Review

The Prevalence, Diagnosis, Management and Prognosis of Peri-Implantitis

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Abstract

Peri-implantitis, characterized by inflammatory reactions resulting in bone loss around dental implants integrated with the jawbone, presents a significant challenge in implant dentistry. While bacterial biofilms are recognized as the primary culprits in peri-mucositis, peri-implantitis is often attributed to mechanical stresses stemming from biomechanical deficiencies, along with a myriad of other contributing factors, including implant positioning, oral hygiene, cement residues, host responses, implant surface quality, bone density, untreated periodontal issues, smoking, alcohol consumption, endodontic problems, and diabetes. Despite extensive research efforts, a universally accepted protocol for effectively managing peri-implant diseases remains elusive. This review provides an overview of peri-implantitis, offering valuable insights into its multifactorial etiology, diagnostic challenges, and evolving therapeutic strategies. By consolidating existing knowledge, this review aims to equip clinicians with a more informed approach to managing this complex and prevalent condition, ultimately improving patient care in implant dentistry.

Keywords: peri-implantitis, dental implants, inflammatory reactions, bacterial biofilms, oral hygiene
Introduction

Peri-implantitis is a broad term encompassing inflammatory reactions triggered by bacterial biofilms, leading to the depletion of bone around dental implants that have integrated with the jawbone (1). While bacterial influence is recognized as the primary instigator of peri-mucositis, the onset of peri-implantitis is believed to be triggered by mechanical stresses arising from an inadequate biomechanical setting. Furthermore, several other contributing factors include suboptimal implant positioning, inadequate oral care, lingering cement residue, host rejection responses, unfavorable implant surface conditions, low bone density, untreated periodontal issues, tobacco and alcohol consumption, unaddressed endodontic problems, and the presence of diabetes, among others (2). Despite numerous research efforts dedicated to determining the optimal treatment methods for these conditions, a universally accepted protocol for their effective and predictable resolution has yet to be established. This review aims to concentrate on regenerative approaches for treating bone defects around dental implants, with the intention of offering valuable insights that can assist clinicians in their management of peri-implant diseases (3).

In this review, we will delve into various aspects of peri-implantitis, including its prevalence, the factors that contribute to its occurrence, the methods used for diagnosis, and the therapeutic strategies employed for its management. We will provide an overview of this condition, shedding light on its various facets and offering insights into both its causes and potential solutions.

Methodology

This study is based on a comprehensive literature search conducted on September 4, 2023, in the PubMed, Web of Science, Science Direct, and Cochrane databases, utilizing the medical topic headings (MeSH) and a combination of all available related terms, according to the database. To prevent missing any possible research, a manual search for publications was conducted through Google Scholar, using the reference lists of the previously listed papers as a starting point. We looked for valuable information in papers that discussed the prevalence, diagnosis, management, and prognosis of peri-implantitis. There were no restrictions on date, language, participant age, or type of publication.

Discussion

Dental implants are a reliable solution for replacing missing teeth, with high survival rates. However, they can develop a complication called peri-implantitis, an inflammation that can lead to implant failure if not detected and treated early. Patients with a history of periodontal disease or who smoke are at a higher risk of peri-implantitis. Detection and timely intervention are crucial for successful implant rehabilitation (4).

Peri-implantitis is a common problem with dental implants, causing inflammation, pocket formation, and bone loss. It's typically diagnosed by changes in the bone level around the implant and bleeding on probing (BOP). This issue affects approximately 13% of implants and 18.5% of patients, with the incidence increasing rapidly over 3 to 5 years. While bacteria play a significant role in its development, there are various factors, both inherent and modifiable, that can increase the risk of peri-implantitis. Identifying these factors is essential for preventing and treating the condition (5).

Mechanism of peri-implantitis

Peri-implantitis is defined by an irreversible and gradually progressing loss of the bone that supports the implant. It's typically accompanied by bleeding and/or discharge when probed. This condition is a primary factor leading to implant failure. The development of peri-implant disease shares similarities with periodontal disease, as both are initiated by an inflammatory response to the accumulation of biofilm. Despite numerous clinical studies assessing the prevalence of peri-implantitis over various follow-up periods, there is currently limited information regarding how implant placement location impacts the prevalence of peri-implantitis (6). As the utilization of dental implants for oral rehabilitation continues to grow, the occurrence of
peri-implant inflammatory conditions has also increased, presenting a considerable clinical concern. Peri-implant disease encompasses two distinct conditions: peri-implant mucositis, which involves reversible inflammation limited to the soft tissues surrounding an implant, and peri-implantitis, which entails the loss of alveolar bone around the implant due to the advancement of the inflammatory process (7).

To ensure the long-term stability of dental implants, they need to integrate firmly with the surrounding bone. Specialized cells called osteoblasts produce bone material and control its mineralization. The balance and mechanical strength of the bone depend on a coordinated process involving osteoblasts that build bone and osteoclasts that break it down. Osteocytes also play a vital role in maintaining implant stability by regulating bone cell activity in response to mechanical stress and local factors in the surrounding environment (8).

Pathogenic biofilms adhering to the implant surface and peri-implant tissues can result in the deterioration of both bone and soft tissues. Certain bacteria, such as Porphyromonas gingivalis, Prevotella intermedia, and Aggregatibacter actinomycetemcomitans, are potential culprits of peri-implantitis and are known for their involvement in biofilm formation on dental implants. In response to bacterial infection, the immune system mobilizes various components, including neutrophils, macrophages, T cells, and B cells, which then migrate to the affected area. Throughout the development of peri-implantitis, the loss of alveolar bone can lead to instability and ultimately result in the loss of the implant (9).

The interaction between the immune system and bone becomes significant when the immune system is activated. In cases of pathological conditions linked to inflammation and immune response activation, immune cells like T lymphocytes, macrophages, and dendritic cells release various pro-inflammatory cytokines. These cytokines disrupt the equilibrium between bone-building osteoblasts and bone-resorbing osteoclasts, frequently resulting in heightened osteoclast activity. Moreover, certain pro-inflammatory cytokines seem to work together to promote osteoclast formation and enhance osteoclast function (10).

Comprehending the infectious causes and development of peri-implant diseases involves two essential steps. First, we must grasp a) the factors causing these diseases and the mechanisms behind them, similar to what we already know about periodontal diseases. Second, we need to consider b) the structural and immunopathological distinctions between periodontal and peri-implant tissues. In essence, our existing knowledge of periodontal diseases should serve as a foundation for understanding peri-implant diseases, while also recognizing that any disparities between the two may lead to new avenues for research (11).

While there are clinical and histopathological similarities between the periodontal and peri-implant mucosa, there are also notable differences. One key distinction is the absence of Sharpey's fibers that anchor the teeth's cementum perpendicularly to the implant surface. Instead, collagen fibers in the submucosal connective tissue run parallel to the implant surface. This results in a deeper peri-implant crevice compared to the gingival crevice, making it easier for bacteria to penetrate. Additionally, natural teeth are anchored in bone via the periodontal ligament and Sharpey's fibers, whereas implants achieve direct osseointegration. The lack of a periodontal ligament has several implications: it reduces the physical barrier against bacterial invasion into submucosal tissue, making peri-implant tissues more susceptible to infection. It also limits blood supply, as the soft peri-implant tissues rely on supra-periosteal vessels rather than the periodontal ligament. This reduced blood supply affects nutrient delivery and immune cell presence, which are crucial for early defense against bacterial colonization (11).

**Prevalence of peri-implantitis**

There is a lack of a definitive definition that effectively illustrates the prevalence of peri-implantitis. Numerous varying case definitions have been in circulation over recent years, making it
challenging to obtain a precise understanding of the true prevalence rate. However, it is estimated that roughly one in every four to five patients is affected (12).

The prevalence of peri-implant diseases is primarily documented through retrospective studies. For instance, Fransson et al. (2005) revealed that 90% of peri-implant tissues exhibited some form of inflammatory response, with a peri-implantitis prevalence of 28% in their research. Similarly, Roos-Jansaker et al. (2006) reported a peri-implant mucositis prevalence of approximately 48%, while 6.6% of the implants in their study were affected by peri-implantitis. However, more recent studies, like the one conducted by Rodrigo et al. in 2018, indicate a 51% prevalence of peri-implant diseases within the Spanish population (13).

A retrospective study with 88 patients conducted by Lv et al. in 2023 found that at the patient level, peri-implantitis had a prevalence of 9.1%, while at the implant level, it was 6.6%. In contrast, the prevalence of peri-implant mucositis was 76.1% at the patient level and 51.1% at the implant level (14).

When comparing dental implants placed in natural bone to those placed in the augmented bone, limited research exists on the prevalence of peri-implant diseases and implant failure rates. Augmentation procedures are common due to complications like bone loss after tooth extraction. A systematic review of eight studies found no significant difference in peri-implantitis prevalence between natural (10.3%) and augmented (17.8%) sites, but variability was higher in augmented sites. Patient-based implant failure rates were 2.5% in natural and 3.6% in augmented sites, with varying definitions of peri-implantitis across the studies (16).

**Risk factors of peri-implantitis**

The potential risk factors linked to peri-implant diseases can be categorized into three groups: patient-related factors, implant-related factors, and long-term factors. Patient-specific factors like a history of periodontitis and smoking have shown clear associations with peri-implant diseases, while evidence regarding other factors such as diabetes and genetic predisposition remains inconclusive. Additionally, implant-specific factors like implant placement, soft tissue qualities, and the choice of implant connection, along with long-term factors like inadequate plaque control and the absence of a maintenance program, have been suggested to significantly influence the maintenance of dental implant health (17).

In one study, a total of 350 individuals were examined. The results indicated that the occurrence of peri-implantitis stood at 18.2% among non-smokers, 19.7% among former smokers, and notably higher at 30.5% among current smokers. It is worth mentioning that the current smoker group exhibited a significantly higher prevalence of periodontitis cases at 54.2% compared to both the former smoker and non-smoker groups (18).

Remarkably, a recent report revealed that the overall DNA methylation level in gingival tissues was greater than that observed in bone tissue, irrespective of whether the bone came from patients with healthy periodontal conditions or from areas surrounding failed implants due to peri-implantitis (19).

**Diagnosis approaches**

The diagnosis of peri-implantitis relies on several clinical parameters, as detailed in research articles. These parameters serve as essential tools for establishing a baseline and detecting the progression of peri-implant disease. Key diagnostic criteria encompass the assessment of pain, implant mobility, and probing depths, with various thresholds proposed by different authors. Radiographic evaluation, including conventional and three-dimensional radiographs, aids in identifying bone loss, with thresholds varying from > 2 mm to > 4 mm for diagnosis. Several
classification systems exist based on bone loss percentage or crestal bone changes compared to baseline data. In cases where prior radiographic records are unavailable, a vertical distance of 2 mm from the expected marginal bone level post-implant placement can be used as a threshold, provided there is evidence of peri-implant inflammation. These parameters collectively form a comprehensive diagnostic framework, guiding clinicians in assessing implant health and determining the severity of peri-implant disease (20, 21).

Defining and diagnosing peri-implant conditions involves distinct criteria. To diagnose peri-implant health, there should be an absence of clinical inflammation, bleeding, suppuration during gentle probing and no increase in probing depth beyond previous measurements, with bone levels remaining within the crestal bone level changes due to initial bone remodeling. For diagnosing peri-implant mucositis, the presence of bleeding and/or suppuration on gentle probing, with or without increased probing depth compared to earlier assessments, is indicative, while bone loss should remain within crestal bone level changes resulting from initial remodeling. The diagnosis of peri-implantitis entails the presence of bleeding and/or suppuration on gentle probing, increased probing depth compared to prior examinations, and bone loss exceeding crestal bone level changes caused by initial bone remodeling (22, 23).

As per the 2018 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions, the diagnosis of peri-implant mucositis involves two key criteria: the presence of visible inflammation around the implant (such as redness, swelling, or bleeding within 30 seconds after probing) and the absence of any additional bone loss beyond the initial healing stage. On the other hand, the clinical diagnosis of peri-implantitis comprises three criteria: the presence of visible inflammation around the implant, radiographic evidence showing bone loss subsequent to initial healing, and an increase in probing depth compared to the depth observed after placing the prosthetic reconstruction. In cases where previous radiographs are unavailable, a radiographic bone level of ≥3 mm combined with both BOP and probing depth of ≥6 mm serves as an indicator of peri-implantitis (24, 25).

**Primary Diagnostic Tools**

**Measuring Pocket Depth**

Residual pockets contribute to the progression of periodontal deterioration. A pocket depth of ≥6 mm following active periodontal treatment has been identified as a risk factor for tooth loss. Similarly, evaluating pocket depth around implants is crucial for monitoring peri-implant conditions. Nevertheless, some clinicians argue that pocket depth (PPD) and BOP measurements are not reliable indicators of peri-implant tissue health and suggest that disturbing the soft tissue barrier at implants could lead to inflammation and bone loss. Additionally, excessive diagnosis and treatment based on unreliable indices may inadvertently harm the implant-tissue interface (26).

**Bleeding on probing**

BOP as a diagnostic tool presents varying levels of accuracy. Around natural teeth, BOP is highly accurate (87%) in detecting attachment losses exceeding 2 mm, but its sensitivity is relatively low (29%), while its specificity is high (88%). However, when applied to dental implants, BOP becomes more challenging to interpret due to the unique peri-implant tissue morphology. Even with deeper probe penetration, BOP positivity does not necessarily indicate disease, as it can also be observed around healthy implants. Therefore, the accuracy of BOP around dental implants is less conclusive, and factors like probing force need careful consideration to minimize false negative results (27).

**Suppuration**

Suppuration, or the presence of pus, is a notable feature in peri-implantitis, a condition characterized by larger and more immune cell-rich lesions than periodontitis. However, suppuration is rare in the absence of disease. Notably, the absence of suppuration doesn't guarantee the absence of disease, as inflammation can transition to new connective tissue formation. Therefore, evaluating both bleeding on probing for acute lesions and
suppuration for advanced or chronic lesions is crucial for assessing peri-implant health (28).

**Molecular tests on the market for peri-implantitis**

Regular monitoring of dental implants is crucial to preventing complications. Molecular tests, particularly MMP-8, show promise for early diagnosis. New PoC test kits like PerioSafe® PRO DRS and ImplantSafe® DR detect active MMP-8 in saliva quickly and affordably. These tests are validated globally, offering high accuracy for screening, distinguishing active sites, predicting disease progression, and monitoring treatment. FDA-approved and available in the US and EU, these tools improve periodontal and peri-implant disease diagnosis, making them accessible to both patients and general clinicians for referrals when needed (29).

Future directions include proteomics and metabolomics for accurate, site-specific diagnosis and prediction of peri-implant disease progression. The assessment of proinflammatory cytokines (IL-1β, TNFα, MMP-8) in the peri-implant crevicular fluid may be of value to diagnose peri-implantitis and peri-implant mucositis, but present investigations are not sufficient to reveal whether biomarkers predict peri-implant disease progression (30) (Table 1).

<table>
<thead>
<tr>
<th>Diagnostic Tool</th>
<th>Use</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Measuring Pocket Depth</td>
<td>Assessing attachment loss around implants; important for monitoring peri-implant conditions; risk factor for tooth loss (≥6 mm) after periodontal treatment</td>
<td>(26)</td>
</tr>
<tr>
<td>Bleeding on Probing (BOP)</td>
<td>Detecting attachment losses exceeding 2 mm; less conclusive around implants; careful consideration of probing force needed to minimize false negatives</td>
<td>(27)</td>
</tr>
<tr>
<td>Suppuration</td>
<td>Notable in peri-implantitis, indicating larger and more immune cell-rich lesions; rare in the absence of disease; presence doesn't guarantee disease absence</td>
<td>(28)</td>
</tr>
<tr>
<td>Molecular Tests (e.g., MMP-8)</td>
<td>Promising for early diagnosis; PerioSafe® PRO DRS and ImplantSafe® DR detect active MMP-8 in saliva, offering high accuracy for screening, predicting progression, and monitoring treatment</td>
<td>(29)</td>
</tr>
<tr>
<td>Future Directions (Proteomics, Metabolomics)</td>
<td>Potential for accurate, site-specific diagnosis and prediction of peri-implant disease progression; research ongoing regarding proinflammatory cytokines (IL-1β, TNFα, MMP-8) in peri-implant crevicular fluid (PICF)</td>
<td>(30)</td>
</tr>
</tbody>
</table>
Management of peri-implantitis

The management of peri-implant infections involves both non-surgical and surgical methods. The choice between these approaches depends on the extent of the peri-implant disease, which can range from mild mucositis to moderate or severe peri-implantitis. In some cases, a non-surgical treatment alone may suffice, while in others, a combination of non-surgical therapy followed by surgical intervention may be required, depending on the severity of the condition (31).

Therapy for mucositis

One randomized controlled trial mentioned that non-surgical cleaning and maintaining oral hygiene proved effective in reducing peri-implant mucositis, although they did not consistently lead to full resolution of inflammation. The additional use of chlorhexidine gel alongside mechanical cleansing did not yield better outcomes compared to mechanical cleaning alone. Notably, implants with restoration margins located above the gingiva exhibited more significant therapeutic improvements compared to those with margins located below the gingiva (32). Another RCT mentions that the necessity of using antibiotics to treat peri-implant mucositis raises questions and should be evaluated in light of the broader risk of antibiotic resistance within the community. Given these considerations and the absence of observable clinical benefits from the supplementary use of antibiotics in this study, it can be inferred that the primary approach to treating peri-implant mucositis should be non-surgical debridement without the concurrent use of systemic antibiotics (33). The establishment of adequate oral hygiene should, therefore, be considered a key issue in the prevention of peri-implant infections. Besides, a maintenance program with regular evaluation of the peri-implant probing depths, supportive professional implant cleaning, and oral hygiene training should be an integral part of every post-operative care plan after implant insertion (31).

Therapy for peri-implantitis

The main goal in treating peri-implantitis is to resolve soft tissue inflammation, reduce probing pocket depth to ≤5 mm, and stop bone loss. The treatment approach depends on the defect's characteristics. Regenerative strategies are considered for containing defects to restore tissue and support. Regardless of the method, reducing probing depth is key. Treatment may lead to tissue recession, especially with surgical methods, but it does not significantly affect horizontal soft tissue dimensions. The presence of keratinized mucosa around the implant plays a crucial role in soft tissue changes (34).

Implant cleaning methods

Cleaning dental implants is important, and different methods have been explored. Conventional curettes can roughen implant surfaces, so softer tip materials are recommended. Both piezoelectric scalers and hand instruments can reduce BOP, plaque, and probing depths effectively. Ultrasonic curettage seems promising and is linked to the use of air polishing systems. Some studies suggest reduced bacteria and biofilm after mechanical curettage and improved results with ultrasonic methods. Air polishing results depend on the medium used, with hydroxyapatite/tricalcium phosphate being the best. Although abrasive air polishing can modify implant surfaces, it doesn't significantly affect cell attachment and viability but may reduce cell response. Re-osseointegration after air polishing therapy ranges from 39% to 46%, with improved implant attachment and reduced pocket depth. BOP can also be significantly reduced in peri-implantitis cases (35-37).

Effective antibiotic treatment for peri-implant infections relies on microbial information. Antibiotic choice (local or systemic) depends on the infection's depth. For instance, mechanical debridement with antiseptics like 0.2% chlorhexidine can work for shallow infections (pocket depth <4 mm), but deeper ones (pocket depth >5 mm) may require local drug delivery or systemic antibiotics like ornidazole, metronidazole, or a combination of metronidazole and amoxicillin for a 10-day duration. Specific antibiotic regimes are recommended for systemic treatment, especially for generalized infections. However, clinical trials
on systemic antibiotic therapy for peri-implantitis are needed (38).

**Laser therapy**

The potential of using dental lasers to promote radiographic bone regeneration appears promising, as the majority of studies have observed increased bone volume compared to initial levels or control groups. The following findings regarding the use of dental lasers for treating peri-implantitis are based on the findings of this systematic review: (1) laser therapy could potentially enhance bone regeneration in peri-implantitis-related defects; (2) laser treatment may lead to reductions in BOP and probing depths (PDs); and (3) laser-based peri-implantitis treatment might be as effective as, if not superior to, mechanical debridement or air abrasion methods (39).

**Photodynamic therapy**

Photodynamic therapy is a non-invasive method for effectively reducing microorganisms in peri-implantitis. One study involved 18 premolars from nine Labrador retriever dogs with dental implants (38, 40). Peri-implantitis was induced after osseointegration, followed by 4 months of plaque formation. The dogs were divided into two groups: one receiving conventional treatment involving scaling and chlorhexidine irrigation, and the other receiving photodynamic therapy (PDT) after scaling with a photosensitizer and low-power laser. Microbiological samples were taken before and after treatment. Both groups showed significant reductions in *Prevotella sp.*, *Fusobacterium sp.*, and beta-haemolytic streptococci. After treatment, there were no significant differences between the two groups. Another study utilized a broad-spectrum light radiator with a water-filtered spectrum between 580 and 1400 nm for irradiation (31, 41). They used Toluidine blue (TB) as a photosensitizer at various concentrations. The irradiance applied was 200 mW cm−2 for 1 minute. The study tested the effectiveness of antimicrobial photodynamic therapy (APDT) on planktonic cultures of *Streptococcus mutans* and *Enterococcus faecalis*, salivary bacteria harvested from human saliva, and initial bacterial colonization on enamel slabs carried in the mouths of six individuals (42). The results showed that APDT, in combination with TB and the specified light spectrum, was highly effective in killing *S. mutans*, *E. faecalis*, and salivary bacteria, with reductions of up to 5 log10. Even in the initial oral bacterial colonization, significant bacterial reduction was observed at all tested TB concentrations, despite individual variations among participants. This study suggests that APDT with TB and VIS+wIRA is a promising method for combating bacteria during the early stages of oral colonization, with potential applications in the treatment of peri-implantitis and periodontitis, considering the healing effects of VIS+wIRA on human tissue. While photodynamic therapy is currently in the experimental phase of development and testing, it has the potential to complement traditional antibacterial approaches in periodontology. Further clinical follow-up studies are necessary to validate the effectiveness of this procedure.

**Surgical treatment**

Surgical approaches for treating peri-implantitis can be categorized into two main methods: non-augmentative and augmentative therapy. Non-augmentative techniques like open flap debridement (OFD) and resective treatment are suitable when there is horizontal bone loss in areas where aesthetic appearance is not a primary concern. Additionally, implantoplasty performed alongside OFD at rough implant surfaces that are exposed above the crest and toward the outer side has been shown to effectively reduce soft tissue inflammation compared to control sites. However, this procedure is associated with more noticeable soft tissue recession. For peri-implantitis sites with intrabony defects, especially those with a minimum depth of 3 mm and when keratinized mucosa is present, adjunctive augmentative measures are recommended. In cases with more complex defect configurations, a combination of augmentative therapy and implantoplasty can be considered, particularly for exposed rough implant surfaces extending beyond the bony envelope (43) (Table 2).
### Table 2. Various therapies for mucositis and peri-implantitis, and their applications.

<table>
<thead>
<tr>
<th>Therapy</th>
<th>Use</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Surgical Cleaning &amp; Oral Hygiene</td>
<td>Effective in reducing peri-implant mucositis, but may not always fully resolve inflammation; use of antibiotics questioned due to potential antibiotic resistance risk</td>
<td>(32, 33)</td>
</tr>
<tr>
<td>Establishment of Adequate Oral Hygiene</td>
<td>Key for preventing peri-implant infections; includes regular evaluation of probing depths, professional implant cleaning, and oral hygiene training</td>
<td>(31)</td>
</tr>
<tr>
<td>Regenerative Strategies for Peri-implantitis</td>
<td>Aimed at resolving inflammation, reducing probing pocket depth, and stopping bone loss; treatment approach depends on defect characteristics; may lead to tissue recession</td>
<td>(34)</td>
</tr>
<tr>
<td>Implant Cleaning Methods</td>
<td>Various methods explored, including softer tip materials, piezoelectric scalers, hand instruments, and air polishing systems; may reduce bacteria and biofilm</td>
<td>(35-37)</td>
</tr>
<tr>
<td>Antibiotic Treatment</td>
<td>Choice (local or systemic) depends on infection depth; mechanical debridement with antiseptics may work for shallow infections, while deeper ones may require specific antibiotics; trials needed</td>
<td>(38)</td>
</tr>
<tr>
<td>Laser Therapy</td>
<td>Shows potential for enhancing bone regeneration, reducing bleeding on probing (BOP), and probing depths (PDs); may be as effective as mechanical debridement</td>
<td>(39)</td>
</tr>
<tr>
<td>Photodynamic Therapy (PDT)</td>
<td>Non-invasive method for reducing microorganisms in peri-implantitis; potential for killing bacteria during early stages of oral colonization; further clinical studies needed</td>
<td>(38, 40-42)</td>
</tr>
<tr>
<td>Surgical Treatment</td>
<td>Non-augmentative techniques (e.g., open flap debridement) and augmentative therapy for intrabony defects; implantoplasty can be considered for exposed rough implant surfaces</td>
<td>(43)</td>
</tr>
</tbody>
</table>

### Conclusion

Peri-implantitis, characterized by bone loss around dental implants, poses challenges for diagnosis and treatment. Diagnosis relies on measuring pocket depth, bleeding on probing, and molecular tests. Treatment ranges from non-surgical cleaning to regenerative approaches and surgery. Emerging technologies like laser and photodynamic therapy show promise. Overall, effective management of peri-implantitis requires a multifaceted approach combining traditional and innovative strategies to ensure long-term implant success and enhance patient care in implant dentistry.

### Disclosure

#### Conflict of interest

There is no conflict of interest.

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#### Ethical consideration

Non applicable

#### Data availability

Data that support the findings of this study are embedded within the manuscript.
Author contribution

All authors contributed to conceptualizing, data drafting, collection, and final writing of the manuscript.

References


