CONTEMPORARY APPROACHES IN PERI-IMPLANTITIS: FROM ETIOLOGY TO THERAPEUTIC PROTOCOL

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ABSTRACT

Introduction:Peri-implantitis is an inflammatory disease of the peri-implant tissues, associated with progressive loss of supporting bone around osseointegrated dental implants. With the increasing number of implant-prosthetic treatments, the prevalence of this condition has become a major concern in clinical dental practice. The etiopathogenesis of peri-implantitis is closely related to bacterial colonization of the implant surface, particularly by Gram-negative anaerobic species, through a mechanism comparable to that of periodontitis. Established risk factors include a history of periodontal disease, poor oral hygiene, smoking, uncontrolled diabetes mellitus, and occlusal overload.
Diagnosis is based on correlating clinical parameters—bleeding on probing, peri-implant pocket depth, and the presence of suppuration—with radiographic evidence of marginal crestal bone changes. Distinguishing peri-implant mucositis from peri-implantitis is essential for selecting an appropriate therapeutic strategy.
Sources: An electronic search was conducted using terms related to “peri-implantitis,” “therapeutic protocol,” and related concepts via PubMed and Google Scholar, prioritizing highly cited articles according to predefined inclusion and exclusion criteria, as well as review articles and the authors’ personal experiences. This review does not adhere to PRISMA guidelines nor does it include a formal risk-of-bias assessment, given its narrative format. The aim of the present study was to analyze the current literature on the treatment of peri-implantitis.

Conclusions: The current therapeutic armamentarium includes non-surgical methods (mechanical debridement, local antiseptics, systemic antibiotic therapy, Er:YAG laser treatment, photodynamic therapy) and surgical methods (resective surgery, regenerative surgery using bone grafts and membranes, implantoplasty). Recent literature supports the need for a sequential and individualized approach focused on implant surface decontamination and control of risk factors.
At present, there is no universally accepted therapeutic protocol; however, effective management of peri-implantitis requires a multidisciplinary approach and a rigorous long-term maintenance program.

Keywords: peri-implantitis, etiopathogenesis, bacterial biofilm, peri-implant bone loss, peri-implant mucositis, risk factors, implant surface decontamination, regenerative therapy, dental implants

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Introduction

Dental implants have become one of the most widely used and predictable options for replacing missing teeth worldwide, with substantial benefits for masticatory function, aesthetics, and overall quality of life. As the number of implants placed each year continues to rise and clinical indications expand, the incidence of implant-related biological complications has also increased. Among these, peri-implantitis is of particular clinical relevance due to its potential to compromise peri-implant tissue stability and jeopardize the long-term success of implant therapy.

Peri-implantitis is an irreversible inflammatory disease of the tissues around an implant that results in progressive peri-implant bone loss, increasing probing depths, and may present with suppuration.[1-3]

The differentiation of peri-implantitis from other inflammatory periodontal conditions cannot be reliably established through the analysis of human salivary biomarkers, including osteocalcin, tartrate-resistant acid phosphatase (TRAP), Dickkopf-related protein-1 (DKK-1), osteoprotegerin (OPG), and cathepsin K (CatK). These markers, despite their established roles in bone metabolism and remodeling, demonstrate insufficient discriminatory capacity when assessed within the salivary medium to distinguish peri-implant inflammatory processes from those associated with periodontal disease.[4,5]

Epidemiological evidence consistently demonstrates a significantly elevated prevalence of periodontitis among individuals with poorly controlled diabetes mellitus compared to systemically healthy controls. This established bidirectional association has prompted scholars to propose periodontitis as a distinct diabetes-related complication, leading to its conceptual designation as the **"sixth complication"** of diabetes mellitus, alongside the classical micro- and macrovascular manifestations of the disease.[6]

Periodontal treatment has recently been shown to potentially improve metabolic control in diabetes, though long-term data are still limited. Conversely, uncontrolled diabetes may adversely affect the response to periodontal therapy and increase the risk of peri-implant diseases. The mechanisms linking diabetes mellitus and periodontal disease include the release of advanced glycation end products secondary to hyperglycemia, along with a spectrum of shared predisposing factors of genetic, microbial, and lifestyle nature. Current evidence indicates an increased risk of periodontal and peri-implant diseases in individuals with diabetes and highlights a role for dental professionals at the diabetes–periodontal interface.[7]

Historically, it has been widely accepted that osseointegration reflects a homeostatic balance between a titanium dental implant and adjacent bone, and that the crestal bone loss observed in peri-implantitis arises from an

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inflammatory process driven by dental plaque.[8]

Since the primary causes of implant failure are peri-implant bone resorption and incomplete osseointegration, research teams and implantologists are focused on clinical and paraclinical studies that evaluate peri-implant bone changes and the success/failure rates of implant systems made from different materials and featuring various macro- and micro-designs and geometries. [9,10]

In this context, this review aims to synthesize current evidence on the definition , etiology and risk factors, as well as preventive strategies and contemporary therapeutic options for peri-implantitis. A clear understanding of these aspects is essential both for reducing disease onset and for optimizing clinical management, thereby supporting the long-term success of dental implants.

Peri-implantitis – Definition, Etiology, and Risk Factors

Definition. Peri-implantitis is a biofilm-induced inflammatory disease affecting the peri-implant tissues, characterized by inflammation of the peri-implant mucosa accompanied by progressive loss of the supporting bone around a dental implant. Clinically, it presents with bleeding on probing and/or suppuration, increased probing depths compared with previous recordings, and radiographic evidence of marginal bone loss. In the absence of baseline data, the diagnosis is based on the presence of bleeding and/or suppuration on probing, increased probing depth (typically ≥6 mm), and marginal bone loss of approximately ≥3 mm relative to the expected bone level following initial post-implant remodeling. These diagnostic criteria were consolidated by the 2017 World Workshop consensus, which standardized the terminology and clinico-radiographic thresholds for peri-implant diseases.[11]

Gram-negative anaerobes and asaccharolytic anaerobic Gram-positive bacilli dominate this heterogeneous mixed infection, though opportunistic microbes like enteric rods and *Staphylococcus aureus* appear in rare cases.[12]

In this regard, Francetti et al. [13] used as evidence for the diagnosis of PI the presence of bleeding/suppuration and a concomitant bone resorption process of 2 mm or more observed radiographically, without considering peri-implant probing depth.

Etiology. The etiology of peri-implantitis is predominantly infectious, arising from the accumulation and maturation of microbial biofilm on implant surfaces, which triggers a host inflammatory response. Experimental and clinical evidence indicates that peri-implant inflammation resembles, but is not identical to, periodontal inflammation; lesion development and progression are influenced by the distinctive characteristics of peri-implant tissues (e.g., connective tissue fiber orientation, vascular supply, and the

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architecture of the implant–mucosa interface). Peri-implantitis is therefore regarded as a multifactorial disease in which biofilm dysbiosis interacts with host susceptibility and local iatrogenic factors (e.g., residual cement, inadequate prosthetic contours, and limited access for oral hygiene), resulting in persistent inflammation and progressive bone resorption.[14]

Histologically, peri-implantitis lesions exhibit a larger size and a greater number of blood vessels, as well as a more extensive connective-tissue infiltrate, compared with periodontitis. The rate of disease progression is faster in peri-implantitis, resulting in more rapid and severe bone loss than in periodontal disease. In peri-implantitis, a non-linear pattern of progressive bone destruction occurs, which may be attributable to the diverse microorganisms present at implant sites, host defense mechanisms, and the absence of a periodontal ligament.[15]

It has been reported that the periodontopathogenic bacteria associated with periodontitis around natural teeth differ from those present in peri-implantitis. *Staphylococcus* species (including enteric strains) and *Candida* spp. have been identified in 55% of peri-implant lesions. In a microbiological comparison between healthy implants and peri-implantitis sites, 19 species were found at higher counts in peri-implantitis, suggesting that a specific bacterial consortium may be associated with its onset. Thus, bacterial loads of *Tannerella forsythia, Treponema denticola, Porphyromonas gingivalis, Staphylococcus aureus, Campylobacter rectus, Campylobacter gracilis, and Prevotella intermedia* were significantly higher, in terms of odds ratios, in subjects with peri-implantitis compared with unaffected implants.[16]

Prevalence

Derks and Tomasi[17] reported a peri-implantitis prevalence ranging from 1% to 47% among subjects treated with implant-supported prostheses. Francetti et al. [13] evaluated a total of 384 implants placed in 77 patients over a mean follow-up period of eight years after loading; at 10 years, the cumulative rate of implants free of peri-implantitis was 86.92%. The reported prevalence figures for peri-implantitis vary widely because they depend on multiple factors, including time in function, patient-related conditions and associated risk factors, as well as differences in diagnostic definitions adopted by investigators.

A.Systemic risk factors

a. Diabetes mellitus. Hyperglycemia may be a potentially important factor in the development of biological complications of implants, as observed in poorly controlled diabetes.Thus, healing processes can be negatively affected by elevated blood glucose levels, causing vascularization alterations, bone remodeling changes, and increased susceptibility to infections, which leads to the conclusion that hyperglycemia has adverse effects on implant integration [18, 19].

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In contrast, the systematic review by Monje et al. [18]found that no study reported the prevalence of peri-implant diseases in patients with hyperglycemia in the absence of other risk factors such as smoking, recent history of periodontitis, or inadequate biofilm control.

b. Smoking. Nicotine has been reported to promote microvascular obstruction, diminish perfusion and nutrient delivery at the surgical implant site, and suppress the proliferation of fibroblasts, erythrocytes, and macrophages, ultimately impairing bone regeneration. Although nicotine exposure in the setting of dental implant surgery is considered minimal, it has been hypothesized that early implant failure may be partly attributable to its vasoconstrictive action. [20]

Smoking significantly affects both innate and adaptive immunity. In smokers, an increase in granulocyte and total leukocyte counts is observed, along with prolonged survival of polymorphonuclear cells, increased production of hydrogen peroxide and protease inhibitors, and altered integrin expression. Smoking also disrupts humoral immune responses by inhibiting the proliferation or function of B and T lymphocytes [21].

In patients diagnosed with peri-implantitis, comparisons between smokers and non-smokers indicated that non-smokers achieved better long-term results after 12 months of post-treatment follow-up [20].

Local risk factors

The 2017 classification identifies several local factors that increase the risk of peri-implantitis, including a prior history of periodontitis, inadequate control of dental biofilm, and failure to attend scheduled post-implant maintenance appointments [22].

The presence of keratinized mucosa (KM) around the implant is considered a protective factor; conversely, its absence is associated with increased risk . [23]

a.A prior history of periodontitis is of particular clinical significance, as the current body of literature consistently reports marked similarities between periodontal and peri-implant diseases with respect to etiology, pathogenesis, risk profile, and clinical expression. Both entities are predominantly biofilm-induced inflammatory conditions, initiated by a dysbiotic microbial community and mediated by an aberrant or heightened host immune–inflammatory response, culminating in the progressive destruction of the supporting tissues (alveolar bone in periodontitis and peri-implant bone in peri-implantitis). Moreover, they share a range of well-established risk determinants and modifiers—most notably inadequate plaque control, previous periodontal disease experience, tobacco use, and poorly controlled diabetes—along with additional local and systemic factors that influence susceptibility and disease progression. From a clinical standpoint, the two conditions exhibit comparable signs of inflammation and tissue breakdown, including bleeding on probing, increased probing depths, possible suppuration,

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and radiographic evidence of marginal bone loss.

An association between periodontitis and peri-implantitis has been reported [24], the study by Ronk et al. [25] yielded contrasting findings. In a cohort of 134 patients receiving a total of 478 implants over a 10-year period (2001–2010), the authors observed that, after five years of functional loading and in the absence of any structured maintenance program, approximately one in five patients would develop peri-implantitis. Accordingly, they concluded that neither the presence of periodontitis nor a prior history of periodontitis constituted statistically significant predictors of peri-implantitis.

b. **Keratinized mucosa surrounding the implant**. Around implants, collagen fibers are chiefly arranged parallel to the implant surface and do not insert directly into it; this limited connective-tissue attachment produces a less effective biological seal. As a result, peri-implant soft tissues exhibit greater vulnerability to inflammatory disease than the soft tissues associated with natural teeth [26].

In their cross-sectional study, Rinke et al. [27] assessed whether risk indicators commonly implicated in peri-implant diseases were associated with peri-implant mucositis and peri-implantitis in patients undergoing implant therapy (IT), at least five years after completion of the implant restoration. Their findings indicated that smoking and the absence of keratinized mucosa (KM) were the strongest risk indicators for peri-implantitis in patients treated with IT.

Furthermore, Gharpure et al. [26] investigated, in 73 patients with 193 implants (mean follow-up: 6.9 ± 3.7 years), the relationship between a thin gingival phenotype (TGP) and a keratinized mucosa width (KMW) < 2 mm as potential risk indicators for peri-implantitis and mucositis. The authors concluded that a thin gingival phenotype and an inadequate KMW (< 2 mm) may represent significant risk indicators for peri-implant disease, together with pain or discomfort during toothbrushing.

Therapeutic Protocol

Dental implant failure is frequently associated with compromised osseointegration. An implant is considered failed when it is malpositioned, exhibits mobility, or shows peri-implant bone loss exceeding 1.0 mm during the first year and 0.2 mm annually thereafter. Peri-implantitis may lead to progressive peri-implant bone resorption and, ultimately, implant loss [28].

The perioperative prescription of oral antibiotics has been widely used to reduce the risk of postoperative wound infection, early implant failure, and the subsequent development of peri-implantitis. However, current scientific evidence does not support the routine perioperative use of oral antibiotics to prevent these complications.[29,30].

The prosthodontist bears substantial responsibility in preventing peri-implant problems by ensuring that implants are restored in a manner that integrates

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biological and biomechanical considerations. When properly balanced, this approach optimizes occlusal forces, enables the patient to perform effective self-care, and permits routine assessment of peri-implant conditions through probing. Accordingly, delivering a reconstruction that impedes plaque removal or monitoring of peri-implant conditions should be regarded as a major error.

There is a growing need to recognize and understand how the implant restorative process may adversely affect peri-implant tissues, to identify strategies to minimize these effects, and to promote and maintain peri-implant health through meticulous attention to detail at every stage of treatment.[31]

It has been demonstrated that certain emergence profiles and prosthetic designs are more prone to plaque and debris accumulation.[32] Consequently, biofilms accumulate and may initiate peri-implant disease. [33]

In the context of the IDRA, the term “cleanable” refers to the ease of access for cleaning the prosthesis by both the clinician and the patient. The patient’s ability to clean the prosthesis depends on their level of skill and dexterity, which may evolve over time [34].Most published strategies for peri-implantitis management are largely derived from therapeutic approaches used in periodontitis. This is because bacterial colonization of tooth and implant surfaces follows similar principles, and it is widely accepted that the microbial biofilm plays an analogous role in the development of peri-implant inflammation [35].

Igiena orală, în general, este precară în cazurile de apariție a periimplantitei, iar un studiu publicat în 2018 la nivelul județului Iași indică…

Overall, oral hygiene is generally poor in cases where peri-implantitis develops, and a study published in 2018 at the level of Iași County indicate that the use of oral hygiene products, rural residents reported using toothpaste, mouthwash, and dental floss at rates of 94.0%, 18.9%, and 3.9%, respectively, compared with 97.2%, 41.4%, and 11.0% among urban residents.

In terms of oral hygiene behaviors, twice-daily toothbrushing was reported by 50.9% of urban respondents and 42.1% of rural respondents. In Iași County, 22.8% of individuals in rural areas indicated replacing their toothbrush annually, while 31.6% of those in urban areas reported changing it four times per year.[49]

Conservative therapy. In addition to adjunctive pharmacotherapy and conventional mechanical debridement (e.g., using curettes, ultrasonic devices, and air-polishing systems), newer minimally invasive approaches have been described as conservative treatment options. These include laser-assisted modalities and antimicrobial photodynamic therapy, which aim to enhance decontamination of the implant surface and support control of peri-implant inflammation.[36]

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Because the inflammatory lesion in peri-implant mucositis is confined to the peri-implant mucosa, non-surgical management is generally considered appropriate. In particular, professional mechanical plaque removal—performed with hand instruments and/or ultrasonic devices—has been shown to be the most effective approach for reducing clinical signs of inflammation.

The non-surgical approach aims to control peri-implant soft-tissue infection and to achieve debridement of the implant surface. Accordingly, various protocols have been described, incorporating mechanical debridement with curettes, ultrasonic instrumentation, air-abrasive systems, and laser-based modalities (including photodynamic therapy), with or without adjunctive administration of antibiotics or antiseptics.

Figuero et al. reviewed these protocols and concluded that, although many interventions produced a measurable short-term reduction in bleeding on probing (BoP), their effect on probing depth was limited.[37]

In a review by Javed et al. encompassing nine studies, both systemic and local antibiotic therapies (e.g., tetracycline, doxycycline, amoxicillin, metronidazole, minocycline hydrochloride, ciprofloxacin, and sulfonamides combined with trimethoprim) were associated with significant reductions in pocket depth over follow-up periods ranging from one to six years.[38]

Owing to their bactericidal mechanism of action, CO2, diode, Er:YAG (erbium-doped: yttrium-aluminum-garnet), and Er,Cr:YSGG (erbium, chromium-doped: yttrium-scandium-gallium-garnet) lasers are being used with increasing frequency in the management of peri-implant diseases. To safeguard the implant surface and surrounding tissues, minimal absorption and limited reverberation must be ensured. Er:YAG and Er,Cr:YAG lasers, operating at a wavelength of approximately 3 microns, have been reported to reduce biofilm levels by up to 90%; however, unlike most mechanical therapies, they do not appear to re-induce biological compatibility or cell-stimulatory properties. [39-41]

Photodynamic therapy (PDT) has gained attention as a valuable adjunctive modality, supported by its combined antimicrobial and anti-inflammatory effects. By pairing a photosensitizing agent with illumination at a specific wavelength, PDT induces the formation of reactive oxygen species that exert bactericidal activity and influence inflammatory signaling pathways [50,51] Recent evidence suggests that PDT may improve clinical parameters—such as bleeding on probing (BOP), probing depth (PD), and plaque index (PI)—and favorably modify peri-implant crevicular fluid biomarkers, including interleukin-1β (IL-1β) and tumor necrosis factor-alpha (TNF-α)[52-58]

Some evidence suggests that males may be at increased risk of peri-implantitis, possibly reflecting sex-related differences in oral hygiene behaviors

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and/or a higher prevalence of systemic comorbidities, such as cardiovascular disease.[59,60,71]]

Some studies have reported favorable outcomes with the combined use of Er:YAG lasers, attributed to their precise ablation and limited thermal injury,[61,62] while also highlighting the advantages of Nd:YAG lasers in terms of deeper tissue penetration.[63] The development of standardized protocols is essential to enhance research consistency, reproducibility, and clinical translation. Future investigations should focus on establishing evidence-based guidelines for laser therapy in peri-implantitis, taking into account laser type, energy parameters, and treatment intervals. [64] Growing attention has been directed toward this approach given its advantages relative to conventional therapies, including pronounced bactericidal effects and the ability to decontaminate implant surfaces effectively while preserving surface integrity.[65-67]

Systemic conditions (including diabetes, cardiovascular disease and smoking) have a significant impact on peri-implantitis progression and on treatment outcomes.[68,69] Independent of the therapeutic approach, smoking is associated with lower peri-implantitis treatment success because it impairs wound healing and bone regeneration.[70]

Laser therapy shows substantial promise for peri-implantitis management; however, methodological limitations, protocol standardization, and evaluation of demographic and systemic modifiers are required to confirm its reliability and long-term efficacy. Future studies should also harmonize success criteria, clarify durability of outcomes, and account for regional practice variability to support robust, evidence-based clinical guidelines.

The surgical approach is widely regarded as effective for implant-surface debridement and decontamination because it provides direct access and enhanced visualization of contaminated areas, which is particularly important for implants with threaded geometry and diverse surface modifications. By elevating a mucoperiosteal flap and exposing the implant circumferentially, the clinician can more predictably remove granulation tissue and disrupt biofilm from the entire affected surface, including the thread valleys, undercuts, and other anatomically sheltered regions that are difficult or impossible to instrument adequately with nonsurgical methods.

Another widely endorsed concept, as proposed by Zitzmann et al., is grounded in a systematic approach to periodontal therapy. In the initial phase, emphasis is placed on optimizing oral-hygiene conditions, accompanied by mechanical debridement and, when indicated, adjunctive local anti-infective measures. Should nonsurgical management prove insufficient, surgical treatment is advised, typically involving open debridement and, depending on the clinical scenario, either resective or regenerative procedures.[42]

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Resective surgical therapy represents a recommended treatment modality for peri-implantitis. Procedures such as ostectomy and osteoplasty, when combined with implantoplasty, have been shown to be effective in reducing disease activity and may arrest further progression. However, because this approach is frequently associated with increased postoperative soft-tissue recession, its indication should be carefully considered, and it is generally less appropriate in cases where esthetic demands are high, particularly within the anterior esthetic zone. [43]

Multiple studies have advocated regenerative surgical approaches for the management of peri-implantitis–associated bone loss and defect morphology, with the primary aim of limiting or preventing soft-tissue recession. In general, these protocols involve the placement of grafting materials—most commonly autogenous bone—used either alone or in combination with barrier membranes to support guided bone regeneration. [44-46]

Chan et al., in their systematic review and meta-analysis, reported that the use of grafting materials in conjunction with barrier membranes is associated with greater reductions in probing pocket depth and increased radiographic bone fill.[47]

The morphology of the peri-implant bone defect surrounding an infected implant is a major determinant of the predictability and overall success of regenerative therapy. In general, regenerative interventions yield superior outcomes in circumferential (contained) or predominantly intrabony defects, as these configurations provide bony walls that help maintain space, stabilize the graft and blood clot, and limit soft-tissue collapse into the defect during healing. Such containment enhances wound stability—an essential prerequisite for bone fill and re-osseointegration to occur.[48]

Conclusion

The available literature indicates that microbial biofilm is the primary etiological factor in peri-implant disease. Nevertheless, the onset, progression, and clinical severity of peri-implant pathology appear to be modulated by a range of predisposing and modifying factors. Among the most frequently reported are systemic metabolic disorders such as diabetes mellitus, which may impair host immune function and wound healing; tobacco use, which adversely affects vascularity and inflammatory responses; and a history of pre-existing periodontal disease, which is associated with increased susceptibility to peri-implant infection and a higher likelihood of colonization by periodontopathogenic microorganisms. Consequently, peri-implant disease should be viewed as a biofilm-induced condition whose clinical expression is strongly influenced by patient-related risk factors and baseline periodontal status. More long-term, high-quality studies are needed to strengthen evidence-based decisions and to make implant therapy more predictable over time.

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