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Contents

President's message

Dear members of the SPIK family,

It gives me immense pleasure and a deep sense of responsibility to address you as the President of the Society of Periodontists and Implantologists of Kerala (SPIK). I sincerely thank all the members for reposing their confidence in me and selecting me for this position. I am truly grateful for the trust and support extended to me, and I look forward to working together with all of you towards the continued growth and advancement of our speciality.

I would also like to express my heartfelt appreciation to every member who supported us with their presence, participation, and encouragement during the Annual Conference of SPIK, held on May 8, 9, and 10, 2026, at Mar Baselios Dental College. The enthusiasm and camaraderie displayed during the conference were truly encouraging and made the occasion memorable.

I extend my warm congratulations and appreciation to Dr Anjhana Narayanan, Editor, JSPIK, for her relentless efforts in bringing out yet another issue of our journal on time. The quality of its academic activities strengthens a professional society, and our journal remains an important platform for sharing knowledge and fostering research among our members. As we actively pursue avenues for indexing JSPIK, I request that all members contribute high-quality original research, reviews, case reports, and other scholarly articles. The quality and consistency of our contributions will be fundamental to establishing JSPIK as a strong and credible academic publication.

As we all know, Periodontics is the backbone of dental practice. The periodontal foundation upon which successful restorative, prosthodontic, orthodontic, implant and other dental treatments are built cannot be overemphasised. Yet, unfortunately, this fundamental role of the periodontist and the importance of periodontal health are still not adequately understood by other dental specialists, general dental practitioners, the medical fraternity and, most importantly, the public at large. This gap in understanding calls for concerted and sustained efforts from all of us.

In the current scenario, the digital domain plays a significant role in shaping perceptions and influencing healthcare-seeking behaviour. Recognising this changing landscape, SPIK intends to establish a stronger presence in digital media, particularly through awareness reels that focus on specific aspects of periodontal health, disease prevention, and the role of the periodontist. We have already released a couple of such initiatives, which have received wide appreciation. I congratulate Dr Anjali Babu, Web Administrator at SPIK, for her initiative and dedicated efforts in bringing our speciality closer to the public through these platforms. I encourage all members to support and actively participate in these initiatives so that the message of periodontal health reaches a much wider audience.

Alongside these new initiatives, we shall continue our regular academic and professional activities, including the SPIK Essay Competition, Mid-term Conference, SPIK Undergraduate Scholarship Examination and the Interdisciplinary Cricket Tournament, before culminating in our next Annual Conference. Each of these programmes provides an opportunity to strengthen academic interaction, encourage young professionals, foster fellowship and bring our members together.

Ultimately, all our endeavours have a common objective—to advance Periodontics, strengthen the professional identity of the periodontist and improve periodontal care for our community. This can be achieved only through collective effort. I therefore seek the continued support, cooperation and active participation of every member of SPIK.

Let us work together to build a stronger academic community, raise awareness of Periodontics, and ensure that the importance of periodontal health receives the recognition it truly deserves.

With warm regards and best wishes,

Dr Jayan Jacob Mathew

President -SPIK

Secretary's Message

Dear Members of SPIK and Esteemed Readers,

Greetings!

As we journey through this vibrant mid-year season — from the auspicious dawn of Vishu to the spirit of reflection during Easter and Eid, and looking forward to the joy of Onam — this period between April and July is truly a time of renewal, joy and collective growth. I extend my warmest wishes to all our members and their families.

It gives me immense pride to present the latest issue of the 'Journal of the Society of Periodontists in Kerala' (JSPIK). Our journal continues to be a beacon of academic excellence and clinical insight for periodontists across the state.

I would like to express my heartfelt gratitude to our President, Dr. Jayan Jacob Mathew, for his visionary leadership, steadfast guidance and tireless dedication in elevating the academic standards of our society.

A special word of thanks to our Editor, Dr. Anjana Narayanan,whose persistent hard work, meticulous editorial eye and unwavering commitment to quality have been instrumental in shaping this issue. Her dedication to bringing forth high-calibre research and clinical articles is steering JSPIK towards becoming one of the premier dental speciality journals in Kerala.

I wish the entire editorial team and all our contributors continued success. May our journal grow from strength to strength, enriching our professional community with evidence-based practice and scholarly excellence.

Wishing JSPIK and all our readers the very best!

Warm regards,

Dr. Deepak Thomas
Secretary, SPIK

Editorial

Dear Members and Colleagues,

It gives me immense pleasure to welcome you to the July issue of the Journal of Periodontology and Implantology of Kerala

As our profession continues to evolve, the practice of Periodontology and lmplant dentistry is increasingly shaped by evidence, innovation, technology, and a deeper understanding of the biological principles that govern treatment outcomes. In this rapidly changing landscape, the role of a scientific journal extends beyond publishing research—it becomes a platform for critical thinking, academic exchange, and continuous professional growth.This issue brings together scholarly contributions that reflect the diversity and evolving nature of our specialty.

I would particularly encourage postgraduate students and young clinicians to embrace research and academic writing as an integral part of their professional journey. Every well-formulated question, carefully conducted study, and thoughtfully documented clinical experience has the potential to contribute to the advancement of our specialty.

I sincerely thank all our contributors and reviewers for their valuable efforts and continued support. I hope this issue provides you with knowledge that is not only intellectually enriching but also clinically relevant.

Dr Anjhana Narayanan
Editor, JSPIK

Smart Dental Implants: A Periodontal Perspective In Modern Implantology

Jasira K.[1], Sanjeev Ravindran[2], Shyamala Devi M.P[3]

ABSTRACT

Smart dental implants (SDIs) represent a significant advancement in implant dentistry by integrating nanotechnology, biosensors, and photobiomodulation therapy into conventional implant systems. Unlike traditional implants, SDIs have been designed to monitor peri-implant conditions in real time while providing therapeutic interventions to enhance tissue healing and prevent disease progression. Piezoelectric nanoparticles embedded within the prosthetic crown harvest mechanical energy generated during routine oral functions such as chewing and tooth brushing. This energy powers integrated light-emitting diodes that deliver localized photobiomodulation therapy, promoting peri-implant tissue regeneration, reducing inflammation, and inhibiting microbial colonization. In addition, embedded micro-sensors continuously assess critical parameters including pH, temperature, and bacterial activity, enabling early detection of peri-implant disease and timely clinical intervention. From a periodontal perspective, SDIs offer a biologically interactive approach that compensates for the absence of the periodontal ligament by providing continuous monitoring and therapeutic support. This review discusses the design, working mechanism, biological principles, periodontal applications, clinical significance, current limitations, and future prospects of smart dental implants.

Keywords: Smart dental implants; Photobiomodulation Therapy; Piezoelectric nanoparticles; Biosensors; Peri-implantitis; Periodontal regeneration.

Introduction

Dental implantology has evolved into a mainstream modality for replacing missing teeth and restoring oral function and aesthetics.[1] Despite the high long-term success rates of conventional dental implants, biological complications such as peri-implant mucositis and peri-implantitis continue to pose significant clinical challenges. These inflammatory conditions are primarily associated with bacterial biofilm accumulation and host immune responses, leading to progressive peri-implant bone loss and, ultimately, implant failure.[14,18] Furthermore, unlike natural teeth, dental implants lack the periodontal ligament (PDL) and its associated mechanoreceptors, resulting in the loss of proprioception and biologic feedback that are essential for regulating occlusal forces and maintaining peri-implant tissue homeostasis.[15]

The development of Smart Dental Implants (SDIs) has emerged as a promising strategy to overcome the biological and functional limitations of conventional dental implants by integrating advanced materials, energy-harvesting technology, embedded biosensors, and therapeutic functionalities for continuous monitoring and improved long-term clinical outcomes.[3,4] Recent advances in nanotechnology and biomedical engineering have enabled the development of these intelligent implant systems, which extend beyond passive tooth replacement by incorporating energy-harvesting materials, biosensors, and photobiomodulation technology into a single implant platform.

Additionally, SDIs are equipped with micro-sensors that monitor oral health metrics such as pH,

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temperature, and bacterial presence. This data is transmitted wirelessly to healthcare providers, enabling proactive intervention.[2] These capabilities facilitate continuous assessment of the peri-implant environment, allowing early detection of pathological changes and timely preventive intervention.

Mechanical forces generated during routine oral activities, such as mastication and tooth brushing, are converted into electrical energy by piezoelectric nanoparticles embedded within the prosthetic crown. The harvested energy powers integrated light-emitting diodes (LEDs) that deliver localized photobiomodulation therapy, promoting peri-implant tissue healing, reducing inflammation, and inhibiting microbial colonization without the need for external power sources.

By combining real-time biosensing with localized therapeutic delivery, Smart Dental Implants represent a transition from passive osseointegration to active biointegration. Their ability to continuously monitor peri-implant health while simultaneously providing therapeutic support has the potential to improve implant longevity, facilitate early disease prevention, and enhance long-term clinical outcomes. This review discusses the structural design, working mechanism, periodontal applications, clinical significance, current challenges, and future prospects of Smart Dental Implants.

Structural Design and Working Mechanism

The Smart Dental Implant (SDI) is based on a conventional screw-retained implant system comprising an implant fixture, abutment, and a modified prosthetic crown integrated with piezoelectric materials and microelectronic components (Fig. 1). Unlike conventional implant restorations, the SDI incorporates an energy-harvesting crown capable of converting functional oral forces into usable electrical energy.

The prosthetic crown contains barium titanate nanoparticles (BTNPs), a lead-free piezoelectric material that generates an electrical charge when subjected to mechanical deformation. During routine oral activities such as mastication and tooth brushing, deformation of the crown activates the BTNPs, producing electrical energy. The generated energy is temporarily stored in miniature capacitors and subsequently powers embedded light-emitting diodes (LEDs), eliminating the need for external batteries or charging systems.

The LEDs emit light at predetermined wavelengths, primarily blue light (405–450 nm) for antimicrobial activity and near-infrared light (810–850 nm) to promote photobiomodulation, tissue healing, and angiogenesis. The implant is designed to ensure uniform light distribution around the peri-implant tissues while preserving the structural integrity of the restoration.

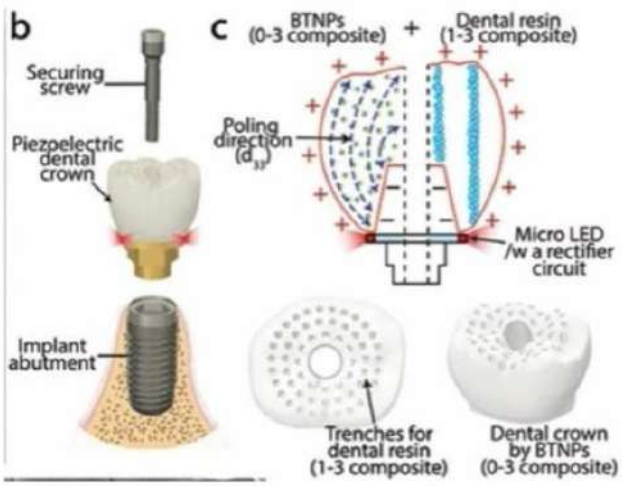

Figure 1.[1] Schematic illustration of the 1–3 composite Smart Dental Implant (SDI) showing the piezoelectric dental crown and comparison of the 0–3 and 1–3 composite designs. **Reproduced from Park et al.[1] **

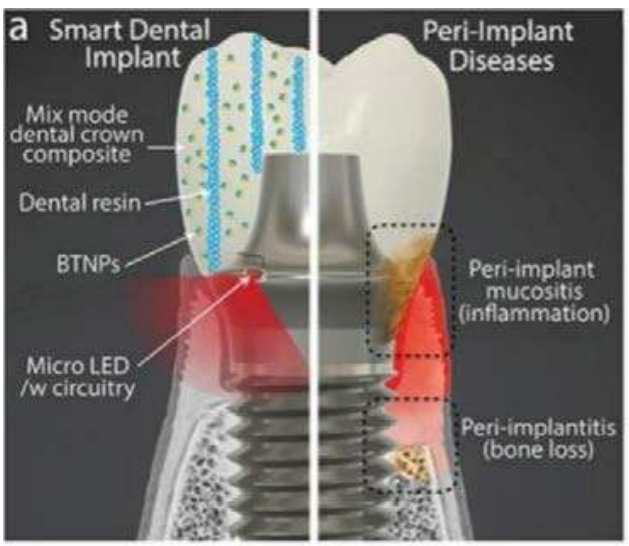

Figure 2. Smart dental implant

Two-Phase Composite Design

0–3 Composite: In this configuration, barium titanate nanoparticles are uniformly dispersed within a three-dimensional polymer matrix, maximizing piezoelectric responsiveness and improving energy-harvesting efficiency.

1–3 Composite: This configuration incorporates one-dimensional resin pillars within a three-dimensional BTNP-based composite, providing the mechanical strength required to withstand functional occlusal loading while maintaining efficient energy conversion (Fig. 1).

Beyond the piezoelectric energy-generation mechanism, SDIs incorporate miniaturized biosensors capable of continuously monitoring peri-implant environmental parameters, including pH, temperature, occlusal loading, and bacterial activity. The recorded data are transmitted wirelessly to external devices, enabling real-time assessment of peri-implant health and facilitating the early detection of pathological changes (Fig. 2).

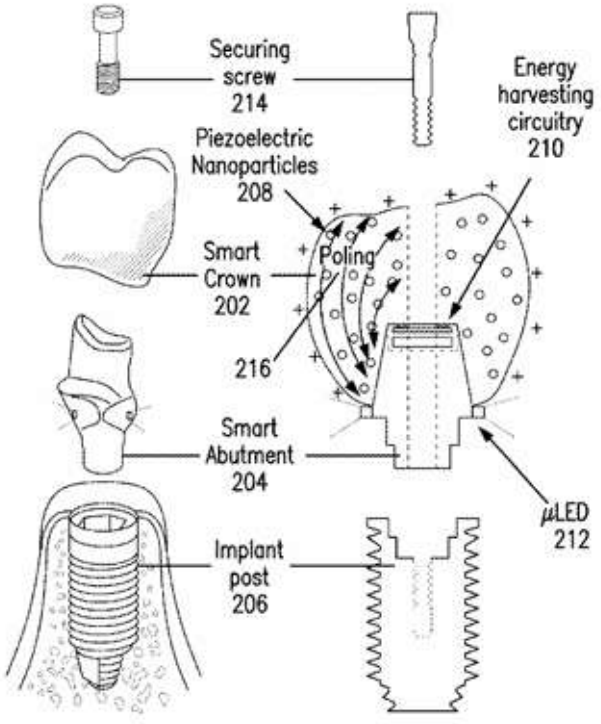

Figure 3.1 Smart Dental Implant (SDI) system showing the piezoelectric crown, energy-harvesting circuit, micro-LED, abutment, and implant post. **Reproduced from Park et al.[1] **

Collectively, the integration of piezoelectric energy generation, biosensing, and localized photobiomodulation transforms the SDI from a passive prosthetic replacement into an intelligent implant capable of continuously monitoring peri-implant health while providing therapeutic support to maintain peri-implant tissue stability.

Biomechanical Considerations and Energy Harvesting Efficiency

The efficiency of energy harvesting in Smart Dental Implants (SDIs) depends on the mechanical forces generated during routine oral function. Studies have shown that mastication produces forces ranging from 70 to 200 N at frequencies of approximately 1–5 Hz, while tooth brushing provides additional shear and compressive forces that contribute to piezoelectric activation. These physiological forces generate sufficient electrical energy to sustain photobiomodulation therapy (PBMT), producing an energy density of approximately 0.77 μJ/cm²/s at 5 Hz, equivalent to 4.1 mJ/cm² during 90 minutes of daily oral activity. This self-powered mechanism eliminates the need for external batteries or charging systems, ensuring continuous therapeutic function while improving patient convenience and long-term sustainability.

Photobiomodulation Therapy and Periodontal Healing

Photobiomodulation Therapy (PBMT), also known as low-level light therapy (LLLT), is one of the most significant therapeutic features of Smart Dental Implants (SDIs). Powered by the implant's piezoelectric energy-harvesting system, integrated light-emitting diodes (LEDs) deliver localized light at specific wavelengths, primarily within the blue and near-infrared spectra, without requiring an external power source.[1]

The therapeutic effects of PBMT are mediated through the absorption of photonic energy by cytochrome c oxidase within the mitochondrial respiratory chain, resulting in increased adenosine triphosphate (ATP) production and enhanced cellular metabolism. This process promotes fibroblast proliferation, collagen synthesis, angiogenesis, and tissue regeneration while modulating the inflammatory response, thereby accelerating peri-implant soft-tissue healing.[12]

In addition to its regenerative effects, blue-light phototherapy exhibits antimicrobial activity against major peri-implant pathogens, including *Porphyromonas gingivalis, Prevotella intermedia,* and *Treponema denticola*. By reducing bacterial colonization and inhibiting biofilm formation, PBMT helps maintain a healthier peri-implant environment and may reduce the risk of peri-implant mucositis and subsequent peri-implantitis.

From a periodontal perspective, localized photobiomodulation serves as a biologically active adjunct that supports peri-implant tissue homeostasis by promoting wound healing, controlling inflammation, and enhancing re-epithelialization of the peri-implant sulcus. These biological effects contribute to improved soft-tissue integration and may enhance the long-term stability of dental implants.

Biosensing and Real-Time Periodontal Monitoring

The integration of micro-sensors within Smart Dental Implants (SDIs) enables continuous, real-time assessment of the peri-implant environment. Embedded during the manufacturing process, these sensors monitor critical physiological and biochemical parameters, including pH, temperature, occlusal loading, bacterial metabolic activity, and implant displacement.[8,14]

Changes in these parameters provide valuable diagnostic information. A reduction in peri-implant pH may indicate anaerobic bacterial proliferation, whereas an increase in temperature is suggestive of an inflammatory response. Continuous monitoring of these biological markers, combined with wireless data transmission to dental professionals or patient mobile devices, facilitates predictive diagnostics and enables timely intervention before clinical signs of peri-implant disease become evident.[3]

From a periodontal perspective, this sensor-based surveillance closely mimics the biological monitoring function of the periodontal ligament (PDL), which continuously detects occlusal forces and provides sensory feedback to maintain tissue homeostasis. Although dental implants cannot fully reproduce the biological functions of the natural PDL, the incorporation of biosensing technology reintroduces an element of biological intelligence by enabling long-term surveillance of peri-implant health. This capability supports early disease detection, individualized maintenance strategies, and improved long-term management of peri-implant tissues.

Antimicrobial Coatings and Surface Optimization

Surface modification plays a key role in determining the biological success of dental implants. Smart

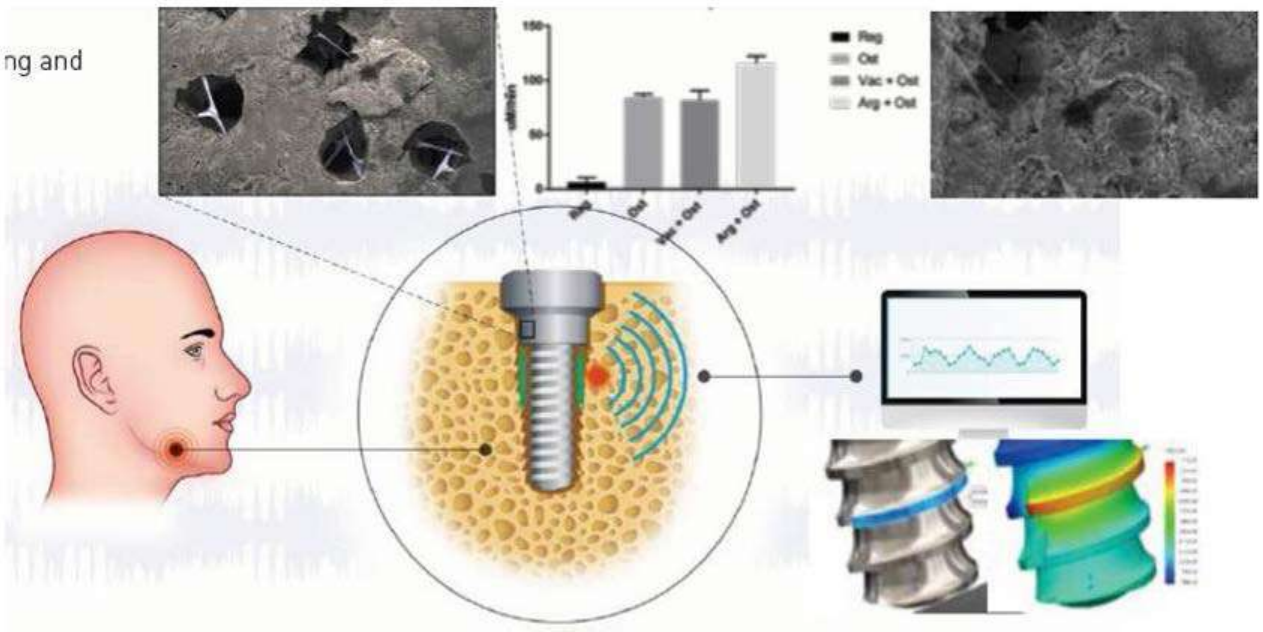

Figure 4.3 Integrated sensors in a Smart Dental Implant (SDI) illustrating real-time monitoring of peri-implant conditions and wireless transmission of physiological data. **Adapted from Varsha et al.3**

implants are often coated with antimicrobial piezoelectric materials, such as barium titanate nanoparticles (BTNPs) or zinc oxide nanoparticles (ZnO NPs), which inhibit bacterial adhesion and biofilm formation. Owing to their high surface area and enhanced surface reactivity, ZnO nanoparticles exhibit potent antibacterial activity through the generation of reactive oxygen species (ROS), including hydrogen peroxide (H_2O_2), hydroxyl radicals (•OH), and superoxide radicals (O_2•$^-$). These ROS induce oxidative stress by damaging bacterial cell walls, increasing membrane permeability, and disrupting intracellular proteins and nucleic acids. Furthermore, the release of Zn^{2+} ions interferes with bacterial metabolic processes, resulting in growth inhibition and eventual cell death.[16,17] Additionally, the implant surface can be engineered at the nanoscale to enhance fibroblast attachment while deterring bacterial colonization. Such nano-topographical cues improve the formation of a tight soft-tissue seal, crucial for maintaining the integrity of the peri-implant mucosa and preventing microbial invasion.

These surface features effectively recreate the protective role of the junctional epithelium and connective tissue attachment found in natural teeth, thereby compensating for the absence of the PDL and reinforcing the biological barrier function.

Periodontal Interface and Biological Integration

The interaction between peri-implant tissues and the implant surface plays a crucial role in determining the long-term success of dental implants. Histologically, the connective tissue fibers surrounding an implant are oriented parallel to the abutment surface, unlike the perpendicular fiber insertion observed around natural teeth. This structural difference renders the peri-implant mucosa more susceptible to bacterial penetration and soft-tissue breakdown. Smart Dental Implants (SDIs) address this biological limitation by combining photobiomodulation therapy with advanced surface modifications that promote fibroblast proliferation, collagen maturation, and soft-tissue regeneration. These biological effects enhance the quality and stability of the peri-implant mucosa, strengthen the mucosal seal, and support the maintenance of the biologic width. Consequently, SDIs establish a more favourable peri-implant tissue interface that improves resistance to microbial invasion, promotes long-term tissue stability, and reduces the risk of peri-implant disease.

Clinical Relevance in Peri-implant Disease Management

Peri-implant diseases, including peri-implant mucositis and peri-implantitis, share similar pathogenic mechanisms with periodontitis, originating from bacterial biofilm accumulation and a host-mediated inflammatory response that may result in progressive soft-tissue destruction and peri-implant bone loss.[18,14] Early diagnosis is critical because peri-implant mucositis is reversible, whereas the management of peri-implantitis is often complex, unpredictable, and frequently associated with limited success following conventional nonsurgical therapy[10].

Smart Dental Implants (SDIs) offer a novel strategy for peri-implant disease management by integrating diagnostic and therapeutic capabilities within a single implant system. Through continuous assessment of the peri-implant environment, SDIs can identify early biological alterations before overt clinical or radiographic manifestations develop, enabling timely preventive intervention[2,3].

In the early stages of inflammation, localized photobiomodulation or photodynamic therapy delivered by the implant may suppress pathogenic microorganisms, modulate inflammatory responses, and accelerate peri-implant soft-tissue healing[4]. In more advanced peri-implantitis, adjunctive phototherapy may complement surgical or nonsurgical debridement by improving local microcirculation, stimulating cellular regeneration, and reducing postoperative inflammation, thereby enhancing treatment outcomes[5].

Furthermore, the real-time biological information generated by SDIs supports individualized supportive periodontal therapy by allowing clinicians to tailor maintenance intervals according to patient-specific risk profiles. This personalized approach has the potential to improve long-term implant survival, minimize peri-implant bone loss, and enhance the predictability of implant maintenance.[6]

Potential Benefits of Smart Dental Implants

The potential benefits of smart implants are vast and far reaching[9]. By continuously monitoring critical physiological parameters, these devices can:

Enable early detection of complications

- Sensors can detect subtle changes in temperature, pressure, or biochemical markers.
- These signals may indicate the onset of infection, inflammation, or implant failure before clinical symptoms appear.

Optimize treatment delivery

- Smart implants can be engineered to release therapeutic agents in response to real-time physiological data.
- Ensures patients receive precise medication dosages at the optimal time, improving treatment efficacy.

Facilitate personalized rehabilitation

- Motion and activity sensors track patient movement and activity levels.
- Provides valuable data for rehabilitation programs, allowing clinicians to tailor interventions to individual needs.

Improve long-term implant performance

- Continuous monitoring offers insights into mechanical and biological behavior of implants over time.
- Supports refinement of implant design and materials, extending longevity and reducing revision surgeries.

Experimental Validation and Biocompatibility

Experimental studies have demonstrated that Smart Dental Implants (SDIs) possess excellent biocompatibility, mechanical stability, and regenerative potential. Both *in vitro* and *in vivo* investigations have shown favourable biological responses following the application of piezoelectric-powered photobiomodulation therapy. Human gingival keratinocyte cultures exposed to this technology exhibit enhanced cell viability and reduced expression of pro-inflammatory mediators, indicating improved cellular compatibility and modulation of the inflammatory response. Furthermore, animal studies have demonstrated enhanced osseointegration, greater peri-implant soft-tissue attachment, and improved healing when compared with conventional implant systems. Collectively, these findings support the potential of SDIs to promote favourable biological integration and improve peri-implant tissue stability, highlighting their promise as a next-generation approach in implant dentistry.

Periodontal Implications and Future Integration

For the field of periodontology, SDIs present an opportunity to redefine the approach to implant maintenance and regenerative therapy. The integration of biosensors allows the monitoring of peri-implant tissue health similar to periodontal probing but in a non-invasive, continuous manner. The data collected could be integrated with artificial intelligence to predict disease patterns, enabling preventive periodontal strategies. Additionally, photo biomodulation could complement traditional regenerative therapies such as guided bone regeneration or platelet-rich fibrin application by enhancing tissue healing responses. The ultimate goal is to create a bio-intelligent implant–periodontal complex that functions symbiotically with the host tissue, capable of self-maintenance and early disease interception.

Challenges and Future Perspectives

Despite their considerable potential, several scientific, technical, and regulatory challenges must be addressed before Smart Dental Implants (SDIs) can be widely adopted in routine clinical practice. The fabrication of implants incorporating piezoelectric materials, biosensors, and embedded microelectronics requires sophisticated manufacturing techniques, resulting in high production costs and increased design complexity. Additional challenges include ensuring the long-term biocompatibility and durability of embedded electronic components, preventing moisture infiltration within the oral environment, maintaining reliable wireless data transmission, and establishing sustainable energy generation within a compact implant design. Furthermore, stringent regulatory approval processes for hybrid biomedical devices and the need to safeguard patient data and cybersecurity remain important considerations.[13]

Future research should focus on optimizing piezoelectric materials and energy-harvesting efficiency, improving the mechanical properties of implant components through advanced biomaterials such as zirconia-based composites, and developing biocompatible encapsulation strategies to protect embedded electronics. Advances in nanostructured

surface modifications, artificial intelligence, wireless communication technologies, and digital health platforms are expected to further enhance the diagnostic and therapeutic capabilities of SDIs. With continued interdisciplinary research and technological innovation, Smart Dental Implants have the potential to become more reliable, cost-effective, and clinically accessible, ultimately facilitating the transition toward intelligent, personalized implant therapy.

Discussion

Peri-implant diseases remain one of the principal biological complications affecting the long-term success of dental implants. Clinical evidence suggests that implant failure is closely associated with an exaggerated host immunoinflammatory response and impaired osseointegration rather than mechanical factors alone[6,7]. Consequently, strategies that can simultaneously promote tissue regeneration, reduce inflammation, and facilitate early disease detection are likely to improve long-term implant outcomes.

Smart Dental Implants (SDIs) represent a significant advancement over conventional implant systems by integrating diagnostic, therapeutic, and regenerative functions within a single implant platform. Unlike traditional implants, which primarily provide mechanical support, SDIs combine piezoelectric energy harvesting, biosensing technology, and photobiomodulation therapy to create a biologically responsive implant capable of interacting with the peri-implant environment. This multifunctional approach has the potential to improve peri-implant tissue homeostasis, enhance osseointegration, and reduce the incidence of peri-implant disease.

From a periodontal perspective, SDIs address several inherent limitations of conventional implants. Continuous assessment of peri-implant biological conditions, combined with localized therapeutic intervention, may facilitate earlier diagnosis and more effective management of peri-implant mucositis before progression to peri-implantitis. Furthermore, the integration of regenerative phototherapy and intelligent biosensing supports a more personalized approach to supportive periodontal therapy, with maintenance protocols tailored according to individual patient risk profiles.

Despite these promising advantages, several challenges remain before SDIs can be routinely incorporated into clinical practice. Manufacturing complexity, long-term durability of embedded electronic components, regulatory approval, data security, and cost-effectiveness require further investigation through well-designed preclinical studies and long-term clinical trials.

Overall, Smart Dental Implants represent an important step toward intelligent implant systems that extend beyond passive tooth replacement. As advances in biomaterials, nanotechnology, biosensing, and artificial intelligence continue to evolve, SDIs have the potential to transform implant dentistry by enabling predictive diagnostics, personalized therapy, and improved long-term peri-implant health[9,12].

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Can Synbiotic Replace Chlorhexidine In Periodontal Care? A Split-Mouth Randomized Controlled Clinical Trial

Lakshmi Damodaran[1], Aswathy M[2], Mohammed feroz TP,[3] Megha Varghese[4], Nikhila TM[5], Anjali Babu[6], Dilsha[7]

ABSTRACT

Background: Periodontitis is a chronic inflammatory disease characterized by the destruction of periodontal supporting tissues due to bacterial infection. Although scaling and root planing (SRP) remains the gold standard treatment, adjunctive antimicrobial therapies are often required for improved clinical outcomes. Synbiotics, a combination of probiotics and prebiotics, have emerged as a promising alternative to conventional antimicrobial agents such as chlorhexidine.

Aim: To evaluate the clinical efficacy of a locally delivered probiotic-prebiotic mixture compared with chlorhexidine gel as an adjunct to scaling and root planing in the treatment of chronic periodontitis.

Materials and Methods: A split-mouth randomized controlled clinical trial was conducted on 15 participants diagnosed with chronic periodontitis. Two periodontal sites in each participant with probing pocket depth (PPD) ≥5 mm were selected. Following SRP, Group A (test) received a locally delivered probiotic-prebiotic paste, while Group B (control) received chlorhexidine gel. Clinical parameters including probing pocket depth (PPD) and clinical attachment level (CAL) were recorded at baseline and after one month.

Results: Both groups demonstrated statistically significant improvements in PPD and CAL after one month. The test group exhibited greater reductions in both parameters compared to the control group. However, intergroup differences were not statistically significant.

Conclusion: Locally delivered synbiotic therapy is as effective as chlorhexidine gel when used as an adjunct to SRP and may serve as a patient-friendly alternative for periodontal maintenance.

Keywords: Chronic periodontitis, Synbiotics, Probiotics, Prebiotics, Chlorhexidine, Scaling and root planing, Local drug delivery.

Introduction

Periodontal disease is a common chronic inflammatory condition affecting the supporting structures of teeth and remains a major cause of tooth loss in adults. The disease is initiated by pathogenic microorganisms residing within periodontal pockets, leading to progressive destruction of periodontal ligament, alveolar bone, and connective tissues.[1]

The primary objective of periodontal therapy is the elimination or reduction of periodontal pocket depth to prevent further disease progression and creating a periodontal environment that facilitates effective plaque control and maintenance of oral hygiene.

Scaling and root planing

(SRP) is the cornerstone of nonsurgical periodontal therapy.[2] However, despite meticulous SRP, residual periodontal pockets and persistent inflammation may remain at certain sites, potentially compromising treatment outcomes. Consequently, the use of adjunctive therapeutic modalities has been advocated to enhance the clinical efficacy of SRP and promote

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periodontal healing.[3]

Local drug delivery (LDD) systems have gained popularity because they provide sustained therapeutic concentrations directly within periodontal pockets while minimizing systemic side effects. Chlorhexidine has long been considered the gold standard antimicrobial agent in periodontal therapy.[4] However, increasing concerns regarding antimicrobial resistance and adverse effects associated with prolonged antimicrobial use have prompted the search for alternative treatment modalities.

Probiotics are defined as live microorganisms that confer health benefits on the host when administered in adequate amounts.[5] Common probiotic genera include Lactobacillus, Bifidobacterium, Streptococcus, Bacillus, and Saccharomyces. Prebiotics are non-digestible food ingredients that selectively stimulate the growth and activity of beneficial microorganisms. Common examples include inulin, fructooligosaccharides (FOS), galactooligosaccharides (GOS), xylooligosaccharides (XOS), resistant starch, and lactulose. microorganisms.When probiotics and prebiotics are combined, they form synbiotics, which provide synergistic benefits by enhancing the survival, colonization, and activity of beneficial microorganisms. The prebiotic component serves as a selective nutrient source for probiotics, improving their viability and therapeutic efficacy. Compared with probiotics alone, synbiotics may more effectively restore microbial balance, suppress pathogenic bacteria, modulate the immune response, and reduce inflammation, making them a promising adjunct in periodontal therapy.[7] Evidence from a systematic review and meta-analysis conducted by Martin-Cabezas et al. demonstrated that probiotic supplementation as an adjunct to SRP significantly improved periodontal clinical outcomes, including CAL gain and PPD reduction, when compared with conventional nonsurgical therapy alone.8

The present study was conducted to evaluate the clinical efficacy of a locally delivered probiotic-prebiotic mixture compared with chlorhexidine gel in patients with chronic periodontitis.

Materials and methods

The present study was designed as a split-mouth randomized controlled clinical trial and was conducted in the Department of Periodontology at Kannur Dental College over a period of six months, as this duration is considered sufficient to evaluate the short-term clinical effects of the intervention, including periodontal healing, changes in clinical parameters, and stabilization of treatment outcomes following non-surgical periodontal therapy. A total of fifteen patients diagnosed with chronic periodontitis were enrolled in the study after obtaining written informed consent. Participants aged between 25 and 50 years with at least one periodontal pocket of ≥5 mm in two different quadrants were included. Smokers, medically compromised individuals, and patients who had undergone periodontal therapy or received antibiotic treatment within one month prior to the study were excluded.

Two periodontal sites with probing pocket depths ≥5 mm were selected from each participant and randomly allocated to either the test group or the control group using a split-mouth design. The test group (Group A) received scaling and root planing (SRP), followed by local delivery of Pre Pro® prebiotic–probiotic capsules containing β-glucan. Each capsule comprised β-glucan 50 mg and fructooligosaccharides (FOS) 100 mg as prebiotic components, along with Lactobacillus acidophilus (200 million), Lactobacillus bulgaricus (100 million), Lactobacillus casei (100 million), Lactobacillus plantarum (100 million), Lactobacillus rhamnosus (100 million), Bifidobacterium breve (100 million), Bifidobacterium infantis (100 million), Bifidobacterium longum (100 million), and Streptococcus thermophilus (100 million) as probiotic components. The control group (Group B) received SRP followed by local application of Hexigel (chlorhexidine gluconate 1% w/w). Baseline clinical parameters, including probing pocket depth (PPD) and clinical attachment level (CAL), were recorded before treatment.

Scaling and root planing were performed using an ultrasonic scaler and universal curettes. In the test group, a commercially available probiotic-prebiotic capsule preparation was mixed with distilled water to achieve a paste-like consistency. The resulting mixture was incrementally delivered into the periodontal pocket until it was completely filled up to the gingival margin. In the control group, chlorhexidine gel (Hexigel®) was placed into the periodontal pocket following SRP. Periodontal dressing was applied to both treatment

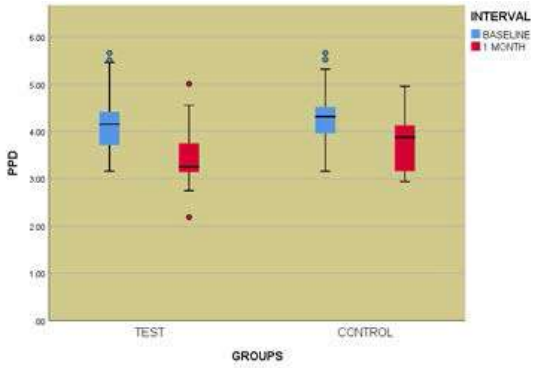
sites after the procedure. Clinical parameters were reassessed after one month to evaluate the outcomes of the interventions.

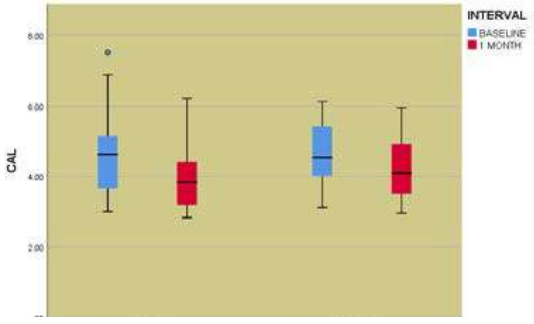
Statistical analysis

Data was analyzed using the statistical package SPSS 26.0 (SPSS Inc., Chicago, IL) and level of significance was set at P<0.05. Descriptive statistics was performed to assess the mean and standard deviation of the respective groups. Normality of the data was assessed using Shapiro Wilk test. Inferential statistics to find out the difference between the groups was done using Independent T test and Paired T test was used for Within group comparison.

Result

For the intergroup comparison, no statistically significant differences were observed between the test and control groups for either probing pocket depth (PPD) or clinical attachment level (CAL) at baseline or after one month . At baseline, the mean PPD was 4.22 ± 0.86 mm in the test group and 4.32 ± 0.91 mm in the control group, with a mean difference of 0.09 mm (p = 0.72), indicating comparable periodontal conditions between the groups. After one month, the mean PPD reduced to 3.49 ± 0.86 mm in the test group and 3.81 ± 0.92 mm in the control group. Although the test group showed a greater reduction, the mean difference of 0.32 mm was not statistically significant (p = 0.21). Similarly, the baseline CAL values were comparable between the groups, with mean values of 4.69 ± 1.07 mm and 4.65 ± 1.20 mm in the test and control groups, respectively, yielding a mean difference of 0.03 mm (p = 0.94). At one month, the mean CAL decreased to 3.98 ± 1.03 mm in the test group and 4.24 ± 1.23 mm in the control group. Despite a greater improvement in the test group, the mean difference of 0.26 mm was not statistically significant (p = 0.46). Overall, both groups demonstrated clinical improvement over the study period, but no significant intergroup differences were observed for PPD or CAL at any time point.





Discussion

The present study evaluated the efficacy of a synbiotic formulation as an adjunct to scaling and root planing (SRP) and compared its clinical outcomes with those achieved using chlorhexidine. The findings demonstrated improvements in periodontal parameters in both groups over the study period. Although the synbiotic group showed numerically greater reductions in probing pocket depth (PPD) and gains in clinical attachment level (CAL), the differences between the groups were not statistically significant after one month. These findings suggest that synbiotics may provide clinical benefits comparable to chlorhexidine while offering additional biological advantages.

Probiotic technology represents a novel and

biologically oriented approach to oral healthcare. Unlike conventional antimicrobial agents that exert their effects through broad-spectrum bacterial suppression, probiotics aim to restore and maintain a balanced oral microbiome by introducing beneficial microorganisms.[5] Meurman et al described probiotics as a natural defense mechanism that utilizes beneficial bacteria commonly present in healthy oral cavities to suppress pathogenic microorganisms associated with oral diseases.[9] This ecological approach is particularly relevant in periodontal therapy, where disruption of the microbial balance plays a central role in disease progression.

The favorable clinical outcomes observed in the synbiotics group may be attributed to the capacity of probiotic microorganisms to modulate the subgingival microbiota and suppress the growth of periodontal pathogens. Teughels et al. proposed that the administration of beneficial bacterial strains following scaling and root planing (SRP) may hinder the recolonization of periodontal pockets by pathogenic microorganisms. Following mechanical debridement, the periodontal environment undergoes a transient phase during which microbial recolonization occurs. The introduction of beneficial bacteria during this period may facilitate competitive exclusion of pathogenic species, thereby reducing their re-establishment and promoting the development of a balanced and health-associated microbial community.

Among the various probiotic strains investigated, Lactobacillus species have received considerable attention because of their documented oral health benefits. Haukioja et al.[8] demonstrated that probiotic lactobacilli can influence oral ecology by inhibiting the adhesion of pathogenic microorganisms and altering the protein composition of the salivary pellicle. Such modifications may reduce bacterial attachment and biofilm formation, thereby contributing to improved periodontal health. Furthermore, Lactobacillus species produce antimicrobial substances, including organic acids, hydrogen peroxide, and bacteriocins, which may directly suppress periodontal pathogens.

An important advantage of synbiotics over probiotics alone is the inclusion of prebiotics, which selectively promote the growth and survival of beneficial microorganisms. The prebiotic component serves as a nutritional substrate for probiotic bacteria, enhancing their viability, colonization potential, and metabolic activity within the oral environment. Previous studies have reported that the addition of prebiotics substantially increases the survivability of Lactobacillus species, thereby prolonging their beneficial effects.[10] Enhanced persistence of probiotic strains may improve microbial stability and contribute to sustained periodontal benefits.[11]

Chlorhexidine has long been regarded as the gold standard antiplaque and antigingivitis agent due to its broad-spectrum antimicrobial activity and substantivity. However, its long-term use is associated with several undesirable effects, including tooth staining, taste alteration, and oral mucosal irritation.[12] Although Hexigel is commercially indicated for topical oral application, Kotwal et al.[13] evaluated its use as a local drug-delivery agent following scaling and root planing in periodontal pockets >5 mm, supporting its subgingival application as an adjunctive chlorhexidine treatment. In contrast, synbiotics exert their therapeutic effects through microbial modulation rather than indiscriminate bacterial elimination and are generally associated with fewer adverse effects.[10] This ecological mechanism aligns with contemporary concepts of periodontal therapy that emphasize restoration of microbial homeostasis rather than complete eradication of oral microorganisms.

The absence of statistically significant differences between the groups in the present study may be attributed to several factors, including the relatively small sample size, short follow-up period, and the substantial clinical improvements resulting from SRP alone. Mechanical debridement remains the cornerstone of periodontal therapy, and the adjunctive effects of biological agents may require longer observation periods to become fully evident. Nevertheless, the comparable clinical outcomes observed suggest that synbiotics may represent a promising alternative or adjunct to chlorhexidine in periodontal care.

The findings of this study are consistent with previous investigations that have reported beneficial effects of probiotics and synbiotics on periodontal parameters, including reductions in plaque accumulation, gingival inflammation, and periodontal pocket depth. Further long-term randomized controlled trials with larger sample sizes and microbiological assessments are required to establish the optimal strains, dosages,

and duration of synbiotic therapy and to determine whether synbiotics can fully replace chlorhexidine in routine periodontal practice.

Within the limitations of the present study, synbiotics demonstrated clinical outcomes comparable to chlorhexidine when used as an adjunct to SRP. Their ability to promote beneficial bacterial colonization, inhibit pathogen recolonization, and avoid the adverse effects commonly associated with chlorhexidine suggests that synbiotics may serve as a biologically favorable alternative in periodontal maintenance and therapy.

Conclusion

Study concluded that both locally delivered synbiotic paste and chlorhexidine gel, when used as adjuncts to scaling and root planing (SRP), resulted in significant clinical improvements in the management of chronic periodontitis. The probiotic-prebiotic admixture, while relatively novel in periodontal therapy, considered as an effective and patient-friendly alternative to chlorhexidine, especially for long-term periodontal maintenance

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Medical practitioners' perspectives on managing patients under antiplatelet therapy during dental procedures: A questionnaire-based study

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ABSTRACT

Purpose: To evaluate medical practitioners' perceptions and practices in managing patients under antiplatelet therapy during dental procedures, focusing on therapy continuation versus interruption, perceived risks, and interdisciplinary collaboration.

Materials and Methods: A cross-sectional descriptive survey was conducted using a structured online questionnaire distributed via Google Forms. The survey included 14 multiple-choice questions and one open-ended item. Fifty actively practicing medical practitioners, including general practitioners, physicians, cardiologists, and neurologists, participated.

Results: Only 12.8% supported routine discontinuation of antiplatelet therapy before all dental procedures. The majority (51.3%) recommended stoppage only for high-bleeding-risk interventions, while 35.9% emphasized individualized decision-making. For minor procedures, 43.6% advised stoppage for 3 days, whereas one-third supported continuation. For high-risk procedures, 51.3% recommended stoppage for 3–5 days. Nearly 90% favored joint decisionmaking between dentist and physician, and thrombotic events were identified as the greatest risk of discontinuation. Open-ended responses reinforced case-by-case assessment, drugspecific considerations, and reliance on local hemostatic measures.

Conclusion: Medical practitioners generally do not favor routine stoppage of antiplatelet therapy before dental procedures. Continuation is widely accepted for low-risk interventions, while short-term interruption is considered for high-risk cases. The findings highlight the importance of interdisciplinary collaboration and evidence-based education to harmonize practice patterns and optimize patient safety.

Keywords: Antiplatelet therapy, Dental surgery, Bleeding, Thromboembolism

INTRODUCTION

Antithrombotic drugs are frequently used to prevent or treat various common cardiovascular disorders like acute coronary syndrome (ACS), stroke, peripheral vascular disease, atrial fibrillation (AF), and venous thromboembolism (VTE). Two main classes of oral antithrombotic drugs are on the market: antiplatelet drugs, which prevent inadvertent or inadequate platelet activation and initial clot formation, and anticoagulants, which slow down clot formation by controlling and reducing thrombin generation and formation of stable clots. Aspirin and P2Y12 inhibitors are the most commonly used antiplatelet drugs, either alone or as dual antiplatelet therapy (DAPT)[1].

Dental procedures in patients taking antithrombotic medications can be challenging, as the risk of bleeding from continuing antithrombotic therapy must be weighed against the thromboembolic risk associated with drug interruption or deescalation.[2] Minor dental procedures, such as simple tooth extraction, scaling and root planing , restoration, and endodontic treatment, generally pose minimal bleeding risk, whereas major oral surgeries, such as multiple complex extractions, gingival recontouring and flap

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raising, carry a higher bleeding risk[3]. Furthermore, some patient-related characteristics (e.g., advanced age, cirrhosis, chronic kidney disease, and a history of bleeding diathesis) are associated with an increased risk of bleeding after dental interventions, while others correlate with markedly elevated thromboembolic risk if antithrombotic drugs are discontinued. Therefore, managing antithrombotic therapy during dental procedures requires consideration of both procedural and patient-specific factors.[3]

Despite many years of experience, perioperative management of antiplatelet and anticoagulant drugs during dental procedures remains a dilemma with respect to balancing bleeding versus thrombotic risks. Multiple guidelines and recommendations by various societies have addressed the optimal management of these drugs in different surgical and invasive settings.

A major concern among dental practitioners is the potential for excessive bleeding after invasive dental procedures, and this often prompts them to stop long-term, low-dose antiplatelet therapy. Interestingly, studies have failed to demonstrate prolonged or excessive post-operative haemorrhage attributable to antiplatelet agents following simple dental extractions when compared to nonmedicated controls[4]. There are published studies highlighting the inherent danger of interrupting antiplatelet therapy. Studies concluded that patients who had clopidogrel therapy interrupted following coronary artery stenting were at a 10 times increased risk of fatality or re-hospitalisation compared to patients who had continuous therapy[5-9]. Some authors suspect the existence of a biological platelet rebound phenomenon when aspirin therapy is interrupted; this creates a prothrombotic state that may ultimately lead to a fatal thromboembolic event.[6-10]

Clinical evidence suggests that most bleeding episodes can be effectively managed with local hemostatic measures, yet concerns about perioperative bleeding still influence practice patterns across medical specialties.

In real-world settings, variability in clinical decision-making is evident, particularly regarding the timing of drug discontinuation and the authority responsible for final decisions. Understanding how general practitioners, physicians, cardiologists, and neurologists approach these scenarios provides valuable insight into current practice trends. This study therefore employed a questionnaire-based survey to map medical practitioners' knowledge and practices in managing patients under antiplatelet therapy during dental procedures, with emphasis on therapy continuation versus interruption, perceived risks, and the role of interdisciplinary collaboration.

Materials and Methods

This study employed a cross-sectional descriptive design using a structured questionnaire distributed via Google Forms. The questionnaire consisted of 14 multiple-choice questions and one open-ended item, designed to assess medical practitioners' knowledge, perceptions, and practices regarding the management of patients on antiplatelet therapy during dental procedures.

The study population included 50 actively practicing medical practitioners—general practitioners, physicians, cardiologists, and neurologists—who had direct or indirect experience in managing patients receiving antiplatelet therapy. The sample size was determined with reference to a study by Ozyilmaz .et .al using a proportion estimation formula with 95% confidence, yielding a minimum of 47.11 To strengthen validity, 50 actively practicing medical practitioners were included.

Inclusion criteria include currently practicing clinicians, while medical students, interns, retired practitioners, and dental surgeons were excluded to avoid bias.

Responses were collected anonymously, and only fully completed questionnaires were included in the analysis. Data were tabulated and expressed as percentages, with descriptive statistics used to summarize practice patterns. Open-ended responses were analysed to identify perspectives and insights.

RESULTS

Data obtained from the 50 respondents were analysed using descriptive statistics. Frequencies and percentages were calculated for categorical variables, including profession, year of clinical experience, prescribing habits, and opinions on antiplatelet therapy management. Results were presented in tabular form to highlight distribution patterns, and key interpretations were drawn from the proportions of responses. No inferential statistical tests were applied as the study was exploratory in nature and aimed at summarizing practitioner perspectives.

TABLE 1: Distribution of responses to survey questions (Q1–Q14) on antiplatelet therapy

Question	Response Options (%)	Percentage
Q1. Profession of respondents	General practitioners	40.0
	Physicians	30.0
	Cardiologists	15.0
	Neurologists	10.0
	Other specialties	5.0
Q2. Years of clinical experience	< 5 years	25.0
	5–10 years	30.0
	11–20 years	25.0
	> 20 years	20.0
Q3. Do you routinely prescribe antiplatelet drugs	Yes	70.0
	No	20.0
	Sometimes	10.0
Q4. How often do dentists refer to you for opinion?	Never	10.0
	Rarely	20.0
	Occasionally	40.0
	Frequently	30.0
Q5. Which dental procedure is most likely to cause significant bleeding?	Tooth extraction	92.3
	Endodontic (root canal)	17.9
	Dental implant placement	15.4
	Scaling	7.7
	Orthodontic treatment	5.1
	Crown preparation	2.6
	Simple restoration	0
Q6. Ranking of dental procedures by bleeding risk	Tooth extraction ranked 1 (highest)	48.7
	Dental implant placement ranked 2–3	35.9
	Endodontic treatment ranked mid-range	28.2
	Scaling & simple restoration ranked 6–7 (lowest)	82.0
Q7. How is bleeding usually controlled during dental procedures?	Pressure pack	48.7
	Depends on severity	46.2
	Combination of methods	35.9
	Sutures	25.6
	Topical agents	17.9
	Spontaneous hemostasis	5.1
	Systemic agents	0

Q8. Should antiplatelet therapy be routinely stopped?	Yes, always	12.8
	No, only for high-risk procedures	51.3
	Depends on patient risk	35.9
	No, never—continue regardless	0
Q9. For low-bleeding-risk procedures	Stop 5–7 days prior	7.7
	Stop 3 days prior	43.6
	Continue without interruption	33.3
	Continue without interruption	15.4
Q10. For high-bleeding-risk procedures	3-5 days	51.3
	5-7 days	38.5
	7-10days	10.2
	Not stopped; manage bleeding with local measures	<5
Q11. Who should make the final decision?	Dentist alone	<5
	Physician alone	<5
	Joint decision	89.7
	Patient	0
Q12. Greatest risk of stopping therapy	Thrombotic events	89.7
	Unsure	10.3
	Patient erratic compliance	0
	none	0
Q13. What is the main risk of not stopping (continuing) antiplatelet therapy during dental procedures	Excessive/ prolonged bleeding	89.7
	No significant bleeding risk	7
	Unsure	3.3
	Hemorrhagic complication requiring transfusion	0
Q.14 How influential are thrombotic risks (e.g., stent thrombosis) compared to bleeding risks in your decision-making	Thrombotic risk are more influential	56.4
	Equally balanced	30.8
	Bleeding risk are more influential	12.8

A total of 50 medical practitioners participated in the study. Of these, 40% were general practitioners, 30% were physicians, 15% were cardiologists, 10% were neurologists, and 5% were from other specialties.

Only 12.8% of respondents supported routine discontinuation of antiplatelet therapy before all dental procedures. The majority (51.3%) recommended stoppage only for high-bleeding-risk procedures, while 35.9% emphasized individualized decision-making based on patient risk factors such as recent stent placement. None favored universal continuation regardless of procedure type.

For minor, low-bleeding-risk procedures, 43.6% recommended stopping antiplatelet therapy three days prior, while 33.3% advised continuation without interruption. Smaller proportions suggested stoppage for 5–7 days (7.7%) or leaving the decision to the dentist based on INR values (15.4%).

When considering high-bleeding-risk procedures, 51.3% recommended stoppage for 3–5 days, 38.5% suggested 5–7 days, and 10.2% opted for 7–10 days.

Very few respondents favored continuation with local hemostatic measures or dentist-based INR decisions.

Decision-making authority was overwhelmingly considered a joint responsibility between dentist and physician (89.7%), with only small minorities supporting unilateral decision-making by either dentist or physician, and none assigning this responsibility to the patient.

Nearly all respondents (89.7%) identified thrombotic events such as stent thrombosis or stroke as the greatest risk of discontinuation. A small proportion (10.3%) were unsure, while none selected patient compliance issues, clot formation elsewhere, or "no risk."

In the open-ended responses, practitioners emphasized that management should be individualized, with decisions based on careful risk–benefit assessment, patient education, and informed consent. Several highlighted that aspirin alone carries a relatively lower bleeding risk, whereas clopidogrel, ticagrelor, and dual therapy combinations are associated with higher bleeding tendencies. A few stressed that stopping antiplatelets within three months of stenting carries a high risk of stent thrombosis, and in such cases, therapy may be reduced from dual to single antiplatelet to balance bleeding and thrombotic risks.

DISCUSSION

The present survey highlights that most medical practitioners do not favor routine stoppage of antiplatelet therapy before dental procedures. Instead, discontinuation was recommended primarily for high-bleeding-risk interventions, with short-term interruption (3–5 days) being the most common practice. This aligns with evidence from randomized controlled trials and systematic reviews, which consistently demonstrate that continuation of aspirin does not predispose patients to major bleeding events during dental extractions, and that minor bleeding can be effectively controlled with local hemostatic measures.[12-14]

In the current survey, 43.6% of respondents advised stopping therapy 3 days before low-risk procedures, whereas one-third supported uninterrupted continuation. Review articles, however, emphasize that most minor dental procedures (e.g., scaling, restoration, single extractions) pose a negligible bleeding risk, and guidelines generally recommend continuing aspirin monotherapy in such cases[15]. The discrepancy suggests a more conservative approach among practitioners, possibly reflecting concern about intraoperative bleeding rather than thromboembolic complications.

For high-bleeding-risk procedures, over half of respondents favored stoppage for 3–5 days, while 38.5% recommended 5–7 days. This is consistent with guideline suggestions that interruption of P2Y12 inhibitors may be reasonable in low thromboembolic risk patients undergoing invasive oral surgery, but continuation is advised in those with recent stent placement or high cardiovascular risk[16]. Importantly, no major bleeding events have been reported in randomized controlled trials where DAPT was continued, reinforcing the role of local hemostatic measures as the primary strategy[17]. It should be considered that high-bleeding-risk procedures (eg, corrective jaw or facial surgery and facial trauma repair by open techniques) were not tested in these RCTs.

Similarly, with newer oral anticoagulants (NOACs/DOACs) including rivaroxaban, apixaban, and dabigatran, most guidelines recommend continuation for minor dental procedures, while a brief 24hour pause may be considered for higher-risk surgeries in low-risk patients; uninterrupted therapy remains the standard in those with elevated cardiovascular risk[18].

Decision-making authority was overwhelmingly attributed to collaboration between dentist and physician (89.7%), which mirrors recommendations in review articles stressing interdisciplinary communication to balance bleeding and thrombotic risks. Finally, thrombotic events were identified as the greatest risk of therapy discontinuation by nearly 90% of respondents, echoing the consensus in published literature that the consequences of stent thrombosis or stroke far outweigh manageable dental bleeding.

Overall, the findings of this survey are broadly consistent with existing reviews and international guidelines. The majority of practitioners supported routine continuation of aspirin for minor dental procedures, while interruption of P2Y12 inhibitors (such as clopidogrel) was considered only in selected lowrisk patients undergoing highbleedingrisk surgery. Local hemostatic measures—pressure packs, sutures, topical agents—were the preferred strategies for managing intraoperative bleeding.

However, the slight tendency among respondents toward shortterm stoppage even in lowrisk procedures

underscores a gap between practice and evidence. According to the latest American Heart Association/American College of Cardiology perioperative guidelines, antiplatelet therapy should not be routinely discontinued for dental procedures, as the risk of thrombotic events (stroke, myocardial infarction, stent thrombosis) outweighs the manageable bleeding risk. Instead, individualized decisionmaking and reliance on local hemostatic techniques are strongly recommended.[19]

This highlights the need for continued education and dissemination of evidencebased recommendations to optimize patient safety and align clinical practice with current cardiovascular standards.

Conclusion

This questionnaire-based survey among currently practicing medical practitioners revealed that routine discontinuation of antiplatelet therapy before dental procedures is not widely supported. Most respondents recommended stoppage only for high-bleeding-risk interventions, with short-term interruption (3–5 days) being the most common practice. For low-bleeding-risk procedures, continuation was considered acceptable by a substantial proportion, reflecting awareness of guideline-based recommendations.

The overwhelming consensus favored joint decision-making between dentist and physician, underscoring the importance of interdisciplinary collaboration in balancing bleeding and thrombotic risks. Practitioners consistently identified thrombotic events as the greatest danger of therapy discontinuation, highlighting patient safety as the central concern.

Overall, the findings emphasize that clinical practice trends are broadly aligned with published reviews and guidelines, but also reveal a tendency toward conservative stoppage even in low-risk procedures. This underscores the need for continued dissemination of evidence-based recommendations to harmonize practice patterns and optimize outcomes for patients undergoing dental treatment while on antiplatelet therapy.

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Comparison of post-operative healing and stability of flap closure using autologous fibrin glue and Vicryl suture following periodontal flap surgery: A randomised controlled trial.

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ABSTRACT

Background: Sutures are routinely used for flap closure following periodontal flap surgery but may contribute to plaque accumulation, tissue reactivity, patient discomfort, and an increased risk of infection. Autologous fibrin glue has emerged as a biological tissue adhesive that may improve wound healing and flap stability.

Aim: To compare postoperative wound healing and flap stability following periodontal flap surgery using autologous fibrin glue and Vicryl sutures.

Materials and Methods: This randomized controlled clinical trial included 40 adjacent surgical sites in 20 systemically healthy patients requiring periodontal flap surgery. Sites were randomly assigned to receive either autologous fibrin glue (Group A) or Vicryl sutures (Group B). Wound healing was evaluated using the Landry Healing Index at 1 and 2 weeks, while flap stability was assessed using the Roll Test at baseline, 1 week, and 2 weeks.

Results: The fibrin glue group showed significantly better wound healing than the Vicryl group at 1 week (4.20 ± 0.41 vs. 3.00 ± 0.32; $p < 0.001$) and 2 weeks (4.90 ± 0.31 vs. 3.60 ± 0.50; $p< 0.001$). Flap stability was also significantly greater at baseline and 1 week ($p < 0.001$). Complete flap stability was achieved in both groups by 2 weeks. $P < 0.05$ was considered as statistically significant.

Conclusion: Autologous fibrin glue provides superior early wound healing and flap stability compared with Vicryl sutures and represents an effective alternative for periodontal flap closure.

Key words: Autologous fibrin glue,vicryl sutures, periodontal flap surgery, wound healing,flap stability

Introduction

Periodontal flap surgery is a widely performed surgical procedure for the treatment of periodontal disease. Successful healing following flap surgery largely depends on precise flap adaptation and stable wound closure. Suturing is considered an integral component of periodontal flap surgery, as it secures the flap in the desired position and facilitates optimal wound healing. However, conventional sutures have several disadvantages, including plaque accumulation, increased postoperative discomfort, tissue reactivity, higher risk of infection, and the need for suture removal[1]. These limitations have prompted the search for alternative methods of flap closure, including sutureless techniques[2].

Fibrin glue is one such biological tissue adhesive that mimics the final stage of the physiological coagula-

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tion cascade by forming a stable fibrin clot. In addition to providing tissue adhesion, it also eliminates dead space between tissue margins, reduces postoperative bleeding and inflammation, and provides excellent flap adaptation, particularly in aesthetically sensitive areas[3,4]. Apart from binding tissues, fibrin sealant also acts as a natural wound bed. This bed acts as a scaffold, allowing mesenchymal and endothelial cells to proliferate and differentiate more easily[5,6].

Vicryl® (polyglactin 910) is one of the most commonly used absorbable suture materials because of its favorable handling characteristics, adequate tensile strength, and minimal tissue reaction, Vicryl suture material is said to have faster wound healing properties and prevent microbial colonization[7,8]. However, sutures provide only point fixation of the flap, may serve as nidus for plaque accumulation, and can contribute to postoperative discomfort, highlighting the need for alternative flap closure techniques.

Fibrin glue is available as either commercially prepared products or autologous preparations. Commercially available fibrin sealants, such as Tisseel® and Reliseal®, have demonstrated favourable clinical outcomes; however, their routine use is limited by high cost, limited shelf life, multiple preparation components, restricted availability, and the potential risk of allergic reactions or transmission of blood-borne infections associated with donor-derived products[9]. In contrast, autologous fibrin glue is prepared from the patient's own blood, thereby minimising immunological and infectious risks while providing a cost-effective and biologically compatible alternative.

Although previous studies[12,15] have reported encouraging results with both autologous and commercially available fibrin sealants for periodontal flap closure, the available evidence remains limited, particularly regarding direct comparisons between autologous fibrin glue and conventional Vicryl sutures. Therefore, the present randomized controlled clinical trial was undertaken to compare postoperative wound healing and flap stability following periodontal flap surgery using autologous fibrin glue and Vicryl sutures.

The aim of the present study was to compare postoperative wound healing and flap stability following periodontal flap surgery using autologous fibrin glue and Vicryl sutures.

Materials and Methods

Study Design

A randomized controlled split-mouth clinical trial was conducted in the Department of Periodontology and Implantology, Educare Institute of Dental Sciences. The sample size was calculated based on a previous study[12], resulting in a total of 40 surgical sites (20 sites per group). Twenty systemically healthy patients requiring periodontal flap surgery were enrolled. Following flap reflection, the surgical flap was divided into two comparable adjacent flap segments (comprising two or more papillae each). Randomization was performed using the lottery method. An independent investigator, who was not involved in the surgical procedure or outcome assessment, prepared identical opaque sealed envelopes containing allocation chits labeled 'Autologous Fibrin Glue' and '5-0 Vicryl Suture'. After flap reflection and segmentation, one envelope was opened to determine the treatment allocation for each flap segment, ensuring allocation concealment until the time of intervention. One flap segment received closure using autologous fibrin glue (Group A), while the adjacent segment was closed using 5-0 Vicryl sutures (Group B). This was a single-blind study, the outcome assessor and statistician were blinded to the treatment allocation.

Ethical Aspects

The study protocol was reviewed and approved by the Institutional Ethics Committee of Educare Institute of Dental Sciences (Approval No. EIDS/IEC/2024-25/030). The trial was prospectively registered with the Clinical Trials Registry–India (CTRI/2026/06/112859). The study was conducted in accordance with the ethical principles of the Declaration of Helsinki and Good Clinical Practice guidelines. Written informed consent was obtained from all participants before enrolment

Study population

Inclusion Criteria

- Systemically healthy individuals aged 18–65 years.
- Patients with probing pocket depth (PPD) ≥ 5 mm requiring periodontal flap surgery.
- Patients demonstrating adequate oral hygiene

following Phase I periodontal therapy.

- Patients requiring flap surgery involving at least four adjacent teeth.
- Patients willing to participate and provide written informed consent.

Exclusion criteria:

- Patients with systemic diseases or receiving medications known to interfere with wound healing.
- Smokers, tobacco chewers, or individuals with other deleterious oral habits.
- Pregnant or lactating women.
- Patients with a known allergy to any medication used during the study.
- Patients unwilling to participate or unable to comply with the follow-up schedule.

Methodology

Patients fulfilling the inclusion and exclusion criteria and diagnosed with Stage III or Stage IV Grade B periodontitis according to the 2017 World Workshop Classification of Periodontal and Peri-Implant Diseases and Conditions[16] were recruited after obtaining written informed consent. All participants underwent Step 1 and Step 2 periodontal therapy in accordance with the European Federation of Periodontology (EFP) S3-level clinical practice guidelines[17], including oral hygiene instructions, supragingival plaque control, and subgingival instrumentation. Patients were re-evaluated following completion of therapy, and those demonstrating satisfactory plaque control, resolution of gingival inflammation, and residual probing pocket depth (PPD) ≥5 mm requiring open flap debridement (OFD) were scheduled for surgery. Open flap debridement was performed under local anaesthesia (2% Lignocaine hydrochloride with 1:80,000 adrenaline) using a conventional Kirkland flap following standard aseptic protocol. After thorough debridement and root surface instrumentation, the surgical flap was divided into two comparable adjacent flap segments (comprising two or more papillae each).

Surgical sites were randomly assigned to Group A or Group B. In Group A, flap closure was performed using autologous fibrin glue, whereas in Group B, flap closure was performed using Vicryl sutures employing simple interrupted suturing technique. After flap closure, flap stability was assessed using the Roll Test.

All patients received standardized postoperative instructions, antibiotic Amoxicillin 500 mg thrice daily and analgesic Ibuprofen 400 mg twice daily was prescribed for 5 days.Clinical evaluations were performed at 1 week and 2 weeks postoperatively.

Outcome Measures

Postoperative wound healing was assessed using the Landry Healing Index (1988) at 1 and 2 weeks. Flap stability was evaluated using the Roll Test immediately after flap closure (baseline), at 1 week, and at 2 weeks postoperatively.

Figure 1: Materials required

(a)calcium chloride (b) Protamine sulfate (c)sterile vacutainer containing 0.9% sodium citrate (d) 5-0 vicryl suture

Figure 2. Platelet-rich plasma and platelet-poor plasma separated after first centrifugation

Figure 3. Fibrinogen precipitate sediment after the second centrifugation.

Figure 4: Prepared Autologous Fibrin Glue ready for clinical application

Preparation of fibrin glue[1]

- Collection of blood: Approximately 10 mL of the patient's venous blood was collected into a sterile vacutainer containing 0.9% sodium citrate as an anticoagulant.
- First centrifugation: The blood sample was centrifuged at 3000 rpm for 10 minutes, resulting in three layers: Platelet-poor plasma (PPP), Platelet-rich plasma (PRP), Red blood cells (RBCs)
- Separation of plasma: The PPP and PRP fractions were carefully aspirated into a sterile test tube without an anticoagulant. The RBC layer was discarded.
- Addition of protamine sulfate: Protamine sulfate (10 mg/mL) was added to the plasma to precipitate fibrinogen.
- Second centrifugation: The mixture was centrifuged at 1000 rpm for 5 minutes, producing an upper serum layer containing autologous thrombin and a lower fibrinogen precipitate.
- Preparation of fibrinogen suspension: Most of the upper serum layer was discarded, leaving approximately 0.5 mL to resuspend the fibrinogen precipitate.
- Preparation of calcium chloride: A separate syringe containing 0.025 mmol/L calcium chloride was prepared.
- Application of fibrin glue: Equal volumes of the fibrinogen suspension and calcium chloride solution were simultaneously applied beneath the reflected periodontal flaps.
- Flap stabilization: Gentle digital pressure was maintained over the flap for 2–3 minutes to facilitate fibrin polymerization and achieve stable flap adhesion.

Statistical Analysis

The collected data were systematically entered in Microsoft Excel and analyzed using JASP statistical software (Version 0.19.7.1). Descriptive statistics were computed and expressed as mean ± standard deviation (SD) for all study variables. As the outcome variables (Roll Test and Wound Healing Index) were ordinal in nature, non-parametric statistical tests were employed. Intergroup comparisons at each time interval were performed using the Mann–Whitney U test, while intragroup comparisons across different time intervals were analyzed using the Friedman test. A p-value of < 0.05 was considered statistically significant for all analyses.

Results

All participants completed the follow-up period, and no postoperative complications or dropouts were reported.

Intergroup comparison of Roll Test scores demonstrated statistically significant differences between the two treatment groups at baseline and 1 week postoperatively. At baseline, Group

A (Autologous Fibrin Glue) exhibited a significantly lower mean Roll Test score than Group

B (Vicryl Suture) (0.65 ± 0.49 vs. 1.60 ± 0.50; $U = 52.0$, $p < 0.001$). Similarly, at 1 week,

Group A maintained significantly lower Roll Test scores than Group B (0.10 ± 0.31 vs. 1.05 ± 0.22; $U = 19.0$, $p < 0.001$. At 2 weeks postoperatively, no papillary movement was observed in either treatment group, indicating complete flap stability. These findings

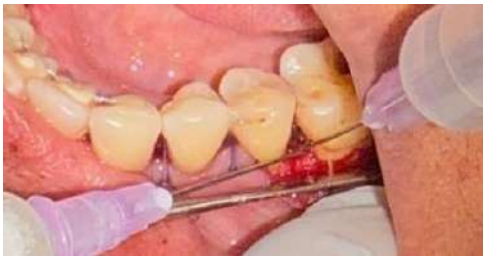

Figure 5: Intra-Operative

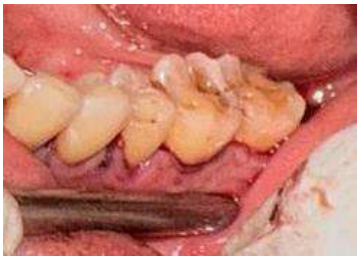

Figure 6: Checking Flap stability after Fibrin Glue application and Vicryl suture placement

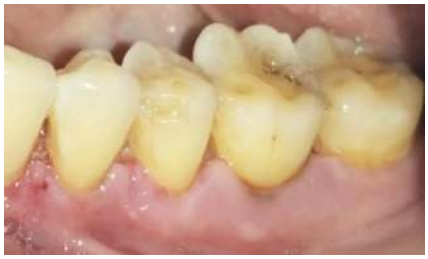

Figure 7: 1week post-operative view

suggest that while both treatment modalities ultimately achieved complete flap stability by 2 weeks, autologous fibrin glue demonstrated significantly better flap stability during the early healing period compared to Vicryl sutures.

Table 1: Criteria for Roll Test

Score	Criteria
Score 1	Barely visible movement of the papilla
Score 2	Clearly visible movement but no retraction of the papilla
Score 3	Clearly visible movement with retraction of the papilla

Table 2: Healing Index by Landry et al (1988)

Score		Criteria
1	Very poor	• ≥50% of the gingiva is red • Bleeding on palpation • Granulation tissue present • Incision margin not epithelialized, with loss of epithelium extending beyond the incision margin • Suppuration present
2	Poor	• ≥50% of the gingiva is red • Bleeding on palpation • Granulation tissue present • Incision margin not epithelialized, with connective tissue exposed • No suppuration
3	Good	• ≥25% and <50% of the gingiva is red • No bleeding on palpation • No granulation tissue • No connective tissue exposed at the incision margin
4	Very good	• <25% of the gingiva is red • No bleeding on palpation • No granulation tissue • No connective tissue exposed at the incision margin
5	excellent	• <25% of the gingiva is red • No bleeding on palpation • No granulation tissue • No connective tissue exposed at the incision margin

Within-group analysis using the Friedman test demonstrated a statistically significant improvement in flap stability over the postoperative period in both treatment groups. In Group A, the mean Roll Test score decreased significantly from 0.65 ± 0.49 at baseline to 0.10 ± 0.31 at 1 week and 0.00 ± 0.00 at 2 weeks (χ^2=22.62, df = 2, p<0.001). Similarly, Group B showed a significant reduction in Roll Test scores from 1.60±0.50 at baseline to 1.05 ± 0.22 at 1 week and 0.00±0.00 at 2 weeks (χ^2=37.21, df=2, p<0.001), indicating complete flap stability by the second postoperative week in both groups.

Intergroup comparison of wound healing scores revealed statistically significant differences between the two groups at both postoperative evaluations. At 1 week, Group A demonstrated a significantly higher mean wound healing score than Group B (4.20 ± 0.41 vs. 3.00 ± 0.32; U = 392.0, p < 0.001). At 2 weeks, Group A continued to show significantly higher wound healing scores than Group B (4.90 ± 0.31 vs. 3.60 ± 0.50; U = 388.0, p < 0.001).

Table 3: Inter-group comparison of Roll Test

Time Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	U-value	p-value
Baseline	0.6500 ± 0.4894	1.600 ± 0.5026	52	<0.001*
1 Week	0.1000 ± 0.3078	1.050 ± 0.2236	19	<0.001*
2 Weeks	0.000 ± 0.000	0.000 ± 0.000	NA	NA

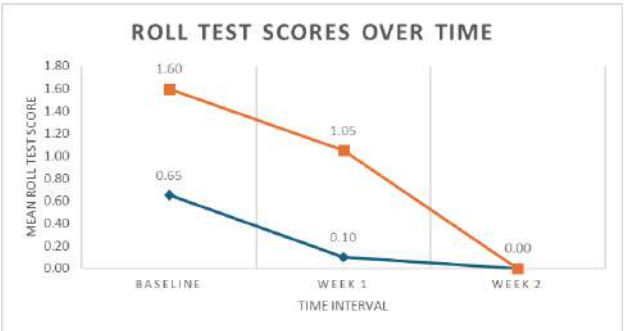

Figure 8: The line diagram depicting the comparison of Roll test score at baseline, 1 week and 2 weeks between two groups.

Table 4: Inter-group comparison of Wound Healing Index Scores

Time Interval	Group A (Mean ± SD)	Group B (Mean ± SD)	U-value	p-value
1 Week	4.200 ± 0.4104	3.000 ± 0.3244	392	<0.001*
2 Weeks	4.900 ± 0.3078	3.600 ± 0.5026	388	<0.001*

Within-group analysis using the Friedman test also demonstrated a statistically significant improvement in wound healing over time in both treatment groups. In Group A, the mean wound healing score increased significantly from 4.20 ± 0.41 at 1 week to 4.90 ± 0.31 at 2 weeks ($\chi^2 = 12.25$, df = 1, $p < 0.001$).

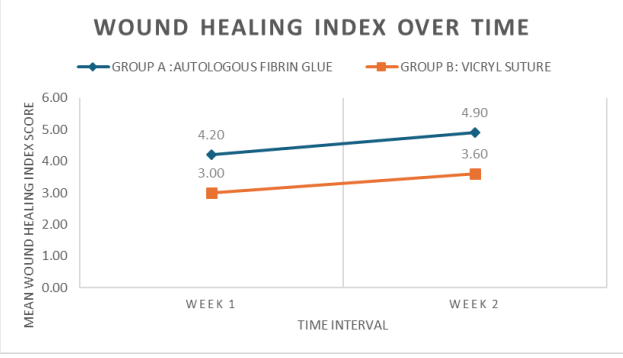

Figure 9: The Line diagram depicting comparison of healing index at 1 week and 2 weeks post-operative between the groups

Likewise, Group B showed a significant increase in wound healing scores from 3.00 ± 0.32 at 1 week to 3.60 ± 0.50 at 2 weeks ($\chi^2 = 12.00$, df = 1, $p < 0.001$), indicating progressive postoperative wound healing in both groups.

Discussion

The present randomised controlled clinical trial was conducted to compare postoperative wound healing and flap stability following periodontal flap surgery using autologous fibrin glue and conventional Vicryl sutures. The findings demonstrated that sites treated with autologous fibrin glue exhibited significantly better wound healing than those treated with Vicryl sutures at both

1 and 2 weeks postoperatively. Although both treatment modalities showed progressive improvement in flap stability during the healing period, autologous fibrin glue provided superior early flap stability, with both groups ultimately achieving complete flap stability by the second postoperative week.

The improved healing observed with autologous fibrin glue may be attributed to its biological properties. Fibrin glue forms a fibrin matrix that closely mimics the final stage of physiological coagulation[10], providing immediate tissue adhesion, eliminating dead space, and serving as a natural scaffold for the migration and proliferation of fibroblasts, endothelial cells, and other reparative cells. These properties promote early angiogenesis, reduce inflammation, and facilitate faster soft tissue healing.[11]

Table 5: Intra-group comparison of Roll Test

Group	Baseline (Mean ± SD)	1 Week (Mean ± SD)	2 Weeks (Mean ± SD)	χ^2 (Friedman)	Df	p-value
Group A (Autologous Fibrin Glue)	0.6500 ± 0.4894	0.1000 ± 0.3078	0.000 ± 0.000	22.62	2	<0.001**
Group B (Vicryl Suture)	1.600 ± 0.5026	1.050 ± 0.2236	0.000 ± 0.000	37.21	2	<0.001**

Table 6: Intra-group comparison of Wound Healing Index Scores

Group	1 Week (Mean ± SD)	2 Weeks (Mean ± SD)	χ^2 (Friedman)	df	p-value
Group A (Autologous Fibrin Glue)	4.200 ± 0.4104	4.900 ± 0.3078	12.25	1	<0.001**
Group B (Vicryl Suture)	3.000 ± 0.3244	3.600 ± 0.5026	12	1	<0.001**

The findings of the present study are consistent with those of Dave and Sathyanarayana (2020), who reported improved healing and flap stability following periodontal flap closure with autologous fibrin glue compared with sutures[2]. Similarly, Soundarajan and Rajasekar (2021) demonstrated significantly better early wound healing with simplified autologous fibrin glue than with 3-0 silk sutures[12]. Comparable outcomes were also reported by Nachimuthu et al. (2023), who found that commercially available fibrin glue (Reliseal®) resulted in superior postoperative healing compared with absorbable Vicryl sutures, suggesting that fibrin sealants are a reliable alternative for non-tension periodontal flap closure. These consistent findings strengthen the evidence supporting the clinical effectiveness of fibrin-based tissue adhesives in periodontal surgery[13].

The present findings are also in agreement with Jathal et al. (2008), who reported enhanced wound healing with fibrin glue, and Manimegalai et al. (2010), who demonstrated that the commercially available fibrin adhesive Tisseel® provided better postoperative healing than conventional silk sutures. Collectively, these studies indicate that both autologous and commercially available fibrin sealants can improve early periodontal wound healing.[3,5]

Unlike conventional sutures, which provide only point fixation, fibrin glue adheres the entire flap surface to the underlying tissues, resulting in uniform tissue adaptation and stabilization. Furthermore, synthetic tissue adhesives such as cyanoacrylate have been associated with tissue toxicity, whereas fibrin glue is biocompatible and promotes physiological wound healing, making it a safer alternative for periodontal flap closure.[14,15]

Although commercially available fibrin glue demonstrated favorable postoperative healing, its routine clinical use may be limited by its high cost, limited availability, short shelf life, and the potential risk of allergic reaction associated with commercially prepared products, making autologous fibrin glue a more economical and biologically safe alternative.

Overall, both treatment modalities resulted in progressive improvement in wound healing and flap stability throughout the postoperative period, with complete flap stability achieved by the second postoperative week. However, autologous fibrin glue consistently demonstrated superior wound healing and enhanced early flap stability compared with Vicryl sutures, suggesting that it may be an effective alternative for periodontal flap closure.

Limitation of study

Using fibrin glue to close the flap is simpler and faster, but additional preparation time away from the chairside in the laboratory, the use of equipment such as a centrifuge, and materials for preparing fibrin glue are required. Additional limitations, such as the relatively small sample size, short follow-up period, and absence of patient-reported outcomes measures.

Conclusion

It was found that healing with fibrin glue is associated with less amount of inflammation during the first week when compared with vicryl suture. The papillae closed with fibrin glue showed better healing and good stability after flap closure. Conventional sutures provide only a marginal fixation, while the fibrin sealing system makes the tissues adhere on its whole surface.

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JSPIK

Peripheral Ossifying Fibroma: Diagnosis and Therapeutic Management-A Case Report

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ABSTRACT

Abstract: Peripheral Ossifying Fibroma (POF) is a reactive, non-neoplastic gingival lesion originating predominantly from the periodontal ligament or periosteum. It commonly presents as a localized gingival overgrowth associated with chronic irritation. This case report describes the clinical presentation, diagnosis, and management of POF in a 68-year-old female patient who presented with a painless gingival swelling in the mandibular anterior region. Clinical examination revealed a firm, sessile, reddish-pink enlargement measuring approximately 8 × 12 mm in relation to 41 and 42. Radiographic findings showed no significant osseous involvement. Following initial periodontal therapy, complete surgical excision of the lesion with thorough curettage was performed. Histopathological examination demonstrated a highly cellular fibroblastic connective tissue stroma containing trabeculae of bone with osteoblastic rimming, confirming the diagnosis of POF. Postoperative follow up for 1 month and 4 months showed satisfactory healing without complications. Early diagnosis, complete surgical excision, elimination of local irritants, and periodic follow-up are essential to minimize recurrence and achieve favourable clinical outcomes in POF management.

Key-words: Peripheral Ossifying Fibroma, Reactive Gingival Lesion, Histopathological Diagnosis, Surgical Excision, Periodontal Ligament.

Key Messages:

- Accurate diagnosis of Peripheral Ossifying Fibroma (POF) requires careful clinical examination supported by definitive histopathological evaluation due to its close resemblance to other reactive gingival lesions.
- Complete surgical excision extending to the periosteum, along with elimination of local irritational factors, is essential to minimize the risk of recurrence in POF.
- Early diagnosis, meticulous periodontal management, and long-term follow-up play a crucial role in achieving favourable healing outcomes and preventing lesion recurrence.

Introduction:

Peripheral ossifying fibroma (POF) is a common, localized, reactive, non-neoplastic gingival overgrowth classified among fibro-osseous lesions, believed to originate from the periodontal ligament or periosteum. First described by Shepherd in 1844 as "alveolar exocytosis" and later termed "peripheral ossifying fibroma" by Eversole and Rovin in 1972, it typically presents as a slow-growing, asymptomatic, sessile or pedunculated gingival mass[1]. Clinically, the lesion appears as a firm nodular growth with its colour ranging from pale pink to reddish and may exhibit surface ulceration due to trauma. It most commonly occurs in young females, with a predilection for the anterior maxillary region, particularly the incisor–canine area[2]. Radiographic findings are variable, often showing no significant bone

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involvement, although scattered radiopaque calcifications or superficial alveolar bone resorption may be observed in some cases. Histologically, it appears as a highly cellular fibroblastic connective tissue stroma containing varying degrees of mineralized material, including bone, cementum-like deposits, and dystrophic calcifications, and is typically covered by stratified squamous epithelium with areas of ulceration and a chronic inflammatory infiltrate. This report underscores the importance of detailed clinical examination and histopathological investigation for the accurate diagnosis of Peripheral Ossifying Fibroma (POF). It further highlights that complete surgical excision extending to the periosteum, is essential to minimize the risk of recurrence and ensure effective management of the lesion.

Case Report

A 68-year-old female patient who is hypertensive, doesn't smoke or chew tobacco, reported to the Department of Periodontology at Annoor Dental College & Hospital, Muvattupuzha, with a chief complaint of a swelling in the lower anterior tooth region which was noticed before 1 month that had progressively increased in size. The lesion was asymptomatic; however, the patient expressed concern regarding the esthetic appearance of the swelling.

On intraoral periodontal examination showed calculus and recession of 4mm in the lower anterior region. On examination of the lesion, a localized gingival enlargement measuring approximately 8 × 12 mm was observed in relation to 41 and 42, extending slightly toward 43. The lesion appeared reddish pink

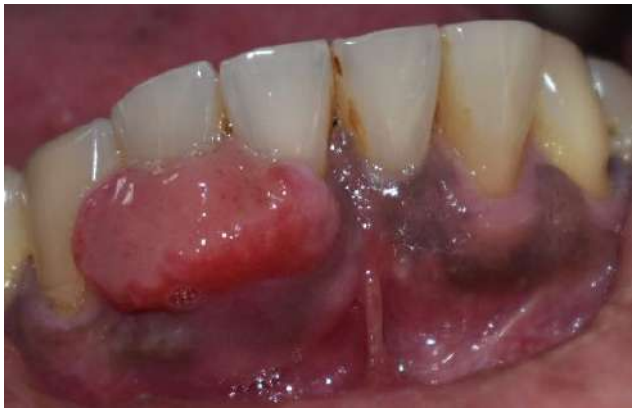

Figure 1: clinical representation of peripheral Ossifying fibroma

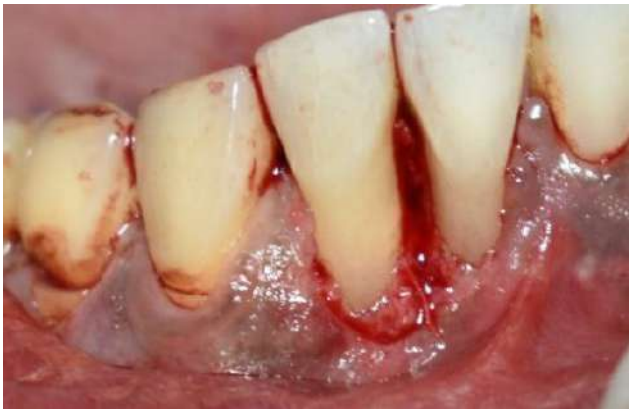

Figure 2: after surgical excision of the lesion

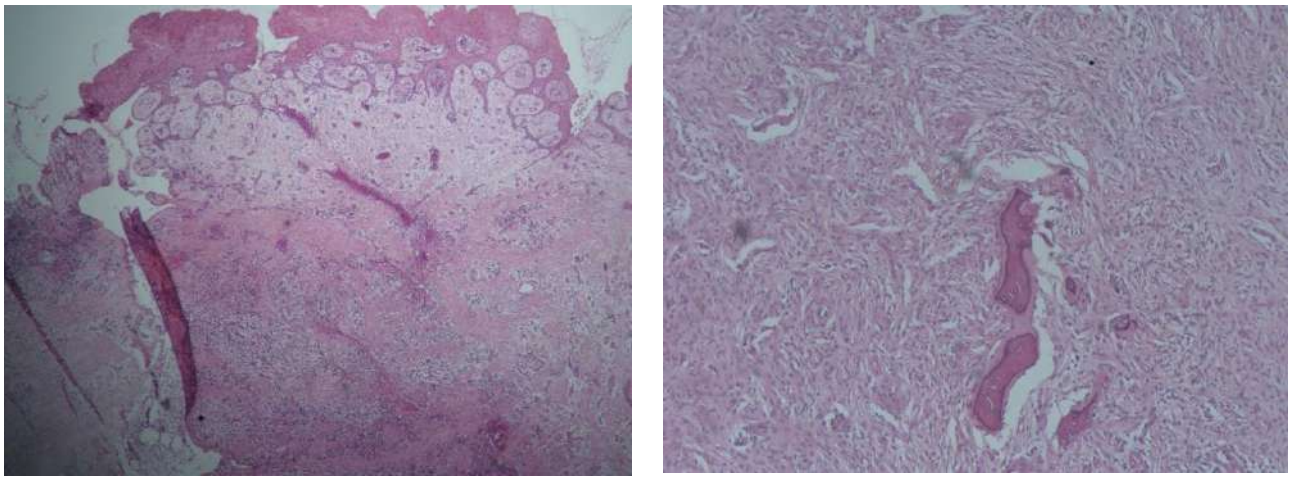

Figure 3: Histological sections showing calcifying deposits

in color with a smooth surface and a sessile base. On palpation, the swelling was firm, non-tender, and did not exhibit bleeding on probing (fig. 1). Radiographic examination revealed no significant alveolar bone loss or other underlying osseous pathology associated with the involved teeth. The patient's dental and medical histories were non-contributory. Based on the clinical findings, a provisional diagnosis of pyogenic granuloma was established, with irritational fibroma and peripheral ossifying fibroma considered as differential diagnosis.

Following the acquisition of written informed consent, an excisional biopsy of the lesion was planned for definitive diagnosis. Initial periodontal therapy comprising oral prophylaxis, including scaling and root planning, was performed to eliminate local irritational factors. One week following initial therapy, complete surgical excision of the lesion was carried out under local anesthesia. Before the surgical procedure, the patient was advised to rinse with 0.2% chlorhexidine gluconate mouthwash to reduce the microbial load within the oral cavity and minimize the risk of postoperative infection. Topical anesthesia was achieved using 20% lignocaine spray at the injection site, followed by local infiltration of 2% lignocaine containing 1:100,000 adrenaline to ensure profound anesthesia and hemostasis during the procedure. After achieving adequate anesthesia, complete surgical excision of the lesion was performed (Figure 2). Special emphasis was placed on thorough removal of the lesion extending down to the periosteum to eliminate residual reactive tissue and reduce the possibility of recurrence. The excised specimen was immediately preserved in 10% formalin and submitted to the Department of Oral and Maxillofacial Pathology and Oral Microbiology at Annoor Dental College & Hospital for histopathological evaluation and to establish a definitive diagnosis. Following surgery suturing of the surgical site was done and a periodontal dressing (Coe-pack) was placed over the surgical site to protect the wound and promote uneventful healing. The patient was prescribed 0.2% chlorhexidine mouthwash and was reinforced with oral hygiene instructions, including proper toothbrushing techniques and maintenance of good oral hygiene. The dressing was removed after 10 days during follow-up evaluation, which revealed satisfactory healing of the surgical area

The patient was recalled for postoperative evaluation after 1 month (Fig. 4) and 4 months (Fig. 5) following the procedure. Healing was uneventful at the 1-month follow-up, and satisfactory healing of the surgical site was observed at the 4-month follow-up, with no postoperative complications noted.

Microscopic examination of hematoxylin and eosin (H&E)-stained sections revealed an ulcerated stratified squamous epithelium overlying a highly cellular connective tissue stroma. The connective tissue consisted predominantly of numerous plump fibroblasts interspersed within a delicate fibrillar stroma. Multiple areas of mineralized tissue formation were evident, showing trabeculae of bone with osteoblastic rimming and osteocytic lacunae containing osteocytes. The newly formed bony trabeculae were surrounded by spindle-shaped fibroblasts with relatively reduced vascularity (Fig; 3).

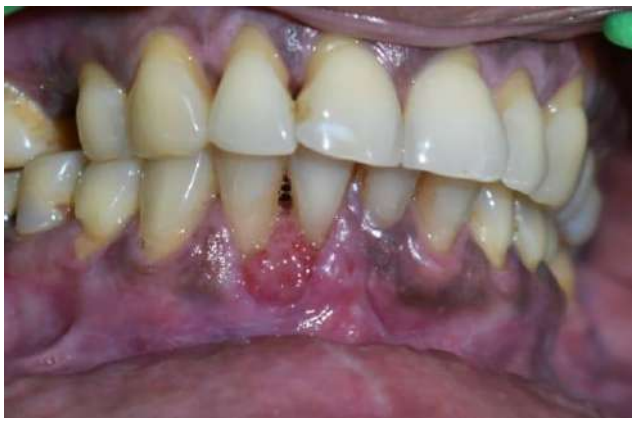

Figure 4: postoperative healing after 1 month

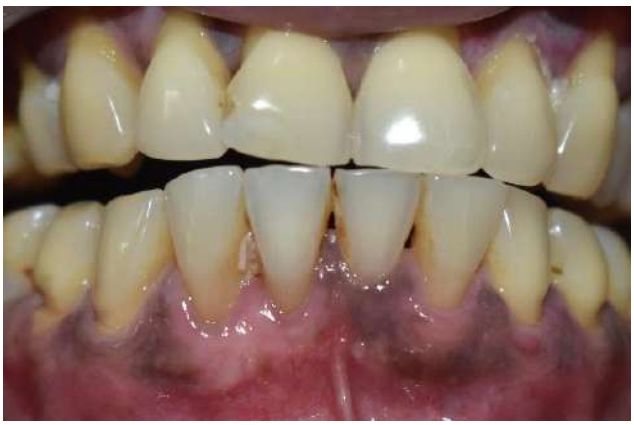

Figure 5: postoperative healing after 4 months

Based on the clinical presentation and histopathological findings, a definitive diagnosis of Peripheral Ossifying Fibroma was established.

Discussion

Peripheral Ossifying Fibroma (POF) also called as peripheral odontogenic fibroma represents a distinctive category of reactive gingival lesions characterized by fibrous proliferation with varying degrees of mineralization[3]. It is classified under reactive hyperplastic lesion rather than a benign neoplastic lesion, as it arises in response to chronic local irritants. The reported prevalence of 2–9% among all gingival growths highlights its relative frequency in clinical practice, although it remains less common compared to other reactive lesions such as pyogenic granuloma.

The origin of POF has been a subject of considerable discussion, with strong evidence supporting its derivation from periodontal ligament (PDL) cells. This assumption is based on several consistent observations, including its exclusive occurrence on the gingiva, particularly the interdental papilla, and the occasional presence of oxytalan fibers within the lesion. These features reinforce the concept that the lesion originates from cells capable of producing both fibrous and mineralized components, reflecting the multipotential nature of PDL fibroblasts.

The etiopathogenesis of POF is closely linked to chronic irritation. Local factors such as dental plaque, calculus, faulty restorations, and orthodontic appliances act as persistent stimuli, initiating a reactive hyperplastic process. Over time, this leads to connective tissue proliferation and subsequent metaplastic changes, resulting in the formation of calcified structures such as bone, cementum-like material, or dystrophic calcifications. This progressive mineralization explains the variation in radiographic appearance observed across different cases. Additionally, the role of functional forces, such as mastication, may further contribute to lesion development by perpetuating local trauma.

A notable feature of POF is its marked female predilection, particularly during the second decade of life[4]. This suggests a possible hormonal influence in its pathogenesis. Estrogen and progesterone may enhance the vascular and cellular response of gingival tissues to local irritants, thereby increasing susceptibility. However, the exact mechanism of hormonal modulation remains unclear and warrants further investigation. Although the lesion most commonly affects the anterior maxilla, atypical presentations in posterior regions and older individuals emphasize the need for careful clinical evaluation irrespective of age or site.

Clinically, POF typically presents as a slow-growing, well-demarcated gingival mass that is often asymptomatic, contributing to delayed diagnosis and treatment. While most lesions remain small, larger lesions can occur and may lead to functional and esthetic complications, including tooth displacement and occlusal disturbances. Such cases underscore the importance of early detection and intervention. Radiographically, findings are inconsistent and depend largely on the degree of calcification. Early lesions may show no detectable changes, whereas more mature lesions may exhibit radiopaque foci or superficial bone resorption, sometimes described as a "cupping" defect. Advanced imaging modalities such as cone beam computed tomography (CBCT) provide valuable insights into the internal architecture and extent of mineralization, particularly in larger or atypical lesions5.

One of the major challenges in managing POF is its clinical similarity to other reactive gingival lesions, including pyogenic granuloma, peripheral giant cell granuloma, and irritational fibroma. This overlap often complicates provisional diagnosis based solely on clinical features. Therefore, histopathological examination remains essential for definitive diagnosis. Microscopically, POF is characterized by a highly cellular fibroblastic stroma interspersed with mineralized components, which may vary from immature woven bone to mature lamellar bone, along with cementum-like deposits and dystrophic calcifications. The diversity of these features reflects different stages of lesion maturation[5].

Another important consideration is the relatively high recurrence rate of POF, reported to range between 8% and 20%. Recurrence is often attributed to incomplete excision, failure to eliminate local irritants, or persistence of periodontal ligament remnants. Hence, treatment should not only involve complete surgical excision of the lesion but also thorough scaling and root planing of adjacent teeth, along with removal of all potential irritants. Long-term follow-up

is crucial to monitor for recurrence and ensure optimal outcomes.

Conclusion

Peripheral Ossifying Fibroma (POF) is a benign yet persistent reactive gingival overgrowth that necessitates early clinical and radiographic intervention to prevent functional impairment and aesthetic deformities. Given its origin from the deeper periodontal structures, particularly the periodontal ligament and periosteum, the most effective management involves complete surgical excision extending to the periosteum and periodontal ligament. This should be accompanied by meticulous elimination of local etiological factors, including plaque and calculus, through thorough scaling and root planning. Despite appropriate treatment, POF presents a notable recurrence rate of approximately 8% to 20%, most commonly attributed to incomplete excision or failure to eliminate underlying irritants. Therefore, long-term periodic follow-up is essential for early detection of recurrence and for maintaining optimal periodontal health.

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Biomechanical and Biological Characteristics of Implant-Abutment Connections in Rehabilitation: A Comprehensive Review

Mohammed Shamil[1], Deepak Thomas[2], Punya Krishnan[3], Aswani Surya K[4], Sreya Jyothibas[5], Shahna N[6]

ABSTRACT

Purpose: To systematically evaluate the biological and biomechanical characteristics of external and internal implant-abutment connections (IACs) and their clinical correlations with marginal bone loss. Investigations: A rigorous synthesis of literature spanning from 1980 to 2022 was conducted to evaluate joint stability under loaded conditions. Basic Procedures: Structural connection mechanisms including butt joints, Morse tapers, slip-fit, and friction-fit joints were analyzed regarding mechanical parameters (rotational misfit, screw loosening, torque loss) and biological width variables (microgaps, leakage, fluid pumping). Main Findings: External hexagon connections demonstrated high rates of rotational misfit (3°-10°) and screw loosening (6%-48%). Internal conical interfaces and Morse taper geometries establish a superior microbial seal, reducing biological microgaps down to 2-3 μm compared to 10 μm in flat butt joints. Platform-switched interfaces significantly limit marginal bone loss (0.22 mm vs 1.18 mm) by horizontal shift of inflammatory cell fronts. Conclusion: Conical and friction-fit connections optimize force distribution, prevent screw complications, and protect peri-implant biological width.

Keywords: Implant Abutment Connections, Morse Taper, Platform Switching, Microgap

Introduction

Prosthetic rehabilitation with osseointegrated implants has evolved from mere bone integration to strict mandates for esthetic and long-term functional success[1]. A critical component governing this longevity is the implant-abutment connection (IAC), the interface between a dental implant and an abutment is stabilized by two mechanical characteristics: a preload of an abutment screw and the friction between the contact surfaces of the implant and the abutment[2]. In the past years dental implants have undergone various design changes surface modifications and material changes to overcome earlier failures. Two-piece configurations are standard clinical choice but inherently feature a microgap interface that can induce biological and mechanical joint challenges[3]. So the dental implant complications are categorized into three general categories: mechanical, biological, and esthetic complications[4]. According to a statement made by Lang et al.[5], Several key terms define implant and prosthesis longevity, including survival, success, and failure. These standards help doctors measure how well an artificial part performs over time and track any problems[5].

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Parts of Implant Abutment Connection:

The implant connections simply defined by Misch et al. by the geometry of connecting elements including components dental implant fixture and an abutment used to support or retain definitive prosthesis[6].

Different classifications of implant abutments

i. Classifications of implant abutment based on available forms of implant abutment including

A. Prefabricated abutment

1. Standard abutment used in non-esthetic zones

Advantage is easy maintenance

2. Conical abutment used in esthetic zones

Figure: Conical abutment

3. Angulated abutments

specialized components used in dental implantology to correct the alignment discrepancies of an implant that cannot be placed in an ideal axial position and helps to avoid positional discrepancy.

4. Cementable core abutments

Per-manufactured dental implant connectors designed to support and retain a cemented artificial crown or bridge

5. Post abutments

6. Ball attachment abutment

It consist of a male spherical metal ball on the implant and a female housing cap with a resilient nylon or silicone that embedded in the denture.

7. Locator abutment

specialized connecting parts used on dental implants to snap and hold removable overdentures or fixed full-arch teeth in place securely

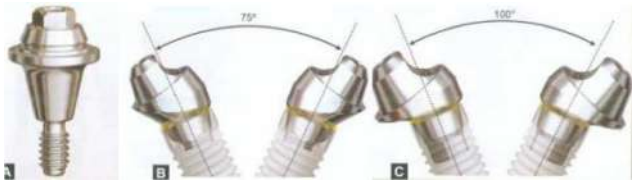
B. Custom made abutments

1. Customizable healing abutment made by either custom copy milling or custom-made CAD/CAM abutments

Figure VII: customisable healing abutment

2. Cylindric abutment/ UCLA abutments

Figure : UCLA abutment (straight, angled at 17 and 30 degree)



3. Castable abutments – for metals including titanium, alumina and Nickel-chromium-cobalt alloys

ii. Classifications of implant abutment based on materials of abutment

1. Ceramic abutments

2. Zirconia abutments

3. PEEK abutments

4. Ti or metal abutments

The implant abutment connection:

This connection determines the joint strength, stability of the joint, lateral and rotational stability, so the pattern of connection may vary depending up on inclusion or exclusion of anti-rotational features also the connection may either be characterized by slip fit joint or friction fit joint[7].

1. The classification of implant abutment connection depending geometrical configuration:

a. octagonal b. Hexagonal

c. Conical d. Cylinder hex

e. Spline

2. The classification of implant abutment connection depending on the space between the connecting parts:

a. The slip fit joint characterized with a space between the mating parts

b. frictional fit doesn't show any space between mating parts[7].

3. The classification of implant abutment connection based on whether or not there exists an extension of a geometric figure above the body of the implant:

External Hex: There is an extension above the implant surface.

Internal Hex: the connection is recessed into the implant body.

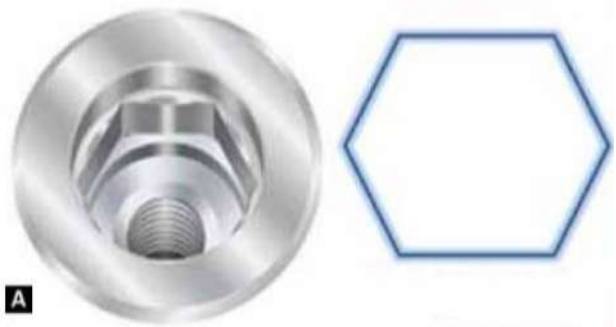
EXTERNAL HEX CONNECTION

External hex on implant and internal hex on abutment combination provides proper implant abutment connection.

Types of external abutment- implant connection:

The Branemark original system external hexagon

The Branemark original system has implant abutment connection of 0.7mm external hex connection which served for the purpose of coupling and act as torque device. The drawbacks of Branemark original system including owing limited height makes it ineffective when excessive axial load is applied and complications like abutment screw loosening, screw fracture and micro-motion at interface[8].

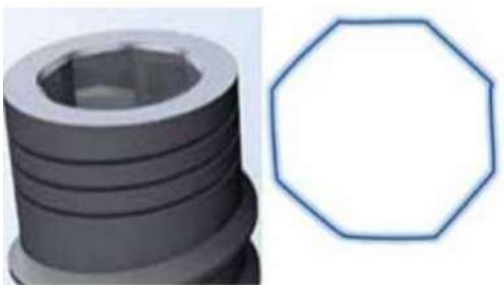


Tapered hexagonal pattern

Designed as an evolution beyond the original Brånemark external-hex system, this internal tapered interface employs a friction-fit hex connection. This configuration offers superior implant-abutment stability and provides high tactile precision during impression transfer procedures[8].

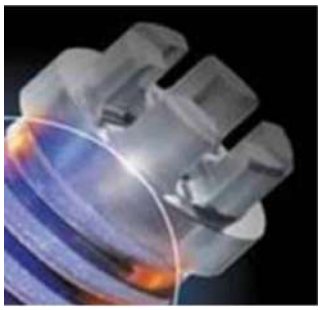
Octogonal pattern: -

It is an 8-sided pattern unpopular design, show no anti-rotational property or very less anti rotational configuration with almost a spherical outline that allows 45° rotation of the abutment[8].



Spline connection

Splines are "Fine-to-groove" anti-rotational configuration with a long & successful history in implantology developed by Calcitek (1992). It consists of 6 spline teeth which project outward from the body fits with the 6 corresponding locking grooves in the abutment. Its advantages includes reduced incidence of screw loosening and minimal rotational movement compared with traditional external design.



INTERNAL HEX CONNECTIONS:

implant feature a chamber within the implant body to which external projection of abutment can engage. The goals of internal connection are to improve connection stability throughout function & placement as well as simplifying the procedure. The advantages include it allows implant cover screw to be held in level with the top of fiction at stage I surgery but for external hex it requires lo hold the cover screw that seat slightly above the level of fixture.

Types of internal connection

Six- point internal hex:

Common type of commercially available connection with abutment fit over implant in every 60° angulation and hexagonal geometry. It distributes force deep within implant effectively and improves joint stability.[7-10]

12-point internal hexagon:

It allows more options for abutment placement over implant in every 30 angulations[8]

Tang et.al stated that 12-point double hexagon connection had better stress distribution & produce smaller displacement compared to other designs.

3-point internal tripod

Represent triangular internal geometry with tri-channel design. But it has a Major disadvantage that is placement of abutment on fixture allowed only at 120 degrees, so it is not very preferred design due to limited options of placement.

Internal octagon

An eight-sided internal geometry similar to circle that allows positioning in very 45° angulation and offers minimal rotational & lateral resistance during funcion.

Morse tape type:

A taped projection from implant abutment that fits into corresponding tapered recess in implant functions by frictional fit & cold welding at 1-A junction. it was proposed by Sutter et al. and has disadvantages like taper interfere with abutment fitting and resisting lateral load.

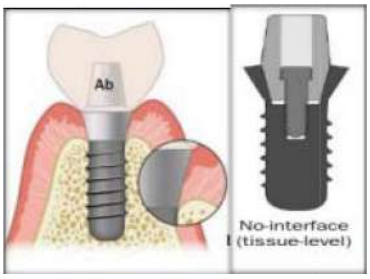
8-degree connection

8-degree internal Morse taper connection used in dental implants, a design pioneered by the ITI (International Team for Implantology) group in Switzerland. To overcome the indexing limitations of a pure Morse taper, researchers Wiskot and Belser modified this design by adding an internal hexagon to provide an anti-rotational mechanism and multi-angle positioning.[11]

IMPLANT ABUTMENT CONNECTION BASED ON ANTI-ROTATIONAL PROPERTY

1. Abutment connection without anti-rotational property:

It has flat surface and requires one piece abutment attachments indicated in multiple implant units united by splinting or by crowns/bars, preventing abutment mal-rotation.



One Piece implant connection type and biological consequences

2. Implant-abutment connection with anti-rotational property

Some anti rotation features incorporated in designs to prevents undesirable movement of overlying abutments[8]

IMPLANT PLATFORMS

The traditional Branemark External hex design maintained same diameter of implant collar to the portion of abutment that connects to the implant design and it is called as platform matched/butt joint implants. Achieving a stable implant-abutment connection while reducing the abutment diameter using an internal conical connection. If the abutment is narrower than implant at connection area then it is known as platform switching, beneficial in reducing bone loss around implant. It allows greater volume of soft tissue at I-A interface & helps to achieve good soft tissue esthetics.[8]

BIOLOGICAL AND MECHANICAL JOINT PERSPECTIVES

The implant-abutment micro gap represents a key environment prone to physical contamination and subsequent biological bone resorption[6]. According to Caricasulo et al implant abutment connection and

peri-implant bone loss has a relation and states that conical connection has lower peri-implant bone loss compared with external hex[12]. Micromovements under continuous chewing generate a hazardous 'pumping effect' that shifts bacteria, metabolic products, and endotoxins into internal implant wells, recruiting a localized inflammatory front[5]. Quaresma finite element study b/w internal hex and conical connection and concluded that conical connection is solid & put lower stress on alveolar bone & prosthesis but has greater stress on abutment than internal hex connection[11].

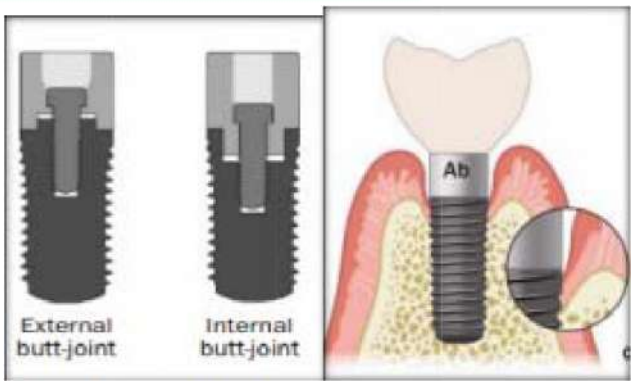
TABLES

Table I: Biomechanical Comparison of Core Connection Architectures

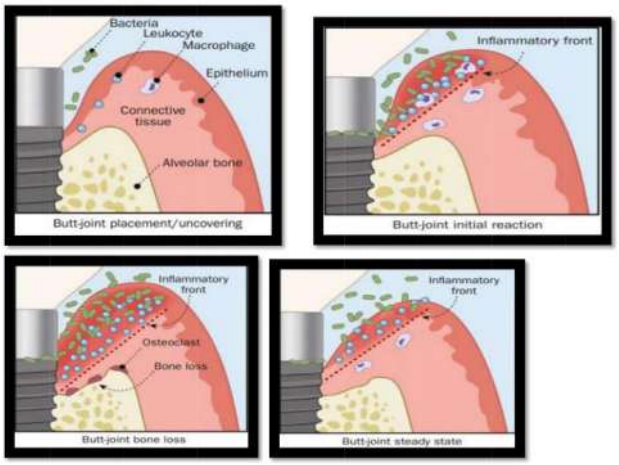
Connection Type	Microgap Width (µm)	Rotational Misfit	Primary Complication Risk
External Hexagon	10.0 µm	3.0° - 10.0°	High Screw Loosening (6%-48%)
Internal Slip-Fit	5.0 - 10.0 µm	1.5° - 3.0°	Fretting Wear / Fluid Leakage
Morse Taper (Friction)	2.0 - 3.0 µm	0.0°	Vertical Height Discrepancy

Traditional flat butt joints allow significant microbial penetration[6]. Conversely, Morse taper (conical) connections establish an intimate cold-welded joint via immense friction, decreasing biological micro-gaps down to 2-3 µm from the standard 10 µm observed in flat junctions[5]. This architecture protects tissue biological width, particularly when coupled with platform switching—the configuration where narrow diameter abutments are adapted on a wide implant shoulder[1].

Schematic representation of the mechanical load transfer distribution and biological cell fronts inside flat butt joints versus conical friction-fit Morse taper interfaces.



Sequence of events involved in Mechanism of bone loss due to inflammatory front created by microbial contamination.



DISCUSSION AND SUMMARY

The comprehensive evaluation of multi-decade data clearly reveals that friction-lock and Morse taper connections provide superior mechanical strength and marginal bone preservation compared to historic external hexagon systems[1,5]. External hexagons possess an inherent vulnerability to high rotational misfit (3°-10°), exceeding the clinical threshold of 2° necessary to secure steady torque preloads, resulting in screw loosening rates between 6% and 48%[10]. The deployment of internal conical connections ensures safe force distribution deep into the body of the implant wall rather than concentrating dangerous mechanical stresses onto the retention assembly[4]. Clinicians must select connection models based on structural alignment criteria, mucosal thickness variations, and functional guidelines to secure predictable outcome

parameters[10]. Implant-abutment interfaces are broadly divided into external and internal connection designs. External connections (ECs), pioneered by Brånemark, feature a projecting hexagonal geometry above the implant platform. Although widely used historically, EC systems are susceptible to interface micromovement, which elevates the risk of microleakage and screw loosening. To mitigate these issues, internal systems were developed, housing the longer abutment connection within the implant body walls. Standard internal connections (ICs) frequently utilize a flat-to-flat hexagonal configuration to prevent rotation and enhance stability. Recently, conical or Morse taper connections (CCs) have gained prominence due to their superior tight sealing ability, which minimizes interface micromovements, screw loosening, and structural fractures. Furthermore, implementing platform switching—using narrower prosthetic abutments—helps preserve marginal bone by shifting the connection center away from the bone crest.

A recent meta-analysis of randomized controlled trials reported that CC configuration resulted in significantly lower peri-implant marginal bone loss (MBL) than ECs after one year of functional loading, whereas differences between EC and IC variants were negligible.

While these trends align with broader literature, conclusive evidence regarding the optimal interface design for preventing clinical and mechanical failure remains limited. This ambiguity stems from a widespread failure across published literature to standardize critical confounding variables, including surface topography, macro- and micro-geometric configurations, subcrestal placement depth, and clinician proficiency, all of which heavily distort clinical outcomes.

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Comparative Evaluation of Two Different Gingival Depigmentation Techniques: Surgical Scalpel & Diode Laser - A Split-Mouth Randomized Clinical Trial

Aswathy M Shenoy[1], Lakshmi Damodaran[2], Mohammed Feroz TP[3], Megha Varghese[4], Deepthi V[5], Neelima Vinod[6]

ABSTRACT

Background: Gingival color plays an important role in dental esthetics, and excessive melanin pigmentation may adversely affect the appearance of the gingiva. Various depigmentation techniques have been employed to improve esthetics, including bur abrasion, scalpel surgery, cryotherapy, electrosurgery, and laser therapy. The scalpel technique involves surgical removal of the gingival epithelial surface, whereas laser therapy uses specific energy and wavelengths to target melanin-containing cells. The choice of method depends on clinical experience, patient preferences and comfort, healing time, and recurrence risk.

Objectives: To compare postoperative pain, healing, and recurrence using surgical scalpel and diode laser techniques for gingival depigmentation.

Methodology: Fifteen patients with gingival hyperpigmentation were enrolled in the study. A split-mouth randomized clinical trial was conducted, with one side treated using surgical scalpel and the other with diode laser.

Results: Gingival depigmentation by diode laser resulted in no bleeding, and patients reported no post-operative pain whereas the scalpel technique led to moderate pain. There was no significant differences in wound healing and pigmentation recurrence at 3 months postoperatively.

Conclusion: The diode laser technique showed minimal bleeding and reduced postoperative pain, making it preferable choice for gingival depigmentation due to its superior patient comfort and similar clinical outcomes.

Keywords: Gingiva, Depigmentation, LASER, Scalpel, Wound healing

Introduction

Gingival colour significantly affects an individual's overall aesthetics. Gingival melanin pigmentation occurs due to excessive melanin production by melanocytes located in the basal and suprabasal layers of the epithelium.[1] Various techniques, such as bur abrasion, scalpel surgery, cryotherapy, electrosurgery, and laser therapy, are used for gingival depigmentation. The scalpel technique, one of the earliest methods, involves the surgical removal of the gingival epithelial surface. Lasers, owing to their specific energy and wavelength characteristics that target soft tissues, have been used to ablate cells that contain and produce melanin pigment.[2] The present study aims to identify the most effective technique for aesthetic gingival depigmentation by evaluating postoperative pain, healing, and recurrence in patients undergoing gingival depigmentation using surgical scalpel and diode laser techniques.

Materials and Methods

A randomised controlled, split mouth study was conducted on 15 outpatients from the Department of

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Periodontology, Kannur Dental College, Anjarakandy for a duration of 12 months. The study was conducted after obtaining ethical approval from the Institutional Ethical Review Board (IECKDC/2024-04/002). Written informed consent was obtained from all the participants.

Sampling procedure: A total minimum calculated sample size of 30(15 per group) would require to yield 80% power to detect a significant difference, with an effect size of 1.1 and a significance level 0.05(5%)

Sample size

$$n = 2 \times \frac{(\frac{Z\alpha}{2} + Z\beta)^2}{\Delta^2} \times SD^2$$

Inclusion Criteria

- Age group of 18-45 years
- Patients with bilateral physiologic melanin hyperpigmentation in the anterior segment of maxillary arch.

Exclusion Criteria

- Patients with a current history of systemic illness, mucosal lesion
- Patient with a history/undergoing chemotherapy
- Pregnant and lactating women
- Patients receiving medications or having medical conditions that may affect bleeding.
- Smokers

All enrolled participants underwent scaling and root planing one week prior to the depigmentation procedure. For each patient, data including age, sex, medical history, and dental history were collected using a case proforma. Gingival pigmentation in all patients wasrecorded using the Dummett Oral Pigmentation Index (DOPI).[3] The maxillary anterior segment was divided into right and left halves and randomly assigned into two groups.Simple randomization was used to allocate the right and left sides of each participant to either the scalpel or diode laser intervention. The allocation sequence was generated using a simple random method.

Group 1: Gingival depigmentation by conventional scalpel technique

Group 2: Gingival depigmentation by diode laser

Depigmentation procedure

Conventional Scalpel Technique

After achieving adequate anaesthesia in the surgical area using 2% lignocaine hydrochloride in group 1 patients, the surface epithelium was removed using a No. 15 surgical scalpel blade. An eyelet curette was used until all visible pigmentation was removed from the gingival margin to the mucogingival junction. Bleeding from the surgical area was controlled by the direct application of a pressure pack.

Laser Technique

In group 2 patients adequate anaesthesia was achieved in the surgical area using 2% lignocaine hydrochloride. Before laser application, appropriate precautions were taken. Protective eyewear was worn by the operator, assistant, and patient. Highly reflective instruments or instruments with mirrored surfaces were avoided to prevent reflection of the laser beam. Additional precautionary measures were taken to avoid the use of the laser in the presence of explosive gases. A diode laser with a wavelength of 980 nm and a power output of 1.5–2.0 W, operated in continuous-wave mode with a flexible fiber-optic delivery system in continuous contact mode, was used. The melanin-pigmented gingiva was ablated using this laser device. The laser parameters were kept consistent across all laser-treated sites to minimize variation in thermal effects and their potential influence on postoperative wound healing.

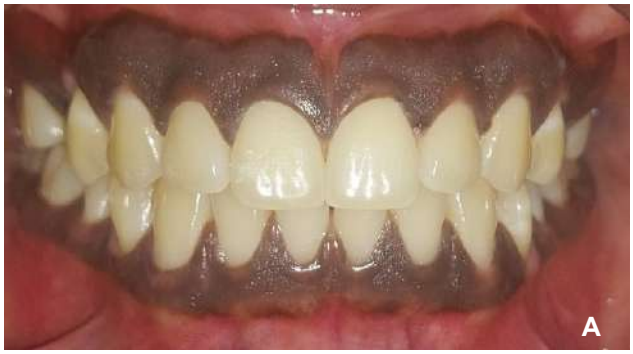
Postoperative analgesics were prescribed to be taken once, 2 hours after the procedure. Patients were evaluated on the 7th, 14th, 21st, and 30th postoperative days and subsequently at 3, 6, and 12 months. Assessment of clinical repigmentation and pigmentation intensity was performed using the Dummett Oral Pigmentation Index (DOPI) at the 3rd, 6th, 9th, and 12th postoperative months.

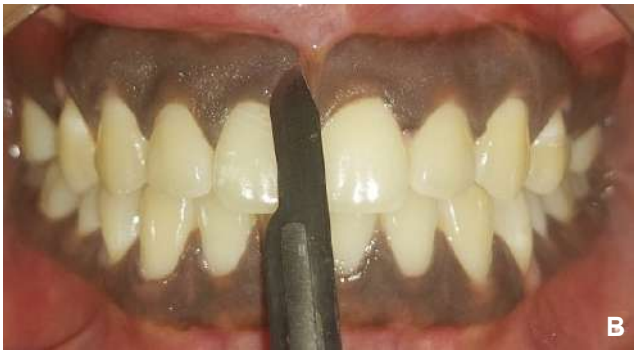
The following clinical parameters were recorded:

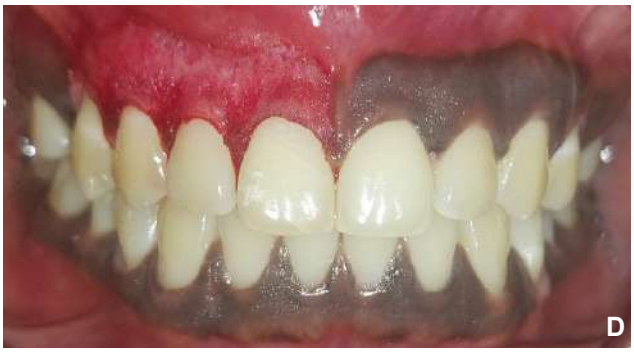
- Dummett Oral Pigmentation Index (DOPI, 1971)
- Visual Analogue Scale (VAS; Hayes and Patterson, 1921) – assessed at 2 hours and 24 hours after the procedure
- Wound Healing Index (Trung et al., 2021) – evaluated on the 7th, 14th, 21st, and 30th

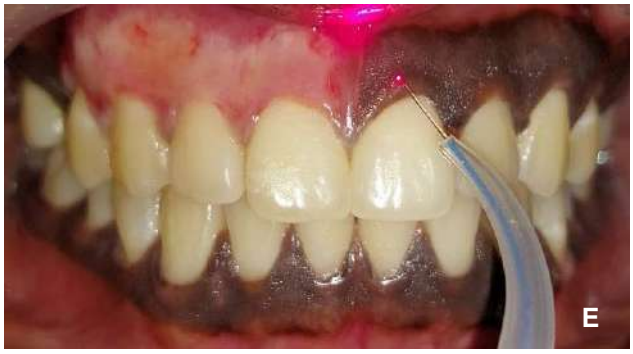
postoperative days.

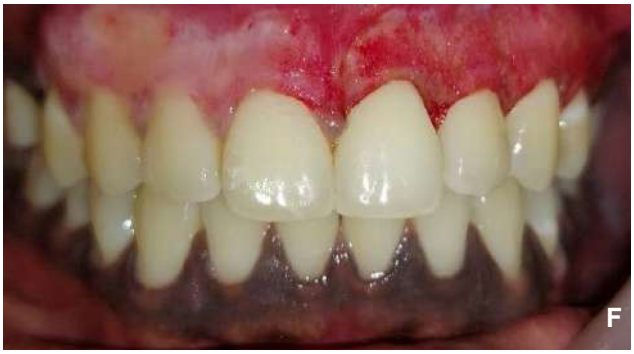
Due to the nature of the surgical interventions, blinding of the patients and operator was not feasible. However, the observer evaluating the study parameters was unaware of the group allocation. The observer was trained and standardized in the evaluation procedures before commencement of the study to ensure consistency and reliability of outcome assessment.

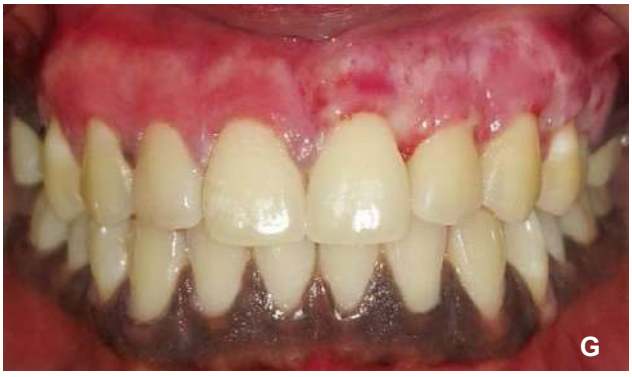












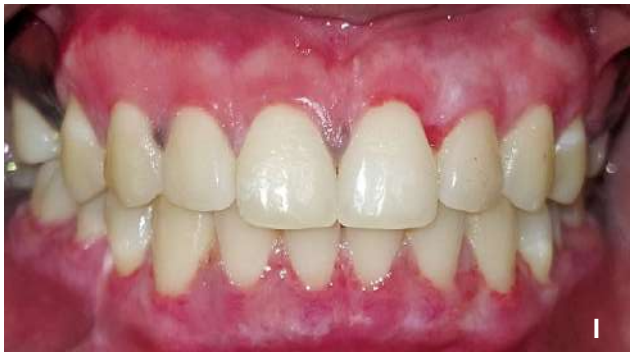
Statistical Analysis

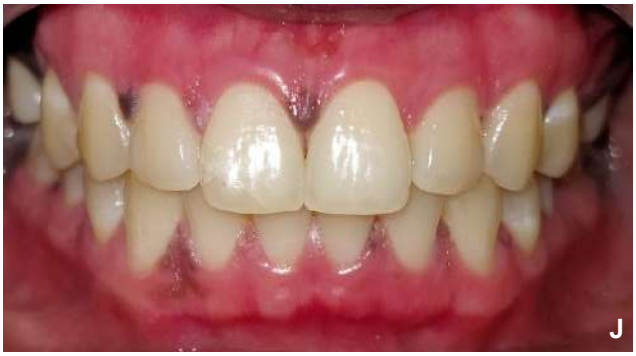
Data was analysed using the statistical package SPSS 26.0 (SPSS Inc., Chicago, IL) and levelof significance was set at P<0.05. Normality of the data was assessed by Shapiro Wilk test.Descriptive statistics was performed to assess the mean and standard deviation of therespective groups.As this was a split-mouth study, observations from the scalpel and diode laser sites were considered paired observations within the same participant. Therefore, paired statistical methods were used for comparisons between the two treatment modalities. For continuous variables, the paired t-test was used for normally distributed data and the Wilcoxon signed-rank test was used when the normality assumption was not met. For paired categorical data, McNemar's test was used, as appropriate.

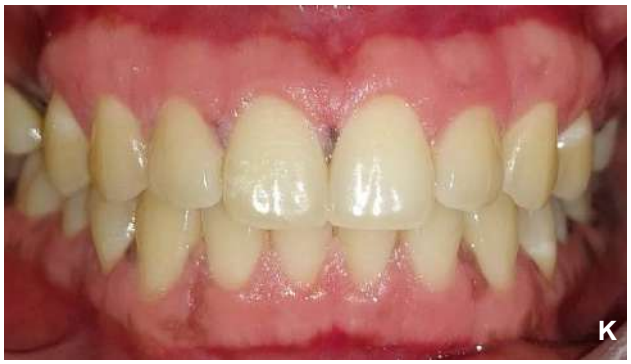
Results

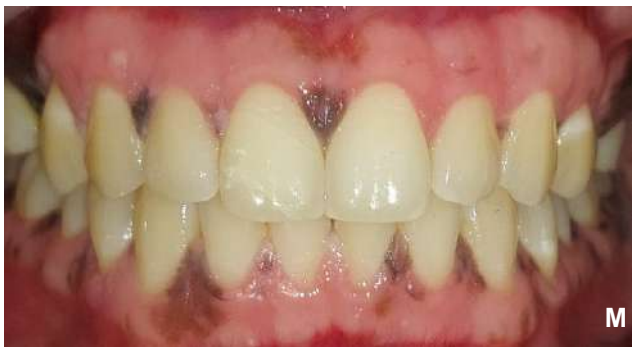
The study group comprised 15 participants, including 8 females and 7 males.

At 2 hours postoperatively, pain was significantly higher in the Conventional Scalpel group, with 60% of participants reporting moderate pain, whereas 66.7% of participants in the Laser group reported only slight pain (Chi-square = 8.45, P = 0.01). At 24 hours, a greater proportion of participants in the Laser group were pain-free (66.7%) compared to the Conventional Scalpel group (26.7%); however, this difference was not statistically significant (P = 0.09). Within-group comparisons over time revealed a significant reduction in pain scores in both the Conventional Scalpel group (P = 0.008) and the Laser group (P = 0.0001).









A-Pre-Operative View, B-Scalpel Depigmentation, C-Use Of Eyelet Curette, D-Immediate Post-Operative View, E-Laser Depigmentation, F-Immediate Post-Operative View, G- 2 Weeks Post-Operative View , H- 3 Weeks Post-Operative View, I- 4 Weeks Post-Operative View, J- 3 Months Post-Operative View, K- 6 Months Post-Operative View, L- 9 Months Post-Operative View, M- 12 Months Post-Operative View

(Right: Scalpel, Left: Laser)

At baseline, both groups exhibited identical distributions of gingival pigmentation, with no statistically significant difference between them (P = 0.99). Complete depigmentation was observed in all participants in both groups at the 3-month follow-up. However, at 6 and 9 months, a significantly greater proportion of participants in the Laser group without recurrence of pigmentation compared to those in the Conventional Scalpel group (P = 0.02 and P = 0.04, respectively), indicating a lower rate of repigmentation following laser treatment. By 12 months, although the Laser group continued to show a higher percentage of long-term maintenance of aesthetic outcomes(20%) than the Conventional Scalpel group (6.7%), the difference was not statistically significant (P = 0.26).

With respect to wound healing, all participants in both groups demonstrated incomplete epithelisation at 1 week postoperatively. At 2 weeks, the Conventional Scalpel group showed significantly higher percentage of complete epithelisation (60%) compared to the Laser group (0%) (Chi-square = 12.87, P = 0.0001). Similarly, at 3 weeks, complete epithelisation was observed in all participants (100%) in the Conventional Scalpel group, whereas only 60% of participants in the Laser group exhibited complete healing (Chi-square = 7.57, P = 0.0001). By 4 weeks, complete epithelisation had been achieved in all participants in both groups, with no statistically significant difference between the two treatment modalities.

Discussion

The present study aimed to comparatively evaluate the conventional surgical scalpel technique and diode laser application with respect to postoperative pain, wound healing, and the recurrence and intensity of gingival repigmentation.

The results demonstrated that the postoperative pain was significantly lower in the laser group than in the conventional scalpel group. At 2 hours postoperatively, 60% of patients in the conventional scalpel group reported moderate pain, whereas 66.7% of patients in the laser group reported only slight pain. At 24 hours, a greater proportion of participants in the laser group were pain-free (66.7%) compared to those in the conventional scalpel group (26.7%), although the difference was not statistically significant. These findings were consistent with those reported by Suragimath et al. (2016)[4] and Chandra et al. (2020)[1]. The reduced pain associated with diode laser therapy may be attributed to the formation of a protein coagulum over the wound surface, which acts as a biological dressing and seals sensory nerve endings. In contrast, the conventional scalpel technique leaves an open, bleeding surface with exposed nerve endings, thereby resulting in greater postoperative discomfort. Administration of analgesics 2 hours postoperatively may have influenced the VAS scores, particularly the 2-hour pain assessment, representing a potential limitation of the study.

With regard to wound healing, the conventional scalpel group demonstrated significantly better healing during the second and third postoperative weeks compared to the laser group. This observation is in agreement with the findings of Lagdive et al. (2009)[5] and Kasagani et al. (2012)[6], who also reported delayed wound healing at diode laser-treated sites. The faster healing observed in scalpel-treated areas may be attributed to delayed epithelial regeneration and the absence of normal wound contraction following laser procedures. Similarly, Grover et al. (2014)[7] suggested that thermal effects associated with laser application may slow the healing process when compared to conventional surgical techniques.

Despite the delayed initial healing, long-term outcomes were more favourable in the laser group. The laser-treated sites exhibited lower rates of repigmentation and better maintenance of depigmentation over time compared to the scalpel-treated sites. These findings were comparable to those reported by Lagdive et al. (2009)[5], who also observed superior long-term results following diode laser therapy. The lower recurrence rate associated with laser treatment may be due to its ability to effectively destroy melanocytes within the basal layer of the epithelium, thereby reducing the potential for melanin re-synthesis.

The delayed healing observed at laser-treated sites has been explained by several authors. Luomanen et al. (1987)[8] in an experimental study, attributed delayed healing to reduced capillary proliferation and slower infiltration of inflammatory cells resulting from thermal coagulation and denaturation of vasculogenic polypeptides. Similarly, D'Arcangelo et al. (2007)[9] emphasized that thermal damage to the surrounding tissues is a major factor responsible for delayed wound healing following laser therapy. The biological response to di-

ode laser treatment is influenced by several parameters, including wavelength, power output, exposure time, mode of operation, number of passes, tip diameter, and energy density. In the present study, a 980-nm diode laser was used at 1.5–2.0 W in continuous-wave contact mode. Repeated or prolonged laser passes and higher energy delivery can increase heat accumulation within the tissues, potentially resulting in thermal coagulation and collateral tissue injury. Such thermal effects may delay inflammatory cell infiltration, vascular proliferation, and subsequent epithelialization. Therefore, standardization of the number of laser passes, tip diameter, exposure time, and energy density is important when evaluating wound healing following laser depigmentation. The delayed epithelialization observed in the present laser-treated sites may therefore be related, at least in part, to the thermal effects associated with laser application. However, because the exact tissue temperature and cumulative thermal exposure were not measured in the present study, this explanation should be considered a possible mechanism rather than a definitive causal relationship. Nevertheless, the advantages of reduced postoperative pain, minimal bleeding, and lower recurrence rates make diode laser therapy an effective and predictable treatment modality for gingival depigmentation.

Conclusion

Both surgical scalpel and diode laser techniques are clinically effective for the treatment of gingival melanin pigmentation. The diode laser offers several advantages, including reduced postoperative pain and discomfort, and better long-term results making it a more comfortable and efficient alternative for patients and clinicians alike.

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Regeneration Redefined: The Role of Hyaluronic Acid in Periodontal Therapy

Nanditha Chandran[1], Anil Melath[2], Subair K.[3], Hemalatha DM[4], Priyadarshni K[5]

ABSTRACT

Hyaluronic acid (HA) is a naturally occurring glycosaminoglycan and a key component of the extracellular matrix, playing vital roles in tissue hydration, cell migration, angiogenesis, wound healing, and modulation of inflammation. Owing to its biocompatibility, viscoelasticity, and non-immunogenic nature, HA has emerged as a valuable biomaterial in periodontal therapy. This review aims to provide a comprehensive overview of the chemistry, pharmacokinetics, biosynthesis, sources, biological functions, clinical applications and recent advances of HA in periodontology. HA has multipurpose use in nonsurgical and surgical periodontal therapy such as guided tissue regeneration, gingival recession management, and peri-implant therapy. Evidence indicates that HA enhances periodontal wound healing by regulating inflammatory responses, promoting fibroblast proliferation, stimulating angiogenesis, facilitating extracellular matrix remodeling, and supporting soft and hard tissue regeneration. Recent advances involving HA-based nanomaterials, stem cell therapies, and bioengineered scaffolds have further expanded its regenerative potential. Commercially available HA formulations have demonstrated favorable clinical outcomes as adjuncts to conventional periodontal treatment. HA appears to be a promising therapeutic and regenerative agent in periodontology, with innovations integrating HA with nanotechnology and stem cell therapies, which are likely to enhance its role in achieving predictable periodontal regeneration, wound healing and improved clinical outcomes.

Keywords: hyaluronic acid, periodontal therapy, periodontal regeneration and wound healing.

INTRODUCTION:

Hyaluronic acid(HA) or hyaluron is one of the important components of our body's extracellular matrix.[1] The Greek word hyalos, which means glass, is where the term "hyaluronic" originates.[2] The average healthy adult weighs about 70 kg and contains roughly 15 grams of HA. It is found majorly in connective tissues, skin, lungs, kidneys, brain, and muscle tissues. HA plays significant and diverse roles in biological functions. It regulates tissue hydration, water transport, elasto-viscosity of connective tissues, and facilitates the supramolecular assembly of proteoglycans in the extracellular matrix. It is also involved in cell communication and migration, blood clotting, angiogenesis, wound healing and reduction of inflammation. HA is the perfect material for clinical and cosmetic applications due to its distinct viscoelastic qualities, biocompatibility, and non-immunogenicity.[3,4]

Nowadays, HA is used to treat long-term inflammatory diseases like periodontal disease, which damages the tissues surrounding teeth. Although there is not much HA in the mineralized portions of these tissues, such as the cementum and alveolar bone, it is crucial for the periodontal ligament and the surrounding gingiva.[5]

This article gives an overview of hyaluronic acid, its chemistry, biological interactions and applications in periodontics.

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HISTORY:

Karl Meyer and John Palmer made the initial discovery of HA in 1934, when they isolated it from a cow's eye fluid. It was subsequently found in humans and other animals. It is mostly present in synovial fluid, the extracellular matrix of connective tissue, and essential tissues like the umbilical cord, cartilage, fascia, and vitreous of the eye. The polymer was extracted and purified from human umbilical cords and rooster combs in 1979 to create pharmaceutical-grade HA.[6]

STRUCTURE OF HA:

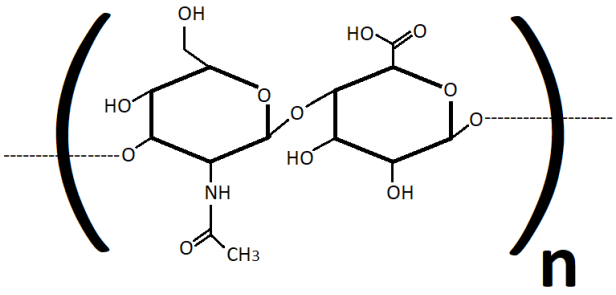

Fig.1: Hyaluronic acid – composed of N-acetyl glucosamine and glucuronic acid linked together via glycosidic bonds.

PHARMACOKINETICS OF HYALURONIC ACID

Hyaluronic acid (HA), when administered orally, is not significantly degraded by gastric or intestinal enzymes but is primarily broken down by gut microbiota, particularly *Bacteroides*, into low-molecular-weight oligosaccharides (<3 kDa) that are absorbed through the intestinal barrier in limited amounts, with microbial metabolism also generating short-chain fatty acids (SCFAs) such as acetate, propionate, and butyrate.[7,8] After absorption, small HA fragments are distributed systemically via the bloodstream to peripheral tissues, including the skin, although only in low concentrations.[9] HA is metabolized through enzymatic degradation by hyaluronidases (HYAL-1 and HYAL-2), which sequentially cleave HA into smaller fragments, as well as by non-enzymatic degradation mediated by reactive oxygen species (ROS), while microbial hyaluronan-degrading enzymes further contribute to its breakdown in the gut.[8,10,11] Most orally administered HA is extensively degraded rather than excreted intact in feces, with its metabolites eliminated through urine and expired air.[9] Hyaluronidases (HYAL-1, HYAL-2) serve as the principal catalysts of HA degradation, gut microbiota and the SCFAs they produce act synergistically by enhancing HA metabolism and systemic signaling, whereas hyaluronidase inhibitors and modified HA formulations resistant to enzymatic degradation can slow HA catabolism.[7,8,10–14]

ORIGIN, DERIVATION, AND BIOSYNTHESIS OF HYALURONIC ACID

Hyaluronic acid (HA) is obtained primarily from animal and microbial sources. Historically, rooster combs have been the richest natural source, while umbilical cords, skin, and vitreous humor contain lower concentrations of HA.[17] Industrially, HA is predominantly produced by *Streptococcus zooepidemicus*, whereas certain plant-derived polyphenols and isoflavones indirectly enhance endogenous HA metabolism by stimulating hyaluronan synthase (HAS) activity and inhibiting hyaluronidase.[17–19] Traditionally, HA is derived from animal tissues through enzymatic extraction and purification, but microbial fermentation has become the preferred method because it provides high-purity HA with improved safety and scalability.[20,21] Recombinant Generally Recognized as Safe (GRAS) microorganisms, including genetically engineered *Bacillus subtilis*, further enhance biosafety, while chemical cross-linking with 1,4-butanediol diglycidyl ether (BDDE) or divinyl sulfone (DVS) improves HA stability and resistance to degradation for biomedical applications.[22,23] Endogenously, HA is synthesized by the membrane-bound hyaluronan synthase (HAS) isoenzymes HAS1, HAS2, and HAS3, which polymerize uridine diphosphate-glucuronic acid (UDP-GlcA) and uridine diphosphate-N-acetylglucosamine (UDP-GlcNAc) into high-molecular-weight HA and simultaneously extrude the polymer into the extracellular matrix.[24,25] In bacteria, HA synthesis is regulated by the hasABC operon, where hasA encodes hyaluronan synthase, hasB encodes uridine diphosphate-glucose dehydrogenase (UDP-glucose dehydrogenase), and hasC encodes uridine diphosphate-glucose pyrophosphorylase (UDP-glucose pyrophosphorylase).[26] Recombinant hosts such as *Escherichia coli*, *Lactococcus lactis*, and *Bacillus subtilis* have been engineered to produce high-molecular-weight HA for therapeutic

use.[27] Although complete chemical synthesis of high-molecular-weight HA remains impractical because of its complex β-glycosidic linkages, shorter HA oligosaccharides can be synthesized chemically or enzymatically for research and specialized biomedical applications.[28]

APPLICATION OF HYALURONIC ACID IN PERIODONTICS:

Hyaluronic acid (HA) not only provides structural support but actively modulates cellular behavior and extracellular matrix (ECM) remodeling during periodontal wound healing. Its effect varies according to the type of periodontal therapy**:** nonsurgical therapy, surgical therapy, guided tissue regeneration, gingival recession treatment, and peri-implant therapy**.** The following sections describe the chronological progression of healing at molecular, cellular, and tissue levels**.**

Nonsurgical Periodontal Therapy (SRP Combined with Hyaluronic Acid)

Following nonsurgical periodontal therapy with scaling and root planing (SRP), adjunctive hyaluronic acid (HA) enhances wound healing through coordinated molecular, cellular, and tissue-level events across distinct phases. During the early inflammatory phase (days 1–3), HA binds to CD44 receptors on neutrophils and macrophages, suppressing pro-inflammatory cytokines such as TNF-α and IL-1β, thereby limiting excessive inflammation while preserving host defense.[53] Neutrophils and macrophages clear bacteria and debris, release reparative growth factors, and HA stabilizes a hydrated fibrin clot that maintains extracellular matrix (ECM) integrity.[54,55] In the transition to the proliferative phase (days 4–7), HA promotes fibroblast proliferation via ERK1/2 and Akt signaling pathways and enhances the expression of VEGF and FGF-2, stimulating angiogenesis, fibroblast and endothelial cell migration, keratinocyte proliferation, and early granulation tissue formation with epithelial migration over the HA-rich matrix.[56–58] During the proliferative and early maturation phase (days 8–14), HA supports collagen type III deposition, regulates matrix metalloproteinase (MMP) activity to prevent excessive ECM degradation, promotes myofibroblast differentiation and peak angiogenesis, resulting in reduced periodontal pocket depth, soft tissue thickening, and progressive re-epithelialization.[59,60] In the remodeling phase (days 15–28), HA is gradually degraded as the provisional matrix is replaced by mature ECM, collagen type III is substituted by collagen type I with increased cross-linking, myofibroblasts undergo apoptosis, and the vascular network matures, ultimately leading to periodontal pocket closure, restoration of functional soft tissue architecture, and marked resolution of inflammation.[61]

Surgical Periodontal Therapy (Flap Surgery Combined with Hyaluronic Acid)

Following periodontal flap surgery, adjunctive hyaluronic acid (HA) enhances wound healing by modulating hemostasis, inflammation, proliferation, and tissue remodeling. During the hemostatic and inflammatory phase (days 1–3), HA stabilizes the blood clot, reduces the release of pro-inflammatory cytokines, and maintains tissue hydration while limiting postoperative edema.[62,63] Neutrophils provide early antimicrobial defense, whereas macrophages release transforming growth factor-β1 (TGF-β1) to recruit fibroblasts and initiate repair.[62,63] In the early proliferative phase (days 4–7), HA activates CD44-mediated signaling, upregulating vascular endothelial growth factor (VEGF) and platelet-derived growth factor (PDGF), which promote fibroblast and endothelial cell proliferation, angiogenesis, and synthesis of a provisional extracellular matrix (ECM), leading to formation of HA-rich granulation tissue.[64,65] During the late proliferative phase (days 8–14), HA enhances collagen type III deposition, regulates matrix metalloproteinase (MMP) activity to preserve newly formed ECM, and supports myofibroblast-mediated wound contraction and keratinocyte migration, facilitating optimal flap adaptation and re-establishment of epithelial continuity.[66,67] In the remodeling and maturation phase (days 15–28), HA is gradually degraded by endogenous hyaluronidases as collagen type III is replaced by collagen type I, increasing tissue tensile strength, while fibroblast numbers normalize and the neovascular network matures, resulting in well-organized gingival tissue with minimal scar formation owing to the antifibrotic and regulatory effects of HA.[68]

Guided Tissue Regeneration (GTR) Combined with Hyaluronic Acid

In guided tissue regeneration (GTR), adjunctive

hyaluronic acid (HA) enhances periodontal regeneration by promoting molecular signaling, cellular recruitment, and organized tissue healing. During the early healing phase (days 1–3), HA binds and protects growth factors such as fibroblast growth factor (FGF) and vascular endothelial growth factor (VEGF) from proteolytic degradation, preserving their biological activity and promoting selective repopulation of the defect by periodontal ligament (PDL) fibroblasts and osteoprogenitor cells, while neutrophils control microbial contamination.[69] HA also serves as a viscoelastic scaffold that maintains regenerative space beneath the barrier membrane and prevents epithelial downgrowth.[69,70] During the proliferative phase (days 4–14), HA stimulates PDL fibroblast proliferation and migration through CD44-mediated signaling, while VEGF-driven angiogenesis supports neovascularization and osteoblasts initiate early bone matrix deposition; simultaneously, fibroblasts align along the root surface and alveolar bone, facilitating integration of connective tissue with the HA-rich scaffold.[70,71] In the maturation phase (days 15–28 and beyond), HA levels gradually decline as collagen type I replaces the provisional matrix, osteoblasts continue differentiation and mineralization, endothelial cells stabilize the new vascular network, and the periodontal defect becomes filled with newly formed bone and functionally oriented PDL fibers, resulting in restoration of stable periodontal attachment.[72]

Gingival Recession Therapy Combined with Hyaluronic Acid

In gingival recession therapy, adjunctive hyaluronic acid (HA) enhances soft tissue healing, root coverage, and gingival thickness by modulating inflammatory and regenerative processes.[73] During the early inflammatory phase (days 1–3), HA suppresses pro-inflammatory cytokines such as interleukin-6 (IL-6) and tumor necrosis factor-α (TNF-α) and binds to CD44 receptors on fibroblasts, promoting their migration and survival.[73] Neutrophils clear debris and control bacterial contamination, while HA stabilizes the blood clot, maintains root surface hydration, and improves flap adaptation and early wound stability.[73,74] During the proliferative phase (days 4–14), HA promotes collagen type III deposition, VEGF-mediated angiogenesis, continued fibroblast proliferation, and coronal migration of keratinocytes, resulting in epithelial coverage, early keratinized tissue formation, and progressive coronal advancement of the gingival margin.[74,75] In the maturation and remodeling phase (days 15–28 and beyond), collagen type III is gradually replaced by collagen type I with increased cross-linking, myofibroblasts undergo apoptosis, and the vascular network matures, leading clinically to complete root coverage, increased gingival thickness, an expanded zone of keratinized tissue, and improved long-term functional and esthetic stability.[76]

Peri-Implant Therapy Combined with Hyaluronic Acid

In peri-implant therapy, adjunctive hyaluronic acid (HA) promotes early wound stabilization, modulates inflammation, and enhances formation of a stable peri-implant soft-tissue seal.[77] During the early inflammatory phase (days 1–3), HA reduces the expression of pro-inflammatory cytokines and binds to CD44 receptors on epithelial and connective tissue cells, promoting cell adhesion and survival.[77] Neutrophils control microbial contamination, while macrophages release transforming growth factor-β (TGF-β) and platelet-derived growth factor (PDGF) to initiate connective tissue repair; HA hydrogels further stabilize the blood clot and peri-implant soft tissues, creating a favorable healing environment.[77,78] During the proliferative phase (days 4–14), HA upregulates vascular endothelial growth factor (VEGF) and fibroblast growth factor-2 (FGF-2), promoting angiogenesis, fibroblast proliferation, connective tissue fiber organization around the implant collar, and epithelial migration to re-establish the peri-implant mucosal seal, resulting in reduced bleeding and inflammation.[78,79] In the remodeling and maturation phase (days 15–28 and beyond), HA is gradually degraded as collagen type I deposition increases, fibroblast density returns to normal, and the vascular network matures, culminating in complete peri-implant mucosal healing with an enhanced soft-tissue seal, improved esthetic contour, and greater long-term implant stability.[80]

RECENT ADVANCES

Hyaluronic acid (HA) is increasingly integrated with nanotechnology and stem cell–based therapies to enhance periodontal tissue engineering by pro-

moting regeneration of both soft tissues (gingiva and periodontal ligament) and hard tissues (alveolar bone and cementum). HA-based nanohydrogels and nano-scaffolds exhibit improved mechanical strength, controlled degradation, and targeted drug delivery, while composites containing nano-hydroxyapatite (nHA), carboxymethyl chitosan (CMCS), and zinc ions mimic the extracellular matrix (ECM), providing osteoconductive, antimicrobial, and sustained drug-release properties.[53–56] HA also enhances the proliferation, migration, and osteogenic differentiation of mesenchymal stem cells (MSCs) through CD44-mediated signaling and activation of the Yes-associated protein/transcriptional coactivator with PDZ-binding motif (YAP/TAZ) pathway.[57,58] When combined with periodontal ligament stem cells (PDLSCs) or gingival mesenchymal stem cells (GMSCs), HA-based scaffolds promote organized periodontal ligament formation, bone regeneration, angiogenesis, and extracellular matrix deposition.[59] Advanced integrated HA–nanotechnology–stem cell constructs, incorporating aligned nanofibers and functionalized nanoparticles, further improve cell viability, spatial organization, immunomodulation, and antimicrobial activity, representing a promising next-generation approach for predictable periodontal regeneration.[54–59]

Commercially Available Hyaluronic Acid Products

Gengigel® (0.2%–0.8% Hyaluronic Acid, Ricerfarma, Italy)
Used in: gingivitis, periodontitis, post-SRP healing, peri-implant mucositis, surgical wound healing.[81,82]

Hyadent® BG / Hyadent® Pro (Regedent AG, Switzerland)
Used in: periodontal pockets, peri-implant soft tissue healing, post-surgical applications.[83]

Hyaloss® Matrix / HYAFF® (Fidia Farmaceutici, Italy)
Used in: guided tissue regeneration, infrabony defects, periodontal regeneration.[84]

Aminogam® Gel (HA + Amino Acids, Errekappa, Italy)
Used in: post-periodontal surgery healing, mucogingival procedures.[84]

Periohyale® (HA-based gel, Italy)
Used in: adjunct to SRP, periodontal maintenance therapy.[85]

Hyalsense® Oral Gel (Hyaluronic Acid, India/Europe)
Used in: gingivitis, post-surgical periodontal care.[85]

Hylodent® Gel (Hyaluronic Acid, dental topical formulation)
Used in: periodontal inflammation, peri-implant soft tissue healing.[85]

CONCLUSION:

The nanotechnology-stem cell-HA constructs represent a next-generation periodontal regeneration strategy, potentially achieving more predictable, functional regeneration than traditional grafts or barrier membranes. Future research should focus on in vivo validation, long-term safety such as immunogenicity, degradation byproducts, and scalable manufacturing of these scaffolds. Translation to the clinic will require regulatory consideration for cellloaded nanomaterials, including controlled release, sterilization, and reproducibility.

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Microneedling in Periodontal Regenerative Therapy - A Review

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ABSTRACT

Microneedling, also known as percutaneous collagen induction, has recently emerged as a promising minimally invasive adjunct in periodontal regenerative therapy. Unlike conventional regenerative approaches that rely primarily on biomaterials and surgical intervention, microneedling stimulates endogenous healing mechanisms through controlled micro-injury, thereby enhancing angiogenesis, fibroblast proliferation, stem cell recruitment, and extracellular matrix remodeling. This review aims to provide a comprehensive overview of the role of microneedling in periodontics, with a particular focus on its applications in periodontal regenerative therapy.

The biological basis of microneedling involves activation of the wound-healing cascade, including platelet aggregation, growth factor release, neovascularization, and mechanotransduction-mediated cellular responses. These processes contribute to both soft- and hard-tissue regeneration. Clinically, microneedling has shown potential in gingival biotype modification, enhancement of wound healing, facilitation of transgingival drug delivery, and as an adjunct in the management of intrabony defects, furcation defects, and peri-implant tissues.

A significant advancement in this field is the synergistic use of microneedling with platelet-rich fibrin (PRF), which enhances growth factor bioavailability, improves scaffold stability, and promotes sustained regenerative signaling. Despite its advantages, including minimal invasiveness, cost-effectiveness, and improved patient acceptance, microneedling is limited by lack of standardized protocols, technique sensitivity, and insufficient long-term clinical evidence.

Future directions involve integration with bioengineered microneedles, advanced biomaterials, and stem cell–based therapies. Within the framework of minimally invasive periodontal regeneration, microneedling represents a biologically driven approach with the potential to improve clinical outcomes. However, further well-designed randomized controlled trials are required to establish its long-term efficacy and standardize its clinical application.

Keywords: Microneedling; Periodontal regeneration; Platelet-rich fibrin; Intrabony defects; Gingival biotype; Angiogenesis; Wound healing; Minimally invasive therapy; Periodontics; Tissue engineering

INTRODUCTION

Periodontal disease remains one of the most prevalent chronic inflammatory conditions affecting humans, characterized by progressive destruction of the supporting structures of teeth, including gingiva, periodontal ligament (PDL), cementum, and alveolar bone.

In recent years, increasing emphasis has been placed on minimally invasive, biologically driven regenerative strategies that harness the body's intrinsic wound-healing potential rather than relying solely on exogenous grafting materials. Among these emerging approaches, microneedling—also referred to as

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percutaneous collagen induction—has gained attention for its ability to stimulate angiogenesis, fibroblast proliferation, stem cell recruitment, and extracellular matrix remodeling through controlled micro-injury. Initially popularized in dermatology and aesthetic medicine, microneedling is now being explored across multiple biomedical disciplines, including orthopedics, neurology, and tissue engineering.[1]

In periodontology, microneedling represents a paradigm shift: instead of merely debriding diseased tissues or placing passive scaffolds, it actively reprograms the local wound environment to favor regeneration. By inducing controlled mechanical trauma, microneedling initiates a cascade of biological events that resemble embryonic and postnatal tissue repair mechanisms. Emerging evidence suggests that microneedling can enhance gingival thickness, improve soft-tissue phenotypes, promote angiogenesis, and potentially synergize with biologics such as platelet-rich fibrin (PRF) and growth factor delivery systems.[2]

HISTORY

The origins of microneedling can be traced back to the concept of controlled skin injury as a stimulus for tissue regeneration. Early observations in dermatology noted that superficial dermabrasion and scarification procedures often resulted in improved skin texture and collagen deposition.[2] In the 1990s, Orentreich and Orentreich formally introduced the concept of subcision wherein needles were used to break fibrotic strands and stimulate neocollagenesis. This laid the foundation for modern microneedling techniques.

The first dedicated microneedling devices were developed in the early 2000s, primarily for aesthetic indications such as acne scars, photoaging, and alopecia. These devices used arrays of fine needles to create thousands of microchannels in the skin without causing extensive epidermal damage. Subsequent histological studies demonstrated increased collagen type I and III deposition, angiogenesis, and dermal remodeling following microneedling.[1]

Controlled micro-injury was shown to activate latent growth factors, including transforming growth factor-β (TGF-β) and vascular endothelial growth factor (VEGF), which are central to tissue repair processes.[3]

In dentistry, early applications of microneedling were largely confined to oral mucosal drug delivery and enhancement of topical agent penetration. However, as understanding of periodontal wound healing evolved, researchers began exploring microneedling as a means of enhancing gingival biotype, accelerating soft-tissue healing, and modulating inflammatory responses.[4]

With advances in device precision, depth control, and combination therapies (e.g., PRF, growth factors, biomaterials), microneedling is now being investigated as an active regenerative stimulus rather than a passive adjunct. Contemporary studies in tissue engineering and biomaterials science have further strengthened this transition by demonstrating the ability of microneedling-induced microenvironments to support stem cell recruitment and osteogenic differentiation.[5]

DEVICES USED FOR MICRONEEDLING

Microneedling devices can be broadly classified based on needle configuration, depth control, mode of operation, and clinical indication. In periodontal applications, device selection is particularly critical due to the delicate nature of gingival tissues and the proximity of vital structures.

Figure 1- Derma Roller

Manual Microneedling Devices

Manual devices include dermarollers and dermastamps, consisting of stainless steel or titanium needles mounted on a cylindrical roller or flat stamp. Needle lengths typically range from 0.25 mm to 2.5 mm.[2] While these devices are cost-effective and simple to use, they offer limited precision and inconsistent penetration depth, making them less suitable for controlled periodontal regenerative procedures.

Motorized Microneedling Pens

Motorized microneedling pens represent the most commonly used devices in medical applications. These devices allow adjustable needle depths, typically between 0.2 mm and 3.0 mm, with high-frequency vertical needle motion. The ability to precisely control penetration depth is crucial in periodontal tissues, where excessive penetration may damage underlying bone or root surfaces.

Recent biomedical engineering advances have led to improved needle designs with tapered tips, reduced tissue drag, and uniform microchannel formation, enhancing both safety and efficacy.[1]

Figure 2- Cartridge for Motorized Microneedling

Fractional Microneedling Systems

Fractional microneedling systems integrate microneedling with energy-based modalities such as radiofrequency. While primarily used in dermatology, their application in periodontology remains experimental. Although deeper tissue stimulation and improved collagen remodeling are theoretical benefits, the risk of thermal injury currently limits their periodontal application.

Figure 3- Fractional RF Microneedling

Emerging Bio-integrated Microneedles

Recent innovations in tissue engineering have introduced biodegradable and drug-loaded microneedles capable of delivering growth factors, antimicrobials, or stem cell-derived exosomes directly into target tissues.[6] These experimental systems offer promising avenues for future applications in periodontal regeneration.

MECHANISM OF APPLICATIONS OF PERIODONTAL REGENERATIVE THERAPY

Microneedling exerts its regenerative effects through a complex and highly coordinated sequence of biological events initiated by controlled mechanical micro-injury. Unlike conventional surgical trauma, microneedling creates precise, standardized, and minimally invasive microchannels that activate endogenous repair mechanisms while preserving overall tissue architecture. In periodontal tissues, this controlled injury acts as a biological switch, transforming a chronically inflamed or biologically inactive environment into one conducive to regeneration.[1]

1. Controlled Micro-injury and Initiation of the Wound-Healing Cascade

The primary biological trigger of microneedling is the induction of uniform micro-wounds within the gingival and periodontal connective tissues. These

micro-injuries do not cause tissue necrosis but instead preserve cell viability while disrupting tissue continuity at a microscopic level.[1]

2. Platelet Activation and Growth Factor Release

One of the earliest molecular events following microneedling is platelet aggregation within the microchannels. Activated platelets release a wide spectrum of biologically active growth factors, including Platelet-derived growth factor (PDGF), Transforming growth factor-β (TGF-β), Vascular endothelial growth factor (VEGF), Insulin-like growth factor (IGF).

3. Angiogenesis and Microvascular Remodeling

Angiogenesis is a cornerstone of successful periodontal regeneration. Microneedling has been shown to significantly enhance neo-vascularization through both mechanical and molecular mechanisms.[4]

4. Fibroblast Activation and Extracellular Matrix Remodeling

Fibroblasts are the principal effector cells in periodontal soft-tissue regeneration. Microneedling stimulates fibroblast activity through mechanotransduction, whereby mechanical stimuli are converted into biochemical signals.[5]

5. Stem Cell Recruitment and Activation

One of the most significant regenerative mechanisms of microneedling is its ability to recruit and activate resident mesenchymal stem cells (MSCs) from multiple sources, including Periodontal ligament, Alveolar bone marrow and Perivascular niches.[6]

6. Modulation of Inflammatory Pathways

Chronic periodontitis is characterized by persistent activation of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6. Microneedling induces a controlled, short-term inflammatory response that paradoxically leads to long-term inflammation resolution.[4]

7. Mechanotransduction and Gene Expression Modulation

At the molecular level, microneedling activates mechanosensitive signaling pathways that regulate gene expression involved in regeneration. These include pathways associated with Cell adhesion, Cytoskeletal remodeling, Osteogenic and fibroblastic differentiation.[7]

8. Synergistic Hard- and Soft-Tissue Regeneration

A unique advantage of microneedling is its ability to simultaneously influence both soft and hard tissues. By coordinating angiogenesis, fibroblast activity, stem cell recruitment, and osteogenesis, microneedling supports true periodontal regeneration rather than isolated tissue repair.[8]

APPLICATIONS OF MICRONEEDLING IN PERIODONTICS

Microneedling has multiple potential applications in periodontics beyond regeneration, primarily related to soft-tissue modulation and healing enhancement.

Gingival Biotype Modification

Thin gingival biotypes are associated with increased risk of recession and poorer esthetic outcomes. Microneedling has been shown to stimulate fibroblast proliferation and collagen deposition, leading to increased gingival thickness and improved tissue resilience.[2]

Management of Gingival Recession

When used as an adjunct to coronally advanced flap or connective tissue graft procedures, microneedling may enhance soft-tissue healing and vascularization, potentially improving root coverage outcomes.

Enhancement of Drug Delivery

Microneedling-induced microchannels facilitate transgingival delivery of anti-inflammatory agents, antimicrobials, and biologics, improving local drug bioavailability without systemic exposure.[4]

Acceleration of Wound Healing

By promoting angiogenesis and reducing inflammatory mediators, microneedling may shorten healing time following periodontal surgery, improving patient comfort and compliance.

Interdental Papilla Regeneration

Loss of the interdental papilla results in unaesthetic "black triangles," open embrasures, phonetic difficulty, and food impaction, and remains one of

the most challenging problems in periodontal plastic surgery. Because papillary tissue is thin and has a limited blood supply, conventional surgical augmentation techniques are technically demanding and often yield unpredictable results. Microneedling has therefore been explored as a minimally invasive means of stimulating papillary fill, either alone or as a delivery adjunct for regenerative agents such as injectable platelet-rich fibrin (i-PRF), hyaluronic acid, and vitamin C. The controlled micro-injury increases local vascularity and fibroblast activity within the papilla while also creating transmucosal channels that enhance uptake of the co-administered biologic, and clinical trials have reported measurable gains in papillary height and reduction of black-triangle area using this approach.[9]

Gingival Depigmentation

Melanin-related gingival hyperpigmentation is not a pathological condition but is a frequent esthetic concern, particularly in patients with a high smile line. Conventional depigmentation techniques—scalpel surgery, bur abrasion, cryosurgery, electrosurgery, and laser ablation—are effective but carry the risk of postoperative discomfort, delayed healing, and, in some techniques, unpredictable repigmentation. Microneedling has emerged as a simple, low-cost, non-surgical alternative in which repeated micro-puncture of the pigmented epithelium is combined with topical application of a depigmenting or antioxidant agent, most commonly ascorbic acid (vitamin C). The microchannels enhance transmucosal penetration of the agent while the induced turnover of the pigmented epithelial layer promotes its replacement by non-pigmented tissue. Case series and comparative trials have reported a pink, esthetic gingival appearance within days of treatment, with patient comfort and minimal morbidity comparable to or better than surgical modalities, making this one of the more clinically mature non-regenerative applications of microneedling in periodontics.[10]

APPLICATIONS IN PERIODONTAL REGENERATIVE THERAPY

Microneedling has emerged as a biologically driven adjunct in periodontal regenerative therapy by enabling controlled activation of endogenous healing pathways at sites traditionally managed through invasive surgical approaches.

1. Management of Periodontal Intrabony Defects

Intrabony periodontal defects represent the most classical indication for regenerative therapy. However, their regenerative outcomes are often compromised by inadequate angiogenesis, limited progenitor cell availability, and chronic inflammation. Microneedling addresses these limitations by biologically activating the defect walls prior to or in conjunction with regenerative procedures.[1] Microneedling of intrabony defect surfaces enhances local bleeding and stabilizes the blood clot, stimulates the release of endogenous growth factors, and promotes the recruitment of osteoprogenitor cells and periodontal ligament stem cells.[3]

2. Enhancement of Periodontal Ligament Regeneration

True periodontal regeneration requires reformation of a functionally oriented periodontal ligament (PDL) between newly formed cementum and alveolar bone. Microneedling has shown promise in enhancing PDL cell migration, proliferation, and alignment, which are critical for re-establishing periodontal attachment.[3]

3. Cementum Regeneration and Root Surface Bioactivation

The root surface plays a decisive role in periodontal regeneration. Microneedling applied adjacent to root surfaces contributes indirectly to cementogenesis by enhancing cellular interactions at the root–tissue interface.[4]

4. Regeneration in Furcation Defects

Furcation defects present unique anatomical and biological challenges due to restricted access, limited blood supply, and complex root morphology. Microneedling has been proposed as a minimally invasive adjunct to enhance regenerative outcomes in early to moderate furcation involvement.[5] In furcation areas, microneedling improves vascular penetration into the furcation zone, enhances soft-tissue adaptation, and promotes progenitor cell migration into anatomically constrained spaces.

5. Synergistic Use with Platelet Concentrates and Biologics

One of the most significant applications of mi-

croneedling in periodontal regenerative therapy is its synergistic use with biologics, particularly platelet concentrates such as platelet-rich fibrin (PRF). Microneedling enhances the regenerative efficacy of biologics by increasing tissue permeability and biological receptivity.

Key advantages include:

- Improved diffusion of growth factors
- Enhanced retention of biologic materials at defect sites
- Prolonged biological activity due to improved vascularization

6. Minimally Invasive Periodontal Regenerative Procedures

Microneedling aligns closely with the principles of minimally invasive periodontal surgery (MIPS) by reducing surgical trauma while maximizing biological stimulation.8

In minimally invasive protocols, microneedling can be performed transgingivally while preserving soft-tissue integrity and minimizing postoperative morbidity.

7. Role in patients with Adverse Systemic Conditions

Systemic conditions such as diabetes and smoking adversely affect periodontal wound healing by impairing angiogenesis and cellular function. Microneedling has been shown to partially counteract these effects by enhancing local vascularization and growth factor signaling.[6]

8. Adjunctive Role in Peri-implant Regenerative Therapy

Although peri-implant tissues differ biologically from periodontal tissues, microneedling has potential applications in peri-implant soft- and hard-tissue regeneration, particularly around early peri-implant defects.[7] This occurs by inducing controlled micro-injury that enhances local bleeding, stimulates the release of growth factors, promotes angiogenesis, and facilitates migration of osteogenic and fibroblastic cells, thereby supporting early regenerative healing around implants.

9. Bioactivation of Regenerative Scaffolds and Biomaterials

Recent advances in biomaterials research suggest that microneedling can be used to bioactivate recipient sites prior to scaffold placement. Microneedling enhances scaffold integration by promoting cellular infiltration and vascular ingrowth.

ROLE OF PLATELET RICH FIBRIN (PRF) IN MICRONEEDLING ASSISTED PERIODONTAL REGENERATIVE THERAPY

The role of PRF in microneedling-assisted periodontal regeneration can be understood through its biological synergy with micro-injury-induced healing cascades, resulting in improved cellular recruitment, angiogenesis, and tissue maturation.

1. Biological Rationale for Combining PRF with Microneedling

Microneedling initiates an acute wound-healing response characterized by platelet activation, growth factor release, and transient inflammation. PRF complements this process by providing a three-dimensional fibrin matrix enriched with platelets and leukocytes, which stabilizes the initial blood clot and serves as a sustained reservoir of bioactive molecules.[2]

2. Enhancement of Growth Factor Bioavailability

PRF contains a concentrated array of growth factors, including platelet-derived growth factor (PDGF), transforming growth factor-β (TGF-β), vascular endothelial growth factor (VEGF), and insulin-like growth factor (IGF). Microneedling further enhances the bioavailability of these growth factors by creating controlled micro-channels within the defect and surrounding tissues, which facilitate deeper diffusion and sustained release of PRF-derived mediators, stimulate local platelet activation, and improve tissue permeability, thereby amplifying the regenerative signaling at the target site.

3. Promotion of Angiogenesis and Vascular Stability

Angiogenesis is a critical determinant of successful periodontal regeneration. PRF is intrinsically angiogenic due to its VEGF content and fibrin architecture. Microneedling further augments this effect by stimulating endothelial cell migration and capillary sprouting.[4]

4. Stimulation of Fibroblast Proliferation and Matrix Formation

Fibroblasts are central to periodontal connective tissue regeneration. PRF provides both biochemical signals and a physical scaffold for fibroblast adhesion and migration. Microneedling enhances fibroblast responsiveness by activating mechanotransduction pathways that regulate cell behavior.

5. Enhancement of Osteogenesis and Bone Regeneration

PRF has been shown to stimulate osteoblastic differentiation and bone matrix formation. Microneedling augments this osteogenic potential by increasing local vascularization and stem cell recruitment from bone marrow spaces.[6]

6. Improvement of PRF Retention and Stability at the Regenerative Site

One limitation of PRF is its susceptibility to displacement and rapid resorption. Microneedling improves PRF retention by:

- Creating micro-anchorage points
- Enhancing fibrin interlocking with connective tissue
- Promoting rapid tissue integration[5]

This improved stability ensures sustained biological activity throughout the critical phases of periodontal regeneration.

7. Role in Minimally Invasive Periodontal Regenerative Protocols

Microneedling in combination with PRF allows transgingival biological activation, while PRF provides localized regenerative support without the need for extensive surgical exposure. This concept is consistent with minimally invasive regenerative approaches such as the study by Pierpaolo Cortellini and Maurizio S. Tonetti which introduced the Minimally Invasive Surgical Technique (MIST), and their later study described the Modified Minimally Invasive Surgical Technique (M-MIST).

ADVANTAGES OF MICRONEEDLING IN PERIODONTAL REGENERATIVE THERAPY

Microneedling offers several biological, clinical, and procedural advantages that distinguish it from conventional periodontal regenerative modalities. Its primary strength lies in its ability to activate endogenous regenerative mechanisms while minimizing surgical morbidity.[1]

1. Minimally Invasive Biological Stimulation

One of the most significant advantages of microneedling is its minimally invasive nature. Unlike traditional regenerative surgeries that require flap elevation, extensive bone manipulation, or membrane placement, microneedling induces regeneration through controlled micro-injury without gross tissue disruption.[1]

2. Activation of Endogenous Regenerative Pathways

Microneedling does not rely on exogenous grafts alone but instead stimulates intrinsic wound-healing cascades, including growth factor release, stem cell recruitment, and angiogenesis.[2]

3. Improved Angiogenesis and Tissue Perfusion

Adequate vascular supply is essential for periodontal regeneration. Microneedling promotes rapid and dense neovascularization, thereby enhancing oxygenation and nutrient delivery to the regenerating tissues.[4]

4. Reduced Patient Morbidity and Improved Acceptance

Due to its minimally invasive nature, microneedling is associated with:

- Reduced postoperative pain
- Minimal swelling
- Faster functional recovery.[5]

These factors improve patient compliance and acceptance, particularly in cases where patients are hesitant to undergo conventional surgical regenerative procedures.

5. Cost-Effectiveness and Accessibility

Microneedling devices are relatively affordable compared to complex regenerative systems. When combined with autologous biologics such as PRF, microneedling enables a cost-effective regenerative

approach that can be readily incorporated into routine clinical practice.[7]

LIMITATIONS OF MICRONEEDLING

Despite its promising advantages, microneedling has several limitations that currently restrict its widespread clinical adoption in periodontal regenerative therapy.

1. Lack of Standardized Clinical Protocols

One of the primary limitations is the absence of standardized protocols regarding:

- Needle length
- Application frequency
- Treatment intervals
- Site-specific indications

This variability leads to inconsistent clinical outcomes and reduces reproducibility across studies.

2. Technique Sensitivity and Operator Dependence

Microneedling outcomes are highly operator-dependent. Excessive penetration depth or improper angulation can lead to:

- Tissue trauma
- Delayed healing
- Unintended bone or root surface damage.[2]

3. Limited Long-Term Clinical Evidence

While short-term outcomes are encouraging, there is a paucity of long-term randomized controlled clinical trials evaluating the stability of regenerated tissues following microneedling-assisted therapy.[3]

4. Inability to Replace Structural Scaffolds in Large Defects

Microneedling primarily functions as a biological stimulator and does not provide structural support. In large or non-contained periodontal defects, microneedling alone may be insufficient without adjunctive graft materials or scaffolds.[4]

5. Risk of Overstimulation and Fibrosis

Excessive or repeated microneedling may lead to:

- Prolonged inflammation
- Fibrotic tissue response
- Disorganized matrix deposition.[5]

This highlights the importance of controlled application and appropriate case selection.

FUTURE DIRECTIONS IN MICRONEEDLING-ASSISTED PERIODONTAL REGENERATIVE THERAPY

The future of microneedling in periodontal regeneration lies in technological refinement, biological integration, and clinical validation.

1. Development of Standardized, Evidence-Based Protocols

Future research must focus on establishing:

- Optimal needle depths for periodontal tissues
- Frequency and timing of applications
- Defect-specific treatment algorithms[6]

Standardization will improve reproducibility and clinical reliability.

2. Integration with Advanced Biomaterials

Emerging biomaterial research suggests that microneedling can be combined with bioactive scaffolds, hydrogels, and growth factor–loaded matrices to enhance regenerative outcomes.[7] Such integration may allow microneedling to serve as a method for conditioning the site prior to scaffold placement.

3. Bioengineered and Drug-Loaded Microneedles

Future microneedling devices are expected to feature:

- Biodegradable needles
- Controlled drug delivery systems
- Growth factor or exosome release platforms.[7]

These innovations could transform microneedling from a mechanical stimulus into a precision-guided regenerative delivery system.

4. Stem Cell Based Therapies

Microneedling-induced microenvironments are well-suited for stem cell homing and exosome-mediated signaling. Future therapies may exploit this synergy to achieve more predictable periodontal regeneration.

CONCLUSION

Microneedling has emerged as a promising biologically based adjunct in periodontal regenerative therapy by utilizing controlled micro-injury to stimulate endogenous wound-healing and regenerative pathways. By enhancing angiogenesis, facilitating stem cell recruitment, promoting extracellular matrix remodeling, and modulating the inflammatory response, it helps transform a chronically inflamed periodontal environment into one that supports regeneration. When used in conjunction with biologics such as platelet-rich fibrin, its regenerative effects can be further enhanced, contributing to improved soft- and hard-tissue outcomes with minimal surgical morbidity. Although current evidence indicates favorable biological potential and safety, the absence of standardized protocols and long-term clinical evidence highlights the need for further well-designed studies. With ongoing progress in biomaterials, mechanobiology, and clinical research, microneedling has the potential to become an important component of predictable and minimally invasive periodontal regenerative therapy.

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Anchoring Beyond the Ridge: The Basal Implant Approach

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ABSTRACT

Basal implants are a specialized form of dental implant designed to obtain anchorage from the dense basal cortical bone of the jaws. Unlike conventional implants, which primarily depend on cancellous and crestal bone for osseointegration, basal implants utilize the highly mineralized cortical bone, which is relatively resistant to resorption. Their design and surgical protocols allow placement in patients with reduced alveolar bone volume, often avoiding extensive bone augmentation procedures. Many basal implant systems permit immediate loading and early functional rehabilitation, making them useful in selected cases requiring rapid restoration of function and esthetics. Different designs, including screw-type basal implants and basal osseointegrated implants (BOI), have been developed to engage the available cortical bone. Depending on the implant design and clinical situation, placement may be performed using flapless or flap-based approaches. Despite their potential advantages, careful case selection, adequate surgical expertise, prosthetic planning, and long-term clinical evaluation are essential for predictable outcomes. This review discusses the principles, types, indications, surgical techniques, advantages, limitations, and clinical considerations associated with basal implantology.

Keywords: Basal implants; Basal cortical bone; Immediate loading; Cortical anchorage; BOI implants; Implantology; Atrophic ridge; Flapless implant placement; Immediate rehabilitation; Dental implants.

Introduction:

Basal implantology is a new implantology technique that engages the basal cortical bone of both jaws to ensure implant retention. Basal implantology is also known as bicortical implantology because it is the permanent, unchanging structure of the maxilla and mandible bone that gives exceptional bicortical anchorage. The advantages of basal implants over conventional implants include the fact that they are performed in a single session utilizing a flapless method, are straightforward, cost-effective, and require the least amount of equipment. It provides an infection-free zone of bone for implants, a larger stress-bearing area, a lower resorption rate, and the ability to give rapid prosthesis, which is not possible with standard crestal implants. Furthermore, basal implantology requires no ridge augmentation procedures, and they are single-piece implants that can be loaded immediately, resulting in great implant strength.[1,2]

History

German and French dentists have long been at the forefront of inventing the idea of basal implants. In 1972, Dr. Jeanmarc Julliet developed and used a single-piece implant with two designs, with the pin threaded fully and giving little resilience. It was only accessible in one standard length, with no uniform cutting tools

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available and were limited to places where the base plate anchors both cortical structures.[2]

Later, in 1975, Dr Clunet-Coste developed the t-shaped single unit implant procedure. These implants were marketed by the Eugen Kuhlman Company and its affiliate Zerca ceased the manufacture of these implants following Juliette's death.[2]

Dr Gerard Scortecci, a French dentist, solved this issue in the mid-1980s by presenting the world with an improved basal implant system and related surgical instruments. He created two types of implants known as "disk implants" that were connected to the prosthetic superstructure via internal and exterior connectors[4].

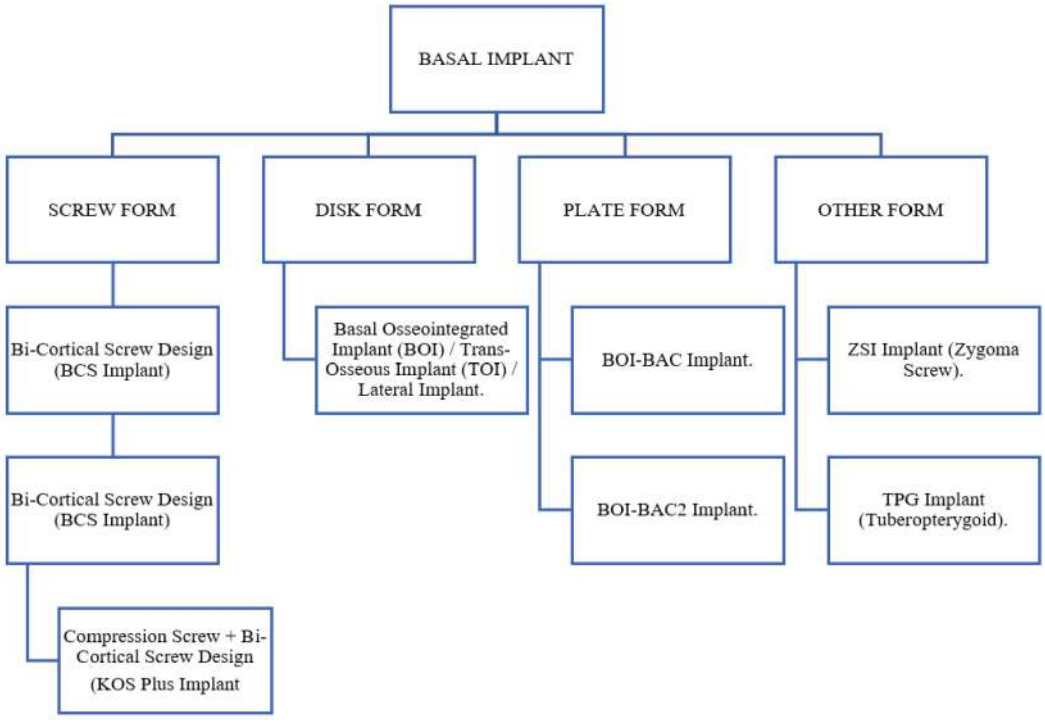
Several German dentists developed unique implants based on the disk implant system in the mid-1990s, together with the necessary surgical instruments and accessories. In 1997, Dr. Stefan Ihde developed the basal osseointegrated implant (BOI) technology, sometimes known as lateral basal implants. These implants were round and had a roughened surface. These implants were designed to transmit masticatory force vertically as well as basally. Soon later, Dr. Ihde modified the basal implant by placing edges on spherical base plates, preventing the implants from rotating in the bone prior to integration.[4]

In 2002, Europe and the United States developed and patented a fracture-resistant base plate. Bending zones appeared immediately in the vertical implant shaft. Screw-able designs (BCS and GBC) were launched in 2005 as technology advanced. In terms of surface structure alterations, the vertical implant part was first polished in the 1990s, but beginning in 2003, the complete basal implant was created with polished surfaces. The polished surfaces had the advantage of being free of mucositis and peri-implantitis, and the implants could be reintegrated if unfastened. Two-piece basal implants were first created, then one-piece devices were introduced.[4]

Definition of basal implants

Basal implants are specially designed dental implants that obtain their primary stability by engaging the dense basal cortical bone of the maxilla and/or mandible, allowing immediate functional loading even in severely resorbed ridges.[1]

Basal implantology is the lateral insertion of disk-shaped implants into basal bone, and more broadly, the anchorage of implants in basal bone.[5]



Flow chart: 1 classification based on basal implant types

Rationale for using basal implants

The concept of basal implantology originates from the presence of two distinct parts of jaw bone

A) the tooth bearing alveolus also called the crestal bone.

B) basal bone

The crestal bone that bears the tooth is less dense in nature and hence, it is exposed to infection from the pathologies of tooth/injuries/iatrogenic factors and has less load bearing capacity. Basal bone is defined as the osseous tissue of maxilla and mandible except alveolar process. The basal bone is densely corticated with less susceptibility to infections and resorption, it offers excellent support for the anchorage of implants. Thus, the rationale of using basal implants is that the cortical bone is more resistant to resorption, which originated from the orthopaedic, hence basal implants are also called orthopaedic implants.[1,3]

Basal implant types based on morphology

There are four basic types of basal implants available[8] Flow Chart:1

1. Screw Form.
2. Disk form.
3. Plate forms
4. Other forms.

Both of the types can be further categorized into-

I. Screw form

a. Compression screw design (KOS implant) [Fig:1]

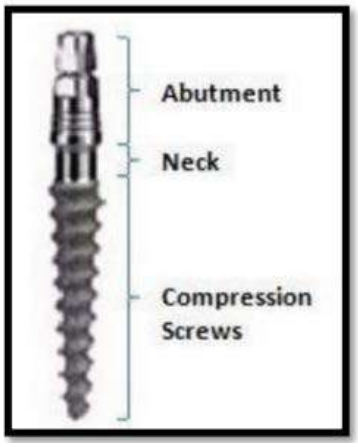


Fig: 1 Compression screw design

b. Bi-cortical screw design (BCS implant) [Fig:2]

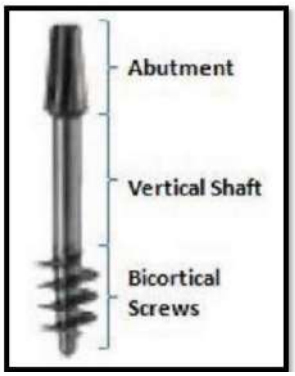


Fig: 2 Bi-cortical screw design

c. Compression screw + bi-cortical screw design [Fig:3]

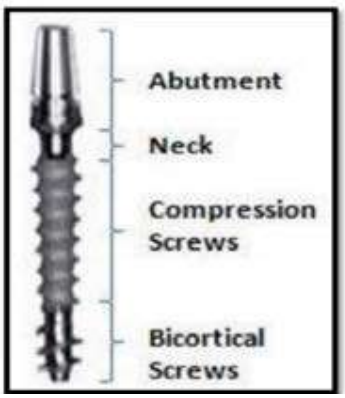


Fig: 3 Compression screw + bi-cortical screw design

II. Disk form

Basal osseointegrated implant (BOI) / trans-osseous implant (TOI) / lateral implant. [Fig:4]

Fig: 4 Basal osseointegrated implant (BOI) / trans-osseous implant (TOI

1) According to abutment connection

A. Single piece implant. [Fig:5]

Fig: 5 Single piece implant

B. External threaded connection. [Fig:6]

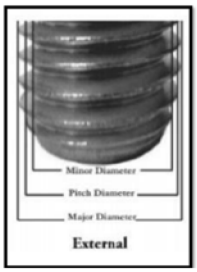


Fig:6 External threaded connection.

a)External hexagon. [Fig:7 a]

b)External octagon. [Fig:7 b]

Fig: 7 (a) External hexagon., Fig:7(b) External octagon.

C. Internal threaded connection. [Fig:8]

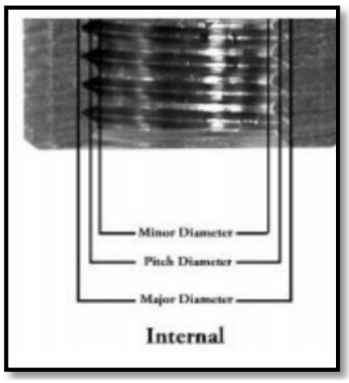


Fig: 8 Internal threaded connection

D. According to basal plate design-

a. Basal disks with angled edges.

b. Basal disks with flat edges also called as s-type Implant.

E. According to number of disks. [Fig:9]

a. Single disk.

b. Double disk.

c. Triple disk.

Fig: 9 (a) Single disk, Fig: 9(b) Double disk., Fig(c) Triple disk.

III.Plate form[Fig:10]

a.BOI-BAC implant.

b.BOI-BAC2 implant.

Fig:10(a) BOI-BAC implant, Fig:10(b).BOI-BAC2 implant

IV. Other forms

a. TPG implant (TUBEROPTERYGOID) [Fig:11].

Fig:11 TPG implant (TUBEROPTERYGOID)

b. ZSI implant (zygoma screw). [Fig:12]

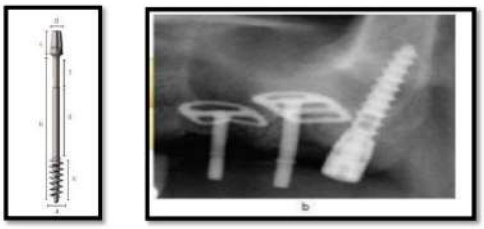


Fig: 12 ZSI implant

Types of basal implants [Fig:13]

There are two types of basal implants

A) basal osseo integrated implants (BOI)

B) basal cortical screw implants (BCS)

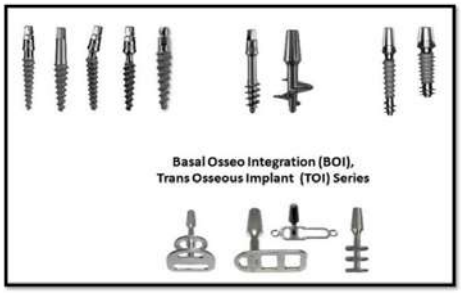


Fig: 13 Basal Osseo Integrated Implants

Basal osseo integrated implants: lateral implants, also known as lateral bone implants, are placed into the jaw bone laterally and are confined to the cortical bone. These implants are specific to certain areas and mainly transmit load to the horizontal segments. There are two types of lateral implants based on the area in which they are used:

■ **Anterior implants:** these are used in the front area of the mouth where there is sufficient vertical space. anterior implants: in the anterior edentulous area with ample of vertical space, two disk implants with crestal and basal disks are used. The crestal and basal disks have a diameter of 7mm and 9-10mm respectively. The purpose of crestal implants is to provide stability until the basal disks are completely ossified to its load bearing capacity. When there is lack of vertical space, then a single disk basal osseointegrated implant is placed with 7-9mm diameter and shaft length between 8- 13.5mm.

■ **Posterior implants:** in the posterior segments, square shaped implants are used as the threaded pins inserted from the side have better medial position and this placement helps to compensate the absorption of distal mandible. The desired vertical dimension of disk 9-12mm or 10-14mm diameter and shaft length of 10-13.5mm can be used according to the availability of horizontal bone. An inferior nerve insertion is done when the vertical bone height above nerve is just 2mm. The disk is introduced below the nerve with the thread carrier located at the side of the nerve.

Basal Cortical Screw Implants: the bi-cortical screw implants are flapless implants that are placed directly through the gingiva, like a conventional implant. These implants transmit the load of the masticatory forces deep into the cortical bone of the opposite side. They provide initial elasticity and show less susceptibility to peri-implantitis as there is thin mucosal penetration diameter and highly polished surface.[6,7,8]

Parts of Basal Implants

The evolution of basal implant design can also be briefly discussed. Early basal implants evolved from single-piece designs, followed by the development of lateral/disk implants (BOI) and subsequently screw-based systems such as BCS, KOS and KOS Plus. Design modifications included changes in the basal plate, introduction of bending zones, development of screwable designs, and adoption of polished surfaces. [8]

The basal implants have 3 main parts:

Implant Body: the implant body is thin with wide threads which helps in the increase of implant bone contact area and increases the vascularity around the implant.

Implant Neck: the part that connects the implant and its abutment is called the neck of the implant. The abutment can be bent to an angle of 15-25 degree depending on the length of the implant.

Implant Surface: the surface of the basal implant is polished with less amount of accumulation of plaque and bacteria.

Implant Morphology:[8]

Basal osseo integrated implants [BOI] implant morphology

The BOI implant is made of either pure titanium or a titanium molybdenum alloy to increase its strength. These might be either single or two pieces. The following are the parts of the BOI implant.

A. The abutment portion

In single-piece BOI implants, the abutment component is conical and remains exposed in the oral cavity, but in two-piece BOI implants, the abutment section can be an externally threaded screw or an internally threaded screw with an exterior hexagonal or octagonal restorative platform.

B. Neck

It is the area just beneath the abutment piece. This section may or may not be constricted in diameter; constriction improves post-healing gingival adaptation, lowers rigidity, and allows for bending by 15°-25°[6].

C. Vertical shaft

This is the portion that connects all of the implant's components. The shaft is smooth and polished to prevent plaque collection and irritation; it can also be elastic or rigid, depending on the diameter and type of titanium utilized. The vertical shaft is only a load bearing component and is typically 10 - 13.5 mm long.

D. Crestal disk

This is the first disk in the implant. It is referred to as a crestal disk because it is located in the crestal bone following implant implantation. This disk serves a twofold purpose: immediately after implant insertion, it supplies and maintains primary stability, and following osseointegration, it transforms into a load bearing and distributing component.

E. Basal disk [Fig:14]

It is the second disk at the base of the implant and is the last component in the implant body. This part is also kept polished and serves as both a load bearing and dispersing component. The section of the shaft that connects to the basal disk is elastic and can bend 15°-25°. The distance between the crestal and basal disks is typically 5 mm.

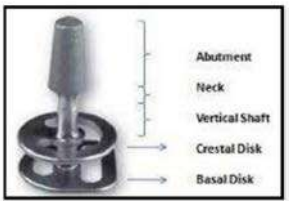


Fig:14 Basal disk

Basal cortical screw implants:

The BCS implants also known as screwable basal implants positioned like a conventional implant i.e. these are flapless implants that are inserted through the gingival without introducing any cut single-piece implant design with modification in abutment design and implant portion[8]. The abutment can be conical straight, conical angled, and multi-unit abutments. They have wide screws that help in engaging the buccal and lingual/ palatal cortical plates to provide primary stability to the implants and later serve as load-bearing and distributing components. Screwable implants are also considered as basal implants because they transmit masticatory forces deep into the bone, customarily onto the opposing cortical bone. This type contributes some elasticity to the implants and by highly polished surface and thin mucosal penetration diameter they are generally not prone to peri- implantitis.[7]

KOS and KOS PLUS implants:

KOS and KOS PLUS implant morphology these implants are made of titanium molybdenumor titanium aluminum vanadium alloy and are single- piece implants. These implants are made to function similarly to compression screws; when inserted into the bone, they will compress the cancellous bone around them to create a denser, more compact bone. These are compressed screw (KOS) and compressed screw with bi-cortical screw design (KOS PLUS). The principle of these implants is that when the implants are placed into the bone it compresses the surrounding cancellous bone to form more compact dense bone.[2]

A) Abutment: the restorative platform is exposed to the oral cavity. The different kinds of abutment available are: conical straight, conical angled, locator abutments, ball abutments, multiunit abutments.

B) Neck: neck is polished and bent at 15-25 degrees.

c) Implant Portion: the implant has a highly polished surface with several threads, wide structure and turns that enables it to apply compressive forces on the cancellous bone to convert it into dense cortical bone.

KOS Plus implant: The apical third of the KOS Plus implant incorporates additional basal cortical screws (BCS) that engage the buccal and palatal/lingual cortical plates. This bicortical engagement provides

primary stability and subsequently contributes to load bearing and load distribution. The BCS component of the KOS Plus implant is maintained in a highly polished condition.[8]

Indications and contraindications[8,9]

Indications:

1. In cases where multiple teeth are absent or to be extracted in future.

2. When the conventional implants or bone augmentation procedure have failed.

3. In cases where either bone height or bone thickness is not sufficient.

Absolute contraindications:

1. Patients on drugs like high dose of IV bisphosphonates for osteoporosis / cancer, anticoagulants, etc.

2. Epileptic patients

3. Patients undergoing radiotherapy for cancer.

4. Severe heart disease or stroke within 6 months.

5. Allergy or hypersensitivity reaction to titanium alloy.

6. Acquired immunosuppressive syndrome (AIDS)

7. Age < 15 years.[19]

Relative contraindications:

1. Bruxism, clenching, malocclusion, history of fracture of tooth associated with psychological problems.

2. Facial or trigeminal neuropathy.

3. Uncontrolled diabetes.

4. Lesion of the oral mucous 3membrane.

5. Smoking

6. Poor oral hygiene

7. Infection of the surrounding teeth (periodontal pockets, cysts, granuloma, etc).

Advantages and disadvantages[8,9]:

Advantages

1. One-piece implants: this concept of one-piece implants has led to a reduction in the failure of implants due to interface problems between the connection between different parts of the implant.

2. Basal-cortical support: these implants take support from the basal bone which has stable and fast repair capacity along with increased resistance to resorption.

3. Compromised ridges: basal implants are the best treatment modality for atrophied ridges, as the augmentation procedures can be avoided, the outcome of which is unpredictable.

4. Distribution of masticatory forces: the masticatory forces are directly transmitted to the cortical bone, which is the load-bearing and distributing area.

5. Peri-implantitis incidence: peri-implantitis is often seen as a complication of conventional implants due to the roughened surface and multiple parts of the implants. The incidence of peri- implantitis is reduced to 98% in case of basal implants due to the polished surface that decreases the accumulation of plaque and bacteria onto the implant surface.

6. Medically compromised individuals: basal implants have shown great results in patients with diabetes, chronic smokers and chronic periodontitis.

7. Immediate loading: the advantage of basal implants is that the prosthesis can be given immediately within 72 hours of surgery.

8. Minimally invasive: the surgery is minimally invasive with fast healing and less post operative complications.

Disadvantages:

1. Compromised aesthetics in case of single tooth replacement.

2. Technique sensitive: a skilled surgeon with good knowledge of the anatomy is required to perform the surgery successfully.

3. May lead to excess bone loss in case of good bone support.

Surgical technique:

Basal implants require a surgical approach that differs from that of conventional crestal implants. The technique involves minimal osteotomy preparation and limited bone drilling, thereby reducing the risk of thermal injury to the surrounding bone. External irrigation is generally used during osteotomy preparation. For KOS, KOS Plus, and BCS implants, a single pilot osteotomy using a pathfinder drill is often sufficient,

while manual drills may be used when more controlled osteotomy preparation is required.[3,10,11]

A flapless approach is generally advocated for screw-type basal implants because their design permits direct engagement of the available cortical bone without extensive exposure of the underlying bone. Fig. 15 Avoiding flap elevation may also help preserve the periosteal blood supply and reduce surgical morbidity. Furthermore, because basal implants are designed for immediate loading, immediate prosthetic rehabilitation can be performed without waiting for conventional osseointegration.[3,10,11]

The surgical approach differs for BOI (basal osseointegrated implant) systems. In these implants, access to the basal cortical bone is obtained by raising a lateral flap and preparing a horizontal osteotomy using disk drills of the appropriate diameter. The osteotomy is prepared in a T-shaped configuration, allowing the disk portion of the implant to be inserted laterally into the basal cortical bone. After placement, the implant is covered by repositioning and suturing the flap. [Fig:15][3,10,11]

Peri-implant healing in basal implants

Peri-implant healing (BOI and BCS implantation) because these implants have a unique design, peri-implant healing is likewise unique. Basal implantologists refer to what conventional implantologists refer to as "osseointegration" as "osseoadaptation". According to basal implantology philosophy, the process of osseoadaptation is carried out by a "bone multicellular unit" (BMU), which is described as resembling a cutting cone with a tail; the cutting cone is made up of osteoclastic cells that eat away the peri-implant bone, and the tail is made up of osteoblastic cells that lay down bone; as this unit moves through the bone, osteoclastic activity is followed by osteoblastic activity. This BMU forms when the boi and bcs implant are subjected to rapid stress, resulting in remodeling under functional stresses, resulting in the development of this unit, which commences the healing phase and leads to the formation of a dense peri-implant bone[15]

The cascade of processes involved is as follows[3]

- activation phase: in this phase the precursor cells/human mesenchymal stem cells develop into osteoblasts and osteoclasts. This phase lasts for 3 days.
- resorption phase: during this phase osteoclastic activity occurs which reveals soft and porous bone. Osteoclastic activity occurs at a rate of 40μm/day.
- reversal phase: in this phase osteoblastic activity takes place. The osteoblasts lay down neo bone in the haversian canals at a rate of 1-2μm/day.
- progressive phase: this phase involves the osteoblasts forming concentric lamella in haversian canals, which leads to reduction in diameter of the canal and increase in bone density. At this stage the diameter of the haversian canal is 40- 50μm. The bone formed is a non-mineralized matrix osteoid and this phase lasts for 3 months.
- mineralization phase: after 10 days of osteoid formation mineralization phase begins.

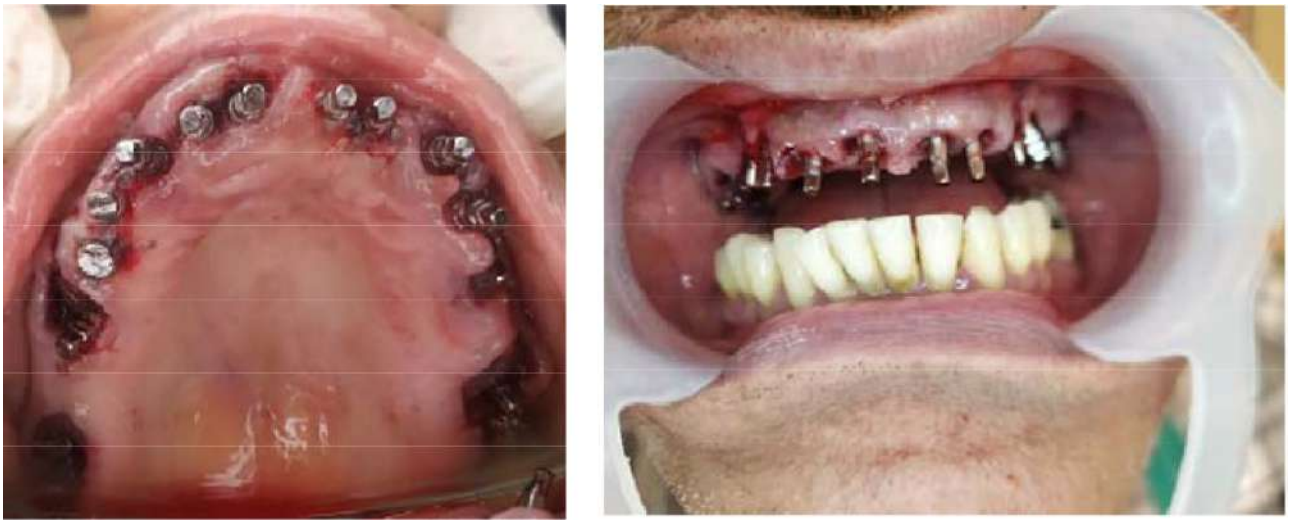


Fig : 15 flapless basal implant placement

This phase involves two stages

a) **primary mineralization stage:** this stage imparts primary hardness to the osteoid and accounts for 60% of all mineralization.

B) **secondary mineralization stage:** this stage imparts the final hardness and final morphology of bone. This phase lasts for 6-12 months.

- Dormant Phase.

During the dormant phase, osteoblasts may differentiate into osteocytes or become bone-lining cells, which subsequently participate in the mechanical, metabolic, and homeostatic functions of bone.[1,2]

In basal implantology, peri-implant bone remodeling is considered a continuous, load-responsive process. Functional loading stimulates the bone multicellular unit (BMU), resulting in coordinated bone resorption and formation and subsequent adaptation of the peri-implant bone to the implant surface. This load-related remodeling and adaptation has been described in basal implantology as "osseoadaptation" and has been referred to as the "fourth dimension" of bone remodeling.[1,3]

The concept of osseoadaptation should be distinguished from conventional osseointegration terminology, although both involve dynamic bone remodeling at the bone–implant interface. Experimental and computational studies have demonstrated that basal implants undergo changes in bone–implant contact and load distribution during healing, with remodeling and maturation of the surrounding bone occurring over time.[3,4]

Basal implants have therefore also been described as "orthopedic implants" because their treatment philosophy emphasizes cortical anchorage, functional loading, and load-induced bone adaptation.[1] However, this terminology represents a concept used within basal implantology and should not be interpreted as evidence that basal and orthopedic implants have identical biological healing mechanisms.[3,4]

Basal implants for atrophied ridges :[1,3]

Rehabilitation of severely atrophied ridges is challenging and may require extensive augmentation procedures in conventional implant therapy.[1,2] Basal implants can utilize the dense cortical and basal bone for anchorage, thereby reducing the need for ridge augmentation in selected cases.[1,3] Different basal implant designs can be selected according to the available bone anatomy and prosthetic requirements.[1,3]

I. General Systemic Considerations[1,3]

According to basal implantologists it doesn't matter until the patient has had a recent myocardial infarction, cerebrovascular accident, immunosuppressant therapy, chemo and/or radiotherapy and bisphosphonate therapy. Diabetes is not a huge concern as long as blood sugar levels are in control, also it doesn't matter if the recipient is a smoker or not.

II. Biomechanical Considerations[3]

The conventional bone-density classifications proposed by Misch may have limited applicability to basal implantology because basal implants utilize a different osteotomy preparation and placement protocol compared with conventional crestal implants.[3] Routine assessment of bone density may therefore be less critical, as the biomechanical environment changes following implant insertion and functional loading. Bone exhibits viscoelastic and adaptive properties, and basal implant designs aim to distribute functional loads over the surrounding cortical bone, thereby reducing excessive stress concentration and the risk of stress shielding.[3]

III. To Load or Not to Load?

According to the philosophy of basal implantology, the craniofacial skeleton is subjected to continuous functional forces, including lateral stresses generated by the surrounding musculature. Therefore, a basal implant may be considered to remain under some degree of functional influence even in the absence of a prosthetic superstructure.[3,15,6] Based on this concept, basal implants may be managed using different loading protocols depending on the clinical situation, implant stability, and prosthetic requirements. They may be left without a superstructure during the healing period or immediately loaded with a provisional or definitive prosthesis. Alternative protocols include prosthetic loading after approximately 3 days, 1 week, or 6–8 weeks, while a temporary restoration may also be maintained for 3–6 months before placement of the definitive prosthesis.[3,15,6]

IV. Which Jaw To Restore First[3]

The stomatognathic system consists of stationery (maxillary bone) and a mobile (mandibular bone) component, the role of the mobile component is to apply forces and the stationery component absorbs a considerable amount of the forces applied. Due to the above mentioned purpose of the jaws, it becomes imperative that the mandible should be restored first, also a conventional mandibular denture on an atrophied foundation is unstable, therefore, chewing function becomes poor and gradually the associated muscles lose their tonicity, because of fixed rehabilitation these adversities are avoided, thus, mandible should be restored first.

A. Atrophied Mandible

Rehabilitation of the severely atrophied mandible presents significant anatomical and biomechanical challenges. Various treatment concepts have been described in basal implantology, particularly with respect to the number and strategic distribution of implants.[3,15,18]

1. Multi-implant concept

The multi-implant concept, associated with the French school and pioneered by Scortecci, advocates the placement of a relatively large number of basal implants, commonly 7–12 implants, to provide extensive cortical anchorage.[3,18] Basal and crestal implants may be combined to provide a rigid prosthetic framework. However, excessive rigidity may restrict physiological mandibular flexure and force redistribution. As mandibular torsion cannot be completely eliminated, unfavorable stress distribution and mechanical overload may potentially develop at the implant–bone interface.[3,18]

2. Strategic implant positioning

An alternative approach, associated with the German school and Ihde, emphasizes strategic implant positioning rather than the use of a large number of implants. In severely atrophied mandibles, approximately four implants may be strategically positioned, preferably in the canine and second-molar regions.[15,19] This configuration is intended to accommodate physiological mandibular flexure and facilitate redistribution

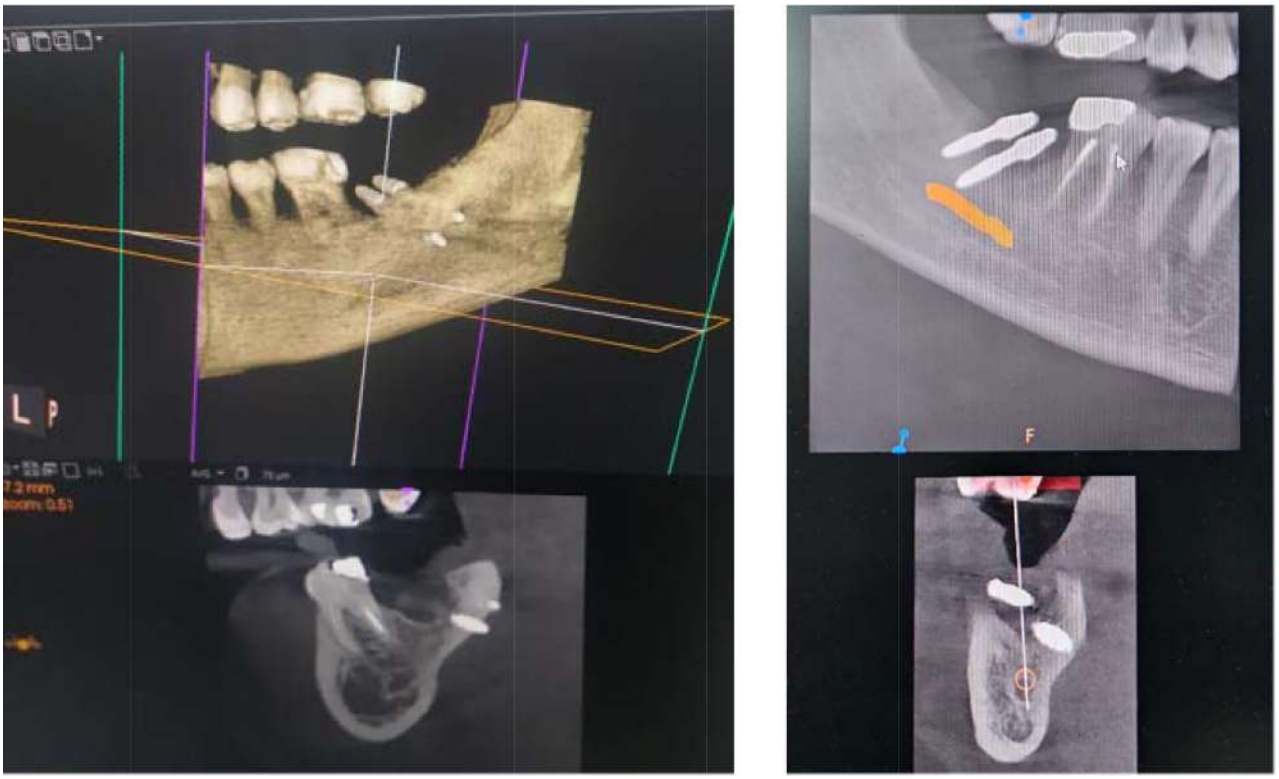


Fig: 16 Lingual Nerve Bypass Technique

of functional forces through an appropriately designed prosthesis.[15,19]

Infraneural Implantation Technique

Infraneural implantation is considered a separate surgical technique for the management of the atrophied mandible rather than a distinct school of implantology.[4,15,22]

With progressive mandibular resorption, the inferior alveolar nerve (IAN) may lie close to the residual alveolar crest, making conventional crestal implant placement challenging without bone augmentation or nerve repositioning.[4,15] In selected cases, BOI implants can be inserted laterally, with the osteotomy modified according to the anatomical relationship between the residual ridge and the IAN. The basal disk is positioned within the available basal bone while maintaining an appropriate relationship with the nerve, thereby potentially avoiding extensive augmentation or nerve-repositioning procedures.[4,15,22] This approach is referred to as infraneural implantation. [Fig:16]

B.Atrophied Maxilla[4,6,19]

The resorbed maxilla poses a considerable challenge for implant restorations. The pneumatized sinus and the porous bone make implant placement a challenging task. The porous bone is taken care of by the compression screw implants, whereas, for the sinus two techniques have been described, which describe alternate techniques of placement

i. Sinus section technique:

In this two/three walls of the sinus are sectioned to facilitate placement of the basal disk in the sinus. Basal implantologists leave the option of lifting the sinus membrane and grafting on the operator. The sole purpose of this technique is to gain bi-cortical support; also only one implant can be placed this way in each sinus.

ii. Tuberopterygoid (TPG) screws:

These implants are placed in the pterygoid bone and aid in providing additional support to the prosthesis. These are used in conjunct with sinus section technique and are placed at 20º-45º in the bone and the angulation between BOI implant and TPG screw should not exceed 90º otherwise prosthesis placement becomes difficult .

iii. Zygomatic screw implant (ZSI)

These are zygomatic implants that are placed in the zygomatic bone and like the BCS implant these also have sharp edged cortical screws that gain bicortical support

iv. Cortically fixed at once[22,21].

This is a very recent treatment proposed by Dr. Henri Diederich in 2013; it is based on basal cortical implantology and is primarily geared toward rehabilitating atrophied jaws regardless of the quantity of bone available, with no augmentations required. This is essentially a plate-shaped implant that resembles mini plates (used for fracture reduction) and has an abutment platform; its unique design allows it to bend and adapt to any surface, and it is fastened to bone with bone-expanding tiny screws . The number of holes necessary can be minimized; another advantage is their isoelasticity, which allows them to resemble bone. These implants are sub-periosteal, and so far, this regimen has demonstrated good results.

Conclusion

Dental implant placement has become a popular restorative therapy among dentists and is now a routine procedure. Basal implants, in particular, are gaining significant attention due to their advantages, such as time efficiency and improved support from the basal cortical bone.

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Glimpses of
SPIK Annual Conference 2026

The SPIK Annual Conference 2026 of the Society of Periodontists and Implantologists of Kerala (SPIK) was successfully conducted from May 8–10, 2026, at Mar Baselios Dental College, Kothamangalam, under the theme "Soft Tissue Excellence: The Periodontist's Signature." The conference brought together Periodontists, Implantologists, academicians, clinicians, postgraduate students, and delegates from across Kerala and neighboring states for three days of academic deliberations, scientific exchange, and professional fellowship.

The conference commenced on May 8, 2026, with two highly engaging pre-conference courses. Dr. R. Viswa Chandra, Professor & HOD of Periodontics at SVS Dental College, Mahbubnagar, conducted a hands-on program titled "Mastering the Mix: Simplifying Protocols for Autogenous Dentine Grafts," supported by Raamah BioCare Pvt Ltd. This was followed by an intensive session on "Mastering the Core Techniques of Root Coverage" by Dr. Nihal Devkar of Regeneration Hub, Pune, supported by Jull-Dent Mumbai. Both programs witnessed active participation and excellent interaction from delegates.

The formal inauguration of the conference was held on May 9, 2026, and was inaugurated by Adv. Dean Kuriakose, Hon'ble

Glimpses of
SPIK Annual Conference 2026

Member of Parliament, Idukki, in the presence of distinguished guests, office bearers of SPIK, faculty members, and delegates.

The scientific sessions featured lectures by eminent speakers covering contemporary topics in Periodontics and Implantology.

Dr. Ambili R, Professor & HOD of Periodontics, PMS College of Dental Science & Research, Thiruvananthapuram, delivered a lecture on "Periodontal Research in Kerala: Opportunities and Challenges."

The prestigious Dr. B. R. R. Varma Oration was delivered by Dr. R. Viswa Chandra on the topic "Extract. Process. Regenerate – Update on Autogenous Dentine Grafts."

Dr. Nihal Devkar, Regeneration Hub, Pune, presented the lecture "God's Own Pink: Peri-implant Soft Tissue Dynamics and Management."

Dr. Devisree Naveen, Professor & HOD of Periodontics, Azeezia Dental College, Kollam, spoke on "Soft Tissue Substitutes – Current Perspectives."

Dr. Anil Melath, Principal, Mahe Institute

Glimpses of
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of Dental Sciences, Mahe, delivered a lecture titled "An Insight into Cortico- Basal Implantology."

On the third day, Dr. Linta Thomas, Associate Professor of Periodontics, Mar Baselios Dental College, Kothamangalam, presented "From Bite to Breakdown: Mandibular Position and the Clockwork of Periodontal Response."

Dr. K. Harikumar Kanakkath, Professor & HOD of Periodontics, Government Dental College, Kozhikode, delivered the lecture "Periodontal Plastic Surgery – Reflections on Evidence & Experience."

A highly interactive panel discussion on "Future Treatment Trends in Periodontics: From Regeneration to Precision Care" was moderated by Dr. Baiju R M, Professor of Periodontics, Government Dental College, Kottayam. The panelists included Dr. Harish Kumar, Professor & HOD of Periodontics, KMCT Dental College, Kozhikode; Dr. Biniraj K R, Professor & HOD of Periodontics, Royal Dental College, Palakkad; and Dr. Jose Paul, Professor & HOD of Periodontics, Annoor Dental College & Hospital, Muvattupuzha.

Dr. Byju Paul Kurian, Principal, Mar Baselios Dental College, Kothamangalam, delivered the lecture "Impression Mastery: Capture & Create Implant Precision."

The conference also featured postgraduate paper and poster presentations, life member paper presentations, and an engaging Perio Quiz led by Dr. Lekshmi A. J., Associate Professor, Department of Periodontics, Mar Baselios Dental College, providing an excellent academic platform for young researchers and clinicians.

A major attraction of the conference was the Gala Banquet & Musical Night held at Hotel Cloud Nine, which enabled delegates to interact and strengthen professional camaraderie in a vibrant atmosphere. The event also included a friendly cricket match between ROYAL SPIK and the MBDC Cricket Team, adding a sporting spirit to the conference.

The conference concluded on May 10, 2026, with the valedictory function, SPIK Annual General Body Meeting, and installation of the new office bearers for the year 2026–27. The organizing committee expresses heartfelt gratitude to all speakers, sponsors, delegates, faculty members, students, and supporting staff for their contributions to the conference's grand success.

The SPIK Annual Conference 2026 successfully fulfilled its objective of promoting academic excellence, clinical advancement, and professional unity among Periodontists and Implantologists.

Journal of the Society of Periodontists, Kerala (JSPIK)

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Reference List: List all references at the end of the manuscript in numerical order. Do not arrange them alphabetically. Include only references cited in the text.

Author Names: Write surname first, followed by initials (without full stops or 'and'). Example: Rao A, Thomas J, Menon S.

Titles: Capitalize only the first word of the article or book title. Do not use quotation marks.

Punctuation: Use commas between authors and a full stop at the end of each reference.

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Chapter in a Book

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Example: Socransky SS, Haffajee AD. Microbial complexes in subgingival plaque. In: Newman MG, Takei HH, Klokkevold PR, editors. Carranza's Clinical Periodontology. 13th ed. St. Louis: Elsevier; 2023. p. 205–220.

Website

Format: Authors (if any). Title of the webpage. Name of the website. Year [cited year month day]. Available from: URL

Example: World Health Organization. Oral health. WHO website. 2023 [cited 2025 Sep 10]. Available from: https://www.who.int/news-room/fact-sheets/detail/oral-health

Quick Tips

• Use standard journal abbreviations as listed in PubMed.

• Check every reference carefully for spelling, punctuation, and accuracy.

• Avoid citing unpublished data or personal communications unless essential.

• Ensure every reference cited in the text appears in the list, and vice versa.

Maintaining consistent referencing reflects the scientific integrity of our journal and enhances its academic value.

— Editorial Board, JSPIK

INFORMATION TO AUTHORS

About the Journal

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

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