Journal of the Society of Periodontists and Implantologists of Kerala

Index Copernicus ID 6818
ISSN 2231- 1823

JSPIK

Volume 15 • Issue 1 • MARCH 2023

www.spik.in

SOCIETY OF PERIODONTISTS AND IMPLANTOLOGISTS OF KERALA

Vol. 15 | Issue 1 | March 2023

Index Copernicus ID 6818

ISSN 2231-1823

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President's message

Warm greetings to all!

We are glad that the third issue of our journal is being released. I would like to congratulate and thank our Editor, Dr Shahana C Mohamed, who is successfully bringing out the third issue of the journal and the contributors /authors for sharing their scientific research. We should encourage research work conducted by post graduate students and PhD scholars in our speciality.

We are all awaiting the gala event of the year,the 15th SPIK Annual conference at Kannur, "Land of looms &lore" on 29th and 30th April 2023. We have renowned faculties like Dr Reshmi Hegde, Dr Bini Raj, Dr V C Santhosh and Dr C K Ashokan as speakers for the event. In addition, we have a point-counterpoint session led by our pandits Dr Anil Melath, Dr Arun Narayanan, Dr Jose Paul, Dr Harish Kumar, Dr Baiju RM and Dr Harikumar K. We also have a cricket match by teams Malabar Perio Kings, Central Kerala Perio Giants and Travancore Perio Titans co-ordinated by Dr Deepak Thomas.

I am sure that this annual meet of Periodontists will prove exceptional in terms of quality and participation. Active involvement and whole hearted co-operation are requested from all the members.

It was indeed an honour and pride to serve you as the President of the Society of Periodontists and Implantologists of Kerala. As my term ends,the time has come to pass the baton to the upcoming President Dr Jose Paul. I congratulate and give all my best wishes and prayers to the new team which takes over from May 2023.

I thank all the past presidents and past secretaries of SPIK and our young dynamic secretary Dr Mohammed Feroz T P, who was supporting SPIK in all the activities. Wishing the entire team of SPIK and each one of you, the very best in future.

Dr. Presanthila Janam
President, SPIK

Secretary's Message

Warm greetings to all !

At the outset, let me extend my sincere thanks to our respected President Dr Presanthila Janam for guiding us in making last SPIK year filled with scientific programs, health awareness programs, scholarship examination for undergraduate students, mid-term conference and annual conference as per schedule.

I am happy to inform that Dr Shahana C Mohamed, our Editor is working hard in a process of indexing JSPIK, our official journal very soon.

Scientific programs and interactive sessions are integral part in updating knowledge at all times. Dr Sameera G Nath, Scientific Programme Convenor charted out many programs for the benefit of the members and post graduate students in the field of Periodontology.

Dr Manikandan GR, Periodontal Health Care Convenor conducted periodontal awareness programs throughout last year.

I would like to thank the organizing committee of SPIK scholarship examination and SPIK Midterm conference in organizing both the events successfully.

I would like to thank all the SPIK members for the wonderful participation and cooperation extended for the conduct of programs during the year 2022-23.

Warm wishes to the COC of 15th SPIK Annual Conference hosted by Kannur Dental College at Kannur in the month of April 2023.

Dr Mohammed Feroz T P
Secretary, SPIK

Editorial

Warm greetings to all!

Let me humbly mention that I am proud to be the editor of the journal of our esteemed organization SPIK. When I took over as editor in 2022, I had a great responsibility, as my predecessors have done an excellent job in streamlining our journal to this level.

As you all know, a high-quality journal is an asset for any organization. It is high time that we take fruitful steps to take our journal to the next level. Indexing the journal is an arduous and systematic process that can only be achieved together, and I humbly ask all SPIKians to work together towards this goal.

I thank our outgoing President and my teacher Dr. Presanthila Janam, our secretary Dr. Mohammed Feroz, and all executive committee members for their unwavering support that they have all provided during this period. I hope that during the tenure of our new president, we will be able to take further steps to advance our indexing process and achieve our goal.

My humble request to all our members: continue to support JSPIK with your scientific contributions.

Dr Shahana C Mohamed
Editor, JSPIK

Is periodontal phenotype clinically significant?

Neenu Krishnan P[1], Ashok Raj VM[2], Rosamma Joseph Vadakkekkutical[3]

ABSTRACT

In 2017 the World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, periodontal biotype has been categorised as "thin scalloped", "thick scalloped" and "thick flat". Periodontal biotype comprises two terms, gingival phenotype (gingival thickness and keratinized tissue width) and bone morphotype (buccal bone plate thickness). Periodontal biotype can be assessed through clinical or radiographic assessments using invasive/non-invasive (for gingival thickness), static/functional (for keratinized tissue width) methods.

Keywords: Gingival phenotype, Periodontal biotype, Phenotype modification therapy, TRAN (transparency of probe) method

Introduction

Over the past years, the concept of assessment of gingival phenotype that can influence the final esthetic result of the treatment have utmost importance in the decision-making process for periodontal restorative, prosthetic, orthodontic, and implant treatment. The "biotype" has been labelled by different authors as "gingival" or "periodontal" "biotype", "morphotype" or "phenotype". Gingival biotype is constituted by gingival thickness and keratinized tissue width and periodontal phenotype is the term used to describe the combination of gingival phenotype (three-dimensional gingival volume), the thickness of the buccal bone plate (bone morphotype) and tooth dimension. The phenotype indicates a dimension that may change through time depending upon environmental factors and clinical intervention and can be site-specific (phenotype can be modified, not the genotype). Both gingival and bone thickness may affect the outcome of therapeutic modalities. Hence, the evaluation of gingival phenotype is one of the most important factors in esthetic outcome prediction and decision-making before periodontal plastic surgery techniques. In the 2017 World Workshop on mucogingival defects in natural dentition, they adopted the term 'periodontal biotype', although they replaced the term periodontal biotype with periodontal phenotype in the 2017 World Workshop on a new classification scheme for periodontal and peri-implant diseases and conditions.

Recently, the World Workshop 2017 recommended evaluating gingival phenotype in a standardized and reproducible way by the transparency of periodontal probe. It is assumed that the probe will be visible when gingival thickness (GT) is thin {≤1 millimetre (mm)} and not visible in a thick GT (>1 mm)(Figures1,2). In World Workshop 2017, one report categorized the periodontal biotype as "thin-scalloped," "thick-scalloped," and "thick-flat" patterns.

[1,2]Postgraduate student, [3] Professor and Head, Department of Periodontics, Government Dental College, Kozhikode, Kerala, India. Corresponding Author: Dr. Rosamma Joseph Vadakkekkutical, E-mail: drrosammajoseph@gmail.com

Although several methods for assessing phenotype were proposed, there is scarce knowledge regarding the evaluation of gingival phenotype in the clinical scenario. Adequate knowledge about confusing facts and different methods of evaluation among dental practitioners aids in the diagnosis and treatment planning of periodontal therapy. The present paper has reviewed historical and current phenotype classifications, methods for evaluation, and clinical significance reported in the literature.

Historical Overview

The anatomical characteristics of periodontium have been studied for more than one century. Hirshfield was one of the pioneers in this field and studied dry human skulls of the American Museum of Natural history and described periodontal breakdown and findings related to differences in alveolar bone.[1] Several investigations were carried out regarding soft tissue and gingival marginal position and certain empirical correlations were identified to predict changes in the position of the free gingival margin. In the 1960s, Ochsenbein and Ross reported the association between gingival anatomy and underlying bone and described the "pronounced scalloped" and "flat" gingival anatomical characteristics.

Weisgold in 1977 reported thin scalloped and thick flat gingival architecture. Claffey and Shanely investigated the relationship between buccolingual gingival thickness and bleeding on probing to loss of probing attachment level following non-surgical therapy.[2] These authors assessed the gingival thickness (thin or thick) using the trans gingival probing method with the use of a stainless-steel wire.

In 1989, Seibert and Lindhe classified the periodontium into thin scalloped and thick flat biotypes and they coined the term 'periodontal biotype'. The thin scalloped biotype was associated with narrow zones of keratinised gingiva and slender teeth, whereas the thick flat biotype was associated with a wide zone of keratinised gingiva and quadratic teeth.

Olsson et al investigated the relationship between crown form and gingival characteristics and found that long-narrow teeth showed a thin biotype, length twice their width, gingival margin more apical (about 1 mm), and more recession compared to short-wide teeth.[3] In 1997, Becker evaluated the relationship of alveolar bone morphology with tooth form and proposed three bone morphological types (flat -2mm, scalloped -3 mm, and pronounced scalloped - 4 mm) considering the measurement from the interdental bone.[4]

Muller and Eger reported three gingival phenotypes named A (normal), B (thick), and C (thin) through cluster analysis.[5] These authors considered GT (gingival thickness), gingival width, and the crown width/crown length ratio and emphasized the existence of three different gingival phenotypes in relation to maxillary anterior teeth. A simple visual method for gingival thickness evaluation based on the transparency of the probe (TRAN) was proposed.[6] Later, three-dimensional imaging like cone-beam computed tomography (CBCT) especially soft tissue CBCT (ST-CBCT) was included for clear visualization and measurement of periodontal structures and dentogingival attachment apparatus.[7] Hence, newer classifications were evolved over the time which mirror the current status in identification of phenotype.

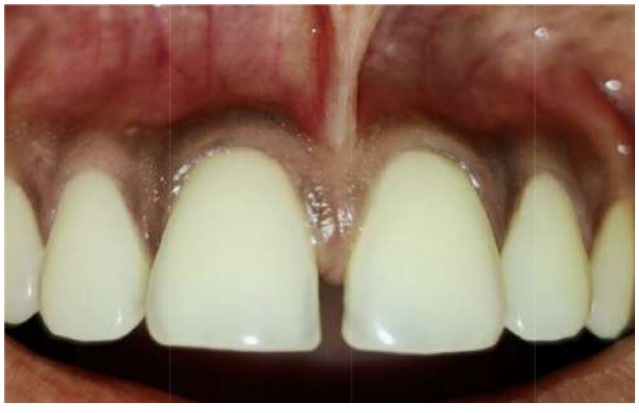

Figure 1: Thin gingival phenotype

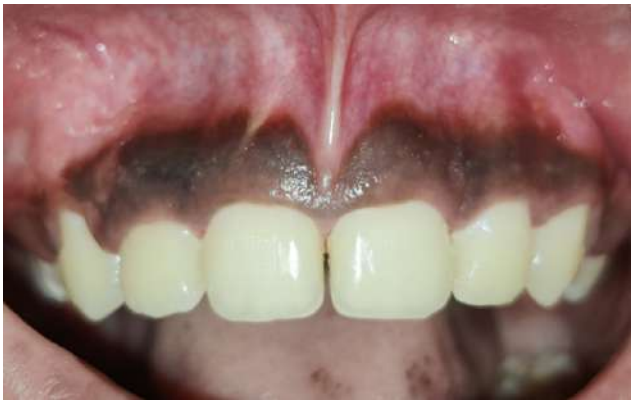

Figure 2: Thick gingival phenotype

Classification

Recently, the 2017 World Workshop on the classification of periodontal and peri–implant diseases and conditions where information about periodontal manifestations of systemic diseases and developmental and acquired conditions highlighted the differences between biotype and phenotype.[11] Biotype is genetically predetermined and cannot be modified by environ-

Table 1: Various classifications for periodontal phenotype

Authors	Classification
Kan *et al.* (2010)[13]	Gingival biotype. "Thin": Delicate, friable, and almost translucent. Visibility of the periodontal probe through the gingival tissue.GT ≤1.0 mm. "Thick": Dense and fibrotic. The periodontal probe is not visible. GT: > 1.0 mm.
Zweers *et al.* (2014)[8]	Periodontal biotype: "Thin scalloped": Clear thin delicate gingiva, narrow zone of Keratinised Tissue (KT), slender triangular-shaped crowns, subtle cervical convexity, interproximal contacts close to the incisal edge, and a relatively thin alveolar bone. "Thick scalloped": Clear thick fibrotic gingiva, high gingival scallop, the narrow zone of KT, and slender teeth. "Thick flat": Clear thick fibrotic gingiva, a broad zone of KT, more square-shaped tooth crowns, pronounced cervical convexity, large interproximal contact located more apically, and a comparatively thick alveolar bone.
Fischer *et al.* (2018)[9]	Gingival biotype: Double-ended periodontal probe. "Thick": The thick probe ending is not detectable through the sulcus. "Moderate": The thick probe ending is visible through the sulcus but the other thin ending is not visible. "Thin": The thin probe is noticeable
Kloukos *et al.* (2018)[10]	Gingival phenotype. Visibility of a periodontal probe with a colored tip (white, green, and blue) through the gingiva. "Thin": White tip visible. "Medium": The white tip is not visible, but the green tip is. "Thick": The green tip is not visible, but the blue tip is. "Very thick": Not even the blue tip is visible.
Cortellini and Bissada (2018)[12]	Periodontal biotype. "Thin-scalloped", "Thick scalloped", and "Thick-flat".
Jepsen *et al.* (2018)[11]	Periodontal phenotype. "Gingival phenotype": Gingival thickness, keratinized tissue width. "Bone morphotype": Buccal bone plate thickness. "Thin": GT ≤1 mm. Probe has been seen through the gingiva. "Thick": GT > 1 mm. Probe not seen.

mental factors whereas phenotype is the appearance of an organ based on a multifactorial combination of genetic traits and environmental factors (its expression includes the biotype). Consequently, periodontal phenotype can be modified by various clinical interventions and environmental factors.

Methods for evaluation of gingival phenotype

Periodontal phenotype can be evaluated in terms of the gingival phenotype (GT and keratinised tissue width (KTW) and the bone morphotype. The identified methods for periodontal phenotype evaluation can be broadly divided into clinical autopsy, clinical or radiographic assessment, and direct vision/measurement assessments.

1. Clinical autopsy

A clinical autopsy has been carried out in dry human skulls[1,4] and extracted teeth and this method was performed in pioneer studies in the evaluation of gingival biotype.

2. Clinical or radiographic assessment

i. Ultrasonic device

This method utilizes ultrasound wave distribution, dispersion, and reflection on an interface. Ultrasonic devices based on the pulse-echo principle were used in important investigations about gingival phenotype.[10,13,5]

ii. Calipers

Calipers have been used with the acrylic template as a guide for measuring changes in pre and postoperative free gingival grafts[17], as well as to measure mucogingival flap reflection at the time of guided tissue regeneration surgery. [18]Boley gauge caliper[19] and Iwanson caliper[13] are the most used calipers. One arm is pushed underneath the gingival sulcus and another arm is over the tissues.[20]

iii. Periodontal probe transparency

GT can be classified as thin and thick based on visibility of the periodontal probe through the gingival margin. GT was categorized as thin if the periodontal probe was visible through the gingival margin and thick if it was not visible.[2,21]

Recently, a double-ended periodontal probe[9] and a periodontal probe with a colored tip (white, green, and blue)[10] were designed to analyze the GT using the TRAN method. The 2017 World Workshop recommended assessing the periodontal phenotype using the TRAN method. The probe will be visible in thin periodontal phenotypes (≤1 mm) and will not be visible in thick periodontal phenotypes (>1 mm). According to the evidence, the TRAN method is the most frequently used. Also, a highly reliable and reproducible method to analyze periodontal phenotypes.[11]

iv. Parallel profile radiographs

Parallel profile radiographs can be carried out for measuring the dentogingival unit on the buccal surfaces of anterior teeth using the long cone parallel technique. In this method, a gutta-percha point cut to the known sulcus depth was inserted into the base of the sulcus; the apical end of the point marks the bottom of the sulcus, and the coronal end marks the gingival margin. Two radiographs were made, one in a frontal projection, and the second in a lateral position using a periapical film holding system. Occlusal films and plastic lip retractors are variants of this radiographic morphometric technique or tangential radiography.[22]

v. Soft tissue – Cone beam computed tomography

The ST-CBCT was proposed to measure dentogingival unit dimensions[23], palatal mucosa thickness[24], crown length[25], and maxillary and mandibular buccal bone plate thickness[25,26] using the cone beam technique.

vi. Transgingival probing

Transgingival probing is used for gingival thickness evaluation. The technique consists of the use of local anesthesia and a periodontal probe positioned perpendicularly to pierce the soft tissue surface until reaching the resistance of the bone. Previously, stainless-steel wire[2], and a disposable sterile syringe needle were used. Subsequently, endodontic reamer, spreader, files, or disposable acupuncture needles were also used. A rubber stopper in contact with the tissue is necessary to assess the GT measurement. A study cast with an acrylic transparent splint was recommended for measuring palatal mucosa thickness.[14,15]

vii. Transformer probe

This method was proposed for measuring GT based on a differential transformer coupled to an os-

cillator and digital voltmeter. The information about this method is scarce.[15,16]

viii. Visual assessment

Visual assessment of the KTW can be performed through clinical appearance evaluation and histochemical staining. The clinical appearance is based on the distance measured mid-buccally from the free gingival margin to the alveolar mucosa. Caliper instrument or periodontal probes[6,27] have been used for the measurement. KTW can be assessed visually after histochemical staining of the mucogingival complex with Schiller 15 or Lugol iodine solution (iodine test). This method is based on the difference in the glycogen content between the keratinized tissue (low glycogen content) and the alveolar mucosa (high glycogen content).

ix. Pushing technique (wrinkle technique)

This technique can be performed using a periodontal probe to assess the mobility of the mucogingival line.[28] The probe is positioned horizontally and moved from the vestibule toward the gingival margin using light pressure.[29]

3. Direct vision/measurement

Dense and fibrotic gingival appearance has been described as thick; whereas friable, delicate, and translucent gingiva is thin.

Clinical Significance

The gingival biotype has substantial importance in maintaining periodontal health. Hence, the evaluation of gingival phenotype has a fundamental importance in the decision-making and predictability of esthetic outcomes in therapeutic modalities in the periodontal scenario.

The thick biotype has a dense and fibrotic wide zone of attachment. According to the literature, the thick biotype possesses resistance to trauma and recession, superior soft tissue handling, promotion of creeping attachment, reduction in clinical inflammation, and high predictability of the surgical outcome. A thin gingival biotype is a delicate, highly scalloped soft tissue and is more prone to recession. The importance of gingival phenotype modification therapy (PMT) has been emerging. GT and KT play a role in obtaining complete root coverage.[30,31] Connective tissue graft (CTG) is the treatment of choice in terms of providing the greatest probability of achieving complete root coverage, increasing KT, better esthetics, and maintaining root coverage outcomes over time in phenotype modification.[32] Acellular dermal matrix (ADM) was also found to be significantly associated with a greater KT gain.[33] For phenotype modification on non-root coverage procedures, while treatment with apically positioned flap (APF) alone shows a significant KT increase compared to untreated sites, the utilization of a graft material (i.e., ADM, collagen matrix, Free Gingival Graft, or Living Cellular Constructs) significantly enhanced the outcomes in terms of KT gain compared to non-grafted APF sites.

Coronal regrowth of the soft tissue margin was significantly noticed in thick biotypes compared to thin tissue biotypes in surgical crown lengthening techniques. Literature shows gingival recession may develop during orthodontic treatment in patients with thin periodontal biotypes. So, sites with less than 2mm attached gingiva should be recommended for gingival augmentation before initiating orthodontic therapy.[34]

Kois described greater thickness of peri-implant mucosa was associated with a thick gingival phenotype compared with a thin biotype. The thick biotype was significantly associated with the presence of the gingival papilla in immediate implants while the thin biotype showed more recession. Peri-implant tissue biotype is a clinical parameter that affects both the esthetic and functional aspects of implant rehabilitation. The modification of mucosal phenotype through soft tissue grafting procedures results in more predictable surgical and prosthetic outcomes. Therefore, these parameters should be evaluated before the development of a comprehensive treatment plan.

Conclusion

Recent classification systems highlighted the "thin scalloped", "thick scalloped" and "thick flat" gingival phenotype. Several invasive and non-invasive method has been proposed to evaluate these factors. By recognizing the nature of these parameters, periodontal biotypes should be taken into account in prompt diagnosis and treatment planning in dental procedures.

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Retrograde Peri Implantitis- What, Why, How?

Keerthana Varma KC[1], Anu John[2], Baiju RM[3], Santhosh Kumar S[4]

ABSTRACT

Biological complications involving dental implants include peri-implant diseases such as peri-implant mucositis and peri-implantitis. Retrograde periimplantitis (periapical implant lesion; implant periapical pathology, apical periimplantitis) was first described in 1992 by McAllister et al. as an infectious-inflammatory process in the tissues surrounding the implant apex. Retrograde periimplantitis (RPI) is a rapid infective process, and it can potentially cause devitalization of adjacent teeth and can greatly reduce bone for future implants if not treated early. Many possible etiological factors have been associated with the periapical implant pathology and most common out of it is associated with infection from adjacent teeth. Any peri-implant radiolucency should be addressed immediately to prevent further loss of osseointegration. To date, there is no consensus for the treatment of RPI; therefore, the treatment is empiric. The objectives of management include treatment of the infection followed by restoration of peri apical health through connective tissue regeneration and re osseointegration. This article tries to review the current evidence related to retrograde peri implantitis and its management.

Keywords: Periimplantitis, Periimplant diseases, Retrograde Periimplantitis

Introduction

Over the last seven to eight decades, dental implant therapy has become a predictable technique to replace missing teeth. High-quality data has been presented by a number of studies to support implant therapy's successful long-term survival and success rate in general populations.[1] Dental implants have a high success rate, however there is a chance of developing infectious complications. Peri-implantitis has been defined as an inflammatory lesion of the mucosa surrounding an endosseous implant and with progressive loss of supporting peri-implant bone.[2] It is frequently accompanied by increased peri implant pocket-probing depth, mucosal recession, and/or suppuration, bleeding at the peri implant margin after the insertion of a periodontal probe into the peri implant space. Retrograde (or apical) periimplantitis and marginal periimplantitis are two different types of infectious dental implant failures. Periimplantitis was reported to occur in 18.8% of subjects and 9.6% of implants.[3] Periimplantitis will worsen and ultimately lead to the loss of the implant if it is not identified and treated.

Retrograde periimplantitis (periapical implant lesion; implant periapical pathology, apical periimplantitis) was first described in 1992 by McAllister et al. as an infectious-inflammatory process in the tissues surrounding the implant apex.[4] In 1993, Sussman and Moss defined it as "localized osteomyelitis secondary due to endodontic pathology". In 1995, Reiser and Nevins described it as "active implant periapical lesion". Piattelli et al., in 1998, histologically examined an implant that was removed due to periapical radiolu-

[1,2] Postgraduate student, [3]Additional Professor, [4]Professor and Head, Department of Periodontics, Government Dental College, Kottayam, Kerala, India. Corresponding Author: Dr Baiju RM, E-mail: baijurm2@gmail.com

cency. They discovered the presence of necrotic bone inside the anti-rotational hole and the demineralization of the bordered trabecular bone. Esposito et al. in 1998 considered the placement of an implant in a previously infected site to be an important factor contributing to implant failure. The pathology develops shortly after implant insertion, while the coronal portion of the implant achieves a normal osseointegration.[4] Of the 539 implants evaluated in a retrospective study, this pathology was found to occur in 1.6% of the implants in the upper jaw and 2.7% in the lower jaw.[5] Retrograde periimplantitis (RPI) is an infection that spreads quickly. If left untreated, it has the potential to destroy nearby teeth and significantly diminish the amount of bone available for future implants. The reported prevalence of RPI is very low (0.26%), but it can be increased up to 7.8% when there is a history of a root canal treatment of an adjacent tooth, next to the implant site.[6]

Etiology

The periapical implant disease has been linked to a wide range of potential etiological variables. Etiological factors can be split into two categories: those that arise during implant placement and those that are brought on by an underlying medical condition. Additionally, an etiological component for RPI was revealed as a human immunodeficiency virus (HIV)-related infection. The various etiological factors are a) residual infection at the site of extraction/tooth removal due to the periapical pathology b) infection from adjacent teeth with periapical pathology and/or incomplete endodontic treatment c) periodontal pathology d) impaired bone healing e) bone overheating f) retained root tip g) excessive tightening during insertion g) incomplete implant depth h) contaminated implant tip.

Nevertheless, it appears from the case reports examined that retrograde periimplantitis is most frequently linked to infection from nearby teeth with periapical pathology and/or incomplete endodontic treatment, as well as to residual infection at the site of tooth extraction when the tooth was extracted due to periapical pathology.

Classification

The periapical implant lesion has historically been divided into two forms and categorised as an endodontic implant pathology. Type 1 is an implant to tooth lesion that occurs when the insertion of the implant results in devitalization of the adjacent tooth either by direct contact or overheating of the surrounding bone, whereas type 2 is a tooth to implant lesion, which occurs when a periapical lesion from a nearby endodontically involved tooth encroaches upon the implant and contaminates it.[7] However, since Sussman introduced the original categorization in 1998, further causes of RPI have been identified, leading to an updated classification (Figure 1) of RPI that may help with selecting the proper treatment. Class 1 and Class 2 will continue to be classified in accordance with Sussman's current system; however, Class 3 can be described as a periapical implant lesion that develops because of improper placement or angulation of the implant (i.e., placed outside the envelope of bone). This can include implants that are placed too far labially or lingually/palatally. Class 4 can be described as a periapical implant lesion that develops despite proper placement in sound bone with adjacent vital teeth postoperatively, which may imply residual bacteria/viruses and/ or necrotic bone/subclinical infection remaining at the site or placement into an infected or inflamed sinus causing either nonhealing of the apical region of the implant or contamination.[8]

Symptoms

Studies show that symptoms might start to appear as early as one week following implant placement and up to four years.[9] The symptoms might range from discomfort and swelling to the presence of a fistulous tract. Pain, soreness, swelling, and/or a fistulous tract are frequently seen in infected lesions. These lesions begin at the implant apex and can progress coronally, proximally, and facially. However the presence of fistulous tract has been identified as the symptom with highest prevalence of 65.6% and maxillary implant sites (78%) seem to be more exposed to retrograde periimplantitis compared to mandibular.[9] Reiser and Nevins connected that finding to the higher frequency of radicular cysts in the maxilla.[10] According to a study conducted by Burdurlu et.al.in 2021, fourteen (3.8%) implants had RPI; of these, 10 (5.8%) were in the maxilla and four (2.0%) were in the mandible.[11]

Diagnosis

The standard diagnostic indicator for an implant

periapical lesion is a periapical radiograph, although it is unable to differentiate between inactive and infected lesions. Reiser et al. classified implant periapical lesions as noninfected - they are diagnosed radiographically but do not present any clinical symptoms. Unless the size of the periapical radiolucency increases, these lesions doesn't need to be treated. Typically, infected lesions come with pain, soreness, swelling, and/or a fistulous tract. Always take a detailed history of the reason for extraction of the tooth prior to implant placement and try to review the previous records including radiographs and clinical photographs if available. The endodontic status of the adjacent teeth are to be assessed. Tracking a fistulous tract using a gutta percha point with the help of an Intra-Oral Periapical Radiograph (IOPA) might be conclusive.

Difference between Retrograde Periimplantitis and Marginal Periimplantitis

According to some research, retrograde and marginal periimplantitis are both site-specific infectious conditions.[12] The main differences, however, relate to the microbial composition, rate of proliferation, and pathway of infection. Contrary to retrograde periimplantitis, which is caused by endodontic pathogens, periimplantitis microbes are closely related to periodontal pathogens. Furthermore, retrograde periimplantitis is initiated apically whereas periimplantitis occurs coronally. Routine probing helps in detecting marginal periimplantitis, whereas retrograde periimplantitis relies on patient complaint and radiographic assessment.[12]

Management

Any peri-implant radiolucency should be addressed immediately to prevent further loss of osseointegration. Bacteria associated with failing implants due to infection are similar to those found in chronic periodontitis cases. Consequently, the dissolution of the biofilm is a must for effective treatment.[13] Reiser and Nevins suggested a classification system for implant periapical lesions differentiating them as either "infected" or "inactive". The authors suggested a surgical intervention for the infected type and monitoring for the inactive lesion.[10]

To date, there is no consensus for the treatment of RPI; therefore, the treatment is empiric.

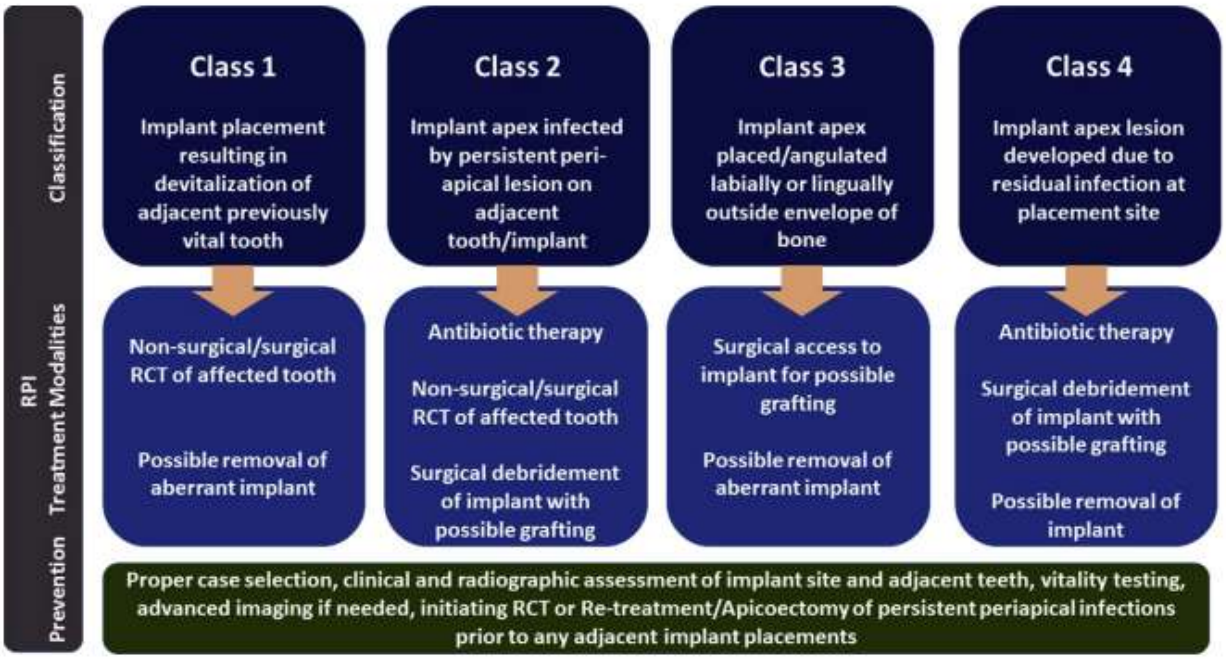


Figure 1 Classification and clinical management of retrograde peri-implantitis associated with apical periodontitis

Figure 1: Adapted from : Sarmast ND, Wang HH, Sajadi AS, Angelov N, Dorn SO. Classification and clinical management of retrograde peri-implantitis associated with apical periodontitis: a proposed classification system and case report. Journal of Endodontics. 2017 Nov 1;43(11):1921-4.

The objectives of management include treatment of the infection followed by restoration of peri apical health through connective tissue regeneration and re-osseointegration. According to published evidence the therapeutic strategies employed include antimicrobial therapy, non-surgical mechanical therapy, lasers and surgical therapy with or without the use of bone grafts and membranes. Romanos et al., based on a systematic evaluation of clinical case reports, showed that the use of antimicrobials only was not successful in any case for the treatment of implant periapical lesions.[14] When compared to alternative treatment techniques for removing the plaque biofilm, the application of air-abrasive powders appears to be an effective method for decontaminating implant surfaces. According to a study by John et al. the use of an air-abrasive device for the treatment of peri-implantitis compared to mechanical debridement showed significantly better results in BOP reduction after 12 months.[15] Nevertheless, it has an increased risk for emphysema.

Quirynen et al. suggested that implant apicoectomy is not required for the treatment of retrograde periimplantitis.[5] The use of augmentation materials is required to induce complete regeneration of bone around peri-implant defects. Following implant decontamination, the concurrent use of Guided bone regeneration (GBR) offers stabilisation of blood clot and space maintenance.

Different types of lasers are available in surgical dentistry, in various wavelengths, such as carbon dioxide (CO_2); diode (810–980 nanometre); neodymium-doped: yttrium, aluminum, and garnet (Nd:YAG); erbium-doped: yttrium, aluminum, and garnet (Er:YAG); and erbium, chromium-doped: yt-

Clinical photographs of management of retrograde periimplantitis

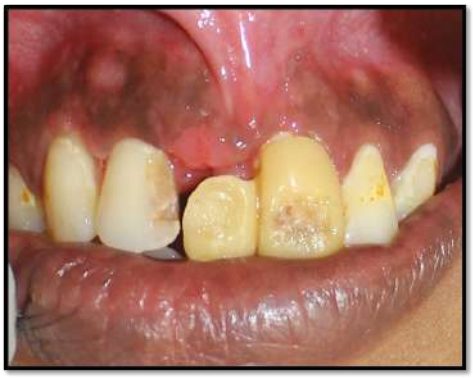


Figure 2a: Patient presented with a swelling in relation to 12 after five months of implant placement i.r.t 11

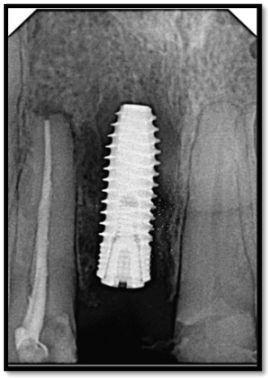
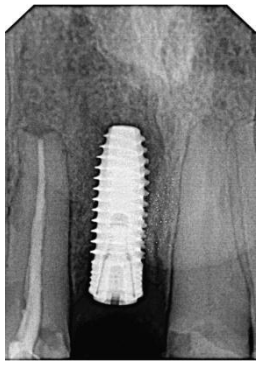


Figure 2b: An endodontic lesion was noted at 12 and peri implant radiolucency noted in relation to 11

Figure 2c: Extensive bone loss and thread exposure following full thickness mucoperiosteal flap elevation



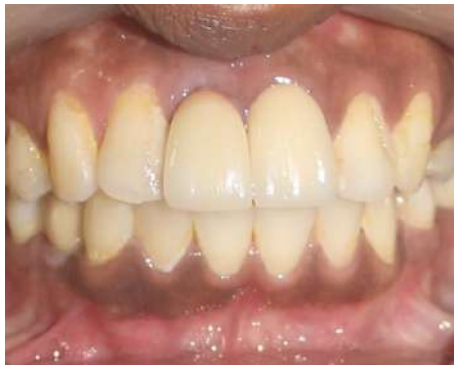


Figure 2d & 2e: Regeneration was attempted and successful periimplant bone fill was obtained in relation to 11 after six months after therapy

trium, scandium, gallium, and garnet (Er, Cr:YSGG). It is essential to exercise caution when applying them so as not to overheat the implant and damage its surface integrity. Er, Cr:YSGG laser ablates tissue through a hydrokinetic process and can be used with radially firing periodontal tip and energy settings up to 2.5 Watt. It does not raise the temperature to dangerously high levels that could harm implant surfaces. Furthermore, in comparison with plastic curettes and chlorhexidine, it effectively eliminates the plaque biofilm over roughened surfaces.[16]

Treatment recommendation according to the systematic review by Ramanauskaite et al. is to use the regenerative treatment approach. They suggest using bone substitute to fill the bone defect that would act as a scaffold for new bone cells to growth into the bony defect, maintain space, and therefore prevent deformities of the soft tissues.[9]

Conclusion

Thorough clinical assessment of the patient and implant site risk assessment are vital for the long-term success of dental implant supported restorations. Endodontic status of adjacent teeth can significantly influence successful osseointegration of dental implants in partially edentulous situations. Retrograde periimplantitis can stay as clinically asymptomatic inactive lesion or may get infected with formation of a fistulous tract. Active lesions require prompt identification and timely management in order not to jeopardise the osseointegrated dental implant. Early intervention employing non-surgical and/or surgical intervention including regenerative therapy may be required.

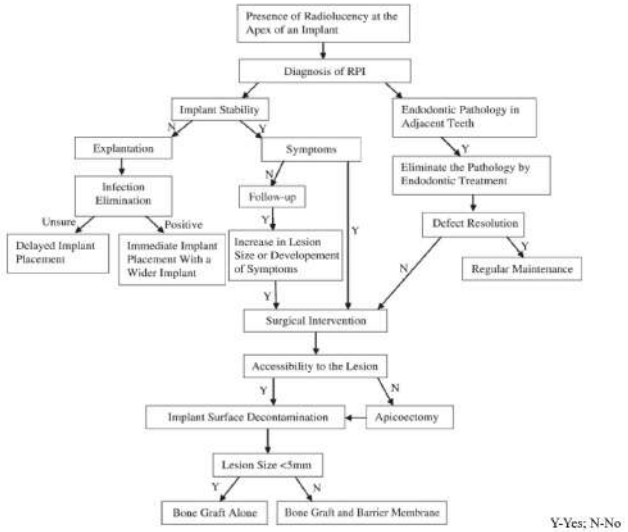


Figure 3 Decision-making flowchart in the management of retrograde periimplantitis

Figure 3: Adapted from: Chan HL, Wang HL, Bashutski JD, Edwards PC, Fu JH, Oh TJ. Retrograde peri-implantitis: a case report introducing an approach to its management. Journal of periodontology. 2011 Jul;82(7):1080-8.

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Photodynamic Therapy: Merging of Drug and Light

Anjali Babu[1], Arun Narayanan[2], Shabeer Ali K P[3], Deepthi V[4]

ABSTRACT

Recent scientific and technological breakthroughs have led to a constant drive to develop new treatment modalities for the treatment of periodontal disease. Microbial biofilms are considered to be the prime culprit for various disease conditions, including periodontal disease. The mechanical removal of this biofilm and adjunctive use of antibacterial disinfectants and antibiotics have been regarded as the conventional methods of periodontal therapy. A novel approach, photodynamic therapy (PDT), seems to be an emerging, non-invasive treatment method, involving photosensitizers, light of a specific wavelength and the generation of singlet oxygen and reactive oxygen species (ROS) to eliminate pathogenic microorganisms. The advantage of this new treatment modality includes rapid bacterial elimination, minimal chance of resistance development and safety of adjacent host tissue and normal microflora. Thus, PDT may be an effective way to treat periodontal diseases and may prove to be a promising alternative to conventional periodontal therapy in near future.

Keywords: Antimicrobial photodynamic therapy, periodontal disease, periimplantitis, photosensitizer, phototherapy

Introduction

The accumulation of biofilm on the tooth surfaces or tooth-gingival interfaces results in dental caries or periodontal disease.[1] It is generally recognized that the growth of bacteria in biofilms imparts a substantial decrease in susceptibility to antimicrobial agents compared with cultures grown in suspension.[2] Periodontal disease caused by dental plaque is characterized by the inflammation of periodontium, which eventually leads to the loss of periodontal tissue support.[3] The removal of the biofilm and adjunctive use of antibacterial disinfectants and antibiotics have been stated as the conventional methods.[4] So the current treatment regimen for periodontal disease involves mechanical debridement and this may be augmented with antibiotic therapy.[5,6] However, the emergence of resistant microorganisms and a shift in the microflora after extended use of antimicrobials restricted its usage.[7] Therefore the development of alternative antibacterial therapeutic strategies seems to be necessary to control microbial growth in the oral cavity.[8]

Photodynamic therapy (PDT) has emerged in recent years as a non-invasive therapeutic modality for the treatment of various infections caused by bacteria, fungi, and viruses.[9] Antimicrobial photodynamic therapy (aPDT), also known as photodynamic inactivation (PDI), photoactivated disinfection (PAD) or photodynamic antimicrobial chemotherapy (PACT), involves the administration of a photoactive dye or photosensitizer (PS) that is able to produce reactive oxygen species (ROS) upon irradiation with light of the correct wavelength to be absorbed by the PS. Allison et al. described PDT as a therapy that "is truly the marriage of a drug and a light". PDT can be applied topically into a periodontal pocket in order to avoid

[1]Post graduate student, [2]Principal, Professor & Head, [3]Reader, [4]Assistant Professor, Department of Periodontology, Kannur Dental College, Anjarakandy, Kannur. Corresponding Author: Dr. Anjali Babu E-mail: anjalibabu626@gmail.com

overdoses and side effects associated with the systemic antimicrobial agent administration. It also minimizes the occurrence of bacterial resistance. Photodynamic antimicrobial chemotherapy represents an alternate antibacterial, antifungal, and antiviral treatment against drug-resistant organisms. Applications of PDT in dentistry are growing rapidly and are also used in the treatment of oral cancer, bacterial and fungal infections, and in the photodynamic diagnosis of the malignant transformation of oral lesions.

In this review, we propose to provide an overview of photodynamic therapy with emphasis on its current status as an antimicrobial therapy to control oral bacteria.

Historical Perspective of Photodynamic Therapy

The concept of treatment with light and photoactive compounds can be traced back over 6000 years to the ancient Egyptians who used light sensitive substances (psoralens) by crushing leaves of plants related to parsley with sunlight to treat sunburns.[3] Reference to the use of a plant extract for the restoration of skin pigmentation was made in 1400 BC, and the phototoxic effects of psoralens were described in 1250 AD. Later the use of phototherapy disappeared for many centuries, and being rediscovered by the western civilization at the beginning of the 20th century. The use of contemporary photodynamic therapy was first reported by the Danish physician, Niels Finsen.[4] He successfully demonstrated photodynamic therapy by employing heat filtered light from a carbon arc lamp (The Finsen Lamp) in the treatment of a tubercular condition of the skin known as Lupus Vulgaris.

The concept of cell death induced by the interaction of light and chemicals was first reported by Osar Raab, a medical student working with Prof. Herman von Tappeiner in Munich. During the course of his study on the effects of acridine on paramecia cultures, he discovered that the combination of acridine red and light had a lethal effect on infusoria (a species of paramecium). Subsequent work in the laboratory of Von Tappeiner coined the term "Photodynamic action" and showed that oxygen was indispensable.

Much later, Thomas Dougherty and Co-workers at Roswell Park cancer institute, Buffalo, New York, clinically tested PDT. In 1978, they published striking results in which they treated 113 cutaneous or subcutaneous malignant tumors and observed a total or partial resolution of 111 tumors. The active photosensitizer used in this clinical PDT trial was called Hematoporphyrin Derivative. It was John Toth, who renamed it as PDT. Wilson M in 1993, proved the effect of cyanide photosensitizer on gram-negative and gram-positive species. PDT was approved by the Food and Drug Administration (FDA) in 1999 to treat pre-cancerous skin lesions of the face or scalp.[1] PDT has emerged in recent years as a new non-invasive therapeutic choice.

Principles of Photodynamic Therapy

PDT is based on the principle that a photoactivable substance (the photosensitizer) binds to the target cell and can be activated by light of a suitable wavelength. During this process, free radicals are formed (among them singlet oxygen), which produce an effect that is toxic to the cell. By irradiation with light in the visible range of the spectrum, the dye (photosensitizer) is excited to its triplet state, the energy of which is transferred to molecular oxygen. The product formed is highly reactive singlet oxygen capable of reacting with biological systems and destroying them. First excited state with the energy of 94 kJ/mol (22 kcal/mol) above the ground state is important and the second excited state does not react.

Mechanism of Action

PDT involves three components: Light, a photosensitizer, and oxygen. When a photosensitizer is administered to the patient and irradiated with a suitable wavelength, a molecule of the photosensitizer in its ground singlet state (S) is excited to the singlet state (S*) and receives the energy of the photon (Figure 1). The lifetime of the S* state is in nanosecond range, which is too short to allow significant interactions with the surrounding molecules. The S* state molecule may decay back to the ground state by emitting a photon as light energy (fluorescence) or by internal conversion with energy lost as heat. Alternatively, the molecule may convert into an excited triplet state (T) molecule via intersystem crossing that involves a change in the spin of an electron. The lifetime of the T state is in microsecond to the millisecond range. Molecules in the T state can emit light (phosphorescence) by returning

to the ground state or can react further by one or both of two pathways (known as the Type I and Type II photo processes), both of which require oxygen.

The Type I reaction involves electron transfer reactions from the photosensitizer triplet state with the participation of a substrate to produce radical ions that can react with oxygen to produce cytotoxic species, such as superoxide, hydroxyl and lipid-derived radicals.

The Type II reaction involves energy transfer from the photosensitizer triplet state to ground state molecular oxygen (triplet) to produce excited state singlet oxygen, which can oxidize many biological molecules, such as proteins, nucleic acids and lipids, and lead to cytotoxicity. Singlet oxygen, probably the major damaging species in photodynamic therapy, has a diffusion distance of approximately 100 nanometer (nm) with half-life of <0.04 microseconds (μs) and a very short radius of action {0.02 millimeter (mm)}. Hence, the reaction takes place within a limited space, leading to a localized response; thus, making it ideal for application to localized sites without affecting distant cells or organs. Thus, the type II reaction is accepted as the major pathway in microbial cell damage.

These two reactions indicate the mechanisms of tissue/cell damage which is dependent on both oxygen tension and photosensitizer concentration. PDT produces cytotoxic effects on sub cellular organelles and molecules. Its effects are targeted on mitochondria, lysosomes, cell membranes and nuclei of tumor cells. Photosensitizer induces apoptosis in mitochondria and necrosis in lysosomes and cell membranes.

Light Source

PDT requires a source of light to activate the photosensitizer by exposure to low power visible light at a specific wavelength. A laser or visible light source is used to activate the photosensitizer.[1] Early laser systems were complex and expensive. Subsequently, diode laser systems that were easy to handle, portable and cost-effective were developed. More recently, less expensive non-laser light sources are used, such as light-emitting diodes (LED), they are small, lightweight and highly flexible. Most photosensitizers are activated by red light between 630 and 700 nm, corresponding to a light penetration depth from 0.5 centimeter (cm) to 1.5 cm. This limits the depth of necrosis. The use of a visible light source is beneficial in visualizing the target area, localization of the photoinactivation without damaging host tissue and causes little damage to the operator. The total light dose, dose rates, and the depth of destruction vary with each tissue treated and photosensitizer used.

Currently, the light source applied in photodynamic therapy are those of helium-neon (He-Ne) lasers (633 nm), gallium – aluminum – arsenide diode lasers (630-690, 830 or 906 nm), and argon laser (488-514

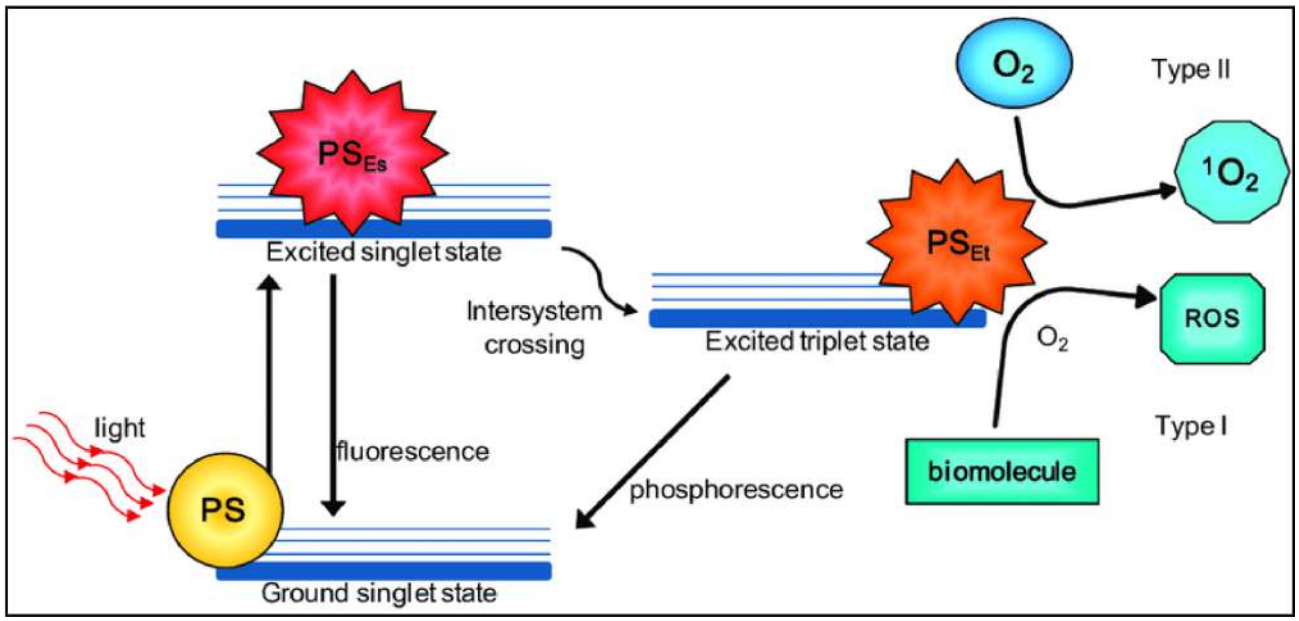

Figure 1: Mechanism of photodynamic therapy action.

nm), and the wavelength of which ranges from visible light to the blue of argon lasers, or from the red of helium - neon laser to the infra-red area of diode lasers.

Sources of light delivery vary depending on the location and morphology of the lesion. The light should be uniform and should deliver precise calculation of the delivered dose. Fibre-optic catheters with terminal cylindrical diffusers or lenses are often used. The tip of the fibre can be formed into various shapes allowing for diffusion in all directions. Currently, the use of fibre optics is very expensive and also it is not approved by FDA. Only diffusing fibres (1–5 cm) are commercially available. Modern fibre-optic systems and different types of endoscopes can deliver light more accurately to the target lesion. Custom-sized and custom-shaped fibres are needed to achieve more homogenous illumination. Overall, the light must penetrate as far as possible into the tissues and not produce thermal effects.

Photosensitizers

PDT uses several photoactive components. An optimal photosensitizer must possess photo- physical, chemical, and biological characteristics. In addition, for the treatment of periodontal infections, the photosensitizer should bind with bacteria and plaque without causing any cosmetic issues, such as unwanted staining of gingiva and other soft tissues. Furthermore, it should be acceptable to patients and personnel and easily access pathogens present in deeper periodontal pockets.

Optimum properties of a photosensitizer[8]

1. High quantum yield of triplet state to obtain large concentrations of the activated drug
2. Should possess high singlet oxygen quantum yield
3. High binding affinity for microorganisms
4. Should possess broad spectrum of action
5. Should have low binding affinity for mammalian cells to avoid the risk of photo destruction of host tissues
6. Low propensity for selecting resistant bacterial strains
7. Minimal risk of promoting mutagenic processes
8. Low chemical toxicity and fast elimination from skin and epithelium
9. High solubility in water, injection solutions and blood substitutes
10. Storage and application light stability

Types of Photosensitizers

Chemically, many photosensitizers belong to dyes and porphyrin-chlorine groups. A variety of photosensitizers include:

1. Dyes: tricyclic dyes with different meso-atoms – methylene blue, toludine blue O (TBO) and acridine orange; and phthalocyanines – aluminum disulphonated phthalocyanine and cationic Zn (II)- phthalocyanine
2. Chlorines: chlorine e6, stannous (IV) chlorine e6, chlorine e6-2.5 N-methyl-d-glucamine (BLC1010), polylysine and polyethyleneimine conjugates of chlorine e6
3. Porphyrines: haematoporphyrin HCl, photofrin and 5-aminolevulinic acid (ALA), benzoporphyrin derivative (BPD)
4. Xanthenes: erythrosine
5. Monoterpene: azulene.

Photofrin and hematophyrin derivatives are referred as first-generation sensitizers. Second generation photosensitizers include 5–aminolevulinic acid (ALA), benzoporphyrin derivative, texaphyrin, and temoporfin (mTHPC). These photosensitizers have greater capability to generate singlet oxygen. Topical ALA has been used to treat precancerous conditions, basal cell carcinoma and squamous cell carcinoma of skin.

Antimicrobial photosensitizers such as porphyrins, phthalocyanines and phenothiazines (e.g., toluidine blue O and methylene blue), which bear a positive charge and they can directly target both gram-negative and gram-positive bacteria. The positive charge seems to promote the binding of the photosensitizer to the outer bacterial membrane, inducing localized damage, which favors its penetration. Toluidine blue O and methylene blue are commonly used for oral antimicrobial photodynamic therapy. Tetracyclines used as antibiotics in periodontal diseases are also observed as effective photosensitizers producing singlet oxygen.

Toluidine blue O (Figure 2) is a vital dye, which is blue – violet in color. It stains granules and proteoglycans/glycosaminoglycans within the mast cells and connective tissues respectively. It has been used for staining mucosal abnormalities of the uterine cervix and oral cavity and for demarcating the extent of lesions before excision. Moreover, it is considered as a potent photosensitizer for killing oral bacteria.

Methylene blue (Figure 3) has been used as a photosensitizing agent since 1920s. It has been used to detect mucosal premalignant lesions and as a marker dye in surgery. The hydrophilicity of methylene blue, along with its low molecular weight and positive charge, allows passage across the porin-protein channels in the outer membrane of gram-negative bacteria. Methylene blue is a redox indicator that is blue in an oxidizing environment and becomes colorless upon reduction. Methylene blue combined with light has been reported to be beneficial in killing the influenza virus, Helicobacter pylori, and Candida albicans.

Photodynamic Therapy and Periodontitis

Biofilms that colonize tooth surfaces and epithelial cells lining the periodontal pocket/gingival sulcus are among the most complex biofilms that exist in nature. These biofilms include a subset of selected species from more than 700 bacterial species that can lead to periodontal diseases. Mechanical removal of this microbial biofilm along antibiotic therapy is the most frequently used treatment modality for periodontal disease. Using antimicrobial agents to treat periodontitis without disruption of the biofilm ultimately results in treatment failures. Unfortunately, the microorganisms in the biofilm are less susceptible to antibiotics and also it is difficult to maintain optimum therapeutic concentrations at the target sites. PDT has the potential to destruct this biofilm as well as to minimize drug resistance, so it seems to be an effective alternative to conventional therapy. Polysaccharides present in extracellular matrix of oral biofilm are highly sensitive to singlet oxygen and susceptible to photodamage. Breaking the biofilm may disrupt microbial colonization and also inhibit plasmid exchange involved in transfer of antibiotic resistance. Antioxidant enzymes produced by bacteria may protect against some oxygen radicals, but not against singlet oxygen. Photodynamic antimicrobial chemotherapy could be an ideal complement to conventional scaling and root planing, because it employs a quick and simple protocol to kill bacteria and also inactivate virulence factors left behind after scaling and root planing. It is used during initial and maintenance therapy for the treatment of periodontitis. The liquid photosensitizer placed directly in the periodontal pocket can easily access the whole root surface before activation by the laser light through an optical fiber placed directly in the pocket. Antimicrobial PDT not only kills the bacteria, but may also lead to the detoxification of endotoxins such as lipopolysaccharide. These lipopolysaccharides, treated by PDT do not stimulate the production of pro-inflammatory cytokines by mononuclear cells. Thus, PDT inactivates endotoxins by decreasing their biological activity.

During inflammation there is venous stagnation and reduced oxygen consumption by tissues. This decrease in oxygen level and change in pH may enhance the growth of anaerobic species. In such cases, PDT may improve tissue blood flow in the microcirculatory system and reduce venous congestion

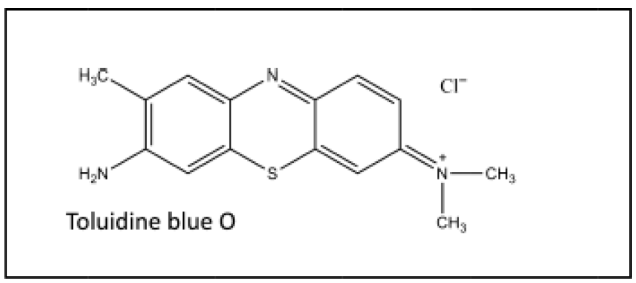

Figure 2: Chemical structures of the phenothiazine photosensitizers Toluidine blue O, C15H16N3SCl (also known as tolonium chloride, basic blue 17, blutene chloride and methylene blue T50 or T extra)

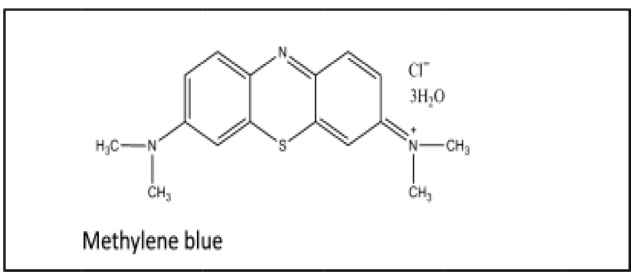

Figure 3: Methylene blue, C16H18ClN3S (also known as methylthionine chloride and 3,7-bis(Dimethylamino)-phenazothionium chloride).

in gingival tissues. Furthermore, PDT may increase oxygenation of gingival tissues by 21–47 per cent. This in turn decreases the time and speed of oxygen delivery and utilization, thus normalizing oxygen metabolism in periodontal tissues.

The susceptibility to destruction of bacteria by PDT is different between gram-positive and gram-negative species. Gram-positive bacteria are more susceptible to photoinactivation than gram-negative bacteria. The structural variations in their cytoplasmic membrane are responsible for the enhanced susceptibility of gram-positive bacteria to binding to photosensitizers. In gram-positive bacteria, the relatively porous outer cytoplasmic membrane, peptidoglycans, and lipoteichoic acid outside the cytoplasmic layer allows the neutral or anionic photosensitizer to bind efficiently into it. In gram-negative bacteria, the structure of the outer membrane is more complex, forming a physical and functional barrier between the cell and its environment, thereby making it difficult for the photosensitizer to gain access into internal target sites. However, this diffusion may be enhanced by:

1) Linking the sensitizer to a polycationic molecule (poly-L-lysine-chlorine, polymyxin B nonapeptide), that leads to weaken the intermolecular interactions of the lipopolysaccharide constituents, disorganize the structure, and render it permeable to drugs by enabling them to cross the outer membrane.

2) Use of membrane active agents [treatment with tris-Ethylene Diamine Tetra-acetic Acid (EDTA)], which release lipopolysaccharide or the induction of competence with sensitized pathogen.

3) Conjugating the sensitizer to monoclonal antibodies that bind to cell-surface specific antigens.

The selective uptake of photosensitizers by bacteria can be enhanced by conjugation with various peptides. For example, Poly-L-lysine (pL)–chlorine e6 conjugates kill *Porphyromonas gingivalis (P. gingivalis)* without affecting the viability of epithelial cells. The polycationic lysine polypeptide is responsible for the initial binding of the photosensitizer to bacteria due its structural similarity to antimicrobial peptides causing cell lysis. Linking the toludine blue O to a monoclonal antibody has been shown to inactivate the lipopolysaccharide of *P. gingivalis*. Hence, conjugated photosensitizers are beneficial in targeting bacteria or particular virulence factors without damaging epithelial cells.

The roles of virulence factors in pathogenesis of periodontal diseases are well documented. PDT has another advantage in inactivating virulence factors secreted by micro-organisms. Following exposure of *P. gingivalis* to low-energy He-Ne laser (632 nm) and TBO (25 um/ ml), the activity of lipopolysaccharide and Interleukin-1 secretion from human peripheral mononuclear cells exposed to such treatment were significantly reduced. In addition, there was a substantial, light dose dependent decrease in the proteolytic activity (94%) of *P. gingivalis*. Such effects may be of beneficial in the treatment of periodontal disease. There are two basic mechanisms that have been proposed to account for the lethal damage caused to bacteria by PDT:

1) Deoxyribonucleic acid (DNA) damage

2) Damage to the cytoplasmic membrane, allowing leakage of cellular contents or inactivation of membrane transport systems and enzymes.

Breaks in both single and double stranded DNA, and the disappearance of the plasmid super coiled fraction have been detected in both gram-positive and gram-negative species after PDT with a wide range of photosensitizer structural types. Although DNA damage occurs, it may not be the prime cause of bacterial cell death. The alteration of cytoplasmic membrane proteins, disturbance of cell-wall synthesis and the appearance of a multi-lamellar structure near the septum of dividing cells, along with loss of potassium ions from the cells may be other possible ways of bacterial death.[1] It has been hypothesized that photosensitizers that operate chiefly via Type I mechanisms penetrate the outer membrane of gram negative bacteria, while the Type II photosensitizers penetrate the outer membrane of gram positive bacteria more efficiently.

The bactericidal activity of PDT depends on various factors such as:

1. The surface charge of the photosensitizer determines its binding with the cell membrane.

2. The electrostatic interaction between the positively charged surface of photosensitizer and the negatively charged membrane of the bacteria.

The environmental conditions surrounding

bacteria may influence the efficient binding of photosensitizers. In vitro studies have shown that blood agar culture media, hemin content and the pH of the medium used may inhibit the binding of photosensitizer with pathogens. Blood contained in the culture media adsorbs the part of laser light, hemin competes with the photosensitizer binding sites and bacterial metabolic byproducts alters the pH of the medium. All of these alter the binding of the photosensitizers to the target sites, resulting in less binding and reduced photoinactivation.

However, the black-pigmented species, such as *P. gingivalis,* Prevotella intermedia and Prevotella nigrescens, are more susceptible to elimination by lethal photosensitization. Intracellularly, they accumulate various amounts of different porphyrin molecules (Prevotella intermedia 267 nanogram/milligram (ng/mg), Prevotella nigrescens 47 ng/mg, Prevotella melaninogenica 41 ng/mg and P. gingivalis 2.2 ng/mg), together with varying amounts of iron free protoporphyrin IX. These photosensitive porphyrins absorb visible light at different wavelength and different energy level and enhance the killing effect.[1]

In 2007, de Oliveira et al., conducted a randomized controlled clinical study to compare the effects of PDT alone without sub gingival scaling root planing (SRP) to sub gingival SRP in subject with aggressive periodontitis and they found that at three months following the therapy, both treatment yielded comparable outcomes in terms of reduction of bleeding on probing and probing depth (PD), gains in clinical attachment level (CAL), thus suggesting a potential clinical benefits of PDT.

According to the study conducted by Braun et al., in 2008, PDT adjunctive to non-surgical periodontal treatment enhances the clinical outcomes.

Effect on mucosal tissues

The epithelium at the dentogingival area acts as a primary barrier for invasion of noxious stimuli and also for the penetration of photosensitizers. The thick layer of stratified keratinized gingival epithelium acts as a barrier for diffusion of water-soluble photosensitizers. Sulcular epithelium shows increased penetration due to its non-keratinization. *Porphyromonas gingivalis* and *Aggregatibacter actinomycetemcomitans* can infiltrate through the epithelial barrier into the periodontal tissues, therefore elimination of these organisms is possible by PDT.[1] Uptake of photosensitizer by epithelial cells depends on incubation time (after application of photosensitizer till its activation by light).

Photodynamic Therapy and Peri-Implantitis

Plaque-induced peri-implantitis is an inflammatory condition that affects soft and hard tissues surrounding an osseointegrated dental implant and may lead to its failure. The incidence of peri-implantitis in patients with chronic periodontitis is up to five times greater than in patients who are free of this disease. In addition, greater proportions of periodontal pathogens have been found in infected and failing implants compared with non-failing implants. The management of peri-implantitis includes the mechanical removal of biofilm from the implants, along with the local application of antiseptics and antibiotics to kill bacteria in the surrounding peri-implant tissues, and also regenerative surgery to reestablish the bone–implant interface.

According to the study by Dortbudak et al.,[10] in the year 2001, photodynamic therapy achieved significant, but incomplete, elimination of *Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis* and *Prevotella intermedia.*

Haas et al.,[11] demonstrated that combination of toluidine blue O-mediated photodynamic therapy with guided bone regeneration resulted in the reduction of bone defects (the mean radiographic peri-implant bone gain was 2 mm) in 21 of 24 implants at 9.5 months following treatment. Thus, these studies show the role of photodynamic therapy as an adjunctive methodology to nonsurgical therapy.

Advantages of Photodynamic Therapy[1,3]

- Minimally invasive technique with least collateral damage to normal cells enhances results and superior healing.
- Exceedingly efficient broad spectrum of action, since one photosensitizer can act on bacteria, virus, fungi, yeasts, and parasitic protozoa.
- Efficacy independent of the antibiotic resistance pattern of the given microbial strain.
- Development of resistance to the PDT is

less as singlet oxygen and other free reactive oxygen species interact with several cell structures and different metabolic pathway.

- As PDT is non-invasive local therapy, disturbances of the microflora at other sites would not occur and damage to the adjacent host tissues can be avoided.
- PDT offers thorough irrigation and elimination of pathogens in inaccessible areas of periodontal pocket within short span of time, thus beneficial to both operator and the patient.
- The risk of bacteremia after periodontal debridement can be minimized.
- There is no need to prescribe antibiotics; therefore, the possibility of side effects can be avoided.
- There is no need to anaesthetize the area and destruction of bacteria is achieved in a very short period (<60 seconds).
- The therapy also causes no adverse effects such as ulcers, sloughing or charring of oral tissues
- Economical to use.

Adverse Effects of Photodynamic Therapy

- Systemic administration of photosensitizer causes a period of residual skin photosensitivity due to the accumulation of photosensitizers under the skin. Therefore, photosensitizers can be activated by daylight causing first or second-degree burns. Hence, direct sunlight must be avoided for several hours until the drug is completely eliminated from the body.
- Most of the dyes adhere strongly to the soft tissue surface of the pocket, even for a shorter period of time, may affect periodontal tissue attachment during wound healing.
- Haematoporphyrin derivative photosensitizer resulted in vesicle formation on tongue with oedema, cellular infiltration and reduction in number of vessels but muscle fibres remained intact.[1]
- Gingival ulceration, muscle necrosis and necrotizing sialometaplasia of salivary glands were observed in rabbits following systemic administration of disulfonated phthalocynanine (5 mg/kg and 20 Joules at 675 nm).[1]

Precautions

- The potential inadvertent irradiation of the patients' eyes must be strictly avoided during treatment, therefore the use of protective glasses by the patient, the operator and the assistant is recommended.[4]

Future Perspectives

The reduced susceptibility of complex oral biofilms to antimicrobial photodynamic therapy may require the development of novel delivery and targeting approaches. Evolving therapeutic strategies for biofilm-related infections include the use of substances designed to target the biofilm matrix, non-growing bacteria (persister cells) within biofilms and/or quorum sensing.[8] The use of bacteriophages and naturally occurring or synthetic antimicrobial peptides may offer the possibility of bacterial targeting without the emergence of resistance. Recently, the advantages of targeted therapy become more apparent, and the use of light alone, antibody–photosensitizer and bacteriophage–photosensitizer conjugates or non-antibody based targeting moieties, such as nanoparticles, are gaining increasing attention.

Phototherapy

Application of a photosensitizer may not be required in some instances, because photosensitizers occur naturally within some microbial species, in particular oral black-pigmented species. Soukos et al., shown that broadband light ranging from 380 to 520 nm was able to achieve a threefold reduction in the growth of *P. gingivalis, Prevotella intermedia, Prevotella nigrescens* and *Prevotella melaninogenica* in dental plaque samples obtained from human subjects with chronic periodontitis. According to Sterer et al., exposure of human salivary microflora to blue light of 400–500 nm showed reduction in levels of volatile sulfide compounds and also demonstrates a selective inhibitory effect on the gram-negative bacteria, suggesting that it may be possible to use light to treat

oral malodor.

Thus, it is evident that very short exposures of periodontal pockets, and of the mucosa of the dorsum of the tongue, to visible light, especially blue light in human subjects with gingivitis, periodontitis and oral malodor may lead to a cumulative suppressive effect on both dental plaque and tongue black-pigmented species by activating their endogenous porphyrins. This may have an impact on the reduction in bleeding and inflammation in periodontitis and also effective against oral malodor and thereby leads to a shift of the microbial composition towards a new one associated with health. This novel technique may offer the advantages such as (i) rapid and painless application of light; (ii) selectivity in its effect; (iii) full penetration of dental plaque by light; (iv) limited penetration of light into gum tissue; (v) absence of phototoxicity to human cells; (vi) no effects on taste; and (vii) possible clinical and microbiological benefit with minimal impact on natural microbiota.

Antibody-targeted antibacterial approaches using photodynamic therapy

Antibody-targeted approaches using photodynamic therapy have been most frequently focused on the treatment of malignant diseases. The therapeutic potential of these approaches for bacterial targeting is based on their ability to demonstrate minimal damage to host cells. Antibodies conjugated with photosensitizers have been used to target *Staphylococcus aureus*. According to Bhatti et al., selective killing of *P. gingivalis* was achieved in the presence of Streptococcus sanguinis or in human gingival fibroblasts using a murine monoclonal antibody against *P. gingivalis* lipopolysaccharide conjugated with toluidine blue O. The combination of pulsed laser energy and absorbing gold nanoparticles selectively attached to the bacterium for killing of microorganisms is the latest innovation. Gold nanoparticles are promising candidates for application as photothermal sensitizers and can easily be conjugated to antibodies.[8] The surface of *Staphylococcus aureus* was targeted using 10- to 40-nm gold nanoparticles conjugated with anti-protein antibodies. The energy that was absorbed by nanoparticles during irradiation was quickly transferred through non radiative relaxation into heat accompanied by bubble-formation phenomena around clustered nanoparticles, leading to irreparable bacterial damage.

Nanoparticle-based antimicrobial photodynamic therapy

One of the intricated issues regarding the PDT is the incomplete penetration of methylene blue in oral biofilms. In order to overcome these deficiencies, new delivery systems that significantly improve the pharmacological characteristics of methylene blue have been developed. According to Pagonis et al., encapsulation of methylene blue within poly (D, L lactide- co-glycolide) (PLGA) nanoparticles (150 – 200 nm in diameter) that may offer a novel design of nano-platform for enhanced drug delivery and photo destruction of oral biofilms. The nanoparticles matrix PLGA is a polyester co-polymer of polylactide and polyglycolide that has received approval by the US Food and Drug Administration as a result of its biocompatibility and its ability to degrade in the body through natural pathways. Once encapsulated within PLGA, the excited state of the PS is quenched, which results in the loss of phototoxicity. When the nanoparticles were incubated with cells, they showed a time dependent release of the PS, which then regained its phototoxicity and resulted in an activatable photodynamic therapy-nanoagent.

The use of biodegradable polymer to synthesize the nanoparticles makes the final product attractive for clinical use. This nanoagent has several favorable properties for use as a photosensitizer, such as (i) a large critical mass (concentrated package of photosensitizer) for the production of reactive oxygen species that destroy cells; (ii) it limits the cells ability to pump the drug molecule back out and reduces the possibility of multiple drug resistance; (iii) selectivity of treatment by localized delivery agents, which can be achieved by either passive targeting or by active targeting via the charged surface of the nanoparticles; and (iv) the nanoparticles matrix is non immunogenic.

PLGA nanoparticles loaded with various compounds (e.g., antibiotics) have been used for bacterial targeting; however, the use of PLGA nanoparticles as carriers of photosensitizers has not been explored in antimicrobial photodynamic therapy until recently.

Conclusion

Antimicrobial PDT seems to be a unique and interesting therapeutic approach towards the

treatment of periodontitis and periimplantitis. The currently available literature illustrates that the use of PDT appears to be very promising, although the development of PDT is still in its "infant" stage. Development of new photosensitizers, more efficient light delivery systems and further clinical studies are required to establish the optimum treatment parameters for PDT. Antimicrobial photodynamic therapy may hold promise as a substitute for currently available chemotherapy in the treatment of periodontal and peri-implant diseases. Further, randomized long term clinical studies and meta-analyses are inevitable to signify the favourable effect of antimicrobial photodynamic therapy, and in comparison, with conventional methods.

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JSPIK

Laser Assisted New Attachment Procedure: The Rising Star

Neelima Vinod[1], Mohammed Feroz T P[2], Sunith Sudhakaran[3], Rizleena Majeed[4]

ABSTRACT

Lasers have emerged as an adjunct in various treatment modalities in dentistry over the last few decades. This minimally invasive technique has been found to produce positive outcomes in a plethora of periodontal treatment procedures. Laser Assisted New Attachment Procedure (LANAP) introduced by Gregg and McCarthy in 1990 is an innovative treatment for periodontal diseases incorporating pulsed neodymium yttrium aluminum garnet (Nd: YAG) 1064 nanometre (nm) wavelength laser. It has been shown that LANAP has the potential to initiate regeneration of the affected periodontal tissues and new connective tissue attachment mediated by cementum.

Keywords: Laser, LANAP, Nd: YAG, Regeneration

Introduction

The periodontal treatment pattern is shifting from a realm of resective to regenerative and reconstructive procedures. Conventional surgical procedures are not always welcomed by the patients due to pain, swelling, root exposure and postoperative discomfort.[1,2] Hence development of effective and less invasive procedures has become the need of the hour for both patients and clinicians.

Millennium dental technologies introduced an innovative therapy LANAP described as Laser Assisted New Attachment Procedure by PerioLase MVP-7 which is a Neodymium Yttrium Aluminium Garnet (Nd: YAG) 1064nm laser in a free running pulsed mode as an alternative or adjunct to the conventional periodontal surgical procedures. It has been noticed that LANAP has the potential for the regeneration of the affected periodontal tissues and new connective tissue attachment mediated by cementum.[1-4] It is a carefully planned treatment protocol which focuses not only on bactericidal and detoxification effect but also intends to diminish the etiology of the disease.[1]

History of LANAP

Dr. Robert H. Gregg II and Dr. Delwin K. McCarthy together with more than 1000 dentists and specialists pioneered the use of the free running pulsed Nd:YAG laser for the treatment of periodontal diseases since 1990s. They were astonished by their ability to regenerate bone and stimulate new attachment for their patients with severe periodontal diseases.[1,3,12,13]

LANAP protocol

LANAP is defined as "Cementum-mediated" new attachment to the root surfaces in the absence of a long junctional epithelium.[3,12,14] It is a minimally invasive well-defined procedure which involves the surgical

[1]Postgraduate student, [2]Professor, [3]Reader, [4]Assistant Professor, Department of Periodontology, Kannur Dental College, Anjarakandy, Kannur. Corresponding Author: Dr. Neelima Vinod, E-mail: neelimachakku@gmail.com

removal of the sulcular epithelium, modification and osteoplasty of bone and perforation of the periodontal ligament (PDL) using piezoelectric bone cutting tips and wound closure via a thermogenic stable fibrin clot without sutures.[12,15] Pocket reduction, new tissue attachment and lack of tissue recession are obtained with LANAP. The procedure combines the PerioLase MVP-7 Free-Running (FR) pulsed Nd: YAG laser with a strict, specific, research-proven protocol that has achieved Food and Drug Administration (FDA) clearance in 2004 for the treatment of all forms of periodontal disease. This single laser surgical treatment is patented by Millennium Dental Technologies Cerritos, CA, USA.[1,13]

LANAP specific clinical steps (Figure 1) must be performed properly and precisely for achieving consistent positive outcomes. Patients which require standard periodontal treatment with pocket depth ≥4 millimetre (mm) are indicated for LANAP. The procedure may be performed in all four quadrants in a single appointment, but for patient comfort, laser treatment is typically limited to no more than two non-adjacent quadrants per visit with several days between visits.[1,4,8-9,12,13] The patient is anesthetized initially with a local anesthetic to properly access the extent of intrabony defect with a probe. An optic fiber tip measuring 0.3-0.4 μ is placed parallel to the root surface to carry away the pocket epithelial lining in coronoapical direction to reflect the gingival flap. The first pass laser or troughing dissipates energy at 4-Watt, free running 100 milliseconds pulse expels the unhealthy lining of the pocket. The duration of the pulse is short. Calcified plaque adherent to the root surface is removed. Selective photo thermolysis removes unhealthy infected and inflamed epithelium of the pocket sparing the intact connective tissue separation of the layers of tissues at rete pegs and ridges level. Laser used with utmost precision in variation of energy density, pulse duration and calculated rate of repetition leads to antiseptic hemostasis and tissue ablation. The second pass with energy dissipation at 4 W, 650 milliseconds pulse allows re-entry of the pocket. This establishes a sticky fibrin blood clot which secures the pocket from detritus matter and facilitates healing from inside out. The pocket is closed by compressing gingival tissues against the root surface which creates a firm fibrin clot. No placement of sutures or surgical glue is required. Splinting of grade II mobile teeth is done. Interferences are minimized by proper occlusal adjustments to decrease traumatic forces simultaneously creating a balance with long axis forces are vital elements of the LANAP protocol.

At the end of the procedure patients are given postoperative instructions pertaining to LANAP protocol including proper diet specifications along with oral hygiene instructions with a lot of emphasis on continued periodontal maintenance. Patients are recalled at an interval of one week, one month and thereafter every three months for periodontal maintenance. In order to allow sufficient recovery time for tissues to heal at cementum fiber PDL interface, probing is ruled out for next six months to one year.

LANAP shows exceptional prediction for regeneration which could be attributed to the selective absorption of laser only by the diseased tissues discriminating healthy adjacent tissues, bactericidal effect on pigmented bacteria, thermal fibrin clot formation which secures the pocket crevice thereby preventing the coronal to apical movement of epithelium.

Figure 1: Schematic Illustration of LANAP

Yukna et al, McAllister, Nevins et al, Brown and Harris et al studies are some of the major studies with definite positive results for LANAP.[1-3,5,10] In one of the largest human histology studies, Yukna et al were the first to publish and prove the positive results of LANAP therapy when compared with conventional periodontal treatment. The results showed that 100% of the teeth treated with the LANAP procedure formed new attachment as opposed to 0% of the control teeth. While treating intrabony defects, new cementum and new connective tissue attachment adjacent to alveolar bone were seen but no long-junctional epithelium was observed.[5]

Nevins et al in 2012 reported another landmark human block study demonstrating highly successful outcomes of patients treated with the LANAP Protocol in cases of severe periodontitis. They studied the histological aspect of healing process while incorporating LANAP protocol to treat intrabony defects. Eight patients with 930 sites were taken up for study with an average of 19.38 teeth per patient (assuming six sites per tooth). Incredible results following LANAP treatment were seen in severe periodontitis.[2]

McAllister conducted a study in 2009 on three cases which were conclusive of positive results of LANAP using the Nd:YAG PerioLase MVP-7 laser for the treatment of moderate to severe adult periodontitis in routine dental practice. All three cases showed clear radiographic bone regeneration following LANAP. He concluded that LANAP presents a less invasive approach and shows better patient compliance.[10]

Brown's research on PerioLase MVP-7 laser demonstrated that LANAP outcomes are phenomenal and comparable to conventional periodontal surgery while facilitating regeneration.[3] A split mouth randomized multicentre study conducted by Harris et al in 2014 advocated that LANAP protocol could produce a systemic effect on subgingival wound healing and minimal postoperative discomfort to the patient.[6]

Western society of Periodontics advocated that proper scientific proofs should be thoroughly checked and reviewed by clinicians before practicing LANAP. There are no proper studies to figure out the substantiation of LANAP in detecting diseased and healthy tissues.[1,15] The American Academy of Periodontology 1999 stated with respect to this technique, "The Academy is not aware of any randomized blinded controlled longitudinal clinical trials, cohort or longitudinal studies, or case-controlled studies indicating that 'laser excisional new attachment procedure (or Laser ENAP)' or 'laser curettage' offers any advantageous clinical results that are not achieved by traditional periodontal therapy".[12]

Behdin et al in 2015 conducted a systematic review and meta-analysis on the effect of laser in periodontal therapy. Their study could not conclude LANAP to have better results as compared to traditional treatment procedures but they also suggested more randomized clinical trials for better assessment.[7] Shah et al in 2015 concluded that Nd:YAG lasers did not have better results over conventional periodontal therapy in the treatment of initial periodontitis.[11] More longitudinal studies are required to prove LANAP to be the best treatment option for daily practice in order to completely resolve the controversies.

Periodontal Healing after LANAP

The periodontal tissue healing after the LANAP procedure is found to be of secondary intention. A stable fibrin clot is formed with the second pass of the laser thereby creating a barrier to prevent the epithelial down growth and ingress of bacteria and deposits. The epidermal cells from both the margins of the wound proliferate and migrate into the wound space to form epithelial spurs and complete re-epithelialization takes place. The granulation tissue elements from the PDL gives rise to the formation of a new cementum and connective tissue attachment. The formation of this new cementum is believed to occur in not less than three weeks.[12]

Advantages of LANAP

- Less invasive
- Less traumatic
- Closure is achieved without sutures
- Minimal postoperative discomfort like pain, bleeding, swelling
- Less prone to recession
- Faster healing is achieved.
- Equally successful results obtained in treating dental implants and natural teeth.
- Sealing of the pocket orifice with a thermal

fibrin clot which acts as a physical barrier preventing the apical growth of epithelium.

- Safe treatment for patients with conditions like haemophilia, HIV and diabetes, or on medications such as cyclosporine.
- Less likely to develop hypersensitivity[1,3,9-10,12]

Disadvantages of LANAP

- The treatment can be expensive.
- If the laser is not used with caution or improperly angled for debridement, it can cause serious alveolar tissue damage or may cause undesirable pulpal changes.[12]

LANAP and ENAP

ENAP- "Excisional new attachment procedure" was described in 1976 as "a definitive subgingival curettage performed with a knife." ENAP results in a long, thin epithelial attachment and a minimal amount of connective tissue attachment. The only published human clinical study comparing the clinical outcomes of gingival curettage to that of ENAP procedure found no significant differences in probing pocket depth reductions or clinical attachment level gain.[12]

Study conducted by Sameera et al in 2018 compared the clinical efficacy of LANAP versus ENAP and also assessed the blood flow in both the procedures using ultrasound Doppler flowmetry. This study was a split-mouth double-blinded controlled clinical trial carried out in fifteen subjects with chronic periodontitis. Results showed a statistically significant reduction in all the clinical parameters when compared baseline to subsequent follow-ups. They observed a greater reduction in all the parameters in the LANAP group compared to that of ENAP group and the rate of revascularization was found to be higher in the ENAP group than that of LANAP group.[16]

Recent Advances in LANAP

LANAP and Implants

Millennium technologies came up with a modification of LANAP for implant known as LAPIP protocol for implants.[1,12]

The LAPIP Protocol

McCarthy put forward the concept of LAPIP, "Laser Assisted Peri-Implantitis Procedure" which could be used in diseased implants. Laser removes inflamed pocket tissue, disrupt biofilms, and decontaminate the root/implant surface. Decrease in inflammation and a laser-induced hemostasis further decontaminates the tissue creating a durable blood clot to close the system. LAPIP results in converting the diseased structure to healthy state, promotes bone and tissue regeneration. The most commendable feature is that the procedure is performed on implant without damaging it. A single appointment might be sufficient. Since no flap is reflected, it even leaves chances for other therapies in the future. The LAPIP protocol recommends the PerioLase MVP-7, Nd:YAG "free-running" pulsed laser to treat periimplantitis.[17] Invitro study conducted by Giannelli on the effects of Nd:YAG laser concluded that the use of Nd:YAG laser appears as a solution to treat peri-implantitis.[18]

LANAP and Diabetes

Long has described the advantage of LANAP protocol performed with the PerioLase MVP-7 in treating diabetic patients with Type IV periodontitis. This therapy has been shown to provide new bone growth and stability in patients with type IV chronic periodontitis, re-establish new cementum-mediated periodontal ligament attachment and induce periodontal regeneration. HbA1c levels and periodontal health of patients exhibited marked improvement after LANAP treatment.[8]

Laser-assisted bacterial reduction in periodontal tissues

Nd:YAG laser has shown bactericidal effects in In vivo studies. The application of laser into deeper periodontal tissues has an effect on the pathogenic microflora in the tissues.[12,19]

Laser-assisted scaling and root planing

The removal of subgingival deposits using laser has been studied and is found to be an effective and simplest way of performing SRP.[12,19]

LANAP Protocol versus Conventional Procedures

The successful treatment of periodontal disease requires thorough debridement of the root surface. Pockets of 5 mm or greater depth make it difficult to remove subgingival plaque and calculus. Hence surgical intervention allows access and visualization for

scaling and root planing in these deep pockets. While scalpel surgery can accomplish access and visualization, it can also result in attachment loss, gingival cratering, gingival recession and associated pain and discomfort. General practitioners most of the times do not consider performing conventional flap surgery due to its invasive nature. LANAP being an alternative is not without its drawbacks. The predominant issues involve cost and time and the dental clinicians must also be willing to undergo procedural training and learn LANAP treatment with live patients. LANAP uses quartz fiber in place of a scalpel to achieve both tissue ablation and antibiotic properties. Hence no cutting means more comfortable recovery. However conventional periodontal treatments for now remain the standard of care in treating periodontal diseases. More studies need to be conducted to prove that any laser system provides clinical value surpassing scaling and root planning and conventional surgical treatments.[13]

Conclusion

LANAP is considered to be an emerging treatment modality in periodontal practice due to its efficacy to promote true periodontal regeneration. LANAP not only regenerates diseased tissues but also promotes antisepsis and increased tissue integrity. It also allows clinicians to achieve predictable positive results including three-dimensional regeneration of bone. The success of LANAP lies in the systematic way in which it is done. Hence the need for professional trainings before performing the procedure is a must to increase positive outcomes. Continued research and careful observation would pave the way for the acceptance of LANAP as a standard of care in treating patients with moderate to severe periodontal diseases.

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Microsurgery In Periodontics – A Concise Review

Gayathri B S[1], Rasha Usman M[2], Sonia P J[3], Archana M K[4], Raseena Beevi N[5], Roshni Ramesh[6]

ABSTRACT

Over the past two decades, Periodontology has seen an increasing refinement of surgical procedures, requiring the development of more intricate surgical and motor skills. The surgical techniques used in Periodontology and implant therapy demand clinical expertise beyond the range of normal visual acuity. These demands are met by the introduction of microsurgery which is characterized by use of improved magnification and precise instrumentation than conventional surgical techniques. The use of microsurgery provides new possibilities to improve the therapeutic outcome of various periodontal surgical procedures. The purpose of this paper is to provide a concise review of periodontal microsurgery, its components and various periodontal surgical procedures that can be performed using microsurgery.

Keywords: Microsurgery, Magnification, Microscope, Loupes

Introduction

Over the past two decades, periodontology has undergone an increasing refinement of surgical procedures, requiring the development of more complex surgical and motor skills. The surgical techniques used in periodontal plastic surgery, guided tissue regeneration, cosmetic restorative crown lengthening, gingival augmentation procedures, soft and hard tissue ridge augmentation, osseous resection, and dental implant placement demand clinical expertise beyond the range of normal visual acuity.

Periodontal microsurgery incorporates the use of a surgical microscope or loupes in an attempt to increase visibility. In 1921, Carl Nylen, who is considered the Father of Microsurgery, first performed eye surgery under a microscope. Apotheker and Jako first introduced the microscope to dentistry in1978.[1] Microsurgery has been practiced in Endodontics since 1986. In 1992, Shanelec and Tibbetts presented a continuing education course on periodontal microsurgery at the annual meeting of the American Academy of Periodontology.[2] In the hands of a trained and experienced clinician, microsurgery offers enhanced outcomes not possible with traditional microsurgery.

Principles of Microsurgery

As a treatment philosophy, microsurgery incorporates three important principles: -

1. Improvement of motor skills with increased precision and reduced tremor.

2. An emphasis on passive wound closure with elimination of the gaps and dead spaces

3. Reduce tissue trauma by the application of microsurgical instrumentation and suturing

Microsurgical Triad (Kim et al. 2001)[3]

There are three elements of microsurgery (Figure 1) :

[1-4]Post Graduate Student, [5]Professor and Head, [6]Professor, Department of Periodontics, Government Dental College, Thiruvananthapuram, Kerala, India. Corresponding Author: Dr. Gayathri B S, E-mail: aryangayathrikrishnan26@gmail.com

1. Magnification
2. Illumination
3. Microsurgical instruments

Magnification

Visual acuity is the ability to perceive two closely lying objects separately. Visualization of fine details can be enhanced by increasing the image size of the object as well. Two obvious methods of increasing image size are either by getting closer to the objects or by magnification.

Magnification systems

Basically, there are two types of optical magnification systems available to dentists which include:

A. Loupes

B. Surgical Operating Microscope

LOUPES

The most common magnification system used in dentistry is magnification loupes.[4] Loupes are fundamentally two monocular microscopes, with side-by-side lenses, angled to focus on an object. The magnified image that is formed, has stereoscopic properties that are created by the use of convergent lens systems.

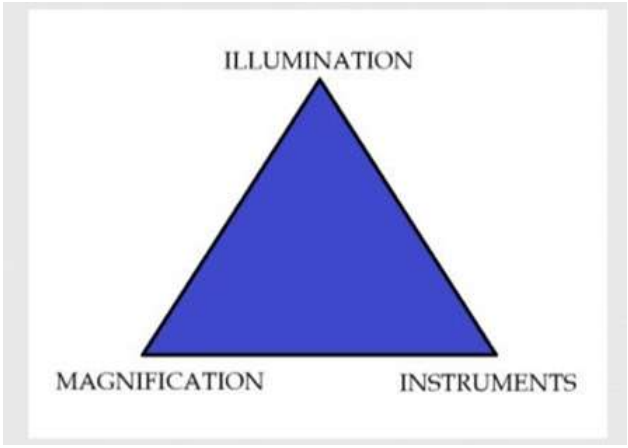

Figure 1 Microsurgical Triad

Three types of loupes are commonly used (Figure 2):

1. Simple loupes.
2. Compound loupes.
3. Prism loupes

Simple loupes

Simple loupes consist of a pair of single, positive, side-by-side meniscus lenses. They are highly subjected to spherical and chromatic aberration, which distorts the image of the object. Because of their size and weight limitations, they have no practical dental application beyond a magnification range of 1.5

Compound loupes

Compound loupes consist of converging multiple lenses with intervening air spaces to gain additional refracting power, magnification, working distance, and depth of field without excessive increase in size and weight. They have better magnification and wider depths of field. But they need an individual light source.

Prism loupes

Prism loupes are the most optically advanced type of loupe magnification presently available. These loupes actually contain Schmidt or roof-top prisms that lengthen the light path through a series of mirror reflections within the loupe. They are superior to other loupes in terms of better magnification, wider depths of field, longer working distances and larger fields of view.[5]

Advantages of loupes

- Less expensive and initially easier to use.
- Loupes also tend to be less cumbersome in the operating field and are less likely to breach

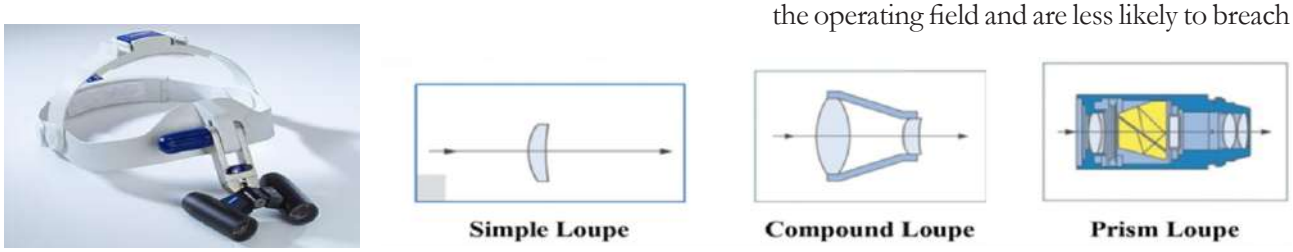

Figure 2 Types of loupes

a clean operative field.

Limitations of loupes

- An individual light source may be required, particularly for magnification in the range of or greater than 4X.
- With loupes, each surface refraction in a lens results in a 4% loss in transmitted light because of reflection. Compound and prism loupes without the protective coating could have as much as a 50% reduction in brightness.
- Discomfort from the heavy weight which has to be borne by the surgeon's nose bridge.
- Higher power magnification often influences posture negatively if the focal length of the magnifiers does not allow the clinician to sit in a normal posture.

SURGICAL OPERATING MICROSCOPE

The operating microscope offers flexibility and comfort superior to magnifying loupes. It is much more expensive and is initially more difficult to use. Surgical microscopes designed for dentistry employ Galilean optics, which have binocular eyepieces joined by offset prisms to establish a parallel optical axis and permit stereoscopic vision without eye convergence or eyestrain.

Optical principles and components of surgical microscope

The surgical microscope is a complicated system of lenses that allows stereoscopic vision at a magnification of approximately 4–40X with an excellent illumination of the working area. In contrast to loupes, the light beams fall parallel onto the retinas of the observer so that no eye convergence is necessary and the demand on the lateral rectus muscles is minimal.

The microscope consists of the optical components, the lighting unit, and a mounting system. The optical unit includes the following components given by Burkhardt & Hürzeler in 2000.[6]

1. Magnification changer
2. Objective lenses
3. Binocular tubes
4. Eyepieces
5. Lighting Unit

To avoid unfavourable vibration of the microscope during use, it should be firmly attached to a wall, ceiling or floor stand.

Magnification changer

The magnification changer or "Galilean" changer consists of one cylinder, into which two Galilean telescope systems (consisting of a convex and concave lens) with various magnification factors are built. These systems can be used in either direction depending on the position of the magnification changer.

Objective Lenses

As processed by a magnification changer, the image is only projected by a single objective. This simultaneously projects light from its source twice for deflection by the prisms into the operation area (i.e. coaxial lighting). The most frequently used objective is 200 mm (focal length, f = 200 mm). The focal length of the objective generally corresponds to the working distance of the object.

Binocular tubes

Depending on the area of use, two different binocular tubes are attached (i.e., straight and inclined tubes). With straight tubes, the view direction is parallel to the microscope axis. Using inclined tubes, an angulation to the microscope axis of 45° is achieved. In dentistry, only inclined, swiveling tubes, that permit continuously adjustable viewing, are feasible for ergonomic reasons. The latest configuration consists of a foldable binocular tube with integrated 360° rotation function. This allows a precise increase or decrease of the working distance and adjusts for eye discrepancies between surgeon and assistant.

Eyepieces

The eyepieces magnify the interim image generated in the binocular tubes. Varying magnifications can be achieved (10x, 12.5x, 16x, 20x) using different eyepieces. Eyepiece selection not only determines the magnification, but also the size of the field of view. Corresponding to the loupe spectacles, an indirect relationship exists between the magnification and the field of view.

Lighting unit

Optimal illumination is necessary with high magnifications. The use of halogen lamps provides a whiter light than lamps using conventional bulbs due to their higher temperature. As halogen lamps emit a considerable portion of their radiation within the infrared spectrum, microscopes are equipped with cold-light mirrors to keep this radiation from the operation area. An alternative to the halogen light is the xenon lamp that functions for up to ten times longer than the halogen lamp. The light emitted has daylight characteristics with an even whiter color and delivers a brighter, more authentic image with more contrast.

Limitations of operating microscope

- Restricted area of vision and loss of depth.
- Loss of visual reference points.
- A steep learning curves.
- Expensive to buy

Classification of Microsurgical Instruments (Figure 4)

Micro surgical instruments[7] (Figure 4):

1. Microneedle holder
2. Microforceps
3. Microscissors
4. Periodontal instruments
   - Knives and scalpel blades
   - Retractors and elevator
   - Micro needle and micro sutures
   - Micro scalpel holder

Knives and scalpel blades: The knives (Figure 5) most commonly used in periodontal microsurgery are those used in ophthalmic surgery or plastic surgery:

1. Blade Breaker Knife
2. Crescent Knife
3. Mini crescent Knife

Figure 3 Optical components of microscope

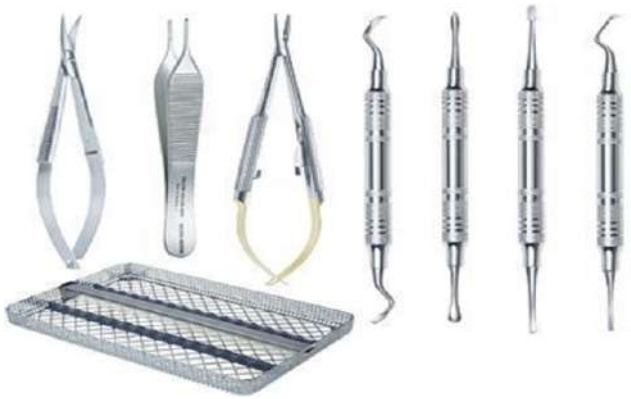

Figure 4 Microsurgical instruments

Figure 5 Periodontal microsurgical knives: 1. Blade breaker; 2. crescent; 3. minicrescent; 4. 260⁰ spoon; 5. lamella and 6. sclera.

4. Spoon Knife
5. Lamellar Knife
6. Sclera Knife

Microsurgical Suturing

Suturing techniques in microsurgery differ from conventional surgery. There is a specific geometry of the suturing required, with the following six well-defined components. (Shanelec DA et al, 1995)

- Angle of entry and angle of exit -The needle should penetrate the tissue at a 90-degree angle, perpendicular to the tissue surface. Passing the needle at an oblique angle cause tearing when the knot is tied.
- Bite size - Proper bite size is between 1 and 1.5 times the tissue thickness. Too small a bite size can tear the wound edges.[8] Too large a bite size can cause under riding or overriding of wound edges. Both outcomes result in impaired healing and unsightly scarring.
- Direction of passage - Once the needle has penetrated one edge of the wound, it must exit the opposing side of the wound with a direction of passage perpendicular to the incision line. This directs the suture force vectors at 90 degrees and prevents lateral dislocation at the wound edge.
- Tension - Suturing should be accomplished with minimal tension. Gentle passage of the suture, in a perpendicular direction of passage, sets the stage for a microsurgical knot that does not cause tissue strangulation through ligation.
- Symmetry - The distance between the bite sizes on either side of the wound edge should be the symmetrical as should the distance between sutures.
- Frequency - Smaller suture material and smaller bite size makes it necessary to place more micro sutures at frequent intervals along the wound edge. This supports wound closure and avoids stress breakage of sutures or tissue tearing

Advantages of microsurgery

- Surgical decision making is enhanced because the quality and quantity of visual data reaching the cerebral cortex is increased by a square of the magnification level.
- Ergonomic and body posture advantages also occur when using the surgical microscope.
- Motor skills are enhanced through instruments designed for a precision grip of the hand.
- An important aspect of periodontal microsurgery is technical improvement in surgical performance.
- Incisions can be accurately mapped, flaps elevated with minimal damage, and wounds closed accurately without tension.
- The resulting appearance of microsurgery is superior to that of conventional surgery.
- Improved view of root surfaces, which permits more definitive removal of calculus and improved smoothness of the root.[9]

Ergonomics

Ergonomic benefit is a significant aspect of microscope use. Issues such as neuromuscular fatigue and occupational skeletal pathology are reduced. Sitting comfort, good body posture, arm support, and controlled breathing are inherent to proper microscope use. Motor skills are enhanced through instruments designed for a precision grip of the hand.[10,11]

Wound healing in periodontal microsurgery

Microsurgery encourages repair through primary healing, which is rapid and requires less formation of granulation or scar tissue. Wound healing studies show anastomosis of microsurgical wounds within 48 hours.

To achieve optimal wound healing, three basic microsurgical principles are needed:

1. Precision tailoring
2. Delicate tissue manipulation
3. Passive primary wound closure

Precise Tailoring

An important feature of microsurgery is the ability to create clean incisions. The design of incisions has significant impact on how the wound edges fit together. A fundamental principle of microsurgery is making incisions perpendicular to the tissue surface,

creating butt joint edges that easily approximate for wound edge stability and maintain blood supply patency.

Delicate Tissue Manipulation

Gentle handling is necessary to reduce cellular injury and subsequent necrosis and inflammation. It is also necessary to maintain tissue hydration and color to maximize healing.

Passive Primary Wound Closure

Specifically designed microsurgical instruments allows a more accurate and atraumatic manipulation of the soft and hard tissues, improves the surgical access and avoids unnecessary removal of tissues, optimizes the defect debridement and the root instrumentation, improves vascularization, and enhances the mobility of flaps and, hence, the possibility of obtaining primary wound closure.

Microsurgical approach may improve the predictability of different periodontal procedures, provide better esthetic results, and cause less postoperative discomfort. The endpoint visual appearance of the typical microsurgical procedure is simply far superior to the end-point appearance of conventional surgery.

Microsurgical Knots

Two basic knots are employed in microsurgery. The square knot, or reef knot, is composed of two single loops thrown in opposite directions. The reef knot has more internal stability than any other knot.[12] If, in tying a knot, both loops are thrown in opposite directions, it becomes the granny knot and unties easily. The English surgeon's knot is composed of two double loops thrown in opposite directions. The first double throw is less likely to loosen when performing the second throw, making it is easier to control tissue apposition.[13] The English surgeon's knot requires a larger bite size. Both knots are tied with instruments.

Microsurgical Indications in Periodontal Surgery

Applications in periodontal flap surgery

With the help of microsurgical techniques, periodontal flap margins can be elevated with uniform thickness. This facilitates precise adaptation of the tissue to the teeth, thus eliminating the gaps and dead spaces circumventing the need for new tissue formation and enhancing periodontal regeneration. Perumal in 2016[14] conducted a randomized controlled trial (RCT) to compare microsurgery and conventional open flap debridement procedures. At 3, 6 and 9 months postoperatively gingival margin level and gingival recession increased in both the groups, but it was not statistically significant. Early healing index score of 1 was found in 85% of test sites and 28% of control sites. The mean pain scale was 0 in test site and 1.07 ± 0.75 in control site.

Studies with enamel matrix proteins by Andrade et al in 2010[15] have shown that enamel matrix derivative could exert better biologic activity in micro surgically treated sites because of reduced tissue trauma and vessel injury to improve vascularization and achieve primary wound closure, which allows optimal retention of enamel matrix derivatives.

Root surface debridement

The primary goals of periodontal surgery include visual access to the root surface for plaque and calculus removal and for removing pathologically altered tooth structures. Magnification greatly improves the surgeon's ability to create a clean, smooth root surface.[16,17] Magnification permits preparation of hard and soft tissue wound surfaces so that they can be joined together according to the accepted microsurgical principle of butt-joint wound approximation. This encourages primary wound healing and enhanced periodontal reconstruction.

Periodontal regeneration

The advantages of microsurgical approach in regenerative therapy relate to improved illumination and magnification of the surgical field that permits proper access to and debridement of the intrabony defect with an increased accuracy and minimal trauma. Isolated interproximal defects that are usually limited to interproximal site are considered ideal for bone grafting with minimally invasive periodontal surgery. Minimal marginal tissue recession and thus improved esthetics and a very limited intra and postoperative morbidity, thereby high patient acceptance and satisfaction, are the other advantages of microsurgical approach for the treatment of intrabony defects.[18,19]

Cortellini in 2001[20] conducted a cohort study to evaluate the outcomes of a microsurgical approach in the regenerative therapy of deep intrabony defect. Study population of twenty-six patients with one deep interdental intrabony defect each were treated with guided tissue regeneration and papilla preservation flaps performed with the aid of an operating microscope and microsurgical instruments. Complete primary closure of the interdental space was achieved in all treated defects and was maintained in 92.3% of cases for the entire healing period. Associated gains in clinical attachment level (CAL) ± were 5.4 ± 1.2 mm on average, corresponding to a CAL gain of 82.8 ± 14.7% of the initial intrabony component of the defect. Average pocket depth reduction was 5.8 ± 1.4 mm and was associated with minimal increase in gingival recession (0.4 ± 0.7 mm).

Application in recession coverage

Periodontal microsurgery has proven to be an effective means of improving the predictability of gingival transplantation procedures used in treating recession with less operative trauma and discomfort. The coronal displacement of the flaps over the defects was found to be easier and had less tension with the microsurgical technique, which facilitates healing and return of the mucogingival line to its original position. Francetti et al in 2005[21] conducted a controlled clinical study for microsurgical treatment in twenty-four cases of gingival recession (depth 2 to 5 mm) in twenty-four patients. Although the outcomes of the test group always showed a major improvement over the controls, no significant differences could be detected between test and control groups. Mean defect coverage at twelve months was 86% and 78% for test and control groups, respectively; complete coverage was achieved in 58.3% and 33.4% of cases, respectively.

A study conducted by Andrade in 2010[15] in which the comparison between the macro and microsurgical techniques was done for the root coverage using the coronally repositioned flap associated with enamel matrix derivative. It was found that there was a significant reduction in the gingival recession height in both the surgical techniques, which were performed. However, the use of microsurgical technique depicted a greater increase in width of keratinized tissue and thickness of keratinized tissue as compared to the macro- surgical techniques performed. Concerning the coverage of mucosal recessions, a comparison between the two approaches (microsurgery and macrosurgery) has been performed in a randomized controlled clinical trial.[6] It was concluded that a microsurgical approach substantially improved the vascularization of the grafts and the percentages of root coverage compared with applying a conventional macroscopic approach. Patient satisfaction about esthetic improvement was reported by Bittencourt 2012[22], that was collected through a questionnaire. All patients in microsurgery group were satisfied from the gingival appearance, compared with 79.1% in the conventional surgery group.

Interdental Papilla Reconstruction

Microsurgical techniques have been developed to replace the lost interdental papilla, which can create phonetic problems, saliva bubbles, and cosmetic deficiencies. A papillary deficiency can be created through iatrogenic surgical removal, as part of tissue collapse following extraction, with periodontal pocket elimination surgery and with periodontal bone loss. Success in the treatment of black triangle with periodontal microsurgery is a significant leap in the field of perio-aesthetics, making it a realistic possibility.

Implant Therapy

Different stages of implant treatment ranging from implant placement to implant recovery and peri implantitis management may be accomplished with more precision under magnification.[23] Microsurgery is associated with enhanced soft tissue procedures and fine suturing. The ability to discern minute dimensional differences permits implant osteotomy preparations centered exactly between reference points such as adjacent teeth, adjacent implants, or buccal and lingual ridge anatomy. The microscope allows immediate detection of subtle changes in drill position and enhanced angular perception so appropriate feedback corrections can be applied.[24]

Sinus Lift

One of the novel applications of microsurgery is in the sinus lift procedure with a success rate of 97%.[25,26] The surgical microscope can aid indirect visualization of the sinus membrane and minimizes the risk of perforations.

Future Perspectives

HDTV Single Camera 3D System

This system involves a three-dimensional (3D) High-Definition Television attached to a stereoscopic microscope which enables 3D visualization and documentation.[27] Viewing of the same stereoscopic vision by assistants and students proves to be advantageous to both clinicians and academicians. It also allows easy printing, organization of data base and adaption to tele-operations.

TOMS-Three-Dimensional On-Screen Microsurgery System

Current advances in video technology permit visualization of the (micro) surgical field on a video monitor three dimensionally without necessitating physical viewing through the microscope.[28] The assembly of the three dimensional on-screen microsurgery system comprises of two single chip video camera mounted on custom fit eyepiece adapters, a dual camera-controller, a view/record image processor, a videocassette recorder (VCR) for optional recording, digital monitor to enable viewing, synchronizing signal emitter and 120 Mega Hertz shutter glasses .

Drawbacks of Microsurgery

- It is much more demanding and technique sensitive and the cost incurred to establish a microsurgical set-up is also high.
- Magnification systems used also pose some difficulties including restricted area of vision, loss of depth of field as magnification increases and loss of visual reference points.
- An experienced team approach mandates microsurgery and is time consuming to develop. Physiologic tremor control for finer movements intra-operatively and a steep learning curve are required for clinical proficiency

Conclusion

Microsurgery has made a great difference in all surgical fields due to its desirable qualities of obtaining cleaner incisions, reduced hemorrhage, reduced trauma at the surgical site and closer wound apposition. Since the surgical procedure is less traumatic and less invasive, healing occurs by primary intention which is rapid with minimal granulation tissue or scar tissue. Optical magnification therefore broadened horizons of dentistry in general and Periodontics in particular. Improvement on visual acuity made possible through optical magnification has become an integral part of modern dental practices.

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Ligaplants: A Stemming Technology in Implants

S Saisuruthi[1], N Gautham Kumar[2], Nivetha R[3], C S Krishnan[4], N Gowrishankar[5]

ABSTRACT

Periodontitis, inflammation of the hard and soft tissues surrounding the tooth, if left untreated, can result in tooth loss. Implant-based tooth replacement has grown in popularity among patients. The introduction of periodontal tissue engineering has revolutionised implant dentistry as a whole, as well as the field of Periodontology specifically. The creation of a periodontal ligament (PDL) attachment around dental implants has recently emerged as a critical new therapeutic option for tooth replacement. PDL is the home of a number of essential cells that are crucial to the dynamic interaction between the tooth and the bone. Ligaplants are now a viable solution to enhance biological performance and increase prosthesis longevity. This review article emphasises the benefits of periodontal ligament coupled dental implants over conventional implants.

Keywords: Ligaplants, implants, tissue engineering, periodontal ligament, osseointegration

Introduction

The multifactorial disease, periodontitis affects 80% of adults, out of which 50% experience early tooth loss, and changes in the tooth supporting structure.[1] Periodontal therapy tends to regenerate the lost structure, especially in cases of severe destruction where the attachment of the tooth has been compromised.[2] The renewal of periodontal ligament fibres and their implantation on the root surface might lead to new attachment.[3] In order to replace missing teeth, the idea of periodontal ligament regeneration is being used on the surface of dental implants.

How do Ligaplants Work?

Since periodontal ligament has the ability to regenerate, a recent development in dentistry known as a "ligaplant" uses tissue-engineered periodontal ligament cells to build a surface that mimics the appearance of a naturaltooth.In contrast to traditional implants, the "ligaplant" combines periodontal ligament cells and implant biomaterial, resulting in the formation of periodontal ligaments that improve the quality of the forces distributed among the tooth abutments and prosthetics supported by the implants.[4]

Advent of Implant

Because of its remarkable long-term clinical survival rate, osseoinegrated implants are currently regarded as the most acceptable implants. The close bone-to-implant contact, or osseointegration, is thought to be the key to optimum healing around implants.[5] Albrektsson et al.[6] defined osseointegration as the direct, microscopic interaction between a living bone and implant. This indicates that without the assistance of the periodontal ligament (PDL), the implants are ankylosed to the bone in a functional manner.

[1,3] Postgraduate student, [2] Professor & Head, [4] Professor, [5] Reader, Department of Periodontics, Madha Dental College and Hospital, Chennai, Tamil Nadu. Corresponding Author: Dr. S Saisuruthi, E-mail: sssmdsperio@gmail.com

Importance of Periodontal Ligament

The PDL is the soft, densely vascularized, and fibrous connective tissue component that lies between the tooth's roots and the alveolar socket's interior wall.[5] The PDL primarily performs a supporting role by securing the tooth to the alveolar bone proper in the area. In addition to aiding in the anchoring of teeth, PDL also contributes progenitor cells to the creation and remodelling of alveolar bone[7] and has been proven to have a remarkable ability for regeneration, restoring the innervations and the strength of biomechanical tissue. Tissue engineering of the PDL is consideredas a new method for replacing missing teeth. The dental implants in this act like a natural tooth because tissue-engineered PDL cells are generated on them. Ligaplants is the name for this new area of implant dentistry.

Tissue Engineering: Foundation of Regeneration

This is an interdisciplinary area that combines engineering and life science principles for the creation of biological substitutes that restore, maintain, or enhance tissue function, according to Langer and Vacanti.[8] According to studies, using tissue engineering in conjunction with an osteoconductive matrix enhances the positive effects of these materials by quickening cellular ingrowth and revascularization of the wound site.

The process of regeneration involves the integration of new transverse fibres into an additional layer of cementum that is connected to the old cementum of the tooth root. The integration of a well attached PDL with the ability to promote the regeneration of nearby alveolar bone would then be possible if a fresh cementum layer were to be applied to the manufactured device's surface. Sonoyama et al.'s[9] demonstration of the feasibility of creating the whole root/periodontal complex by implanting a hydroxyapatite/tricalcium phosphate block coated with PDL-derived mesenchymal stromal cells into the tooth sockets of minipigs provided evidence in support of this concept.In order to increase the success of implant treatment, there has recently been a lot of scientific study and development in implant design, geometry, materials, and methods.

Ligaplants' Advent

During extraction, PDL cells are lost along with natural teeth. Therefore, these cells are unable to take part in the healing of wounds around endosseous implants that are placed to replace missing teeth. Dental implants have a good survival rate, yetfailures do howeverhappen, because any inflammation in their vicinity may result in more severe bone loss than inflammation in the vicinity of natural teeth with PDL. Osseointegrated implants are surrounded by localised bone loss, which is a significant clinical problem.[3] Hence,the goal of implant dentistry has now shifted from not only achieving osseointegration, to the preservation and reduction of peri-implant hard- and soft-tissue loss.[4] As a result, the concept of and development for an implant with a PDL have been considered. Although attempts have been made for years to make up for this obvious difference by "shock-absorbing systems" built into the implant or its superstructure, osseointegrated implants are "ankylosed" and do not have the same mobility as natural teeth with a PDL. If an implant with a PDL could be developed, Ligaplants, a combination of PDL cells with implant biomaterial, could be used to achieve this.[4]

Historical Context

Numerous studies have been conducted in order to create a bio-root that would create the optimum conditions for future implant-supported therapies, i.e., for the growth of a potential periodontal ligament around an implant.

The periodontal ligament and its cells have the ability to re-establish the connective tissue attachment, according to Nyman et al.[11] in 1982. The regeneration potential of periodontal ligament-derived cells was further supported by Nunez et al. in year 2012 in their main investigation.

In a 1990 study, Buseretal discovered that the periodontal ligament cells might be a source of regeneration since they can cover the surface of dental implants while they're forming. When dental implants were positioned close to tooth roots in a number of in vivo tests, cementum-like tissue and an intervening periodontal ligament were shown to form by Buser et al, Caiazza et al, and Warrier et al.[24]

In fact, a growing body of research is validating the significant potential of the in vivo formation of ligamentous attachments to the biomaterials. Park et al. have demonstrated this potential of customised periodontal biomimetic hybrid scaffolds for engineering human tooth-ligament interfaces to be used clinically.[11]

The ability of various tooth bud-forming cells to stimulate the development of dental hard tissues and PDL around titanium implants was proven.

By reusing periodontal ligament-derived cells in an animal study, Takata et al[10] investigated whether connective tissue attachment could develop on implant materials. They discovered that while new connective tissue attachment was observed on bioactive materials such as bio glass and hydroxyapatite, little or no cementum deposition was observed on bio inert materials such as titanium alloy and partially stabilised zirconium.

In order to show that cultured periodontal ligament cells can produce tissue that resembles a true periodontal ligament around implants, Choi, (2000) implanted implants containing cultured autologous periodontal ligament cells in the canine mandibles.[16] Histological examination after three months of healing revealed that some implant surfaces had developed a layer of cementum-like tissue with collagen fibres inserted there.[11]

Properties of Ligaplants

It performs the role of a shock absorber by allowing the tooth to move within the socket. Furthermore, it offers proprioception. The PDL interacts with the neighbouring bone in a significant way as the periosteum on the side of the bone that faces the root. It contains essential cells such undifferentiated mesenchymal stem cells, osteoclasts, osteoblasts, fibroblasts, cementoblasts, and cementoclasts. Each of these cells plays a vital role in the dynamic interaction between the bone and the tooth.[12]

Advantages

1. It resolves issues including gingival recession and bone abnormalities caused by missing teeth.
2. Simulates the natural placement of tooth roots in the alveolar process.
3. Although initially fitted loosely to spare PDL cell cushion, Ligaplants become securely integrated without interlocking and without direct bone contact.[13]

Disadvantages

1. Care should be taken when cultivating ligaplants. i.e., the temperature, the cells used for culturing, how long it takes etc. The ligaplants may fail if a problem arises during the culturing process because other non-periodontal cells may form.
2. Implant failure may occur as a result of unpredictability in the parameters influencing the host's ability to accept the implant or the growth of PDL in the socket.
3. Long-term cell culture may encourage the development of non-PDL cell types.[14]

Are the Ligaplants Successful?

Site-specific signalling, which in turn is mediated by an anatomic code encoded in the expression patterns of homeogene-coded transcription factors, is necessary for the establishment of a regenerative PDL. Thus, the homeoproteins regulate the expression of the homeogene, which establishes cell identities in accordance with the anatomic site and tissue type, as well as the production of cell surface and signalling components and signals from the cell surface feedback.[15]

Implant and Periodontal Ligament Tissue Interface

Site-specific signalling, which in turn is mediated by an anatomic code encoded in the expression patterns of homeogene-coded transcription factors, is necessary for the establishment of a regenerative PDL. The synthesis of cell surface and signalling components is thereby influenced by homeoproteins, and signals from cell surface feedback are used to modify the expression of homeogene, which allows cell identities to be determined based on anatomical site and tissue type. In reality, homeogene Msx2 has been taken into account for separating mineralized bone from non-mineralized PDL. Asporin, asmall leucine rich proteoglycans protein found in the extracellular matrix, has been given a function in the suppression of PDL mineral production.[16]

Preparation

In vitro technology is used to produce laboratory-made tissues. With the aid of signalling molecules, the cells are cultivated on biodegradable scaffolds or matrix before being transplanted into the body. In contrast, in vivo technique refers to the placement of all the developed important components in a tissue defect where they go through a natural healing process in the body and lead to regeneration.

Manufacturing of Ligaplants

One of the clearest instances of its healing potential is the stimulation of double PDLs during tooth transplantation. The donor tooth is removed and immediately replanted in its original alveolus fourteen days prior to transplantation. The PDL goes through a healing phase that involves cell proliferation and differentiation as a result of this intentional stress. After 14 days, the in vivo cell culture achieves its peak of activity, at which point the tooth transplant can be done with millions of active cells linked to the root of the tooth by fresh Sharpey's fibres.[14] Now,tissue engineering techniques can be applied to a comparable cell culture surrounding an artificial root employing this model in its biology and therapeutic aspects.

1) Creation of culture dishes that react to temperature

On polystyrene culture dishes, N-isopropylacylamide monomer in 2-propanol solution is used. After that, an Area Beam Electron Processing System irradiated the dishes with an electron beam. After being rinsed in cold water to remove any remaining ungrafted monomer, the temperature-responsive polymer-grafted (poly isopropylacylamide) dishes are sterilised with ethylene oxide.[16]

2) Cells and cell culture

From an extracted tooth, human periodontal ligament cells are recovered. Following extraction, a scalpel blade is used to remove periodontal tissue from the middle part of the root. The collected tissues are put into culture jars with Dulbecco's modified Eagle's minimal essential medium, 10% foetal bovine serum, and 100 units/mL of penicillin streptomycin, along with other supplements. These outgrowth cellsarethen grown for 48 hours at 37°C in a humidified atmosphere with 5% CO2 to enable cell attachment to the dishes. The medium is changed three times each week, and the plates are cleansed to remove contaminants. Human periodontal ligament cells are plated on temperature-responsive culture to collect the cell sheet. Dishes (35 mm in diameter) with 1x105 cells are grown at 37°C in a medium that serves as an osteodifferentiation medium and contains 50 mg/mL ascorbic acid 2-phosphate, 10 nM dexamethasone, and 10 nM -glycerophosphate.[14,16]

3) PDL cell culture in a bioreactor

A titanium pin with a hydroxyapatite (HAP) coating is inserted into a hollow plastic cylinder with a 3-mm space around it. The gap is continuously flooded with culture medium. Human single cell suspensions are first seeded into plastic tubes with a growth media flow for 18 days.[14,16]

Ligaplant Risk Factors

The bioreactor has been designed to imitate the PDL scenario during cell growth; cells are positioned in a small area between the ligaplants and surrounding hollow cylinder in order to sustain the cell differentiation state and to achieve enough cell stimulation. Thus, it was anticipated that the PDL phenotype would be preferred, suggesting a close bond between the cells and the implant. Therefore, to create successful ligaplants that significantly improve the implant system, the preparation of the ligaplants should have minute mechanical movements of medium flow, ideal spacing between the implants and the culture, and optimal surface treatment duration.[14]

New Research on Ligaplants

In 2005, the impact of residual PDL and the viability of the PDL around a dental implant were also investigated by Parlar et al. and Jahangir et al.The ability of the anatomical substitute to restore lost physiological activities, as well as its mechanical and biological capabilities, are key factors in the occurrence of a tooth being replaced by a dental implant.[25-27] L. Carvalho et al. in 2006 assessed the equivalent dynamic stiffness of the ligament structure to determine the impacts of PDL on the dynamic load transfer mechanism from the tooth to the alveolar bone.[17] A fresh pig mandible with a tooth was employed in this investigation, and an experimental process was run to determine the system's dynamic transmissibility. A better dental implant system can be designed with the help of the knowledge on the PDL's stiffness and

damping offered by the transmissibility function.[17]

Another study by Mareiin 2009 demonstrated that undifferentiated mesenchymal stem cells were capable of differentiating to provide the three essential tissues needed for periodontal tissue by implanting a titanium fixture with a porous hollow root-form poly (DL-Lactide-co-Glycolide) scaffold seeded with autologous bone marrow-derived mesenchymal stem cells in goats. Both at ten days and after one month, periodontium-like tissue with newly formed bone.[18]

In order to promote the formation of implant-ligament biological interfaces, or ligaplants, which are capable of genuine and functional loading, Galt et al. demonstrated tissue engineering of the periodontal ligament and cementum-like structures on oral implants in people for the first time in 2010.[14] According to Gault's research, it is possible to harvest periodontal ligament fibroblasts from mature hopeless teeth and cultivate them in bioreactors while maintaining their differentiation condition. Eight implants were placed in all, and one of them remained in place and continued to operate even after five years, as well as demonstrating significant bone regeneration in the nearby bone defect two years following implantation. Thus, it can be inferred that in the future, using ligaplants in a therapeutic setting may also be able to assist in avoiding bone grafting and the expense, inconvenience, and discomfort that it causes the patient.[14]

By conducting an animal investigation, Rinaldi and Arana Chavez in 2010 demonstrated results showing that the PDL in touch with the implant caused a thin cement-like layer to form more slowly after implantation.[28] Javier Caton et al.in 2010 examined the clonogenic potential of human dental and periodontal tissues, such as the dental pulp and the PDL, as well as their potential for tooth and periodontal repair and/or regeneration.They also suggested cutting-edge therapy strategies that use stem cells or progenitor cells and are intended to replace lost periodontal or dental tissue.[19] Kano et al. in 2012 proposed that implants surrounded by tissue similar to periodontal ligament could develop when tooth-shaped titanium implants with hydroxyl-apatite coatings were immediately placed into the tooth socket where some periodontal ligament still existed; maintenance of original periodontal tissue domains was most likely the cause of the prevention of the implants' osseointegration.[20] Due to its ability to regenerate periodontal tissue, ligaplants for tooth replacement offer clear advantages over osseointegration devices, according to Kiong and Arjun Kumar in 2014.[29]

The capacity of PDL-derived cells cultivated with osteoinductive media to generate cementum production was proven by Kaorowashio et al. in 2018. An environment resembling the one found surrounding a normal tooth developed around a titanium implant, resembling periodontium. When used in a clinical setting, dental implants with a cell sheet approach may prove to be a workable alternative to traditional implant therapy. Additionally, the use of this approach may play a cutting-edge role not only in the field of periodontics but also in areas of dentistry like prosthetics and orthodontics.[21]

One of the most significant topics in the scientific field has been the occurrence of the regeneration of the lost periodontal tissues. Since their discovery, periodontal ligament stem cells (PDLSCs) have been used by Kengo Iwasaki et al. in 2019 to explore the regenerative capacity of periodontal bone abnormalities. Using periodontal ligament stem cell sheets, which were created through cell sheet engineering in animal models and are currently the subject of clinical human trials, periodontal deficiencies were successfully repaired.[22]

Conclusion

The advent of periodontal tissue engineering has revolutionised implant dentistry. To address the drawbacks of the traditional implants, ligaplants have recently been introduced based on the modality. The majority of studies on ligaplants conducted on animals have yielded positive results, but it is still necessary to provide a more practical and attainable method of obtaining periodontal ligament linked implants. Additionally, more human studies must be conducted to assess the effectiveness of this technique. Due to ligaplants' advantages over traditional implants, they may become a new, ground-breaking component of implant dentistry.

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Address for communication

Dr Shahana C Mohamed, Editor JSPIK, Assistant Professor, Department of Periodontics, Government Dental College, Medical College P. O, Thiruvananthapuram - 695 011, Kerala, India. E-mail: editorspik@gmail.com