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President's message

Dear members of the SPIK family,

The Indian Society of Periodontology [ISP] has now been renamed as the Indian Society of Periodontology and Implantology [ISPI]. This was a change that was long overdue. The field of Implantology has been usurped by many specialities like Oral Surgery and Prosthodontics. But to me, the best person to place an implant and care for it - is a Periodontist. We have so many Periodontists who are skilled Implantologists, which I believe is due to a combination of factors.

1. **Our Training:** As we focus on the supporting tissues of the tooth, Periodontists are the most adept at the evaluation of soft tissues and alveolar tissues especially in patients with periodontal issues.
2. **Tissue handling:** Periodontists are the best at handling soft tissues
3. **Bone grafting and regeneration:** Periodontists routinely do a variety of regenerative procedures as part of their clinical practice, therefore graft placement during implant placement is easy for them. Soft tissue augmentation around an implant is a domain for us.
4. **Long-term maintenance:** As Periodontist focus on periodontal health, they are well-suited for implant maintenance and if issues occur, manage peri-implant disease

Therefore, I can conclude by saying that Periodontists do Implants better than other specialities. Hope my other speciality friends don't see this write up!!

Regards,

Dr Arun Sadasivan
President -SPIK
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Secretary's Message

Dear esteemed readers,

As we bid farewell to another year and welcome the final edition of our journal of this year ,it's a pleasure to reflect on a remarkable year. The past 12 months have showcased resilience and teamwork. I'm grateful to our contributors, editorial team, and readers for making our journal a success.

Celebrating Christmas and stepping into the new year, let's embrace new opportunities, fresh perspectives, and growth. May it bring joy, prosperity, and inspiring moments.

Wishing you a Merry Christmas, happy, healthy, and successful new year!

Dr. Deepak Thomas
Secretary, SPIK

Editorial

Dear Members and Colleagues,

It gives me immense pleasure to address you through this issue of the Journal of the Society of Periodontology and Implantology – Kerala (JSPIK). A professional journal is not merely a collection of articles; it is the academic voice of our society. It reflects our scientific growth, clinical expertise, research aptitude, and collective commitment to advancing periodontal and implant care. Through the exchange of evidence-based knowledge and innovative ideas, the journal continues to strengthen our specialty and inspire excellence among clinicians, academicians, and students alike.

The recently conducted mid-term conference at Mahe Dental College stands as a testament to our unity, enthusiasm, and dedication to continuous learning. Periodontists today play a pivotal role not only in preserving oral health but also in promoting overall systemic well-being. With increasing awareness of the oral–systemic link, our responsibility towards society extends beyond clinics and classrooms. From prevention and early intervention to advanced regenerative and implant therapies, we contribute significantly to enhancing patients' quality of life. As specialists, educators, and researchers, we must continue to uphold ethical practice, encourage research, and reach out to the community through awareness and service initiatives.

I sincerely appreciate the contributions of our authors, reviewers, editorial team, and society members who consistently support the growth of JSPIK. Let us continue to work together to elevate the standards of our journal and our specialty.

Wishing everyone continued academic excellence and professional fulfilment.

Warm regards,

Dr Anjhana Narayanan
Editor, JSPIK

JSPIK

Smart Dentistry: Harnessing Artificial Intelligence in Contemporary Periodontal Practice: A Narrative Review

Anil Melath[1], Kamali T.[2], Subair K.[3], Jilu Abraham[4], Sukesh AK[5], Arjun Manoj[6]

ABSTRACT

Artificial intelligence (AI) is increasingly influencing contemporary periodontal practice by improving diagnostic accuracy, refining treatment planning, and supporting personalized patient care. By leveraging machine learning, deep learning, and natural language processing techniques, AI systems are capable of analyzing complex datasets—including radiographic images, periodontal measurements, biological markers, and patient records—with high precision. These technologies assist clinicians in early disease identification, risk stratification, and continuous monitoring of periodontal health. The aim of this narrative review is to explore the role of artificial intelligence in contemporary periodontal practice, highlighting its applications in diagnosis, treatment planning, disease prediction, and clinical decision-making, while discussing the drawbacks and future prospects.

Keywords: ANN, CNN, machine learning, deep learning, image analysis, omics, prediction

Introduction

Artificial Intelligence has rapidly emerged as a transformative tool across multiple industries, with dentistry standing out as a field poised for significant advancement[1]. By analyzing diverse clinical inputs ranging from radiographs and photographs to periodontal probing charts, AI supports clinicians in making faster, data-driven clinical decisions while simultaneously improving patient engagement and understanding[2,3]. Neural network-based systems emulate aspects of human cognitive processing, empowering machines to execute tasks previously reliant on expert judgment[4].

Machine learning enables the interpretation of extensive datasets to anticipate clinical outcomes, whereas deep learning algorithms improve diagnostic accuracy through hierarchical feature extraction[5]. Decision-support systems built on expert knowledge and individualized data enhance diagnostic reliability and promote tailored care. Modern imaging systems, including intraoral scanners and Convolutional neural networks (CNN) based tools, have streamlined evaluation workflows. Artificial neural networks (ANNs), which emulate human cognitive functioning, can detect subtle pathological changes often missed during early examinations. As these technologies evolve, they are reshaping dental care into a more efficient, predictive, and patient-focused discipline[6].

Core Pillars of Artificial Intelligence

AI development has historically followed two conceptual pathways: strong AI and weak AI[7]. Strong AI aims to recreate human-level recognition even if it is an artificial system, striving toward machines capable of adaptive reasoning in uncertain environments[8]. In contrast, weak AI centers on task-specific computational models that emulate isolated cognitive processes without replicating human intelligence as a whole. This approach accepts that artificial and human cognition differ fundamentally but asserts that functional performance does not require full cognitive mimicry[7].

Weak AI, which underpins most contemporary

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AI applications, leverages data-driven learning and adaptive problem-solving[7]. Bio-inspired neural networks epitomize this paradigm, serving as the conceptual backbone for modern machine-learning and deep-learning architectures[9].

Evolutionary Framework of Modern AI

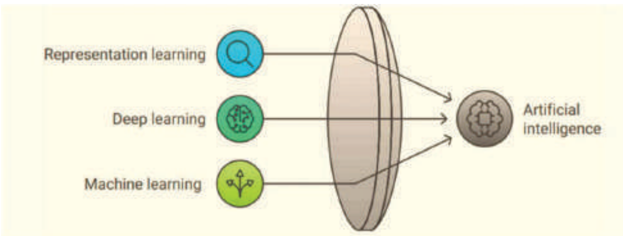

Figure 1 working mechanism of artificial intelligence

Machine learning Figure 1, grew out of efforts to understand human intelligence and sensory processing, inspiring the development of artificial neural networks capable of learning through predefined computational rules[10]. Early innovators recognized that successful machine performance does not depend on replicating biological systems, much like aviation deviated from imitating birds, forming the philosophical foundation for weak AI.

Although strong AI remains an aspirational long-term goal, contemporary progress is overwhelmingly driven by weak AI and cognitive-science principles that integrate psychology, philosophy, and computational theory. Many AI architectures mirror elements of human perception: speech-recognition models parallel language acquisition, while computer-vision systems process visual information analogous to human vision. These advancements illustrate AI's evolution from theoretical inquiry to a sophisticated collection of perceptual and decision-making technologies[11].

Role of AI in Modern Medicine

The integration of AI into healthcare can be traced to pioneering expert-system architectures developed in the 1970s. Seminal programs such as MYCIN, INTERNIST, and CASNET were among the first computational tools designed to support clinical reasoning by organizing and applying specialist knowledge to patient-specific problems. Developed within the Stanford medical informatics initiative, these systems laid the conceptual groundwork for subsequent advances in diagnostic decision-support. Although rudimentary by today's standards, they demonstrated how structured clinical rules and inference engines could meaningfully augment medical judgment and helped shape the evolution of modern medical AI.[12,13]

Despite its rapid expansion, AI remains comparatively young, with much of its foundational progress occurring since the mid-20th century[14]. Today, expert systems remain a cornerstone of medical AI, offering robust frameworks for organizing clinical knowledge[7]. Modern applications include diagnostic support, risk estimation, and predictive modeling, made possible through machine learning, Bayesian inference, and advanced data-mining techniques[15]. These innovations underpin the transition toward intelligent, data-driven healthcare systems[7].

AI-Driven Transformations in Periodontics

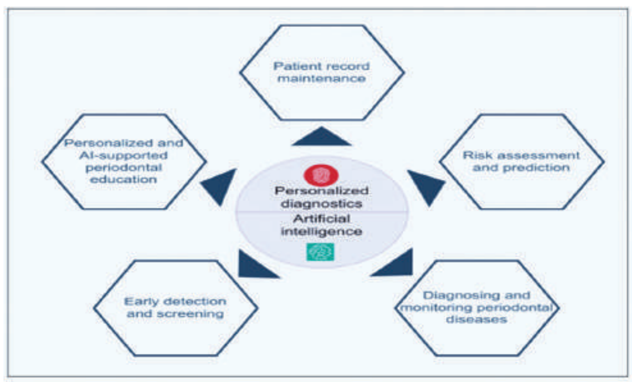

Figure 2 Schematic representation of personalized diagnostics in artificial intelligence

1 Diagnosis of Periodontal Diseases

AI algorithms, particularly CNNs, have demonstrated significant capability in interpreting radiographic and three-dimensional datasets for early detection of periodontal abnormalities[1]. Numerous studies show that AI models can identify periodontal bone loss, stage and grade periodontitis, and classify implant systems with accuracy comparable to or exceeding clinical experts[17]. Automated analysis of intraoral photographs further aids in identifying gingival inflammation, plaque accumulation, and recession[16].

Deep-learning systems have also shown considerable promise in implant diagnostics Figure 2. In a large collaborative investigation, Park and colleagues evaluated radiographic datasets aggregated from multiple dental college hospitals and private clinics to train a neural network capable of identifying a wide range of implant systems. Their model successfully differentiated 25 implant designs with high accuracy and required only a fraction of the time taken by clinicians to complete the same task. This demonstrates the practical value of AI-assisted screening tools in managing the increasing diversity of implant systems encountered in clinical practice[18].

2 Risk Assessment and Predictive Analytics

AI enhances periodontal risk prediction by integrating electronic health records, behavioral data, systemic health indicators, and potentially genomic and multi-omics profiles. Traditional risk calculators are limited by their reliance on clinical and lifestyle factors alone; however, ANN-based models have already achieved strong predictive performance. Incorporating multi- omics layers, such as micro biome and proteomic data, offers promise for highly precise, personalized risk assessment[17].

3 Early Detection and Screening

Periodontal disease is often identified only after irreversible damage occurs, making early detection essential. Noninvasive diagnostic fluids like saliva and GCF, combined with omics-based biomarker discovery, hold potential for AI-enhanced early screening Figure 3. Although current biomarker-driven AI research is limited, emerging studies show strong potential for integrated multi- omic prediction models. Smartphone-based AI tools and intelligent toothbrushes further empower patients in monitoring oral health[17].

4 Personalized Medicine

AI facilitates individualized treatment planning by integrating clinical, genetic, and behavioral data to recommend targeted therapy[17]. Unlike traditional "one-size-fits-all" approaches, precision periodontics relies on patient-specific biomarkers, risk profiles, and pharmaco genomic insights[19]. The principles of P4 medicine—predictive, preventive, personalized, and participatory care—align closely with AI-enhanced periodontal strategies[17].

5 Patient Record Management

Accurate record keeping remains a cornerstone of quality care Figure 2. AI-enabled digital documentation systems enhance accuracy, streamline administrative workflows, and support clinical decision-making by consolidating and analyzing patient data[17].

6 Drug Discovery

AI accelerates drug-development pipelines by modeling chemical structures, predicting biological activity, and identifying therapeutic targets with high speed and efficiency (1).

7 Clinical Decision Support

AI-based decision-support platforms synthesize real-time clinical data and evidence-based knowledge to enhance diagnostic accuracy, reduce errors, and improve workflow efficiency, positioning AI as an essential component of future healthcare systems[1].

8 AI-Enhanced Periodontal Education

Virtual and augmented reality technologies are beginning to reshape periodontal training by offering immersive environments in which learners can practice diagnostic and therapeutic procedures. Early systems, such as Periosim, provided one of the first opportunities to experience tactile feedback while exploring periodontal structures in a simulated setting.

Although the platform enabled trainees to rec-

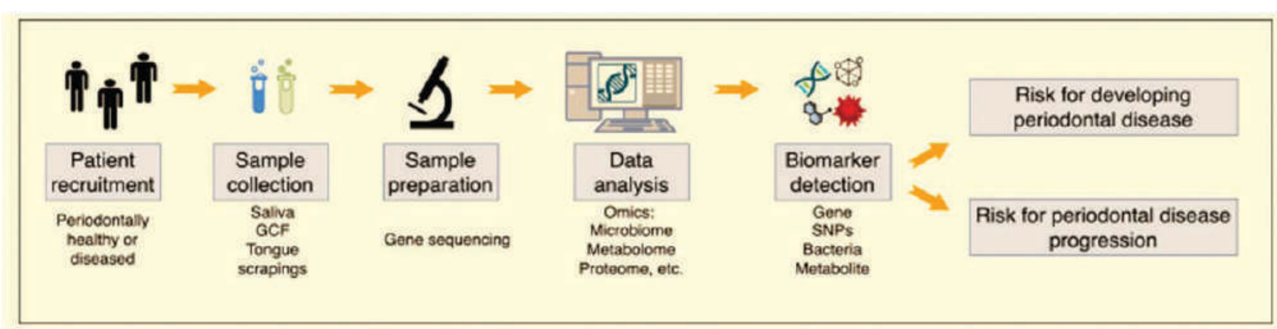

Figure 3 Early detection and screening

ognize calculus and gingival contours, its capacity to quantify clinical findings such as probing depth or furcation involvement was limited. More advanced simulators have since improved force feedback and measurement accuracy, enabling students to develop appropriate probing pressure and refine diagnostic skill under standardized, reproducible conditions[17].

9 Transformative Impact on Periodontal Research

AI is central to advancing personalized periodontal diagnostics. Its success hinges on interdisciplinary collaboration, standardized data-sharing frameworks, and integration of systemic health information such as metabolic markers and inflammatory profiles. AI supports the identification of disease-related biomarkers, modeling of risk trajectories, and validation of novel diagnostic approaches. Collaboration with regulatory bodies and industry partners is essential to ensure ethical use, clinical applicability, and long-term reliability[17].

Methodological Approaches

Gingivitis and periodontitis are prevalent inflammatory diseases with significant implications for both oral and systemic health. Conventional periodontal diagnostic approaches largely depend on subjective clinical evaluations, contributing to variability in diagnosis and treatment outcomes.

1. Subjectivity in Diagnosis:

Conventional periodontal assessments based on probing depths, bleeding on probing, and radiographs are highly operator-dependent, leading to variability and inconsistency in diagnosis[20].

The integration of artificial intelligence (AI) offers a transformative advancement by improving diagnostic accuracy, standardizing assessments, and supporting evidence-based decision-making. Consequently, AI enables more precise treatment planning and personalized periodontal care.

2. Enhanced Diagnostic Accuracy:

Chang HJ, Lee SJ, Yong TH, et al. in 2020 reported a novel hybrid framework that combined deep learning architecture and the conventional CAD approach demonstrated high accuracy of range referenced (e.g., high 80s–90s%), excellent reliability in the automatic diagnosis of periodontal bone loss and staging of periodontitis, significantly reducing clinician-dependent variability[21].

Drawbacks

1. Lack of Standardization:

Despite technological progress, the absence of standardized protocols for data acquisition, model development, training, and validation limits the reproducibility and generalizability of AI applications across diverse clinical environments[22].

2. Data Heterogeneity and Bias:

Variability in datasets and underrepresentation of certain populations in training data may introduce bias, potentially exacerbating existing healthcare disparities[22].

3. Ethical and Legal Concerns:

Issues related to patient data privacy, informed consent, algorithm transparency, and accountability remain significant barriers to widespread clinical adoption of AI[23].

4. Need for Multidisciplinary Collaboration:

Addressing these challenges requires coordinated efforts among researchers, clinicians, policymakers, and regulatory bodies to develop robust standards, ethical frameworks, and governance models that ensure safe, equitable, and effective integration of AI into periodontal care[23].

Conclusion

Artificial intelligence is playing an increasingly significant role in the evolution of modern periodontics by enhancing diagnostic precision, enabling individualized treatment strategies, and improving clinical workflow efficiency. Through the application of machine learning and deep learning techniques, AI supports early disease detection, accurate risk assessment, and personalized therapeutic planning. These advancements strengthen evidence-based clinical decision-making and promote proactive periodontal care. As AI continues to integrate with digital dentistry and tele health platforms, it is poised to advance periodontics toward a comprehensive precision-care model that prioritizes safety, efficiency, and patient-centered outcomes.

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Socket Shield Technique as a Root-to-Ridge Bone Armor in Contemporary Implant Therapy- Review

Nagasurthi K.J.[1], Kadhiresan R.[2], Arunmozhi U.[3], Preetha D.[4]

ABSTRACT

Following tooth extraction, alveolar ridge resorption presents a significant biological challenge in implant dentistry, particularly affecting the buccal plate, which undergoes the most notable dimensional loss. This resorption occurs due to the disruption of the periodontal ligament (PDL) and the subsequent remodeling of the bundle bone. Traditional ridge preservation methods, such as socket grafting and the use of barrier membranes, can help minimize dimensional alterations but are unable to completely prevent ridge collapse since they do not maintain the integrity of the PDL. Partial Extraction Therapies (PET), which keep a portion of the root segment, have been developed to address this issue. Among these, the Socket Shield Technique (SST) enables fast implant placement while protecting buccal bone and peri-implant soft tissues. This review covers the biological foundation of post-extraction alterations, the evolution and rationale for PET and SST, clinical data, indications, differences in SST preparation, surgical protocols, biological integration, benefits, limitations, and future directions in implant therapy.

Keywords: socket-shield, partial-extraction, implant, bone-preservation

Introduction:

Tooth extraction, a common dental treatment, causes predictable biological mechanisms that result in alveolar ridge resorption. The buccal bone plate, which is particularly thin in the maxillary anterior region, is highly vulnerable because it is primarily composed of bundle bone, which relies on the PDL for vascular supply and mechanical stimulation. Removal of the PDL during extraction causes fast bone remodeling, resulting in horizontal and vertical dimensional loss of the alveolar ridges.[1,2,3] which may complicate the placement of dental implants or unesthetic appearance. Traditional ridge preservation procedures like socket shield, root submergence, pontic shield involving bone grafts, allografts, xenografts, and barrier membranes may reduce bone resorption.[4,5] The Socket Shield Technique (SST) is a specialized Partial Extraction Therapy [PET] that has gained popularity due to its ability to maintain ridge dimensions while allowing for quick implant placement.[12,13] This review discusses the various protocols involved in Socket Shield Technique.

Post Extraction Changes: Biological Basis:

Immediately after extraction, the socket forms a blood clot made up of fibrin, platelets, erythrocytes, and inflammatory cells. This clot acts as a scaffold for the migration of cells involved in wound healing. Within the first week, the clot is replaced by granulation tissue made up of proliferating fibroblasts, endothelial cells, and inflammatory cells. Osteoclastic activity on the socket walls starts early, primarily affecting the thin buccal plate.[6]

After about two weeks, the granulation tissue is replaced by a temporary matrix, and early woven bone begins to grow within the socket lumen. Internal bone filling may occur, but it does not prevent exterior ridge

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collapse because loss of PDL support causes bundle bone resorption on the outside socket walls.

Histological and clinical investigations show that the most significant dimensions changes occur within the first three to six months after extraction. Schropp and colleagues found that about half of total horizontal ridge decrease happens within the first three months, with a mean width loss of 3.8 mm, and that approximately two-thirds of total resorption occurred during this time.[7]

Tan et al. and Van der Weijden et al. conducted systematic evaluations of human studies and measured these changes, reporting 29-63% horizontal bone loss and 11-22% vertical bone loss by six months, with minimal subsequent alterations. (8,9) These predictable patterns of resorption highlight the limitations of traditional ridge preservation techniques and emphasize the necessity for physiologically driven strategies to minimize bone loss.

The buccal alveolar bone is primarily made of bundle bone, which contains Sharpey's fibers from the periodontal ligament (PDL) that inserts into it. This biologic unit offers vital vascular supply and mechanical stimulation to keep bone homeostasis. When the PDL is severed after tooth extraction, the bundle bone loses its functional stimulation, resulting in remodeling and resorption.[3]

Emergence of Partial Extraction Therapies and Socket-Shield Technique:

Conventional socket grafting procedures may aid in internal bone fill, but they do not restore the PDL or keep it functionally intact. As a result, ridge collapse occurs even after grafting, especially on the buccal aspect.[3,4] The idea that preserving the PDL itself may be critical to preventing ridge collapse led to the development of PET procedures, such as SST, which attempt to keep a root segment intact with its PDL to support the adjacent alveolar bone and soft tissue.

Partial Extraction Therapy (PET) intentionally retain a root fragment to protect the alveolar ridge. These include root submergence, pontic shielding, proximal shielding, and the socket shield technique (SST). Root submergence is mostly employed beneath pontics to preserve ridge contours, whereas pontic shield attempts to maintain ridge shape and papillae.[1,14]

Hurzeler and colleagues proposed the Socket Shield Technique (SST) in 2010 as a proof-of-principle strategy for fast implant insertion. In their study, a narrow buccal section of the root was kept with its PDL while an implant was put palatally. Histological investigation demonstrated the creation of cementum-like tissue between the implant surface and the residual dentin, with no evidence of inflammation or fibrous encapsulation.[12] This study contradicted the long-held idea that total root removal was required for successful osseointegration, implying that maintaining a portion of the root with an intact PDL might sustain ridge dimensions.

Subsequent clinical investigations and case series have expanded on this concept, revealing that SST can preserve buccal bone and soft tissue shapes while allowing for rapid implant placement with excellent survival rates and better esthetic outcomes.[13,15]

Indications for Socket-Shield Technique:

The primary rationale for SST is to protect the buccal plate by keeping the PDL on the retained root fragment, limiting physiologic remodeling and bundle bone resorption. The retained piece serves as a biological shield to support the buccal ridge, while the implant integrates with bone on the palatal and apical surfaces.[12] This segmented healing system allows dentin, PDL, bone, and implant to coexist in a stable manner.

Clinical indications for SST include[13,15]:

a. Immediate implant placement in sites with intact buccal plate.

b. Areas with a thin buccal bone where dimensional preservation is critical.

c. High esthetic demand, particularly in the anterior maxilla.

d. Cases requiring preservation of papillae and soft tissue contours.

Contraindications include[13]:

a. Active periapical or periodontal infections at the extraction site.

b. Vertical root fractures or ankylosed teeth.

c. Poor oral hygiene or non-compliant patients.

d. Severe bone defects where primary implant stability cannot be achieved.

Types of Socket-Shield Preparations:

SST has been adapted into several variations to suit clinical scenarios and anatomical challenges.[12,14,18] (Figure 1)

i. Buccal Shield: Retention of a buccal root fragment to preserve buccal bone.

ii. Full C Buccal Shield: Retention of buccal and interproximal root walls, useful when adjacent teeth or implants are present.

iii. Partial C Buccal Shield: Retains buccal and one interproximal surface, indicated when one side is edentulous.

iv. Proximal (Interproximal) Shield: Retains a root fragment on the mesial or distal aspect to preserve interproximal bone and papillae.

v. Lingual/Palatal Shield: Retains lingual or palatal root fragments in posterior sites with compromised buccal anatomy.

vi. Multiple Buccal Shields: Used in multirooted teeth to maximize preservation of alveolar volume.

vii. Modified Shields (Root Membrane, T-Profile, Concave Designs): Tailored shields for complex prosthetic or esthetic requirements.

Pre-Surgical Evaluation and Case Selection:

CBCT imaging is useful for evaluating buccal plate thickness (preferably >1 mm), root shape, and closeness to critical tissues. To determine the viability of shield preparation, consider esthetic variables such as soft tissue biotype and smile line, as well as tooth morphology and root structure.[13,14]

Armamentarium:

The armamentarium for SST includes: (Figure 2)

a. CBCT / IOPA radiographs - for assessment of buccal plate and root morphology

b. Local anesthetic syringe with cartridges

c. Periotomes - for atraumatic root fragment separation

d. Long-shank diamond fissure bur - for root sectioning

e. Long-shank round diamond bur - for shield thinning and contouring

f. High-speed handpiece with copious irrigation

g. Surgical curettes / spoon excavator - for removal of palatal root fragment

h. Implant surgical kit (osteotomy drills & drivers)

i. Implant physiodispenser

j. Suture material (4-0/5-0) with needle holder

Surgical Technique:

The surgical technique for SST requires precision and familiarity with atraumatic extraction principles.[12,13,20] (Figure 3)

Steps include:

i. Anesthesia: Local block or infiltration.

ii. Decoronation: Remove the clinical crown at or slightly below the crestal margin.

iii. Root Sectioning: Mesiodistal sectioning of the root using long-shank diamond burs.

iv. Palatal Fragment Removal: Atraumatic removal with periotomes or micro-elevators, preserving the buccal fragment.

v. Shield Shaping: Buccal fragment is reduced to a thickness of 1-1.5 mm and contoured flush with or slightly below the crest.

vi. Osteotomy Preparation: Palatal to the retained shield, avoiding contact with it.

vii. Implant Placement: Implant is placed palatal to the shield with primary stability.

viii. Soft Tissue Management: Flapless or minimal flap approach to preserve blood supply.

ix. Healing: Allow natural clot formation around the implant and shield. Bone grafting between dentin and implant is typically not required.

Healing after SST:

Within 24 hours, clots form in the gaps around the implant and shield. The maintained PDL maintains vascularity, which promotes osteogenic cell migration and angiogenesis. Within the first week, osteoid deposition occurs on implant surfaces that are not facing the shield, while cementoblast-like cells differentiate along the shield's dentin surface. Mineralization consolidates osseointegration after 6-8 weeks, resulting in a physiologically stable contact between dentin and bone, with remodeling continuing without inflammation or fibrous tissue development. Evidence suggests that this compartmentalized healing permits dentin, PDL, and bone to coexist on the implant surface, promoting long-term stability and esthetics.[12]

Rule of 5 Triangles:

SST preserves the buccal bone during immediate implant placement by retaining a portion of the root. Successful outcomes depend on the 5 key factors / "triangles" (Figure 4). This technique minimizes buccal bone resorption, maintains soft tissue contours, and enhances esthetics in the anterior region.

1. Buccal plate presence - ensures structural support.

2. Primary stability - immediate implant anchorage.

3. Implant design - tapered/threaded for optimal fit.

4. Jumping distance - gap between implant and socket wall.

5. Tissue biotype - thick biotype favors predictable healing.

Advantages of Socket-Shield Technique:

SST offers several clinical benefits[13,15]:

a. Preservation of buccal bone and alveolar ridge contour, reducing the need for extensive grafting.

b. Maintenance of peri-implant soft tissue volume, including gingival margin and papillae.

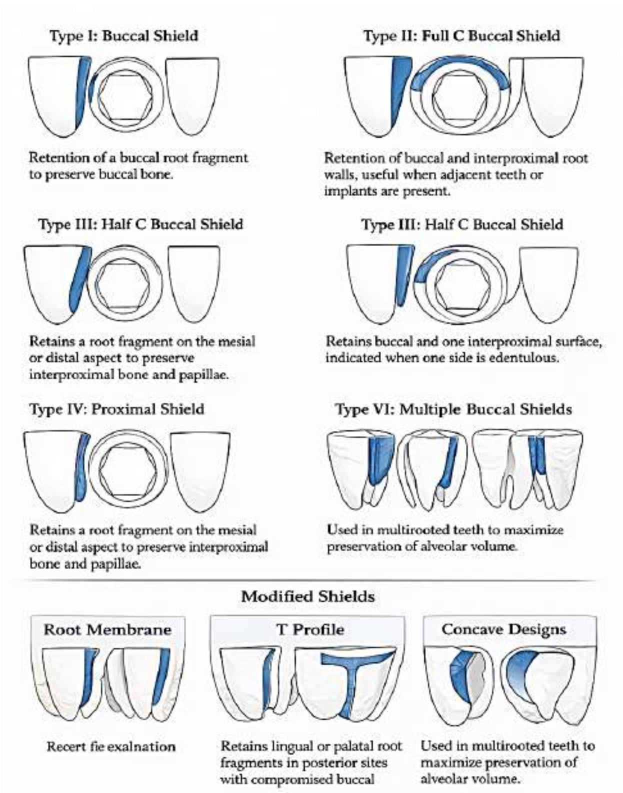

Figure 1

Figure 2

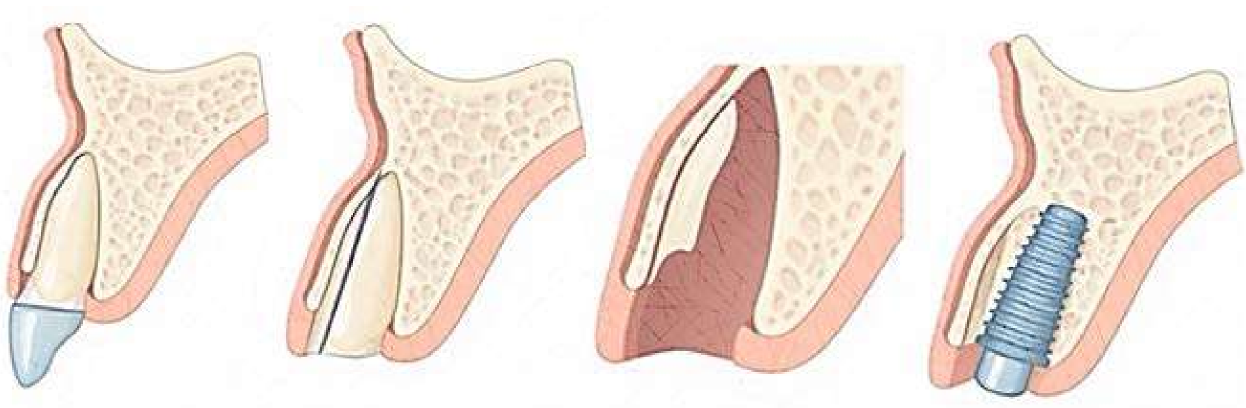

Figure 3

c. Support for immediate implant placement with predictable primary stability.

d. Superior esthetic outcomes, particularly in high-demand anterior cases.

Limitations and Complications:

Despite its advantages, SST has limitations[13,14]:

a. It is technique-sensitive, requiring precise root sectioning and shield preparation.

b. Risks include shield exposure, mobility, or infection if not executed properly.

c. Limited high-level evidence and long-term data exist, particularly in posterior and multirooted sites.

d. Retreatment is technically challenging if complications arise.

DISCUSSION:

Gluckman and Salama's retrospective study in 128 SST cases with a follow up of 4 years found remarkable implant survival rates (>95%) with preserved buccal bone and soft tissue profiles.[15]

Atieh et al., in a systematic review and meta-analysis, observed that SST cases showed minimal dimensional changes in buccal bone and improved esthetic scores, without any increase in implant failure rates or biological complications[13]

Gustavo Avila-Ortiz in 2018 has done a meta-analysis and found that SST improves aesthetic outcomes, particularly in the anterior region. Partial Extraction Therapy research supports the concept of root retention for alveolar preservation. However, despite the favourable outcomes, many research are observational or retrospective in nature, emphasizing the importance of bigger randomized controlled trials to create standardized techniques and better evidence.[3]

Lu et al. (2025) conducted a systematic review and meta-analysis including 27 clinical studies comparing SST with conventional immediate implant placement in the esthetic zone and reported implant survival rates comparable to conventional techniques (approximately 95-98%), along with significantly reduced buccal bone loss and improved pink esthetic scores[21].

Altalhi et al. (2025), in a systematic review and meta-analysis of over 20 clinical studies, observed implant survival rates ranging from 95% to 98% with SST, while also identifying shield exposure as the most commonly reported complication[22].

Wu et al. (2024) reviewed more than 30 human clinical studies on partial extraction therapy and demonstrated that SST-based approaches consistently achieved implant survival rates exceeding 95%, with superior ridge dimensional stability due to preservation of the periodontal ligament[23].

Clinical validation was provided by Dhondt et al. (2025) in a prospective case series of 23 implants placed in 20 patients, followed for 18 months, which showed an implant survival rate of 95.7%, minimal buccal bone loss (approximately 0.3-0.5 mm), and high esthetic outcomes[24].

In addition, Shaheen et al. (2024), through a systematic review, concluded that SST consistently demonstrates implant survival rates greater than 95% with favorable hard- and soft-tissue outcomes, although the technique remains highly technique-sensitive and supported predominantly by observational studies[25].

Future Directions:

Future research should focus on standardized surgical protocols, longer follow-up randomized controlled trials, and the integration of digital planning and guided surgical techniques to improve predictability and clinical acceptance.[13,14,16,19]

Conclusion

The Socket Shield Technique is a biologically grounded breakthrough in implant therapy that preserves hard and soft tissues in partially extracted sites for better esthetics and future implant placement. The

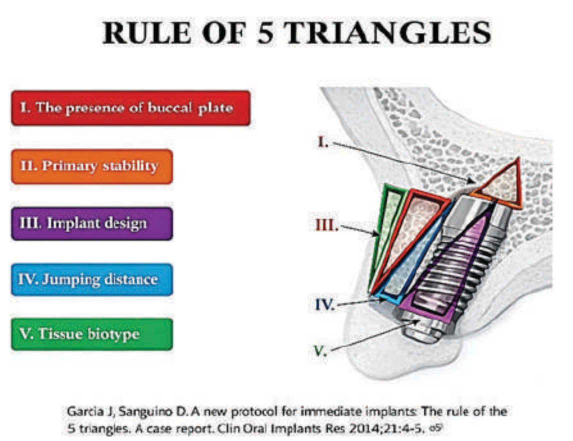

Figure 4

advantage of this technique is preventing the ridge collapse after extraction and maintaining the integrity of the supporting structures around the teeth. Continued research using high-level evidence will help to clarify its position in clinical practice.

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JSPIK

Comparative Evaluation of the Efficacy of Commercially available Mouthwashes in the Management of Patients with Gingivitis: A Randomized Control Trial

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ABSTRACT

Context: Gingivitis, an early stage of periodontal disease, is primarily caused by plaque biofilm accumulation. Mechanical plaque control is effective but often benefits from adjunctive chemical methods. Chlorhexidine remains the gold standard but is associated with many side effects. Chlorine dioxide and herbal alternatives such as tulsi and clove have gained attention for their antimicrobial and anti-inflammatory properties with fewer adverse effects.

Aims: To compare the efficacy of 0.1% chlorine dioxide and combination of tulsi-clove mouthwashes with 0.2% chlorhexidine in reducing plaque and gingival inflammation in patients with biofilm-induced gingivitis.

Settings and Design: A randomized, single-blind, parallel-group controlled clinical trial

Methods and Material: The trial was conducted on 90 subjects with biofilm-induced gingivitis. After scaling and root planing, subjects were randomly assigned to three groups:Group A: 0.1% Chlorine Dioxide; Group B: 0.2% Chlorhexidine &Group C: Tulsi-Clove combination. Plaque Index and Gingival Index were assessed at baseline and after 15 days.

Statistical analysis used: Data were compiled and analyzed using Statistical Package for the Social Sciences (SPSS) version 23.0.The paired t-test was used to evaluate intragroup differences between baseline and post-intervention scores for each mouthwash group, assessing the effectiveness of each intervention over time. A p-value < 0.05 was considered statistically significant

Results: All the groups showed reduction in both plaque and gingival indices. The present study resulted out statistically significant differences with plaque index between chlorhexidine, chlorine dioxide and herbal mouthwash($p>0.005$).

Conclusions: The tulsi-clove combination was effective in reducing plaque but did not show a statistically significant reduction in gingival inflammation. Chlorine dioxide may serve as a promising alternative in reducing plaque and gingival inflammation with fewer side effects.

Key-words: Gingivitis, Chlorine dioxide, Chlorhexidine, Tulsi, Clove, Plaque Index, Gingival Index, Mouthwash

Key Messages : Chlorine dioxide demonstrated significant efficacy in reducing plaque and gingival inflammation, making it a viable, patient-friendly alternative to chlorhexidine with fewer side effects . The tulsi-clove mouthwash significantly reduced plaque levels, supporting its role as a natural, well-tolerated adjunct for long-term maintenance in gingivitis management . While chlorhexidine remains the gold standard, chlorine dioxide and tulsi-clove formulations present effective and safer alternatives, especially for patients requiring extended oral hygiene support.

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Introduction

Periodontal diseases, which encompass both gingivitis and periodontitis, are inflammatory conditions primarily initiated by microbial activity. The fundamental cause for these pathologies is the accumulation of plaque biofilm at or below the gingival margin, which subsequently triggers an excessive and detrimental inflammatory immune response from the host[1]. Dental plaque is a complex biofilm, characterized by diverse bacterial communities encased within a host-derived matrix, adhering to dental surfaces and other oral structures. The metabolic byproducts of these bacteria contribute to tissue damage and alveolar bone loss. Implementing effective plaque control strategies can lead to a reduction in both the amount and composition of the plaque biofilm. Bacteria in the subgingival region form a complex and well-structured microbial biofilm. These biofilms are highly resilient, challenging to manage, and play a crucial role in the development and progression of plaque and periodontal disease. Therefore, the primary goal of treatment is to eliminate these bacterial biofilms.

Conventional nonsurgical periodontal therapy primarily involves the mechanical debridement of supragingival and subgingival deposits, supplemented by comprehensive patient education on optimal oral hygiene practices(Darby et al., 2011). These interventions aim to reduce the bacterial load and promote a shift in microbial composition towards a healthier oral flora. Chemical plaque control, on the other hand, serves as a supplement to mechanical methods, with various agents being effectively used to manage and prevent gingivitis. Mechanical debridement, the primary treatment approach for managing periodontal disease, has shown considerable success in treating most patients but carries a higher risk of recurrence, particularly in cases involving systemic comorbidities.

Antimicrobial mouthrinses have gained increasing importance as adjuncts in maintaining oral hygiene, due to their proven effectiveness in reducing microbial load in the mouth(Gizani et al., 2025_)

Chlorhexidine is considered the gold standard for plaque control, but its long-term use is associated with several side effects, including tooth and tongue staining, altered taste perception, and increased calculus formation, which can discourage its continued use[2,3].

Chlorine dioxide (ClO_2) is a water-soluble, light-sensitive compound known for its remarkable stability under protected conditions. ClO_2 interacts with specific amino acids found in saliva—such as pyruvate, methionine, trimethylamine, tyrosine, and glycine—which are essential for the survival and growth of odor-causing bacteria. By disrupting the availability of these compounds, ClO_2 inhibits bacterial growth and reduces halitosis[4]. Additionally, it neutralizes precursors of volatile sulfur compounds (VSCs), such as hydrogen sulfide, which are key contributors to oral malodor. What sets ClO_2 apart from conventional antimicrobials is its selective oxidizing action and capacity to penetrate biofilm (Shinada et al., 2010). This allows for more efficient disruption of mature biofilms, which are often resistant to treatment. Studies and systematic reviews support its effectiveness in lowering oral malodor, as evidenced by improvements in organoleptic assessments and reductions in VSC concentrations. Given that prolonged use of chlorhexidine is limited by adverse effects—such as staining, taste alteration, and calculus formation—ClO_2 emerges as a favorable alternative. Its minimal cytotoxicity and enhanced biofilm-targeting capabilities suggest its potential usefulness in the management of chronic, biofilm-induced gingivitis and in promoting long-term periodontal health.[5]

Tulsi (Ocimum sanctum), a medicinal herb widely employed in traditional and contemporary oral care, contains a range of bioactive constituents including eugenol, caryophyllene, germacrene-A, clemence, and caryophyllene oxide. Notably, linolenic acid (43–64%) and eugenol (up to 71.3%) are key components that exert anti-inflammatory effects by inhibiting cyclooxygenase-2 (COX-2) and lipoxygenase pathways. Tulsi extracts—particularly in alcoholic form—have shown antimicrobial efficacy against several periodontal pathogens, such as Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Prevotella intermedia, Fusobacterium nucleatum, and Escherichia coli. Beyond its antimicrobial activity, Tulsi also enhances immune function by increasing levels of interferon, interleukin-4 (IL-4), and T-helper cells, thereby supporting host resistance to infection[6].

Clove (Syzygium aromaticum) has a long-standing history in traditional medicine, especially in dental applications. Its active constituent, eugenol, imparts strong antiseptic, anti-inflammatory, and analgesic effects. Research, including quasi-experimental studies, has shown that clove-based mouthrinses can significantly reduce gingival inflammation and plaque accumulation, particularly in adolescent populations, indicating its potential as a natural therapeutic agent in gingival health maintenance[7].

Given the lower risk of adverse effects associated with these newer materials compared to the clinical gold standard, chlorhexidine, it is valuable to assess their effectiveness in patients with biofilm induced gingivitis. Therefore, we compared the efficacy of 0.1% chlorine dioxide and combination of tulsi-clove mouthwashes with 0.2% chlorhexidine in reducing plaque and gingival inflammation in patients with biofilm-induced gingivitis.

Materials & Methodology

A randomized single blind, parallel group, controlled clinical trial was carried out in the Department of Periodontology at Annoor Dental College & Hospital, Muvattupuzha, Kerala, India. This study received ethical clearance from the Institutional Ethics Committee. All participants were thoroughly informed about the clinical procedures and the follow-up requirements before their enrolment in the study.

Eligibility criteria consisted of participants in the age group of 18 to 60 with biofilm induced gingivitis. Exclusion criteria included participants with multiple restorations or severe dental caries, recent use of any antibiotics (topical or systemic) within the past two weeks, current tobacco users, current users of other mouth rinses or antimicrobials, and those on any medication.

Ninety participants with biofilm induced gingivitis [2017 AAP classification] with 2 weeks washout period were randomly selected based on inclusion and exclusion criteria. The examiner responsible for recording the clinical parameters was blinded to the group allocation, while the participants were explained about the study and written informed consent was obtained from the patient. Participants were then randomly allotted to one of the three groups based on lottery method.

Group A: Chlorine dioxide [0.1%] [Freshclor antimicrobial mouthwash, marketed by Group Pharmaceutical Ltd, Kolar District, Karnataka]

Group B: Chlorhexidine [0.2%][Hexidine mouthwash, manufactured & marketed by ICPA Health Products Limited, Mumbai]

Group C: Tulsi-clove mouthwash [Niyamba Cura mouthwash, marketed Onika organics, sold by Niyamba pharmaceutical pvt limited]

In the first stage, all participants received scaling and root planing [SRP] and oral hygiene instructions that include, Modified bass brushing technique. Participants were recalled after 15 days [wash out period] and evaluation of the gingival status of all participants were done using Plaque Index (Silness and Loe,1963) and Gingival Index (Loe and Silness, 1963), then randomly allotted to Group A, B and C, who received

BASELINE				
GROUP	INDICES	MEAN	St. Deviation	P-value
CHX	GI	1.657	0.308	0.001
CHX	PI	1.328	0.465	0.0031
ClO2	GI	1.418	0.359	0.0034
ClO2	PI	1.382	0.479	0.00001
Tulsi Clove	GI	1.492	0.353	0.099
Tulsi Clove	PI	1.291	0.207	0.000001

Table 1: Mean plaque index and gingival index score of baseline in Group A, B & C

AFTER 15 DAYS				
GROUP	INDICES	MEAN	St. Deviation	P-value
CHX	GI	1.332	0.385	0.001247
CHX	PI	0.952	0.382	0.0031
ClO2	GI	1.167	0.163	0.0034
ClO2	PI	0.875	0.218	0.00001
Tulsi-Clove	GI	1.364	0.291	0.099
Tulsi-Clove	PI	0.87	0.291	0.000001

Table 2: Mean plaque index and gingival index scores after 15 days in Group A, B & C

0.1% ClO2, 0.2% CHX and combination of tulsi-clove mouthwash respectively. They were instructed to rinse with 10 ml of mouth rinse for 1 min, twice daily for 15 days, half an hour after brushing. Participants were recalled after 15 days, and gingival index and plaque index was recorded again.

Results

In the Tulsi-Clove group, the mean Gingival Index decreased from 1.49 at baseline to 1.36 at 15 days. However, the p-value (0.099) is greater than 0.05, indicating that this reduction is not statistically significant. The mean Plaque Index significantly decreased from 1.29 at baseline to 0.87 at 15 days, with a p-value of 0.000001, which is highly statistically significant.

In the Chlorhexidine group, the mean Gingival Index significantly decreased from 1.66 at baseline to 1.33 at 15 days, with a p-value of 0.001, which is statistically significant. The mean Plaque Index significantly decreased from 1.33 at baseline to 0.95 at 15 days, with a p-value of 0.003, which is statistically significant.

In Chlorine Dioxide group, the mean Gingival Index significantly decreased from 1.42 at baseline to 1.17 at 15 days, with a p-value of 0.003, which is statistically significant. The mean Plaque Index significantly decreased from 1.38 at baseline to 0.87 at 15 days, with a p-value of 0.00001, which is highly statistically significant

Discussion

Effective plaque control is essential in the prevention and management of gingivitis, as dental plaque is the primary etiological factor in the initiation and progression of periodontal inflammation. While mechanical plaque removal remains the cornerstone of oral hygiene, the role of adjunctive chemical plaque control has been well established in contemporary periodontal therapy.

The results of present study confirm that chlorhexidine remains the most effective agent for reducing both gingival and plaque indices. Malhotra et al. (2011) reported that 0.2% chlorhexidine significantly inhibited plaque formation compared to herbal rinse, despite lower patient preference due to side effects like taste alteration and staining[8]. Similarly, studies by Jones (1997) and Santos (2003) have consistently validated CHX as the gold standard for chemical plaque control, highlighting its broad-spectrum antimicrobial activity and substantivity[9,10].

Chlorine dioxide showed a significant reduction in both plaque and gingival indices, particularly in plaque control where the effect was highly statistically significant. Its antimicrobial properties, as well as its ability to neutralize volatile sulfur compounds and disrupt biofilms, make it a promising alternative to CHX. It is consistent with research by Shinada et al. (2010) and Grootveld et al. (2001), which demonstrated ClO_2's strong antibacterial effect and its ability to neutralize volatile sulfur compounds involved in halitosis4,5. Paraskevas et al. (2008) also compared ClO_2 to CHX in a three-day plaque accumulation model and reported comparable short-term effects, suggesting ClO_2 as a

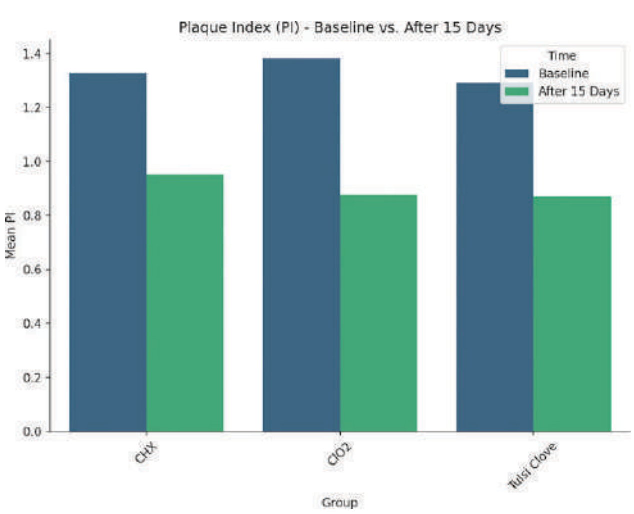

Fig 1: Inter group comparison of plaque index

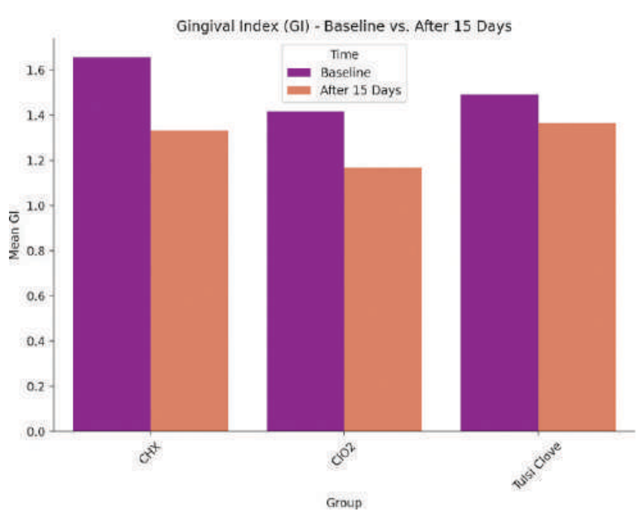

Fig 2: Inter group comparison of gingival index

viable alternative, particularly for patients intolerant to CHX side effects[11].

Our results showed a statistically significant reduction in plaque levels with the herbal mouthrinse but not in gingival inflammation. Malhotra et al. (2011) similarly found that while the herbal mouthrinse was less effective than CHX in reducing plaque, it was favoured by patients for its taste and ease of use. The herbal components in our formulation, particularly tulsi and clove, are known for their anti-inflammatory, antiseptic, and analgesic properties. Studies by Haffajee et al. (2008) and Kaim et al. (1998) have demonstrated the antimicrobial efficacy of herbal formulations, particularly against anaerobic pathogens like P. intermedia and T. forsythia[12,13].

Taken together, our findings suggest that while CHX remains the most efficacious, chlorine dioxide presents a strong alternative with fewer side effects and high biocompatibility. The herbal tulsi-clove rinse, though less potent in inflammation control, offers a natural and patient-preferred option, especially in long-term or preventive care.

The present study has certain limitations that should be considered while interpreting the results. The follow-up period was limited to 15 days, which restricts evaluation of the long-term effectiveness and sustainability of the observed outcomes. In addition, microbiological analysis was not performed, thereby limiting insight into the specific changes in oral microbial load associated with the interventions. Furthermore, long-term clinical outcomes were not assessed. The study also did not include subjective patient-reported parameters such as taste acceptability, comfort, or compliance, which limits a comprehensive patient-centered evaluation. Future research incorporating longer follow-up durations, microbiological assessments, and patient-reported outcome measures is warranted to provide a more holistic and clinically relevant assessment of the efficacy and acceptability of the evaluated mouthrinses.

Conclusion

The tulsi-clove combination was effective in reducing plaque. Though it showed reduction in gingival inflammation, it was not statistically significant. The herbal tulsi-clove rinse, though less potent in inflammation control, offers a natural and patient-preferred option, especially in long-term or preventive care. Chlorine dioxide may serve as a promising alternative in reducing plaque and gingival inflammation with fewer side effects.

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JSPIK

Biological Insights into Peri-Implant Soft Tissue Health

Anjali Babu[1], Mohammed Feroz T P[2], Nikhila T M[3], Neelima Vinod[4]

ABSTRACT

Surgical healing and implant surface design collectively shape peri-implant tissues, giving rise to distinct histological and morphological traits. The biological seal at the mucosa-implant interface prevents pathogen penetration, protects the underlying peri-implant bone, and reduces susceptibility to inflammatory peri-implant diseases. This review provides an overview of the structure, biology, and function of peri-implant soft tissues, outlining their morphogenic features and their relevance to peri-implant health.

Keywords: Peri-implant epithelium, Peri-implant connective tissue, Supracrestal tissue attachment, Periimplant mucositis, Periimplantitis

Introduction

The favourable predictability and widespread acceptance of implant therapy have contributed to a substantial rise in the utilization of dental implants for prosthetic rehabilitation. Nevertheless, although dental implants demonstrate high survival and success rates, peri-implant diseases continue to represent a major clinical concern. A systematic review and meta-analysis, based on the 2017 World Workshop criteria, estimated that approximately two-thirds of adults with dental implants suffer from peri-implant mucositis, while nearly one-quarter develop peri-implantitis.[1] Evidence suggests that maintaining a peri-implant mucosal thickness of at least 2 mm is essential for sustaining peri-implant tissue stability.[2] Adequate keratinized mucosal width combined with a thick peri-implant mucosal biotype supports effective integration and formation of a stable biological seal at the mucosa–implant interface. However, even under optimal conditions, the peri-implant biological seal exhibits less mechanical resistance, stability, and hermeticity compared to that formed between periodontal tissues and dental surfaces.[3]

Biology of Peri-Implant Soft Tissues in Health

Peri-implant tissues exhibit distinct structural and morphological features, including the absence of a periodontal ligament, a deeper peri-implant sulcus, reduced vascularity due to their scar-like nature, and notable histological and functional differences at the peri-implant epithelium (PIE) and peri-implant connective tissue (PCT), all of which contribute to the formation of the biological seal around dental implants. Consequently, these biological structures have a lower capacity for integration and adhesion around dental implants compared to the biological seal of periodontal tissues.[4]

Peri-Implant Epithelium (PIE)

The epithelium surrounding dental implants is composed of three types of stratified squamous epithelia: (a) the PIE, (b) the peri-implant sulcus epithelium (PISE), and (c) the oral epithelium (OE). The peri-implant epithelium, which develops from the oral epithelium within 2–3 weeks after placement of a transmucosal abutment, is characterized by several layers of non-keratinized flattened epithelial cells with wide intercellular spaces.[5] Like the junctional epithelium (JE), the peri-implant epithelium is sup-

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ported by a basal membrane composed of structural proteins, including type IV collagen, fibronectin, and laminin. The basal membrane comprises two layers namely, internal basal lamina (IBL) and external basal lamina (EBL) and each subdivided into a lamina lucida and lamina densa, which facilitate epithelial cell adhesion to underlying surfaces. The internal basal lamina, primarily composed of laminin 332 and type VIII collagen, anchors the peri-implant epithelium to the implant surface, with its lamina lucida inserting into the plasma membrane of PIE cells, reinforced by hemidesmosomes and epithelial adhesion plates,

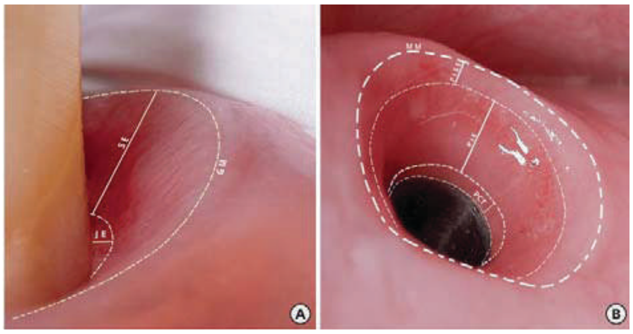

Figure 1: Anatomical Structure of (A) Periodontal Tissues; (B) Peri-Implant Tissues[3]

JE: junctional epithelium, SE: sulcular epithelium, GM: gingival margin, MM: mucosal margin, PISE: peri-implant sulcular epithelium, PIE: peri-implant epithelium, PCT: peri-implant connective tissue.

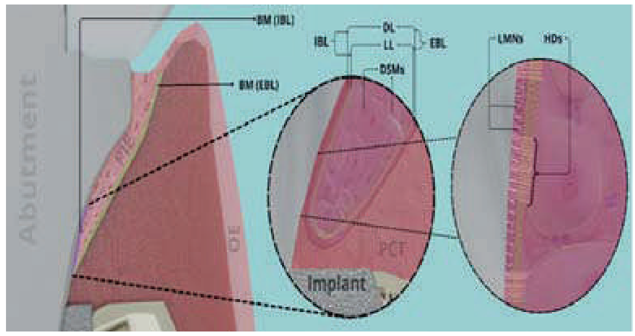

Figure 2: Schematic Representation of the PIE3

- **Left:** PIE's BM consists of IBL and EBL. Notably, the IBL only encompasses the PIE at its most apical level.
- **Middle:** The IBL and EBL are divided into LL and DL, through which the epithelial cells adhere to different surfaces. The inter-epithelial cell adhesive junctions are mediated by DSMs.
- **Right:** The IBL adheres to the implant surface via HDs and LMNs

PIE: peri-implant epithelium, BM: basement membrane, IBL: internal basal laminae, EBL: external basal laminae, LL: lucid lamina, DL: dense lamina, DSM: desmosome, HD: hemidesmosome, LMN: laminin.

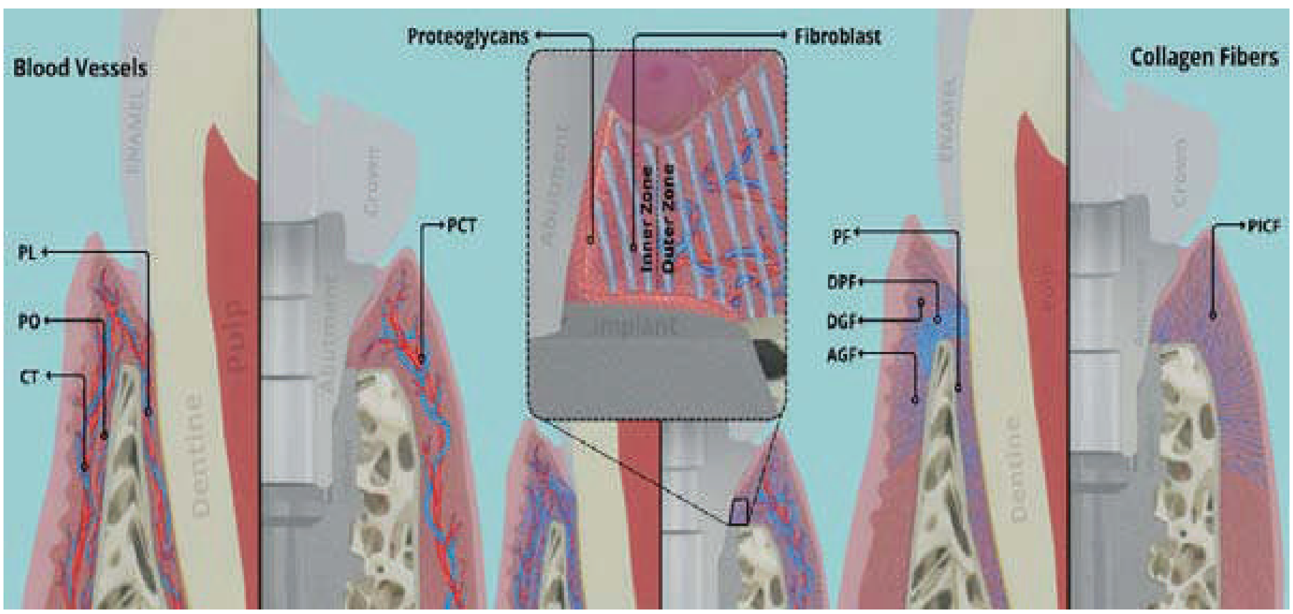

Figure 3:Schematic Comparison of The Periodontal& Peri-Implant Connective Tissue[3]

- ***Left:*** *Periodontal tissues have a broad blood flow from the PL, the PO, and the CT; Vascular supply of the peri-implant mucosa is predominantly from its CT.*
- ***Middle:*** *Histological zones of the PCT, separated from the implant surface by a 20-nm proteoglycan layer.*
- ***Right:****Schematic representation of collagen fibres running through the periodontal CT (DGF,* **DPF, AGF, PF) and in** *PCT, PICFrunning parallel to the dental implant.*

PCT: peri-implant connective tissue, CT: connective tissue, PL: periodontal ligament, PO: periosteum, DGF: dento-gingival, DPF: dento-periosteal, AGF: alveolo gingival, PF: periodontal fibres, PCT: peri-implant connective tissue, PICF: peri-implant collagen fibres.

while the lamina densa connects directly to the implant surface via hemidesmosomes, collectively forming the biological seal (BS) between the epithelium and implant. However, it has been reported that the internal basal lamina and hemidesmosomes are found only in the most apical region of the PIE-titanium interface, unlike periodontal tissues, where these structures are present along the entire junctional epithelium-tooth interface [Ikeda H et al., 2000][5]. The external basal lamina, rich in extracellular matrix proteins, connects the peri-implant epithelium to the connective tissue of the peri-implant mucosa, with its lamina lucida anchoring to the basal PIE cells via hemidesmosomes and its lamina densa connects to the fibroreticular or sub-dense lamina.

Peri-Implant Connective Tissue (PCT)

The peri-implant connective tissue (PCT) beneath the PIE is composed mainly of type I and III peri-implant collagen fibres (PICF), while the supracrestal PCT contains predominantly type I collagen fibers. In both regions, these fibres are oriented parallel to the implant surface and do not attach to it. The lamina propria of the peri-implant mucosa contains abundant type V collagen fibres, which are highly resistant to collagenase. This collagen profile suggests a scar-like, chronically inflamed connective tissue rather than a protective or sealing structure comparable to periodontal tissues, and may contribute to accelerated horizontal recession.[6] Evidence suggests that a 200-μm zone of PCT adjacent to the implant is composed of two layers: (i) an inner 40-μm avascular layer rich in flattened fibroblasts and thin, parallel collagen fibers, separated from the titanium surface by a 20-nm proteoglycan layer, resulting in weaker adhesion than natural teeth; and (ii) an outer 160-μm layer containing mainly peri-implant collagen fibers, with fewer fibroblasts and limited vasculature [Moon et al., 1999][7].

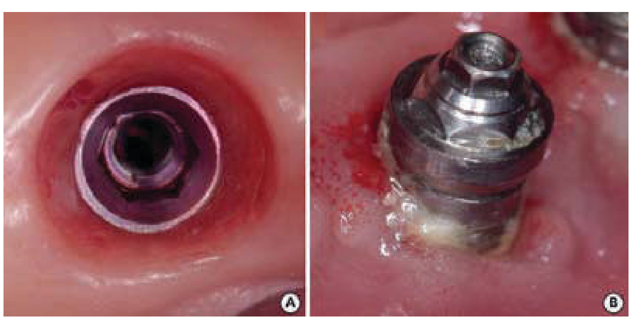

Figure 4: (A) Healthy Peri-implant Mucosa; (B) inflammatory peri-implant disease[3]

Peri-implant Sulcus (PS)

Peri-implant probing depth is influenced by various factors such as the peri-implant phenotype, anatomical conditions, implant design, and the position of the implant platform relative to the crestal bone.8 Thus the new classification framework concluded that "it is not possible to define a range of probing depths compatible with peri-implant health".[9] In a long-term follow-up study, healthy peri-implant mucosa frequently presents with probing depths greater than 4 mm [Winitsky N et al., 2018][10]. Furthermore, the delicate attachment between the peri-implant epithelium and the implant surface is easily disrupted by minimal probing pressure, often causing bleeding. Therefore, bleeding on probing and probing depth alone may not accurately reflect peri-implant pathology. Similar to the gingival crevicular fluid, the peri-implant sulcus contains an inflammatory exudate known as peri-implant crevicular fluid (PCF), which primarily originates from the vessels of the peri-implant mucosal plexus.3 Clinically healthy implants usually exhibits higher volume of peri-implant crevicular fluid and a greater concentration of pro-inflammatory cytokines compared to the gingival sulcus of periodontally healthy teeth.[3] During peri-implant mucositis and peri-implantitis, there is

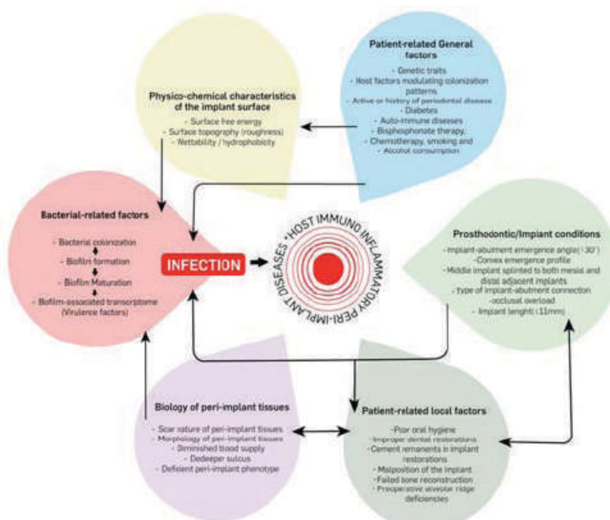

Figure 5: Risk factors associated with the onset and progression of inflammatory peri-implant diseases[3]

a significant increase in the volume of peri-implant crevicular fluid, accompanied by elevated levels of proinflammatory mediators and enzymatic activity. This leads to a substantial increase in the inflammatory infiltrate, characterised by a higher proportion of neutrophils, macrophages and plasma cells compared to those found in periodontitis.

Peri-implant biological width (supra-crestal tissue attachment)

The formation of biological width in peri-implant soft tissue is a sequential and regular process and its dimensions have been found to vary from 3 to 4 mm, with the peri-implant component ranging from 2.02 to 2.3 mm and the peri-implant connective tissue component from 1.1 to 1.49 mm across various studies.[6] The dimensions may also vary depending on the implant design and surface characteristics. In conical implants with an internal hex, this attachment ranges from 3.13–3.34 mm.[6] In monobloc implants, it is about 2.55±0.16 mm, compared with 3.26±0.15 mm in two-component implants. Surface treatments, such as sand-blasting and acid-etching, can further alter the dimensions, which may range from 2.84–3.80 mm.[6] Like the biological width in natural teeth, the peri-implant tissue attachment also provides key immunological functions, such as (a) the endocytosis and decomposition of exogenous substances by neutrophils, (b) the endocytosis and decomposition of exogenous factors by the JE itself, (c) the antibacterial action of crevicular fluid, and (d) the exfoliation of the JE cell layer.[3]

Inflammatory Peri-Implant Diseases

Peri-implant mucositis and peri-implantitis are the two inflammatory conditions that can lead to failure of osseointegrated implants. Peri-implant mucositis is characterised mainly by bleeding on probing, along with erythema, swelling and sometimes suppuration. Without early control of etiologic factors, peri-implant mucositis can progress to peri-implantitis. Peri-implantitis is a plaque-associated disease involving progressive bone loss after implant loading. Clinically, bleeding on probing is the key tool to distinguish between healthy and inflamed peri-implant mucosa, while bone loss is used to differentiate between peri-implant mucositis and peri-implantitis. The progression of peri-implantitis occurs in a non-linear and accelerating pattern.

Conclusion

Peri-implant mucosal tissue plays a critical role in the long-term maintenance of dental implants, acting as a biological barrier against bacterial invasion and contributing to both functional and esthetic outcomes. The shift from the early emphasis on osseointegration to the recognition of mucosal integration highlights its increasing importance. Therefore, a comprehensive understanding of peri-implant soft tissue biology is essential for formulating strategies that prevent or minimise the impact of peri-implant diseases on implant treatment outcomes.

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Effect of Smokeless Tobacco on Periodontium

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ABSTRACT

Smoking is one of the most important causes of preventable death. The prevalence of habits are increasing day by day especially in young individuals. Smoking has systemic as well as oral manifestations. Tobacco can be smoke and smokeless. Both of them cause major illness. This article is based on the effect of smokeless tobacco on periodontium. Smokeless tobacco can be snuff and chewing. Nicotine is one of the major constituents in tobacco that causes periodontal destruction. Tobacco also has an effect of neutrophils, T-lymphocytes, monocytes, fibroblasts, anaerobic species etc. On periodontium it causes mucosal lesions, acute necrotising ulcerative periodontitis and gingivitis, gingival recession, plaque accumulation, attachment loss, change in subgingival bacterial colony, local immunosuppression etc. Numerous tobacco cessation programs has been introduced which includes the 5A's approach (ASK, ADVISE, ASSESS, ACT, ASSIST) or 5R's (RELEVANCE, RISK, REWARDS, ROADBLOCKS , REPETITION). There are other pharmacological as well as non pharmacological treatment approaches towards tobacco cessation. Prevention is better than cure so as healthcare professionals it is our duty to create awareness so that the smoking habit can be completely eradicated from the world.

Keywords : smokeless tobacco, nicotine, periodontium, tobacco cessation, periodontal therapy

Introduction

Habits are repetitive action that is done unknowingly. Some of the oral habits that can cause destructive changes to the periodontium include smoking, pan chewing, snuffing, thumb sucking, finger biting, mouth breathing, tongue thrusting, lip biting etc.[1] Smoking behaviour in itself can be a habit.[2] Smoking is known to cause lung diseases, cancer, cardiovascular diseases and poor pregnancy outcomes.[3] Although smoking habit has decreased nowadays various statistics has shown that more than one quarter of the nation's population use tobacco in one or other form. Smokeless and smoke forms of tobacco most commonly used include khaini and beedi.[4] Smokers experience widespread destruction of the periodontium whereas the effects of smokeless tobacco is confined to its site of placement which may be manifested as white mucosal lesions and gingival recession. Alteration in the oral microbiome is the main factor that leads to this kind of periodontal destruction.[3]

Composition of smokeless tobacco.

There are two types of smokeless tobacco. They are snuff and chewing tobacco. There are moist as well as dry snuff. Moist snuff is administered orally where as the dry snuff is inhaled through the nose. Chewing tobacco can be of three varieties: looseleaf, plug and twisted. Smokeless tobacco contains carcinogens such as polynuclear aromatic hydrocarbons (e.g., benzo[a] pyrene), polonium-21, and N-nitrosamines.[5] One of its primary alkaloids, nicotine, can affect both wound healing and immune function.[6]

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Pathogenesis of smoking related periodontal destruction.

Exposure of nicotine leads to vasoconstriction which leads to impaired angiogenesis as follows

Exposure of nicotine

↓

1. Vasoconstriction

↓

Impaired Angiogenesis

Has an effect on neutrophils by favouring the shedding of adhesion molecules, alteration in f-actin kinetics , decreased neutrophil migration, phagocytosis and defect in oxidative killing.

2. Neutrophil dysfunction

↓

- Shedding of adhesion molecules
- Alter the f-actin kinetics

↓

- Reduced neutrophil migration
- Inhibit phagocytosis
- Impaired oxidative killing

Also influences the proliferation and functioning of T-lymphocytes

3. T-lymphocytes

↓

- Reduced proliferation
- Reduced functioning

Reduced phagocytosis and pro-inflammatory cytokines and oxygen radicals synthesis by monocytes.

4. Monocyte suppression

↓

- Decreased phagocytosis
- Decreased synthesis of pro-inflammatory cytokines and oxygen radicals

5. Increased tissue destructive cytokines

6. Decreased antibody production

↓

Decreased response to periodontal pathogens

7. Defective periodontal ligament fibroblast attachment.[6]

8. Neutrophils release protease

↓

Tissue destruction.[9]

Smoking also lowers the oxygen tension in periodontal pockets which favours the growth of anaerobic species that hydrolyse trypsin like BANA substrate leading to periodontal disease progression.

9. Smoking

↓

Lower oxygen tension in periodontal pocket

↓

Growth of anaerobic species

↓

More prevalence of :

- P.gingivalis
- T.denticola
- B.forsythus

↓

Hydrolyse trypsin like BANA substrate

↓

Periodontal disease progression.[3]

Effect of smokeless tobacco

A) On periodontium

Most commonly associated with acute necrotizing ulcerative gingivitis, periodontitis and gingivitis.[5] Smoking also contributed to the development of plaque.[7] The calcium content in plaque was also increased in smokers. Development of gingival inflammation which may later affect other parts of periodontium leading to periodontitis. Initially changes are seen at the blood vessels by capillary dilation and increase in blood flow.[8] Mucosal lesions are commonly found in smokeless tobacco users. Among these users severe gingival recession and attachment loss was found in sites near to the mucosal lesions.[10] Attachment loss is mainly localized in the form of gingival recession.[11]

B) On prevalence of periodontitis

Increased presence of periodontal pathogens in the soft tissues of smokers with periodontitis.[12] Fusobacterium is more abundant in smokers than in non smokers which brings about a change in the subgingival bacterial colony(F.nucleatum). It also contributes to formation of subgingival biofilm due to its “bridging species” role among other microorganisms and also it has local immunosuppressive capability all of which favours the development of various periodontal diseases.[13]

C) On periodontal treatment

After non surgical therapy such as scaling and root planing , the healing in relation to gingival bleeding reduction and pocket depth reduction is decreased in smokers than non-smokers.[8] Antimicrobial therapy may be considered in smokers since the subgingival pathogens are difficult to remove by scaling and root planing in smokers. Antimicrobial therapy act as an adjunctive to mechanical therapy. Studies have shown that systemic amoxicillin and metronidazole, local delivery of minocycline microspheres further increased the results of mechanical therapy.[7] The clinical attachment gains after regenerative procedures are also comparatively less in smokers than non smokers.[11] Gutka chewing leads to delayed post extraction wound healing.[16] Non surgical leads to decreased response to scaling and root planing, pocket depth, attachment levels. Surgery and implants leads to decreased pocket depth, increased destruction of furcations, decreased clinical attachment levels, reduced bone fill, increased recession, increased membrane exposure after GTR, decreased pocket depth reduction, increased clinical attachment levels after open flap debridement.[18]

Cessation of smokeless tobacco

Use of smokeless tobacco has to be controlled by implementing certain policies, by proper education on the potential hazards of smokeless tobacco use, training of the health professionals in cessation programmes.[14] An evidence based intervention called 5 A's approach to tobacco cessation is implemented by several countries which is as follows :

ASK about tobacco use at every visit

↓

ADVISE users to quit

↓

ASSESS their willingness to quit

↓

ACT on patient's response

↓

Ready to quit Not ready to quit

↓

ASSIST with quit plan or HELP motivate the patient

↓

Arrange follow-up contact.15

If not 5 A's then 5 R's :

- RELEVANCE : Prompt the patient to explain why quitting tobacco is personally meaningful to them.
- RISK : Encourage the patient to recognize the possible harmful effects of continued tobacco use.
- REWARD : Invite the patient to consider the potential advantages of quitting.
- ROADBLOCKS : Help the patient identify any challenges they might face in the quitting process.
- REPETITION : Reinforce the motivational discussion at every clinical encounter with patients who are not yet ready to quit.[15]

Other recommended treatment approaches include :

1. Non-pharmacological strategies for smoking cessation
   a. Counselling
   b. Cognitive behavioural therapy
   c. Written or telephone or digital advices
2. Pharmacological approaches for smoking cessation

   First-choice agents
   a. Nicotine replacement therapy
   b. Transdermal patch
   c. Gum
   d. Lozenge
   e. Oral inhaler
   f. Nasal spray
   g. Combined NRT
   h. Varenicline (combined or not with NRT)
   i. Bupropion sustained release (combined or not with NRT)

j. Varenicline+Bupropion sustained release

k. Cystisine

Second-choice agents or adjuvants

a. Anxiolytics (benzodiazepines)

b. Other antidepressants

c. Selective serotonin reuptake inhibitors (SSRIs)

d. Serotonin or norepinephrine reuptake inhibitors (SNRIs)

e. Atypical antidepressants[19]

Conclusion

Smoking has both systemic as well as oral effects. Oral effects especially seen in relation to the periodontium. Public health strategies focusing on education, early detection, and cessation support are crucial in mitigating these risks. Individuals are encouraged to avoid smokeless tobacco use and seek regular dental check-ups to maintain optimal oral health.

Smokeless tobacco increases the risk of oral potentially malignant disorders in smokers. It is the duty of all healthcare providers to assess the tobacco usage and advise the patient about the adverse effects of smoking.[20]

In conclusion, the detrimental impact of smokeless tobacco on oral health is profound and multifaceted. Combating this issue necessitates concerted efforts from healthcare providers, policymakers, and communities to implement preventive measures, promote cessation, and ensure early detection of smokeless tobacco-induced oral diseases.

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Z-Plasty Frenectomy in an 18-Year-Old male with aberrant labial frenum: An Eight-Month Follow-Up

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ABSTRACT

One of the most demanding aspects of periodontal plastic surgery is the management of aberrant labial frenum that interferes with aesthetics and function. High or papillary frenum attachment may cause midline diastema, gingival recession, and compromised orthodontic outcomes.

Case summary: An 18-year-old male patient presented with a midline diastema in the maxillary anterior region associated with a high papillary frenum attachment. Clinical examination confirmed blanching on tension. A Z-plasty frenectomy was performed under local anaesthesia to correct the frenum. The triangular flaps were transposed and sutured to achieve tension-free closure. Healing was uneventful, and follow-up at eight months demonstrated a stable gingival contour, absence of hypertrophic scar. Compared with conventional frenectomy techniques, Z-plasty offered superior advantages, including redistribution of tissue tension, lengthening of soft tissue, enhanced vestibular depth, and camouflaging of scars, thereby ensuring both functional and aesthetic success.

Conclusion: Z-plasty frenectomy provides predictable functional and aesthetic outcomes in cases of aberrant frenum. This case report highlights the effectiveness of Z-plasty in ensuring optimal healing, enhanced aesthetics, and long-term stability.

Keywords: Z-plasty, frenectomy, aberrant frenum, midline diastema

Introduction

A frenum is a fold of mucous membrane composed of muscle and connective tissue fibres that connect the lip and cheek to the alveolar mucosa, gingiva, and underlying periosteum.[1] Aberrant frenum attachments are recognised as important etiological factors for functional and aesthetic problems, most notably midline diastema and gingival recession.[2] These issues arise due to plaque control interference, muscle pull, or abnormal insertion levels, which may also compromise orthodontic outcomes and contribute to diastema relapse.[3]

Placek et al. classified frenum attachments based on their anatomical position into four types: a) mucosal, b) gingival, c) papillary, and d) papillary penetrating. The severity of functional and aesthetic disturbances often correlates with the level of attachment, with papillary and papillary penetrating types posing a higher risk of periodontal complications.[4] Clinically, an aberrant frenum can be identified by applying tension to the area and observing tissue movement or blanching due to ischemia.[6]

Management includes frenotomy, which involves incision and repositioning of the frenum, or frenectomy, which entails complete removal along with its bony attachment.[6] Several techniques have been developed to enhance surgical outcomes, including classical methods, Miller's technique, V-Y plasty, electrocautery, laser,

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and Z-plasty. Among these, Z-plasty—a plastic surgical modification—provides superior functional and aesthetic results by redistributing tension, lengthening tissue, and camouflaging scars through the transposition of two triangular flaps created around a central incision.7 This technique has gained prominence in periodontal plastic surgery due to its ability to achieve primary intention healing and optimal postoperative aesthetics. This case report presents the management of a high papillary frenum in an 18-year-old male patient using Z-plasty, with an eight-month follow-up to evaluate functional and aesthetic results.

Case Report

An 18-year-old male patient presented with aesthetic concerns due to a midline diastema between the maxillary central incisors and was particularly concerned about the risk of relapse after diastema closure. Clinical examination revealed a high papillary frenum attachment with blanching on tension and mild plaque accumulation in the interdental region. (Figure 1(A, B)) Routine haematological investigations were within normal limits. Labial frenectomy can be performed before, during, or even after the orthodontic closure of the maxillary midline diastema, depending on the individual case. In this case, frenectomy was planned to be performed during orthodontic therapy. A thorough family and medical history was obtained to rule out any contraindications to the surgery. The surgical procedure was explained to the patient, and informed consent was obtained. Following professional mechanical plaque removal, the surgery was performed under aseptic conditions.

Surgical Procedure

After oral prophylaxis, local anaesthesia was administered using 2% lignocaine with adrenaline (1:80,000) via infiltration in the maxillary anterior labial region. The surgical site was disinfected with povidone-iodine and isolated.

A central vertical incision was made along the length of the frenum, extending to its base at the interdental papilla. Two oblique lateral incisions were then placed at approximately 60° angles to the central incision, creating two triangular mucosal flaps of equal dimension, giving the characteristic Z-shaped design (Figure 2-Diagramatic representation). Submucosal undermining was performed to release the muscle fibres and achieve flap mobility, while preserving the interdental papilla and minimising trauma to the underlying periosteum.

The triangular flaps were transposed across the central incision and positioned in opposite directions, effectively lengthening the vestibule and relocating the frenal attachment away from the midline. (Figure 3 A). Hemostasis was achieved with gauze pressure. The flaps were then approximated and sutured using

FIGURE 1. Pre-operative view of high labial papillary type frenal attachment (A) frontal (B) lateral view

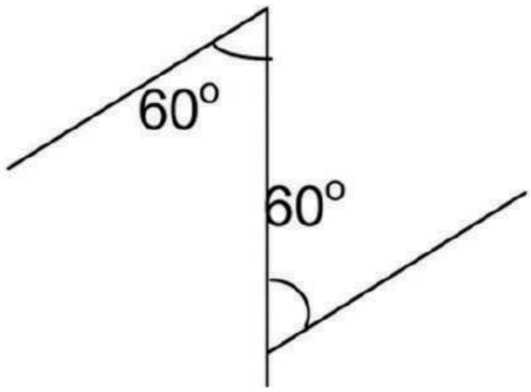
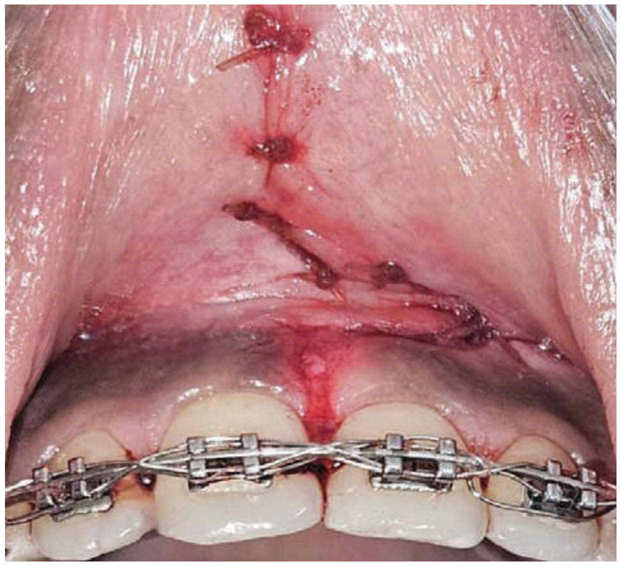


Figure 2 : Diagrammatic representation of the Z-plasty frenectomy design.

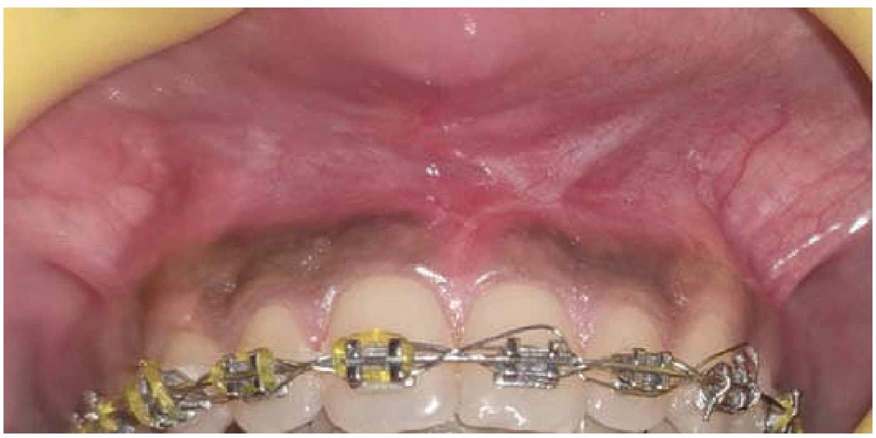
resorbable chromic gut sutures, ensuring tension-free closure and eliminating the need for suture removal (Figure 3 B). A periodontal dressing was applied to protect the surgical site. Postoperatively, the patient was prescribed analgesics, advised on meticulous oral hygiene, and instructed to use warm saline rinses.

At the two-week follow-up, healing was satisfactory with no signs of edema, infection, or discomfort. By one month, the site demonstrated adequate epithelialization, improved vestibular depth, and no hypertrophic scar formation (Figure 4 A, B). At the 8-month review, the mucosa appeared healthy, the interdental

Figure 3 A: Two flaps were relocated to the side opposite each flap's apex



3 B: Sutures were placed by securing two opposing flaps.



A Frontal view

(B) Lateral view

FIGURE 4 (A),(B) : Two-month postoperative view reveals a zone of attached gingiva, with no evidence of scarring or loss of interdental papilla.

papilla was intact, and no recurrence of diastema or scar tissue was observed. Orthodontic space closure remained stable, and the gingival contour was harmonious, reflecting both functional and esthetic success of the procedure. (Figure 5)

Discussion

An aberrant maxillary labial frenum is a well-recognised etiological factor for midline diastema and may also contribute to gingival recession, interference with plaque control, and relapse after orthodontic treatment.[5,8,9] Conventional frenectomy techniques, though effective, often result in unesthetic scarring or inadequate vestibular depth, which can compromise both periodontal health and orthodontic stability.[8] In contrast, the Z-plasty modification provides functional and esthetic advantages by redistributing tissue tension, lengthening the vestibule, and minimizing postoperative scar formation.[5,9]

The technique involves creating two triangular flaps at 60° angles to a central vertical incision, which are then transposed and sutured in a new position. This flap design allows redirection of tensile forces, thereby promoting scar camouflage and improving mucogingival harmony.[8,9] In the present case, the use of resorbable chromic gut sutures ensured tension-free closure and eliminated the need for suture removal, supporting uncomplicated healing. Follow-up at eight months demonstrated stable closure of the diastema, preservation of the interdental papilla, and a healthy gingival contour, consistent with previous reports of uneventful healing and esthetic success with this technique.[5,9]

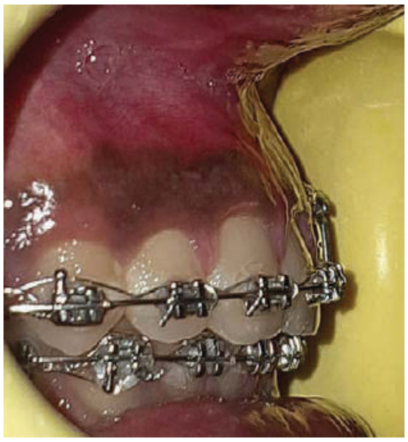
Recent literature emphasizes that frenectomy timing relative to orthodontic therapy plays a critical role in preventing relapse of midline diastema.[5] Performing the procedure after or during orthodontic space closure, as in the present case, avoids premature scar tissue formation that might otherwise interfere with tooth movement.[9,11,12] Additionally, the interdisciplinary approach—combining periodontal surgery with orthodontic management—has been shown to improve long-term functional stability and patient satisfaction.[5,7]

While alternative methods such as electrocautery and lasers are available, Z-plasty remains advantageous in cases requiring both functional release and esthetic refinement. Compared with conventional frenectomy, it provides greater predictability in deepening the vestibule and reducing scar tissue, which are particularly important in young patients with high esthetic demands.[5,7,8,9,11,12] The outcomes of this case further support Z-plasty as a reliable technique in periodontal plastic surgery for managing aberrant frenum, offering stable results over an extended follow-up period.

Conclusion

This case report demonstrates that Z-plasty frenectomy is a reliable technique for managing an aberrant maxillary labial frenum, providing functional stability and esthetic improvement.[5,6,10] The use of resorbable sutures enabled uneventful healing, and

A. Frontal view



(B) Lateral view

Figure 5 (A),(B) : Eight-month postoperative view the 8-month review confirmed stable diastema closure, healthy gingiva, and scar-free results.8 When performed in conjunction with orthodontic therapy, Z-plasty not only prevented relapse but also highlighted the value of an interdisciplinary approach in achieving long-term functional and esthetic success.7,8,9,10,11

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Emerging Biomarkers In Periodontal Disease: Decoding The Molecular Signatures Of Inflammation

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ABSTRACT

Periodontitis, also known as gum disease, is a chronic inflammatory condition that leads to the destruction of the tooth-supporting tissues, including the gums, bone, and periodontal ligament.Periodontitis it still considered to cause connective tissue and bone destruction. Furthermore, monitoring its progression is a difficult and technically demanding process involving measurement of bleeding on probing, probing depth and attachment loss along with radiographic assessment and (subjective) visual observations. The implementation of biomarkers for identifying periodontal disease would be highly advantageous, as current diagnostic methods fail to indicate ongoing disease activity Biomarkers are indicators with high prognostic property and predictive value. Biomarkers must be able to detect the presence of a disease or its progression.

Introduction

Periodontal disease is a persistent bacterial infection that leads to inflammation-driven destruction of the periodontal ligament and alveolar bone, which serve as the supporting tissues of the teeth.According to the 2009- 2010 data from the National Health and Nutrition Examination Survey (NHANES)[1] the prevalence of periodontitis is calculated as 46% in adults.[2] Being the most common disease affecting the oral cavity after dental caries and the major cause of tooth loss, it affects the quality of individual's life. Therefore, early diagnosis and control of the disease is the paramount goal.[3]

A biomarker is a quantifiable indicator that has been assessed and validated as a sign of normal biological function, disease-related activity, or a response to therapeutic intervention.[5] Disease biomarkers are essential in the life sciences and are increasingly contributing to diagnosis, assessment of treatment effectiveness, and pharmaceutic innovations[6]. For biomarkers to take their appropriate place in regular clinical use, it is crucial to gain a deeper understanding of their connection to disease progression mechanisms and treatment interventions.

Classification of Biomarkers[4]

Based on Biological Source (Sample Origin)

Classifies periodontal disease biomarkers into four major groups based on their biological nature and origin:

Proteomic biomarkers

Genetic biomarkers

Microbial biomarkers

Other biomarkers

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Each group reflects a different aspect of periodontal pathogenesis.

Proteomic Biomarkers	Genetic Biomarkers	Microbial Biomarkers	Other biomarkers
• Cathepsin G, glucuronidase • Acid phosphatase • Alkaline phosphatase • Lactoferrin, lactoferrin receptor • Interleukin-1β, interleukin-6, interleukin-17 • MMPs (MMP-8, MMP-9, MMP-13) • Cathepsin B, cathepsin K • Osteocalcin, Osteonectin • Osteoprotegerin, Osteopontin • Receptor activator of nuclear factor kappa B ligand (RANKL) • Platelet-derived growth factor, platelet growth factor • Porphyromonas *gingivialis* hemagglutinin antigen • C-reactive CRP, Telopeptide • IgG, EGF, macrphage factor inflammatory protein-1α Tumor necrosis factor, IgG • Epidermal growth factor, SH3 macrophage inflam proein-1α, Tumor necrosis factor, IgG	Cathepsin G gene Mutation • IL-1 gene polymorphism • IL-6 gene polymorphism • TNF α gene Mutation • TLR genetic factors, Polymorphisms	• Aggregatibacter actinomycetencomitans • Porphyromonas gingivalis • Tannerella forsythia • Treponema denticola • Peptostreptococcus micros • Fusobacterium species • Prevotella intermedia • Parvimonas micra • Treponema socranskii	• Calcium • Cortisol • Hormones • Hemoglobin

Proteomic biomarkers are host-derived proteins and enzymes detected in saliva or gingival crevicular fluid. They primarily reflect inflammatory response, connective tissue breakdown, and bone metabolism, and are useful indicators of ongoing periodontal destruction and response to therapy.

Genetic biomarkers represent variations or polymorphisms in genes involved in immune regulation and tissue metabolism. These markers indicate individual susceptibility to periodontal disease rather than current disease activity.

Microbial biomarkers consist of specific periodontopathogenic microorganisms associated with disease initiation and progression. These biomarkers reflect the etiologic bacterial burden and are useful in identifying high-risk microbial profiles.

Other biochemical biomarkers include inorganic and metabolic substances present in oral fluids that indicate changes in the local periodontal environment.

sources of biomarkers

- ✓ Sub gingival bacteria and their products .
- ✓ Host inflammatory and immune products .
- ✓ Proteolytic and hydrolytic enzymes.
- ✓ Enzymes released from dead cells.
- ✓ Connective tissue degradation products.

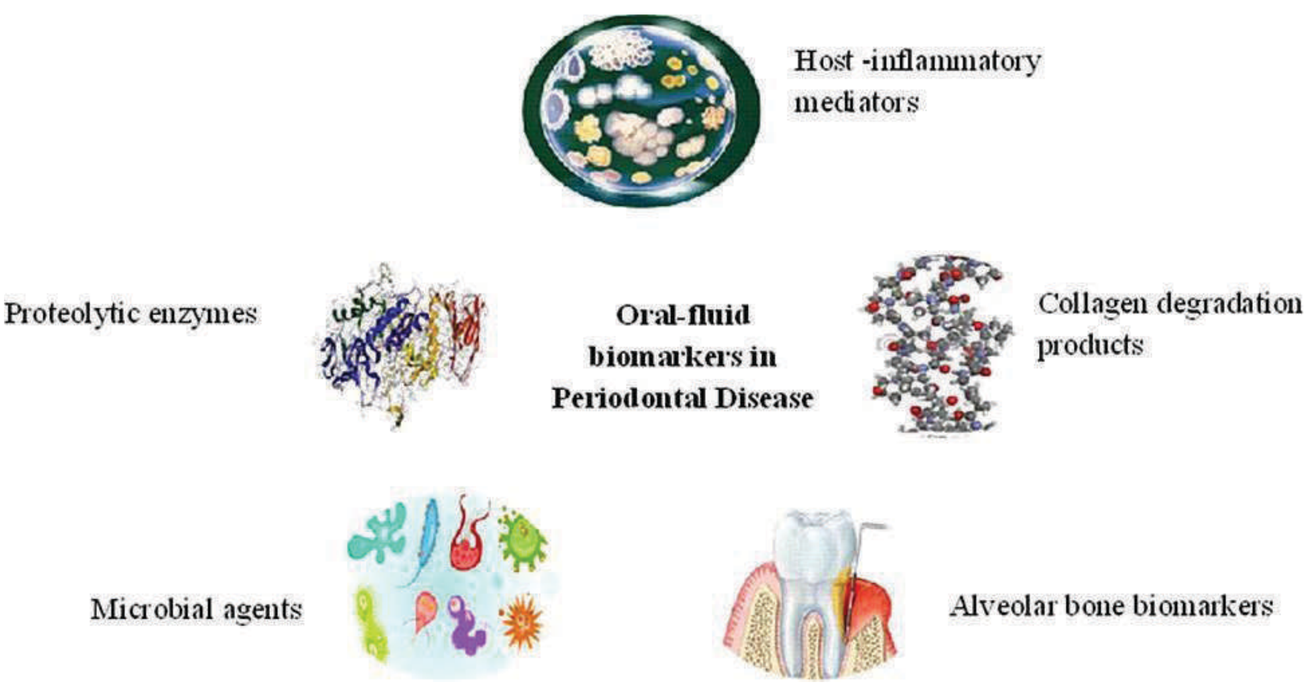


Figure 1: Types of oral fluid biomarkers in periodontal disease

Role of Biomarkers in Diagnosing Periodontal Disease

Investigators in the fields of biotechnology and medicine are actively exploring the application of oral fluids for diagnosing both oral and systemic conditions, as well as for drug development. In the pharmaceutical sector, biomarkers are being extensively researched for their potential in personalized dosing and studies of drug metabolism. Experts from diverse fields—including the insurance sector, the Environmental Protection Agency, and Homeland Security—have shown interest in the potential application of oral fluids for monitoring biomarkers. Under investigation are possible uses of GCF and saliva in the preliminary screening for biological/chemical warfare agent exposure, environmental toxin detection, and screening for metabolites of drugs of abuse.

Due to the non invasive and simple nature of their collection, analysis of saliva and GCF may be especially beneficial in the determination of current periodontal status and a means of monitoring response to treatment.[7] Studies related to the pathogenesis of periodontal diseases usually examine whether and immunologic markers in saliva or GCF might reflect the extent of periodontal destruction and possibly predict future disease progression.[8]

Need for a Periodontal Diagnostic Indicator

A periodontal diagnostic instrument offers relevant insights for distinguishing between conditions, identifying disease location, and determining the extent of infection. These diagnostic tools, in turn, form the foundation for treatment planning and serve as a method for evaluating the success of periodontal therapy.

Proteolytic and Hydrolytic Enzymes as the Markers of the Disease Activity

Aspartate aminotransferase

According to Oringer (2001), AST—an indicator of tissue damage released by necrotic cells present in GCF—shows a significant relationship with periodontal disease severity.[9,10]

Alkaline phosphatase

Gibert (2003) proposed that alkaline phosphatase (ALP) could serve as a predictive marker for future periodontal tissue destruction and disease progression.[11,12]

Lactate dehydrogenase

Wong (2006) observed that lactate dehydrogenase (LDH) activity is elevated in individuals with deeper

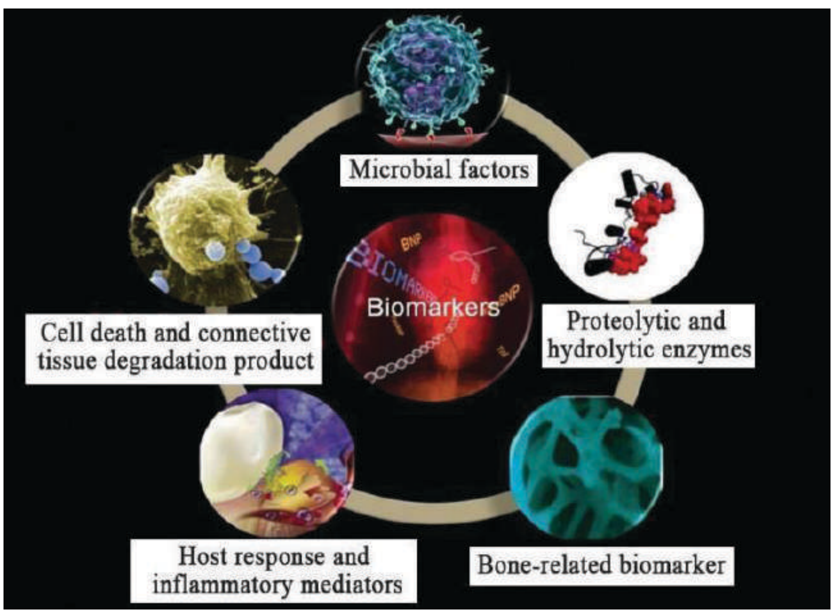


Figure 2 : Biomarkers In Periodontal Disease

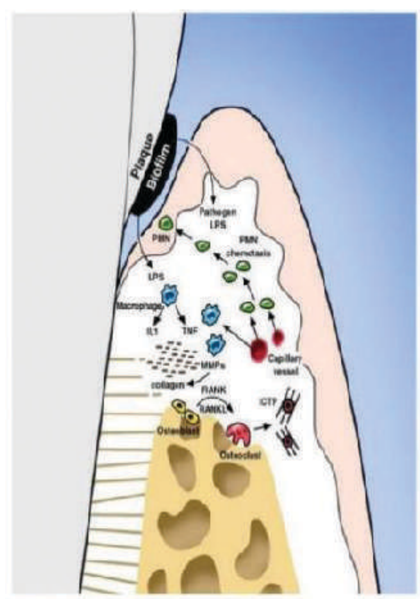


Figure 3 : Pathogenesis of Periodontal Bone Loss — The Cellular and Molecular Events Occurring in Periodontitis.

probing depths compared to those with a healthy periodontium.[13,14]

Collagenase-2 (MMP-8)

Rai (2008) reported significantly higher levels of salivary MMP-8, a known biomarker, in individuals with gingivitis and periodontitis compared to healthy subjects.[15,16]

Other markers include

Cathepsin B [17]

Gingival crevicular fluid (GCF) and saliva show elevated cathepsin B levels in patients with gingivitis and periodontitis compared to healthy controls. Levels correlate with clinical attachment loss (CAL), bleeding on probing (BOP), and pocket depth (PD).[18]

Hence, Cathepsin B serves as a potential biochemical indicator of:

Disease activity and severity and Response to periodontal therapy

Gelatinase (MMP-9)[19]

MMP-9 degrade denatured collagens (gelatin), type IV and V collagen, and elasetin, key comments of the basement membrane and gingival connective tissue. Their expression increases in response to: Bacterial lipopolysaccharides (LPS), Inflammatory cytokines (IL-1β, TNF-α, Oxidative stress and mechanical irritation.[20]

Collagenase-3 (MMP-13)[21]

Elevated MMP-13 levels have been detected in: Gingival crevicular fluid (GCF), Saliva, Inflamed gingival tissues. Levels correlate strongly with Clinical attachment loss (CAL), Probing pocket depth (PPD), Radiographic bone loss. After non-surgical or surgical periodontal therapy, MMP-13 levels decrease, reflecting healing and reduced inflammation.

Bone-Related Biomarkers from Oral Fluids associated with Periodontal Diseases

Pro-inflammatory cytokines released in

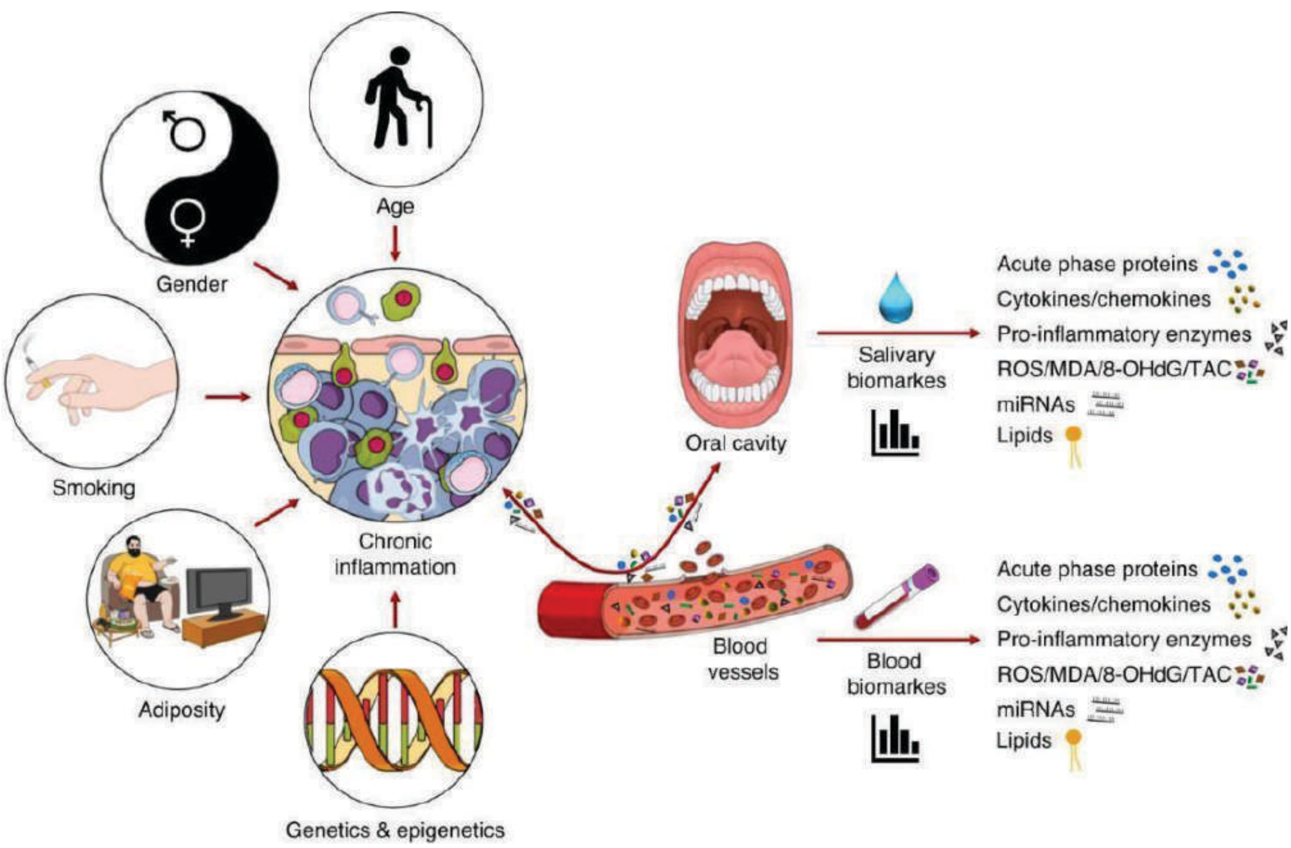


Figure 4 : Influence of Systemic and Lifestyle factors on Chronic inflammation and their reflection in Oral and Blood Biomarkers

periodontal lesions stimulate osteoclast differentiation and activation, leading to progressive bone loss. These biological events are reflected by the presence and altered levels of bone-related biomarkers in oral fluids such as gingival crevicular fluid and saliva.[22]

Biomarkers of bone resorption, including collagen degradation products and osteoclastic enzymes, increase during active periodontal breakdown and correlate with disease severity.

Osteocalcin

A significant relationship was noted between osteocalcin levels and clinical parameters such as pocket depth, gingival index scores, and GCF concentrations of ALP and prostaglandin E2 in periodontitis patients.[23]

Calprotectin

Calprotectin concentrations in gingival crevicular fluid (GCF) were significantly increased in individuals diagnosed with periodontal disease relative to those without the condition.[24]

Osteonectin

Osteonectin seemed to be a more sensitive indicator for detecting periodontal disease status than any other biomarkers.[25]

Osteopontin

OPN may serve as a promising indicator of periodontal disease advancement, facilitating early diagnosis.[26]

Markers of Cell Death and Connective Tissue Degradation Products

Fibronectin

Fibronectin and its breakdown products are believed to contribute to the inflammatory process.

Salivary Biomarkers

The key challenge in salivary diagnostics lies in unlocking its potential and refining technological innovations for application with this biological fluid. Scientists anticipate that various aspects of human health and disease will be detectable in saliva through proteomic and genomic markers.[27]

GCF as a Diagnostic Marker

Gingival crevicular fluid (GCF) contains increased concentrations of numerous biochemical markers that provide valuable insight for accurately diagnosing disease activity[28]." The future method of GCF analysis :

Prostaglandin E2

In 1997, Page observed significantly increased levels during the active phase of periodontitis.[29]

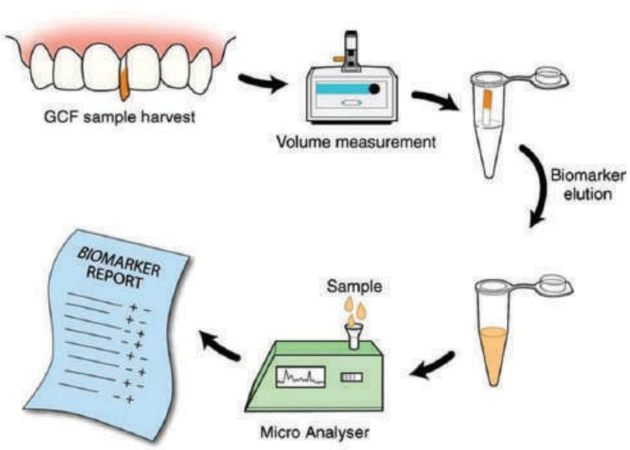


Figure 5 : Workflow for Analysis of Biomarkers from Gingival Crevicular Fluid (GCF)

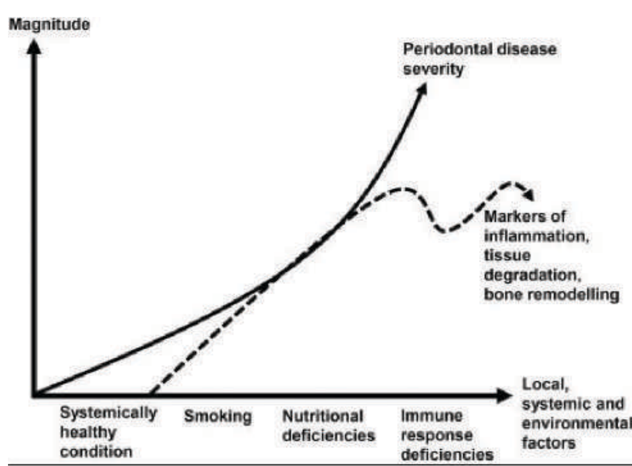


Figure 6: Relationship Between Local, Systemic, And Environmental Factors And The Progression/Severity Of Periodontal Disease, Alongside The Biomarker Response Pattern.

β-glucuronidase

Studies confirmed that the levels increase sixfold in periodontal diseases.[30]

Neutrophil elastase

Human studies have shown that NE levels increase in GCF in periodontitis.[31]

Collagen in GCF

Garnero (2003) identified changes in collagen breakdown products associated with disease conditions, which are evident in GCF and indicate active periodontal disease.

Proteoglycans

Research by Page and Kornman demonstrated elevated levels of proteoglycans in gingival crevicular fluid (GCF) during the active phase of periodontitis.

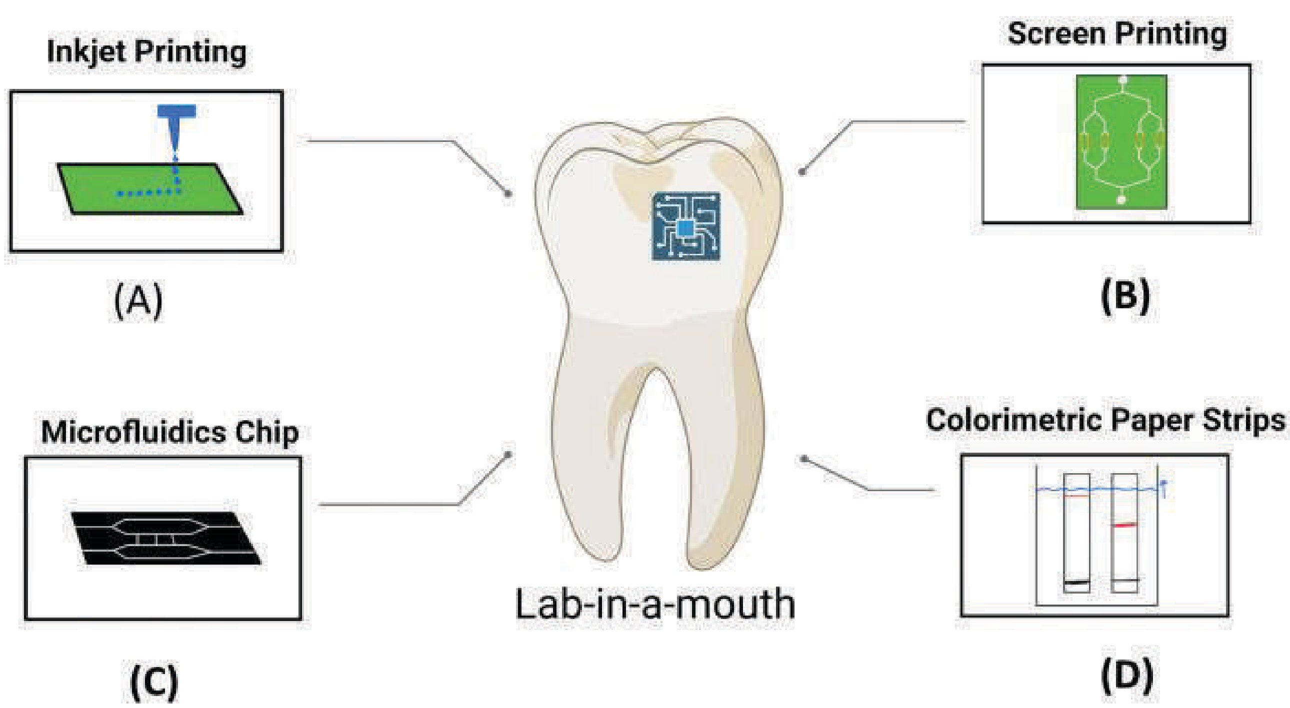
Glycoproteins

Paster in 2001 reported that the glycoprotein concentration decreases in GCF when the healing process takes place in periodontium.[32]

Recent Advances in Biomarkers[33]

Oral fluids hold vast potential for future healthcare applications, particularly in diagnostics. Advances have been made—from traditional periodontal probing to advanced genetic susceptibility testing and molecular biomarker arrays—that enhance our understanding of the mediators involved in periodontitis progression. The discovery of biomarkers has facilitated the development of new therapies that integrate diagnostic and treatment strategies for periodontal disease.

Furthermore, innovative diagnostic tools like microarray and microfluidics are now accessible for biomarker screening and risk evaluation. The future looks promising for rapid, user-friendly diagnostics that will improve patient evaluation and enable personalized, targeted dental treatments, ultimately enhancing oral healthcare. salivary diagnostics is recently, based on development of microfluidics or micro/nanoelectromechanical systems (MEMS/NEMS) consisting of electrical, mechanical, and functional elements such as sensors, actuators, and microelectronics which permit to measure proteins, DNA, transcripts (mRNA), electrolytes, and small molecules present in the saliva. The modern tools also includes electrochemical sensing, lab-on-chip, RT-PCR, fibre optic microsphere-based arrays, high-throughput DNA microarrays, resonance-based fiber optic sensors, and microchip-based electrophoretic immunoassay.[34] The new possibility of point-of-care diagnostics for "lab on-a- chip" helps



in detecting multiple biomarkers, and diagnosis of many diseases. It seeks to integrate and automate the complexities of a laboratory procedure into a device of the size of a computer chip.

Point of care (POC) diagnostic devices represent a method of clinical monitoring and testing that allows for quick and accurate identification of the medical malady in the office or while the patient is close at hand. POC diagnostic devices can rapidly identify biologically relevant substances like blood glucose levels, liver enzymes, blood in the stool, pregnancy, and the presence of drugs in the blood or urine amongst others.[42] Though many varieties of POC devices exist, diagnosis of infectious diseases is of great global interest. The World Health Organization's criteria for an effective POC test for such cases are summarized by the acronym ASSURED; the device must be affordable, sensitive, specific, user-friendly, rapid and robust, equipment-free, and deliverable to end users. This has been updated to account for real-time health monitoring with a revision to the ASSURED criteria, now termed REASSURED, to include real-time connectivity and ease of specimen collection to the initial standards.40,.41The oral cavity is more easily accessible to health care providers in a less invasive manner than the GI tract, vascular system, or other body parts. Rapid and accurate diagnosis of oral pathologies and their pathogenic basis is beneficial to ensure that the most appropriate treatment is used and the illness can be treated quickly. Fluids accessible and assessable in the mouth, like saliva, mouth rinse, peri-implantitis fluid (PISF), and gingival crevicular fluid (GCF), allow noninvasive testing for notable biomarkers relevant to both specific oral diseases like periodontitis and systemic issues.[43]

Recent Biomarkers

Obesity Related

Chemerin - an adipokine

Leptin - The decreasing leptin level in GCF and gingival tissue was associated with a deteriorated periodontal status, and smokers also showed reduced GCF leptin levels in recent studies.[35]

Systemic Disease Related

Progranulin – CP & type 2 DM.

Capsase 3 - GCF and the serum concentration of caspase-3 proportionally increases with the progression of periodontal disease.[36]

IL-29 - antiviral IL-29 level was highest in GCF of aggressive periodontitis patients while that of chronic periodontitis lying in between.

After non-surgical periodontal therapy, IL-29 levels increased both in chronic and aggressive periodontitis patients a potential therapeutic agent in treating periodontitis.

Conclusion

This is a promising area for further investigation, and more research is needed to identify the set of biomarkers with the most favourable combination of sensitivity, specificity, and positive/ negative predictive values. Recent advances in salivary proteomics and gene transfer techniques targeting the salivary glands will promote the creation of biomarkers with diagnostic and/or prognostic significance.[37]

Though several products show potential benefit; which gives a clue as to which tissue components are at risk, most of the test kit, or biomarkers yield little or no additional information, at high costing. It is also clear that no single marker has been able to fulfil all the criteria necessary for assessment of the clinical state of the periodontium. Future research should be directed possibly at the production of "marker packages" As of now various efforts are on to develop an ideal test, but actual use as a chairside diagnostic is still illusive. Therefore the development of a wide spectrum of markers is the primary goal of periodontal research.[38,39]

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SPIK MARVEL–2025:
A Milestone Midterm Conference at MAHE

The Department of Periodontics, MAHE Institute of Dental Sciences & Hospital (MINDS), in association with the Society of Periodontists and Implantologists of Kerala (SPIK), successfully organized the SPIK Midterm Conference – MARVEL 2025 on 18th and 19th October 2025 at MAHE.

The two-day academic event was designed to help postgraduate students integrate seamlessly into their academic journey, understand evolving program expectations, and gain insights from experienced clinicians and academicians. The conference featured seven scientific lectures and two engaging panel discussions, offering participants a comprehensive perspective on contemporary periodontology and implantology.

The program was inaugurated by Dr. Anil Melath, Principal, MAHE Institute of Dental Sciences & Hospital. Dr. Arun Sadasivan, Professor and Head, Department of Periodontics, and President, SPIK, graced the occasion as the Chief Guest. The formal inauguration ceremony was held at Navatara Auditorium, MAHE.

The first day comprised four enlightening lectures delivered by distinguished speakers, including Dr. Arun Sadasivan, Dr. Harikumar, Dr. Raj A.C., and Dr. Biniraj. A thought-provoking panel discussion on "Basal versus Conventional Implants" followed, stimulating active academic interaction among delegates. The day concluded with a grand banquet hosted at Ritz International, MAHE, fostering camaraderie among the nearly 100 registered delegates and faculty members from across Kerala.

The second day commenced with a keynote address by Dr. R.V. Chandra, followed by lectures from Dr. Deepthi Cherian and Dr. Baiju R.M. A dynamic panel discussion on the "Role of Biomarkers in Periodontal Diagnosis and Treatment," moderated by Dr. Arun Sadasivan, featured Dr. Harish Kumar, Dr. Jayan Jacob, and Dr. Baiju, and provided valuable clinical insights.

Scientific sessions included e-poster and paper presentations across review, original research, and case report categories, showcasing the academic rigor and enthusiasm of postgraduate delegates. Certificates of participation were awarded to all delegates, and certificates of merit (First, Second, and Third) were presented to winners in the respective scientific categories.

The conference received overwhelmingly positive feedback from participants, reflecting its academic depth, organizational excellence, and relevance to contemporary periodontal practice. The program concluded with a valedictory function at 2:00 PM, marking the successful culmination of SPIK MARVEL 2025.

Journal of the Society of Periodontists, Kerala (JSPIK)

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