Volume 3 | Issue 1 | November 2009

JSPIK

Journal of the
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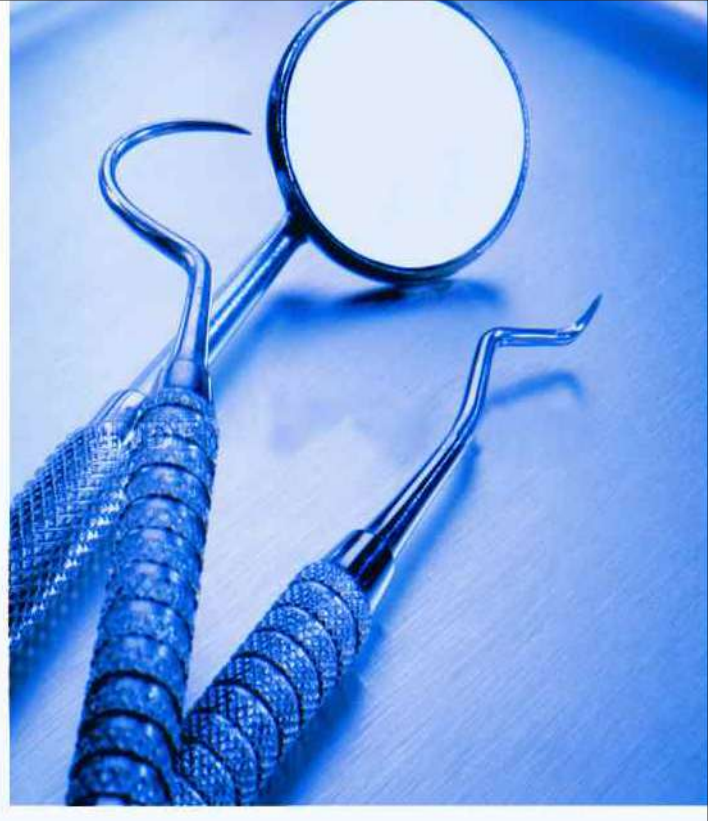

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President's Message

Dear Colleagues,

I would like to share with you all, the philosophy of a great English Neuro Surgeon named Trotter; which has greatly influenced my professional work.

"As long as medicine is an art ;its chief and characteristic instrument must be human faculty. We come therefore to the very practical question of what aspects of human faculty it is necessary for the good doctor to cultivate. The 1 st to be named must always be the power of attention: of giving one's whole mind to the patient without the interposition of oneself. It sounds simple but only the very greatest doctors ever fully attain it. It is an active process and not either mere resigned listening or even politely waiting untill you can interrupt. Disease often tells its secrets in a casual paranthesis"

I appeal to SPIK family to achieve excellence and high ethical standards in your professional work. A Merry X' mas and wishing you all a wonderful new year, 2010.

Truely yours,

Raju Kurian Ninan
President, SPIK

Editorial

Warm Greetings To All Of You.

The first issue of JSPIK brings with it the wishes for prosperity, joy, and contentment to all its members. I hope that all of you will agree with me in that with growing periodontal awareness among the community, our profession is gaining more and more appreciation. So from our part, it is our professional obligation to rise to the expectations, adhere to strict quality maintenance measures in our office and also update our knowledge by subscribing to as many as periodontal magazines, find out time to go through all articles and attending scientific sessions.

Also let me request all of you to send articles and interesting case reports to JSPIK, so that we can widen our knowledge base and manage clinical problems more effectively by learning from others. Let us stand united, brainstorm vigorously to improve oral health of our people.

A Merry X' mas and prosperous new year.

Yours Truely,

Dr. Siby T Chennankara

Message from the Secretary

Welcome you all to the first issue of our Journal - 'JSPIK' of this current SPIK year.

Our society, SPIK have been in the path of steady progress since the very beginning and no doubt the whole credit goes to our sincere, committed and active office bearers. The meritorious contribution of the senior members and past presidents to the overall development of our society is highly appreciable.

The first half of this SPIK year have already passed, we could conduct the Installation of office bearers along with the Second Annual Conference, Family get together, three Executive committee meetings, three Scientific programmes and also release of official publications - News letter and Journal.

At the same time I would like to remind you that active involvement and whole hearted co-operation are required from all the members to keep up the momentum of our activities.

It shall be our endeavor to bring in more and more members into our fold which will not only strengthen our Society but also will boost, our professional ethics and standards.

Let us enjoy every aspect of our profession and our association. Let us all enjoy being together, enjoy our interaction with other professional bodies and enjoy lending a helping hand to improve the community around us.

Your creative ideas, positive suggestions, healthy criticism will definitely improve our SPIK activities.

Looking forward to your continued support,

Happy X' mas and new year.

With regards,

Dr. Santhosh Sreedhar
Secretary - SPIK

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An Interdisciplinary Approach in the Management of a True Combined Lesion - "Merging to Emerge"

Dr. Rosamma Joseph

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Abstract

Endoperiodontal lesion is effectively managed by endodontic and regenarative periodontal therapy. A relevent case report is presented in this article.

Introduction

The periodontal tissues and root canal system have a common developmental, anatomic, and functional relationship. They are connected by anatomic structures such as apical foramina, accessory canals, and dentinal tubules as well as by pathologic pathways such as tooth perforations and fractures[1,2]. Therefore, an infection originating in one tissue will affect these other related tissues. An "endo-perio" lesion involves a condition where both the pulp and the periodontium are diseased simultaneously in what appears to be a single periodontal lesion. First described by Simring and Goldberg in 1964[3], such lesions are very common and pose diagnostic and therapeutic challenges to the clinician.

Case Report

A 35 year old female reported to the OPD at the Dept. of Periodontics, Govt. Dental College, Calicut with complaints of mobile lower teeth of few months duration. It was associated with bleeding from gums during brushing and frequent pus discharge in relation to her upper front tooth. The patient gave a history of trauma to her upper front tooth 5 years back. She had previously undergone extraction of 37 due to mobility. Her systemic history was non contributory.

Clinical examination revealed moderate plaque and calculus scores and generalized gingival inflammation. Generalised periodontal pockets ranging from 5mm-8mm were present associated with Grade III mobility of 31,32. Discoloration and extrusion of 12 was also noted along with a draining sinus in the attached gingiva. A 12 mm deep periodontal pocket was detected in relation to the distal aspect of the same tooth; however no mobility was noted.

Full mouth IOPA radiographs revealed generalized loss of alveolar bone with combined angular and horizontal defects. The upper anterior region showed large periapical radiolucency associated with an angular bone defect along the distal aspect of 12. A pulp viltalty test on 12 gave a negative result. Based on these findings a diagnosis of generalized chronic periodontitis and a Periodontal – endodontic lesion associated with 12 was made.

Treatment Plan

As the prognosis of 31, 32 was poor, extraction was advised. Plaque control was instituted and scaling and root planing was performed. Endodontic therapy was done on 12 and the patient was revaluated after 6 weeks. On revaluation, persistent periodontal pockets were detected and therefore full mouth periodontal surgery was advised. The periapical lesion in relation to 12 was also persistent.

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Surgical Periodontal Therapy

Full mouth periodontal surgery was performed using full thickness access flaps. A regenerative technique using alloplastic bone graft containing a combination of bioactive glass and hydroxyapatite granules (Grabio Glascera, Dorthom Medi Dents, India) was employed to address the Grade II furcation involvement in 36.

Combined Periradicular- Regenerative Periodontal Surgery in 13-23 region

A combination of Peri-radicular surgery (apicoectomy) and regenerative periodontal surgery was carried out for resolution of Periodontal- endodontic lesion on 12. Internal bevel incisions initiated from the crest of the gingival margin and 2 vertical incisions were used to elevate a full thickness flap. On reflection, a large peri radicular defect associated with a dehiscence was observed in relation to 12. Apicoectomy was carried out to permit complete debridement of the defect and thorough root planing was performed. Root conditioning was performed using a solution of doxycycline and the defect was filled using an alloplastic bone graft containing a combination of bioactive glass and hydroxyapatite granules (Grabio Glascera, Dorthom Medi Dents, India). A bioabsorbable GTR membrane (Periocol GTR, Eucare Pharmaceuticals P. Ltd., India) was then used to cover the bony defect. Multiple interrupted silk sutures were used to secure the flap to their original position. A non eugenol periodontal dressing was applied. The patient was prescribed antibiotics and analgesics and a soft diet was instituted. Sutures were removed on the 7th post operative day and the healing was uneventful. Occlusal rehabilitation using esthetic ceramic crown on 12 and a direct composite veneer on 11 was performed 5 months after surgery. The patient is currently on maintenance periodontal therapy.

Discussion

Various classifications have been put forward for classifying periodontal- endodontic lesions. Simon, Glick and Frank (1972)[4] classified such lesions based on the primary source of infection into 5 groups:

1. Primary periodontal lesions
2. Primary endodontic lesions
3. Primary periodontal lesions with secondary endodontic involvement
4. Primary endodontic lesions with secondary periodontal involvement
5. True combined lesions

True combined lesions occur when an endodontically induced periapical lesion exists at a tooth that is also affected by periodontitis. The two lesions can either merge or exist separately. Merged lesions form by ongoing marginal attachment loss or by exacerbations of apical periodontitis.

In the present case the history of trauma and discoloration of 12 provide evidence for the presence of a persistent endodontic lesion. The patient also had generalized loss of attachment as indicated by the probing pocket depths and bone destruction. Therefore in this case the periodontal and endodontic pathologies were coexistent and hence the lesion may be classified as a true combined lesion.

The long term prognosis for the combined lesion is related to the extent and configuration of periodontal attachment loss. In such cases the endodontic treatment is more predictable and likely to be successful. However, the periodontal component is more difficult to treat. It cannot resolve as long as the endodontic lesion is present, but effective endodontic treatment cannot eliminate the periodontal pocket. The ability to eliminate the periodontal component of the defect ultimately determines the success of therapy. After successful endodontic therapy, the residual periodontal pocket can be more predictably treated. For these reasons, endodontic therapy should precede periodontal pocket elimination[5].

The goals of periodontal therapy include the reduction or elimination of tissue inflammation induced by bacterial plaque and its by-products, correction of defects or anatomical problems caused by the disease process, and regeneration of lost periodontal tissues as a consequence of disease destruction.

Guided tissue regeneration employs barriers, nonresorbable or bioabsorbable, to control the cell and tissue repopulation of the periodontal wound[6]. The biological rationale of the procedure is based on prevention of migration of the epithelial periodontal tissues into the osseous defect, allowing time for bone and other attachment tissues to heal. During normal healing, it appears that the epithelial tissues migrate rapidly into the wound, preventing regeneration. The placement of a barrier membrane would thus ensure that the detached root surface becomes repopulated with cells from periodontal ligaments capable of forming bone, periodontal ligament and cementum. Also, the membrane provides sufficient space for optimal wound stability, an essential prerequisite for periodontal regeneration to occur[7].

The case presented here highlights the use of combination of a resorbable GTR membrane and an alloplastic bone graft in the treatment of an intrabony defect associated with periodontal- endodontic lesion. Trombelli L[7] and Needleman I et al[11], based on their systematic reviews in 2005 have concluded that GTR provides an additional benefit, in terms of clinical attachment level gain, probing depth reduction and defect fill, when compared to Open flap debridement in the

treatment of deep intraosseous defects. Studies indicate the additional use of a bone grafting material provide superior results than barrier membranes used alone. The combination of barrier membranes and grafting materials result in histological evidence of periodontal regeneration, predominantly bone repair.[8,9] GTR combined with bone substitutes results in slightly more probing depth reduction and greater gain in hard tissue probing as compared to GTR alone at re-entry surgery.

von Arx T, Cochran DL in 2001[10] proposed a novel classification system (fig 1) of periodontal – endodontic lesions based on application of guided tissue regeneration as a treatment option. The authors proposed that for treatment of communicating apicomarginal defects (class IIb lesions); a barrier technique might become an important aid to regenerate tooth-supportive structure.

Conclusion

The pulp and the periodontal attachment are the two components that enable a tooth to function in the oral cavity. The simultaneous existence of pulpal problems and inflammatory periodontal disease can complicate diagnosis and treatment planning. The present case provides proof that combined endodontic and regenerative periodontal surgery using GTR is successful to conserve strategic teeth which are of esthetic and functional importance.

Class I:	Periapical bone defect without marginal lesion
Ia	Lingual/palatal cortex not eroded
Ib	Lingual/palatal cortex eroded (with a buccal surgical approach, this will result in a transosseous or through-and-through bone defect)
Class II:	Periapical lesion (with or without lingual erosion) and concomitant marginal lesion
IIa	No communication between the separate lesions
IIb	The two lesions are fused = communicating apicomar ginal or endodontic-periodontal lesion
Class Iil:	Lateral or furcational lesion (with or without marginal lesion)
IIIa	No communication to alveolar crest/marginal periodontium
IIIb	Communication to alveolar crest/marginal periodontium

Fig 1: Classification of Periodontal – Endodontic Lesions on application of Guided Tissue Regeneration[10]

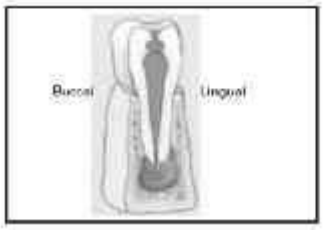


Fig 3 *Class Ia lesion (bone defect confined to periapical region).*

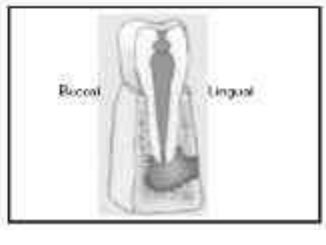


Fig 4 *Class Ib lesion (periapical bone fdefect with erosion of lingual cortical plate).*

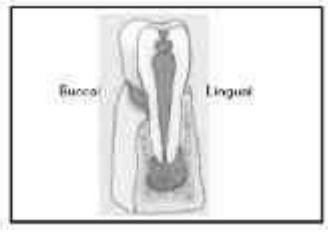


Fig 5 *Class IIa lesion (periapical and concomitant marginal lesion without communication).*

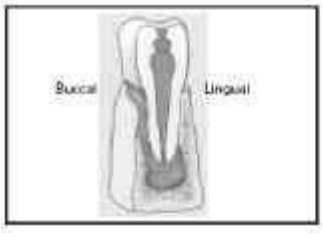


Fig 6 *Class IIb lesion (periapical and concomitant marginal lesion with communication).*

Fig 7 *Class IIIa lesion (lateral juxtara dicular lesion).*

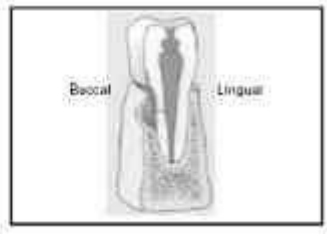
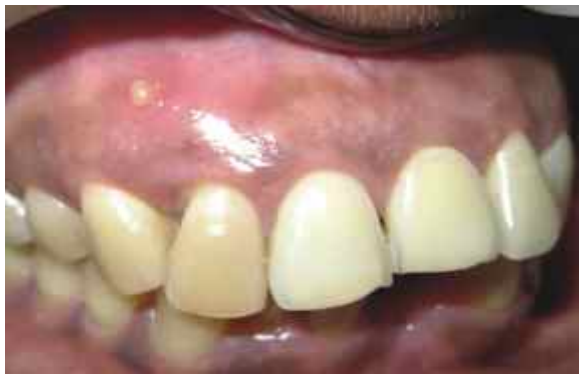
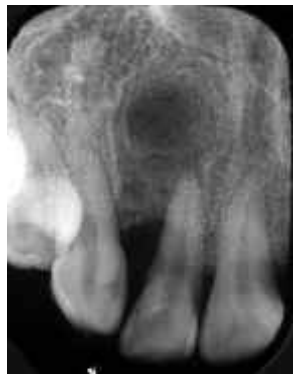


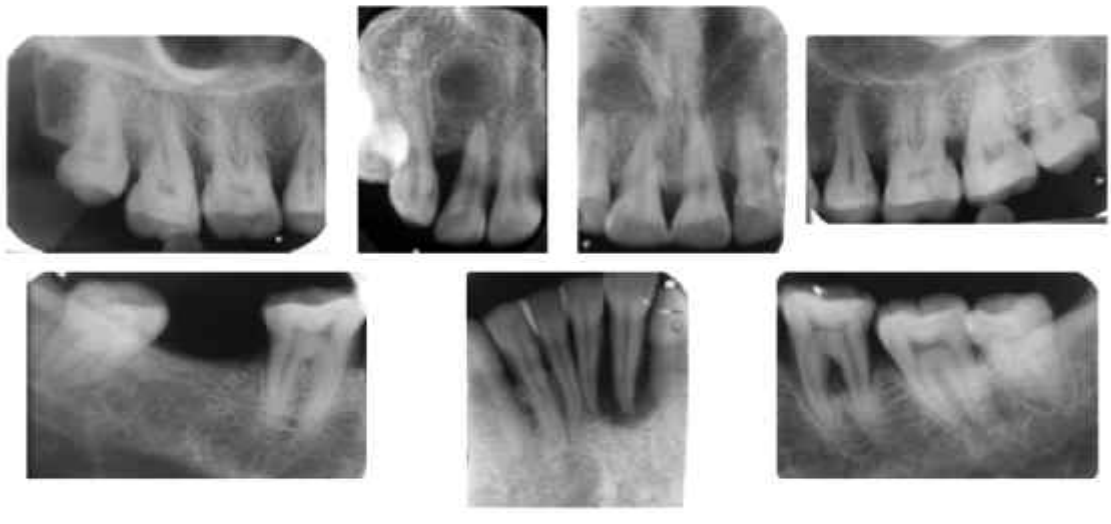
Fig 8 *Class IIIb lesion (lateral juxtaradicular lesion with communication to marginal lesion).*

Pre- Treatment



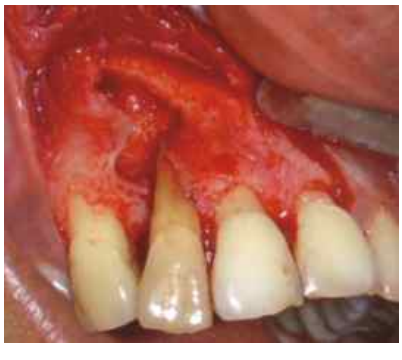


Full Mouth IOPA Radiographs

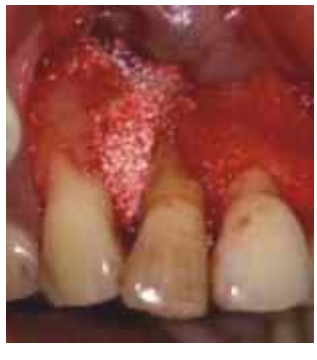


Combined Periradicular- Regenerative Periodontal Surgery in 13-23 region

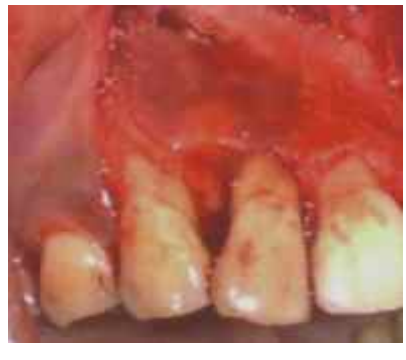
Exposure of the defect after Apicoectomy



Defect filled with alloplastic bone graft

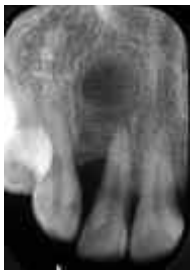


Defect covered with GTR membrane

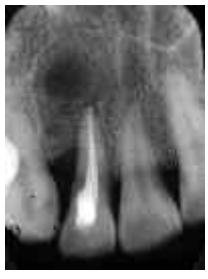


Sutures Placed

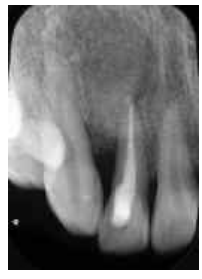
5 weeks post operative



Pre- Treatment

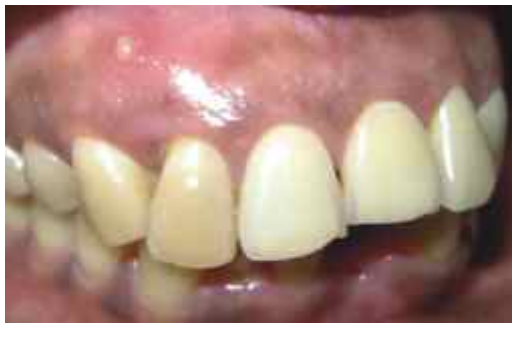


Post- Endodontic Treatment



5 months after surgery

Pre-Treatment



Post Treatment

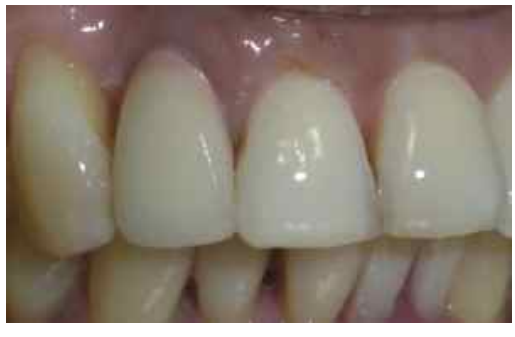


Fig 1: Classification of Periodontal – Endodontic Lesions on application of Guided Tissue Regeneration[10]

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TOOTH MOBILITY

DR P. JAYACHANDRAN*

Abstract

Tooth mobility is a challenging factor in connection with periodontal diseases. This review reaches all aspects of tooth mobility including terminologies, etiology, pathogenesis, diagnosis and management.

Introduction

"Mouth is the mouthpiece of mind". For the power of speech and beautiful smile, healthy gums and teeth are essential. Mobile teeth are concerned not only for the patient, but also to the dentist, because it is the critical stage where the tooth lies between two "S" i.e. to be saved or sacrificed.

Tooth mobility is an important feature of periodontal disease. This is evidenced by the large number of devices and method of tooth mobility assessment that have been developed and tested. Tooth mobility has been considered and investigated as an indirect measure of the functional condition of the periodontium as well as possible aggravating co-factor for periodontal disease.

When the percentage of teeth extracted as a result of caries and periodontal disease were compared from age 1-74 by Kelly and Harvey (1974) it was found that, teeth recommended for extraction due to periodontal disease acceded that of caries or other reasons. Tooth mobility is considered to be of paramount significance of establishment of diagnosis, prognosis and treatment plan.

Definitions

(AAP periodontal literature review 1996).

Fremitus

A palpable or visible movement of a tooth when subjected to occlusal forces.

Tooth mobility

The degree of looseness of tooth beyond physiologic movement.

TERMINOLOGIES

1. Physiologic / Normal Mobility

It refers to the limited tooth movement or tooth displacement that is allowed by the resilience of an intact and healthy periodontium when a moderate force is applied to the crown of the tooth examined. (Muhlemann 1951).

2. Pathologic tooth mobility

It is any degree of perceptible movement of a tooth faciolingually, mesiodistally or axially when a force is applied to the tooth. (M.J. Perlitsh 1980)

3. Altered tooth mobility

It is an alteration of the mobility characteristics of a tooth, which represents a transient or permanent change in periodontal tissues. (Giargia and Lindhe 1997). An increased tooth mobility may be associated with different physiologic or pathologic phenomena while decrease mobility usually is result of therapy.

4. Functional mobility

Functional mobility is the movement of teeth during function or parafunction.

5. Normal tooth mobility

It is more during early mornings and progressively decreases. Muhlemann (1960) reported that tooth mobility was 0.4 – 0.12 mm for 500 gm force applied. The incisors have the highest (0.1 – 0.12 mm) and molars the lowest (0.4 –0.8mm). Children and females exhibit higher values than adults and males respectively.

6. Increased / Static tooth mobility:

It is a form of stabilized mobility. It is usually due to trauma from occlusion but may be due to periodontal diseases. But the periodontal structures have become adapted to an altered functional demand. It is self limiting and normal for that tooth with remaining bony support.

7. Increasing / Progressive tooth mobility:

It is of progressive nature and can be identified only through a series of repeated tooth mobility measurements carried out over a period of several dogs or weeks.

8. Hypermobility

A form of increased mobility persisting after completion of periodontal treatment. It is often referred to as "residual mobility". It has 2 phases, a developing phases and a permanent phase.

9. Reduced tooth mobility

As seen in ankylosed tooth after failing replantation or if autogenous bone grafts are placed in contact with detached root surface.

STAGES OF TOOTH MOBILITY

Tooth mobility occurs in 2 stages, namely

a. Initial / Intra socket stage (ITM)
b. Secondary stage (STM)

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a. Initial / Intra socket stage (ITM):

When a small force is applied to the crown of a tooth, the resistance of tooth supporting structures against displacement of root is low in the initial phase of force application and crown is moved by 0.5 mm to 0.1 mm which was called initial tooth mobility (ITM) by Muhlemann (1954) and is the result of intra alveolar displacement of root. ITM depends on structure and organization of periodontal ligament. In the pressure zone there is a 10% reduction in the width of the periodontal ligament and in the tension zone there is a corresponding increase.

Muhlemann and Zander (1954) stated that "There are good reasons to assure that the initial displacement of the roots (ITM) corresponds to a reorientation of periodontal membrane fibers into a position of function redness towards tensile strength".

The magnitude of the "Initial tooth mobility" varies from individual to individuals from tooth to tooth, and is mainly dependent on the structure and organization of PDL. The ITM value of ankylosed teeth is therefore zero.

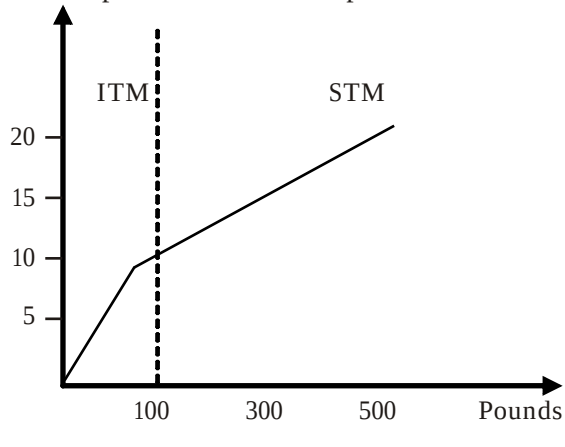
b. Secondary stage: (STM)

When a large force (up to 500 pounds) is applied to the crown, the fiber bundles on the tension side can not offer sufficient resistance to further root displacements. The additional displacement of the crown that is observed in "Secondary tooth mobility" is allowed by distortion and compression of the periodontal ligament in the pressure side.

Tooth mobility Curve (Lindhe 4h edition)

Muhlemann (1954) in a series of studies, used progressively increasing forces (50 – 1500 g) and observed that the crown displacement was directly, but not linearly related to the magnitude of the force applied. The results from a large number of assessments showed that this force / displacement relationship had a typical pattern which could be illustrated by a double sloped curve. The 2 parts of this curve were defined as "Initial" (ITM) and "Secondary" (STM) tooth mobility components.

The ITM part described the displacement obtained by the use of forces smaller than 100 gm and was represented by a steep curve indicating that, within this range, small increments of the force resulted in a relatively marked dislocation of the tooth. On the other end STM part of the curve i.e., forces ranging between 100-50g or >100g was represented by less steep line. The STM component of the curve slope indicated that, in the 2nd phase, a comparatively large force increment was needed to obtain a certain additional tooth movement.



- According to Muhlemann (1960) the magnitude of "Secondary – TM" i.e. the excursion of the crown of the tooth when a force of 500 pounds is applied.
- Forces varies between different types of teeth
  (Example incisors 10-12 / 100 mm)
  Canines 5-9 / 100 mm.
  Premolars 8-10 / 100 mm
  Molars – 4-8 / 100 mm
- The force applied is larger in children than in adults and It is also larger in females than males and increases during certain conditions like pregnancy. Further more tooth mobility seems to vary during the course of the day; the lowest value is found in the evening and largest in the morning.

Aetiology

Local and systemic factors are implicated.

Local factors:

1. Bone loss or loss of tooth support:

Loss of tooth support (bone loss) can result in mobility. The amount of mobility depends on the severity and distribution of bone loss at individual root surfaces, the length and shape of the roots, and the root size compared with that of the crown. A tooth with short, tapered root is likely to be loose than one with normal size or bulbous roots with the same amount of bone loss.

2. Trauma from occlusion:

Mobility from trauma from occlusion occurs initially as a result of resorption of the cortical layer of bone, leading to reduced fiber support and later as an adaptation phenomenon resulting in a widened periodontal space.

3. Hypofunction:

As seen in teeth without antagonist causing widening of periodontal ligament initially due to reduced stresses.

4. Extension of inflammation from the gingiva or from the periapex into the periodontal ligament

The spread of inflammation from an acute periapical abscess may increase tooth mobility in the absence of periodontal disease.

5. Periodontal surgery temporarily increases tooth mobility

6. Pathology of jaws like tumors, cysts, ostemylitis etc.

7. Tooth morphology (Crown and root shape)

· Flat contacting surface – thin, narrow septa – less bone support.

· Convex or bell shape – flat and wide septa more bone support increase in number, size of roots – more one support.

8. Overjet and over bite are directly proportional to tooth mobility

II. Systemic causes:

1. Age: Mobility is positively related to age of the individual (Wasserman 1973) changes in the PDL that have been reported with aging include decreases numbers of fibroblasts with more irregular structure.

2. Sex and Race: Slightly higher incidence seen in females and blacks. (Wasserman 1973).

3. Menstrual cycle: Burdine and Friedman (1970) observed increased horizontal tooth mobility during 4th week of menstrual cycle.

4. Oral contraceptives: Studies by knight and wade, Das, Bhowmick and Dutta indicate that periodontal disease and attachment loss were more common among women on pills. However Friedman (1972) found tooth mobility to be less among ovulatory drug users.

5. Pregnancy: Ratietschak (1967) has reported tooth mobility in pregnancy and has attributed it to physico-chemical changes in periodontium.

6. Systemic disease: Certain systemic diseases aggravate periodontal disease i.e. Papillon Lefevere syndrome, Down's syndrome, Neutropenia, Chediak Higashi syndrome, Hypophosphatasia, Hyperparathyroidism, Acute leukemia, Paget's disease etc.

7. Bone factor concept of Glickman: "When a generalized tendency toward bone resorption exists, bone loss initiated by local inflammatory processes may be magnified. This systemic influence on the response of alveolar bone has been termed the bone factor in periodontal disease. The bone factor concept, developed by Irving Glickman in early 1950s, envisioned a systemic component in all cases of periodontal disease. In addition to the amount and virulence of plaque bacteria, the nature of the systemic component, not its presence or absence, influences the severity of periodontal destruction.

Although the term "Bone factor" is not in current use, the concept of a role played by systemic defense mechanisms has been validated, particularly by studies of immune deficiencies in severely destructive types of periodontitis, such as juvenile forms of the diseases.

Factors affecting development of tooth mobility:

1. Magnitude, frequency and character of masticatory forces.

2. Amount of fiber bundles, in the periodontium and strength of alveolar bone.

3. Physical resistance of the periodontium.

4. Direction of the masticatory stress.

5. Physiological and systemic factors, which influence metabolic process of cells such as blood circulation in periodontium, age, nutrition and general health.

6. Para-functional habits and forces.

MICROSCOPIC FEATURES OF TOOTH MOBILITY –

(M.J. Perlitsh, DCNA 1980; Vol.24, No.2: 177)

The excessive forces produce molecular physico-chemical alteration of the ground substance and fibrous components of the tissues, atrophic, degenerative and necrotic changes. Increased compression and tension of the periodontal ligament are seen. With severe tension, widening, thrombosis, hemorrhage and tearing of the periodontal ligament and bone resorption are seen. There is temporary depression, in mitotic and the rate of proliferation and differentiation of fibroblasts, collagen and alveolar bone.

Grant et al (1995) found significant proportions of Campylobacter rectus and Peptostreptococcus microbes and elevated levels of Porphyromonas gingivalis in pockets of mobile teeth than that of non mobile ones.

Dynamics of tooth mobility

(Periodontal literature Review 1996 AAP)

Tooth mobility seems to occur in two stages (Muhlemann 1967):

- First, there is an initial or intra vascular stage where movement within the socket is associated with redistribution of the fluids, interstitial contents and fibers.

- The second stage occurs gradually and includes elastic deformation of the alveolar bone proper in response to increased forces.

FORCES ON TEETH:

1. Normal or Physiologic forces:

Teeth and their supporting structures are subjected to severe occlusal forces of up to 50 mg during mastication. The presence of tissue fluids and arrangement of PDL fibers are such that these intermittent heavy forces can be properly accommodated without tissue destruction. These forces are transmitted through PDL fibers to the alveolar bone proper.

2. Orthodontic or Pathologic forces:

When unidirectional orthodontic force exceeding the adaptive capacity is applied to a tooth, pressure is exerted on side of the periodontal ligament in the direction of the force and tension on the opposite site. In undermining resorption, bone is resorbed towards the socket with eventual resorption of the socket wall, where as on tension

side, new bone is opposed on the socket lining maintaining the constant width of the periodontal space.

3. Jiggling Pathologic Forces:

Forces applied to a tooth during function and parafunction may exceed the adaptive capacity. These jiggling forces may move a tooth in a faciolingual, mesiodistal, or vertical direction, along the X, Y or Z axis. As a result of pressure being exerted in all direction, the entire periodontal ligament behaves as it is subjected to pressure only. A force that exceeds the tooth's adaptive capacity leads to the lesion of trauma from occlusion.

CORRELATION BETWEEN TOOTH MOBILITY AND OCCLUSION

Trauma fromOcclusion

"When occlusal forces exceed the adaptive capacity of the periodontal tissue, injury results." The resultant injury is termed trauma from occlusion.

So occlusal trauma is described as trauma to the periodontium from functional and para-functional forces causing damage to the attachment apparatus of the periodontium by exceeding its adaptive and reparative capacities.

- Generally, two forms of occlusal trauma are recognized:

1. Primary occlusal trauma

It is a condition in which the pathologic occlusal forces considered the principal etiology for occlusal changes in the periodontium.

2. Secondary occlusal trauma

It occurs when the periodontium is already compromised by inflammation and bone loss. Teeth with a reduced adaptive capacity and compromised periodontium may then migrate when subjected to certain occlusal forces. Factors such as frequency, duration and velocity of those occlusal forces, not just their magnitude, may be of greater significance in the development of tooth hypermobility. This mobility is a common clinical sign of occlusal trauma.

TOOTH MOBILITY INDICES

a. Miller's Index (1938):

1. The first distinguishable sign of movement.

2. The movement of the tooth which allows the crown to deviate within 1 mm of its normal position.

3. Easily noticeable and allows the tooth to move more than 1mm in any direction or to be rotated or depressed in the socket.

b. Modified Miller's index:

Score of 0, 0.5, 1, 1.5, 2.5, 3 are utilized.

c. Prichard's index (1972)

1. Slight mobility.

2. Moderate mobility.

3. Extensive movement in a lateral or mesiodistal direction combined with vertical displacement in the alveolus.

4. Or sign can be used for added refinement.

d. Wasserman's Index (1973)

1. Normal

2. Slight mobility less than 1 mm of buccolingual movement.

3. Moderate mobility – up to approximately 2 mm of buccolingual movement.

4. Severe mobility – more than 2 mm of movement.

e. Nyman's Index (1975)

Zero degree – Normal – less than 0.2 mm

Degree 1 – Horizontal / Mesiodistal mobility of 0.2 – 1mm

Degree 2 – Horizontal / Mesiodistal mobility of 1-2 mm.

Degree 3 – Horizontal / Mesiodistal mobility exceeding 2mm and / or vertical mobility.

f. Flezar's Index (1980)

M_o - Firm Tooth

M_1 - Slight increased mobility

M_2 - Definite to considerable increase in mobility but not impairment of function.

M_3 - Extreme mobility, a loose tooth that would be incomparable in function.

g. Glickman's Index (1972)

- Normal mobility
- Pathologic mobility
- Grade I – slightly more than normal
- Grade II – moderately more than normal
- Grade III – Severe mobility faciolingually and or / mesiodistally combined with vertical displacement.

h. Lovdal's Index (1959)

First degree – teeth that were somewhat more mobile than normal.

Second degree – teeth showing conspicuous mobility in transversal but not axial direction. Third degree – teeth being mobile in axial as well as on transversal direction.

Measurement of tooth mobility

(JISP 2003 Vol. 6 No.2, Panejjer JP 1973)

Measurement of tooth mobility is important to evaluate the condition of periodontium in research oriented studies and for diagnosis and treatment planning.

There are numerous mobilometers, to name a few.

1. Elbrecht's indicator (1939)
2. Werner's Oscillator (1942)

3. Dreyfus vibrator (1947)
4. Zinrner's oscillograph (1949)
5. Manly's device (1951)
6. Muhlemann's Macro-periodontometer and Micro-periodontometer, Pictons gauge (1957)
7. Parfitt's transformer (1958)
8. Joel's technique (1958)
9. Goldber's device (1961)
10. Korber's transducers, USAFSAM periodontometer (O'Leary and Rudd 1963)
11. Pameijer's device (1973)
12. Laser method (Ryden 1974)
13. Persson and Svensons device (1980)
14. Periotest (Schulte 1987, Simons AG, Germany)

PERIODONTOMETER: (Muhlemann 1957)

By means of the "Periodontometer" a small force (100 pounds) is applied to the crown of a tooth. The crown starts to tip in the direction of the force. The resistance of the tooth supporting structures against displacement of the root is low in the initial phase of force application and the crown is moved only5 / 100 to 10 / 100mm.

PERIOTEST

➢ The Periotest device dynamically measures the reaction of the periodontium to a defined percussive force applied to the tooth produced by a tapping device.

➢ It is connected by table to a unit which controls functions and analyses measurements. A metal rod housed in the interior of the hand piece, the tapping head is accelerated to a present speed of 0.2 m/s (meters per second) and maintained at constant speed by compensation for the influence of friction and gravitation. Upon impact, the tooth is slightly deflected and the tapping head is decelerated.

➢ The contact time between the tapping head and the tooth varies between 0.3 and 2 ms (milli seconds). The contact time is shorter for teeth whose alteration ability of the periodontium is greater and which are less mobile. The tapping head is electro magnetically retracted into the hand piece. In 4 seconds, 16 exact defined tapping impulses are applied to the tooth and 10,000 signals for deceleration are registered and analyzed by the measuring unit. Invalid measurements are recognized as such and eliminated.

➢ Since the contact times are not clinically meaningful, the unit displays a value called the "Perio test value" (PTV). The value is calculated from the contact time between tapping head and tooth and ranges from – 8 to +50, corresponding to four different degrees of mobility. (JDR 1992, Vol.27;184) Schulte et al)

➢ Goodson (1988) confirmed the correlation between PTV and clinical mobility index (MI). He showed the periotest differentiates between 39 units for the mobility indices 0 to 3.

➢ In a comparative study, stepwise multiple linear regression analysis at Periotest values compared with clinical parameters demonstrated that the influence of bone loss is far more important than other clinical parameters, indicating that the periotest value dependent to a large extent on bone loss. The greater the alveolar bone height, the lower the periotest value.

➢ Of the other diagnostic valves tested, the pocket depth is correlated somewhat more strongly with periotest value than recession and the papillary hemorrhagic index. The correlation between the PTV and bone loss was stronger in the maxilla than in the mandible. Periodontally healthy teeth also had higher periotest value in the maxilla.

➢ In tooth with non inflammatory recession and simultaneously TMJ dysfunction syndrome contribute to a significant increase in the periotest values for incisors. This is possibility due to increased alveolar destruction by bruxism.

➢ In addition, the high sensitivity of the periotest method provides a means for early recognition of changes in the periodontium on a result of periodontal diseases.

➢ Even though standardization of the grading of mobility would be helpful in diagnosing periodontal disease and in evaluating the outcome of treatment, these devices are not widely used.

As a general rule, mobility is graded clinically with a simple method such as the following:

The tooth is held firmly between the handless of two metallic, instruments or with one metallic instrument and one finger, and an effort is made to more it in all directions.

"It is not the length of the excursive movement of the crown that is important from a biologic point of view, but the displacement of the root within the remaining periodontal ligament". Increased crown displacement (tooth mobility) may also be detected in a clinical measurement where a "Horizontal" force is applied to teeth with angular bony defects / or increased width of the periodontal ligament. If this mobility is not gradually increasing – from one observation interval to the next – the root is surrounded by a periodontal ligament of increased width but normal composition. This mobility should be considered "Physiologic" since the movement is a function of the height of the alveolar bone and the width of the periodontal ligament.

Only progressively increasing tooth mobility which may occur in conjunction with trauma from occlusion and which is characterized by active bone resorption and which indicates the presence of inflammatory alterations with in the periodontal ligament tissue, may be considered "Pathologic".

Miller's original classification	Mobility index	PTV
No movement distinguishable	0	-8 to + 9
First distinguishable sign of movement	1	10 to 19
Crown deviates with 1 mm of its normal position	2	20 + 29
Mobility is easily noticeable, and the tooth moves more than 1 mm in any direction or can be rotated in its socket.	3	30 + 50

TOOTH MOBILITY REVISTED:

(According to Charles Anderegg and David Metzler, J.P. July 2001)

- Our commonly used parameter, degrees of millimeter movement, gives incomplete diagnostic and prognostic information. Current methods of grading or classifying mobility give no indication of the mobility is pathologic, physiologic or adaptive in nature.

- So adding the designator (A) for adaptive and (P) for pathologic to the current grading or classification scheme would add the critical element for determining necessary additional occlusal or periodontal treatment.

- Pathologic mobility, as defined, would include any degree of movement that may be reduced or eliminated once the pathologic factors is identified and corrected. Such etiologic factors would include inflammatory disease such as periodontitis, occlusal factors, parafunctional habits and iatrogenic factors.

- Adaptive mobility, as defined, would include the absence of an etiologic factor that might be improved upon to directly improve stability by decreasing or eliminating tooth mobility. While pathologic mobility would certainly require treatment, adaptive mobility might or might not. Progressing mobility, whether adaptive or pathologic, would of course require treatment to stabilize the situation.

- So we feel that adding the designator (A or P) to current descriptive terminology would, in a broad sense, address the etiology of existing mobility and complement current methods used to measure the degrees of mobility.

TREATMENT OF INCREASED TOOTH MOBILITY

A number of situations will be described here which may call for treatment aimed at reducing an increased tooth mobility.

Situation – I:

Increased mobility of a tooth with increased width of the periodontal ligament but normal height of the alveolar bone:

- If a tooth is fitted with an improper fitting a crown restoration, occlusal interferences develop and the surrounding periodontal tissues become the seat the inflammatory reactions, i.e. trauma from occlusion.
- If the restoration is so designed that the crown of the tooth in occlusion is subjected to undue forces directed in a buccal direction, because resorption phenomena develop in the buccal - marginal and lingual - apical pressure zones with a resulting increase of the width of the periodontal ligament in these zones.
- The tooth becomes hyper mobile or moves away from the "traumatizing" position. The resulting increased mobility of the tooth should be regarded as a physiologic adaptation of the periodontal tissues to the altered functional demands.

Fig. (a). contact relationship between a mandibular and a maxillary premolar in occlusion. Occlusion results in horizontally directed forces (arrows) which may produce an undue stress concentration within the "brown" areas of the periodontium of maxillary tooth. Resorption of alveolar bone and a widening of the periodontal ligament can be detected, leading to increased mobility. Following adjustment of the occlusal correction, the horizontal forces are reduced. This results in apposition "red areas" and normalization of the tooth mobility.

Situations – II:

Increased mobility of a tooth with increased width of the periodontal ligament and reduced height of the alveolar bone:

- If a tooth with a reduced periodontal tissue support is exposed to excessive horizontal forces, inflammatory reactions develop in the pressure zones of the periodontal ligament with accompany bone resorption. These alterations are similar to those which occur around a tooth with normal height of the supporting structures (as seen in situation 1). The alveolar bone is resorbed, the width of the PDL is increased in the pressured tension zones and tooth becomes hyper mobile.
- If the excessive forces are reduced or eliminated by occlusal adjustment bone apposition to the "pre trauma" level will occur, the periodontal ligament will regain its normal width and the tooth will become stabilized.

Conclusion

Situations I and II oculusal adjustment is an effective therapy against increased tooth mobility when such mobility is caused by an increased width of periodontal ligament.

Situation – III:

Increased mobility of a tooth with reduced height of the alveolar bone and normal width of the periodontal ligament:

- In this case tooth mobility can not be reduced or eliminated by occlusal adjustment. If such increased tooth mobility does not interfere with the patients chewing function or discomfort, no treatment is required.
- If the patient experiments the tooth mobility as disturbing, however the mobility can in this situations be reduced only by splinting, i.e. by joining the mobile tooth / teeth together with other teeth in jaw into a fixed splint for example "A – splint". A-splint, according to Glossary of Periodontal terms (1986) is an appliance designed to stabilize mobile teeth". A-splint can be fabricated in the form of joined composite filling, fixed bridges removable partial prosthesis etc.

Situations – IV:

Progressive (increasing) mobility of a tooth (teeth) as result of gradually increasing width of reduced periodontal ligament:

- Often in cases of advanced periodontal disease the tissue destruction may have reached a level where extraction of one or several teeth cannot be avoided. Teeth which in such a dentition are still available for periodontal treatment may, after therapy, exhibit such a high degree of mobility or even signs of progressively increasing mobility – that there is an obvious risk that the forces elicited during function may mechanically disrupt PDL components and cause extraction of the teeth. Only by means of a splint will it be possible to maintain such teeth.
- In such cases fixed splint has two objectives.
  1. To stabilize hyper mobile teeth and
  2. To replace missing teeth.
- Splinting is indicated when the periodontal support is so reduced that the mobility of the teeth is progressively increasing. i.e., when a tooth or a group of teeth during functions are exposed to extraction forces.

Situations – V:

Increased bridge mobility despite splinting:

- In patients with advanced periodontal disease it can often be observed that the destruction of the periodontium has progressed to varying levels around different teeth and tooth surfaces in the dentition. They may also be distributed in the jaw in such a way as to made it difficult, or impossible, to obtain a proper splinting effect even by means of a cross arch bridge The entire bridges splint may exhibit mobility in frontal and / or lateral directions. Neither progressive tooth mobility nor progressive bridge mobility can be accepted.
- In cases of extremely advance periodontal disease, a cross arch splint with an increased mobility may be regarded as an acceptable result of rehabilitation. It requires particular attending regarding the design of the occlusion.
- In cases of severity advanced periodontal disease it is often impossible to anticipate in the planning phase whether a bridge / splint after insertion will show signs of instability and increasing mobility. In such cases, a provisional splint should always be inserted.
- Any alteration of the mobility of the bridge / splint can be observed over a prolonged period of time and the occlusion continuously adjusted. Until, after 4-6 months, it is known whether stability can be achieved (i.e. no further increase of the mobility).
- Conclusion: An increased mobility of a cross arch bridge / splint can be accepted provided the mobility does not disturb chewing ability or comfort and then mobility of the splint is not progressively increasing. Splints can be temporary, permanent, extra coronal, Intra coronal, removable, fixed or fiber or resin bonded etc.
- However through are 2 schools at thought regarding mobility and splinting. Waerhaug and co-workers feel that increase in mobility do not necessarily represent a state of pathology and does not require splinting. But Muhlemann strongly advocates that increase in mobility is pathological which requires treatment. However, a study of Kegel et al (1979) revealed no difference in mobility reduction between splinted and unsplinted teeth over 17 weeks period.

CONCLUSIONS

Irrespective of age, periodontal disease could crawl-in at any point of time, severity of which would vary. Prompt diagnosis is therefore, a necessity so as to prevent further deterioration of the periodontium. It is therefore the duty of periodontist to preserve the perfect alignment of teeth and to prevent any ugliness that could merits sanctity.

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GENERAL ANESTHESIA

AN OPTION FOR PERIODONTAL SURGERY

A case report


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Key words:

Gingival hyperplasia, gingivectomy, general anesthesia

Abstract:

Gingival hyperplasia is a recognized side effect in individuals on anti- convulsant therapy, the management of which is complex. Most of these procedures are carried out in outpatient setting under local anesthesia. The use of general anesthesia as an option for periodontal procedures like gingivectomy is very rare. It is an option when surgery is likely to be prolonged or requires carrying out in multiple stages because of the generalized extensive gingival growth and more so when patient is apprehensive or less cooperative. Presenting a case where the use of general anesthesia was necessitated for gingivectomy since a full mouth surgery was required and the patient was apprehensive.

Introduction:

Periodontal surgeries are wide ranging and many of these procedures are time consuming. Most of these procedures are carried out in outpatient setting under local anesthesia. There are occasions when conscious sedation is used as a second choice. But, use of general anesthesia is very rare for periodontal surgery.

Presenting a case where use of general anesthesia (GA) was necessitated for gingivectomy, and a brief review of such periodontal situations where GA would be required and the requisite procedure(s) to be followed when it is utilised.

Case report:

A 17-year-old mentally challenged girl reported to the department of Periodontics, with complaints of swollen gums, difficulty in eating and an inability to carry out routine oral hygiene procedures.

On examination she was found to be moderately built and was deaf and dumb. History reveals similar gum swellings noticed when she was of 5 and 11 years, requiring excision to let the deciduous and permanent teeth, respectively, to erupt. The last surgical intervention was two and a half years back. Her parents gave history of convulsions in her since she was of 2 years age and she was on Diphenyl hydantoin since then. But, a history of change in the drug to Sodium valproate since 18 months was reported.

On oral examination, firm and fibrotic gingiva was seen covering 2/3 rd of the crown surfaces especially in the lower anterior region. There was evidence of minimal inflammation in the lower anterior region. Drifting of anterior teeth was also noticed due to fibrotic enlargement. (Fig.1). Orthopentamogram revealed impacted 3rd molars and erupting 2nd molars. But, there was no evidence of periodontal bone loss. (Fig .2).

Supragingival scaling was performed and gingivectomy was planned under general anesthesia since a full mouth surgery was required and the patient was apprehensive.

After due preoperative workup and consent procedures, under induction anesthesia, single long ribbon

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gauze was placed as a throat pack to prevent the aspiration of any fluid, blood or debris. Gingivectomy was carried out on the maxillary and mandibular anterior teeth both on the labial and lingual/ palatal surfaces. It was carried out using bard parker blades, no. 11 and 15, gingivectomy knives (Kirkland and Orbans) and curettes.

Periodontal dressing was placed after recovery from general anesthesia. The postoperative recovery was uneventful and the patient was discharged from hospital 2 days after the procedure.

The histopathologic examination of the excised tissue showed increased fibroblast proliferation and elongation of rete pegs into lamina propria, commensurate with the diagnosis of fibrous gingival hyperplasia. (Fig.3)

Discussion:

Repeated injections, pain and unpleasant experience result in fear which may become difficult to overcome and make the patient unwilling or be unco-operative in the dental chair.

The choice of general anesthesia (GA) for this case was made as the patient had already undergone gingival excision several times since her childhood. These attempts had left fear and apprehension in her mind. Use of GA also curtailed the number of patient visits for completion of surgery.

Our patient was on Valproic acid since 18 months and was on Diphenyl hydantoin earlier. Gingival overgrowth is one of the most common side effects associated with administration of phenytoin, the most frequently used anti – epileptic drug. Gingival enlargement occurs in about 50 % of the patients receiving Diphenyl hydantoin[14], although different authors have repoted incidences from 3% - 84.5% [3,8,12]. The onset of symptoms with Diphenyl hydantoin use varies from 2 weeks to 3 months, with maximum severity at 12 to 18 months. The mechanism of hyperplasia may be related to a stimulation of fibroblast proliferation or to a presence of genetically determined phenytoin-sensitive subpopulations of fibroblasts[7]. There are reports of regression of hyperplasia on stopping treatment with phenytoin.[5,13]

But, gingival hyperplasia is a rare side effect of Sodium valproate therapy. There are only 3 cases which have been reported so far[2,11,15]. However, in each of these cases the patients had never received Diphenyl hydantoin and the gingival hyperplasia regressed on stopping treatment with sodium valproate.

In the presented case, sodium valproate cannot be considered as the only cause for hyperplasia since the patient had been on Diphenyl hydantoin earlier and a similar history of gingival hyperplasia was noted even then.

There are anecdotal case reports of general anesthesia having been used in periodontal practice. In each of these, the reason for using general anesthesia for the gingivectomy procedures was fear and unco-operative nature of the patients.[1,4,9,10,16]

However, the use of GA is not without risks. The cases need to be meticulously selected, proper preoperative care given, and intensive close monitoring during and immediately after the procedure should be provided.

Patient selection is based on American Society of Anesthesiologists (ASA) Physical Status Classification where age of the patient, nature of surgical procedure, treatment site and required anesthetic procedure are the influencing variables.[6] (Table I).

Patients of ASA class I or II will be able to tolerate any surgical or anesthetic procedure. ASA class III patient requires the most critical decision making. A patient determined to an ASA IV classification should undergo only brief emergent or necessary palliative dental treatment.

Since use of general anesthesia in periodontal procedures is not very common, it is necessary to recall the pre-requisites for it.

A clear, concise, understandable preoperative patient communication is fundamental to ensure a smooth transition from the preoperative phase through recovery. The patient must have a complete understanding of the planned anesthesia protocols and what is expected of him/ her both preoperatively and postoperatively. A signed informed consent in an understandable language having the details of the procedure, the risks involved including that of anesthesia and the alternative treatment options must be obtained. In a situation where the patient is mentally challenged, the parent/ guardian needs to be explained the details of the procedure and the informed consent to be signed by him/her.

Preoperative workup involves the assessment of cardiovascular, renal, liver and coagulation system disorders. For an elective surgery obvious infections should be treated beforehand.

Preoperative fasting is advised for atleast 4 hours prior to the procedure. This is done to reduce the volume of gastric contents and thus reducing the risk of aspiration pneumonia.

Premedications include administering sedative (benzodiazepines) the night before surgery to reduce preoperative anxiety; vagolytics (atropine) to block unwanted autonomic effects and to prevent excessive secretions, given usually before shifting for surgery.

Postoperatively, clear fluids should be given after 3-4 hours of the procedure. Patient should be checked for bleeding or extraoral swelling. Analgesics are prescribed to alleviate pain and antibiotics to prevent infection.

It is emphasized that general anesthesia remains an option for periodontal procedures like gingivectomy when surgery is likely to be prolonged or requires carrying out in multiple stages because of the extensive gingival growth and more so when patient is apprehensive or less cooperative. It is necessary for the periodontist to be aware of the preoperative considerations and postoperative care to be provided for such procedures.

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Table I: The American Society of Anesthesiologists (ASA) Physical Status Classification

ASA	HEALTH STATUS
I	A normal healthy patient
II	A patient with mild systemic disease and no functional limitations. (well controlled sick patient)
III	A patient with moderate to severe systemic disease that results in definite functional limitation (poorly controlled sick patient)
IV	A patient with severe systemic disease that is a constant threat to life and functionally incapacitating.
V	A morbid patient who is not expected to survive 24 hours with or without surgery
VI	A brain dead patient whose organs are being harvested
E	If the procedure is an emergency, the physical status is followed by "E" (for example, 2E)

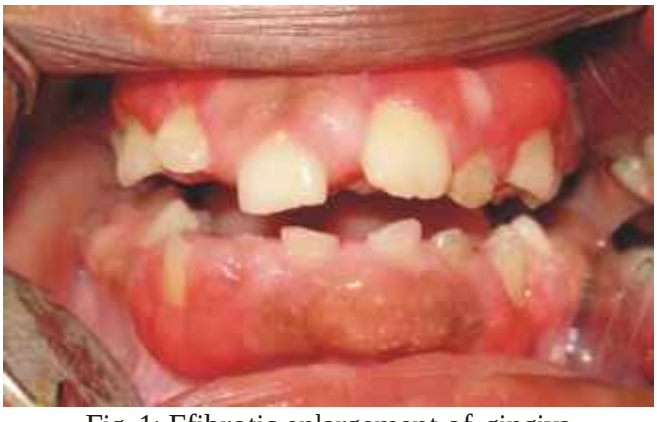

Fig. 1: Ffibrotic enlargement of gingiva

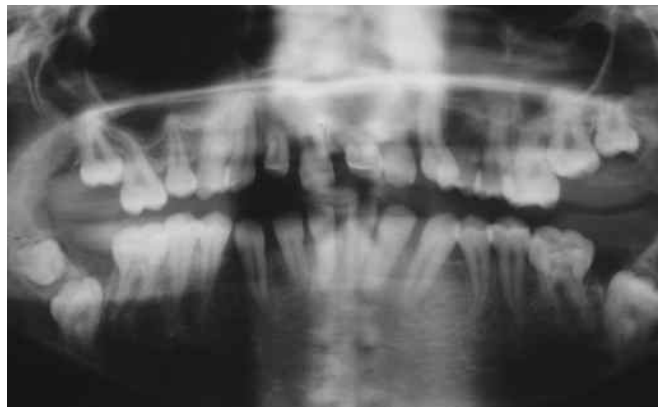

Fig.2: Orthopentamogram showing impacted 3rd molars and erupting 2nd molars

Fig.3: Microphotograph of excised gingival growth

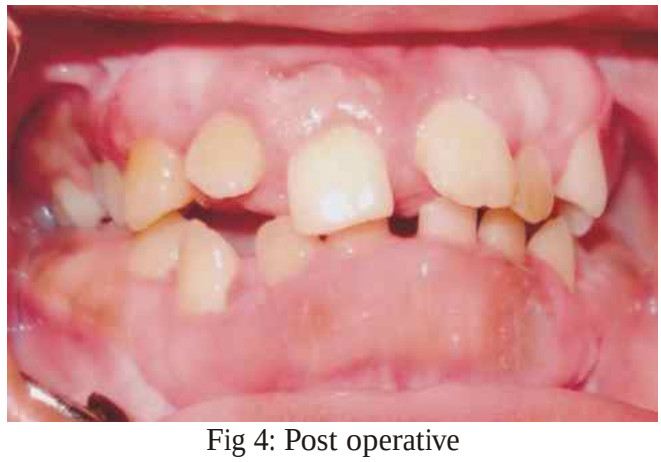

Fig 4: Post operative

Upcoming Events

- P G National Convention at Thirupathy, June 2010
- I S P National Conference 2010 at Bangalore, November 2010
- I S P National Conference 2011 at Ahemedabad

LOCALIZED PERIODONTITIS ASSOCIATED WITH PALATO-GINGIVAL GROOVES IN ADJACENT MAXILLARY INCISORS; A CASE REPORT

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Abstract

Aim: *Presentation of a case of localized periodontitis associated with palato-gingival grooves in adjacent maxillary incisors and its management*

Background: *Palato-gingival grooves are developmental morphological defects predominantly affecting maxillary incisors which can be a predisposing factor to localized severe periodontal destruction.*

Case Description: *A 23 year old patient presented with a chief complaint of proclination and shaking of upper front teeth. Examination revealed the presence of palato-gingival grooves in relation to maxillary left central and lateral incisors with associated periodontal pockets along the grooves. After the initial therapy, odontoplasty was performed to eliminate the grooves and bone grafting was done to correct the bony defect.*

Summary: *A rare case of palato-gingival grooves occurring on adjacent teeth with associated periodontal problem and its management has been presented. The management must include the elimination of the grooves along with routine periodontal treatment*

Clinical Significance: *Although the occurrence of palato-gingival grooves on adjacent teeth is rare, early recognition of these localized tooth-related factors that predispose to periodontitis is important for preventing more severe complications such as endodontic involvement and subsequent tooth loss.*

Keywords: *Palato-gingival groove, maxillary incisors, periodontitisAim*

To report a rare case of palato-gingival grooves occurring on adjacent maxillary central and lateral incisors associated with localized periodontitis and its management.

Background

Palato-gingival grooves (palato-radicular grooves, distolingual grooves, radicular lingual grooves) are developmental morphological defects affecting maxillary incisors. Developmentally, this is thought to represent an infolding of the enamel organ and Hertwig's epithelial root sheath[1]. It was first described in the literature by Oehlers in 1958 as a radicular invagination of an upper lateral incisor in a Chinese female[2]. Prichard was the first to state that lingual grooves on maxillary teeth are a predisposing factor to localized severe periodontal destruction[3]. The presence of this anomaly compromise the patient's ability to perform adequate plaque control in the area, thereby leading to plaque accumulation, which in turn predisposes to localized periodontal destruction[4]. The possibility of a long junctional epithelium instead of a proper connective tissue attachment can also render a certain degree of weakness to the area. The inflammatory process may extend apically along the groove resulting in endodontic involvement, which unfavorably affects the prognosis of the tooth[5].

The reported prevalence of palate-gingival groove varies from 2.8 to 8.5%[6,7].out of these; the maxillary lateral incisors are affected up to 90% of the times[7]. The central incisors are affected less frequently. The presence of palate-gingival grooves in both the central and lateral incisors is considered to be a rare condition as reported in the literature. The only study that reported the occurrence of palato-gingival grooves in both the central and lateral

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incisors was conducted by Hou and Tsai in a group of 101 Chinese adults[8]. They found a 4% prevalence of cases in which palato-gingival grooves were present on all the four maxillary incisors. However, the periodontal status of those teeth was not specifically mentioned. Here we report a case of palato-gingival grooves occurring on adjacent maxillary central and lateral incisors associated with localized periodontitis and its management.

Case Presentation

A 23 year old male patient presented to the out-patient department with the chief complaint of proclination and shaking of upper front teeth since one year. There was occasional bleeding from the affected teeth while brushing. This was his first dental visit. No significant medical or family history was elicited. Extra-oral examination of the patient showed the presence of incompetent lips. There was proclination and extrusion of the left maxillary central incisor with midline diastema. On intra-oral examination, the oral hygiene was found to be fair except on the palatal aspect of maxillary anteriors that showed plaque accumulation and gingival inflammation. Examination of the maxillary incisors revealed the presence of palato-gingival grooves in relation to the central and lateral incisors on the left side. The grooves were present on the mesiopalatal aspect of central incisor and the distopalatal aspect of the lateral incisor. On probing, 8 mm deep pocket was present on the central incisor and 5 mm deep pocket on the lateral incisor. The pockets were present only along the grooves and other surfaces showed minimal probing depth. The central incisor showed grade II mobility and the lateral incisor was grade I mobile. Radiographic examination revealed angular bone loss in relation to the mesial aspect of the left central incisor.

In the initial phase of the therapy thorough scaling and root planing were carried out. Occlusal interferences on the incisors were eliminated by coronoplasty. Evaluation of the patient after 6 weeks showed a reduction in the probing depth to 4 mm on both the teeth as well as a visible reduction in mobility. It was then decided to raise a flap to gain access for elimination of the grooves as well for correction of the bony defect.

Following local anesthesia, a full thickness mucoperiosteal flap was reflected on the palatal aspect of the affected teeth. Flap reflection and debridement allowed the complete visualization of the grooves. The groove on the central incisor extended up to 3 mm on the root surface terminating short of the marginal bone, whereas, the groove on the lateral incisor extended up to a distance of 5 mm stopping at the level of the marginal bone. There was a 3 mm deep intrabony defect on the mesiopalatal aspect of the left central incisor. As the grooves were shallow, odontoplasty was carried out using citric acid at pH 1 applied for 3 minutes. The bony defect was filled using a porous hydroxyapatite alloplastic graft material. The flap was replaced and sutured and a periodontal dressing was placed. Postoperatively, the patient was prescribed doxycycline (200 mg first day followed by 100 mg once daily for 4 days) and ibuprofen (400 mg thrice daily for 3 days). Chlorhexidine rinses were also advised. The dressing and sutures were removed one week postoperatively. The post surgical healing period was uneventful. The patient was seen regularly at 1 month, 3 months, and 6 months postoperatively. Review after 6 months showed reduction in probing depth to 3 mm in relation to both the teeth. Radiograph showed correction of the bony defect. The patient was referred to orthodontics department for correction of diastema and migration. Presently, the patient is undergoing fixed orthodontic treatment.

Discussion

The palato-gingival groove is a developmental anomaly predominantly affecting the maxillary incisors. It starts at the junction of the cingulum with one of the lateral marginal ridges and continues apically to the proximal surface of the root[4, 6]. The funnel-like shape of the groove provides a well-protected niche for the pathogenic bacteria, which subsequently results in periodontal breakdown. Periodontal disease can jeopardize the pulp depending on the depth and apical extension of the groove[5].

According to their extent, the palato-gingival grooves are generally categorized into mild, moderate, and complex grooves[9]. Mild grooves are gentle depressions of the coronal enamel that terminate at or immediately after crossing the cementoenamel junction. Moderate grooves continue to extend some distance apically along the root surface in the form of a shallow or fissured defect. Complex grooves are deeply invaginated defects that involve the entire length of the root or that separate an accessory root from the main root trunk. In the present case, both the grooves were of the moderate type.

In majority of the cases, the maxillary lateral incisors are affected by palato-gingival grooves. However, the maxillary central incisors are also affected, although less frequently. In a study of 625 extracted maxillary lateral incisors, Everett and Kramer described a prevalence of 1.9% of palato-gingival grooves[4]. Withers et al examined 531 military trainees for the prevalence of palato-gingival grooves in maxillary incisors and their relationship to localized periodontal disease. There was a total prevalence of 8.5% in the group, with 4.4% prevalence in lateral incisors and 0.28% in central incisors. Bilateral grooves on maxillary lateral incisors were found in 0.75% of cases. It was also found that palato-gingival grooves were associated with more plaque accumulation and poorer

periodontal health[7]. In another study of 3168 extracted maxillary central and lateral incisor teeth, Kogon found a total prevalence of 4.6% with 5.6% prevalence for lateral incisors and 3.4% in central incisors[6].

The occurrence of palato-gingival grooves in adjacent incisors is a rare phenomenon. Hou and Tsai examined 404 maxillary central and lateral incisors in 101 Chinese subjects for the presence of palato-gingival grooves. A total prevalence of 18.1% was found and in 4% cases, the grooves affected both the central and lateral incisors bilaterally[8]. There was however no specific mention of the periodontal condition of the teeth in those cases.

The palato-gingival grooves are most commonly located on the midpalatal surface rather than on the lateral surfaces. However, grooves on the mesial and distal surfaces are more frequently associated with periodontal pockets[8, 10]. Distal and mesial location of the groove can be considered a clinically adverse factor, since plaque more rapidly accumulates in these regions and are removed with greater difficulty[10]. In the present case, the grooves were located on the mesiopalatal and distopalatal surfaces of maxillary central and lateral incisors respectively.

Various treatment modalities have been advocated for the treatment of periodontal lesions associated with palato-gingival grooves. They include scaling and root planing, flap curettage, bone grafts, guided tissue regeneration, and use of enamel matrix proteins[11, 12, 13]. The grooves can be eliminated by odontoplasty in the case of shallow ones or by restoring with amalgam, composite, or glass ionomer cements[9, 14, 15]. In our case, an odontoplasty was carried out to flatten the grooves, as they were shallow. The associated bone defect was filled with a hydroxyapatite graft.

Summary

To summarize, we have reported a rare case of localized periodontitis associated palato-gingival grooves on adjacent teeth, which to the best of our knowledge has not been reported previously in the literature. The management must include the elimination of the grooves along with routine periodontal treatment, which, in the present case was achieved by odontoplasty

Clinical Significance

Although the occurrence of multiple palato-gingival grooves is uncommon, when present, they can be risk factors for localized periodontal destruction as reported in the present case. Early recognition of these localized tooth-related factors that predispose to periodontitis is important for preventing more severe complications such as endodontic involvement and subsequent tooth loss.

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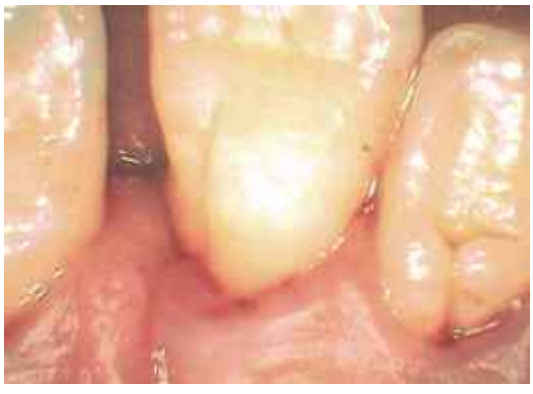

Fig 1: Palato-gingival grooves in relation to maxillary left central and lateral incisors

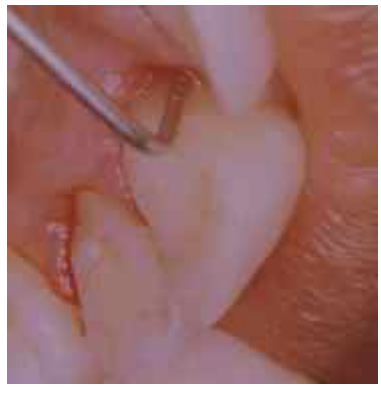

Fig 2: 8 mm deep pocket in relation to 21 along the groove

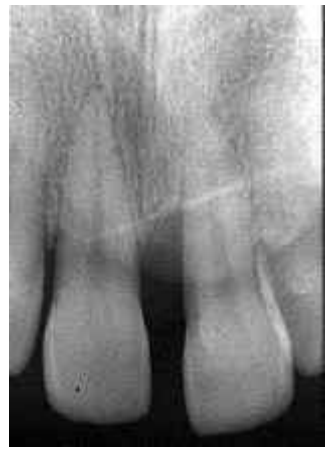

Fig 3: IOPA x-ray showing angular bone loss in relation to mesial aspect of 21

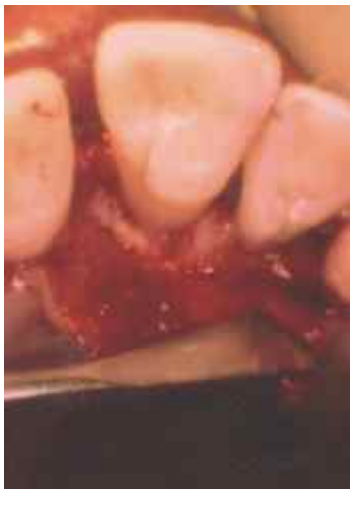

Fig 4: Flap reflection and curettage shows the extent of grooves along with the associated bony defect

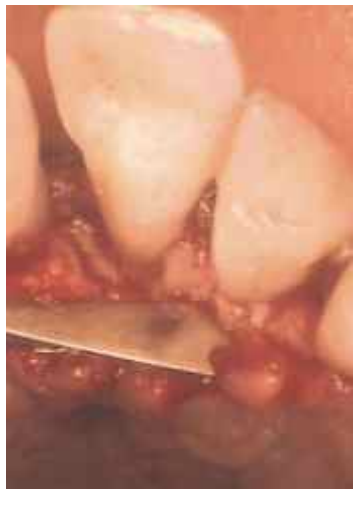

Fig 5: Odontoplasty performed to eliminate the grooves

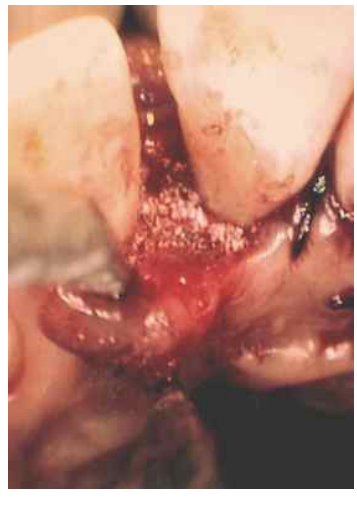

Fig 6: Bony defect filled with alloplastic graft material

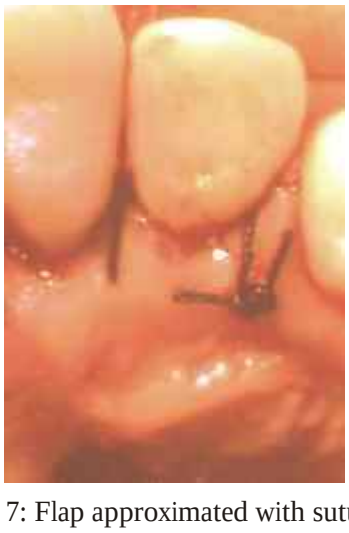

Fig 7: Flap approximated with sutures

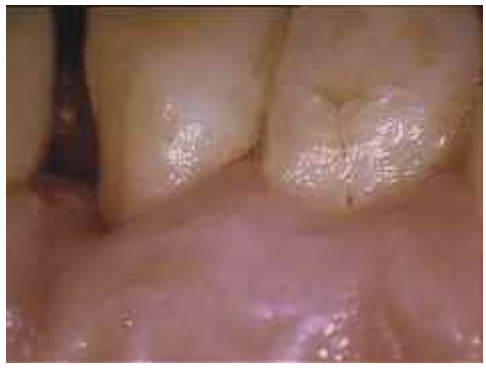

Fig 8: Clinical picture 6 months postoperatively

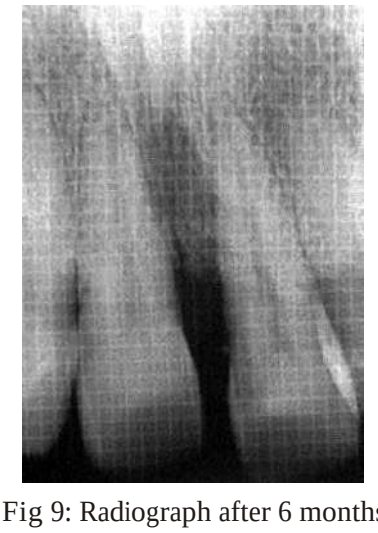

Fig 9: Radiograph after 6 months

COMPARATIVE EVALUATION OF GRAM NEGATIVE SPECIES IN THE SUBGINGIVAL MICROBIOTA OF SUBJECTS WITH HEALTHY AND CHRONIC PERIODONTITIS — A MICROBIOLOGICAL STUDY

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Abstract:

Purpose of the study: *Of the innumerable bacterial species which inhabit the human oral cavity less than 10 bacterial species are associated with the involvement of periodontal disease. Hence this study was designed to investigate the occurrence and concentration of 3 major periodontopathic bacteria, from the subgingival plaque samples of subjects with chronic periodontitis by microbiological analysis.*

Materials and methods: *In this study 40 subjects were divided into two groups of 20 each Group I being the control (healthy periodontal conditions) and Group II (subjects with chronic periodontitis). Subgingival plaque samples were collected from 2 teeth in each subject using sterile paper points and these samples were incubated anaerobically and the colonies formed were confirmed by Grams staining and by a series of biochemical tests.*

Results: *The results of the study showed significant difference in the occurrence of these periodon-topathogens in subjects with chronic periodontitis than in healthy subjects. There was a definite increase in the concentration of the three major periodondopathogens in the study group in comparison with the control group.*

Conclusion: *Monitoring the different bacterial species that are implicated as periodontopathogens in advanced periodontal lesions may greatly assist the assessment of treatment efficacy and risk of further periodontal breakdown.*

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INTRODUCTION

Chronic Periodontitis may be defined as a mixed infection affecting individual or multiple sites within the oral cavity and leading to the loss of the supporting periodontal tissues. This disease is chronic in nature and can persist in the absence of treatment[2]. The micro-organisms of dental plaque have been shown to be capable of initiating the mechanisms of destruction of the periodontal tissues, while their effective control has been shown to be most appropriate means of arresting the progression of periodontal disease. Although over 400 different bacterial species have been detected in the oral cavity, only a limited number have been implicated as periodontal pathogens. Among them Porphyromonas gingivalis, Aggregatiacter actinomycetemcomitans and Prevotella intermedia, species have been associated with chronic periodontitis.

The present study is designed to investigate the occurrence of these three periodontal pathogens in patients with chronic periodontitis and healthy subjects.

OBJECTIVE OF THE STUDY

1. To evaluate the concentration of the three major periodontopathogens in periodontally healthy subjects.
2. To evaluate the concentration of the three major periodontopathogens in subjects with chronic periodontitis.
3. To compare the concentration of these periodontopathogens in healthy and chronic periodontitis subjects.

MATERIALS AND METHODS:

SOURCE OF DATA:

Subjects reporting to the Department of Periodontics, A.B.Shetty Memorial Institute Of Dental Sciences.

METHOD OF COLLECTION OF DATA

Sample size of 40 subjects taken and divided into 2 groups

Group I:

Control group: Subjects with healthy periodontal conditions.

Group II:

Study group: Subjects with clinically diagnosed chronic periodontitis.

The subjects are to be registered for the number of teeth present and 2 teeth in each subject are selected for microbiological sampling.

SCREENING EXAMINATION INCLUDES:

Medical History

Dental history

CRITERIA FOR SELECTION:

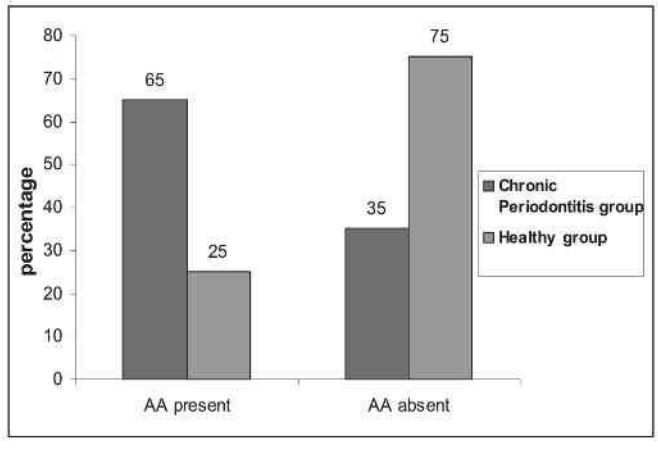
INCLUSION CRITERIA:

1. Each subject should have atleast 20 standing teeth none of which had untreated periapical lesion.
2. Subjects who have not received previous subgingival periodontal debridement or periodontal surgery in the preceding 6 months.

Table 1

Group * Aggregatibacter Actinomycetemcomitans

			AA		
			Present	absent	Total
Group	Chronic Periodontitis	Count	13	7	20
		% within Group	65.0%	35.0%	100.0%
		% within AA	72.2%	31.8%	50.0%
		% of Total	32.5%	17.5%	50.0%
	Healthy	Count	5	15	20
		% within Group	25.0%	75.0%	100.0%
		% within AA	27.8%	68.2%	50.0%
		% of Total	12.5%	37.5%	50.0%
Total		Count	18	22	40
		% within Group	45.0%	55.0%	100.0%
		% within AA	100.0%	100.0%	100.0%
		% of Total	45.0%	55.0%	100.0%



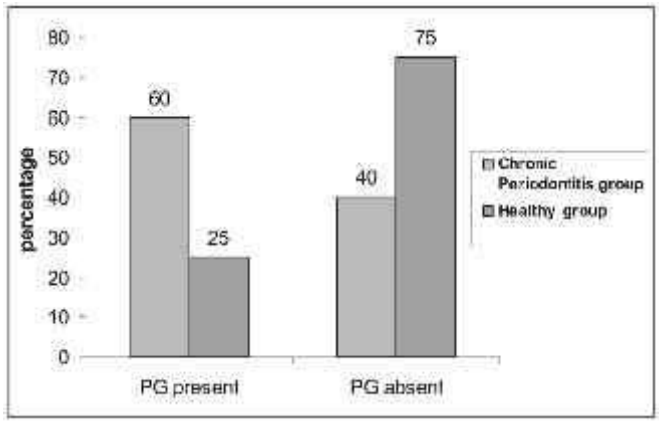
Fishers exact P-value= .025 sig

There is significance difference in presence of AA between chronic periodontitis & healthy (p=.025)

Table 2

Group * Porphyromonas gingivalis

			PG		Total
			Present	absent	
Group	Chronic Periodontitis	Count	12	8	20
		% within Group	60.0%	40.0%	100.0%
		% within PG	70.6%	34.8%	50.0%
		% of Total	30.0%	20.0%	50.0%
	Healthy	Count	5	15	20
		% within Group	25.0%	75.0%	100.0%
		% within PG	29.4%	65.2%	50.0%
		% of Total	12.5%	37.5%	50.0%
Total		Count	17	23	40
		% within Group	42.5%	57.5%	100.0%
		% within PG	100.0%	100.0%	100.0%
		% of Total	42.5%	57.5%	100.0%

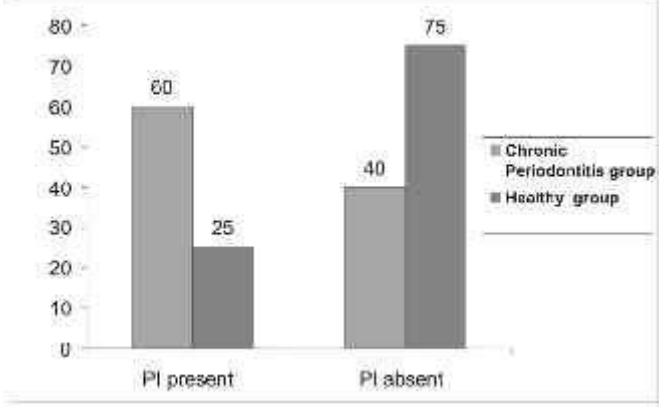


Fishers exact P-value= .049 sig

Table 3

Group * Prevotella intermedia

			PI		Total
			Present	absent	
Group	Chronic Periodontitis	Count	8	12	20
		% within Group	40.0%	60.0%	100.0%
		% within PI	88.9%	38.7%	50.0%
		% of Total	20.0%	30.0%	50.0%
	Healthy	Count	1	19	20
		% within Group	5.0%	95.0%	100.0%
		% within PI	11.1%	61.3%	50.0%
		% of Total	2.5%	47.5%	50.0%
Total		Count	9	31	40
		% within Group	22.5%	77.5%	100.0%
		% within PI	100.0%	100.0%	100.0%
		% of Total	22.5%	77.5%	100.0%



Fishers exact P-value= .020 sig

3. Subjects who had no antimicrobial therapy in previous 6 months.
4. Subjects with clinical attachment loss =3mm.
5. Subjects with age ranging from 25 to 50 years.

EXCLUSION CRITERIA:

1. Patients suffering from any chronic inflammatory or immunological conditions.
2. Subjects with any other systemic diseases.
3. Pregnant women are excluded from the study.

SUBGINGIVAL SAMPLING:

The subgingival plaque samples are to be collected using sterile paper points and are transferred on to the thioglycolate broth and should be processed within 36-48 hours. The samples are further inoculated into Columbia blood agar plates and are incubated anaerobically for 5-7 days at 35 ° C using the anaerobic gas pack system(Anaero gas pack 3.5;LE002A-5NC)in the Mcintosh& fildes pattern anaerobic culture jars. The samples are also inoculated into the TSBV agar plates and are incubated for 5-7 days at 35° C, and the colonies of anaerobic bacteria

formed are further confirmed by grams staining and by a series of biochemical tests. The colonies of Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans and Prevotella intermedia are specifically looked for using these techniques.

RESULTS:

The results of the study showed significant difference in the occurrence of these periodontopathogens in subjects with chronic periodontitis than in healthy subjects.

The incidence of the isolated bacteria among them are as shown in the Tables 1,2 and 3 Statistical analysis was done using Fishers exact test, the Fishers exact P-value for all the species were less than .05,which shows a significant difference in the concentration of these periodontopathogens.

Statistical analysis is done using Fishers exact test.

DISCUSSION

Periodontal diseases are inflammatory conditions resulting in the loss of the supporting structure of the teeth caused by micro-organisms. It has been known that both supragingival and subgingival plaque deposits are populated by heterogenous groups of micro-organisms5. A report from the World Workshop in Periodontics examining microbial factors in periodontal disease has suggested that there is sufficient evidence for two of these organisms, Porphyromonas gingivalis and Aggregatibacter actinomycetemcomitans to be considered as etiologic agents4 .

Results from this evaluation showed statistically significant difference between the case and control groups. On the basis of our findings in this study Aggregatibacter actinomycetemcomitans was found to be the most prevalent organism associated with chronic periodontitis, compared to Porhyromonas gingivalis and Prevotella intermedia which were less prevalent. This is in variance with the recent studies that suggest Porphyromonas gingivalis as the predominant organism associated with chronic periodontitis. The findings in this study are consistent with those given by Hamlet et al and Hossain et al²´4 , and thereby it is proposed that chronic periodontitis is associated with the increased concentration of periodontopathogens i.e. Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans and Prevotella intermedia. Hence further longitudinal studies are needed to confirm the association of these peridontopathic bacteria with chronic periodontitis.

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ANTIOXIDANTS-

A NOVEL CONCEPT IN DENTAL TREATMENT

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ABSTRACT

In todays society most of the individuals are affected by several autoimmune and infectious disease, this may be attributed to the change in lifestyle from the indigenous times. Many predisposing factors have led to the increase in certain harmful "free radicals" which cause detrimental impacts on health wherein they affect the internal milieu and reflect as systemic diseases. They also augment the aging process, which may be a major drawback in the present times where aesthetic image is a prime importance. However the possibilities to combat these effects are on the rise and the "return to nature" seems to provide the 'apt' solution. There is a resurgence of interest in this field and scientific work is being done to implement "antioxidant" therapy for health benefits which is inclusive of dental treatment. Several over the counter products are being developed and may be recommended as powerful adjunctive therapies. This article hopes to alleviate myths and apprehensions regarding the use and abuse of antioxidants

Antioxidants – A Novel Concept in Dental Treatment

"Health is the optimal wealth" and no efforts should be spared towards the uplifting of health related issues. Traditionally the focus was on endemic diseases like cholera and tuberculosis for which universal cures and vaccines have already been established but now it has shifted to autoimmune and infectious diseases which are more prevalent and on the rise and is the basic or prime concern in this modern age. A host of factors have been implicated but the limelight is now on certain disease promoting molecules known as 'free radicals'.[2,9,13,14,18,19,22,24,25,26,27,31,32]

Free radicals bring about many derogatory effects and have the ability to translate into disease by manifesting itself in several internal organs. They are not only restricted to the internal milieu but also express on the exterior thus contributing to one of the drastic effects due to the ravages of time on skin which is "wrinkles and skin damage". In the present era where cosmetics, aesthetics and longevity of life is a "numero uno" concern they may be a cause of "emergency".[10]

A free radical may be defined as "a species capable of independent existence that contains one or more unpaired electrons." The reactive oxygen species are those damaging molecules including oxygen radicals and other highly reactive forms of oxygen that can harm biomolecules and contribute to disease. These reactive oxygen species have a double edged mode of action where they are protective in just the right amount, if not they can trigger off a series of destructive actions and can harm biomolecules, the list is endless!![2,9,13,14,18,19,22,24,25,26,27,31,32]

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The reactive oxygen species in the body are elaborated during inflammation, which is a response to wall of noxious stimuli in the body. The neutrophils are the first line of defense and they require oxygen to function but when the oxygen consumption is at a rapid rate due to the progressive inflammatory condition the cell is under oxidative stress and undergoes the "respiratory burst" phenomenon which leads to the production of reactive oxygen species which can cause damage.[1, 11, 21, 33]

Superoxide, hydroperoxyl, hydroxyl, nitric oxide, peroxynitrite, hydrogen peroxide, lipid peroxide are some of the many examples of these species.

Internal sources of reactive oxygen species are from inflammatory conditions, ischaemic conditions, within the mitochondria; by phagocytes and during strenuous exercise.

External sources may include cigarette smoke, environmental pollutants, radiation, ultraviolet light, drugs, pesticides and ozone. They are extremely reactive, have a short life span, and may assist in damage to different targets by lipid peroxidation by the generation of new species by chain reaction.[8,9,14,15,22,25,31,32,34]

Inflammatory diseases and conditions are on the rise due to the perils of a modern lifestyle and the impact it has on nutrition, environment and psychological stress which promotes oxidative stress induced conditions like rheumatoid arthritis, atherosclerosis, macular degeneration, diabetes, cancer and even the gums are not spared!![26,31]

Periodontal disease!! which is so ubiquitously present!! is under the shadow of these reactive oxygen species who would have thought so?? A decade ago it would not have been easy for one to fathom the role of these species at such a broad level but it is time now to broaden our horizons and consider the etiological role of reactive oxygen species even in periodontal disease!!

At present the "armour of protection" which can control and harness the activity of these reactive oxygen species are certain powerful substances which may be the magic bullet on the horizon called the "ANTIOXIDANTS."[2,9,13,14,18,19,22,25,26,27,31,32]

So very rightly named they tend to keep in check these deleterious reactive oxygen species molecules. The fact that they are omnipresent in colourful fruits and herbs inclusive of medicinal plants has brought about a revival of interest in its medicinal property especially in India where medicinal herbs are part of the traditional therapies like "Ayurveda". Certain Chinese herbs like Ginseng & Ginkgo Biloba have shown potent antioxidant activity and the famous Chinese green tea has also shown surplus benefit of weight loss along with the antioxidant activity.[7,8]

There is widespread media coverage regarding the consumption of "red wine" as a healthy alterative to carbonated beverages like coke, Why is this? The elements of wine which can be traced back to certain Mediterranean fruits like raspberries, cranberries, blueberries and bilberries are powerhouses of antioxidants. They contain polyphenols which have immense benefit and have a protective effect. Apple polyphenols for example have shown to have a role against hair loss.[5,36]

Rediscovery of old concepts which were always present have been subject of emphasis in the recent times starting by shelving of most refined and fast foods with artificial elements and returning to the staple traditional organic diets with an ample portion of fruits like oranges and vegetables like yams, carrots, tomatoes and leafy greens which will replenish and boost the body supply of antioxidants like Vitamin C, Vitamin E and -carotene.

Future prospects of utilizing antioxidants for treatment of conditions like cancers and other inflammatory conditions including the retardation of the signs of aging is gaining stable ground , it will not be shocking to see its therapeutic implications in the field of dentistry too!![10]

ANTIOXIDANTS AND THEIR PROMISING USAGE IN DENTISTRY

A proper judicious balance is essential between the inherent pro-oxidant and antioxidant mechanisms to maintain homeostasis.[2,9,13,14]

The literature on the usage of antioxidants has shown promising implications in treating dental ailments.

Coenzyme Q_{10} topical gel which contains ubiquinone has shown to be effective in the treatment of gingival diseases like gingivitis and periodontitis. Topical application of Co-enzyme Q_{10} has shown significant improvement in periodontal health. They have been used as mouth rinse, topical gels and tablets.[3,4,12]

Natural indigenous products like tinctures and gum paints have been synthesized from natural herbs like Thyme, Fennel seeds, Myrrh, Ginseng and Ginkgo Biloba.

Propolis which is obtained from conifer buds in combination with bees wax is seen to have an analgesic action which controls dental pain and it has a plaque inhibitory action.

Pycnogenol which is a polyphenolic component also obtained from coniferous trees has been incorporated into chewing gums and this has shown superior plaque inhibitory action; also the oil derived form it is used to formulate mouthwashes that control bad breath.[29]

Apple polyphenols – are also being incorporated into chewing gums to control plaque and prevent periodontal

disease.[30,35]

Certain mouth washes and tooth powders are coming into the market with dual advantage; probiotics are being combined along with antioxidants since they inhibit the alkaline environment and prevent the growth of resistant bacteria.

Green tea is another natural product which has anti-cariogenic action, it contains natural fluoride and polyphenols that inhibit cares, it has direct bactericidal effect on streptococcus mutans.[6]

Green tea prevents the adherence of bacteria to the tooth surface; prevents plaque matrix synthesis, it inhibits bacterial enzymes also.

New oral sprays which have a dynamic combination of antioxidants i.e. Propolis, Co-enzyme Q_{10}, Green tea catechins and Aloe vera have been released in the International Drug Market with an antibacterial and anti-inflammatory role. These sprays effectively control gingivitis and periodontitis.

Pharmaceutical research took a major leap when it was realized that these natural products had got such untapped potential. The sudden resurgence in the literature and increased scope of monetary benefit has triggered the race and most pharmacologists, microbiologists and biochemists began to unravel the chemistry of natural processes in humans, animals, plants and microorganisms. The identification of many key antioxidant molecules opened a new avenue to futuristic concepts in therapy and numerous applications in industrial products.

Many studies on animals are showing promising results for instance the superoxide dismutase and catalase enzyme capsules along with normal oral prophylactic procedures showed superior results and improved condition of the inflamed tissues, the application of topical mercaptoalkyl guanidines also showed a significant improvement of periodontal health.[16]

In summary "antioxidants" may be recommended as a strong adjunctive therapy and this will in addition have a greater impact, because people tend to abide increasingly to preventive protocol programmes rather than curative regimens. However people are still skeptical about the usage of these products regardless of the numerous benefits. Therefore more awareness to the general public and clinicians in particular may go a long way in alleviating some of the myths and apprehensions on the use and abuse of antioxidants. However this arena is yet to be fully explored since natures bounty cannot be underestimated there are many secrets yet to be revealed. Research is still underway regarding this aspect and emphasis is upon the longitudinal studies and their results.

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SODIUM CALCIUM PHOSPHOSILICATE (NOVAMIN®)

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ABSTRACT

Tooth hypersensitivity is an unpleasant sensory experience. This review points towards the management of tooth hypersensitivity with the help of bioactive glasses.

INTRODUCTION

Tooth hypersensitivity is a very common problem encountered in our daily practice. It is the sharp pain arising from exposed dentin, as a result of various stimuli such as heat, cold, chemical or osmotic and that cannot be ascribed to any other pathology. Unfortunately few epidemiological studies have highlighted its prevalence in our community. In the US, studies have shown 8 to 30% of the adult population suffers from tooth hypersensitivity(Gillam et al1999 J oral rehab). Patients with periodontal disease have a much higher incidence of tooth hypersensitivity.

PHYSIOLOGY

Though the etiology of dentin hypersensitivity is multifactorial, it is seen that open dentinal tubules are a major cause in sensitivity. The currently accepted theory for tooth hypersensitivity is the Hydrodynamic theory proposed by Brannstrom. (Brannstrom et al 1986 J Endo) Open dentinal tubules allow fluid flow through the tubules, that produces pressure changes which excite the nerve endings in the dental pulp. There are studies showing that in patients with dentin hypersensitivity there is a greater number of tubules per area and the diameter of the tubules is greater than in patients with no sensitivity(Absi EG et al 1987 J Clin Per) Usually the dentinal tubules of the root are covered by a layer of cementum or by a smear layer of 2 to 5µ thick. When the smear layer is present the fluid flow that occurs in dentin is only a few percent of that possible in its absence. Smear layer may be removed physically or by acids leading to the opening of dentinal tubules and thereby sensitivity. In periodontitis bacteria and their products also play a role leading to a higher percent of these patients reporting sensitivity. Addy et al has reported rates of dentin hypersensitivity as high as 93% in periodontitis patients.

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MANAGEMENT

There have been 2 basic approaches to the treatment and prevention of dentinal hypersensitivity. The first approach is to treat the tooth with a chemical agent that penetrates into the dentinal tubule and depolarizes the nerve synapse, which reduces sensitivity by preventing the conduction of pain impulses. eg: Potassium nitrate.

The second approach is to treat the tooth with a chemical or physical agent that creates a deposition which mechanically occludes dentinal tubules, thereby preventing pulpal fluid flow. eg: Strontium chloride, Potassium oxalate.

Although both approaches can significantly reduce or eliminate hypersensitivity, the duration of relief is highly variable. Hypersensitivity usually reappears due to tooth brush abrasion, presence of acid challenges in mouth etc.

The future of dentin hypersensitivity management lies in a material that will chemically react with the surface of dentin, intimately adhering to the tooth structure and significantly reducing the possibility of reopening the dentinal tubules either due to acidic food or overzealous brushing.

BIOACTIVE GLASS- A RETROSPECTIVE

Bio active glass was developed as a bone replacement material. Numerous studies have shown that this material will induce bone formation in a physiologic system. The material has been widely tested in bone and soft tissue. Toxicology evaluation of the glasses has shown no toxic effects in bone or soft tissue in numerous invitro and invivo models.

It has also been reported that it has antibacterial properties, most likely due to the change in pH induced by the dissolution of the ions from the surface of the glass and lack of bacterial adherence to the glass surface.

In osseous defects bonding of glass to bone begins with the exposure of the glass to aqueous solution. Na+ in the glass exchanges with H+ from the body fluids causing the pH to become more alkaline. Ca and P migrate from the glass forming a Ca- P rich surface which will form an apatite layer. Underlying this Ca- P rich layer on the glass is an area which becomes increasingly silica rich due to the loss of Na, Ca and P ions.

Bioactive glass has not previously been described as a material for use in the treatment of dentin hypersensitivity. Also the antibacterial properties of this material will be of benefit in periodontal treatment where bacterial colonization will be reduced.

SODIUM CALCIUM- PHOSPHOSILICATE (NOVAMIN®)

Is a patented product which is chemically bioactive glass. Novamin® containing dentrifices which hit the market recently can occlude the dentinal tubules thereby eliminating sensitivity. The Ca- P layer crystallizes into hydroxyapatite which is chemically and structurally equivalent to biological apatite. The combination of residual bioactive glass particles and the newly formed hydroxyapatite layer results in the occlusion of dentinal tubules thereby relieving hypersensitivity.

The antimicrobial action against periodontal pathogens could be of significant benefit to the patient in periodontal maintenance therapy.

In an experimental gingivitis study it was found that the material also have some local anti-inflammatory action as determined by a reduction in gingival inflammation (Eberhard et al Jr Biomaterials 2004).

Studies have shown Novamin® did not induce a TNF , IL-, IL-10 or white cell recruitment when endotoxin was injected into the Novamin® pre exposed peritoneal cavity of mice. The researchers concluded that there is a transient suppression of the inflammatory response to endotoxin possibly through early induction of IL-6.

CONCLUSION

These properties of Novamin® viz desensitizing agent, antibacterial and anti-inflammatory makes the material an attractive product for use in periodontal therapy.

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MANAGEMENT OF PALATORADICULAR GROOVE

2 CASE REPORTS


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Abstract: The palatal groove is a developmental anomaly that predisposes the tooth involved to a severe periodontal defect. When further complicated by pulp necrosis, these grooves often present a diagnostic and treatment planning challenge that requires an interdisciplinary treatment approach. This case report describes the successful collaborative management of maxillary lateral incisor with palatal groove using a combination of nonsurgical endodontic therapy, odontoplasty, and periodontal regenerative techniques.

Periodontitis is an inflammatory disease of the supporting tissues of the teeth resulting in progressive destruction of periodontal ligament and alveolar bone resulting in pocket formation, recession or both. Dental plaque has been established as the prime etiological factor in the development of periodontal diseases. Numerous local anatomical factors cause plaque accumulation and retention; such factors include marginal ridge discrepancies, food impaction, cervical enamel projections, open contacts, and palatoradicular grooves.

Palatoradicular grooves (PRGs) are developmental anomalies of the maxillary incisor teeth which usually begin in the central fossa, cross the cingulum, and extend varying distances apically[1], and possibly reaching the root apex [2, 3, 4]. They can be present in the midpalatal, mesial, or distal region of the tooth [5, 6, 7]. PRGs extending apical onto the root[8, 9,10,11,12] have been regarded as funnels for the retention of microbial dental plaque.

Withers (1981) reported presence of palatogingival grooves on 2.3% of maxillary incisor[1]

The predominance of the PRG in the lateral incisor[1, 6] can be due to the undesirable position of the tooth during the growth of the maxilla. While it is still a tooth germ, it becomes trapped between the central incisor, canine and first premolar, that are in a more advanced phase of dental development. Mineralization of the crown of the maxillary lateral incisor starts later, compared with the others making this germ, under these conditions, highly susceptible to folding[13,14].

It originates with alteration in the growth of Inner Enamel Epithelium (IEE) and Hertwig's Epithelial Root Sheath (HERS) and involves commonly the maxillary lateral incisors. Although the pathogenesis of palatoradicular groove is similar to dens invaginatus, it differs from the latter in that it results in a groove, rather than in an opening. Palatoradicular groove formation presumably represents an aborted attempt to form an additional root[10].

The presence of PRG does not always indicate the development of pathology. In most cases the epithelial attachment remains intact across the groove and the periodontium remains healthy[12]. Once the attachment is breached, a periodontal pocket forms along the length of the groove. The prognosis of teeth affected by this anomaly depends upon the depth and extension of the groove. The attachment could be breached due to endodontic involvement or periodontal involvement[6]. The

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former results in a primary endodontic - secondary periodontal lesion whereas the latter results in a primary periodontal - secondary endodontic lesion.

Although no formal classification has been proposed for palatoradicular groove, Goon suggested that there are two types of PRGs which he classified as simple and complex[15]. The simple PRGs do not communicate with the pulp and represent a partial unfolding of HERS. Complex PRGs are characterized by direct communication with pulp and extend the length of the root. In rare cases, the groove may lead to a minor accessory root which may contain a root canal.

The funnel-like shape of the PRG promotes the accumulation of plaque and calculus, which are difficult to remove, at times making proper cleaning by the patient or even the dentist nearly impossible[9,10]. Hence management of PRG poses a challenge to the dentist.

PRG can be treated successfully using an interdisciplinary management comprising endodontic and periodontal procedures and in some cases odontoplasty and restorative procedures.

CASE REPORT 1:

A 52 year old male reported to Dept. of Periodontics, Govt. Dental College, Trivandrum complaining of perception of salty taste in his mouth for the past 6 months. He had consulted many dentists for the same and was prescribed various mouthwashes but he had no relief from his symptoms. He reported to the department with the same complaint. He had no relevant medical history and showed good oral hygiene.

On examination, there was a change in color and appearance of 12. The tooth showed the presence of a palatogingival groove originating from the cingulum running across the CEJ and extending on the root (Fig 1). A deep periodontal pocket measuring 11mm was present distal of 12. Also, the tooth exhibited grade II mobility. There was suppuration from the periodontal pocket. On vitality testing, 12 was found to be non vital. 22 was also seen to have a palatogingival groove but it did not show any periodontal disease.

On radiographic examination, a bone defect was seen distal of 12. Remaining bone height upto half of the root length was seen with periapical radiolucency with 12 suggestive of an endo-perio lesion.

A treatment strategy was planned that comprised supra and sub-gingival scaling with root debridement and adjunctive antibiotics and analgesics, followed by root canal treatment and periodontal surgery for pocket elimination and groove repair.

Fig 7 shows the IOPA x-ray of the region after completion of RCT. The mobility of 12 reduced after root canal treatment (RCT) to grade I and there was no need of splinting. Under local anesthesia, a full thickness mucoperiosteal flap was reflected after giving a crevicular incision from mesial aspect of 13 to mesial aspect of 11. Upon reflection, the bony defect along the palatal root became evident. The apical extent of the palatoradicular groove was detected up to the middle 3rd of the root (Fig 2).

All the granulation tissue was removed using a Gracey curette no. 1 / 2 and the root was conditioned using tetracycline. The palatoradicular groove was deep in the coronal aspect and shallow along the root surface. Odontoplasty was done along the root using a straight fissure diamond bur and air rotor handpiece (Fig 3). The deep groove along the crown of 12 was filled with Glass ionomer cement to obliterate the groove (Fig 4).

Demineralised bone matrix (Osseograft ™) mixed with the patient's blood was placed in the bone defect (Fig. 5) and the flaps were approximated and sutured using 3-0 chromic catgut sutures (Fig. 6). The wound site was covered with a zinc oxide eugenol dressing. One week following surgery, the dressings and sutures were removed. Healing after surgery was uneventful.

After 3 months, the pocket probing depth had reduced from 11 to 7 mm. There was no exudate or bleeding on probing. The patient continues to be under review.

CASE REPORT 2:

A 35-year-old woman was referred to the Department of Periodontics, Government Dental College, Trivandrum with a complaint of pain and swelling in the region of the upper right lateral incisor since one week. The patient revealed a history of recurrent episodes of pain and swelling on the palate accompanied by pus discharge over the past one year.

Clinical examination revealed a swelling on the anterior portion of the palate close to the midline (Fig.1). A sinus opening was also detected on the labial aspect of 12 (Fig.2.). A groove extending apically from the cingulum on the palatal aspect of the tooth was observed. The tooth was found to be tender to percussion and failed to respond to vitality testing. Periodontal probing of the palatal gingiva of this tooth showed a 10-mm pocket with exudate and bleeding. At all other points around the tooth the sulcus depth was found to be shallow. The tooth had grade I mobility. Radiographic examination indicated a tear drop shaped radiolucent area centred around the apex of the root of 12 (Fig.3).

Our treatment plan consisted of root canal therapy followed by surgical curettage of the periodontal defect. Following the endodontic treatment a resolution of the palatal swelling was observed although the periodontal pocket persisted (Fig.4.).

On reflection of the labial flap and degranulation, a large fenestration type defect was observed (Fig.5.) Palatally a deep isolated intrabony defect extending along the length of the radicular groove was detected (Fig.6). Granulation tissue was removed and careful scaling and root planing of the exposed root surface was carried out. The coronal portion of the groove was sealed with glass ionomer cementum (Fig.7). This technique was advantageous as it effectively eliminated the groove as a pathway for bacterial ingress, without loss of tooth substance and without exposure of cut dentin.

A dense particulate hydroxyapatite alloplastic graft material was placed in the labial and palatal osseous defects (Fig.8, Fig.9). The flap was replaced to its original position and fixed by silk sutures which were removed 8 days later (Fig.10).

Upon recall 2 weeks later the patient was free of pain. The tooth was asymptomatic and unresponsive to percussion. The palatal swelling and labial sinus tract had healed completely by this time. (Fig.11,12)

Discussion:

Palatoradicular groove is a rare developmental anomaly with a prevalence of 2.8–8.5%[9]. Although rare, the clinical implications are important, since such grooves may lead to combined endo-perio lesions. These grooves usually begin in the central fossa, cross the cingulum and extend to various distances, depths and directions along the root. These grooves may present radiographically as a radiolucent parapulpal line. However, this was not seen in the present cases.

When the groove is entirely located in the crown of the tooth, terminating at the CEJ and when it is shallow, the prognosis is termed good since there will be absence of deep bony defect and pulpal damage. Simple treatment may be considered, including curettage of granulation tissue, improvement of oral hygiene and sometimes elimination of the groove by means of saucerization[16]. Saucerization involves elimination of the defect with rotary cutting and polishing instruments. It has been a helpful method in eliminating shallower grooves as was done in case 1.

However, when grooves are deeper, materials such as composite and amalgam[17], Glass ionomer cement[18] can be used to fill the palatoradicular groove. In these cases, the groove was sealed with glass–ionomer cement. Glass–ionomer cement (Fuji I) was used, since it has an antibacterial effect, chemical adhesion to the tooth structure and good sealing ability[19]. Clinical and histological studies have reported that there is an epithelial and connective tissue adherence to glass–ionomer cement during the healing process[20]. The clinical observation found in these cases correlated with this study.

In the present cases, since the groove was extended onto the root surface with substantial periodontal destruction, a flap procedure including curettage of granulation tissue and root planing was undertaken. Since there was an advanced circumferential bony defect, a demineralised bone matrix was placed to promote bone regeneration. Demineralised bone matrix has osteoinductive potential and clinical studies have shown considerable bone fill and gain in attachment level following use of demineralised bone matrix[21].

The key factors which may have contributed to the success of this case are:

- Effective root canal treatment
- Periodontal attachment facilitated by the elimination of the groove
- Placement of bone graft into the osseous defect
- Periodontal maintenance.

Conclusion

Palatoradicular grooves are rare developmental anomalies, but can initiate pulpal and periodontal disease that can be difficult to diagnose and manage. The management would vary according to the type, extent and complexity of the palatoradicular grooves. The key to long term success is accurate diagnosis and elimination of the contributory factors. A clinician's awareness of the existence of such anomalies can go a long way in avoiding improper treatment of such cases.

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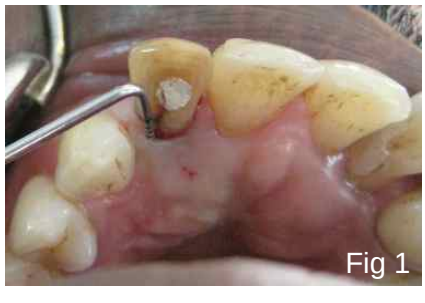
CASE 1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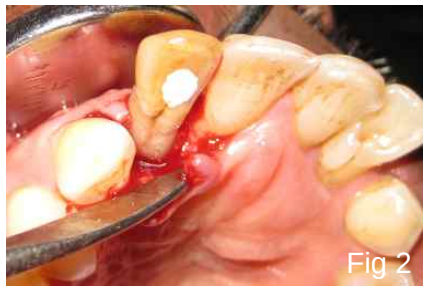
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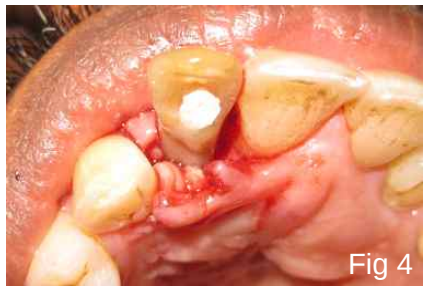
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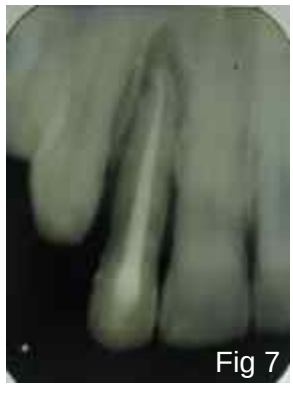
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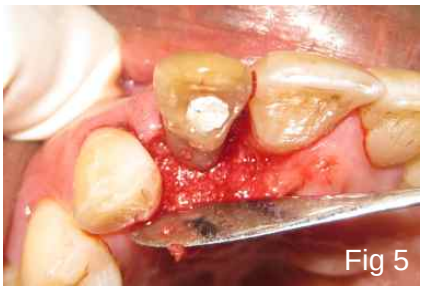
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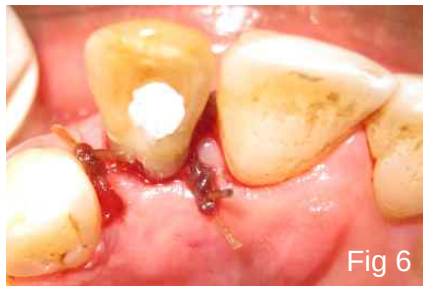
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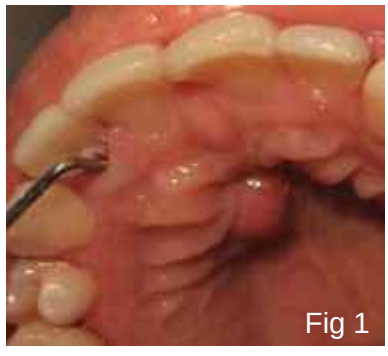
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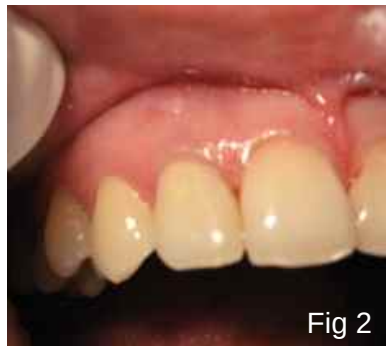
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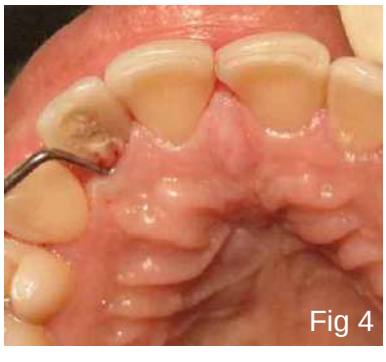
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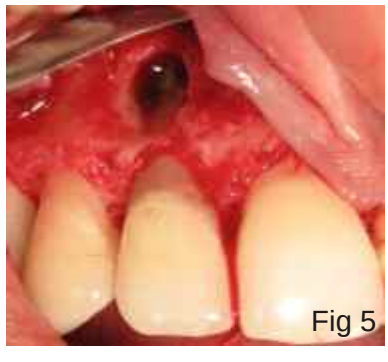
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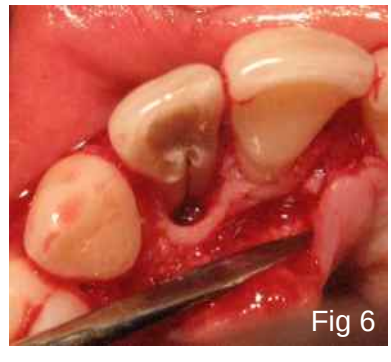
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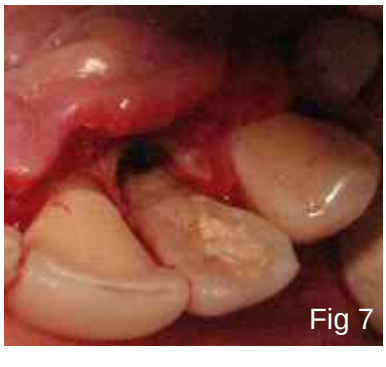
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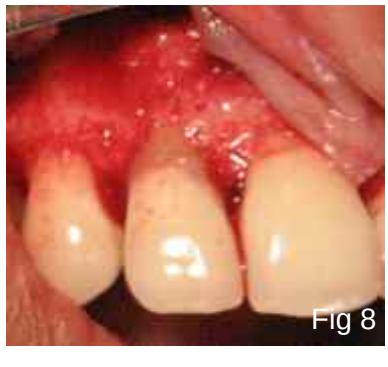
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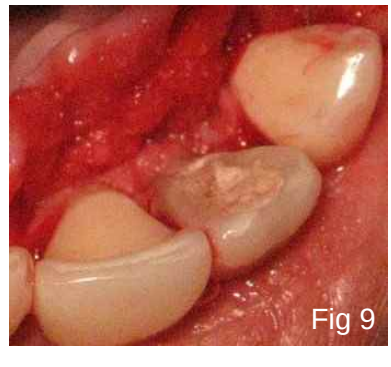
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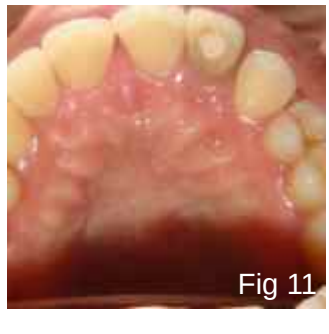
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Fig 12

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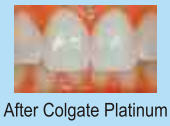

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