Peri-Implantitis Management: Current Therapies and Future Perspectives

Abstract

Peri-implantitis is a biofilm-associated inflammatory disease characterized by progressive peri-implant bone loss and represents a growing clinical concern as dental implant therapy becomes increasingly prevalent. Despite high implant survival rates, peri-implantitis affects a substantial proportion of implant patients and is associated with complex interactions between microbial biofilms, host immune responses, implant surface characteristics, and patient-related risk factors. This review provides a comprehensive overview of the etiology, diagnosis, and contemporary management strategies for peri-implantitis. Conventional non-surgical therapies, including mechanical debridement and adjunctive antimicrobial approaches, remain first-line interventions but demonstrate limited effectiveness in moderate to advanced disease due to restricted access to contaminated implant surfaces. Surgical therapies, encompassing resective and reconstructive approaches, offer improved infection control and pocket reduction, although long-term predictability remains variable. Regenerative strategies, particularly guided bone regeneration combined with bone grafts and biologic modifiers such as recombinant human platelet-derived growth factor-BB and enamel matrix derivative, show promise in contained defect morphologies but remain technique-sensitive. Emerging adjunctive strategies, including advanced biomaterials, implant surface engineering, probiotics, and host-modulation therapies, aim to enhance antimicrobial efficacy, modulate inflammation, and promote peri-implant tissue regeneration. Current evidence highlights the absence of a universally predictable treatment protocol and underscores the importance of early diagnosis, defect-specific therapy, and structured supportive maintenance. Future advances in biologically responsive implant surfaces, host-modulatory interventions, and personalized treatment planning are expected to improve long-term peri-implant health and implant survival.

Keywords

Peri-implantitis; Peri-implant disease; Implant surface decontamination; Non-surgical therapy; Surgical therapy; Guided bone regeneration; Bone grafts; rhPDGF-BB; Enamel matrix derivative; Biomaterials; Implant surface engineering; Host modulation; Probiotics; Supportive periodontal therapy

Introduction and Disease Overview

Dental implants have achieved remarkable success rates; however, the increasing prevalence of peri-implant complications presents a significant clinical challenge.[1] Peri-implantitis affects approximately 22% of patients within a decade of implant placement, with prevalence rates continuing to rise as implant procedures increase.[2]

Inflammatory diseases around dental implants comprise two distinct entities with differing prognoses.[3] Peri-implant disease concepts and classifications have been revisited and refined over time to improve diagnostic clarity and clinical decision-making.[8] Peri-implant mucositis is a

reversible inflammatory condition limited to peri-implant soft tissues, whereas peri-implantitis is an irreversible disease characterized by inflammation with progressive loss of supporting bone. Clinically, peri-implantitis presents with bleeding or suppuration on probing, increased probing depths, and radiographic evidence of bone loss. Diagnosis is based on clinical and radiographic assessment, with emerging protocols incorporating biomarker analysis and advanced three-dimensional imaging modalities.[2]

The pathogenesis of peri-implantitis is multifactorial, involving complex interactions between microbial biofilms, host immune responses, and patient-related risk factors.[4] Established risk determinants include poor plaque control, smoking, prior periodontal disease, implant surface characteristics, residual cement, and systemic conditions such as diabetes mellitus.[5] Evidence consistently indicates increased disease susceptibility among patients with a history of periodontitis, inadequate biofilm control, and limited adherence to supportive maintenance care.

Treatment approaches typically follow a sequential protocol beginning with non-surgical interventions. Non-surgical therapy includes mechanical debridement, antiseptic therapy, and antibiotics; however, mechanical debridement alone may fail to eliminate causative bacteria and should be combined with adjunctive treatment modalities.[5] Despite these measures, non-surgical therapy demonstrates limited efficacy, particularly in moderate to severe cases, due to restricted access to contaminated implant surfaces.[6]

When non-surgical approaches fail, surgical intervention becomes necessary, with treatment strategies broadly categorized as resective therapy, reconstructive therapy, or combined approaches.[7] Surgical interventions include resective procedures for pocket elimination and regenerative techniques aimed at restoring lost bone, although current evidence suggests that regenerative approaches remain unpredictable.[5]

Future perspectives in peri-implantitis management focus on emerging technologies and innovative therapeutic strategies designed to overcome current treatment limitations. Promising developments include antibacterial implant surface coatings, photodynamic therapy, and artificial intelligence-assisted diagnostic systems with the potential to enhance clinical outcomes.[2] Advanced diagnostic modalities incorporating matrix metalloproteinase-8 biomarker assays, microbial polymerase chain reaction analysis, and sophisticated imaging techniques are expected to improve early disease detection and treatment planning.[2] Ongoing research explores novel surface decontamination methods, host-modulation strategies, and regenerative approaches using biologics and growth factors to achieve more predictable outcomes.[7]

This review aims to synthesize current evidence on peri-implantitis management strategies, evaluate the effectiveness of contemporary treatment modalities, identify gaps in existing therapeutic approaches, and discuss future directions to support evidence-based clinical decision-making. Long-term implant success depends on structured maintenance protocols, including three-month recall visits, professional biofilm control, and radiographic surveillance, while future advances may revolutionize both preventive and therapeutic strategies.[2]

Conventional Non-Surgical and Surgical Therapies for Peri-Implantitis

The management of peri-implantitis focuses on disrupting microbial biofilm on implant surfaces while achieving shallow peri-implant pockets (≤5 mm) that can be effectively maintained during

long-term care. Depending on the severity and extent of peri-implant tissue destruction, treatment may be undertaken using either non-surgical or surgical approaches.[10,11,12]

Conventional Non-Surgical Therapy:

Conventional non-surgical therapy aims to control peri-implant infection by removing subgingival biofilm through mechanical debridement of peri-implant pockets, in combination with appropriate oral hygiene measures to reduce inflammation. Implant surface decontamination during non-surgical therapy typically involves a combination of mechanical, chemical, and adjunctive modalities.[10]

- **Mechanical debridement:** Using titanium instruments, ultrasonic scalers with non-metallic tips, or air-abrasive systems employing glycine or erythritol powders.
- **Chemical debridement:** Using antimicrobial agents such as chlorhexidine or hydrogen peroxide.
- **Adjunctive therapies:** Laser treatment or antimicrobial photodynamic therapy to enhance surface decontamination, particularly in the presence of complex implant surface topography.[10]

Clinical studies have demonstrated improvements in probing depth reduction, bleeding on probing, and implant surface cleanliness when antimicrobial photodynamic therapy combined with hydrogen peroxide (OHLLT) is used as an adjunct to conventional non-surgical treatment.[12,13] This approach provides an antimicrobial effect while preserving the integrity of the implant surface.[12]Laser therapy may further support mechanical debridement by aiding in the decontamination of both implant surfaces and inflamed peri-implant tissues.[14]

Although adjunctive systemic or local antibiotics may be used to reduce pathogenic microorganisms; however, their effect on clinical parameters such as bleeding on probing and peri-implant pocket depths remains limited in patients with deeper peri-implant pockets. The major limitation of non-surgical therapy is restricted access to the apical portion of the peri-implant pocket, which can hinder complete biofilm disruption. Consequently, implants presenting with deep pockets or complex surface characteristics often require surgical intervention to achieve effective decontamination.[9,10] Current EFP clinical guidelines emphasize the importance of initiating treatment with non-surgical interventions to improve peri-implant soft tissue health before surgical options are considered.[10]

Multiple investigations have assessed a wide range of mechanical, chemical, and adjunctive decontamination strategies, reporting variable levels of success in achieving complete biofilm removal, and to date, no single gold-standard method has been established.[16] Nevertheless, favorable outcomes following non-regenerative surgical procedures have been reported, including reductions in probing depth, absence of bleeding on probing or suppuration, and stability of peri-implant bone levels in a substantial proportion of implants and patients receiving regular supportive periodontal therapy.[13]

Early identification of peri-implantitis significantly influences the success of non-surgical therapy. Chang et al. reported higher success rates of non-surgical treatment when peri-implantitis is detected at an early stage. Similarly, Schwarz et al. suggested that non-surgical therapy is more predictable when marginal bone loss is limited to less than 2mm, whereas surgical approaches—such as access flap surgery or apically positioned flaps—are more appropriate when bone loss exceeds this threshold. Timely detection may therefore reduce the need for more invasive surgical interventions.[12]

Surgical Therapy:

Surgical therapy allows direct access to the base of the peri-implant pocket, typically through open flap debridement or access flap procedures. Alongside mechanical debridement, the use of laser treatment as an adjunct provides several benefits, including antibacterial and anti-inflammatory effects, reduction of postoperative pain and discomfort, and acceleration of wound healing through stimulation of fibroblasts.[11]

Access Flap Debridement with Resective Procedures:

- **Implantoplasty:** Refers to the mechanical reshaping of exposed implant parts through removal of threads and surface roughness to reduce plaque retention and lowers the risk of reinfection.[13]
- **Osteoplasty/Osteotomy:** Involves the removal or recontouring of peri-implant bone to facilitate access for plaque control and reduce biofilm accumulation.[17]
- **Apically Repositioned Flap:** Often performed in conjunction with osteoplasty/osteotomy to reduce peri-implant pocket depths, improving long-term cleansability and plaque control.[17]

Systematic reviews indicate that conventional non-regenerative surgical procedures can effectively reduce peri-implant inflammation in the short term; however, long-term predictability remains variable. Implantoplasty performed as part of non-regenerative surgical therapy has been associated with significant reduction in bleeding on probing and probing depth, with improvements in clinical and radiographic outcomes maintained for up to three years compared with mechanical debridement alone. In contrast, the adjunctive use of systemic antibiotics, chemical agents, or diode laser therapy has not demonstrated consistent long-term clinical or radiographic benefits.[17]

Long-Term Outcomes:

Long-term follow-up studies have demonstrated favorable clinical and radiographic outcomes following access flap debridement combined with osseous recontouring, with reported stability extending up to 11 years. Treatment outcomes appear to be influenced by implant surface characteristics, with turned (smooth) surfaces demonstrating more favorable responses compared with roughened surfaces. Evidence suggests that implantoplasty does not exhibit clear superiority over glycine air-polishing. Moreover, implantoplasty poses concerns regarding

residual titanium particles in peri-implant tissues, and caution is advised when performing this procedure on narrow-diameter implants.[10] However, adjunctive laser therapy helps to remove residual titanium particles and accelerates tissue healing.[14]

Moreover, multimodal treatment approaches that combine implantoplasty, apically repositioned flaps, free gingival grafts, and laser-assisted therapy have been associated with favorable microbiological changes at peri-implant sites, characterized by reductions in pathogenic genera such as Porphyromonas, Treponema, and Fusobacterium, along with an increased levels of Streptococcus.[14]

Supportive Measures for Long-Term Prognosis:

Achieving long-term peri-implant stability requires ongoing supportive measures following both surgical and non-surgical therapy. Insufficient width of keratinized mucosa may compromise effective plaque control, as brushing over non-keratinized, mobile tissue can cause discomfort and limit oral hygiene practices. Additionally, a lack of adequate keratinized mucosa provides a weaker soft-tissue barrier against bacterial penetration, leading to increased plaque accumulation, inflammation, and subsequent peri-implant tissue breakdown and bone loss. Maintaining an adequate width of at least 2mm is therefore considered beneficial for peri-implant health.[14]

Soft-tissue augmentation procedures, including free gingival grafts, may be indicated to enhance peri-implant tissue stability and facilitate plaque control.[14] Following active treatment, patient education and supportive periodontal therapy play a vital role in maintaining peri-implant health by controlling biofilm accumulation through regular periodontal maintenance visits. In selected cases, adjunctive interventions, such as soft-tissue augmentation may further contribute to the long-term control of peri-implant inflammation.[14,15]

Regenerative management of peri-implantitis

Advances in bone grafting materials, barrier membranes and biomaterials have expanded the options available for managing complex osseous defects and restoring lost tissues to their original anatomy. The success of regenerative therapy for peri-implantitis depends on the morphology of defect, with four-walled intrabony defects ≥ 3mm demonstrating favourable prognosis.[18]

Guided bone regeneration:

Guided bone regeneration (GBR) is a surgical technique used to stimulate new bone formation at sites of intrabony defects with the help of bone grafts and barrier membranes. Barrier membranes prevent epithelial downgrowth into the defect site and provide stability; they can be resorbable or non resorbable.

Bone grafts:

Bone grafts play a key role in periodontal regeneration, acting as a structural framework.They can be categorized into the following types:

- **Autogenous bone graft**: Autogenous bone graft remains the gold standard for bone regeneration procedures, as it is osteogenic, osteoconductive and osteoinductive.
- **Allografts**: Allografts such as mineralized dehydrated bone allograft (MDBA) can be utilized in guided bone regeneration for peri-implantitis management.[19]
- **Xenografts**: Bovine derived xenografts are osteoconductive and are widely used for the management of intrabony defects in peri-implantitis.[20]
- **Alloplasts**: Synthetic bone grafts composed of calcium phosphate or bioactive glass are primarily osteoconductive, lack osteoinductive properties and are used less frequently in guided bone regeneration.[21]

Growth factor modulation:

Growth factors are proteins that can stimulate mesenchymal and osteoblast proliferation at implant sites by acting as signaling molecules, particularly platelet derived growth factor (PDGF), bone morphogenic proteins (BMPs), transforming growth factor beta (TGF-β), insulin like growth factor (IGF) and vascular endothelial growth factor (VEGF). Although current results look promising, further research is required to address potential long term outcomes and safety.[22]

- **Recombinant human platelet derived growth factor(rhPDGF-BB):**

Recombinant human platelet derived growth factor (rhPDGF-BB) is a synthetic form of platelet derived growth factor (PDGF). PDGF is a widely used growth factor due to its ability to stimulate angiogenesis, chemotaxis and mitogenesis. It is delivered using bone grafts or synthetic matrices which help localize its activity while providing support and expediting regeneration.[23]

- **Growth factor enhanced matrix (GEM 21S):**

Growth factor enhanced matrix(GEM 21S) is a bone grafting material consisting of FDA approved recombinant human platelet derived growth factor (rhPDGF-BB) and osteoconductive β tricalcium phosphate scaffold. GEM 21S is utilized in the treatment of peri-implantitis as it acts as an osteoconductive matrix promoting angiogenesis and osteogenesis thereby increasing the survival rate of implants.[23]

Enamel matrix derivative (EMD):

Enamel matrix derivative (EMD), an amelogenin-rich biologic material derived from porcine enamel matrix plays a key role in bone regeneration. It is delivered using Propylene glycol alginate (PGA) aqueous solution which enhances the precipitation of EMD. EMD restricts epithelial downgrowth and promotes regeneration by growth of mesenchymal cells and angiogenesis. EMD also possess anti-inflammatory and anti-bacterial properties. EMD is typically used in combination with bone grafts to prevent rapid degradation and flap collapse due to its lack of structural rigidity.[24,25]

Adjunctive and Emerging Strategies in Peri-Implantitis Management

Conventional mechanical and surgical approaches remain the cornerstone of peri-implantitis management; however, their effectiveness is often limited by complex implant surface characteristics, persistent biofilm formation, and a dysregulated host inflammatory response. As a result, adjunctive and emerging therapies have gained increasing attention for their potential to enhance treatment outcomes by targeting microbial colonization, modulating host immune responses, and promoting peri-implant tissue regeneration. Advances in biomaterials, surface engineering, and biological modulation represent a shift toward more comprehensive and biologically driven treatment strategies. These emerging approaches are primarily intended to complement established therapies, improve long-term peri-implant stability, and reduce disease recurrence rather than replace conventional interventions.[26,27]

1. Biomaterials and Implant Surface Engineering:

Biomaterials and implant surface engineering play a pivotal role in the prevention and management of peri-implantitis by targeting the earliest pathogenic event—bacterial adhesion—while supporting peri-implant bone integration. Since implant surface characteristics directly influence microbial colonization and host tissue responses, surface modification strategies have emerged as both preventive and therapeutic adjuncts.[28]

Anti-adhesive surface modifications aim to inhibit bacterial attachment through physicochemical alterations rather than bactericidal mechanisms. Hydrophilic polymer grafting, nanoscale topographical patterning, and titanium nitride (TiN) coatings have demonstrated reduced bacterial adhesion and biofilm formation without inducing antimicrobial resistance. Clinical and in vivo studies confirm the efficacy of TiN-coated surfaces in limiting oral bacterial colonization. However, excessive anti-fouling properties may also impair osteoblast adhesion, necessitating the incorporation of bioactive molecules to restore osteogenic potential.[29]

Bactericidal surface modifications provide active antimicrobial effects through contact-dependent or release-based mechanisms. Nanopatterned surfaces, antimicrobial peptides, graphene-based materials, and metal or metal oxide nanoparticles disrupt bacterial membranes or generate reactive oxygen species, effectively preventing biofilm maturation. Several in vivo studies demonstrate that these surfaces maintain antibacterial activity while supporting osseointegration. Controlled-release coatings incorporating antimicrobial agents or ions further enhance antibacterial efficacy, although challenges remain regarding sustained release and potential cytotoxicity.[29]

Intrinsic antibacterial alloys, particularly titanium–copper (Ti–Cu) systems, offer drug-independent antimicrobial activity through ion release and contact sterilization. These alloys reduce biofilm stability, suppress bacterial virulence gene expression, and resist infection-induced bone resorption while promoting osseointegration. Externally triggered strategies, such as near-infrared light–activated titanium oxide surfaces, provide on-demand antibacterial effects and have shown promise in reducing peri-implant inflammation without inducing resistance.[29]

Despite the widespread use of moderately rough titanium surfaces to enhance osseointegration, increased surface roughness may predispose implants to microbial accumulation. Contemporary biomaterial strategies therefore aim to balance antibacterial efficacy with biological safety and osteogenic capacity, representing a shift toward biologically responsive implant systems for peri-implantitis prevention and management.

2. Host Modulation and Biological Adjuncts:

Peri-implantitis is not solely a biofilm-induced condition but also the result of an exaggerated host immune response leading to peri-implant soft tissue inflammation and progressive bone loss. Host modulation strategies aim to control this dysregulated inflammatory response and preserve peri-implant tissues.[30]

Biological adjuncts, including probiotics, growth factors, and immunomodulatory agents, have been explored to regulate peri-implant inflammation.[31]

Probiotics function by competitively inhibiting peri-implant pathogens, modifying local microbial ecology, and downregulating proinflammatory cytokines and matrix metalloproteinases. Clinical studies suggest that probiotics may reduce bleeding on probing and peri-implant mucosal inflammation when used adjunctively with nonsurgical therapy, particularly in peri-implant mucositis. However, evidence supporting their effectiveness in established peri-implantitis remains limited and inconsistent.[30]

Growth factor–based therapies, such as recombinant platelet-derived growth factor and enamel matrix derivatives, contribute indirectly to host modulation by enhancing wound healing, angiogenesis, and bone regeneration. These agents may improve peri-implant tissue stability when used in regenerative surgical protocols, although their direct anti-inflammatory effects are secondary.[31]

Emerging host immune-modulatory approaches include cytokine regulation, oxidative stress modulation, and immune pathway targeting. While preclinical data are promising, clinical translation remains limited due to variability in delivery systems and lack of long-term outcome data.[31]

From a clinical perspective, host modulation should be considered an adjunctive strategy integrated with mechanical debridement and surgical therapy rather than a standalone treatment. Future research should focus on implant-specific delivery systems, establishment of standardized treatment protocols, and evaluation of long-term effects on peri-implant bone preservation and implant survival.

Discussion

The management of peri-implantitis remains a significant clinical challenge due to its multifactorial etiology, complex microbial profile, and limited regenerative capacity around implant surfaces. Despite improved implant designs and preventive strategies, peri-implantitis

continues to demonstrate unpredictable treatment outcomes, particularly in advanced cases with extensive bone loss and soft tissue inflammation.[32,33]

Non-surgical therapy is widely regarded as a first-line approach, especially in early disease stages; however, its effectiveness in established peri-implantitis is limited. Systematic reviews have consistently reported modest improvements in clinical parameters such as bleeding on probing and probing depth, with negligible radiographic bone gain.[34,35] The inability to adequately decontaminate rough implant surfaces and deep peri-implant defects remains a critical limitation, often necessitating surgical intervention.[35]

Surgical access therapy allows direct visualization and thorough debridement of contaminated implant surfaces, resulting in improved infection control compared to non-surgical approaches.[36] Resective surgical techniques, including apically positioned flaps and implantoplasty, aim to reduce pocket depths and facilitate plaque control; however, they primarily achieve disease stabilization rather than true regeneration and may compromise esthetic outcomes.[37]Additionally, concerns regarding titanium particle release during implantoplasty and its potential biological effects warrant further investigation.[38]

Regenerative surgical approaches have gained increasing attention due to their potential to restore lost peri-implant bone and improve long-term implant prognosis. Guided bone regeneration (GBR), when combined with particulate bone grafts, has demonstrated favorable outcomes in contained and semi-contained peri-implant defects.[39] Xenografts and slowly resorbing biomaterials are commonly preferred due to their superior space-maintaining properties, although clinical outcomes remain highly dependent on defect morphology and surgical technique.[40]

The incorporation of biologically active agents has further expanded regenerative possibilities. Recombinant human platelet-derived growth factor-BB (rhPDGF-BB), delivered via GEM21S, promotes angiogenesis, chemotaxis, and proliferation of osteogenic cells. Recent clinical studies suggest that rhPDGF-BB, when used adjunctively with bone grafts, may enhance radiographic bone fill and clinical attachment levels in peri-implant defects, although long-term, implant-specific randomized controlled trials remain limited.[41,42]

Enamel matrix derivative (EMD) has also been proposed as an adjunctive regenerative agent due to its anti-inflammatory properties and ability to enhance soft tissue healing. While EMD alone does not appear to induce significant peri-implant bone regeneration, its use in combination with surgical debridement and grafting has been associated with improved clinical outcomes, including reduced probing depths and inflammation.[43,44]

Emerging strategies targeting biomaterials and implant surface engineering seek to overcome the challenge of re-osseointegration. Novel surface modifications, antibacterial coatings, and bioactive materials are under investigation to promote favorable host–implant interactions while limiting bacterial adhesion.[45] In parallel, host modulation therapies—such as photobiomodulation, probiotics, and local delivery of anti-inflammatory agents—aim to control the host inflammatory response and improve treatment stability.[46]

Despite these advances, current evidence underscores the absence of a universally predictable treatment protocol for peri-implantitis. Variability in diagnostic criteria, defect morphology, and outcome measures continues to limit comparability across studies.[32] Future research should focus on well-designed randomized controlled trials with long-term follow-up and standardized reporting. Ultimately, a personalized, risk-based treatment approach integrating surgical, regenerative, biological, and maintenance strategies is likely to offer the greatest potential for long-term peri-implant health.

Abbreviations

GBR-Guided bone regeneration
MDBA-Mineralized dehydrated bone allograft
PDGF-Platelet derived growth factor
BMP-Bone morphogenic protein
TGF-β-Transforming growth factor beta
IGF-Insulin like growth factor
VEGF-Vascular endothelial growth factor
rhPDGF-BB-Recombinant human platelet derived growth factor-BB
GEM 21S-Growth factor enhanced matrix
EMD-Enamel matrix derivative
PGA- Propylene glycol alginate

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