

## Peri-Implantitis: A Review

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### ABSTRACT

Peri-implantitis is a plaque-associated pathological condition occurring in tissues around dental implants, characterized by inflammation and progressive bone loss. With the increasing prevalence of implant therapy, peri-implantitis has emerged as a significant clinical concern that threatens long-term implant success. This review summarizes current knowledge regarding etiology, risk factors, pathogenesis, diagnosis, and management of peri-implantitis. The role of microbial biofilm, host response, systemic and local risk factors, and surgical and non-surgical treatment modalities are discussed. Emphasis is placed on preventive strategies and emerging therapeutic approaches aimed at preserving peri-implant health and improving implant longevity.

### Introduction:

Dental implants have become a predictable and widely accepted treatment modality for the replacement of missing teeth, with reported success rates exceeding 90% over ten years [1]. However, biological complications such as peri-implant mucositis and peri-implantitis have gained increasing attention due to their impact on implant survival [2]. Peri-implantitis is defined as an inflammatory process affecting the soft and hard tissues surrounding an osseointegrated implant, leading to loss of supporting bone [3]. As implant therapy becomes more common, understanding the mechanisms, diagnosis, and management of peri-implantitis is crucial for clinicians.

### Epidemiology

Epidemiological studies indicate that peri-implantitis affects approximately 10–20% of implant sites and up to 30% of implant patients [4]. The variability in prevalence across studies is attributed to differences in diagnostic criteria, population characteristics, and observation periods [5]. Mucositis, the reversible inflammation of peri-implant soft tissues, occurs in up to 80% of patients with implants [6]. The transition from mucositis to peri-

implantitis underscores the importance of early diagnosis and preventive care.

### Etiology and Pathogenesis

The primary etiological factor in peri-implantitis is the accumulation of bacterial biofilm at the implant surface [7]. The microbiota associated with peri-implantitis resembles that of periodontitis, including *Porphyromonas gingivalis*, *Tannerella forsythia*, and *Treponema denticola* [8]. However, studies also show higher proportions of opportunistic pathogens such as *Staphylococcus aureus* and *Candida albicans* [9]. These microorganisms adhere to rough implant surfaces and trigger an inflammatory host response leading to tissue destruction.

### Host Response

The host immune response plays a central role in the pathogenesis of peri-implantitis. Pro-inflammatory cytokines such as interleukin-1 $\beta$  (IL-1 $\beta$ ), tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), and prostaglandin E<sub>2</sub> (PGE<sub>2</sub>) are elevated in peri-implant crevicular fluid [10]. The imbalance between pro- and anti-inflammatory mediators contributes to connective tissue breakdown and bone resorption. Genetic polymorphisms, such as IL-1

genotype variations, have been implicated as susceptibility factors [11].

### **Implant Surface and Material Factors**

Implant surface characteristics influence bacterial colonization and tissue response. Rough surfaces promote osseointegration but may also enhance microbial retention [12]. Additionally, titanium particles released due to mechanical wear or corrosion may exacerbate inflammation [13].

### **Risk Factors**

#### **Patient-Related Factors**

Smoking remains one of the strongest risk factors for peri-implantitis, with smokers exhibiting up to a threefold increased risk compared to non-smokers [14]. Systemic diseases such as uncontrolled diabetes mellitus also predispose individuals to peri-implant inflammation due to impaired wound healing and immune response [15]. Poor oral hygiene and a history of periodontitis are consistent predictors of peri-implantitis development [16].

#### **Prosthetic and Surgical Factors**

Improper implant positioning, residual cement, and over-contoured restorations can create plaque-retentive areas, facilitating biofilm accumulation [17]. Inadequate keratinized mucosa and excessive occlusal load have also been implicated [18].

### **Clinical and Radiographic Features**

Clinically, peri-implantitis is characterized by bleeding on probing, increased probing depth (>5 mm), suppuration, and peri-implant bone loss beyond initial remodeling [19]. Radiographically, vertical or circumferential bone loss around the implant is evident [20]. Disease progression may be rapid and non-linear, often leading to implant failure if left untreated [21].

### **Diagnosis**

Diagnosis relies on clinical and radiographic assessment. Probing pocket depth, bleeding on probing, and suppuration are key clinical parameters [22]. Baseline radiographs taken at prosthesis placement are essential for detecting progressive bone loss [23]. Advanced imaging modalities such as cone-beam computed

tomography (CBCT) provide detailed three-dimensional assessment of bone defects [24]. Microbiological and biomarker analyses, while promising, are not yet standardized for routine use [25].

### **Prevention**

Preventive measures focus on controlling plaque accumulation and maintaining peri-implant health. Regular professional maintenance visits, patient education, and reinforcement of oral hygiene are essential [26]. Antimicrobial mouth rinses containing chlorhexidine or essential oils may reduce bacterial load [27]. Early management of peri-implant mucositis prevents progression to peri-implantitis [28].

### **Management of Peri-Implantitis**

#### **Non-Surgical Therapy**

Non-surgical approaches aim to reduce bacterial load through mechanical and chemical means. Debridement using titanium or carbon-fiber curettes, air-abrasive systems, or ultrasonic devices can disrupt biofilm [29]. Adjunctive use of local or systemic antibiotics, such as minocycline or amoxicillin/metronidazole, has shown short-term benefits [30]. However, complete resolution of peri-implantitis through non-surgical therapy alone is uncommon, especially in advanced cases [31].

#### **Surgical Therapy**

Surgical intervention is often necessary to achieve access for decontamination and regeneration. Open-flap debridement facilitates thorough cleaning of the implant surface [32]. Various decontamination methods, including laser therapy, photodynamic therapy, and chemical agents like citric acid and hydrogen peroxide, have been proposed [33]. Regenerative techniques using bone grafts and membranes aim to restore lost peri-implant bone [34]. The success of regenerative therapy depends on defect morphology, implant surface, and maintenance compliance [35].

#### **Implant Surface Decontamination**

Effective surface decontamination remains a challenge due to the complexity of implant topography. Studies suggest that a combination of mechanical and chemical cleaning yields better outcomes than either method alone [36]. Emerging technologies, such as cold plasma and

electrolytic cleaning, show promise for biofilm removal without damaging the implant surface [37].

### Emerging and Adjunctive Therapies

Recent research has explored the use of probiotics, photodynamic therapy, and laser-assisted protocols as adjuncts to conventional treatment [38]. Additionally, biologic agents such as enamel matrix derivatives, platelet-rich fibrin (PRF), and growth factors have been investigated for their regenerative potential [39]. The use of locally delivered statins and antimicrobial peptides also represents a growing area of interest [40]

### Prognosis

The prognosis of peri-implantitis depends on disease severity, patient compliance, and treatment approach. Early detection and intervention improve outcomes significantly [41]. Implants with circumferential defects and residual bone loss exceeding 50% often exhibit compromised survival [42]. Long-term success is highly dependent on stringent maintenance protocols [43].

### Future Directions

Future research should focus on developing standardized diagnostic criteria and evidence-based treatment protocols. Personalized medicine, incorporating genetic and microbiome profiling, may help identify at-risk individuals [44]. Advances in biomaterials and surface engineering may produce implants with enhanced resistance to microbial colonization [45]. Furthermore, digital monitoring tools and artificial intelligence-assisted diagnostics hold potential for early disease detection and better patient management [46].

### Conclusion

Peri-implantitis represents a complex multifactorial disease with significant clinical implications. Effective management requires an understanding of microbial, host, and implant-related factors. Prevention through meticulous plaque control, regular maintenance, and risk factor modification remains the cornerstone of care. Although numerous treatment modalities exist, long-term evidence supporting their predictability is limited. Continued research into novel regenerative and antimicrobial therapies, along with individualized treatment planning, will be essential to improving outcomes and ensuring the longevity of dental implants.

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