Peri-implant diseases: a narrative review of etiology, diagnosis, and contemporary management strategies

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Abstract

The advent of dental implants in the 20th century revolutionized restorative dentistry, providing patients with a practical and aesthetically pleasing solution for tooth loss. However, this innovation has been overshadowed by the rise of peri-implant diseases, such as peri-implant mucositis and peri-implantitis, which compromise the longevity of implants and present significant challenges for clinicians. These conditions impact millions globally, with up to 65% of patients having peri-implant mucositis and some developing peri-implantitis. The economic impact is substantial, with treatment costs in the U.S. alone exceeding $3,000 per case. This review explores the historical development of peri-implant diseases, their multifactorial etiology, advancements in diagnostics, and current and emerging treatment strategies. By integrating insights from microbiology, immunology, and biomechanics, the review aims to provide a comprehensive framework for the prevention, early detection, and personalized management of these complex conditions.

Keywords: peri-implantitis, dental implant, inflammation, periodontal probing, decontamination, lasers, narrative review, mucositis.

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Introduction

The introduction of dental implants in the 20th century marked a paradigm shift in restorative dentistry, offering patients a durable and aesthetically pleasing solution for tooth loss. However, this innovation has been shadowed by the emergence of peri-implant diseases—inflammatory conditions that threaten the longevity of implants and challenge clinicians worldwide. Peri-implant mucositis, characterized by reversible soft tissue inflammation, and peri-implantitis, defined by progressive bone loss, now afflict millions globally(1–8). Recent epidemiological studies estimate that 19–65% of patients develop peri-implant mucositis, while 1–47% progress to peri-implantitis, depending on risk factors such as smoking, diabetes, and oral hygiene practices (9–19). The economic burden is staggering: in the United States alone, treatment costs for advanced peri-implantitis exceed $3,000 per case, not accounting for indirect costs like lost productivity or psychological distress (20–24). This narrative review traces the historical evolution of peri-implant diseases, unravels their multifactorial etiology, evaluates diagnostic advancements, and critically appraises contemporary and emerging therapies (25–29). This review aims to provide a comprehensive roadmap for prevention, early intervention, and personalized management (30–41) by integrating insights from microbiology, biomechanics, and clinical trials.

Historical Evolution

The story of peri-implant diseases is inextricably linked to the rise of implant dentistry (32,42–55). In the 1960s, Swedish orthopedic surgeon Per-Ingvar Brånemark serendipitously discovered osseointegration—the fusion of titanium with living bone—while studying blood flow in rabbit femurs (22,56–65). This discovery laid the foundation for modern implantology, and by the 1980s, dental implants were hailed as a revolutionary solution for edentulism (22,66–71). Early enthusiasm, however, was tempered by observations of inflammatory lesions around implants, which puzzled clinicians accustomed to the biology of natural teeth (72–79).

In 1987, Mombelli and colleagues published a scientific paper describing “peri-implantitis” in patients with bleeding, suppuration, and radiographic bone loss (80–92). Their work drew parallels to periodontitis, attributing the condition to microbial plaque accumulation (31,93–96). However, histopathological studies soon revealed critical differences (72,97–108). Unlike natural teeth, implants lack a periodontal ligament, which usually acts as a shock absorber and bacterial barrier (109–112). Instead, a fragile mucosal seal forms around the implant, creating a vulnerable interface for bacterial infiltration (113–116). Additionally, peri-implant lesions exhibit , a distinct immune response dominated by neutrophils’ rapid-response immune cells that release proteolytic enzymes rather than the lymphocyte-driven inflammation seen in periodontitis (117–121). This neutrophil dominance explains the aggressive bone destruction observed in some patients, even those with meticulous oral hygiene (122–132).

The 1990s and early 2000s were marked by diagnostic chaos (28,133–136). Clinicians debated whether minor bone loss (1–2 mm) represented pathology or normal remodeling, while researchers struggled to standardize criteria (137–142). A pivotal moment came in 2017 when the World Workshop on Periodontology established clear definitions: peri-implant mucositis was defined by bleeding on probing (BoP) without bone loss, while peri-implantitis required BoP, probing depths ≥6 mm, and radiographic bone loss ≥3 mm (143–148). These criteria unified the field and catalyzed epidemiological studies (149–154). A 2020 meta-analysis found that 22% of patients developed peri-implantitis within five years of implant placement, with smokers and diabetics at the highest risk (155–161) (Figure 1).

Etiology: A Symphony of Microbial, Host, and Mechanical Factors

Peri-implant diseases arise from a complex interplay of

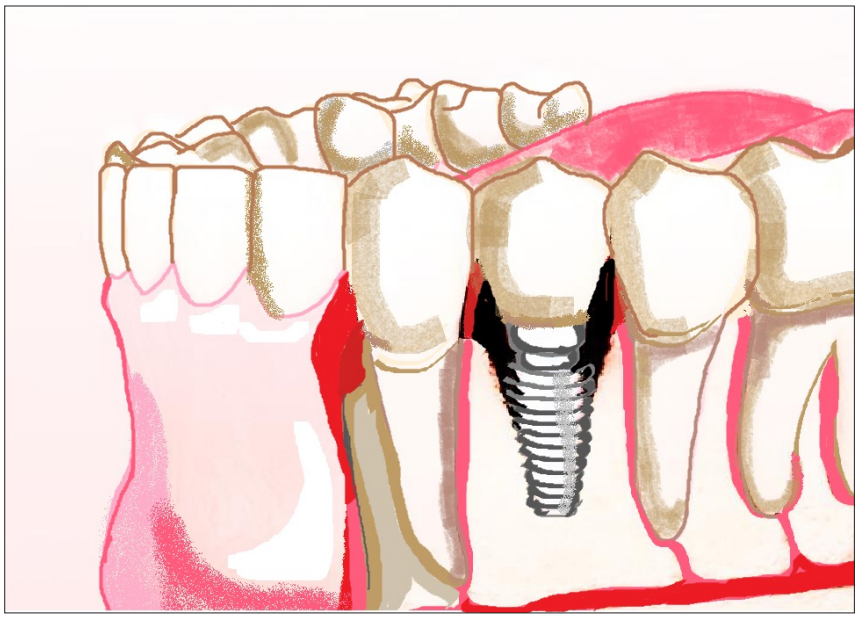

Figure 1. Peri-implantitis at the site of an implant insertion.

microbial biofilms, host susceptibility, and biomechanical stressors (105,106,162–170). At the core lies dysbiosis a collapse of the symbiotic relationship between the oral microbiome and host immunity (171–174). The primary culprits are microbial biofilms, which colonize implant surfaces with remarkable tenacity (175–180).

While periodontitis-associated bacteria like Porphyromona gingivalis and Tannerella forsythia are often present, peri-implant biofilms harbor unique pathogens. Staphylococcus aureus, a bacterium rarely found in periodontal pockets, thrives on titanium surfaces, forming dense aggregates resistant to antibiotics. Candida albicans, a fungal species, further complicates the picture by forming mixed biofilms that evade immune detection (181–190). These pathogens exploit local risk factors such as rough implant surfaces, subgingival cement remnants, and prosthetic overhangs, transforming the peri-implant sulcus into a reservoir of inflammation (108,191–196).

Host susceptibility plays an equally critical role. Genetic polymorphisms in inflammatory mediators like interleukin-1β (IL-1β) and tumor necrosis factor-α (TNF-α) amplify the body's response to biofilm, leading to collateral bone destruction(40,197–200). For example, carriers of the IL-1β +3954 T allele face a threefold increased risk of peri-implantitis (201–208). Systemic diseases act as force multipliers: diabetes mellitus impairs neutrophil function and collagen synthesis, while osteoporosis disrupts bone remodeling through altered RANKL/OPG ratios (209–220). Even psychological stress, via cortisol-induced immunosuppression, has been implicated in disease progression (71,221–232).

Biomechanical forces complete this pathogenic triad (233–242). Excessive occlusal loads-whether from bruxism, malocclusion, or poorly designed prostheses—induces microfractures in the crystal bone (243–248). Over time, these mechanical stresses synergize with inflammation, creating a vicious cycle of bone resorption (249–254). Finite element analysis studies illustrate how off-axis loading increases strain distribution by 40%, overwhelming the bone's adaptive capacity (255–263). This biomechanical-immune interplay explains why even patients with impeccable hygiene can develop peri-implantitis if their implants are biomechanically compromised(170,263–267) (Figure 2).

Diagnosis: Navigating the Gray Zones

Diagnosing peri-implant diseases remains an art as much as science. Clinical examination is the cornerstone: bleeding on probing (BoP) signals peri-implant mucositis, while deeper probing depths (≥6 mm) and radiographic bone loss ≥3 mm define peri-implantitis (268–275). Yet, these criteria are fraught with subjectivity (276–283). Probing depth measurements vary with instrument type (e.g., plastic vs. metal probes) and operator technique, while conventional radiographs often underestimate bone loss by 20–30% due to their two-dimensional nature (284–292).

Emerging technologies are bridging these diagnostic gaps (293–301). Cone-beam CT (CBCT) visualizes bone defects, revealing intricate details like defect morphology (circumferential, crater-like, or horizontal) and grippauproximity to vital structures (302–311). Radiomic algorithms, powered by machine learning, analyze CBCT datasets to quantify bone loss volume with 89% accuracy, offering objective metrics for monitoring progression (312–316). At the molecular level, in peri-implant crevicular fluid, biomarkers like interleukin-1β (IL-1β) and matrix metalloproteinase-8 (MMP-8) correlate firmly with disease activity (317–322). Chairside lateral flow assays for MMP-8, already validated for periodontitis, are now being adapted for peri-implantitis, promising rapid, non-invasive diagnostics (323–326). Microbial DNA analysis via PCR or next-generation sequencing adds another layer of precision, identifying pathogen profiles to

Figure 2. Peri-implantitis visible in the second quadrant on X-ray: OPT.

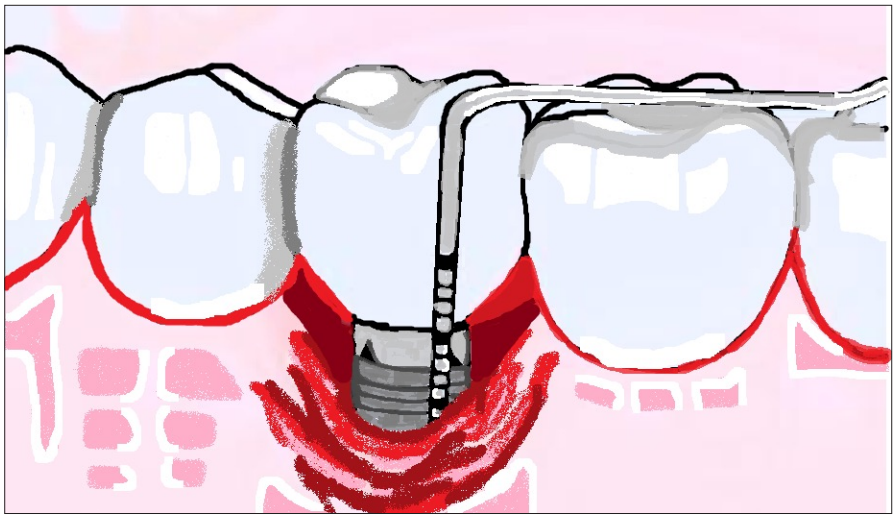

Figure 3. Periodontal probing of peri-implantitis

Table 1. Diagnostic Tools for Peri-Implant Diseases

Diagnostic Method	Key Features	Advantages	Limitations
Clinical Probing	Measures pocket depth and bleeding on probing (BoP)	Low cost, immediate results	Operator-dependent, may underestimate bone loss
Radiography (2D)	Periapical/panoramic X-rays to assess bone loss	Widely available, low radiation	Limited to 2D views, misses early defects
Cone-Beam CT (CBCT)	3D visualization of bone morphology and defect extent	High accuracy in defect characterization	Higher cost and radiation exposure
Biomarker Analysis	Detects IL-1β, MMP-8 in peri-implant crevicular fluid	Early disease detection, chairside potential	Requires specialized equipment
Microbial DNA Testing	PCR/NGS to identify pathogens (e.g., *S. aureus*, *Candida*)	Guides targeted antimicrobial therapy	Time-consuming, not routine in clinical practice

guide targeted therapy (312,327–333). For instance, detecting S. aureus might prompt the use of vancomycin rinses, while Candida-dominant cases may benefit from antifungal agents(334–339) (Figure 3) (Table 1).

Management: From Prevention to Regeneration

The management of peri-implant diseases demands a nuanced, stepwise approach. For peri-implant mucositis, mechanical debridement remains first-line. Titanium-coated brushes (e.g., TiBrush®) and glycine powder air-polishing devices effectively disrupt biofilms without scratching implant surfaces. Adjunctive therapies like localized drug delivery-minocycline microspheres (Dentomycin®) or chlorhexidine chips (PerioChip®)-suppress pathogens for months, reducing bleeding and inflammation. Photodynamic therapy (PDT), which combines light-activated dyes (e.g., methylene blue) with low-level lasers, eradicates 99.9% of P. gingivalis in vitro and has shown promise in reducing probing depths by 1.5 mm in clinical trials(268,340–345).

Advanced peri-implantitis often necessitates surgical intervention (346,346,347,347–353). Respective surgery, involving flap elevation and surface decontamination, remains the gold standard for shallow defects. Citric acid (pH 1) and Er lasers (2,940 nm) are preferred for decontaminating titanium surfaces, though debates persist over their relative efficacy(354–357). For complex defects, regenerative techniques like guided bone regeneration (GBR) combine bone grafts (xenogenic, alloplastic, or autogenous) with collagen membranes to restore lost architecture. A 2023 meta-analysis reported 2.1 mm of vertical bone gain with GBR, though long-term stability remains variable. Lasers are increasingly integrated into these procedures; ER lasers decontaminate surfaces and stimulate osteoblast activity, enhancing bone formation(358–363).

Adjunctive therapies are broadening the therapeutic arsenal. Probiotics like Lactobacillus reuteri lozenges modulate the oral microbiome, reducing bleeding by 34% in early trials. Systemic interventions, such as vitamin D supplementation in deficient patients, address underlying risk factors, potentially curbing recurrence. Even psychological support through stress-reduction programs has been hypothesized to improve outcomes by mitigating cortisol's immunosuppressive effects(364–367).

Controversies and Future Directions

The field of peri-implant diseases is rife with unresolved debates. The role of systemic antibiotics, for instance, remains contentious. While short courses

of metronidazole (500 mg/day for 7 days) reduce anaerobic bacterial loads, overuse risks Clostridioides difficile colitis and antimicrobial resistance. Similarly, the optimal method for implant surface decontamination—chemical agents like EDTA versus mechanical methods like lasers—lacks consensus. A 2022 randomized trial found no significant difference in recurrence rates between the two approaches, underscoring the need for personalized strategies(368–373).

Looking ahead, the horizon is bright with innovation (252,374–382). Precision medicine approaches, powered by machine learning, aim to predict individual risk by integrating genetic data (e.g., IL-1β polymorphisms), microbiomics profiles, and CBCT-derived radiomic features (383–390). Bioactive implant coatings embedded with silver nanoparticles or quorum-sensing inhibitors (e.g., furanone) are promising in preclinical studies, reducing biofilm formation by 70%. Immunomodulatory therapies, such as anti-IL-6 antibodies (tocilizumab) and RANKL inhibitors (denosumab), are in early-phase trials, offering hope for halting bone resorption at the molecular level (391–403). Even teledentistry is making inroads, with AI-powered apps analyzing patient-submitted photos for early signs of inflammation.

Conclusion

Peri-implant diseases underscore the intricate challenge of harmonizing human biology with technological innovation in dentistry. While current strategies focus on mitigating inflammation and regenerating bone through techniques like guided bone regeneration and laser therapy, these approaches often address symptoms rather than root causes.

The future of peri-implant disease management lies in a paradigm shift toward prevention and personalization, driven by collaboration across disciplines such as microbiology, biomechanics, and biomaterials science. Emerging advancements, including bioactive implant coatings embedded with antimicrobial agents like silver nanoparticles, aim to revolutionize implant design by creating surfaces that actively resist biofilm formation. Similarly, predictive models integrating genetic data (e.g., IL-1β polymorphisms) and advanced 3D imaging techniques could enable clinicians to identify high-risk patients early, tailoring prophylactic strategies such as probiotic regimens or immune-modulating therapies.

However, translating these innovations into clinical practice requires overcoming significant hurdles. Regulatory frameworks must be adopted to evaluate novel biomaterials, while cost barriers and the need for specialized training could delay widespread adoption. For example, anti-IL-6 antibodies, though promising in preclinical trials for halting bone loss, demand rigorous safety assessments and cost-effectiveness analyses before clinical use. Ethical considerations, including patient data privacy in genetic testing, require careful navigation as personalized therapies evolve.

Equally vital is the human-centric approach to care. Clinicians must balance technological progress with empathetic patient engagement. Digital monitoring tools, such as remote imaging platforms, could enhance follow-up care, but their success depends on patient adherence and accessibility. Addressing systemic health factors from diabetes management to stress reduction may improve outcomes by mitigating immune dysregulation linked to disease progression.

Economically, investing in preventive technologies like bioactive coatings or advanced diagnostic imaging may reduce the long-term costs of treating advanced peri-implantitis, alleviating burdens on healthcare systems. Longitudinal studies tracking the decade-long outcomes of regenerative therapies remain critical to validate their efficacy and refine guidelines.

In summary, the evolution of peri-implant disease management reflects a broader shift in healthcare—from reactive interventions to proactive, patient-centered care. By embracing innovation while respecting biological and behavioral complexities, the dental community can ensure implants fulfill their promise as enduring solutions for oral health. This journey demands creativity, collaboration, and a commitment to bridging scientific discovery with clinical practice—for the benefit of patients worldwide.

Abbreviations

Abbreviations	Definition
BoP	Bleeding on probing
CBCT	Cone-beam computed tomography
EDTA	Ethylenediaminetetraacetic acid
GBR	Guided bone regeneration
NGS	Next-generation sequencing
PDI	Peri-implantitis
PDL	Periodontal ligament
PIM	Peri-implant mucositis
PDT	Photodynamic therapy)
PCR	Polymerase chain reaction
WWP	World Workshop on Periodontology

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Informed Consent Statement

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Data Availability Statement

Data are contained within the article.

Conflicts of Interest

The authors declare no conflicts of interest

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