Management of Peri-Implantitis: A Literature Review on Diagnosis, Therapy, and Long-Term Maintenance

Abstract

Peri-implantitis is a biologically mediated, inflammatory disease that undermines the long term survival of dental implants by causing progressive bone loss within osseointegrated fixtures. Although implant-supported prostheses have achieved more than 90% long-term survival, prevalence of peri-implantitis continues to increase, with about 22% of patients developing the disease within a decade of implant placement. The current evidence-based review literature discussed modern strategies for diagnosis, treatment, and long-term management of peri-implantitis. The disease is a multifactorial effect of interaction among microbial biofilms, host immune response, genetic susceptibility, and iatrogenic variables. Diagnosis is confirmed on the basis of a combination of clinical parameters and sophisticated modalities such as cone-beam computed tomography, MMP-8 biomarker assays, and microbial PCR analysis. Treatment depends on the stage of the disease: early peri-implantitis can be managed non-surgically by mechanical debridement, antiseptics, and lasers, whereas advanced disease most commonly requires resective or regenerative surgical interventions based on defect morphology. The Cumulative Interceptive Supportive Therapy (CIST) protocol is an evidence-based clinical strategy for intervention. Emerging technologies such as antibacterial surface coatings, photodynamic therapy, and artificial intelligence augmented diagnostics have the potential to enhance clinical outcomes. Yet, long-term success is dependent significantly on organized maintenance such as three-month recall visits, professional biofilm control, and radiographic surveillance. This review emphasizes the value of incorporating conventional and innovative therapies into a stage-specific, prevention-based model for optimizing peri-implant health and preventing implant loss.

Keywords: Peri-implantitis, dental implants, implant complications, diagnosis, non-surgical therapy, regenerative surgery, maintenance protocols, biofilm control, Er:YAG laser, CIST protocol.

INTRODUCTION

Dental implants have transformed oral rehabilitation with the provision of long-term,

functional, and cosmetically acceptable solutions to tooth loss. With survival rates of more than 90% at 10 years, as determined by Pjetursson et al. (2012), implant-retained prostheses are now widely accepted worldwide as the gold standard for the replacement of missing teeth [1]. Their widespread global use is not only a sign of surgical progress but also patient satisfaction and long-term functional success.

But this therapeutic success comes at a biological price. Peri-implantitis, or inflammation of the peri-implant tissues followed by bone loss, has emerged as a major clinical problem around the practice of implant dentistry. While the disease can still remain undetected in its initial stages, its progression could be rapid and would eventually compromise the stability and survival of the implant. As noted by Smeets et al. (2014), peri-implantitis is a distinct clinical entity with a complex interrelation of risk factors that make both its diagnosis and treatment challenging [2].

The burden of an epidemiological nature is high. Derks and Tomasi (2015) state that about 22% of patients develop peri-implantitis during a period of ten years post-implant placement. Interestingly, patients with systemic risk factors—e.g., diabetes or smoking history—have a 3.2× greater chance of developing the disease, highlighting the need for patient selection and continuous risk evaluation [3].

In addition to its clinical implication, peri-implantitis also plays a huge economic impact. Salvi et al. (2017) indicated that the treatment of advanced lesions may cost as much as 350% higher than preventive treatment, often requiring complex retreatment or surgery [4]. Such repeated treatment can be detrimental to patient satisfaction, prolong healing, and diminish the perceived value of implant therapy. Current research has highlighted the contribution of avoidable iatrogenic factors. Wilson (2009) identified the presence of residual cement, commonly unseen under the prosthetic margins, as being accountable for 81% of the initial cases of peri-implantitis [5]. This has generated renewed interest in careful prosthetic technique and careful observation after the placement.

Microbiologically, the disease is most directly linked to specific pathogens that promote inflammatory breakdown. It has been shown through research that pathogens such as Porphyromonas gingivalis and Staphylococcus aureus have virulence factors that are capable of compromising host tissues and promoting inflammation within the peri-implant space [6]. While this microbial insult is not implant-specific, it appears to progress more quickly because of the absence of periodontal ligament defenses and the relatively compromised blood supply around the implant interface [2].

AIM OF THE REVIEW

In light of these growing concerns, this literature review seeks to synthesize current clinical and scientific perspectives on peri-implantitis treatment. It surveys current diagnostic methods, critically assesses non-surgical and surgical treatment options,

and highlights the importance of early prevention, prosthetic planning, and long-term maintenance protocols. Through the integration of evidence from landmark studies, this review offers clinicians a stage-specific, evidence-based strategy to minimize disease progression and enhance implant survival.

ETIOLOGY AND PATHOGENESIS

Peri-implantitis pathogenesis is a triad of factors: microbial colonization, host immune response, and biomechanical overload. Natural teeth have a periodontal ligament that prevents pathogens from traveling freely along the implant-bone interface [2].

- **Microbial Factors**

Gram-negative anaerobes prevail:Porphyromonas gingivalis has collagenases that break down connective tissue [6]. Staphylococcus aureus in 28% of cases increases inflammation through lipoteichoic acid [7].Biofilms quickly develop on rough surfaces of titanium, particularly in microgaps at abutment connections [2].

- **Host Factors**

Primary risk modulators are ,Genetic susceptibility is significant, especially IL-1 gene polymorphisms like IL-1β +3953 and IL-1α -889, which have been reported to increase TNF-α production, enhancing susceptibility to peri-implant inflammation and bone loss [8].
Systemic conditions: Diabetes (HbA1c >7%) affects neutrophil function [9].
Smoking decreases tissue perfusion by 40% [10].

- **Iatrogenic Triggers**

Endoscopic study of failed implants supports the link between residual cement and 81% of early-onset peri-implantitis patients. [11].Prosthetic design: Crowns that are overly contoured make it difficult to maintain proper hygiene [12].Occlusal overload occurs when excessive forces lead to bone resorption [13].

HISTOPATHOLOGICAL PERSPECTIVE

Peri-implant lesions show more plasma cell infiltration compared to periodontitis, which accounts for their quicker progression (2.5 times faster bone loss) [2].

DIAGNOSTIC PROTOCOL.

The clinical examination for peri-implantitis includes the use of plastic probes with a force of 0.25 N. The diagnostic criteria include detecting blood or pus while probing, probing depths of 6 mm or more, and movement, which implies a later stage of the illness [5]. The

evaluation through radiographs involves standardised periapical images to measure bone loss, and cone-beam computed tomography (CBCT) is used to examine the three-dimensional shape of defects [14]. Advanced diagnostics feature MMP-8 point-of-care tests, providing 89% sensitivity in predicting active bone loss [15], alongside microbial PCR testing that reveals pathogen profiles for more precise antibiotic therapy [16]. It's important to remember that obtaining baseline radiographs when loading a prosthetic is crucial. If bone loss surpasses 0.2 mm per year from this initial point, it suggests the presence of a problem [17].

NON-SURGICAL THERAPY

For early peri-implantitis and mucositis with probing depths of 5 mm or less, first-line therapy is recommended. Jepsen and colleagues (2015) emphasise that managing mechanical plaque is still essential [18]. Key methods include erythritol air-polishing, which reduces biofilm by 67% at a 60° angle [19], using titanium curettes to remove calculus without harming implant surfaces, and using ultrasonics with PEEK-coated tips in conjunction with glycine irrigation. Adjunctive therapy includes local antiseptics using chlorhexidine chips, which prevent infections for 21 days [20] and systemic antibiotics, especially amoxicillin-metronidazole (500 mg TID for 7 days), for severe cases [21]. With a 2940 nm wavelength, the Er:YAG laser destroys 89% of biofilm [22].Nonetheless, non-surgical treatment has its limitations, achieving resolution in only 37% of pockets exceeding 5 mm because of threads that are difficult to access [23].

SURGICAL MANAGEMENT

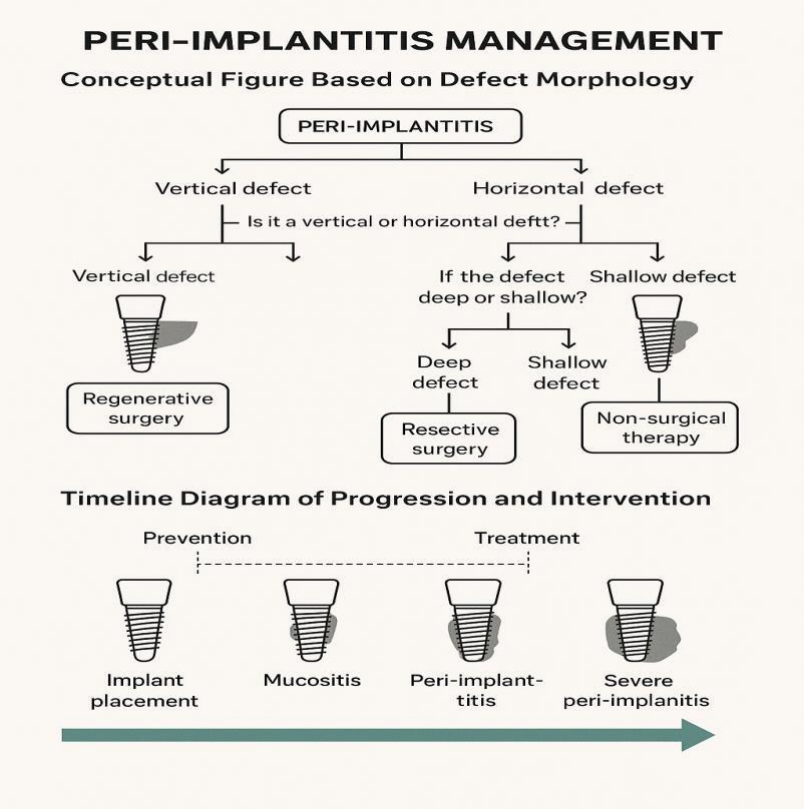
Reconstructive surgery is recommended for horizontal defects [24] and involves techniques including osteoplasty to produce positive architecture, implantoplasty with diamond burs to smooth exposed threads, and an apically positioned flap. At three years, results indicate a 72% disease remission rate, despite the absence of bone fill [25].During regenerative surgery, which follows the gold standard procedure [26], flap elevation and degranulation, surface decontamination with an Er:YAG laser, application of a xenograft (DBBM) combined with a collagen membrane, and primary closure are all steps that are taken. Results show that 81% of bone had filled in by the 24-month mark [27]. In a recent study, Roccuzzo et al. (2024) demonstrated that the combination of bone grafts and platelet-rich fibrin (PRF) not only accelerates the process of angiogenesis but also cuts the amount of time needed for healing by 35 percent [28].

CLINICAL CONSIDERATIONS AND MAINTENANCE

One important consideration for choosing the right treatment is the morphology of the peri-implant defect. With a reported success rate of 81% [27], regenerative methods are most suited for treatments that involve abnormalities with three or four walls. A combination of

regenerative procedures and guided bone regeneration (GBR) is effective in treating crater-shaped lesions, with a 68% success rate. Resective surgery is the most effective way to treat horizontal abnormalities, with a 72% success rate [25]. Decontamination of the implant surface is another essential component towards achieving excellent outcomes. Considered the gold standard due to its 98% bacterial kill rate, the Er:YAG laser is Chemical agents including 24% EDTA gel [30] can effectively remove endotoxin from the implant surface. Consequently, it is imperative to avoid damaging or changing the surface of the implant by not utilising metal curettes during the cleaning procedure.

Supportive maintenance is essential in preventing the recurrence of peri-implantitis. Jepsen et al. (2015) emphasised that implementing a structured recall every three months can lead to a 58% reduction in recurrence rates [18]. During maintenance visits, clinicians are required to evaluate probing depths (PD), bleeding on probing (BoP), and the mobility of implants. Re-instrumentation generally involves the application of air-polishing in conjunction with titanium curettes. It is recommended that high-risk patients receive antimicrobial support, such as 0.12% chlorhexidine rinses. Annual periapical radiographs are recommended to



assess bone levels and identify early changes. Long-term outcomes indicate that survival rates are significantly higher with supportive care, achieving 92.3% at five years with regular maintenance, in contrast to 64.7% without such care. [31]

EMERGING INNOVATIONS

New technologies are playing an important role in the management of peri-implantitis. Antibacterial coatings using zinc oxide nanoparticles show promise in reducing microbial adhesion on implant surfaces [32]. Photodynamic therapy, using methylene blue combined with 660 nm wavelength light, shows promise for the eradication of pathogenic

microorganisms [33]. The diagnosis accuracy of peri-implant bone loss detection has been much enhanced by the use of artificial intelligence, most especially by convolutional networks. The stated general accuracy spans 61% to 94.74%. Early and accurate detection of peri-implant illness enabled by these artificial intelligence models helps doctors enhance the results and planning of treatment.
[34].

CLINICAL PROTOCOL: THE CIST FRAMEWORK

From the clinical presentation, the Cumulative Interceptive Supportive Therapy (CIST) protocol, developed by Lang and Berglundh [35], is a structured, phase-oriented approach for the treatment of peri-implant disease. Throughout treatment, it allows for timely intervention at every level, ranging from the initial mucosal inflammation to the late stages of bone loss.

Stage	Clinical Presentation	Interventions	Key Reference
A	BoP+ without bone loss	Oral hygiene reinforcement	Jepsen et al. 2015 [18]
B	PD 4–5 mm + bone loss <2 mm	Mechanical debridement + antiseptics	Smeets et al. 2014 [2]
C	PD 5–7 mm + bone loss 2–4 mm	Local antibiotics + occlusal adjustment	Roccuzzo et al. 2024 [28]
D	PD >7 mm + bone loss >4 mm	Resective/regenerative surgery	Berglundh et al. 2018 [5]

As an example, a diabetic patient who presents with a probing depth of 6 millimetres and a bone loss of 3 millimetres is considered to be in Stage C. Because of this particular circumstance, the intervention that would be most suitable would be mechanical debridement in conjunction with minocycline microspheres [36]. Later, if the problem doesn't go away, Stage D treatment would be done, which includes access flap surgery, decontamination with an Er:YAG laser, and the use of a xenograft with platelet-rich fibrin (PRF) [28].

CONCLUSION

Peri-implantitis has a higher prevalence and clinical severity, thus it requires a systematic, evidence-based diagnosis, intervention, and maintenance. Early diagnosis with diagnostic tools such as MMP-8 testing and initial x-rays is essential to enable physicians to observe when the disease progresses in a timely way. Treatment should be led by defect morphology; non-surgical interventions are acceptable for early disease, while regenerative treatments are ideal for contained abnormalities. Structured maintenance, involving recalls every quarter, has been shown to decrease recurrence by 58% [18], underlining the importance of long-term follow-up.

Antibacterial coatings, photodynamic therapy, and artificial intelligence (AI)-aided diagnostics are some of the novel technologies that hold potential as augmentation of the fundamental concepts presented by Smeets et al. (2014) [2]; disease prevention by meticulous control of biofilm and improved prosthetic design remains the most critical aspect. In the future, additional research should concentrate on predictable and protocol re-osseointegration regimens as well as risk-based personalised approaches to improve clinical outcomes even further and implants to endure longer.

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LEGENDS

Figure 1: Visual guide to peri-implantitis management. Top: Treatment decision tree based on defect morphology (vertical vs. horizontal, deep vs. shallow). Bottom: Timeline of peri- implantitis progression from implant placement to advanced bone loss, with corresponding intervention points. [5,18,26,28]Adapted by author based on data from Jepsen et al. (2015), Berglundh et al. (2018), Schwarz et al. (2010), and Roccuzzo et al.

(2024).

Figure 2 : CIST Protoco