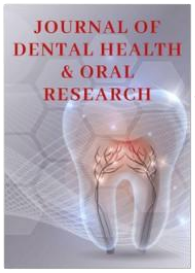

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*Review Article*

# Peri-Implant Mucositis and Peri-Implantitis: A Narrative Review of Etiology, Diagnosis and Current Treatment Modalities

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## Abstract

Peri-implant diseases represent one of the most prevalent and clinically significant complications in contemporary implant dentistry. Despite the widespread adoption of dental implants, peri-implant mucositis and peri-implantitis continue to affect a substantial proportion of implant-bearing patients, with reported prevalence rates of approximately 50% and 19.53%, respectively. The absence of universally standardized diagnostic criteria and treatment protocols has historically limited both clinical management and research comparability. This narrative review aims to synthesize current evidence on the etiology, diagnosis and treatment of peri-implant diseases, with the goal of providing clinicians with an evidence-informed framework for clinical decision-making and long-term implant maintenance. A peer-reviewed literature was conducted, encompassing studies on the microbial, host-related, prosthetic and systemic etiological factors contributing to peri-implant disease; diagnostic criteria established by the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions; and current non-surgical and surgical treatment modalities including mechanical debridement, adjunctive antiseptic and antibiotic therapies, laser therapy, resective surgery, regenerative surgery and implantoplasty. Bacterial biofilm remains the primary etiological trigger, while host immune response, prosthetic design, smoking, uncontrolled diabetes and a history of periodontitis are key risk modifiers. Peri-implant mucositis is reversible through non-surgical intervention; peri-implantitis requires a stepwise approach escalating to surgical management based on defect morphology and treatment response. Regenerative and resective surgical approaches demonstrate comparable long-term implant survival. A validated prognostic classification system for peri-implantitis remains absent from the literature. Effective management of peri-implant diseases requires individualized risk assessment, early diagnosis and structured maintenance protocols. Standardized treatment guidelines and long-term randomized controlled trials are needed to strengthen prognostic tools and improve patient outcomes.

**Keywords:** Peri-Implantitis; Peri-Implant Mucositis; Dental Implants; Biofilm; Surgical Treatment; Non-Surgical Treatment; Implant Maintenance; Bone Loss

## Introduction

Despite their well-documented success, dental implants are increasingly affected by inflammatory conditions involving the surrounding tissues [1]. Depending on whether the supporting bone is involved, these conditions are classified as peri-implant

mucositis or peri-implantitis [2]. Reported prevalence figures vary considerably, largely because a universally accepted case definition remains absent from the literature. A recent systematic review and meta-analysis confirmed that both conditions are common and that their frequency increases proportionally with implant loading time [3,4].

At the site level, peri-implant mucositis affects approximately 50% of evaluated locations, establishing it as a clinically significant and widespread condition [1]. This inflammatory response is associated with marked shifts in the submucosal microbiome, in which anaerobic pathogenic species progressively displace commensal flora [2]. At the patient level, the prevalence of peri-implantitis is estimated at 19.53%, dropping to 12.53% when only single-implant cases are considered [4]. These figures, however, underrepresent the true burden of disease, as risk is further compounded by behavioral and systemic host factors, including smoking and a prior history of periodontal disease [5]. Specific biomarkers identified in peri-implant crevicular fluid have also been associated with early pathological progression and their diagnostic utility continues to be explored [2]. When broader infection-related bone loss thresholds are applied, prevalence estimates exceed 10% across implant populations [5]. Collectively, the literature underscores the importance of structured preventive maintenance protocols and individualized risk stratification in managing these conditions over time [3,6].

Peri-implantitis has become one of the most consequential challenges in contemporary implant dentistry. As one of the leading causes of late implant failure, it is defined by progressive bone loss driven by a dysregulated host inflammatory response to microbial biofilm [7]. Beyond its biological impact, the condition carries substantial economic implications, repeated surgical interventions, prosthetic modifications and implant replacement represent a growing financial burden for patients and healthcare systems alike [6].

Accurately diagnosing peri-implant disease requires integrating clinical parameters, including probing depth and bleeding on probing, with radiographic evidence of crestal bone loss [8]. This distinction matters clinically, as mucositis remains a reversible condition while peri-implantitis, if left unaddressed, follows a nonlinear and accelerating pattern of destruction [9]. Among the most consistently identified risk factors are a history of periodontitis, tobacco use and poorly controlled diabetes, all of which modulate the host immune response and compromise soft tissue integrity around implants [3,9].

Treatment strategies have evolved considerably, shifting from conventional mechanical debridement toward more comprehensive approaches that include adjunctive antimicrobial therapy and regenerative surgery [10]. Non-surgical intervention remains appropriate for early-stage disease, while advanced osseous defects typically require resective or regenerative surgical correction. Emerging technologies, including biodegradable magnesium membranes for guided bone regeneration, represent promising adjuncts in the surgical management of complex defects [9,11]. This narrative review aims to synthesize current evidence on the etiology, diagnosis and treatment of peri-implant diseases to support clinical decision-making and improve long-term implant outcomes.

**Etiology and Risk Factors**

*Bacterial Biofilm and Microbial Composition*

Bacterial biofilm remains the primary etiological driver of peri-implant disease and its composition differs meaningfully from that associated with natural tooth periodontitis. Gram-negative anaerobic species predominate in established peri-implant infections, with *Porphyromonas gingivalis, Treponema denticola, Tannerella forsythia, Prevotella intermedia, Prevotella nigrescens, Streptococcus constellatus* and *Aggregatibacter actinomycetemcomitans* consistently identified in early disease development [12]. Beyond this classical consortium, *Staphylococcus aureus* has received increasing attention due to its particularly strong binding affinity to titanium alloy surfaces, the material used in the vast majority of contemporary implant systems, which facilitates early and persistent colonization that compromises peri-implant tissue stability [13].

Bacterial contamination at the time of implant placement also contributes to long-term disease risk. Prevotella intermedia, in particular, has demonstrated consistent short-term recurrence following initial treatment, suggesting it can persist in the oral environment and re-establish pathogenic biofilm even after therapeutic intervention [13]. This capacity for recurrence underscores the importance of monitoring microbial profiles over time, not only during the acute phase of disease management but throughout long-term maintenance.

*Host Immune Response*

While biofilm is the initiating factor, the magnitude and trajectory of peri-implant tissue destruction are ultimately determined by the host inflammatory response. In susceptible individuals, dysregulation of innate and adaptive immune pathways leads to an exaggerated cytokine cascade, including elevated levels of interleukin-1β, tumor necrosis factor-$\alpha$ and prostaglandin E2, that promotes osteoclastic activity and progressive marginal bone loss disproportionate to the bacterial load present [14]. This immunological amplification explains why two patients with comparable biofilm accumulation can present with markedly different clinical outcomes. The implant surface itself may also modulate this response, as surface roughness and material characteristics influence early protein adsorption and subsequent immune cell recruitment [14,15].

*Prosthetic Design and the Implant-Supracrestal Complex*

Prosthetic design is an underappreciated but clinically significant contributor to peri-implant disease. Restoration emergence angle and contour convexity directly affect the patient's ability to perform effective oral hygiene and this relationship has been quantified in the literature. Restorations with an emergence angle exceeding 30 degrees combined with high prosthetic convexity were associated with a 37.8% incidence of peri-implantitis, while recontouring those restorations to improve cleansability resulted in resolution of bone and soft tissue inflammation in 66.6% of cases [16]. Furthermore, 48% of implants diagnosed with peri-implantitis were classified as uncleansable, meaning neither the patient nor the clinician could adequately access the restoration for debridement [11].

Cement-retained prostheses introduce an additional risk vector. Residual subgingival cement, which can mimic the radiographic and clinical appearance of calculus, has been consistently associated with peri-implant inflammation and its removal frequently produces rapid improvement in the surrounding periodontium [17]. These findings collectively reinforce the principle that restorative decisions made at delivery have long-term biological consequences that extend well beyond aesthetics and function.

*Systemic Risk Factors: Diabetes Mellitus and Obesity*

The relationship between diabetes mellitus and peri-implantitis is nuanced and depends critically on glycemic control. Controlled diabetes, in isolation, has not been shown to significantly increase the prevalence of peri-implant disease. However, uncontrolled diabetes confers a 2.3-fold higher incidence of peri-implantitis, likely through impaired neutrophil function, delayed wound healing and a chronically elevated inflammatory baseline [18]. Obesity has similarly emerged as an independent risk modifier and its co-occurrence with uncontrolled diabetes further compounds susceptibility [13,18].

*Smoking*

Tobacco use represents one of the most consistently documented risk factors for peri-implant disease across all stages of severity. Smoking alters the submucosal microbiome, suppresses local immune surveillance and impairs the vascular response necessary for proper wound healing around the implant site [19]. These mechanisms collectively accelerate the transition from reversible mucositis to irreversible bone-destructive peri-implantitis. The evidence supporting smoking cessation as a component of implant therapy planning is compelling and clinicians should address this risk factor proactively, ideally before implant placement, rather than managing its consequences after disease has established [20].

**Diagnosis and Classification**

*Clinical and Radiographic Parameters*

Diagnosing peri-implant disease requires the integration of clinical and radiographic findings; no single parameter is sufficient in isolation. Clinically, the hallmark signs include visual inflammation of the peri-implant mucosa, erythema, edema and occasionally hyperemia; accompanied by Bleeding on Probing (BoP) and, in more advanced cases, suppuration [21]. Probing Depth (PD) is recorded at six sites per implant using light, standardized force and while increased PD is informative, it must be interpreted alongside inflammatory indicators rather than used as a standalone diagnostic criterion [17]. BoP remains the most sensitive clinical marker of active mucosal inflammation and its consistent presence across multiple probing sites strengthens the diagnostic case for disease [22].

Radiographic assessment is essential to confirm peri-implantitis and to characterize the extent of bone involvement. Crestal bone levels are evaluated relative to the implant platform and a critical distinction must be made between the physiological bone remodeling that occurs in the first year following implant placement, typically 1 to 2 mm and progressive pathological bone loss

that continues beyond this initial phase [23]. The latter is the defining radiographic hallmark of peri-implantitis. Comparison with baseline radiographs, ideally taken at the time of prosthetic delivery, substantially improves diagnostic accuracy and is considered best practice in implant maintenance protocols [24]. Severity staging based on the extent of bone loss has been proposed in the literature, with early disease defined as loss of less than 25% of implant length, moderate as 25–50% and advanced as exceeding 50% [18].

*The 2017 World Workshop Classification*

A major advancement in standardizing peri-implant diagnosis came with the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, whose updated case definitions were published in 2018 [19]. This framework addressed a longstanding limitation in the field: the absence of universally accepted diagnostic thresholds that could be applied consistently across clinical and research settings. For cases in which baseline radiographs are unavailable, a frequent scenario in referral-based implant management; the consensus criteria establish probing depths of 6 mm or greater, bone levels at least 3 mm apical to the implant platform and the presence of BoP and/or suppuration as the minimum diagnostic requirements for peri-implantitis [25]. The reliability of these 2018 case definitions has since been evaluated in prospective clinical studies, which have supported their utility while identifying areas where further refinement may be warranted, particularly regarding threshold sensitivity in early-stage disease [24,25].

*Classification: Mucositis vs. Peri-Implantitis*

Peri-implant diseases are classified into two distinct entities that differ fundamentally in their tissue involvement, reversibility and clinical management. Peri-implant mucositis is defined as a reversible inflammatory condition confined to the soft tissues surrounding a functioning implant, characterized by BoP with or without suppuration in the absence of radiographic bone loss beyond initial remodeling [20]. It is widely considered the precursor to peri-implantitis and its resolution through targeted biofilm disruption is a primary goal of preventive implant care [25].

Peri-implantitis, by contrast, involves progressive loss of supporting marginal bone in addition to mucosal inflammation, rendering it a non-reversible condition that requires more intensive and often surgical intervention [26]. Following surgical treatment of peri-implantitis, the resulting clinical picture may not conform neatly to existing classification categories, a recognized gap in the literature that has prompted proposals for post-treatment classification schemes to better capture treatment outcomes and guide ongoing management decisions [27].

*Differential Diagnosis*

An accurate diagnosis of peri-implantitis also requires ruling out conditions that can mimic its clinical presentation. Occlusal overload, for instance, may produce radiographic bone loss without the inflammatory mucosal signs characteristic of biofilm-driven disease [16]. Foreign body reactions to residual cement or other submucosal contaminants can produce localized inflammation and probing depth increases that resolve entirely upon removal of the offending agent, without the need for surgical intervention [18,28]. Distinguishing these entities from true peri-implantitis has direct implications for treatment selection and for avoiding unnecessary surgical procedures.

**Non-Surgical Treatment Approaches**

*Overview and Treatment Goals*

Once the diagnosis of peri-implant disease has been established, the treatment sequence should begin with non-surgical therapy as the first line of intervention. The primary objectives at this stage are infection control, reduction of peri-implant inflammation and assessment of tissue response before any decision about surgical escalation is made [29]. Non-surgical treatment also provides a structured opportunity to reinforce patient-specific oral hygiene measures and to identify modifiable local factors that may be contributing to disease progression. Evidence supports that a peri-implant maintenance protocol at six-month intervals can preserve stable peri-implant attachment in the majority of cases when disease has been adequately controlled [30]. If clinical resolution is achieved through non-surgical means, the patient should transition to a structured supportive maintenance program; if significant inflammation or bone loss persists, surgical intervention should be planned accordingly [22,30].

*Mechanical Debridement*

Mechanical debridement remains the foundation of non-surgical peri-implant therapy. This includes subgingival instrumentation of the implant surface to disrupt and remove calculus and biofilm, along with minor soft tissue curettage where indicated. Titanium curettes, ultrasonic scalers fitted with plastic tips and carbon fiber instruments are the preferred tools, as they minimize the risk of surface damage to the implant and abutment [31]. Clinical improvements, including reductions in bleeding on probing and probing depth, have been consistently reported within relatively short follow-up periods, establishing mechanical debridement as an indispensable component of any non-surgical protocol [32].

*Adjunctive Antibiotic Therapy*

The role of antibiotics in non-surgical peri-implantitis management remains an area of active investigation. Current evidence indicates that local or systemic antibiotic adjuncts, most commonly metronidazole, amoxicillin or their combination; do not consistently confer additional long-term clinical benefit beyond mechanical debridement alone [33]. A randomized controlled trial comparing non-surgical treatment with and without systemic antibiotics found no statistically significant difference in outcomes at 12 months, reinforcing the need for restrained antibiotic prescribing in this context [24,25]. Adjunctive antibiotics may be considered in cases of acute infection or when systemic involvement is suspected, but this decision should always account for the patient's medical history, concurrent medications and the broader goal of antimicrobial stewardship [34].

*Air-Polishing Therapy*

Air-polishing with erythritol powder represents a minimally invasive adjunct that has demonstrated effectiveness in reducing biofilm burden at peri-implant sites. It can be used alone or in combination with saline irrigation and clinical studies have reported favorable outcomes for both peri-implant mucositis and early-stage peri-implantitis [26]. However, erythritol air-polishing is best understood as a complementary modality rather than a definitive treatment and its long-term efficacy is significantly enhanced when integrated into a comprehensive debridement protocol [35].

*Laser Therapy*

Laser-based systems including Er,Cr:YSGG, Nd:YAG and diode lasers, have been investigated as non-surgical adjuncts for implant surface decontamination and peri-implant inflammation control. Clinical and radiographic improvements, including reductions in BoP and PD, have been reported across multiple study designs [28]. A systematic review and meta-analysis of non-surgical laser therapy concluded that meaningful clinical gains are achievable, though outcomes are consistently stronger when laser treatment is combined with mechanical debridement rather than applied in isolation [29,36]. At present, no single laser system has demonstrated clear superiority and the choice of device should be guided by available evidence, clinician training and patient-specific considerations [37].

*Chemical and Antimicrobial Adjuncts*

Several chemical agents have been evaluated as subgingival adjuncts to mechanical debridement. Chlorhexidine at concentrations of 0.12% to 2% provides broad-spectrum antimicrobial activity and is among the most widely used agents in clinical practice [38]. Hydrogen peroxide (3%) and citric acid (4%–40%) have demonstrated efficacy in endotoxin removal and implant surface decontamination, while sodium hypochlorite (1.5%) has been associated with favorable outcomes in terms of defect depth reduction in some preclinical models [39]. Local antimicrobial delivery systems, including doxycycline gel and minocycline microspheres, offer targeted pharmacological activity within deeper infected pockets and may serve as useful adjuncts when systemic antibiotic use is not warranted [31].

*Treatment Outcomes*

Overall, non-surgical therapy is most effective in the management of peri-implant mucositis, where resolution of inflammation and restoration of tissue health are achievable in the majority of patients through mechanical biofilm disruption and adjunctive measures [40]. In cases of established peri-implantitis; particularly where moderate to advanced bone loss is present, non-surgical treatment can meaningfully reduce inflammatory parameters and may arrest disease progression in the short term, but it rarely achieves complete resolution of the osseous defect [22,23]. A 12-month prospective study of non-surgical peri-implantitis outcomes confirmed that while clinical improvements were measurable, a substantial proportion of cases required surgical re-evaluation within the follow-up period [41]. These findings reinforce the importance of realistic treatment expectations and the value of structured re-evaluation checkpoints within the overall management plan.

**Surgical Treatment Approaches**

*Rationale and Treatment Goals*

When non-surgical therapy fails to achieve adequate infection control or when the extent of bone loss precludes resolution through conservative means, surgical intervention becomes necessary. The overarching goals of surgical peri-implantitis treatment are to eradicate tissue inflammation, arrest disease progression, regenerate lost supporting bone where defect morphology permits, and, where possible, restore functional osseointegration [42]. Surgical access allows direct visualization of the implant surface and underlying defect, enabling more thorough debridement than is achievable through a closed approach.

*Implant Surface Decontamination*

Effective biofilm removal from the contaminated implant surface is the most critical step in any surgical protocol and the various decontamination strategies are broadly classified as chemical or physical. Within the physical category, mechanical methods; including titanium brushes, ultrasonic instruments and sandblasting systems using abrasive particles, aim to eliminate microbial toxins and modify surface texture to reduce susceptibility to future bacterial adhesion [43]. Antimicrobial Photodynamic Therapy (aPDT) represents an alternative physical approach in which a photosensitive substance binds to biofilm bacteria and, upon laser irradiation, generates cytotoxic singlet oxygen capable of disrupting bacterial cell membranes [33]. Chemical decontamination agents, including chlorhexidine, citric acid and hydrogen peroxide, are frequently used as adjuncts to mechanical methods, though no single decontamination protocol has yet demonstrated clear superiority in the literature [44].

*Access Flap Debridement and Resective Surgery*

The surgical procedure typically begins with the elevation of a full-thickness mucoperiosteal flap to expose the contaminated implant surface and the extent of the osseous defect. Access Flap Debridement (AFD) alone involves thorough removal of granulation tissue and surface decontamination and is appropriate when defect morphology does not favor a regenerative approach [44]. When combined with resective techniques; including osseous recontouring and apically repositioned flap placement, the goal shifts toward creating a shallow, maintainable peri-implant environment with a positive osseous architecture [35]. For implants located in non-aesthetic zones, this resective approach represents the treatment of choice, as pocket depth reduction and improved cleansability are achievable and predictable outcomes. More than half of cases treated with resective surgery combined with systemic antimicrobial therapy have been reported to achieve successful outcomes defined as disease resolution [45].

*Implantoplasty*

Implantoplasty; the mechanical smoothing of the exposed implant surface using rotary instruments, has been evaluated both as a treatment modality for established peri-implantitis and as an adjunct to resective surgery. By reducing implant surface roughness, it aims to decrease bacterial adhesion and facilitate long-term maintenance. Evidence supports its microbiological efficacy in biofilm elimination; however, this benefit must be weighed against a meaningful mechanical cost [36]. Multiple studies have demonstrated that the implantoplasty process reduces the load-bearing cross-sectional thickness of the implant body and can introduce surface micro-cracks that predispose the implant to fatigue fracture under functional loading. Additionally, the release of titanium particles during the procedure raises concerns about local and systemic biological effects that remain incompletely understood [46]. Implantoplasty should therefore be reserved for specific clinical scenarios; particularly exposed implant threads in non-aesthetic regions and applied with an understanding of its structural implications.

*Regenerative Surgery*

Reconstructive surgical approaches aim to restore lost peri-implant bone using grafting materials and, where indicated, barrier membranes. A range of bone replacement materials has been applied in this context, including autogenous bone, alloplastic fillers, xenografts and titanium granules, used with or without resorbable and non-resorbable membranes [47]. The selection of grafting material and membrane type is guided primarily by the morphology of the osseous defect; circumferential intrabony defects with three or more remaining walls are considered the most favorable configuration for regenerative therapy [48].

Short-term comparative data suggest that regenerative surgery with bone grafting offers advantages over resective surgery in terms of radiographic bone fill and probing depth reduction [37]. However, a meta-analysis evaluating long-term clinical outcomes found no statistically significant difference between regenerative and resective approaches in terms of implant survival or overall prognosis [49]. This finding does not diminish the value of regeneration in appropriately selected cases, but it does

underscore the importance of realistic patient expectations and careful defect morphology assessment before committing to a more complex surgical approach [47].

*Implant Removal*

When both non-surgical and surgical therapies fail to achieve disease resolution, implant removal becomes the necessary endpoint. This decision is not a treatment failure in isolation; rather, it reflects the limits of biological recovery in the context of advanced bone loss, unfavorable implant positioning or compromised host response [50]. Early recognition of cases unlikely to respond to salvage therapy, combined with timely removal and appropriate site rehabilitation, often produces better long-term prosthodontic outcomes than prolonged attempts to maintain a failing implant [44]. Based on the current understanding of peri-implant diseases, the following clinical recommendations are proposed [30,32,33,46] (Fig. 1).

| Peri-implant mucositis | Peri-implantitis | Failed implant |
|---|---|---|
| **Diagnosis** | **Diagnosis** | **Diagnosis** |
| Bleeding and/or suppuration upon gentle probing, without bone loss beyond changes attributable to initial bone remodeling. | Bleeding and/or suppuration upon gentle probing, increased probing depth, and radiographic bone loss extending beyond changes attributable to initial bone remodeling. | Implant presenting with severe peri-implant bone loss (greater than 50% of fixture length), loss of osseointegration, or fixture fracture. |
| **Treatment** | **Treatment** | **Treatment** |
| • Non-surgical treatment and disease prevention<br>◦ Sufficient quality and quantity of surrounding mucosa and bone<br>◦ Properly positioned implant fixture to ensure adequate Supracrestal mucosal thickness<br>◦ Appropriately designed prosthesis with cleansable contours<br>◦ Removal of excess cement in cement-retained restorations | • Non-surgical treatment<br>◦ Removal of prosthesis prior to therapy when access is limited<br>◦ Adjunctive local and/or systemic antibiotics when clinically indicated<br>• Re-evaluation at 3 months following non-surgical therapy<br>• Surgical treatment<br>◦ Access surgery with implant surface decontamination<br>◦ Resective or regenerative surgery based on defect morphology<br>• Supportive peri-implant care tailored to individual patient risk profile (IDRA) | • Removal of the affected implant<br>• Identification and control of modifiable systemic and local risk factors contributing to failure<br>• Reconstruction of affected hard and soft tissues prior to rehabilitation<br>• Reimplantation only after etiological factors have been corrected |

**Figure 1:** Clinical recommendations for the management of peri-implant diseases based on diagnosis category. IDRA: implant disease risk assessment. [30,32,33,46].

## Conclusion

With the increasing ubiquity of dental implants, peri-implant conditions are becoming a growing challenge in contemporary dentistry. The definitions established during the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, encompassing peri-implant health, mucositis and peri-implantitis; provide a much-needed common language for both researchers and clinicians. This framework is not merely a formality; it represents a meaningful step toward improved diagnosis and more appropriate patient care.

Understanding who is at risk remains equally important. Smoking, diabetes mellitus, a personal history of periodontitis, inadequate maintenance and excess cement in cement-retained restorations are not isolated variables; they reflect the complex, multifactorial nature of peri-implantitis and remind us that each patient requires an individualized approach from the very beginning of implant therapy.

In spite of advances in the recognition of the clinical and radiographic characteristics of peri-implant disease, a reliable prognostic system in this area is still lacking and this remains an urgent gap requiring further research. Non-surgical management provides sufficient resolution in cases of peri-implant mucositis, but treatment does not always succeed and recurrence and incomplete resolution remain a reality even in treated peri-implantitis. Surgical approaches including open flap debridement, apically repositioned flap and guided bone regeneration may offer clinical benefit, but the absence of standardized, evidence-based protocols prevents their consistent application in routine practice. Moving forward, well-designed randomized clinical trials with long-term follow-up are needed to refine treatment algorithms, strengthen prognostic tools and ultimately improve outcomes for patients living with peri-implant disease.

**Conflict of Interest**

The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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**Data Availability Statement**

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

**Ethical Statement**

The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore was exempt.

**Informed Consent Statement**

Not Applicable.

**Authors' Contributions**

All authors contributed equally to this paper.

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