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Contents

President's message

Society of Periodontists and Implantologists of Kerala is a speciality organization whose ultimate objective is improving the periodontal health of the public. SPIK organizes regular scientific activities pertaining to the current concepts in Periodontology and Implantology. It has organized and conducted several programs, webinars and conferences. This year we had organized a webinar on the updates in Periodontal Plastic Surgery by Dr Prakash P. S. G from Chennai. The Periodontology Scholarship Examination was held on October 22, 2022 at Amrita School of Dentistry by Dr Rajesh Vyloppillil and team. SPIK essay contest was also conducted for life members and post graduate students.

We have organized and conducted mid-term conference at PMS College of Dental Science and Research, Thiruvananthapuram on 10th & 11th of December 2022. The invited speakers were Dr Kaarthikeyan, Dr Biju Thomas, Dr K. Harikumar Menon and Dr Baiju R.M, who shared their in-depth knowledge in the subject which was an inspiration to the young aspirants and faculty members. The conference was an ideal platform for the post graduate students for showcasing their academic, clinical and research talents by presenting scientific papers. A quiz program was also organized during the conference by Dr Manikandan G R for the post graduate students of all dental colleges in Kerala.

The upcoming activities of this SPIK year include a post graduate orientation program-a webinar and the SPIK annual conference. A webinar will be organized on January 29, 2023 by Dr Ashish Nichani and Dr Arun Sadasivan. We are planning to conduct the SPIK annual conference on 29th & 30th of April 2023 at Kannur Dental College, Kannur.

Our society is growing in strength and stature,and we require more members to our SPIK family to further strengthen it. Congratulations to the winners and participants of various programs conducted this year and I urge everyone of you to regularly attend activities of SPIK. I would like to thank all the past presidents, secretaries and also our present dynamic secretary Dr Mohammed Feroz T P, who have successfully steered this organization all these years until now.

Dr. Presanthila Janam
President, SPIK

Secretary's Message

Warm greetings to all SPIK members!

At the outset, let me wish all members a *Happy and Prosperous 2023*.

SPIK is an excellent scientific learning platform for all postgraduate students. I would like to congratulate the organizing committee of SPIK Midterm Conference held at PMS College of Dental Science and Research, Thiruvananthapuram in December 2022. It was a well-organized program with scientific deliberations on current concepts in the field of Periodontology.

Let me congratulate the Editor for publishing the second issue of our journal of this SPIK year. All the contributions were subjected to a blind scrutiny by peer reviewers before acceptance for publication.

We would further like to increase the membership strength as well as implement new ideas and innovations to improve the scientific activity of SPIK in the coming days. Looking forward for a fabulous Annual conference at Kannur in the month of April and your continued support.

Dr Mohammed Feroz T P
Secretary, SPIK

INFORMATION TO AUTHORS

About the Journal

JSPIK accepts articles from dentists, dental specialists (any speciality) and students. The articles submitted must have relevance to the speciality of Periodontics. Authors are encouraged to submit research papers, interdisciplinary case reports, interesting case discussions, letters to editor review articles or short communications.

Manuscripts

Articles should be type written on one side of A4 size (21x28cm) white paper in double spacing with a sufficient margin. Use a clear and concise reporting style. SPIK reserves the right to edit, manuscript, to accommodate space and style requirements. A soft copy of the article also has to be send to the editor's email: **editorspik@gmail.com**

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- **Tables:** Tables should be self explanatory, numbered in roman numbers, according to the order in the text and type on separate sheets of paper.
- **Illustrations:** Illustrations should be clearly numbered and legends should be typed on a separate sheet of paper, while each figure should be referred to the text(figure 1,2 etc.)
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Management of Plasma Cell Gingivitis: A Case Report

Gayathri B S[1], S Santhosh Kumar[2], Roshni Ramesh[3], K C Ajith Kumar[4], Suchitra A[5], Lekshmi G[6]

ABSTRACT

Plasma cell gingivitis is a benign inflammatory condition of the gingiva, which usually arises as a hypersensitive reaction to chewing gums, dentifrices or other dietary components. Histologically, the connective tissue is infiltrated by abundant plasma cells. This case report depicts the clinical presentation of plasma cell gingivitis due to the use of an anti-sensitivity tooth paste. The presented case is of a 45-year-old male patient with swelling in relation to lower anterior region which bleeds on pressure and was unresponsive to conventional periodontal therapy. Periodontal surgical management was done after discontinuation of anti-sensitivity tooth paste. The patient was under regular follow up and no recurrence was reported till date. Cases have been reported earlier in literature, related to plasma cell gingivitis caused by dentifrices. They can be due to the components inadvertently added for flavor and taste. Dentists should be aware of the possible side effects of these products for arriving at a proper diagnosis.

Keywords: Plasma Cell Gingivitis, Gingiva, Plasma cells, Dentifrice

Introduction

Plasma cell gingivitis is an infrequently observed benign inflammatory condition of the gingiva, which is also known as atypical gingivitis and plasma cell gingivostomatitis.[1] This condition has also been reported on lips, tongue, conjunctiva and nasal aperture. Plasma cell gingivitis is so named because of the prominent plasma cell infiltrate in the affected tissues.[2,3] It was first reported in 1970's as an allergic reaction to cinnamon used in chewing gum.

Allergic reaction seems to be a plausible explanation for plasma cell gingivitis. Various allergens documented are certain components of tooth paste,[4,5,6] cinnamon,[7] chewing gums,[8,9] mint etc. Clinically, it is characterized by sharply demarcated erythematous and edematous gingiva often extending to mucogingival junction. Moreover, the gingiva appears red and friable and bleeds easily on provocation.

The aim of this report is to present a case of plasma cell gingivitis on the mandibular labial anterior region in a 45-year-old male patient caused by the use of dentifrice, that was accurately diagnosed, successfully treated and maintained surgically. This case report emphasizes the adverse effects that may be caused by inadvertent use of herbal or chemical agents in dentifrices and the importance of detection of possible antigenic source for the successful management of plasma cell gingivitis.

Clinical Presentation

A 45-year-old male patient presented to the Department of Periodontics with a chief complaint of swollen gums of lower front teeth noticed since past two weeks. He reported a gradual increase in size of the swelling with tenderness and severe bleeding

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while brushing. After consultation from a nearby hospital he was referred to our department for further management. He gave a history of oral prophylaxis for painful and bleeding gums and changing of tooth paste for teeth sensitivity as advised by a dentist three weeks back. Medical and family history was noncontributory.

On intra oral examination, diffuse proliferative gingival enlargement of Grade III extending from distal aspect of the 43 to distal aspect of 33 noticed on the labial aspect. The surface was erythematous with fibroedematous consistency and borders shows friable appearance (Figure 1A and B). Enlargement was associated with an inflammatory component probably due to his inability to maintain an adequate personal oral hygiene. Enlargement of interdental papilla in relation to maxillary incisors was also present. On palpation the enlargement was non tender with mobility of mandibular anteriors. Periodontal probing revealed deep pockets in relation to involved teeth.

Provisional diagnosis was inflammatory gingival hyperplasia and the differential diagnosis included plasma cell gingivitis, Wegener's granulomatosis, sarcoidosis, leukemic gingival enlargement, scorbutic gingivitis, human immunodeficiency virus (HIV) associated gingivitis and tuberculosis associated

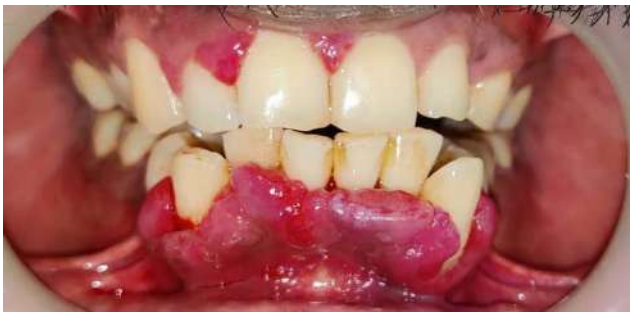

Figure1A: Pre-operative frontal view of gingival enlargement.

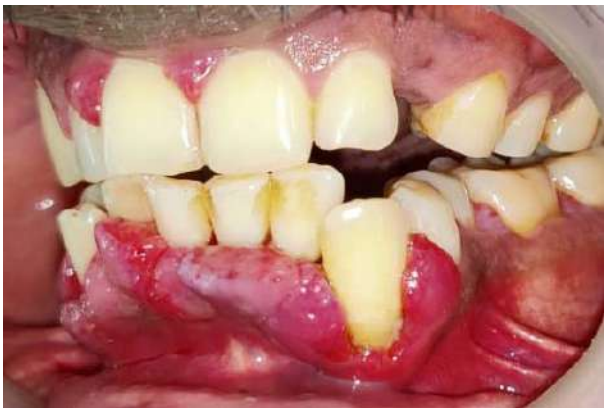

Figure 1B: Pre-operative lateral view

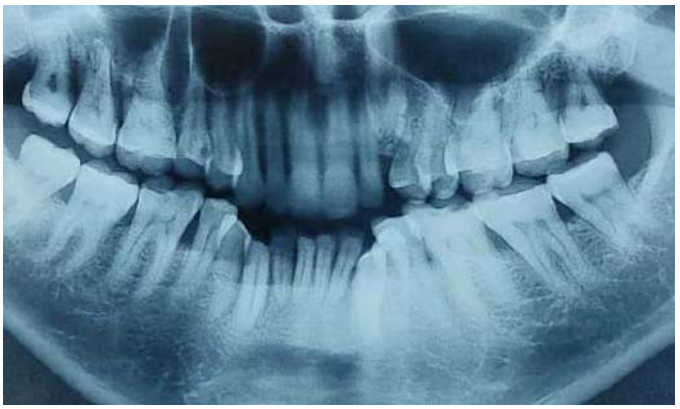

Figure 2: Panoramic view showing bone loss in relation to mandibular anterior and posterior regions.

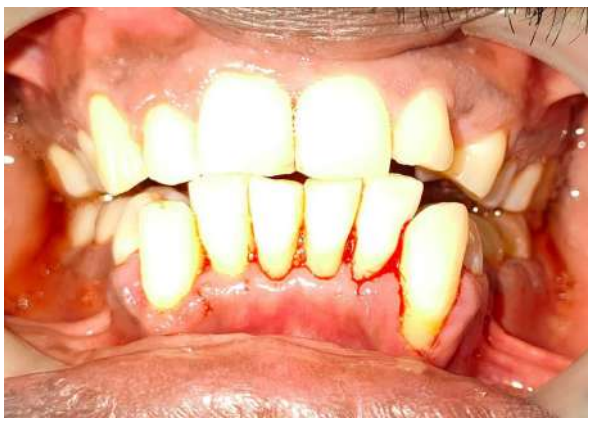

Figure 3: Post-operative view one week after gingivectomy showing complete resolution of enlargement.

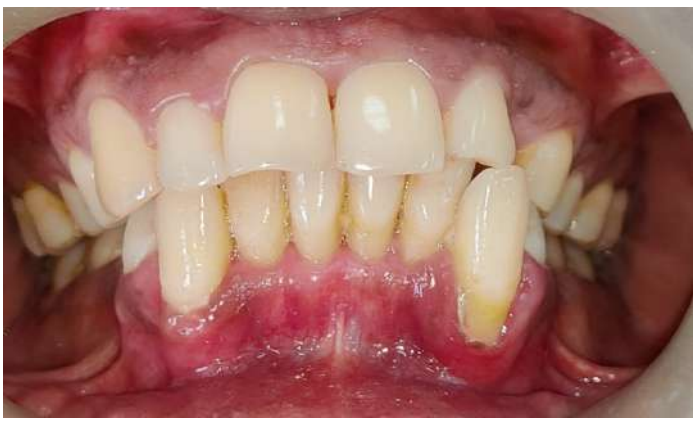

Figure 4: Two months post-operative view showing normal contour of gingiva.

gingival enlargement.

Case Management

On radiologic examination, panoramic view revealed bone loss in relation to mandibular anterior region (Figure 2). Hematological examination including complete blood count and differential count were performed to rule out leukemic gingival enlargement and found to be within normal limits. Viral markers were negative and ruled out HIV associated gingivitis. Assessment of vitamin C level in blood was done to rule out scorbutic gingivitis and found to be insignificant in this case. A negative Mantoux test report ruled out tuberculosis associated gingival enlargement.

To eliminate the inflammatory component of the enlargement non-surgical periodontal therapy was performed. After scaling and root planing, patient was instructed to use 0.2 % chlorhexidine mouth wash and gum paint for two weeks and advised to discontinue the antisensitivity tooth paste. On reevaluation after two weeks, erythema and bleeding subsided markedly and enlargement of the maxillary interdental region subsided but lesion on the mandibular labial region persisted. Hence surgical removal by external bevel gingivectomy was planned. After giving mental nerve block, enlargement was excised from labial aspect of 43 to 33 and sent for biopsy. Curettage was done in the area. Adequate hemostasis was achieved by suturing with 4-0 silk, local application of tranexaemic acid and pressure pack.

Clinical Outcomes

Post-surgical healing was uneventful. No recurrence was seen at the 3-month follow-up (Figure 3 and 4)

Histological Examination

Microscopically, the specimen showed hyperplasic para-keratinized stratified squamous epithelium overlying a moderately collagenous connective tissue stroma (Figure 5A). Within the connective tissue stroma diffuse and very intense inflammatory cell infiltrate was noted, chiefly composed of plasma cells. Vascularity of stroma was moderate with formed blood vessels. Extravasated red blood corpuscles were also noted. Histological evaluation revealed the diagnosis as plasma cell gingivitis (Figure 5B).

Discussion

Plasma cell gingivitis is a rare inflammatory condition characterized by diffuse and massive infiltration of the plasma cells into the connective tissue. In plasma cell gingivitis, gingiva appears swollen, erythematous and friable with loss of stippling.[1] It is classified into three categories – due to allergens, due to neoplastic origin, and plasma cell gingivitis due

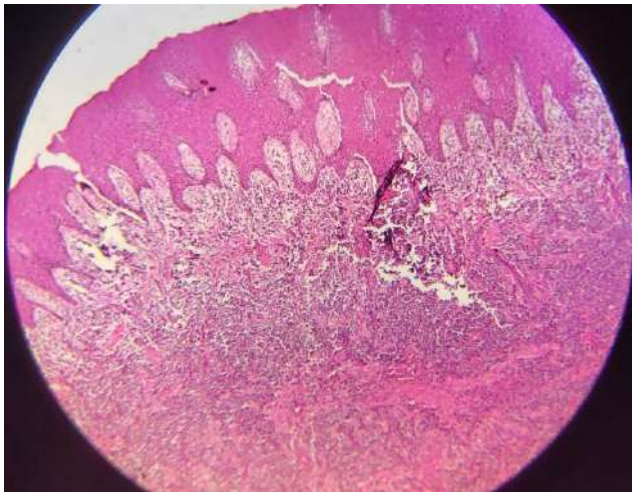

Figure 5A: Histopathologic analysis of the biopsy specimen showing hyperplastic para - keratinized stratified squamous epithelium overlying a moderately collagenous connective tissue stroma (hematoxylin and eosin staining, 10 x magnification)

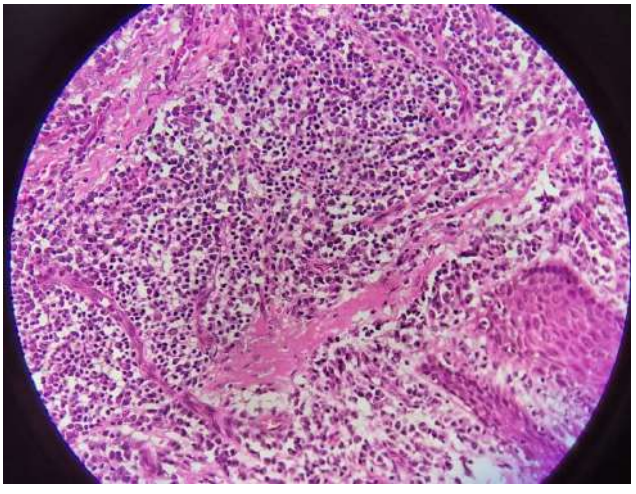

Figure 5B: Histopathologic analysis of the biopsy specimen showing dense infiltration of plasma cells in the connective tissue (hematoxylin and eosin staining, 40 x magnification)

to unknown cause.[8] It commonly presents as mild marginal gingival enlargement sometimes extending to involved attached gingiva.[1] It usually arises as a hypersensitivity reaction to components of chewing gum, dentifrices or dietary components. Some authors propose that bacterial plaque can also act as an allergen.[10] In our case, patient gave a history of usage of new dentifrice which could be the triggering factor.

The differential diagnosis of plasma cell gingivitis includes clinically inflammatory gingival hyperplasia and histologically mucous membrane pemphigoid, lichen planus, lupus erythematosus, multiple myeloma, solitary plasmacytoma. In the present case, since the lesion persisted after phase I therapy, inflammatory origin was ruled out. Complete remission of symptoms occurred after excision of the tissue along with cessation of the use of anti-sensitivity tooth paste and there was no recurrence after that. Histological evaluation ruled out pemphigus and pemphigoid lesions. Absence of atypical plasma cells ruled out plasma cell neoplasms. Normal hematological investigations ruled out leukemia. No attachment loss and bone loss are usually seen in plasma cell gingivitis.[10] Poor oral hygiene and associated periodontal disease can be the explanation for attachment and bone loss in this case.

Conclusion

The case described here is of plasma cell gingivitis that may be caused by inadvertent use of synthetic or herbal agents in dentifrices. Treatment of plasma cell gingivitis includes cessation of the possible allergen responsible for the condition along with non-surgical and surgical management as required. The importance of diagnosing plasma cell gingivitis is that the appearance of the gingiva can mimic a variety of more serious conditions such as pemphigoid, leukemic gingivitis, multiple myeloma, plasmacytoma, HIV associated gingivitis etc. Confirmation of diagnosis is done by histopathologic and immunohistochemical examination. Once the histologic diagnosis of plasma cell gingivitis is made, it is still difficult to identify the possible source of the allergen. So, this case report underscores the necessity of a comprehensive history taking, examination and appropriate diagnostic tests in order to arrive at a definitive diagnosis and treatment plan for gingival conditions which are nonresponsive to conventional therapy.

Declaration of Patient Consent

We certify that we have obtained all appropriate patient written consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Conflicts of Interest

There are no conflicts of interest.

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The Crevice Fluid - A Review

Aneta K Abraham[1], Jose Paul[2], Johnson Prakash D'Lima[3], Senny Thomas Parackal[4], Sruthi K Nair[5]

ABSTRACT

Serum, leukocytes, oral microorganisms, and structural cells of periodontium form the components of "gingival crevice fluid". Gingival crevicular fluid (GCF) components can be used to identify or diagnose active disease, as well as to forecast periodontal disease risk in individuals. The production of GCF rises both as a result of orthodontic tooth displacement and during the healing phase following periodontal therapy. Therefore, determining the levels of GCF constituents could be a better way in assessing individuals' risk for the infection.

Keywords: Inflammatory exudate, Gingival crevicular fluid, Periotron, Periodontitis, Biomarker.

Introduction

"Periodontitis" is a chronic inflammatory bacterial disease that weakens the periodontium, the tissue that supports the teeth, over time.[1] The oral cavity's defence mechanism includes gingival crevicular fluid (GCF), saliva, leukocytes, and the gingival sulcus epithelial barrier, all of which are generally successful in minimizing the detrimental consequences of high concentration of bacteria found in dental plaque.[2] The physiological fluid known as "crevice fluid" or "GCF" is produced by the gingival plexus, which is located next to the epithelial lining of the dentogingival region, in the gingival corium.[3] GCF is made up of periodontal structural cells, leukocytes, serum, and oral microorganisms.[2] GCF is a transudate of gingival tissue interstitial fluid that transforms into true inflammatory exudate during gingivitis and periodontitis.[4]

Historical Background

Since the nineteenth century, the presence of crevicular fluid has been recognised. Brill and coworkers laid the groundwork for future research into the physiology and composition of GCF with a series of pioneering investigations between the late 1950s to the early 1960s. In the 1970s, there was a surge in GCF research. Sueda, Bang, and Cimasoni were the first to investigate existence and function of proteins in GCF, notably enzymes. The Crevicular Fluid, a monograph by Cimasoni, was first released in 1974. An update appeared in 1983.[5] This extensive analysis is an attempt to detail the different aspects of GCF and its function in health, periodontitis and diagnosis of oral diseases.

Formation of Crevicular Fluid

GCF is regarded as a "serum transudate" and an "inflammatory exudate".[3] It has been proposed that

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GCF is produced (figure 1) by inflammation of the connective tissue beneath the junctional and sulcular epithelium.[6] In early experiments, fluorescein dye administered systemically was observed in GCF recovered from healthy gingival crevices in dogs.[7] Because the fluorochrome was unable to pass through the other oral epithelia, it was concluded that their permeabilities differ from those of the gingival pocket epithelium. Nonetheless, Brill's[8] research focused on the potential benefits of GCF, and he hypothesized that GCF was a crucial part of the crevicular region's defense systems. This hypothesis was reinforced by GCF's flushing effect, which was proved to be capable of eliminating the bacteria and carbon particles, put into the gingival crevice.[9] Equally comprehensive studies by Egelberg on the underlying sulcular and junctional epithelia's vascular histology backed up this extensive work.

According to Alfano's[3] work and Pashley's[10] hypothesis, "the initial fluid produced could simply be interstitial fluid that appears in the crevice due to an osmotic gradient". This fluid, which has a "pre-inflammatory" character, was previously categorized as a transudate but transformed to an inflammatory exudate when stimulated.

Table 1 Diverse elements of Gingival Crevicular Fluid(GCF)[12]

Cellular components	Epithelial cells, Neutrophils, Monocytes/ Macrophages, Lymphocytes, T-Lymphocytes, B-Lymphocytes
Electrolytes	Sodium, Potassium, Calcium
Inflammatory mediators	Total and subgroups of immunoglobulin IgG, Substance P, Prostaglandin E, Acute phase proteins, C-reactive protein, Leukotriene B4, Cytokines such as Interleukin -1, 2, 4, 6, 8,10, Tumour Necrosis Factor -α, Interferon -α
Enzymes generated from the host and their inhibitors	Aspartate aminotransferase, α2-Macroglobulin, α1-Proteinase inhibitor, Alkaline phosphatase, Acid phosphatase, Elastase, Elastase inhibitors, Serine proteinase, Cathepsin D, Immunoglobulin degrading enzyme, Dipeptidyl peptidases, Non specific neutral proteinases, Trypsin like enzymes, Collagenases, Matrix metalloproteinase-1, Matrix metalloprotenase-3, β glucuronide, Cathepsins, Cysteine proteinases (B,H , and L) MMP-8, Tissue inhibitor of MMP-1, MMP-13, MMP-2,MMP-9, Stromelysins, β-N-acetyl-hexosaminidase , Myeloperoxidase, Lactate dehydrogenase, Arylsulfatase
Bone- specific proteins	**'Bone formation markers':** Type -I procollagen propeptides , Alkaline phosphatase, Osteocalcin, Bone Gla proteins (BGP) **'Bone resorption markers':** Galactosyl hydroxylysine(GHYL), Tartrate-Resistant Acid Phosphatase (TRAP), N-terminal osteocalcin fragments, Glycosaminoglycans (GAGs), Pyridinium cross-linked collagen peptide fragments, Hydroxyproline
Bacterial and metabolic products	Lactic acid, Hydroxyproline, Endotoxins, Urea, Cytotoxic substances, Antibacterial factors
Tissue breakdown products	Osteonectin, Osteocalcin,Type I collagen peptides, Osteopontin, Calprotectin, Hemoglobin β-chain peptide, Pyridinoline crosslinks (ICTP, Polypeptide growth factor) Hydroxy proline, Fibronectin fragments Laminin, Glycosaminoglycans (Hyaluronic acid, Chondroitin -4-sulfate, Chondroitin-6-sulfate, Dermatan sulfate)

The Crevicular Fluid Composition

The composition of GCF can be seen broadly as the outcome of an interplay between the periodontal tissue cells and bacterial biofilm adhering to the tooth surface. GCF constituents (table 1) can be used to detect or identify active disease, as well as to identify patients who are likely to develop periodontal disease. GCF contains over forty different compounds. These substances may come from the host or be generated by bacteria that are present in the gingival crevice, but determining their origin can be challenging.

GCF is made up of[11]

- Cellular components
- Enzymes
- Bacterial products
- Organic components
- Inorganic components

Functions of GCF

- Wash out of foreign bodies:
  - It washes the crevice, epithelial cells, bacteria and other debris.
  - Its coronal flow pushes particulate matter including the microorganisms from the crevice.
- Antimicrobial actions:
  - It plays protective role for the host
  - It contains antimicrobial agent. eg. lysozyme
- Anti – inflammatory action:
  - It transports immunoglobulin A (IgA), IgG, and IgE of the immune systems.
  - Serum protein α2 macroglobulins inhibit proteolytic enzymes.
  - It contains polymorphonuclear leukocytes (PMNs) cells, leucocytes and macrophages which are capable of phagocytosis of bacteria and other microbes.
- Maintains the pH conditions
- Have plasma proteins, which may strengthen the epithelium's bond to the tooth.[11]

Methods to collect GCF

Using Absorbent paper strips

The collection methods are broadly classified as

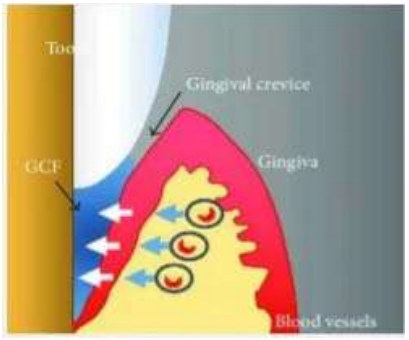

Figure 1 Origin of GCF

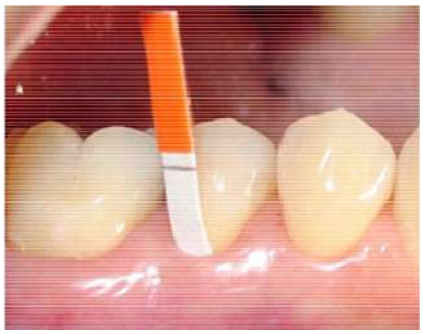

Figure 2 Intra-crevicular technique [4]

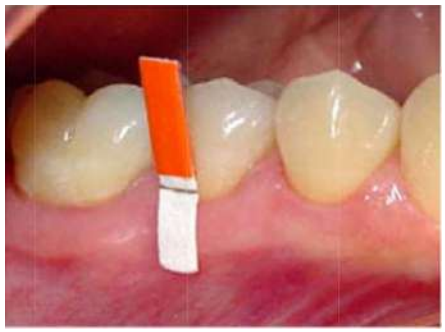

Figure 3 Extra-crevicular technique[4]

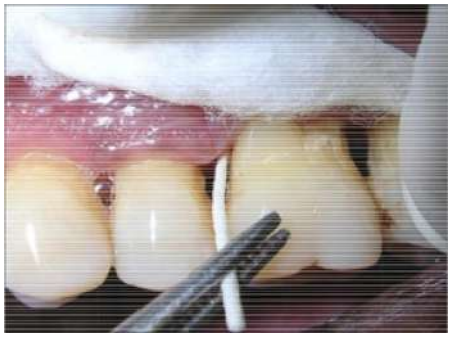

Figure 4 Pre weighed thread technique[11]

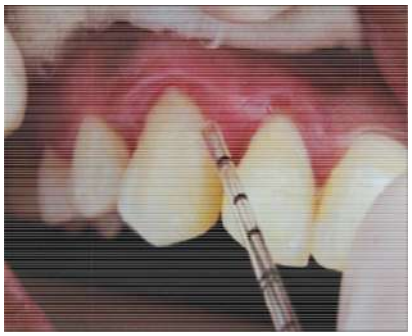

Figure 5 GCF collection using a capillary tube[4]

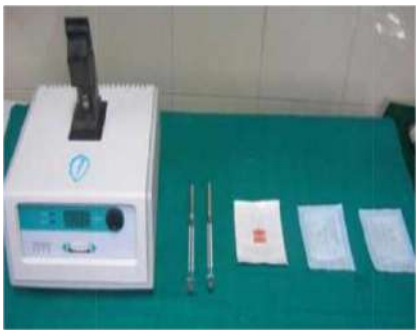

Figure 6 †Periotron[5]

†(Dental product, Winnipeg, Manitoba, Canada.)

intracrevicular (figure 2) and extra-crevicular (figure 3). The most common method is intracrevicular approach, and it can be characterized further according to whether, if the strip is placed merely at the entry of the sulcus or in the pathological pocket[13] (Loe & Holm Pedersen), or at the bottom of the pocket or till a minimal resistance is felt [14] (Brill's technique).

Using twisted threads which are preweighed

The twisted threads are maintained in the gingival crevice surrounding the tooth, and their weight is used to determine how much fluid has been collected[15] (figure 4).

Using Micropipettes

Micropipettes allow fluid to be absorbed through capillary action (figure 5). Capillary tubes with predetermined lengths and diameters were placed within the pocket, and their contents were centrifuged and examined later.[16]

Using Gingival Washing

This method involves perfusing an isotonic fluid into the gingival crevice, such as "Hanks' balanced salt solution." Cells and soluble components such plasma proteins are present in the resultant fluid, which is a diluted crevicular fluid.

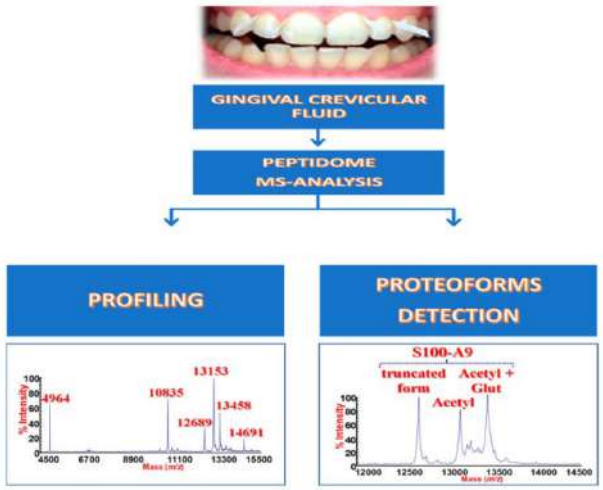

Figure 7 Overview of the main informational data extrapolated by peptidomics experiments on gingival crevicular fluid (GCF). Peptidomics workflow, without a digestion step, allows to preserve the endogenous information of peptides from GCF, including post-translational modifications and proteolytic products.[24]

Evaluation of the Amount of Fluid collected

- Direct viewing or staining:
  - The amount of collected GCF on the absorbent strip was calculated based on the distance the fluid travelled up the strip.
- Weighing the Strip
- Use of Periotron :
  - Harco electronics developed Periotron®† (figure 6) is an electronic technology for determining gingival fluid absorption on paper strips.

Problems in the Collection of GCF and Data Interpretation

Contamination

Blood, saliva, and tooth plaque are common contaminants in GCF samples, and their presence reduces the precision of both volume measurement and GCF composition.[5]

Sampling time

The protein concentration of the initially collected GCF will change if the collection time is extended.[4]

Determination of the volume

Evaporation is a notable issue in accurately determining the volume of GCF samples. Typically, the total volume collected is between 0.5 and 1 microliter. The percentage of error is deemed to be more important, as the total sample of GCF gathered is so little.[11]

Clinical Significance of Gingival Crevicular Fluid

Circadian Periodicity

The total volume of GCF, increases gradually between 6 a.m. and 10 p.m. and then declines. Interleukin-1beta concentrations and total amounts vary throughout the day in gingival crevicular fluid, with the lowest concentrations and total amounts in the morning and the highest in the evening.

Sex hormones

GCF flow is increased by female sex hormones, possibly because they create an increase in vascular permeability. Fluid production is increased by pregnancy, ovulation, and hormonal contraception.

Mechanical stimulation

GCF flow is stimulated by chewing and vigorous gingival brushing. Even minor stimuli, such as intrasulcular placement of a paper strip, increase fluid production.

Smoking

Smoking causes a transient and immediate, but significant increase in flow.

Chairside Diagnostic Kits

Chairside periodontal test kits can be divided into:

- Microbiological test kits
- Genetic kits
- Biochemical test kits

Commercially available diagnostic kits

1. Periocheck - Neutral Proteinases - Approved by (Food and Drug Administration) FDA
2. Pocket watch - aspartate aminotransferase (AST)
3. TOPAS – Toxicity Pre-screening assay (bacterial toxins and proteases)
4. Prognostik- Elastase - Not Approved by FDA and American Dental Association (ADA)
5. Dipstick method - MMPs
6. Under development, for β - glucuronidase and proteinases
7. Periogard – AST

Periodontal therapy

During the healing period after periodontal therapy, the production of gingival crevicular fluid increases. The findings of *Arnold et al* demonstrates that GCF dissipation is improved when the gingival sulcus has a larger area of keratinized gingiva; near closeness of alveolar mucosa and gingival margin, influences tissue fluid dissipation via the porous and movable alveolar mucosa, limiting the primary defence of the gingival sulcus by GCF concentration; and a close proximity of gingival margin.[17]

Orthodontic Forces

Gingival fluid production may increase as a result of orthodontic tooth displacement.[18] Analysis of various enzymes and cell mediators present in GCF can be used to track the biological response to orthodontic forces. Particularly during orthodontic treatment, levels of Interleukins-1, 2, 6, and 8 have been found to rise.[19] One to two days after the stimulus is provided, MMPs and their inhibitors have been demonstrated to peak in the GCF, and after about a week, they return to baseline.[20,21]

GCF and Implants

Neutral protease levels were discovered to be greater in implant sites that were moderately to severely irritated compared to mildly inflamed implant sites. Myeloperoxidase, neutrophil elastase, and levels were all noticeably higher in failing implants when compared to successful implants.

Interleukin -1 (IL-1) levels were nearly three times greater in GCF around implants than in healthy areas. In osseointegrated implants, elastase and collagenase activity was significantly lower than in adult periodontitis.

Current Status

As a fluid lying in close proximity to the periodontal tissue, the gingival crevicular fluid is the principal target in the search for periodontal disease biomarkers because its protein composition may reflect the disease pathophysiology. Biochemical marker analysis of GCF is effective for objective diagnosis in the early and advanced stages of periodontal disease. The proteomic analysis of GCF in different periodontal conditions demonstrates marked differences according to disease profile.[22]

Mass spectrometry (MS) is a powerful analytical tool used in numerous clinical laboratories worldwide. Recent advances in proteomics and MS technologies have provided promising methods for transdisciplinary discovery of novel markers. Peptidomics research represents one of the most interesting and challenging area of proteomics (figure 7). GCF peptidome could be a goldmine for the discovery of novel biomarkers of periodontal diseases. Peptidomics investigations, mainly based on profiling (top-down) strategies, which are well suited for proteoforms detection, are very few and therefore, till date, the picture of naturally occurring peptides and their role in GCF is still incomplete. In recent years it was proposed that GCF should be kept in consideration in "the wider contexts of oral

and systemic health.[23]

Future Directions

Gingival crevicular fluid is arguably the ideal biological fluid for the identification and measurement of biomarkers for periodontal disease because it can be easily and noninvasively sampled from the immediate vicinity of the affected tissues. An ever expanding pool of potential gingival crevicular fluid protein biomarkers for periodontal health and disease has been devised over the years, particularly with the recent implementation of proteomic technologies.

In the future, gingival crevicular fluid proteomic analyses will hopefully complement the clinical examination, with the aspiration that this approach will give added value in the personalized prevention, diagnosis and management of periodontal diseases.[25]

Conclusion

GCF, an exudate obtained from the periodontal pocket or sulcus, has been promoted as a useful tool for identifying periodontal disease activity. Because GCF constituent levels reflect the form and sensitivity of the hosts to the microbial plaque challenge, furthermore, because the periodontal disease progression is primarily determined by host reactions, determining levels of GCF constituent could be a better way to assess a person's risk for the disease.

Currently, there is no gingival crevice fluid indicators, either alone or in combination can be used to assess if periodontal therapy is adequate and/or essential to avoid additional periodontal deterioration. Despite their current limited utility, we believe that several components of gingival crevice fluid will have a role in periodontal diagnostics in future. Such markers, we believe, will be most valuable at the patient level.

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Trauma from Occlusion-A Review

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ABSTRACT

The impact of excessive occlusal load and the initiation and advancement of periodontitis has always been a subject of debate, with the occlusal damage being the primary focus which is caused by high forces applied to the periodontium. When traumatic occlusion was considered as a major etiological factor in periodontal disease, occlusal adjustments were thought to be one of the most significant aspects of periodontal therapy. However, only few researches have investigated excessive occlusal forces in humans because this would require treatment of one group of patients while other set of patients left untreated. This review article discusses historical context, etiological aspects, classifications, tissue response, signs and symptoms, diagnostic techniques, management, and current perspectives of trauma from occlusion.

Keywords: occlusal forces, periodontal disease, traumatic occlusion, occlusal traumatism

Introduction

The impact of occlusion and its subsequent effects on the periodontal tissue has been a big question for many years. Although this interaction has been linked to a range of occlusal disorders, the main focus has been on occlusal damage caused by high forces applied to the periodontium.[1] So, injury to tissues occurs in trauma from occlusion. And a traumatic occlusion is an occlusion that results in such an injury.[2] In an effort to explain and comprehend this condition, previous investigators used human autopsy specimens and a number of animal studies as a baseline for histological and clinical research. The results were frequently contradictory and varied. Differences between animals, forces employed, and a lack of controls were all concerns in the animal trials.[1] Excessive occlusal forces have only been studied in humans in a few studies since it would require treating one group while other set of patients remained untreated to examine the impact of increased occlusal pressure coupled with periodontal disease. This resulted in an unacceptably ethical quandary.[3]

Definitions

"A condition where injury results to the supporting structures of the teeth by the act of bringing the jaws into a closed position".[4] (Stillman,1917)

"When occlusal forces exceed the adaptive capacity of the tissues, tissue injury results. This resultant injury is termed Trauma from occlusion".[5] (Glickman 1962)

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Historical Perspective

For almost a century, occlusal trauma has been associated to periodontal disease. Karolyi identified a relationship between periodontal damage and high occlusal forces in 1901.[6] Stillman was the first to link traumatic occlusion to periodontal disease.[4]

Orban and Weinman established an approach for assessing the influence of traumatic occlusal loads on periodontium in 1956.[7,8] They concluded that occlusal forces had no effect on destruction of periodontal tissues. Instead, they interpreted the data showing the influence of gingival inflammation that advanced to the supporting bone tissue as the basis of periodontal disease.

Traumatogenic occlusion was not the reason for formation of soft tissue abnormalities like McCall's festoons and Stillman's clefts according to Goldman.[9] In the early 1960s, Glickman and Smulow presented the "co-destructive theory" stating that "traumatic occlusion could act as a cofactor in progression of periodontitis".[10]

Lindhe and colleagues in 1974 (in beagle dogs) and Polson and colleagues in 1976 (in squirrel monkeys) conducted studies on heavy occlusal loads. When plaque was not there on the tooth surface, severe occlusal stresses were found to induce a reduction in bone density and tooth mobility, but there was no evidence that it may cause attachment loss on its own.[11,12]

Waerhaug in 1979 investigated the association of plaque quantity, bone pattern and existence or absence of heavy loads of occlusion using number of autopsy specimens collected from cadavers. Waerhaug reached the conclusion that bone loss was always linked to plaque downgrowth, and that high occlusal forces had no association with vertical bone loss.[13]

According to Wolffe et al. "despite the constant stresses that caused the drifting of the teeth, the periodontium remained healthy".[14,15]

Excessive occlusal loads do not develop destructive periodontal diseases, but they can cause periodontal injury[16], according to the current debate on the impact of occlusal trauma in periodontitis. Many studies carried out in animals have looked at the impact of occlusal traumatism on periodontium and found out that these forces may not have any impact in advancement of periodontal disease, leaving certain questions unexplained.[17]

Etiology

Ross has separated the variables that cause chronic destructive periodontitis into two categories[1,2]:

a) Precipitating factors: Irritants and strong occlusal loads act as precipitating factors which causes destruction of periodontal tissues that have already been compromised by predisposing factors.

b) Predisposing factor: Developmental variables, systemic component and functional mechanisms are all predisposing variables that contribute to the histopathologic changes. Again, divided into:

A. Intrinsic factors:

- Relationship between the long axis of teeth and the stresses they could be exposed to
- Morphology of tooth root is an important factor (short, conical, fused or narrow roots are more susceptible)
- Alveolar process architecture. (Alveolar bone quantity and quality)

B. Extrinsic factors:

- Local conditions that promote to alveolar bone loss, such as plaque
- Injudicious bone excision during surgical periodontal treatments
- Presence of lengthy span fixed denture prosthesis.
- Neurosis-induced parafunctional tendencies
- Over hanging margins of filling and presence of food impaction
- Improperly shaped prosthesis
- Partial denture prosthesis which is improperly constructed

Other Predisposing Factors

Tooth loss: Early tooth loss due to caries or an accident is common and can lead to occlusal traumatism.

Faulty restorations: If the tooth or teeth can drift or rotate into a harmonious occlusal relationship, the

traumatism may be transient; if not, the traumatic situation may become chronic. As a result, during restorative procedures, the principles of good functional occlusion must be followed.

Improper occlusal adjustment: If employed indiscriminately, therapeutic measures to rectify faulty functional occlusion can cause further aggravation. Further stress is caused by occlusal correction methods that result in occlusal contact relationships with forces that are not directed axially.

Dysfunction of Temporomandibular joint: Minor functional occlusal discrepancies combined with psychoneurotic behaviours, as well as severe dysfunctional occlusal relationships alone, can cause temporomandibular joint (TMJ) ailment.[18]

Classification:

According to duration of course: Acute or chronic

Acute occlusion trauma: A sudden occlusal pressure, like chomping down on a heavy surface, e.g., olive pit. Restorative materials and some prosthesis that shift directions of forces on the teeth can potentially cause this type of occlusal traumatism.[2]

Chronic occlusion trauma: Prevalence is more, this also has a significant role therapeutically. Also, more commonly caused by occlusal changes induced by wearing, drifting, and extrusion of tooth as well as parafunctional activities like bruxism.[2]

According to nature of course: Primary trauma and Secondary Trauma

"If occlusion trauma is regarded the primary etiologic cause in periodontal damage and occlusion is the only local modification to which a tooth is subjected", categorized as "primary occlusal trauma". Examples are injury to tissues caused by:

1. "High filling"
2. Increased pressures on the abutment and antagonistic teeth due to prosthetic replacement
3. Tooth extrusion into areas of edentulous space that have not been replaced
4. Orthodontic movement causes teeth to move into functionally unfavourable positions.[19]

Box's classification

Physiologic occlusion:

It's a state of equilibrium where the pressures exerted on the tooth while in occlusion are coordinated and do not disrupt the tooth's natural connection with its supporting tissue structures, according to Box.

Traumatogenic occlusion:

As the consequence of the occlusion, the periodontal tissue is overstressed, resulting in periodontal damage.[20]

Tissue reaction to Trauma caused by Occlusion

There are three phases of injury:

Stage I: Injury

Around rotational axis tooth rotates under occlusal load, creating pressure and tension zones on opposing fulcrum sides. When pressure is raised, hyalinization happens. Areas of the ligament necrosis occur after disruption to fibroblasts as well as other connective tissue cells.[21]

Stage II: Repair

The natural periodontium is continually repairing itself. The wounded tissues generate greater reparative activity during trauma from occlusion . An attempt is made to repair the periodontium that has been damaged. Forces are only traumatic if the damage they cause exceeds the tissues' ability to repair themselves. Nature seeks to repair the thinning bony trabeculae with new bone when bone is resorbed by high occlusal stresses. Buttressing bone production is an important aspect of the TFO reparative process because it attempts to compensate for missing bone.[9]

Stage III: Adaptive remodeling

If mechanism of repair cannot maintain destruction induced by occlusal load, periodontal remodeling takes place in order to establish a structural relationship which is not harmful to periodontium that led to thicker ligament component along with crest which is funnel shaped and also angular defect in junctional epithelium, but there is no pocket formation.[22]

Trauma from occlusion: Diagnostic criteria[1]

1. The most prevalent presentation of "primary trauma" is tooth mobility of tooth. To assess mobility, mechanical and electrical devices can be utilised. The Miller classification system1 is used to perform subjective mobility evaluations, with values ranging from 0 to 3.
2. Teeth can tilt and migrate individually or as a group. The percussive effect teeth upon tapping using an instrument shift to a dull one from a resonating tone along with sound supportive framework when primary TFO has noticed.
3. Masticatory muscle examination for evaluation of hypertrophy or symptoms of hypertonicity, as well as spasms observed in muscle groups.
4. The TMJ examination, as well as evaluation of mandibular deviation.
5. Fremitus test: The vibrating characteristics with respect to dentition are measured once the teeth being positioned in contact orientations and movement pattern. Damp forefinger is put on the buccal aspect and labial aspect of maxillary dentition. Maintaining maximal inter cuspal position is critical. Instruct the patient to grind in the lateral, protrusive, and lateral protrusive contacting actions and postures systematically. According to fremitus test scale, movements are graded.

Grading of Fremitus[1]

- Class I: Detection of "mild vibration"
- Class II: Vibrations that are clearly felt on palpation, however no observable movements noticed.
- Class III Movement that can be appreciated with the naked eye[1].

Table 1:

Occlusal trauma: Clinical indicators[23]
• Positive fremitus
• Mobile tooth
• Discrepancies in occlusion
• Wear facets on teeth
• Pathologic migration of tooth
• Fracture of tooth/Teeth
• Thermal sensitivity

Radiographic Indications[1]

1. Periodontal ligament widening, often accompanied by lamina dura thickening, that occurs laterally along the root in the apical region and at bifurcation sites.
2. The destruction of interdental septum vertically rather than horizontally, resulting in intrabody abnormalities.
3. Radiolucency of the alveolar bone
4. Root resorption
5. Hypercementosis
6. Secondary dentin laid down in the pulp chamber

Abfraction and its developmet

The term abfraction refers to pathological loss of hard tooth material caused by biomechanical loading forces. These are angular or wedge-shaped flaws that develop on cementoenamel junction due to enamel and dentin flexure and eventually fatigue. It's also worth noting that occlusal stresses that causes cervical flexure might break hydroxyapatite crystal connections, resulting in microfractures and ultimately enamel loss.[24]

Pathologic Tooth Migration (PTM)

Pathologic migration is the term used to describe tooth displacement caused by periodontal disease disrupting the balance of elements that preserve physiologic tooth position. Pathologic migration is a typical symptom of disease and can occur alone or in conjunction with gingivitis and formation of pocket as the disease progresses. The most prevalent site of pathologic migration is the anterior teeth; however, it can also affect the posterior teeth. The teeth may move in any direction, and they are commonly mobile and rotated.[2]

Trauma from Occlusion around Implants

In the last few decades, the use of dental implants have increased the prosthetic options for edentulous

patients.

A case report by Uribe et al[25] in 2004 reported that occlusal stress was associated with marginal peri-implantitis. Erythema and a periodontal pocket were present when the implant was placed.An abnormal contact was observed while assessing the occlusion with the aid of articulating paper. So, the patient received occlusal correction along with surgical therapy.[25]

Occlusal overload was shown to be the cause of peri-implant bone loss in a research from 2008, which may be corrected by treating the traumatic occlusion.[26]

In 2014, Merin et al. presented one case study showing bone deterioration around implant can be reversed by just correcting the occlusion. The subject of this case report was a 63-year old female patient. On occlusal examination, the implant showed traumatic occlusion. Occlusal correction was done by the author. Routine periodontal evaluations, according to the authors, should include not just periodontal and radiographic data, but also the evidence associated to occlusion.[27]

Passanezi E et al.[28] in 2018 reported that the necessity for an accurate diagnosis to treat the condition adequately should be done by taking into account that,

Occlusal trauma and mucositis or peri-implantitis may coexist without producing a combined lesion; Trauma from an occlusal lesion may be produced in the crestal bone around implants and the bone matrix remaining in the lesion of occlusal trauma in the marginal area of the implant can have complete recovery after controlling the traumatogenic occlusion.

To diagnose and treat lesions that resemble peri-implantitis, care must be taken. also, care should be given while correlating the peri-implant probing, crest of the bone, soft tissue margins to produce differential diagnosis of peri implant mucositis or peri-implantitis either associated or not associated with trauma from occlusion.[28]

Management of Trauma from Occlusion

Despite the fact that occlusal trauma and other types of trauma might occur at the same time, the two conditions can be treated separately, with unique treatment aims and outcomes. The management of occlusal traumatism has the following objectives:

- To reduce or eliminate tooth mobility
- To maintain a stable and reproducible maximal inter cuspal position
- To establish a proper occlusion with appropriate phonation and aesthetics, as well as efficient masticatory function
- To eliminate parafunctional habits

At any point of time during periodontal treatment, occlusal trauma can be managed. In the treatment of chronic periodontitis, excessive loads are avoided or minimised. Occlusal therapy can be done in a variety of methods that include:

- Occlusal correction; parafunctional habits management
- Stabilization of movable teeth on a temporary or long-term basis
- Tooth movement in orthodontic therapy

After completion of treatment,

- Teeth movement is reduced or eliminated.
- Progression of tooth migration is prevented
- Consistent physiologic occlusion is achieved.
- Results should be aesthetically pleasing.
- Radiographic signs and symptoms should be eliminated/reduced.

Eliminating premature contacts, fremitus, and occlusal interferences done, which reduced pain and improved patient comfort.[29]

Current Concepts

After periodontal health is re-established, occlusal therapy may be used, if necessary, to aid in reduction of mobility when such a problem is linked to a widening periodontal ligament space, according to Bhola et al.[30] (2008). Redirecting occlusal forces and bringing tooth mobility down to physiological limits may have an impact on the long-term regeneration of bone and attachment of connective tissue.

A clinical example of a 13 mm pocket probing depth on the mesial surface of a lower molar with Grade II clinical mobility and bleeding on toothbrushing was documented by Santamaria et al.[31] in 2009. The patient had nonsurgical periodontal therapy, which

included occlusal correction, scaling, and root planing. After six months, there were decreases in bleeding on probing, tooth mobility, and pocket probing depth to 4 mm. These findings suggest that minimising occlusal interferences and receiving the proper periodontal treatment improve periodontal parameters, and that clinicians should carefully investigate excessive occlusal forces.

Occlusal adjustment to remove occlusal interferences in centric relationships, protrusion or lateral excursions, mandibular eccentric movements, and contacts at balancing sides led to an improvement in periodontal parameters, according to a systematic review (Foz et al.[32] in 2012) of four studies that investigated the effects of occlusal adjustment in the reduction of periodontal disease progression. However, more research is required to fully comprehend the advantages of occlusal modification in periodontal therapy.

Ben-Gal[33] in 2013 came to the conclusion that "contact distribution between the prosthesis and opposing jaw play a substantial role in preserving the prosthesis, but have a lesser effect on implant survival and bone loss" after reviewing the literature in relation to current concepts in implant occlusion. There are several reviews on the subject, but they all seem to express the perspectives and clinical experience of the authors.

The 2017 world work shop defined excessive occlusal force and changed the name as traumatic occlusal force[34].

Definition of Excessive occlusal force: "Occlusal force that exceeds the reparative capacity of the periodontal attachment apparatus, which results in occlusal trauma and/or causes excessive tooth wear (loss)".

Definition of Traumatic occlusal force: "Any occlusal force resulting in injury of the teeth and/or the periodontal attachment apparatus. These were historically defined as excessive forces to denote that the forces exceed the adaptive capacity of the individual person or site"[34].

A classification for occlusal traumatism was approved by the work group

2017 World Workshop Classification for occlusal traumatism[34]

Table: 2 TRAUMATIC OCCLUSION
Primary causes
Secondary causes
Occurrence of trauma due to orthodontic forces

Consensus report published on "Classification of Periodontal and Peri-Implant Diseases and Conditions"[34]:

- No conclusive evidence exists to support the claim that occlusal traumatism damages human periodontal tissue, and having minimal proof that traumatic loads of occlusion can cause PDL inflammation in human being and animals.
- Traumatic occlusion has been shown in animal studies that enhance bone loss, and observational studies reveal that such traumatogenic occlusal loads are linked to the rate of destruction of attachment apparatus.
- In humans, there is lack of evidence showing the relationship of traumatic occlusal pressures and non-carious cervical lesions (NCCL)., nor that occlusal trauma accelerate the advancement of periodontitis in people.
- Abfraction, "a wedge-shaped defect: that develops at the "cementoenamel junction", is caused by tooth structure flexure and presence is unclear according to recent findings.
- According to observational research, traumatic occlusion does not induce gingival recession.

One randomised clinical research found that limiting tooth movement can help with periodontal treatment outcomes. There isn't enough clinical evidence to assess the effects of removing symptoms of traumatic occlusal stresses on periodontal treatment response.

The evidence for the link between traumatic occlusal forces and periodontitis was examined in a systematic review by Jorge Iván Campio[35] in 2019. Following a review of thirty articles, cross-sectional studies found a significant correlation between clinical attachment level, probing depth, and occlusal discrepancies. When groups with and without occlusal discrepancies are compared, the magnitude of the effect

is however insignificant. They came to the conclusion that there is only weak evidence from available human studies that proves traumatic occlusion is related to periodontitis.[35]

Conclusion

In both animal and human research, occlusal trauma has been associated to the advancement of the periodontal disease. According to all investigations, stresses on occlusion are not able to promote "plaque-induced" periodontal disease or periodontal attachment destruction, with latest research backups. If occlusion and the status of the periodontium are linked in periodontal health and tissue loss in periodontal disease, they should be linked in periodontal treatment and the maintenance of post-treatment health.

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JSPIK

Management of Lobular Capillary Hemangioma - A Case Report

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ABSTRACT

Oral soft tissue enlargements are often a diagnostic challenge to the clinician due to its diverse pathogenesis. Lobular capillary hemangioma (LCH) is a variant which clinically resembles pyogenic granuloma (PG). However, it is histologically different and is organized as lobular aggregates of blood vessels. This article focuses on a case of LCH which was clinically diagnosed as pyogenic granuloma.

Keywords: benign gingival enlargements, pyogenic granuloma, lobular capillary hemangioma, diode laser, gingivectomy

Introduction

Diverse groups of pathologic lesions cause oral soft tissue enlargement. Pyogenic granuloma (PG) is a common non neoplastic tumor like growth of oral soft tissue, especially attached gingiva. Pyogenic granuloma represents an exuberant connective tissue proliferation to a known stimulus or injury. The term pyogenic granuloma is a misnomer because it doesn't produce pus nor does it represent granulomatous inflammation.[1,7] Lobular capillary hemangioma (LCH) is a relatively rare variant of pyogenic granuloma, currently categorized as vascular tumor under the classification scheme of the International society for the study of vascular abnormalities.[2] It is usually pedunculated, lobulated, pink to purple soft tissue mass of size varying from few millimeters to several centimeters. Typically, a painless growth which often bleeds easily. The lesion may exhibit rapid growth, may alarm the clinician to suspect malignancy.[2] This case report is that of a LCH in a 68-year-old female hypertensive patient which was treated by a combined conventional gingivectomy and diode laser and under follow up.

Case Report

A 68-year-old female patient reported to the Department of Periodontology, at Government Dental College, Kannur with a chief complaint of painless growth of three months duration, distal to the maxillary left canine (Figure 1). The lesion was initially small and gradually increased in size. Intraoral examination revealed 1cm x 0.6 cm x 0.5 cm sized sessile, smooth surfaced, pinkish red nodular mass involving the marginal and attached gingiva, extending mesiodistally from the distal aspect of the canine and involving one centimeter (1cm) to the edentulous alveolar ridge. The swelling was soft to firm on palpation with intact surface without bleeding spot or ulceration. Oral hygiene and periodontal status of the patient were satisfactory. Intraoral periapical radiograph (IOPA) findings were normal. Routine blood investigations were done. Patient was hypertensive (under medication), without any other systemic illness. The blood investigations came to be normal and hypertension of the patient was under control. Two weeks later after accessing the medical status, the patient was scheduled for surgical excision.

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As the initial phase of treatment, thorough oral prophylaxis was carried out. Excision of the gingival lesion was done by means of external bevel gingivectomy under local anesthesia (two percentage lignocaine with adrenaline injection) (Figures: 2 & 3) and the area was thoroughly curetted and debrided. Diode laser (CHEESE medical diode laser system) with 1.9 Watt was also used for debridement, irrigated with betadine and saline, Vitamin E topically applied, followed by periodontal dressing with Coe pack. On the fifth day periodontal dressing was removed and irrigated. The surgical site healed uneventfully and near normal appearance of gingiva was seen after three weeks. (Figures: 4,5 & 6)

Histopathologic Examination

Gross specimen was grey brown tissue bits each average measuring 1cm x 0.5 cm. Microscopically revealed tissue fragments lined by squamous epithelium. Subepithelial connective tissue shows proliferated capillaries lined by endothelial cells organized in a lobular aggregate, intervened by edematous fibrous stroma containing inflammatory cells (Figures: 7 & 8). Impression- lobulated capillary hemangioma (pyogenic granuloma)

Discussion

Pyogenic granuloma is a tumor like gingival enlargement that is considered as an exaggerated conditioned response to minor trauma. The lesion varies from discrete spherical, tumor like mass with pedunculated attachment to a flattened keloid like enlargement with a broad base. The lesion tends to involute spontaneously to become fibroepithelial polyp or it may persist relatively unchanged.[3] Depending on the stage of the lesion, histopathologically it may be inflammatory to fibrous. Hormones and local irritants can also affect its histopathological presentation. LCH is a variant of pyogenic granuloma which usually appears as a raised bright red lesion that may grow rapidly and bleed profusely. These lesions are sometimes caused by injury or use of certain drugs and often recur after the treatment. Histopathology helps to differentiate LCH from PG, in which the connective tissue is composed of numerous blood vessels that are organized in lobular aggregates.[2]

Elimination or correction of injurious agents, effective plaque control, patient motivation, precise surgical excision etc. are methods for effective management of such lesions.[8,9,10] Various modalities such as conventional scalpel, electrosurgery, cryosurgery etc.

Figure 1- Preoperative

Figure 2 - Excised gingival tissue

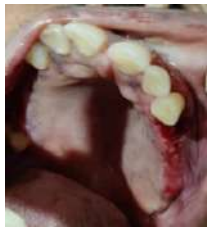

Figure 3 Immediate Postoperative view

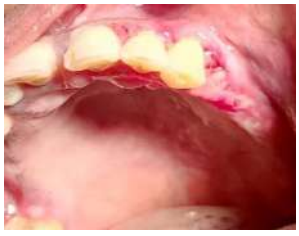

Figure 4 - 1 week postoperative

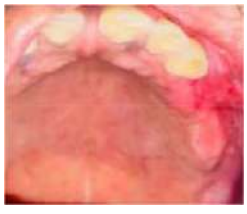

Figure 5 - 3 weeks post operative

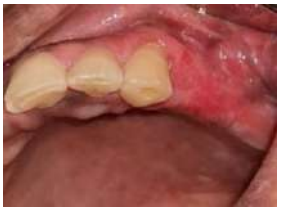

Figure 6- 1.5 months postoperative

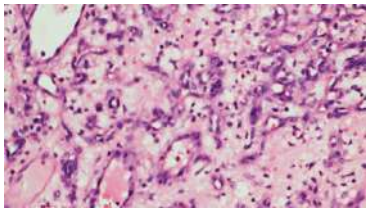

Figure 7- photomicrograph of histopathological section

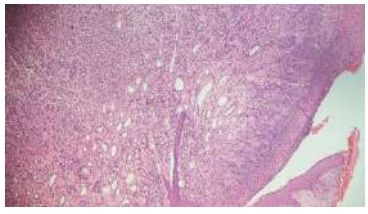

Figure 8- photomicrograph of histopathological section

are used for surgical excision for decades. Introduction of laser is an innovative approach for surgical management of such lesions.[4,5,6,8,9,10]

Diode laser has "hemoglobin" as one of their target chromophores, hence they provide effective hemostasis at surgical site.[8,9,10] In the present case we used a combination of conventional surgical gingivectomy and diode laser. A good hemostasis was achieved in present case using diode laser. Usage of laser provides added advantage of minimizing hemorrhage during excision and increased patient comfort. Diode laser was used around 1.9 Watts after conventional gingivectomy in this case. Definite steps should be practiced to safeguard the use of laser. Wave length specific eye gear should be used by operators, assistants and patient to prevent the retinal damage.

Conclusion

The present case report highlights the advantage of combined conventional surgical and diode laser assisted excision. The various treatment modalities should be evaluated for treating such cases. The authors emphasized using combination of both conventional surgery and diode laser with better predictable results without any recurrence of growth. However, more cases with longer follow up should be carried out to establish the outcome.

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JSPIK

Relationship between Periodontitis and Atherosclerosis

Veena Venugopal[1], Annie V Issac[2]

ABSTRACT

Periodontal disease is characterized by the deterioration and loss of the tissues that support teeth. Periodontitis has a complex etiology that includes a number of risk factors and systemic disease. Putative risk factors for atherosclerosis are obesity, inflammation, and infection. Periodontal disease is a candidate risk factor that shares many of these traits and periodontitis can initiate progression of atherosclerosis.

Keywords: Periodontitis, Atherosclerosis, Bacteremia, Cytokines

Introduction

Periodontitis is an infectious disease resulting in inflammation within supporting structures of teeth resulting in progressive attachment loss and bone loss. Periodontitis may affect initiation and progression of systemic conditions and vice versa. The total surface area of pocket epithelium usually exposes to subgingival bacteria and their product is estimated as the size of the palm of an adult hand.[1] Subgingival microbiota in patient with periodontitis provides a significant and persistent gram-negative bacterial challenge. Inflammation plays a significant role in atherogenesis pathology starting from endothelial cell damage to plaque rupture.

Atherosclerosis is a slow and progressive disease of blood vessels characterized by deposition of fatty plaque on the inner wall of vessel lumen leading to obstruction of normal blood circulation. It is a condition characterized by deposition of low-density lipoproteins, cholesterol, cellular waste products and calcium which accumulate in the blood vessel wall resulting in narrowing of vessel wall and thus obstructs blood flow. Atherosclerotic plaque is composed of fat laden macrophages called foam cells, Tmcells, mast cells and matrix metallloproteinases.

Periodontitis and atherosclerosis share common risk factors like obesity, advanced age, stress, lower socioeconomic status cigarette smoking, hypertension, diabetes and genetic alterations.

Oral hygiene is a lifestyle indicator that has an impact on personal hygiene and health, according to De Stefano et al.[2] This further explains the link between periodontal diseases and cardiovascular diseases. Therefore, factors responsible for increased risk for periodontitis may also be responsible for increased risk of atherosclerosis. There is increased risk for cardiovascular diseases among patients who have periodontal diseases. Periodontal infection can lead to mild bacteraemia and endotoxemia in affected patients.

Pathways Linking Periodontitis and Atherosclerosis

Four major pathways have been suggested to explain the relationship between atherosclerosis and

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periodontitis, these pathways include (Fig. 1)[3]:

1. Direct bacterial impact on platelets: This pathway is supported by the virulence factors of two types of oral bacteria Porphyromonas gingivalis and Streptococcus sanguis. These bacteria produce the collagen-like platelet aggregation protein which in turn stimulates platelets aggregation both in vitro as well as in vivo. Some strains of Porphyromonasgingivalis and Streptococcus sanguinis produce platelet aggregation-associated protein (PAAP), which aids in the aggregation of platelets on the innermost layer of the vessel lumen. In the pathogenesis of thrombogenesis and thromboembolism that result in acute myocardial infarction and stroke, platelet aggregation is a key player.[4]

2. Autoimmune responses: Antibodies that cross-react with periodontitis causing bacteria and human heat -shock proteins have been recognized. In addition, some oral bacteria such as P. gingivalis can invade aortic and cardiac endothelial cells through fimbriae.

3. Invasion of bacteria into endothelial cells and macrophages: Numerous surveys found that there were certain oral bacteria in the atheromatous plaque.

4. Endocrine-like effects of pro-inflammatory mediators: Pro-inflammatory mediators have an endocrine-like effect on vascular endothelial tissue as well as an elevation in C-reactive protein (CRP) and fibrinogen among patients with periodontal diseases.

Acute-phase proteins

Localized inflammation that is brought on by bacterial infection is a hallmark of periodontitis. This inflammatory response is thought to be greatly influenced by acute-phase proteins. C- reactive protein and fibrinogen are two important acute phase proteins produced by the liver, which are considered as independent risk factors for coronary artery disease. Periodontitis-induced elevation in level of acute phase proteins suggests the potential role of periodontal disease in pathogenesis of cardiovascular disorders.[5]

Porphyromonas gingivalis has certain proteins called heat shock proteins that are similar to corresponding human proteins. When these proteins travel to another site, antibody directed towards bacterial heat shock protein, because of structural homology react with human heat shock protein and thus induces an autoimmune reaction called Molecular Mimicry.

Plasma fibrinogen, plasma lipoproteins, white blood cell count and Von Willebrand factor increased during periodontitis which increases blood viscosity. Increased viscosity of blood leads to increased risk of thrombus formation and subsequently leads to ischemic heart disease and cerebrovascular accidents.

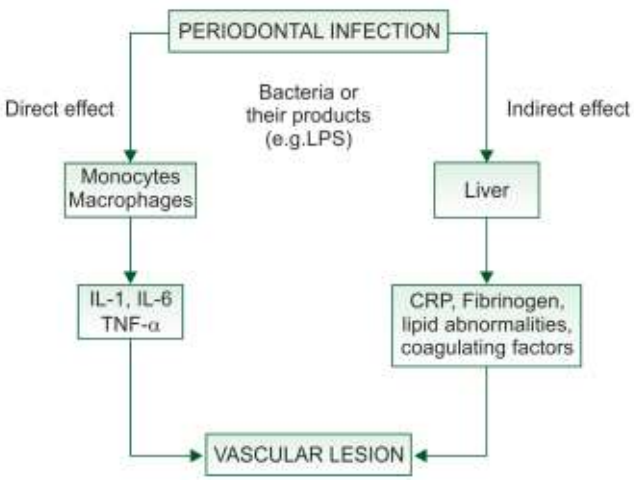

Fig 1: Systemic Spread of Periodontal Infection[3]

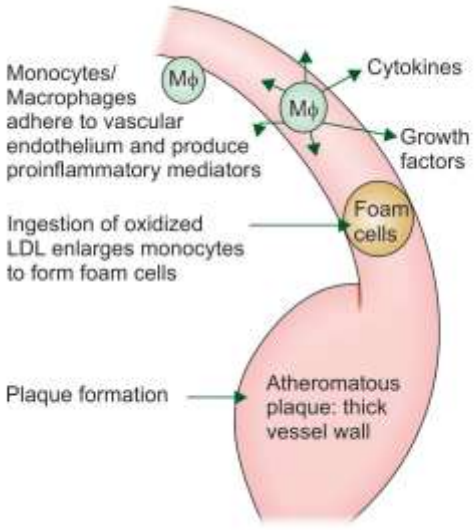

Fig 2: Pathogenesis of Atherosclerosis[7]

Adhesion molecules play a significant role in adhesion of circulating monocytes to vascular endothelium. Proinflammatory cytokines and mitogenic factors further play a role in atheroma progression and thickening of the arterial wall.[6] Examples are Intercellular adhesion molecules (ICAMs), Endothelial leukocyte adhesion molecule-1 (ELAM-1) and Vascular cell adhesion molecule-1 (VCAM-1). Monocytes adhere to vascular endothelium through these adhesion molecules penetrate epithelium and migrate under the arterial intima, Ingestion of oxidised LDL enlarges monocytes to form foam cells which constitute atherosclerotic plaque. (Fig: 2)[7]

Leishman et al.[8] determined relationship between periodontal pathogen load and anti-human heat shock protein 60 (hHSP60) antibodies in patients with cardiovascular disease. Periodontal pathogen load of Porphyromonas gingivalis, Tannerella forsythia, Fusobacterium nucleatum and Aggregatibacter actinomycetemcomitans was determined in seventy-four patients with myocardial infarction using quantitative real-time polymerase chain reaction. The anti-hHSP60 levels and T. forsythia populations showed the most positive relationships. Higher concentrations of P. gingivalis and T. forsythia with noticeably raised anti-hHSP60 levels were found in patients with periodontal pockets of more than 4mm depth.

According to Bokhari et al.[9] there may be a link between systemic biomarkers of coronary heart disease and clinical periodontal parameters of periodontitis. Three hundred and seventeen patients with periodontitis and angiographically confirmed coronary heart disease who were older than thirty and free of other systemic illnesses were included in this study. Clinical attachment level (CAL), bleeding on probing (BOP), probing depth (PD) are periodontal markers. Systemic parameters like C-reactive protein (CRP), fibrinogen (FIB), and white blood cell (WBC) levels were recorded. The strongest correlation was reported between BOP and CRP levels, and linear regression demonstrated a connection between periodontal and systemic parameters. BOP was the only periodontal indicator significantly linked with each systemic parameter, and stepwise regression analysis models showed that BOP was a predictor of systemic CRP levels (CRP, FIB, and WBC).

In order to better understand how periodontal disease affects an individual's vulnerability to atherosclerotic lesions, Beck J et al. developed a model of hyperinflammatory macrophages that are genetically determined.[10]

Conclusion

The periodontal medicine is an emerging field that offers insight into the concept of oral cavity as on system interconnected with the whole human body. Periodontal infection is a potential risk factor for Atherosclerosis. Fortunately, it is a modifiable risk factor unlike age, sex and genetic factors.

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JSPIK

Titanium Activated Platelet-Rich Fibrin (T-PRF) - New Elixir of Periodontics

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ABSTRACT

Oral and maxillofacial surgery, plastic surgery, and sports medicine are among the surgical specialties where platelet concentrates (PC) platelet-rich plasma (PRP), and platelet-rich fibrin (PRF) are widely employed. All of these technologies aim to extract from a blood sample every component that could be employed to enhance healing and encourage tissue regeneration. Since its debut in 1954 and the subsequent introduction of Titanium platelet-rich fibrin, Advanced platelet-rich fibrin, and Injectable platelet-rich fibrin, Platelet concentrates have advanced significantly. These PC also successfully find a variety of uses in implant dentistry and Periodontics. The purpose of this study is to provide a concise overview of T-PRF, preparation methods, new developments, and their numerous clinical and technical aspects and applications.

Key words: Platelet concentrates; Platelet rich plasma; Platelet-rich fibrin; Pure-platelet-rich fibrin; Leukocyte- and platelet-rich fibrin; Titanium-platelet-rich-fibrin

Introduction

The use of platelet-rich products to treat a variety of clinical dental disorders has gained popularity in recent years. In France, platelet-rich fibrin (L-PRF) was initially used as an autologous leukocyte biomaterial in 2001.[1,5] L-PRF doesn't need anticoagulant or bovine thrombin, in contrast to other platelet concentrates (nor any other gelling agent). L-PRF is therefore regarded as a second-generation platelet concentration [2] L-PRF has been used successfully by J. Choukroun et al., 2001,[5] Z. Mazor et al., 2009[15], and A. R. Pradeep et al., 2012[14], although some research suggest that glass evacuated blood collection tubes with silica activators may pose a health risk (S. M. O'Connell et al., 2007).[17] The silica particles in the tube are small enough to remain colloidally suspended in the buffy coat, fibrin, and platelet plasma layers even though they are dense enough to sediment with the red blood cells when the product is used for treatment. This means that the patient may be exposed to these particles. Titanium Activated PRF (T-PRF), a more recent platelet concentrates, aids in regeneration as a 3rd generation platelet concentration.[5] This substance is used to dispel any rumours concerning silica and to prevent any negative consequences, either immediate or long-term, of dry glass or tubes made of plastic coated with glass. The distinguishing PRF's features, such as its higher biocompatibility, result from platelet activation with titanium as opposed to activation with silica particles.

Evolution of Platelet Concentrates

- Platelet Concentrates as fibrin glue in the 1970s[2]
- Autologous fibrin adhesive Tayapongsak 1994[3]
- Platelet-rich Plasma (PRP) by Whitman1997[4]
- Platelet Rich Fibrin (PRF) Choukron et al 2001[5]

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❑ Advanced Platelet Rich Fibrin (A-PRF) by Ghanaati in 2014[16]

❑ Albumin Platelet Rich Fibrin+ (Alb-PRF+) by Fujoka- kobayashi in 2020[7]

❑ Injectable Platelet Rich Fibrin (i-PRF) by Mourao in 2015[6]

❑ Titanium–platelet rich fibrin (T-PRF) Tunali and co-worker 2014[18]

❑ Titanium Prepared Platelet Rich Fibrin (T-PRF)

Titanium-prepared Platelet-rich fibrin (T-PRF); a third-generation platelet concentrate was developed by Tunali in 2013 to overcome the hazardous effects of silica in the glass tubes used for PRF preparation. Histomorphometric analysis of T-PRF showed more polymerized fibrin formation with a longer resorption rate in the tissues as titanium seems to be more effective in activating platelets than the silica activators in glass tubes.[5] This has been shown to increase the duration of release of growth factors with that of PRF, attributing to the thicker fibrin meshwork. T-PRF can be used in conjunction with bone grafts, which offers several advantages including promoting wound healing, bone growth and maturation, graft stabilization, hemostasis, and improving the handling properties of graft materials.

Figure 1: Method of preparation of T-PRF

Preparation (Figure 1)

a. By venipuncture of the antecubital vein, intravenous blood was drawn and placed in a 10 ml sterile titanium test tube without the use of an anticoagulant.

b. The tubes were immediately spun in a centrifuge for 12 minutes at 2800 rpm.

c. The composition of a structured fibrin clot in the middle of the tube, in between the red corpuscles at the bottom and the acellular plasma (Platelet Poor Plasma (PPP)) at the top, is made possible by centrifuging blood as soon as it is collected.

d. A stable fibrin membrane was obtained by squeezing serum out of the newly formed T-PRF clot.

e. After it had been separated using sterile tweezers and scissors and put onto the PRF box.[8]

Discussion

A second-generation platelet concentrate called Platelet Rich Fibrin (PRF) was created by Choukroun et al. in 2001. The Third-generation platelet concentrate is Activated Platelet Rich Fibrin with Titanium (T-PRF). Recent studies have shown that it aids in sinus lifting, periodontal regeneration, faster epithelization, improved wound healing, and tissue regeneration. It has been discovered that when compared to earlier generations of platelet concentration, it has greater biocompatibility, a thin fibrin meshwork, and a prolonged resorption period (Table 1). In an experiment, a T-PRF clot was created on flocculation at 200 rpm for 12 minutes. In clinical research on gingival recession conducted in 2018, Uzun BC, et al. discovered that T-PRF increased gingival thickness and keratinization.[9] In 2020, Valladao CA, et al. studied variations in verti-

cal faults in the maxilla and mandible, but they found no appreciable change.[12] Relative attachment level increases and Gingival Marginal Level alterations are caused by titanium-rich platelet fibrin, as demonstrated by Arabaci T. et al. in 2018.[10] The studies mentioned above demonstrate the value of titanium platelet rich fibrin in current dental procedures.

Table 1 - APPLICATIONS OF T-PRF

STUDY	AIM	METHODOLOGY	RESULT
Uzun BC, et al. Clinical trial: 2018[9]	To evaluate T- PRF effectiveness of treating gingival recession	Clinical periodontal indices, epithelial tissue, and other examinations were performed on patients with Class I/ II Gingivalrecession with abrasion defects. Prior to the actual surgical surgery, the gingival width and recession depth was measured, as well as at 1-6 and twelve monthsfor review.	T-PRF raised gingival thickness and KTW both by 93.29%; it is a secure and efficient procedure.
Arabaci T, et al. A randomized split mouth clinical study. 2018[10]	To evaluate how GCF biological markers and periodontal outcomes were changed by T-PRF combined with open flap debridement (OFD).	People with periodontal diseases were managed by autologous Ti-PRF+OFD or Open flap debridement alone was compared on clinical measures.	OFD+Ti-PRF sites had majorly larger mean PD decreases, relative attachment level gains, and GML changes than OFD sites. (p=0.033, p=0.029, p=0.026)
Chatterjee, et al. A randomized controlled study. 2017[11]	To compare the effects of titanium produced platelet-rich fibrin on infrabony deformities (Titanium derived PRF).	Each group (Test group=T-PRF) and Control group=PRF, was given ten sites at random. Clinical, radiographic, and histological parameters were evaluated.	Between baseline and nine months, both the groups showed a substantial reduced PPD and an increase in CAL in intragroup comparisons
Valladao CA, et al. A backward-looking clinical study. 2020[12]	To characterize the bone gain associated with Guided Bone Regeneration operations for vertical and horizontal bone augmentation that combines membranes, bone transplants, and PRF.	Study comprised individuals who required vertical or horizontal bone regeneration before receiving dental implants. CBCT measured bone gain at baseline and after 7.5 (± 1.0) months.	The maxilla gained more than mandible in horizontal deformities, and anterior portion gained more than posterior. There was no variation in vertical faults.
Ustaoglu G, et al. RCT:2020[13]	Toseehow T-PRF compared into Open Flap Debridement (OFD) and GT Rin Intra Bony Defects (IBDs) (GTR).	The Depth of Probing (PD), Clinical Attachment Level (CAL), and Inflammatory Bowel Disease (IBD) were all observed. The radiographic depth of IBD was also measured	T-PRF was as successful as GTR for therapeutics outcomes of IBDs with endo-periolesion

Conclusion

In recent years, L-PRF has become more important and has been used in a number of clinical trials. The T-PRF is described as a robust autogenously produced fibrin product that is rich in platelets and leukocytes. Although more in vitro and in vivo studies are required to pinpoint its exact significance. T- PRF has been shown to be successful in promoting sinus lifting, periodontal regeneration, wound healing, and soft tissue regeneration surrounding implants. We anticipate that T-PRF will be used more frequently, especially when compared to the more expensive titanium tube preparation done in the past. Compared to glass tubes, its adoption might be constrained. T-Platelet Rich Fibrin metrics, such as resorption duration and clinical success, were required because the product is undoubtedly an intriguing experimental one that needs more clinical trials.

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9TH SPIK PERIODONTOLOGY SCHOLARSHIP EXAMINATION 2022

The Society of Periodontists and Implantologists of Kerala in association with Amrita School of Dentistry, Kochi, organized and conducted the 9th SPIK Periodontology Scholarship Examination on October 22, 2022. Dr Sameera G Nath, Scientific Program Convener- SPIK, Dr Rajesh Vyloppillil, Professor and Head, Department of Periodontics, Amrita school of Dentistry - Exam Convener SPIK along with Dr Presanthila Janam- President SPIK, Dr Mohammed Feroz T P- Secretary SPIK, worked towards the success of the program. They were assisted by faculty members of Department of Periodontics, Amrita School of Dentistry- Dr Biju Balakrishnan, Dr Maya Rajan Peter, Dr Reshma Suresh, Dr Swetha V R, Dr Anjali R Nath, Dr Sreelekshmi M R, Dr Pallavi Menon, Dr Prajula Mithwin, and Dr Shilpa Ramachandran. There were twenty participants from twenty different dental colleges of the state of Kerala and Mahe, who were selected as the best student in Periodontics in their respective colleges. The inaugural function commenced at 9:00 am with the Vice Principal, Amrita School of Dentistry Dr Rakesh S, inaugurating the event along with the esteemed panel of examiners Dr K R Biniraj, Dr Vivek Narayan, Dr Deepak Thomas and Dr Rajesh Vyloppillil. The examination began at 9.30 am. The examination process comprised of spotters, MCQs, short essay and clinical case scenarios at the lecture hall followed by grand viva in the mini auditorium with the individual examiners. The exam concluded at 2:00pm. The participants, examiners, and the organizing faculty were provided refreshments during the course of the day. At the completion of the rigorous evaluation process, three students emerged as the winners. Haritha K R of Royal Dental College secured the first place, Sankar Mahadev S B of Government Dental College Thiruvananthapuram won the second place and Aiswarya K N of Sree Sankara Dental College won the third place. The results were announced and all the participants were given certificate of participation during the valedictory function.

SPIK ESSAY COMPETITION 2022

Society of Periodontologists & Implantologists of Kerala organized an essay competition for life members and post graduate students in the month of October 2022. The competition was designed to foster the interest of the next generation of dental professionals. A total of twenty two entries were received and was evaluated by faculties from KMCT Dental College - Dr Navia George, Dr Aparna T K and Dr Subisha S B.

WINNERS OF SPIK ESSAY COMPETITION 2022		
LIFE MEMBER CATEGORY	**Dr Subair K,** Professor, Mahe Institute of Dental Sciences & Hospital	
POST GRADUATE STUDENT CATEGORY	1st Prize	**Devika Gopakumar,** PMS College of Dental Science and Research, Thiruvananthapuram
	2nd Prize	**Beena Vijayan Parvathy,** Azeezia Dental College, Kollam
	3rd Prize	**Anjali Babu,** Kannur Dental College, Kannur

SPIK MIDTERM CONFERENCE 2022

The Midterm Conference of SPIK 2022 was hosted by Department of Periodontology, PMS College of Dental Science and Research, Thiruvananthapuram on the 10th & 11th December 2022. The programme started with the inaugural function at 10.30 am with collaring of the President by the Secretary followed by invocation.

The organizing chairman - Dr Arun Sadasivan welcomed the gathering followed by presidential address by Dr Presanthila Janam- President SPIK. The chief guest of the program, Director of Medical Education, Dr Thomas Mathew inaugurated the conference by lighting the lamp. Dr P.S. Thaha, Chairman, PMS College of Dental Science and Research, Principal Dr Rajesh Pillai and Secretary-SPIK Dr Mohammed Feroz T.P addressed the gathering.

The second (July) issue of Journal of Society of Periodontists and Implantologists of Kerala (JSPIK) 2022 was released by Dr Thomas Mathew by presenting the journal to Dr P.S. Thaha in presence of Editor JSPIK -Dr Shahana C Mohamed. Dr. P.S. Thaha, a founder member of SPIK was felicitated for his lifetime contributions to society and to the specialty. Dr. Ambili R. was also felicitated for achieving the 'Best Teacher Award' from the Kerala University of Health Sciences. Prize distribution of essay competitions conducted by SPIK was done by the Chief Guest. The vote of thanks was proposed by organizing secretary Dr.Ambili R. The executive committee meeting of SPIK was conducted after the scientific program on the first day.

The scientific session was impressive with the following topics by the renowned speakers :

Topics	Name of the Speaker
Current trends and future directions in non-surgical periodontal therapy	Dr Biju Thomas
Principles and practice of guided bone regeneration	Dr Baiju R.M
Role of occlusion in periodontal and implant dentistry	Dr Kaarthikeyan
Optimizing soft tissue around teeth and Implants	Dr K. Harikumar Menon
Periodontal considerations in implant dentistry	Dr Kaarthikeyan

The conference was well attended by 126 participants including teaching faculty, practitioners, and postgraduate students from fifteen dental colleges in and around Kerala. Eighty scientific presentations were made by postgraduate students and life members of society. The invited speakers were Dr Kaarthikeyan, Dr Biju Thomas, Dr K. Harikumar Menon and Dr Baiju R.M, who shared their in-depth knowledge in their respective topics. The first day ended with a gala dinner at Hotel SP grand days, Thiruvananthapuram.

A quiz program was conducted by Dr. Manikandan G R for the postgraduate students on the second day. The conference concluded on the second day with the valedictory function where the certificates for winners of the paper presentation and quiz competition were distributed. The conference was well attended by the delegates in terms of scientific content, hospitality, infrastructure facilities of the college and entertainment.

Inaugural function

Felicitating Dr P.S Thaha

Release of JSPIK

Felicitating Dr Ambili R

Organizing Committee

Dr Biju Thomas

Dr Baiju R M

Dr Kaarthikeyan

Dr K. Harikumar Menon

Quiz program by Dr Manikandan G R