to increase the number of these cells at the injured site to accelerate the tissue regeneration and repair. Although, suture-based cell delivery appears to be a feasible approach to transplant stem cells into the soft tissues of the body, retaining the desired mechanical, and physical properties of such sutures would be a major challenge.

Smart sutures

Electronic sutures[13]

Electronic sutures with the capability to monitor, sense, and actuate typical biological responses in the body would be very useful in improving localized tissue health monitoring. Electronic suture can accurately measure the elevated temperature which can be used to identify the infection status and also helps in the maintenance of ideal temperature to support healing process with microheaters at the wound site.

Sutures in Microsurgery

The microsurgery offers new possibilities for periodontal surgery can improve therapeutic result for a variety of procedure & gives benefits of improved cosmetics, rapid healing, minimal discomfort & enhanced patient acceptance. Most periodontal microsurgical suturing is done with sutures ranging in size from 6-0 to 9-0 (Fig. 4). Increasing number of zeroes correlates with decreasing the suture diameter and strength. Monofilament suture material is preferable to polyfilament (high capillarity). Polypropylene & its newest development polyhexafluoropropylene & polytetrafluoroethylene materials with excellent tissue properties, commonly used in microsurgery.

Alternatives of sutures

Staples

Staples are made of stainless steel and combine the highest tensile strength of any suture material in use today with a low tissue reactivity. Metal staples also provide excellent wound edge eversion without strangulation of tissue and result in minimal scarring.[15] It provides faster closure than sutures. Contaminated wounds closed with staples have a lower incidence of

infection than those closed with sutures. Staple closure also eliminates the risk that a health care provider will experience a needle prick, which is a particularly important consideration in caring for trauma patients with unknown medical histories.

Tapes

Steri-Strips- Tapes are strips of microporous non occlusive material (eg, paper, plastic, rayon fabric) backed by a thin film of acrylic polymer adhesive. They are useful as an adjunct to or a substitute for other wound closure materials. Although they are used most often to reinforce a wound after the removal of sutures or staples, they also be used alone for wounds that are small, non exudative, and under minimal tension. Closure with microporous tape produces far more resistance to infection. Prepare the skin with tincture of benzoic compound to aid adhesion. Place strips with sufficient space between each to allow drainage of fluid from the wound to avoid infection. It should keep dry for 72 hours. Linear wounds in areas with little tension are easily approximated with tape alone, whereas wounds in areas where the skin is more taut generally require that tape skin closure be supplemented with dermal sutures. In addition, tape will not adhere to mobile areas under tension or moist areas.

Topical tissue adhesives

Cyanoacrylate (Dermabond) Tissue adhesive provides an excellent, strong, and flexible method of approximating wound edges. Compared with sutures, staples, and tapes, adhesives provide faster closure. Available as Drops/ sprays, ideal to be used as Periodontal dressing. Dermabond used over sutures at the time of surgery provides extra support, creates an impermeable suture line, decreases the need for postoperative care, and may reduce redness on the suture line.

Advantages include reduced cost, ease of application, absence of needles and suture removal, and higher rate of patient satisfaction;

Disadvantage is lack of strength. This are not good for suturing mucous membranes, contaminated wounds, deep wounds, or wounds under tension.[16]

Conclusion

The choice of suture material is one of the decisive factors in the success of overall surgical treatment. The evolution of suture materials has provided many options in selecting the correct suture for a particular procedure. To date there is not an ideal suture material that could be used under all circumstances in every surgical procedures. Therefore compromises must be made in selecting a suture material. The selection of suture material is based on the condition of the wound, the tissues to be repaired, and the characteristics of the suture material. A factor that should not be forgotten is the price of a suture. In practice, the selection depends on experience of the surgeon and his knowledge of the material.

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Management of abnormal frenum - an overview

Vidya Vishnu[1], Suchithra A[2]

ABSTRACT

The maxillary labial frenum is a normal anatomic structure in the oral cavity, formed by mucous membrane and connective tissue. A thick, hypertrophic or broad fibrous frenum has been accused of causing maxillary midline diastema, interfering with plaque removal, causing tension and gingival recession. A surgical removal of the frenum is indicated in order to prevent these situations or facilitate orthodontic closure of the diastema. The management of such an aberrant frenum is accomplished by performing frenectomy. Frenectomy is the complete removal of the frenum, including its attachment to the underlying bone. There has been a controversy among researchers regarding the need of frenectomy and the time of the surgery. This article gives an overview of the management of abnormal frenum and a short insight to advantages and disadvantages of different techniques.

Key words: Maxillary Labial Frenum, Frenectomy, Frenotomy, Midline diastema

Introduction

Aesthetic concerns have led to an increasing importance in seeking dental treatment, with the purpose of achieving perfect smile. The continuing presence of a diastema between the maxillary central incisors has often been considered as an aesthetic problem. Aberrant frenal attachment is one of the most common causes of midline diastema[1]. Thus the focus on frenum removal has become essential. Although it is a normal structure, its presence has been associated with some unpleasant and even pathological situations. The frena may jeopardize the gingival health by causing a gingival recession when they are attached too closely to the gingival margin, either because of an interference with the proper placement of a toothbrush or through the opening of the gingival crevice because of a muscle pull[2].

Muscular anatomy of the frenum

A frenum is a mucous membrane fold which contains muscle and connective tissue fibres that attach the lip and the cheek to the alveolar mucosa, the gingiva and the underlying periosteum. It extends over the alveolar process in infants with the growth forms a raphe that reaches the palatal papilla. Through the growth of alveolar process as the teeth erupt, this attachment generally changes to assume the adult configuration[3]. But in some instances,the infantile arrangement is retained.

Knox and Young histologically studied the frenulum, and they have reported presence of both elastic and muscle fibres (Orbicularis oris - horizontal bands and oblique fibres). However, Henry, Levin and Tsaknis have found considerably dense collagenous tissue and elastic fibres but no muscle fibres in the frenulum[2].

Classification of maxillary labial frenal attachment

By Placek et al (1974)4–[Fig.1]

1. Mucosal – when the frenal fibres are attached up to the mucogingival junction.

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2. Gingival – when the fibres are inserted within the attached gingiva.

3. Papillary – when the fibres are extending into the interdental papilla.

4. Papilla penetrating – when the frenal fibres cross the alveolar process and extend up to the palatine papilla.

Clinically, papillary and papilla penetrating frena are considered as pathological.

Other variations in frenal attachment are[5][Fig.2]

- Simple frenum with a nodule
- Simple frenum with appendix
- Simple frenum with nichum
- Bifid labial frenum
- Persistent tectolabial frenum
- Double frenum
- Wider frenum

Diagnosis of abnormal frenal attachment

The abnormal frena are detected visually by applying tension over the frenum to see the movement of the papillary tip or the blanch which is produced due to ischaemia in the region.[6]

The frenum is characterized as pathogenic when it is unusually wide or when there is no apparent zone of the attached gingiva along the midline or the interdental papilla shifts when the frenum is extended.

- Pull syndrome (Placek et al, 1974)

A detaching movement of the marginal gingiva transferred from the lip by the frenum has been termed the pull syndrome.

- Tension test

It is used to describe the movement or displacement of marginal gingiva when tension is applied to the lip in an outward, downward & lateral direction.

Indications of frenectomy[7,8]

The frenum is characterized as pathogenic and is indicated for removal in the following situations :

1. An aberrant frenal attachment causing a midline diastema.

2. A flattened papilla with the frenum closely attached to the gingival margin, which causes a gingival recession and a hindrance in maintaining the oral hygiene.

3. An aberrant frenum with an inadequately attached gingiva and a shallow vestibule.

4. In cases with too short labial frenum, which creates problems in upper lip movement, speech etc.

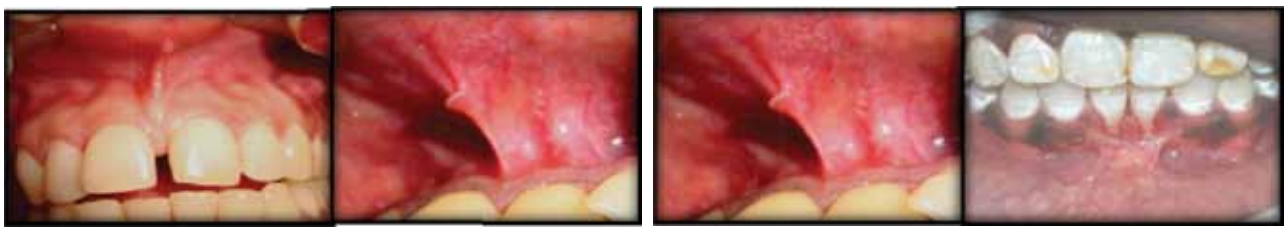
5. When a maxillary labial frenum prevents the installation of a removable denture.

6. To facilitate lip lengthening procedure.

7. In rare occasions, for aesthetic reasons in

(i)Mucosal (ii)Gingival (iii) Papillary (iv)Papilla penetrating

Fig.1. Classification of maxillary labial frenal attachment by Placek



(i)Simple frenum with a nodule (ii)Simple frenum with appendix (iii)Simple frenum with nichum (iv)Wider frenal attachment

Fig.2. Variations in frenal attachment

patients with high smile line.

Management of abnormal frenum

The aberrant frena can be treated by frenectomy or by frenotomy procedures.

Frenectomy is the complete removal of the frenum including its attachment to the underlying bone.

Frenotomy is the incision and the relocation of the frenal attachment.

Frenectomy can be accomplished either by one of the following techniques:

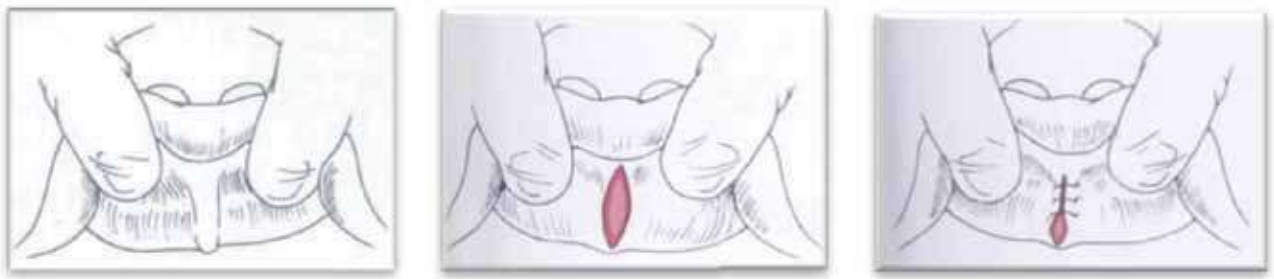
- Conventional (Classical) frenectomy
- Two haemostat technique
- Miller's technique
- V-Y Plasty
- V Rhomboid plasty
- Z Plasty
- Frenectomy with soft tissue graft
- Frenectomy by using electrocautery
- Frenectomy using Lasers

Classical frenectomy

In the classical frenectomy proposed by Archer and Kruger[9], the frenum, interdental tissue and palatine papilla are completely excised leading to exposure of underlying alveolar bone and thus leading to scarring and an unaesthetic scar, but this approach was advised to ensure removal of muscle fibers, supposedly connecting the orbicularis oris with the palatine papilla. [Fig.3]

Two haemostat technique

In this technique, after local anaesthesia, raise the lip, put a haemostat parallel to the alveolar ridge. Another haemostat is placed parallel to the lip at right angle to the first. Then the labial frenum is excised



(i) Abnormal Frenum (ii) Excised Wound (iii) Suturing

Fig.3. Classical Frenectomy

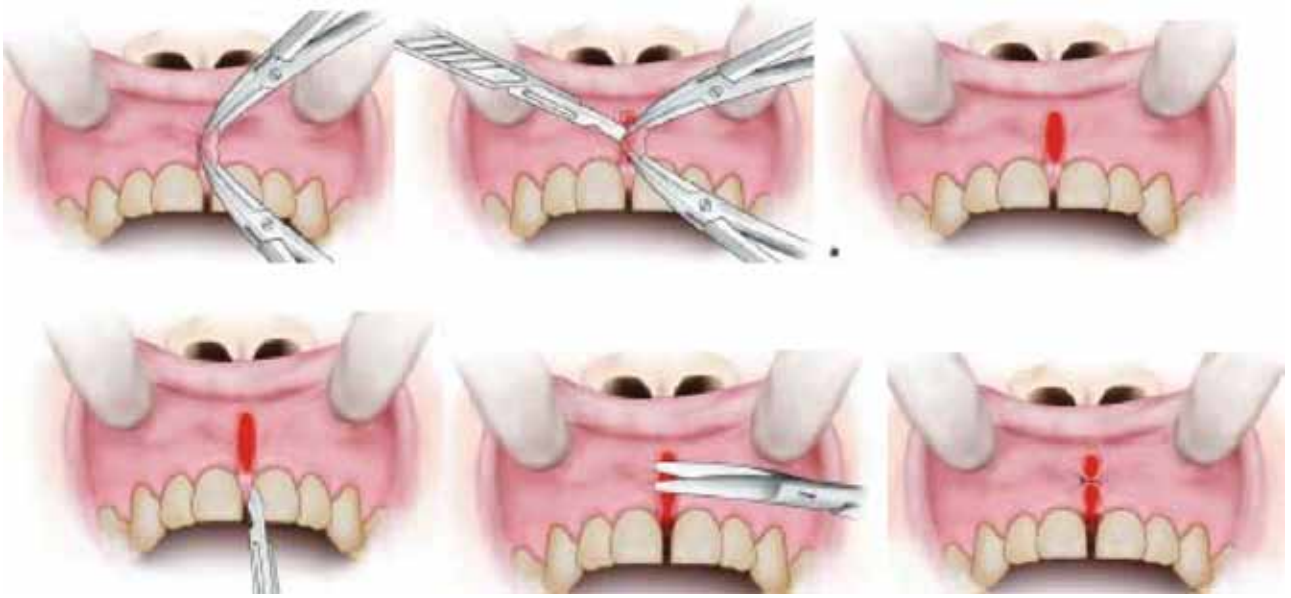


FIG.4. Two Haemostat Technique

by cutting around the outside surfaces of the two haemostat leaving behind a diamond shaped cut with no. 11 or 15 blade. [Fig.4]

Miller's technique

This technique was advocated by Miller PD[2] in 1985 for the post-orthodontic diastema cases. The ideal time for performing this surgery is after the orthodontic movement is complete and about 6 weeks before the appliances are removed. This not only allows healing and tissue maturation, but it also permits the surgeon to use orthodontic appliances as a means of retaining a periodontal dressing.

Procedure:

Excision of the frenulum and exposure of the labial alveolar bone in the midline is done after anaesthetizing the area. A horizontal incision is now made to separate the frenulum from the interdental papilla. Then a laterally positioned pedicle graft (split thickness) is obtained and it is sutured across the midline and a periodontal dressing is placed. [Fig.5]

Advantages of Millers technique :

- Healing takes place by primary intention
- A zone of attached gingiva, matching with adjacent tissue, forms in midline which is pleasing to the individual.
- No unaesthetic scar formation
- No recession of interdental papilla occurs because the transseptal fibers are not severed out.
- The attached gingiva in midline may have a bracing effect which helps in prevention of orthodontic relapse.

Z plasty/ frenuloplasty

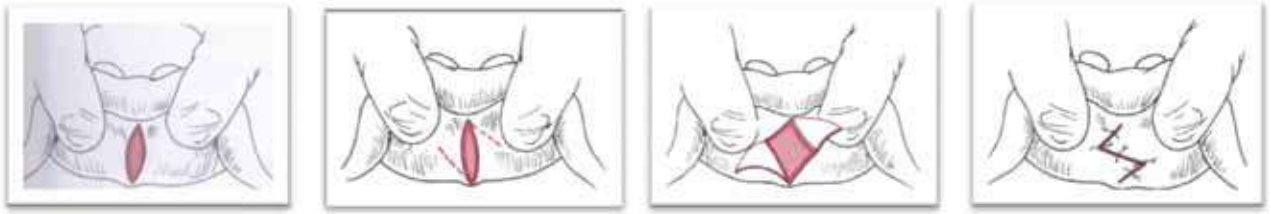
The risk of failure of frenectomy can be reduced by using a technique known as Z-Frenuloplasty, which is a soft tissue surgery used to lengthen a frenulum. It works best when used for hypertrophic thick frenula with a low insertion and a shallow sulcus[10].

Procedure :

The length of the frenum is incised with the scalpel & at each end, limbs at between 60° & 90° angulation, incisions are made in equal length to that of the band. By using fine tissue forceps, with care not to damage the apices of the flaps, the submucosal tissues are dissected beyond the base of each flap, into the loose non-attached tissue planes. Thus, double rotation flaps which are at least 1 cm long are obtained. The resultant flaps which are created are mobilized & transposed through 90° to close the vertical incisions horizontally. Absorbable 5-0 vicryl sutures are placed, first through the apices of the flaps, to ascertain the adequacy of the flap repositioning and

(i) Preoperative view (ii),(iii)Vertical incision mesial to lateral incisor and undermining of the pedicle (iv)Suturing of the pedicle at midline (v) Postoperative

Fig.5. Millers Tehnique



(i)Excision done (ii) Oblique incisions at each end of excision (iii) Flaps reflected (iv) Vertical flaps positioned horizontally and sutured

Fig.6. Z PLASTY

then they are evenly spaced along the edges of the flaps, to close the wound along the cut edges of the attached mucoperiosteum and the labial mucosa. A periodontal dressing is placed. Healing is uneventful, with no hypertrophic scar formation and tension at the frenum area. [Fig.6]

Advantages of Z plasty

- Less soft tissue tension,
- Lengthening of the lip
- Minimal scarring
- Improved lip function.
- Safe, cost effective and results in better functional & aesthetic appearance.
- Allows for tissue healing by primary intention; increasing recovery & reducing the risk of tissue contractures.

Vestibuloplasty with secondary epithelization

This technique is used for extremely wide base of frenal attachment. Incision is made through mucosal tissue and underlying submucosal tissue, without perforating the periosteum. Supraperiosteal dissection is completed by undermining the mucosal and submucosal tissue with scissors. Mucosal flap is then sutured in the most depth of vestibule and the exposed periosteum is allowed to heal by secondary epithelialization. [Fig.7]

(i)Incision starting from apex to base

(ii) Suture placed in the depth of vestibule

Fig.7. Vestibuloplasty with secondary epithelization

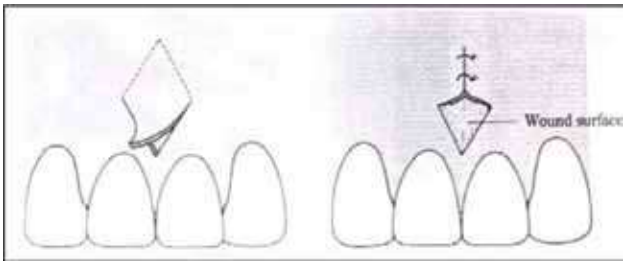


FIG.9. Modification of V-Rhomboid Plasty

V-Rhomboid Plasty

Procedure involves engaging frenum in a haemostat followed by excision of the frenum coronal & apical to haemostat with a blade. Circumferential submucosal tissue is dissected and excised frenum is removed. [Fig.8]

Modification of V-rhomboid plasty

In v-rhomboidplasty, vestibule may become shallow and the band of attached gingiva is narrowed after completion of sutures. So modified V-rhomboid plasty was introduced in which triangular wound is left open in the attached gingiva. Sutures are placed at the alveolar mucosa. Open wound surface is created in the attached gingiva. [Fig.9]

V-Y plasty

V-Y plasty[7] can be used for lengthening the localized area, like the broad frena in the premolar-molar area. The frenum is held with the haemostat and an incision is made in the form of V on the undersurface of the frenum. The frenum was relocated at an apical position and the V shaped incision was converted into a Y, while it was sutured with 4-0 silk sutures. [Fig.10]

Edward's technique[11]

Henry et al. studied thoroughly the histological constituents of frenum & found considerably dense collagenous tissue, loose connective tissue, and elastic

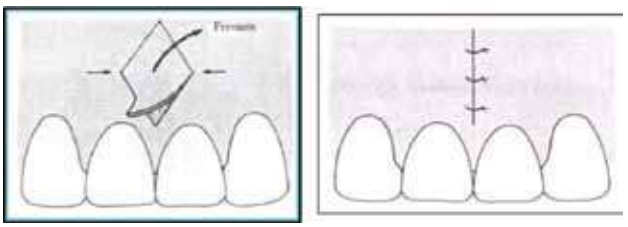


Fig.8. V-Rhomboid Plasty

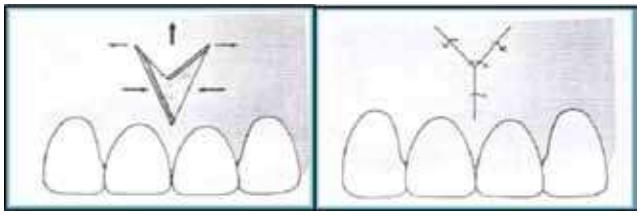


Fig.10. V-Y Plasty

fibers, but no muscle fibers. So Edward, evaluating 308 patients who demonstrated either a diastema or an abnormal frenum or a combination of both, advocated a "conservative surgical procedure" which consists of

1. Apically repositioning the frenum (with denudation of alveolar bone),

2. Destruction of the transseptal fibers between the approximating central incisors,

3. Gingivoplasty of any excess labial and/or palatal tissue in the interdental area.

One of the salient aspects of Edward's technique was the aesthetic maintenance of the interdental papilla. But the healed scar in the midline appeared unaesthetic to the subjects.

Frenectomy with soft tissue graft[12]

This technique was proposed by Coleton & Lawrence. They used free gingival graft combined with frenectomy. This procedure avoids the scar, but a mismatched gingival colour in midline and need of a second surgical site to achieve donor tissue complicate the technique. This technique may result in an unsatisfactory colour match by producing a "keloid," "tattoo-like" or "tirepatch" appearance at the grafted area. Lopes et al evaluated clinically and histologically, the difference in color and blood supply of FGG with or without frenectomy. [Fig.11]

Electrosurgery

Electrosurgery[13] is recommended in patients with bleeding disorders, where the conventional scalpel technique carries a higher risk which is associated with problems in achieving a haemostasis and also in non-compliant patients. Electrocautery offers the advantage of minimal procedural bleeding and there was no need of sutures. [Fig.12]

Frenectomy using soft tissue lasers[14]

Diode lasers are attracted to pigment and frenum are typically thicker fibrous tissue with minimal pigments. The lack of pigment and more fibrous nature of the tissue means that higher energies and some patience are required to ablate this tissue. Er:YAG lasers may ablate frenums faster and can be used in non-contact mode, but the drawback compared to diode lasers is an increased risk of bleeding. Er:YAG (hard tissue) lasers are not well absorbed in hemoglobin as the soft tissue diode lasers are, so hemostasis can be an issue with these wavelength. [Fig.13]

Advantages of Laser over Conventional technique:

- No need of local anaesthesia.
- Painless procedure.
- Less patient apprehension.
- Bloodless operative field, thus better visibility.
- No need of periodontal dressing, therefore no patient discomfort as a result of irritation from the dressing.
- Better healing & less scarring.

Fig.11. Frenectomy With Soft Tissue Graft

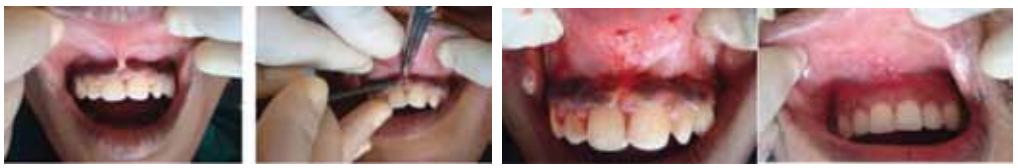


Fig.12. Electrosurgery

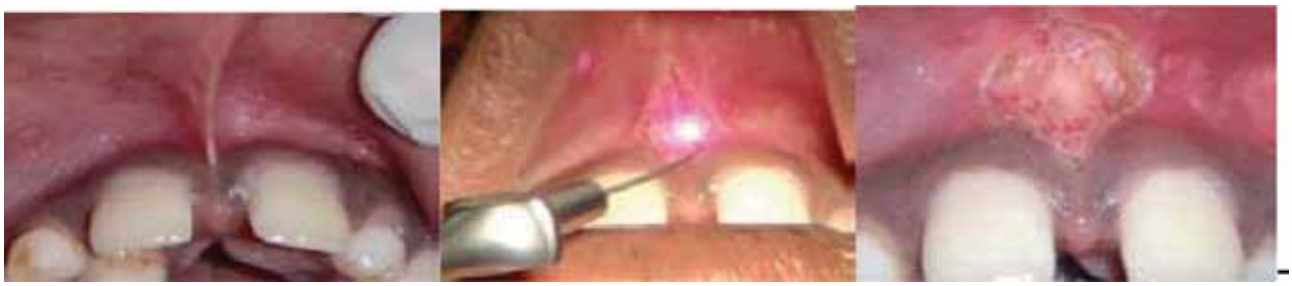


Fig.13. Laser

- Less time consuming.

Controversies in frenectomy

Over the years, the relationship between the maxillary midline diastema and the labial frenum has been the subject of much controversy and confusion. Many orthodontists support the idea that even in cases of an abnormal frenum we should wait till the eruption of all six permanent anterior teeth. During the primary dentition phase, surgical intervention of the labial frenum is not recommended. If the eruption of the permanent canines has failed to close the diastema, frenectomy has a clinical validity only in conjunction with orthodontic treatment[15].

There has been a controversy even among orthodontists concerning the need of all, and the timing for a frenectomy. Some orthodontists support the viewpoint that there is a need for an early removal of the frenum, so as to prevent any obstacles to complete diastema closure. Other orthodontists propose to close the diastema first, and then carry out frenectomy in the hope that the resultant scar tissue will hold together the teeth in close apposition. The ideal time for this technique is after the beginning of orthodontic treatment and about 6 weeks before the appliances are removed. That allows healing, tissue maturation and does not prolong orthodontic treatment[16]. A third body of clinicians rarely, if ever, considers surgical removal of the frenum. They prefer to combat the undeniably increased relapse potential when a diastema is closed, by using bonded retainers on the two central incisors[17].

The advantage of an early excision prior to orthodontic treatment is the ease of surgical access. Access to the surgical procedure is more limited after orthodontic closure and it will not be possible to remove all the residual fibrous tissue thoroughly from the interdental suture area[16].

Since there is quite no evidence concerning the fact that the maxillary labial frenum is the main causative factor for a midline diastema, some orthodontists propose the following therapeutic methodology[16]: Initially, it is necessary for the dentist to make a diagnostic trial, in order to find out whether the frenum is implicated in the pathogenecity of the diastema based on blanch test and intraoral periapical radiograph. If we find out that the diastema in our case is related to the frenum, a maxillary labial frenectomy is indicated.

Periodontists concentrate on the issue of the adequate zone of the attached gingiva. In case of inadequate zone of the attached gingiva, the increased tension causes gingival recession and a frenectomy is recommended. Oral surgeons suggest that in case of a maxillary midline diastema, a small intervention of the frenum is useful. In this way, the closure of the diastema is facilitated and the orthodontic treatment is not affected[18].

Conclusion

There is still a controversy among researchers concerning the need for frenectomy, as well as the right time for frenectomy. Today, the belief that the presence of a maxillary midline diastema does not prompt an early frenectomy predominates. We must wait for a short period, specifically until the eruption of all [6] permanent anterior teeth. This is acceptable if the frenum is not responsible for other pathological situations in the oral cavity. While an aberrant frenum can be removed by any of the modification techniques that have been proposed, a functional and an aesthetic outcome can be achieved by a proper technique selection, based on the type of the frenal attachment. Conventional scalpel method of frenectomy is an age old method and even though the literature is replete with evidence of achieving good results they have their own pitfalls. Though the approaches to the problem of not using the traditional scalpel, like electro surgery and lasers have merits, further improvements can still be attempted.

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Biologic Width or Biologic Barrier: A Review

Mary Diana [1], Divya P V, [2]

ABSTRACT

Although the term biologic width is familiar to most clinicians, many of them are ignorant about the concept and its clinical relevance,the understanding of which can eliminate many restoration and implant associated periodontal complications. A healthy periodontium is a prerequisite for successful outcome of any dental treatment. Encroachment of biologic width can result in gingival inflammation, attachment loss and bone loss necessitating more invasive treatment. Restorative margins often need to be placed subgingivally for esthetic reasons or due to caries. Giving due importance to biologic width assessment prior to any restorative procedures can prevent adverse effects on the attachment apparatus and helps to reinstate teeth with a healthy periodontium. The paper is a review on the concept, clinical significance of biologic width around natural teeth and implants and methods for correction of its violation.

Keywords: Biologic width, Crown lengthening, orthodontic extrusion, biologic width and dental implant.

Introduction

The fundamental function of gingiva is to protect the underlying anatomical structures from mechanical and biological stress. This task is carried out by connective tissue and epithelial attachment respectively. Although science have proved that biologic width is indeed a reality, many clinicians still fail to accept its existence. The biologic width is defined as the physiologic dimension of the junctional epithelium and connective tissue attachment[1]. Nevins and Skurow (1984) defined biologic width as the total of 'supracrestal fibers, junctional epithelium and sulcus'[2].

In dentistry the area of biological width along with sulcus, around natural teeth or an implant is sometimes called Bermuda Triangle or Devil's Triangle. It extends from gingival crest, with tooth/implant on one side and biological width on the other side. This biological width area is most exploited and misused area in dentistry. Like the Bermuda triangle where a number of aircraft and sea vessels are said to have disappeared, the margins of the prosthetic crowns are extended so much that the dentist loses the access and vision where the margin is actually located in the sulcus region[3].

Encroachment of biologic width can result in gingival inflammation, attachment loss and bone loss. In any individual epithelial attachment and connective tissue attachment measurements will change with time and in response to variations in the local and systemic environment. Walton T (2011) suggested name change to 'Biologic Barrier', one that reflects the dynamic and mutable nature of the supra-alveolar connective tissue[4].

Concept of biologic width

In 1977, Ingber et al first described Biologic Width and credited D. Walter Cohen for first coining the term. This term was based on the work of Gargiulo et al.(1961),who described the dimensions and relationship of the dentogingival junction in humans. Measurements were made from the dentogingival components of 287 teeth(325 surfaces were examined histologically and Quantified) from 30 human cadaver jaws, with an age range of 19 to 50 years. He reported

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the following mean dimensions: a sulcus depth of 0.69mm, an epithelial attachment of 0.97mm, and a connective tissue attachment of 1.07mm[5]. Based on his work, the biologic width is commonly stated to be 2.04mm, which represents the sum of the epithelial and connective tissue measurements. This complex protects the subjacent periodontal ligament and the alveolar bone from the attack of a pathogenic biofilm present in the oral cavity[6]. However significant variations of dimensions were observed, particularly the epithelial attachment, which ranged from 1.0 to 9.0mm. The connective tissue attachment, on the other hand, was relatively constant[7].

Similar biologic width dimensions were also reported by Vacek et al. (1994). Heevaluated 171cadaver tooth surfaces, and observed a mean measurements of 1.34mm for sulcus depth, 1.14 for epithelial attachment, and 0.77mm for connective tissue attachment.[8] (Fig. 1)

Maynard & Wilson (1979) divided the periodontium into three dimensions[9];

1. Superficial physiologic: Free and attached gingival.

2. Crevicular physiologic: Gingival dimension from gingival margin to junctional epithelium.

3. Subcrevicular physiologic: Analogous to biologic width described by Gargiulo et al.

Categories/profiles of biologic width

Kois proposed three categories of biologic width based on the total dimension of attachment and the sulcus depth following bone sounding measurements[10,11]: (Table 1)

- Normal crest.
- Low crest.
- High crest.

	Normal crest	High crest	Low crest
Mid-facial measurement	3 mm	<3 mm	>3 mm
Proximal measurement	3-4.5 mm	<3 mm	>4.5 mm

Table 1. Categories of biologic width based on bone sounding measurements

Normal crest patient (85%)

The gingival tissue tends to be stable for a long-term. The margin of a crown should be placed no closer than 2.5 mm from alveolar bone. (Fig. 2)

High crest patient (2%)

This is seen more often in a proximal surface adjacent to an edentulous site. In this situation, it is commonly not possible to place an intracrevicular margin because the margin will be too close to the alveolar bone. (Fig. 3)

Low crest patient (13%)

The low crest patient has been described as more susceptible to recession secondary to the placement of an intracrevicular crown margin. (Fig. 4)

Biologic width in periodontal disease

Attachment loss and increased probing depths has been shown to influence the dimensions of the biologic width. Gargiulo et al. (1961) compared the measurements with respect to periodontal disease and reported a mean biologic width of 2.43 mm at sites without attachment loss. In contrast, the mean biologic width was reduced to 1.71 mm at sites with an attachment loss of up to 6.08 mm.[12]

Similarly, the mean biologic width was smaller at sites with increased probing depths. In the presence of gingival inflammation, the average biologic width was 1.25 ± 0.19 mm[13].

The presence of attachment loss was reported to influence the dimensions of the junctional epithelium. The mean junctional epithelium around teeth without attachment loss was 1.35 mm, it was reduced to 0.71 mm in the case of an attachment loss of up to 6.08 mm. Differences in the mean values of the connective tissue dimensions were not documented for teeth with and without attachment loss. However, significant variability of individual values of the connective tissue dimensions was observed[5].

Evaluation of biologic width violation

Clinical method

The signs of biologic width violation are chronic progressive gingival inflammation around the restoration, bleeding on probing, localized gingival hyperplasia with minimal bone loss, gingival recession,

pocket formation, clinical attachment loss and alveolar bone loss.[14]

Transgingival probing

The biologic width can be identified by probing under local anesthesia to the bone level (referred to as "sounding to bone") and subtracting the sulcus depth from the resulting measurement. If this distance is <2 mm at one or more locations, a diagnosis of biologic width violation can be confirmed.[15]

Radiographic Evaluation

Radiographic interpretation can identify interproximal violations of biologic width. However, on the mesiofacial and distofacial line angles of teeth, radiographs are not diagnostic because of tooth superimposition. Parallel profile radiographic technique has been devised which could be used to measure both length and thickness of the dentogingival unit with accuracy.[16]

Effects of biological width violation

Placing restorative margin within the biologic width frequently leads to: [17]

- Gingival Inflammation.
- Clinical Attachment Loss.
- Bone Loss.

Clinically these sign of biological width violation appear as pain around the restoration margin, Bleeding from the inflamed gingival margin area of involved tooth, gingival recession[18].

Restorative considerations:

Restorative margin placement:

Although supragingival margin is preferred, subgingival margin placement is often unavoidable due to restorative and esthetic reasons.

Margin placement — Rules[19]

1. If the sulcus probes 1.5 mm or less, the restorative margin could be placed 0.5 mm below the gingival tissue crest

2. If the sulcus probes >1.5 mm, the restorative margin can be placed in half the depth of the sulcus.

3. If the sulcus is >2 mm, gingivectomy could be performed to lengthen the tooth, and create a 1.5 mm sulcus. Then the patient can be treated as per rule1.

Restoration Overhangs:

Overhanging dental restorations pose a significant concern as their prevalence has been estimated at 25–76% for all restored surfaces[20]. Most overhanging restorations can be recontoured without replacing the restoration, and this should be considered a standard component of nonsurgical treatment.[6]

Artificial Crown Contours:

Becker & Kaldahl (1981) opined that buccal and lingual crown contours should be "flat", not "fat", usually 0.5mm wider than the CEJ, and that furcation areas should be "fluted" or "barreled out" to accommodate oral hygiene in these areas.[21]

Proximal Contact Relationships:

It is generally accepted that tight interproximal contacts are important for gingival health.

Hancock et al. (1980) evaluated 40 naval recruits to determine the relationship of interdental contacts on periodontal status. Results revealed no significant relationship between contact type and gingival index or probing depth. A significant relationship was seen between food impaction and contact type (greater food impaction at sites with open or loose contacts)22.

Correction of biologic width violations

The concept of tooth lengthening was first introduced by D.W. Cohen (1962). Biologic width violation occurred during restoration margin placement

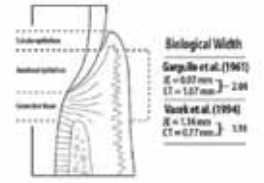

Fig. 1 Biologic width dimensions

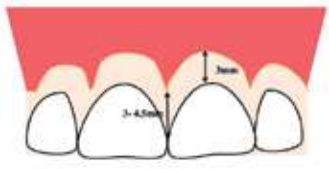
Fig. 2 Normal crest

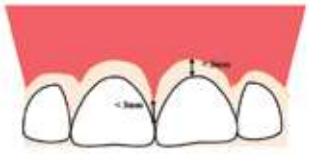
Fig. 3 High crest

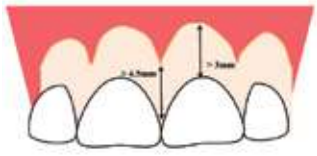
Fig. 4 Low crest

can be corrected by two methods[23]. (Fig. 5)

- Surgically removing bone away from proximity to the restoration margin.
- Orthodontic extrusion of the tooth and then moving the margin away from the bone.

Surgical crown lengthening

Surgical methods for crown lengthening include[6]

(A) Gingivectomy,

(B) Apically positioned flap surgery (APF),

(C) APF with osseous reduction.

Gingivectomy and APF without osseous reduction are limited because bone removal is often necessary to provide adequate distance from the osseous crest to the anticipated restoration margin, allowing for biologic width. APF with osseous surgery is the most common technique for crown-lengthening surgery.

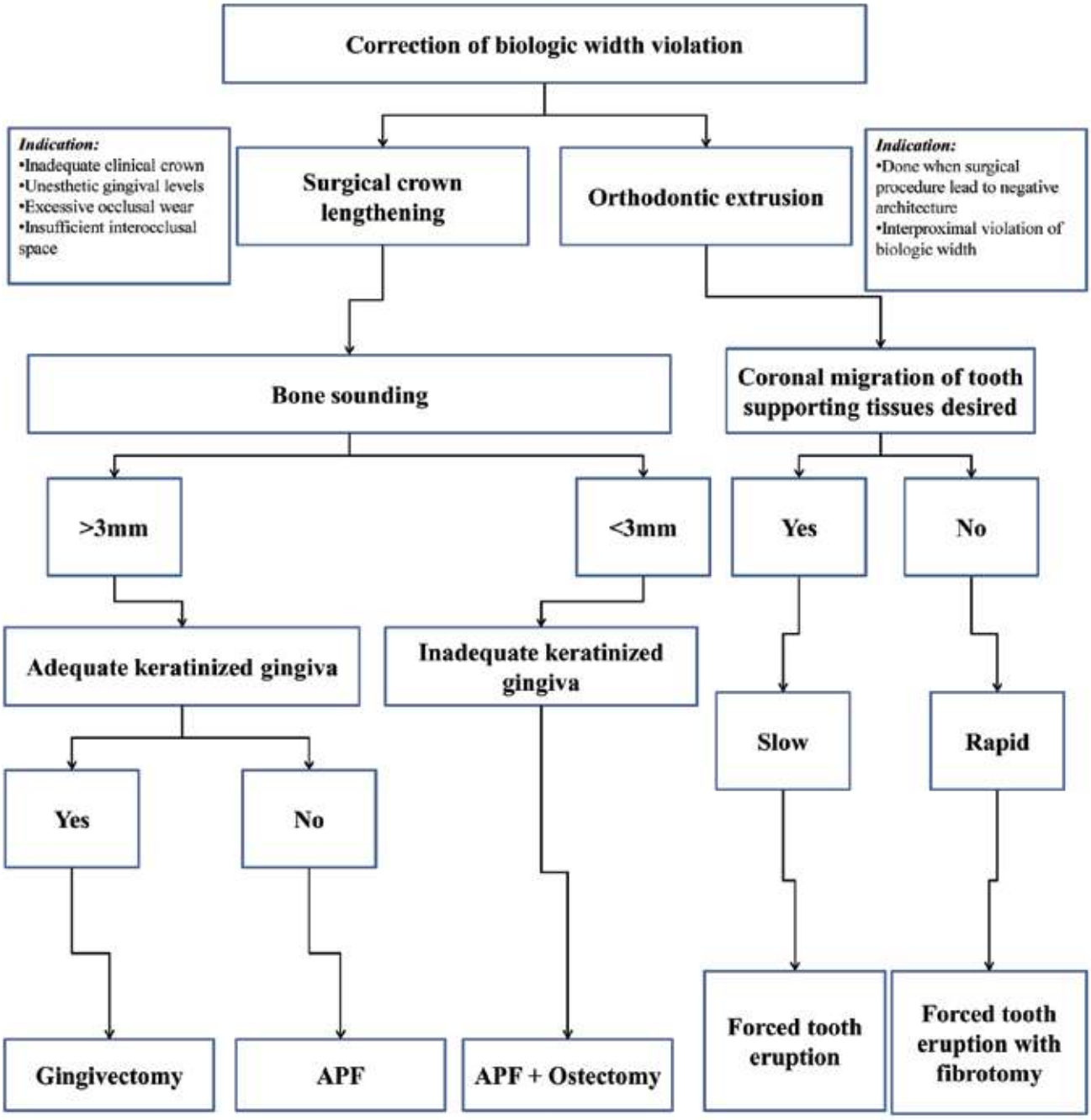

Fig. 5 Decision tree for selection of method for correction of biologic width violation

RESTORATION	PRINCIPLE
Amalgam/ Composite	4mm distance between restorative margin and alveolar crest
Post and Core	5-6mm of exposed tooth above osseous crest (including 1.5mm ferrule length)
Crown	Enough coronal tooth exposure for adequate retention + 4mm distance from restorative margin to alveolar crest

Table 2. Tips for biologic width considerations in different restorative situations

Complications after crown lengthening include poor esthetics due to gingival recession creating "black triangles", root hypersensitivity/resorption and transient mobility[24].

Healing after crown lengthening

Bragger et al. (1992) reported a mean tissue recession of 1.32 mm following surgery, while 29% of sites demonstrated 1–4mm gingival recession between 6 weeks and 6 months postoperatively. Attachment levels or probing depths did not change after 6 weeks of healing. This study lends support to the concept of refraining from restorative treatment for at least 6 weeks following crown-lengthening surgery. Furthermore, due to the possibility of recession, delaying margin placement for 6 months following surgery in areas of esthetic concerns may be indicated[25].

Orthodontic procedures

Forced tooth eruption can be performed in two ways:

- Slow eruption- By applying low orthodontic force, the tooth erupts slowly, bringing the alveolar bone, and gingival tissue along with it[26].
- Rapid eruption- The tooth is erupted to the desired amount over several weeks, with supracrestal fibrotomy performed weekly in an effort to prevent the tissue and bone from following the tooth. Then the tooth is stabilized for atleast 12 weeks prior to surgical correction[27].

Biologic width and dental implants

The protective function of peri-implant mucosa is to establish the required biologic width around an implant. Peri-implant biologic width is 3.08 mm which can have significant influence on the character of soft tissues and depends on a variety of characteristics that include implant design, presence of adjacent teeth and quality of soft tissue[28].

Berglundh and Lindhe (1996) suggested that the soft tissue attachments (biologic width), once established, were nature's mechanism for protecting the zone of osseointegration from the bacterial and mechanical challenges of the oral cavity[29].

Difference between bw around natural tooth and implants		
	Tooth	Implant
Biologic width	Supracrestal- 2.04mm	Subcrestal – 3.08mm
CT composition	Low collagen and high Fibroblast	High collagen and low Fibroblast
Connective tissue fibres	Perpendicular insertion into cementum	Collagen fibres parallel to tooth surface

Peri-implant biologic width

From biologic and esthetic perspectives, the goal must therefore be to prevent loss of perimplant bone during the establishment of biologic width and is part of an important endogenous fall back system. Stability of the biologic width is chiefly dependent on the type of the implant (one piece versus two-piece) and the crestal bone[30].

The fact that one-stage implants have no implant abutment interface leads to less bone remodeling, hence a stable biologic width. This phenomenon is not related to loading and will occur whether the implant is loaded or unloaded (Cochran et al 1997).

It was suggested that as long as the soft tissue covering the implant remains closed (sealed) during healing, crestal bone remodeling does not occur and the crestal height is maintained at the pre-surgical levels Biologic width formation takes place since the time of placement of the implants[31]. Factors affecting the crestal bone loss are surgical trauma, microgap, occlusal overload and crest module design[32].

The development of an implant capable of allowing the growth of biologic width in the coronal direction based on the existing bone level is called perio-integration.

'Platform switching' – the concept

Studies have shown that a minimum thickness of 3 mm of soft tissue is required to allow the formation of biologic seal[33]. The discovery of this concept lies in the simple fact of horizontally repositioning the biologic width by using undersized diameter of prosthetic component in relation to the implant diameter in order to limit peri-implant bone resorption. This in turn maximizes the surface area desired for the soft tissue to attach.

Conclusion

Maintenance of gingival health is one of the keys for the longevity of teeth, as well as for the longevity of restorations. In this context, the biologic width functions as a barrier against the entrance of microorganisms into the internal medium of the periodontal ligament and into the gingival and osseous connective tissue. In addition, owing to its dynamic nature, the terminology of biologic barrier seems more apt. It is extremely difficult to estimate how far intrasulcularly a preparation margin can be placed owing to inter- individual variation in biologic width. Best method is to measure total dentogingival complex width by bone sounding. It provides a better means than following mere statistical values. A lower value should raise concern while a higher value permits extension of preparation margin intrasulcularly. As a general rule, limit the margin placement 0.2- 0.5mm into sulcus.

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Oil pulling: a natural healer

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ABSTRACT

Oil pulling is considered as a natural remedy to improve both oral and systemic health, which has its origin in Ayurvedic medicine. When considering oral aspect, it has been exclusively used for strengthening gums and jaws, and to prevent tooth decay, bleeding gums, oral malodour, cracked lips and dryness of throat, etc, as a traditional Indian folk remedy for many years. It is found to alleviate the accumulation of plaque, prevents cavities, halitosis and gingivitis. Oil pulling with various essential oils improves oral health and overall general health. According to recent literatures, in addition to the use of sesame oil, it shows that coconut oil is also found to be effective for both oral and many systemic diseases and also against fungal, viral and bacterial infections, because of the presence of Monolaurin, a medium chain fatty acid. Other than oral health, oil pulling also helps in alleviating asthma, allergies, diabetes, migraine headaches, chronic fatigue, and chronic skin problems. It works by detoxifying or cleansing the body.

Key words: Oil pulling, Gingivitis, Halitosis, Ssesame oil.

Introduction:

There is a tremendous wealth of information on internet regarding the benefits of oil pulling in systemic and oral health. Nowadays, people search for complementary and alternative medicinal methods to improve or maintain their health, either as the main way to do so or as an adjunct to conventional medicine. Recently, oil pulling has generated a great deal of interest as a way to promote oral health.

Oil pulling is an age-old process mentioned clearly in Charaka Samhita and Sushrutha's Arthashastra. It is referred to as Kavala Graha and Kavala Gandoosha. In Gandoosha, the mouth is completely filled with oil so that, gargling is impossible and is spitted after 3-5 minutes, whereas in Kavala Graha, a comfortable amount of fluid is retained in the mouth and gargled.[1] According to Ayurveda, this procedure is said to cure about 30 systemic diseases and basically down regulates the ageing process.[2]

Oil pulling is considered as a powerful detoxifying Ayurvedic technique that become very popular recently as a complementary and alternative medicinal remedy for many different health ailments. Oil pulling is an ancient Ayurvedic dental technique that involves swishing a tablespoon of oil, such as sesame, coconut, or sunflower oil, in the mouth on an empty stomach for about 15-20 min.[3] Literature also depicts about the use of other oils such as coconut oil, ground nut oil,olive oil, mustard oil and extracts of gooseberries and mango leaf extracts.[4] The regular usage of these edible oils as an oil puller have many advantages over various commercially available mouth washes, as edible oils do not cause any staining, causes no allergic reactions, no bad taste after use,and is readily available in the household.

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Oil pulling is considered as a traditional home remedy to prevent oral malodor, bleeding gums, teeth decay, dryness of throat and cracked lips and for strengthening the teeth, gums and jaws. In addition, this practise is claimed to cure many diseases ranging from thrombosis, eczema, intestinal infection, and diabetes, to bronchitis and asthma.[2]

Methods to perform oil pulling therapy

It is critical to understand that during the oil pulling/oil swishing process, one's metabolism is intensified, which may leads to improved health. The oil pulling is carried out best before breakfast. Inorder to accelerate the healing process, it can be repeated[3] times a day, but always on an empty stomach before meals.

For performing oil pulling therapy, a tablespoon (teaspoon for young children above 5 years of age) of sesame oil is taken in the mouth, sipped, sucked and pulled between the teeth for around 10 and 15 min. The viscous oil turns thin and milky white, and it should not be swallowed as it may contain bacteria. Oil pulling therapy should be followed by proper tooth brushing and is preferably performed on an empty stomach in the morning.[5]

Mechanism of action

Asokan et al. conducted a study inorder to check the antibacterial activity of sesame oil and lignans isolated from sesame oil on oral microorganisms and to check whether saponification or emulsification occurs during oil-pulling therapy.[6] Sesame oil and lignans does not exhibit any antibacterial activity, whereas emulsification of sesame oil occurs during oil pulling therapy and increased consumption of NaOH in titration is a definite indication for a possible saponification process, which might enhances the cleansing action of the sesame oil during oil pulling therapy.

The main mechanisms by which the oil-pulling works are:

1. REDUCES LIPID PEROXIDATION - It is by decreasing free-radical injury to the tissues.

2. VISCOCITY OF OIL -It greatly inhibits bacterial adhesion & plaque coaggregation.

3. SAPONIFICAION -It takes place by alkali-hydrolysis of fat by salivary alkali like bicarbonates.

4. EMULSIFICATION-Insoluble fats gets broken down into minute droplets & dispersed in water. It greatly enhances the surface area of oil, thereby increasing its cleansing action.

5. PRESENCE OF UN-SAPONIFIABLE SUBSTANCE (Sesamin, Sesamolin)-It protects the oral cavity from infections by its anti-oxidant property.

Antibacterial activity of sesame oil

Anand et al. determines the efficacy of oil pulling on dental caries causing S.mutans and Lactobacillus acidophilus. Sesame oil shows a significant antibacterial activity by inhibiting the growth of S. mutans and L.acidophilus.[7] The study shows a significant decrease in the total count of bacteria and the oilpulling practise reduces the susceptibility of a host to dental caries.

Role of coconut oil in "pulling" therapy

Based on a study, as reported by the British Dental Association,shows that "pulling" with coconut oil can reduce cavities.[8] They came up with a conclusion that "coconut oil strongly inhibits the growth of most strains of Streptococcus bacteria including S. mutans – a causative organism of dental caries."

Recognition of the antimicrobial activity of coconut oil has been first reported by Hierholzer and Kabara since 1982.[9] This early research was directed at the virucidal effects because of possible problems related to food preservation.

Recently, results from many studies revealed that monolaurin, the monoglycerides of lauric acid from coconut oil had antimicrobial activity against various gram positive and gram negative organisms, including Escherichia vulneris, Enterobcater spp., Helicobacter pylori, Staphylococcus aureus, Candida spp., including C. albicans, C. glabrata, C. tropicalis, C. parapsilosis, C. stellatoidea and C. krusei, as well as enveloped viruses.[10]

Coconut oil has an unique role as an important functional food in the diet. The difference between coconut oil and other edible oils is that, the presence of medium chain fatty acid in coconut oil, whereas other edible oils are composed of long chain fatty acids. The medium sized monoglycerides is hypothesized to inhibit enzymes involved in energy production, alter bacterial cell wall, leading to the death of the bacteria. Also found that medium-chain saturated fatty acids and

their derivatives act by disrupting the lipid membranes of the organisms.[11,12] Electron microscopic images showed that 15 minutes exposure to monoglycerides caused gram positive cocci cell shrinkage and cell membrane disintegration.

The coconut oil consists of lauric acid, which is used by the body to make the same disease fighting fatty acid derivative monolaurin that babies make from the lauric acid they get from their mother's milk. This monoglyceride monolaurin is the substance that keeps infants from getting viral or bacterial or protozoal infections.[13]

However, the coconut oil may need to be "pre-digested" with an enzyme to make it most effective.[14]

Oil pulling therapy for halitosis

A study has been conducted by Asokhan et al, to elucidate the effect of oil pulling with sesame oil on halitosis and the microorganisms that is responsible for it and to compare its efficacy with chlorhexidine mouthwash. Group I (oil pulling) and Group II (chlorhexidine)included 10 adolescents each.[14]

The parameters assessed were: plaque index, Marginal gingival index, organoleptic (ORG) breath assessment (ORG 1), self assessment of breath (ORG 2) and benzoyl-DL-argininenaphthylamide (BANA) test from tongue coating samples on days 0 and 14 of the experimental period.

The result showed comparisons of both the pre- and post therapy values of plaque and modified gingival index score showed a statistically significant difference (P =0.005 and 0.007, respectively) in Groups I and II. There was a definite reduction in the ORG 1, ORG 2 scores and BANA test Score in both Groups I and II. From the above mentioned study, it can be concluded that oil pulling therapy has been equally effective like chlorhexidine on halitosis and organisms,associated with halitosis.

Oil pulling on plaque and gingivitis:

A study on the effect of oil pulling on plaque and gingivitis has been conducted by Amith et al. Study showed swishing with refined sunflower oil in the mouth greatly reduces gingivitis and plaque load. They found out that there was a net decline in mean plaque scores from baseline to 45 days amounting to 0.81 ± 0.41. Hence, plaque scores have reduced by 18-30% and gingivitis has reduced by 52-60%.[15]

Thaweboon et al. evaluated the effect of oil pulling using rice bran oil, palm oil, sesame oil,coconut oil, corn oil, sunflower oil and soy bean oil on the biofilm models formed by S. mutans, Candidaalbicans, Lactobacillus casei. It was shown that antimicrobial activity against S. mutans and C. albicans was exhibited by coconut oil. Sunflower oil had antifungal activity against C. albicans. whereas, Sesame oil had antibacterial activity against S. mutans,also L. casei found to be resistant to the oils tested. The use of edible oils for oil pulling as a home remedy to treat dental caries is based on evidence based research.[5]

Oil pulling therapy with sesame oil:

A randomized controlled triple blind clinical trial study done by Asokan et al. to evaluate the efficacy of oil pulling therapy by sesame oil on dental caries causing S. mutans count in plaque and saliva, plaque induced gingivitis. Sesame oil was given to the study group to suck and pull in the morning 10 min before tooth brushing whereas the control group was asked to swish with 0.2% chlorhexidine mouthwash 30 min before tooth brushing.

Study showed a significant reduction in colony count of S. mutans in plaque sample and decrease in plaque, gingival index score in the study and control group. Hence,based on the clinical study, it is reported that the use of sesame oil (idhayam oil) pulling therapy is an effective home remedy than chlorhexidine mouthwash and recommended.[16]

Advantages and disadvantages of oil pulling:

It causes no staining, has no lingering aftertaste, and causes no allergic reactions. It is five to six times more cost-effective than the commercially available mouthwashes and is,moreover, readily available in the household. It has got therapeutic effects against various systemic conditions.[17]

The downside of this is, that it has got a longer time of application and has shorter substantivity.

Systemic effects:

Oil pulling is considered as to have beneficial effects on systemic health also and has been claimed to cure diabetes, eczema, thrombosis, intestinal infection, respiratory diseases, etc..

The proponents of oil pulling would agree and would add that oil pulling could have positive systemic effects on other, more serious conditions. They feel that toxins and bacteria from the body might be expelled through the tongue and trapped in the oil and removed from the body.[18]

Conclusion:

Ayurvedic medicine has grown successively despite the negligible amount of scientific evidence. Oil pulling is considered as a natural alternative. Growing sector of the population desires natural products for their health care. So,oral health care professionals should be knowledgeable enough about the alternative and complimentary products. The exact mechanism of action of oil pulling therapy is still not clear hence further research studies with various essential oils like sesame oil will lead to wonderful approach in the field of dentistry for maintenance of oral health. More long term studies are needed in larger populations, to assess the wide-ranging effects, that oil pulling may have on various oral conditions.

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Transcription factors NF-κB and STAT3 in periodontal disease

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ABSTRACT

The importance of host response in periodontal disease is well recognized in the recent times. The microorganisms in dental plaque can activate host factors and initiate a series of cellular events resulting in periodontal tissue destruction. Transcription factors are present in cytoplasm of most mammalian cells and mediate cellular events on activation. NF-κB and STAT3 are two important transcription factors implicated in many inflammatory immune diseases. Activators of these factors are abundant in periodontal disease and many of their target genes can mediate periodontal tissue destruction. This comprehensive review is aimed at detailing the role of transcriptions factors NF-κB and STAT3 in periodontal disease.

Key words: periodontal disease, NF-κB, STAT3, transcription factors, host response

Introduction:

Periodontal disease is a chronic inflammatory disease of multifactorial etiology. Microbes in the dental plaque initiate periodontal inflammation, but host response to microbial insult is mainly responsible for progression of the disease and periodontal tissue destruction. [1] Genetic and environmental risk factors influence this host response and can aggravate the disease process.

Host response to microbial challenge occurs through a series of cellular events ultimately resulting in production of inflammatory mediators. During these cellular events transcription factors play an important role. They are factors present in cytoplasm of most cells and upon activation they are transported to nucleus to regulate the production of multiple inflammatory mediators. The role of inflammatory mediators of periodontal tissue destruction has been reviewed extensively in the past, but little is known about transcription factors in periodontal pathogenesis. NF-κB (nuclear factor kappa) and STAT3 (Signal Transducer and Activator of Transcription) are two critical transcription factors in chronic inflammatory diseases and their role in periodontal disease is detailed here.

NF-κB

NF-κB is a family of transcription factors comprising of NF-κB1 (p50), NF-κB2 (p52), RelA (p65), RelB and cRelfirst described by Sen and Baltimore in 1986 as the regulator of kappa light chain gene in murine B lymphocytes. [2, 3] The classic form of NF-κB is the combination of p50 and p65. [2] NF-κB exist as a complex with a protein known as inhibitor kappa B (IκB) inside the cytoplasm. During activation IκB degrades and the released NF-κB enters the nucleus to activate its target genes. [2]

STAT3

The Janus kinase-signal transducers and activators of transcription (JAK-STAT) signaling is also activated during inflammation. When the receptors associated with STAT signaling are stimulated by appropriate cytokines, receptors get dimerized and

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activates members of the STAT family via tyrosine phosphorylation resulting in formation of homo or heterodimers of STAT.[4] These dimeric molecules are transported to the nucleus by exposure of nuclear localization signal to stimulate transcription of the responsive genes. [4] At present, seven STAT family members have been identified i.e., STAT1, STAT2, STAT3, STAT4, STAT5a, STAT5b and STAT6. They have similarity in their molecular structure and function, but play diverse physiological roles in a wide variety of biological processes.[4] STAT3 is mainly activated by gp130 cytokines like interleukin-6, oncostatin M (OSM), leukemia inhibitory factor (LIF) and ciliary neurotrophic factor. [5]

NF-κB and STAT3 synergistically control a common set of genes encoding for cytokines and chemokines.[6] NF-κB and STAT3 crosstalk was observed in many inflammatory conditions and cancers.[6, 7]

NF-κB and STAT3 in periodontal pathogenesis

Many activators of NF-κB are seen in periodontal disease like bacterial LPS, prostaglandin E2, IL-1β, TNF alpha, stress, viruses etc. [8] Target genes of NF-κB including cytokines, matrixmetalloproteinases (MMP), cyclooxygenase2 (COX 2), inducible nitric oxide synthase (iNOS), RANKL etc are beneficial to the progression of periodontal disease. [8] NF-κB activation by various activators like IL-1b, TNF áhas been observed in cultured periodontal cells which lead to production of inflammatory mediators like prostaglandins and MMP. [9, 10]

NF-κB activation plays an important role in connective tissue destruction as well as bone resorption [11].

STAT3 pathway is activated mainly by IL-6 and IL-6 family of cytokines which signal through gp130 receptors. [5]Other activators of STAT3 like IL-22, INF-γ, TNF-α, IL-1, IL-4, IL-10, IL-17/23 and LPS etc are also seen associated with periodontal disease. [1]Signaling target of STAT3 include INF-γ, TNF-α, IL-1,IL-4, IL-6, and IL-10, MMP-2 and MMP-9, iNOS, VEGF,COX-2 etc and many of these molecules have biologically significant role in periodontal pathogenesis. [1]

Risk factors of periodontal disease like diabetes, smoking, obesity etc activate STAT3 and NF-κB leading to persistent inflammation. [12-15] STAT3 and NF-κB activation has also been reported in many chronic inflammatory diseases like rheumatoid arthritis which share similarities with periodontal disease in pathogenesis. [16]Periodontal disease is recently identified as a risk factors for various systemic diseases like cardiovascular disease, diabetes mellitus, pre term low birth weight and even cancers [17, 18]. NF-κB and STAT3 activation can be considered as an important molecular mechanism to explain the link between periodontal disease and systemic diseases. NF-κB and STAT3 activation in periodontal cells has been reported in many experimental studies [10,11]. But the evidence from human studies are scarce and future studies are required in this area.

Clinical implications

Plaque control and antimicrobial therapy were the conventional periodontal management strategies. But due to better understanding of the role of host response in periodontal pathogenesis, host modulation therapy (HMT) is also introduced recently as an adjunct to conventional methods. HMT is beneficial mainly in high risk category patients [1] Drugs mainly utilized for host modulation were sub antimicrobial dose doxycycline (SDD), NSAIDs and bone sparing drugs like bisphosphonates and the molecular targets include MMPs, cytokines and prostaglandins. A more recent approach for HMT is by inhibiting major signaling pathways of inflammation where many molecules can be simultaneously targeted. [19]

Signaling blockade has been attempted and found to be useful in experimental periodontitis and also in other similar chronic inflammatory diseases. [20, 21] Inhibitors of NF-κB and STAT3 can be utilized for blocking the signaling pathways in chronic inflammatory diseases like periodontitis. Local delivery of these agents in periodontal therapy may reduce the side effects associated with systemic exposure and minimize the risk of general immunosuppression. Gene delivery also may appear promising for periodontal therapy. Future controlled clinical trials are required to identify safe and effective NF-κB and STAT3 inhibitors that can revolutionize the existing periodontal treatment strategies and thereby help to improve patient's oral as well as systemic health.

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JSPIK

Tooth fracture- A comprehensive management protocol

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ABSTRACT

Tooth fracture presents an esthetic, functional and psychologic derangement to the patient. Optimal management of fractured tooth requires a team effort, in which the periodontist plays a pivotal role. Extensive tooth fracture compromising the retention and resistance of the planned restoration is a primary indication for crown lengthening. Crown lengthening aims to increase the clinical crown length of a tooth or teeth for either esthetic or restorative (functional) purposes or a combination of both. Surgical crown lengthening should be done based on two important principles: preservation of biologic width and adequate keratinized gingiva so as to maintain a healthy periodontium. This review serves as a guide for the clinician to select the right treatment modality for fractured tooth and provides two decision trees for the above purpose.

Keywords: Tooth fracture, Crown lengthening, Biologic width.

Introduction

Tooth fracture presents an esthetic, functional and psychologic derangement to the patient. The majority of dental injuries involves the anterior teeth, especially the maxillary central incisors.1 A number of techniques have been reported in literature for rehabilitation of grossly mutilated teeth including restorative, orthodontic and surgical techniques. Extensive tooth fracture compromising the retention and resistance of the planned restoration is a primary indication for crown lengthening. Crown lengthening aims to increase the clinical crown length of a tooth or teeth for either esthetic or restorative (functional) purposes or a combination of both .The concept of tooth lengthening was introduced by D W Cohen in 1962 and is presently a procedure that often employs some combination of tissue reduction/removal, osseous surgery, and /or orthodontics for tooth exposure. The amount of tooth structure exposed above the osseous crest must be enough to provide for a stable dentogingival complex and biologic width to permit proper tooth preparation and account for an adequate marginal placement, thus ensuring a good marginal seal with retention for both provisional and final restorations.[2]

Surgical crown lengthening

Surgical crown lengthening procedure is based on two principles: biologic width (BW) establishment and maintenance of adequate keratinized gingiva (KG) around the tooth. The Biologic width is defined as the dimension of soft tissue that is attached to the portion of the tooth coronal to the alveolar bone crest. Studies indicate that a minimum of 3 mm of space between restorative margins and alveolar bone would be adequate for periodontal health, allowing for 2mm of BW space and 1 mm for sulcus depth.[3,4] An adequate width of KG (>2 mm) should be maintained around a tooth for gingival health whenever possible.

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The aesthetic rehabilitation of patients with mutilated teeth generally involves a multidisciplinary approach. However, during the restorative procedures, negligence of periodontal tissues is of common occurrence, and is often the cause of failure. Hence, it is essential to maintain a healthy periodontium and the biologic width while restoring teeth. Following are the factors to be considered before formulating a treatment protocol.[2]

Clinical evaluation

1. Sulcus/ pocket depth
2. Biologic width
3. Gingival margin- Osseous crest distance
4. Pulpal involvement.
5. Apical extent of fracture
6. Gingival health
7. Furcation involvement
8. Loss of mesial, distal or occlusal space
9. Final margin placement

Radiographic evaluation

1. Alveolar crest to CEJ distance
2. Apical extent of fracture line
3. Pulpal involvement
4. Root length
5. Root form
6. Root trunk length
7. Crown root ratio

Treatment protocol

The treatment for fractured tooth for each patient is different. To determine which is the best available treatment option, is a perplexing question to the clinician. For that, we have to address the above mentioned clinical and radiographic parameters. Primarily, we have to assess the apical extent of the fracture line. If this fracture lies 2mm or more above the Cemento enamel junction, then proper restorative procedures can be carried out, as it will not violate the health of periodontium. But, if the distance between fracture line and CEJ is less than 2mm, periodontal treatment modalities for crown lengthening have to be considered before restorative procedures.

Before embarking on periodontal procedures, crown root ratio has to be assessed. A 1:1 ratio has been defined as the minimum acceptable ratio when the periodontium is healthy and the occlusion is controlled.[5, 6] A crown root ratio less than or equal to 1:1 is considered for periodontal management. A crown root ratio greater than 1:1 is not ideal for crown lengthening and treatment option is extraction followed by immediate implant placement / socket preservation or fixed prosthesis later on.

Assess the gingival architecture of the concerned tooth. If there is disharmonious gingival architecture on presentation or expected after surgical crown lengthening or if there is an esthetic need to preserve interdental papilla, orthodontic extrusion is the best treatment modality.[7] Forced eruption allow the crown margins to be placed on the sound tooth structure while maintaining a uniform gingival contour that provides improved aesthetics without involving adjacent teeth. It also maintains acceptable crown-root ratio, biological width and good periodontal health.[7]

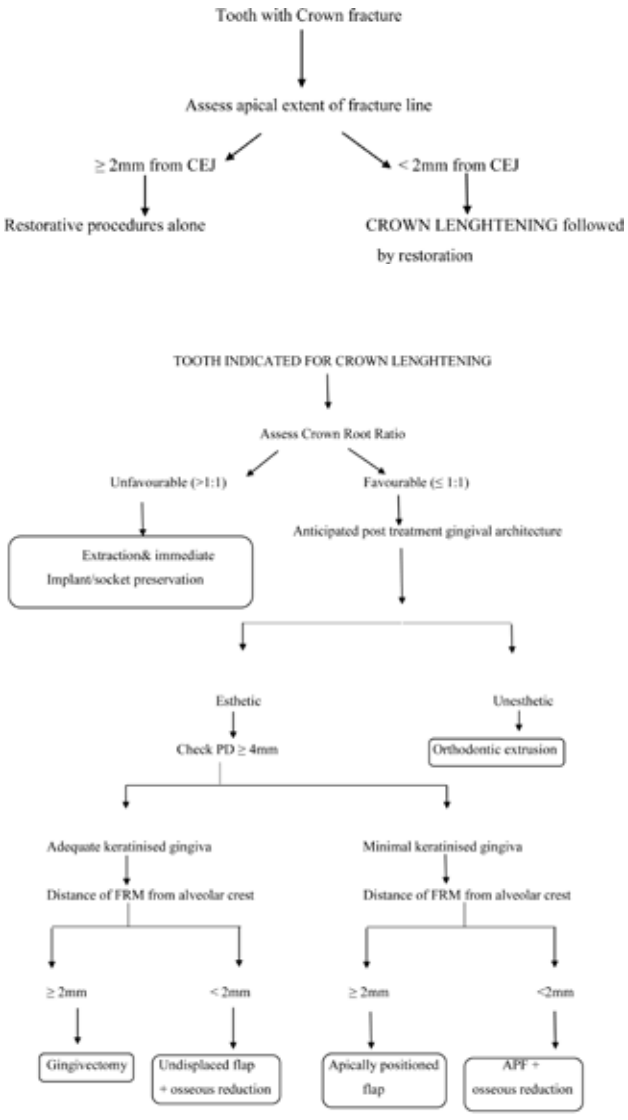
Another important factor that needs clinician's attention is final restorative margin (FRM). The reference line with respect to making incisions and performing osseous surgery for restorative crown lengthening is FRM. With the final restoration margin (FRM) as a reference, the BW is established around the tooth by removing bone to ≥3 mm below the FRM. Literature review on the FRM – alveolar bone distance suggests various measurements given by different authors. To avoid encroachment of the biological width, the dentist should measure probing depths before starting treatment.[8] Bragger and colleagues (1994) showed that creating a distance of 3mm from FRM to alveolar crest was necessary for stable periodontium. Rosenberg and colleagues (1999) and Weinburg and Eskow (2000) recommended a distance of 3.5-4mm; whereas Wagenburg (1989) recommended at least 5-5.25mm. According to Fugazzotto (2011) and Cohen (2007) distance between FRM and alveolar crest should be a minimum of 4mm. If a ferrule effect is required for endodontically treated teeth that need to be restored with a cast post and core, then 4 to 5 mm of clearance is needed from the finish margin to the alveolar bone.

Depending on the clinical and radiographic presentation, the following are the periodontal

treatment modalities.

Clinical findings	Treatment approach
Adequate Keratinised Gingiva FRM to alveolar crest > 2mm	Gingivectomy
Adequate Keratinised Gingiva FRM to alveolar crest < 2mm	Undisplaced flap with osseous reduction
Inadequate Keratinised gingiva FRM to alveolar crest > 2mm	Apically positioned flap
Inadequate Keratinised gingiva FRM to alveolar crest < 2mm	Apically positioned flap with osseous reduction.
Need to preserve adjacent structures and interdental papillae on neighbouring teeth. Crown root ratio < 1	Orthodontic extrusion
Fracture line close to CEJ or below with unfavourable crown root ratio for orthodontic extrusion	Extraction immediate implant/ socket preservation and delayed implant

Decision tree



Conclusion

the treatment of traumatised teeth require a close collaboration of different dental specialities. Comprehensive fracture management requires good clinical planning and a coordinated multi-disciplinary approach. The restoration and rehabilitation of traumatised teeth requires concerted efforts of the restorative team in which the periodontist plays a central role. This review is a ready reckoner for right decision making, which is the key to treatment success.

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