

Peri-implant Diseases

A Review of Treatment Interventions



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KEYWORDS

- Guided bone regeneration • Laser • Nonsurgical • Peri-implantitis
- Peri-implant mucositis • Surgical • Treatment

KEY POINTS

- Risk factors of peri-implant mucositis and peri-implantitis are comparable to those of gingivitis and periodontitis.
- The ideal management of peri-implant diseases focuses on infection control, detoxification of implant surfaces, regeneration of lost tissues, and plaque control regimens via mechanical debridement.
- Implantoplasty (modification in implant surface topography), when used in combination with resective surgery, has been reported to significantly reduce the clinical parameters of peri-implantitis.
- A new technique, laser-assisted peri-implantitis protocol, is under investigation.
- There is lack of standardized treatment protocols for peri-implant disease.

Several studies^{1–6} have reported that dental implants are functionally stable and have long-term success rates, and are therefore increasingly being used in the oral rehabilitation of partially and completely edentulous individuals. However, with the increasing number of patients receiving dental implants, the prevalence of inflammatory conditions around a dental implant has also escalated.⁷ The consensus report from the 6th European Workshop on Periodontology has defined peri-implantitis as the presence of inflammation of the peri-implant mucosa and simultaneously loss of supporting bone.⁸ In addition, it has also been described as a site-specific infection that

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exhibits features comparable to those of chronic adult periodontitis.⁹ In indexed literature, there is a variance in the prevalence of peri-implantitis. Mombelli and colleagues¹⁰ estimated the prevalence of peri-implantitis to be in the order of 10% implants and 20% patients during 5 to 10 years after implant placement; and Koldstad and colleagues¹¹ reported the prevalence to range between 11.3% and 47.1% in their study population. Moreover, in a recent study on Belgian adults, the prevalence of peri-implant mucositis and peri-implantitis was 31% and 37%, respectively.¹² It has been suggested that peri-implant mucositis and peri-implantitis are analogous to gingivitis and periodontitis, respectively.¹³ However, it is pertinent to mention that biologic differences exist between natural teeth and implants. Therefore, the progression of infection around implants and natural teeth is also divergent. Moreover, tissues around implants are more prone to plaque-associated infections that spread into the alveolar bone.¹⁴

The ideal management of peri-implant diseases focuses on infection control, detoxification of implant surfaces, regeneration of lost tissues, and plaque-control regimens via mechanical debridement (with or without raising a surgical flap).^{15,16} However, a variety of other therapeutic modalities also have been proposed for the management of peri-implantitis. These treatment strategies encompass use of antiseptics and/or antibiotics, laser therapy, guided bone regeneration, and photodynamic therapy.¹⁶⁻²³ The aim of this article was to review indexed literature with reference to the various therapeutic interventions proposed for the management of peri-implant diseases.

PERI-IMPLANT DISEASES: PERI-IMPLANT MUCOSITIS AND PERI-IMPLANTITIS

Peri-implant diseases are categorized into 2 types: peri-implant mucositis and peri-implantitis. Peri-implant mucositis is characterized by inflammation of the soft tissues surrounding the implant without any signs of bone loss (**Fig. 1**).⁸ The clinical signs of peri-implant mucositis include bleeding on probing (BOP) and/or suppuration, which are usually associated with probing depth (PD) of at least 4 mm with no evidence of radiographic loss of bone.^{24,25} It has been reported that inflammatory cell lesions in sites with peri-implant mucositis are dominated by T cells and have an apical extension that is restricted to the barrier epithelium.²⁶ Peri-implant mucositis is usually reversible (when early diagnosis and removal of etiology are implemented)^{27,28}; however, it is considered as a precursor to peri-implantitis.⁸

The term “peri-implantitis” was introduced in literature more than 3 decades ago.⁹ This term was modified in the 1990s to describe an inflammatory process around an implant that includes both soft tissue inflammation and progressive loss of supporting bone beyond biological remodeling (**Figs. 2 and 3**). In general, the clinical definition of



Fig. 1. Clinical presentation of peri-implant mucositis (yellow arrows).



Fig. 2. Clinical presentation of peri-implantitis with extensive calculus deposition (*arrow*) around dental implants.

peri-implantitis has varied among studies. For example, in studies by Roos-Jansåker and colleagues,^{29,30} peri-implantitis was described as a condition in which implants with varying degrees of bone loss are accompanied by a PD of at least 4 mm, BOP, and purulent discharge on gentle probing. However, in a systematic review by Berglundh and colleagues,³¹ peri-implantitis was defined by PD of more than 6 mm and in combination with BOP and clinical attachment loss or bone loss of at least 2.5 mm (**Fig. 4**).

RISK FACTORS OF PERI-IMPLANT DISEASES

Risk factors of peri-implant mucositis and peri-implantitis are comparable to those of gingivitis and periodontitis.^{27,32} The following risk factors have been associated with the etiology of peri-implant diseases.

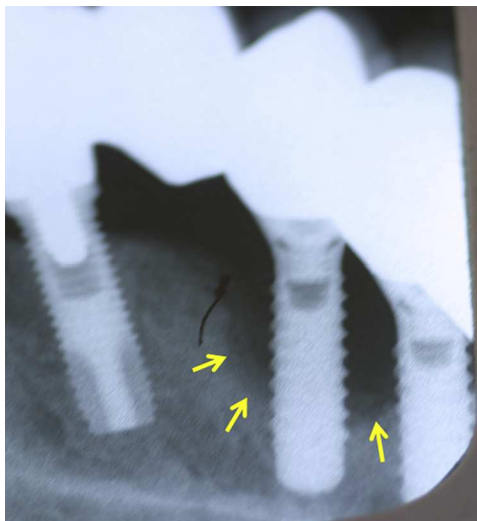


Fig. 3. A peri-apical radiograph showing crestal bone (*yellow arrows*) loss around dental implants.

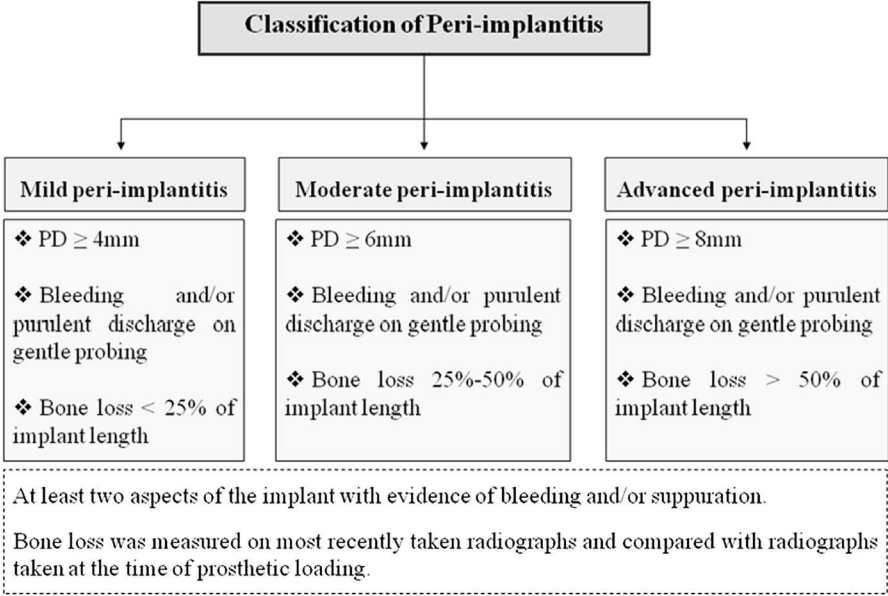


Fig. 4. Classification of peri-implantitis. (Modified from Froum SJ, Rosen PS. A proposed classification for peri-implantitis. *Int J Periodontics Restorative Dent* 2012;32:533–40.)

Poor Plaque Control

Implant geometry and surface characteristics may obligate patients to adopt non-conventional plaque-control regimens, such as interdental brushing and flossing in addition to traditional tooth-brushing techniques. The primary focus of these nonconventional plaque-control protocols is to maintain the periodontal health of the remaining dentition, thereby minimizing the possibilities of plaque accumulation and formation of periodontal and/or peri-implant pockets. However, it may be challenging for some patients to get accustomed to nonconventional plaque-control regimens (as stated previously) without ado. This may in turn compromise their ability in executing plaque control (see Fig. 2). In this regard, it is imperative for dental health care providers to educate and regularly monitor their patients (through routine check-ups) to ensure proper plaque control. This would in turn help patients in maintaining their periodontal and/or peri-implant health.³³

Previous History of Periodontal Disease

It has been claimed that peri-implantitis is a common finding in patients with a history of periodontitis.^{34,35} Results from a systematic review and meta-analysis showed that the relative risk for peri-implantitis was significantly higher in patients with a previous history of periodontitis compared with patients without a history of periodontal disease.³⁴ However, in a recent study, Meyle and colleagues³⁶ investigated the long-term clinical and radiographic parameters of osseointegrated implants in nonsmoking patients with a previous history of chronic periodontitis. The results showed that patients with a previous history of periodontitis regularly attending an oral hygiene maintenance program displayed implant survival rates up to 100% after 5 and 10 years. Similarly, in a systematic review, Pesce and colleagues³⁷ concluded that there is a lack of consensus regarding the role of periodontitis in the etiology of peri-implantitis.

Nevertheless, because several periodontopathogens (such as *Aggregatibacter actinomycetemcomitans*, *Prevotella intermedia*, and *Porphyromonas gingivalis*) associated with the etiology of periodontitis also have been isolated from peri-implant sulci of patients with peri-implantitis,^{38–41} it is arduous to disregard the hypothesis that peri-implantitis is more common in patients with a history of periodontitis.

Stagnation of Residual Material in/Around the Gingivae Following Implant Prosthesis Cementation

A conventional approach toward restoration of dental implants using fixed prosthesis is the use of cement-retained restorations. In the absence of occlusal screw access openings, cement-retained restorations are useful in enhancing the number of occlusal contacts and simultaneously improving aesthetics.⁴² However, inadequate removal of excessive cement at the time of implant cementation may lead to a complication: cement-induced peri-implantitis.⁴² The probability of cement to remain in the peri-implant sulcus is high when margins of the restoration are placed 1.5 to 3 mm subgingivally.⁴³ Stagnation of residual cement in peri-implant sulcus has been associated with peri-implant tissue inflammation, BOP and/or suppuration, increased PD, evidence of bone loss on radiographs, and patient discomfort.⁴⁴ Shallow intracrevicular placement of the gingival margins reduces the occurrence of peri-implant inflammation and related complications.

Occlusal Overloading

Occlusal overloading is a major cause of biomechanical implant complications, including fracture and/or loosening of the implant (Fig. 5). Occlusal overloading in combination with plaque accumulation may also disturb the intricate bond between the implant surface and bone leading to peri-implantitis and, if left untreated, implant failure.^{45–47} However, conflicting results also have been reported.^{48,49} In a study on

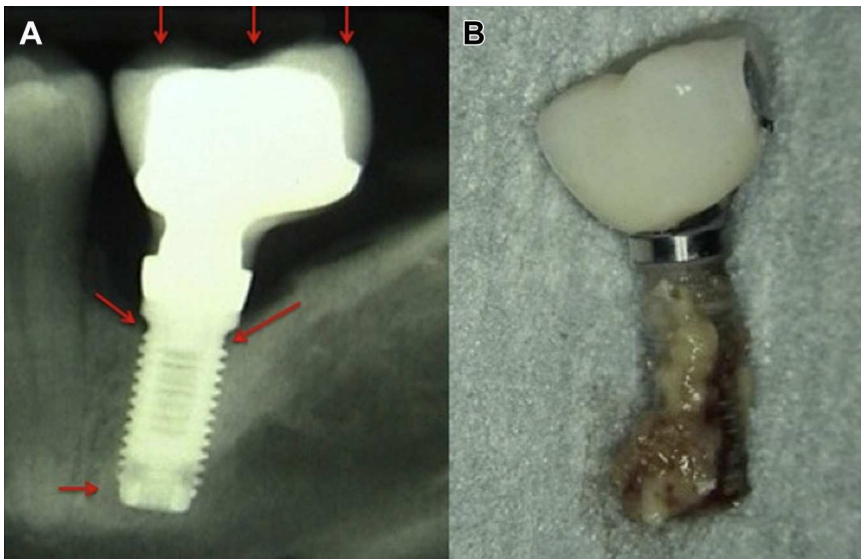


Fig. 5. Implant failure due to inflammation and occlusal overload. (A) Red arrows are showing the crown-implant relation and the bone loss. (B) The extracted implant surrounded by inflammatory tissue and plaque.

dogs, there was no loss of osseointegration and/or peri-implantitis following a period of 8 months of excessive occlusal load on titanium (Ti) implants.⁴⁹ Occlusal overload may be prevented by performing comprehensive examinations, treatment planning, well-defined surgical and prosthetic treatments, and regular maintenance. However, Pesce and colleagues³⁷ reported in a recent systematic review that there is a lack of clinical evidence to support a cause-effect relationship between peri-implantitis and occlusal overload.

Habitual Tobacco Smoking

Tobacco smoking is a significant risk factor for periodontitis and peri-implant diseases,^{50–53} and pathogenesis of periodontitis closely resembles that of peri-implantitis.⁵⁴ Tobacco smoking has been associated with an increased expression of receptor of advanced glycation end products (RAGE) in gingival tissues.^{53,55} Experimental results by Katz and colleagues⁵⁵ showed an increased expression of RAGE in gingival epithelial cells of smokers as compared with nonsmokers. Moreover, it also has been reported that nornicotine (a metabolite of nicotine) upregulates RAGE expression in the gingivae of smokers. This stimulates the secretion of cytokines and reactive oxygen species, which enhance alveolar bone loss.^{55,56} Furthermore, there is a synergistic effect of tobacco smoking and carriage of interleukin (IL)-1 gene polymorphism that results in increased risk of peri-implantitis.^{57,58}

Galindo-Moreno and colleagues⁵⁹ reported that the rates of marginal bone loss around implants are significantly associated with smoking. Studies^{60–64} have reported that rates of implant failure range between 6.5% and 20.0% among smokers compared with nonsmokers. In smokers, implant failures occur more often in the maxilla as compared with implants placed in the mandible^{61,63,65}; most likely due to the presence of poor-quality trabecular bone in the maxilla.⁶⁶ Results from a recent systematic review and meta-analysis also reported a significantly higher risk of peri-implantitis in smokers as compared with nonsmokers.⁶⁷

Diabetes

It is well acknowledged that periodontal inflammatory conditions are worse in patients with poorly controlled diabetes as compared with systemically healthy individuals.^{4,50,68,69} This is primarily because chronic hyperglycemia impairs tissue repair and host defense mechanisms. In addition, an increased formation and accumulation of advanced glycation end-products in the periodontal tissues raises cellular oxidative stress and increases the production of proinflammatory cytokines (such as IL-6, IL-1 β , IL-18, matrix metalloproteinase [MMP]-8 and MMP-9) in the serum, saliva, and gingival crevicular fluid of patients with chronic hyperglycemia.^{68,70–73} These proinflammatory cytokines may jeopardize the osseointegration and long-term survival of implants in patients with chronic hyperglycemia. However, it is pertinent to mention that degree of glycemic control (in patients previously diagnosed with diabetes) may play a role in a successful osseointegration and survival of implants in diabetic patients.⁴

Genetic Factors

A correlation between IL-1 gene polymorphism and peri-implantitis has been reported by some studies.^{74–76} However, in a systematic review, Dereka and colleagues⁷⁷ reported no apparent association between specific genetic polymorphism and dental implant failure in terms of biological complications (peri-implant diseases). Hultin and colleagues,⁷⁸ Rogers and colleagues,⁷⁹ and Campos and colleagues⁸⁰ also reported no association between gene polymorphism and peri-implantitis. Although the

association of genetic factors with peri-implantitis cannot be completely disregarded, further studies are warranted to assess the role of genetics as a potential risk factor of peri-implantitis.

INCIDENCE AND PREVALENCE OF PERI-IMPLANT DISEASES

There is a lack of information regarding the incidence of peri-implant mucositis and peri-implantitis, as the interpretation of data is difficult. In this regard, the incidence of peri-implantitis may be underestimated. Renvert and colleagues⁸¹ investigated the incidence of peri-implantitis over a period of 13 years between 2 dental implant systems (TiOblast AstraTech [AT] and machine-etched Brånemark Nobel Biocare [NB] system, Gothenburg, Sweden, respectively). In this study,⁸¹ the incidences of peri-implantitis for AT implants were 26.2% and 7.1% between years 1 and 7; and 30.4% and 11.5% for NB implants between years 7 and 13. There was no significant difference in the incidence of peri-implantitis over a period of 13 years between the 2 implant systems.⁸¹ The investigators considered a history of periodontitis as a risk for future incidence of peri-implantitis.⁸¹ The consensus report of the 7th European Workshop on Periodontology⁸² also emphasized that there is a need to develop new and more efficient preventive and treatment approaches for periodontal disease. This report⁸² also accentuated such preventive and treatment protocols should be based on recent advances in understanding of host modulation and inflammation resolution with direct management of the microbiota.

To our knowledge, only a limited number of studies have assessed the prevalence of peri-implant disease.^{83–88} Moreover, we observed that the prevalence reported in these studies varied between patients and implant systems. For example, in the 5-year follow-up results by Fransson and colleagues,⁸⁸ the prevalence of peri-implant diseases was reported to be 92%, whereas in the study by Scheller and colleagues,⁸³ the prevalence of peri-implant diseases at 5-year follow-up was 24%. Zitzmann and Berglundh⁸ reported the prevalence of peri-implantitis to vary between 12% and 43% of implants. This variation may be associated with a lack of standardization in the patient selection and criteria for diagnosing peri-implant mucositis and peri-implantitis among these studies. Therefore, a precise estimation of the prevalence of peri-implant disease remains debatable.

CLASSIFICATION OF PERI-IMPLANTITIS

To our knowledge from indexed literature, there is no globally accepted classification of peri-implantitis. However, Froum and Rosen⁸⁹ recently proposed a classification that differentiated peri-implantitis according the severity of inflammation and extent of bone loss around the implant. This classification was based on 3 different clinical stages of peri-implantitis: (1) early peri-implantitis, (2) moderate peri-implantitis, and (3) advanced peri-implantitis (see [Fig. 4](#)).⁸⁹ Early peri-implantitis was defined by PD of at least 4 mm and radiographic bone loss of less than 25% of the total implant length. Moderate peri-implantitis was defined by PD of at least 6 mm and radiographic bone loss of 25% to 50% of the total implant length, whereas advanced peri-implantitis was defined by PD of at least 8 mm with more than 50% of radiographic bone loss with reference to implant length. In this classification, BOP and/or suppuration on 2 or more aspects of the implant were selected as the markers of inflammation. The investigators conjectured that their proposed classification would help strengthen communication between clinical practitioners and researchers and help attain a better understanding of peri-implantitis⁸⁹; however, further evidence-based clinical studies are needed to authenticate this classification.

ASSESSMENT OF END POINTS AMONG STUDIES ON THE TREATMENT OF PERI-IMPLANTITIS

End points are commonly used to determine the effectiveness of a clinical treatment. In studies addressing the treatment of peri-implantitis, “true end points” are preferred over “surrogate end points,” because they are able to depict a considerable proportion of the effect of treatment on an outcome of interest, such as implant failure.^{90–92} In a systematic review, Faggion and colleagues⁹¹ explored indexed literature to determine the type of end points reported regarding peri-implantitis therapy and their frequency of use. Studies included in this systematic review⁹¹ chiefly reported implant failure as a consequence of peri-implantitis therapy and not as an objective of an investigation. The investigators concluded that there is a lack of consensus regarding the efficacy of treatment of peri-implantitis for reducing the risk of implant failure.²⁷ A similar conclusion was reported by Lee.⁹²

ETIOLOGY AND PATHOGENESIS OF PERI-IMPLANTITIS

Microbial colonization is the chief predisposing factor in the etiology of peri-implantitis. Subgingival microbes (such as *Aggregatibacter actinomycetemcomitans*, *Prevotella intermedia*, *P gingivalis*, and *Treponema denticola*) associated with the etiology of periodontitis are similar to those of peri-implantitis.⁷⁸ In addition, it also has been reported that patterns of plaque formation and accumulation on implant surfaces are comparable to those seen on teeth.⁹³

The pathogenesis of peri-implantitis is similar to that of periodontitis.⁹⁴ Microbes in plaque stagnated on implant surface(s) release chemotactic peptides that attract neutrophils toward the peri-implant pocket(s). Moreover, cytokines are released into the peri-implant crevicular fluid as a result of microbial damage to epithelial cells, which attract more leukocytes (predominantly neutrophils) toward the affected site.⁹⁵ If neutrophils become overloaded with microbes, they degranulate and the toxic enzymes released from neutrophils cause tissue damage and gingival inflammation.⁹⁴ If the inflammation persists, it may progress to marginal gingiva and ultimately cause bone loss, a classical feature of peri-implantitis.⁹⁴ Stromal cells (such as granulation tissue fibroblasts) also may participate in the pathogenesis of peri-implantitis by upregulating vascularity and matrix breakdown, thereby promoting migration/maintenance of infiltrates (proinflammatory cytokines) into the inflamed site.⁹⁶

MANAGEMENT OF PERI-IMPLANTITIS

A variety of treatment protocols have been proposed for the management of peri-implantitis.⁹⁷ The management of peri-implantitis can be divided into (1) nonsurgical management and (2) surgical management; surgical management encompass resective and regenerative treatment.^{97–100} However, there is lack of standardized treatment protocols for peri-implant disease. The following text comprehensively reviews the different therapeutic protocols that have been proposed for the management of peri-implant diseases.

Nonsurgical Management of Peri-implant Diseases

Nonsurgical (closed debridement) treatment of peri-implant disease involves the mechanical removal of plaque and calculus (using mechanical instruments, such as scalers and curettes) from the implant surfaces (Fig. 6).¹⁰¹ The significance of antibiotics and/or antiseptic oral rinses (including chlorhexidine [CHX]) as adjuncts to mechanical debridement remains debatable.^{23,101,102} Renvert and colleagues⁹⁸ systematically reviewed



Fig. 6. Nonsurgical treatment of peri-implant diseases using hand instruments.

indexed literature that assessed the nonsurgical management of peri-implant mucositis and peri-implantitis. They reported that nonsurgical mechanical debridement alone is effective in the treatment of peri-implant mucositis. The adjunct use of antiseptic oral rinses in combination with nonsurgical mechanical debridement improved the treatment outcome of peri-implant mucositis as compared with the use of nonsurgical mechanical debridement alone.⁹⁸ In a study on cynomolgus monkeys, Trejo and colleagues¹⁰¹ reported mechanical debridement with or without the adjunct use of CHX is effective in the treatment on experimental peri-implant mucositis. In addition, histologic results by Trejo and colleagues¹⁰¹ showed minimal inflammation in groups treated by mechanical debridement (regardless of CHX use) as compared with the control group (no treatment group). However, Schwarz and colleagues¹⁰³ reported limited effect of nonsurgical mechanical debridement on the treatment of peri-implant diseases irrespective of type of adjunct treatment used.¹⁰³ Similar results were reported by Karring and colleagues.¹⁰⁴ Clinical results by Khoury and Buchmann¹⁰⁵ showed a temporary improvement in PD following nonsurgical treatment of peri-implant diseases. This could possibly have occurred due to an incomplete decontamination of implant surfaces during the nonsurgical treatment approach.

Although mechanical implant surface debridement constitutes the basic element for treatment of peri-implant diseases, other factors, such as implant positioning, geometry, and surface characteristics, may influence the overall outcomes of mechanical treatment of infected implants.¹⁰⁶ There is a need for further randomized controlled trials assessing treatment modalities of nonsurgical management of peri-implant mucositis and peri-implantitis.

Surgical Management of Peri-implant Diseases

Surgical management of peri-implantitis is usually recommended in patients with severe forms of peri-implant diseases (PD >5 mm and bone loss) (Fig. 7).^{89,105,107} Three-year follow-up results from a clinical trial showed that surgical treatment of peri-implantitis was significantly more effective in reducing PD as compared with nonsurgical therapy.¹⁰⁵ However, there are no long-term clinical trials in indexed literature that have compared nonsurgical with surgical interventions for peri-implantitis.¹⁰⁸

The surgical treatment approach is performed when an acute infection has resolved and appropriate oral hygiene of the patient is achieved. Guided bone regeneration (GBR) is a surgical approach in which a particulate graft material (such as allograft, xenograft, or alloplast) is directly placed into the osseous defect and covered with a barrier membrane (such as collagen membrane or expanded polytetrafluoroethylene



Fig. 7. Surgical treatment of peri-implant diseases using flap surgery and diode (810 nm) laser decontamination.

[ePtFE] membrane).^{109–111} Schou and colleagues^{110,111} investigated the efficacy of GBR by using autogenous bone graft and ePTFE membrane in the treatment of peri-implantitis in cynomolgus monkeys. In these studies,^{105,106} experimental peri-implantitis was induced in the jaws of monkeys after 3 months of the placement of Ti plasma-sprayed implants. Plaque-control protocols were executed and implants were divided into 4 different surgical treatment groups that comprised: (1) placement of autogenous bone grafts over the defect and covered by membranes, (2) covering the defect with autogenous bone grafts alone, (3) covering the defect with membrane alone, or (4) access flap procedure as a control group. Euthanasia was performed after 6 months, and jaw segments were assessed by quantitative digital subtraction radiography. The results showed that bone levels corresponding to levels that existed before induction of peri-implantitis were achieved in cases in which autogenous bone graft particles were placed over the defect and covered with membrane as compared with other groups.^{110,111}

The use of growth factors as adjuncts to conventional GBR protocols also has been reported to enhance osseointegration and promote new bone formation around induced peri-implant defects as compared with when GBR is performed without adjunct growth factor therapy.^{112–115} Although studies^{89,91,116} have reported that surgical management of peri-implant diseases yields promising results, long-term controlled studies are needed to validate the efficacy of these surgical treatments.

Lasers in the Treatment of Peri-implant Diseases

Some studies^{17,19,117–119} have recommended the use of lasers as an adjunct to the conventional nonsurgical treatment of peri-implantitis. It has been shown that carbon dioxide (CO₂) and 980-nm diode lasers do not affect implant surfaces during irradiation of implant surfaces as compared with other lasers, such as Neodymium-doped Yttrium-Aluminum-Garnet (Nd:YAG) and Erbium:YAG laser.^{119,120} In a case-series involving 15 patients, Romanos and Nentwig¹⁹ investigated the efficacy of CO₂ laser in the decontamination of failing implants. Clinical and radiologic parameters were evaluated at baseline and approximately after 2 years. The results showed almost complete bone fill in the peri-implant defects and the study concluded that decontamination of the implant surfaces with the CO₂ laser is an effective treatment for the management of peri-implantitis.¹⁹ Roncati and colleagues¹¹⁷ compared the effect of using a 810-nm diode laser as adjunct to conventional nonsurgical and nonsurgical debridement alone (control) in the treatment of peri-implantitis. The 5-year follow-up

outcomes showed nonsurgical debridement with adjunctive 810-nm diode laser therapy is a more effective treatment strategy for the management of peri-implantitis as compared with nonsurgical debridement alone.¹¹⁷ Likewise, results from another study¹²¹ reported osteoblastic attachment to implant surfaces and bone formation is higher in irradiated as compared with nonirradiated implant surfaces.

Recently, a new technique termed laser-assisted peri-implantitis protocol (LAPIP) is under investigation. The LAPIP technique is an implant-specific modification of the laser-assisted new attachment protocol (LANAP). LAPIP and LANAP protocols use a laser-ablation step to eradicate inflamed sulcular tissues (using Nd:YAG laser) and decontaminate the implant/root surface followed by nonsurgical periodontal therapy (scaling and root planing). The LAPIP works on the concept of creation of a blood clot, which allows the defect area to heal apico-coronally by preventing the down-growth of gingival epithelium. However, there are no controlled trials in indexed literature regarding the efficacy of LAPIP for the management of peri-implantitis.

Photodynamic Therapy in the Treatment of Peri-implant Diseases

The concept of photodynamic therapy (PDT) is based on the application of light (generally red light with wavelength ranging between 630 and 700 nm) on a chemical dye (photosensitizer [PS]), which leads to the production of singlet oxygen molecules under aerobic conditions (**Fig. 8**).¹²² These molecules have been reported to cause oxidative damage to the target cells, such as microbial and tumor cells.¹²³ PDT has been shown to be a noninvasive and practical and feasible treatment option for periodontitis and cancer.^{124,125} Although clinical^{126–128} and experimental^{129–131} studies have investigated the efficacy of PDT in the management of peri-implantitis, there seems to be a lack of consensus among clinicians and researchers in nominating PDT as a treatment of choice for peri-implantitis. The factors associated with this discrepancy include variation in the study design, duration/frequency of treatment, selection criteria of control group and the laser-related parameter, such as type of laser PS. Thus, further long-term randomized controlled trials are needed to assess the role of PDT in the treatment of peri-implantitis.

Decontamination of Implant Surfaces

It has been reported that implant surface roughness influences the healing process by promoting osteogenesis and cell surface interactions.^{132–135} However, implant surface roughness has a significant impact on the quantity and quality of the plaque formed.¹³⁶ In addition, bacterial adhesion in the grooves and pits of rough-surfaced

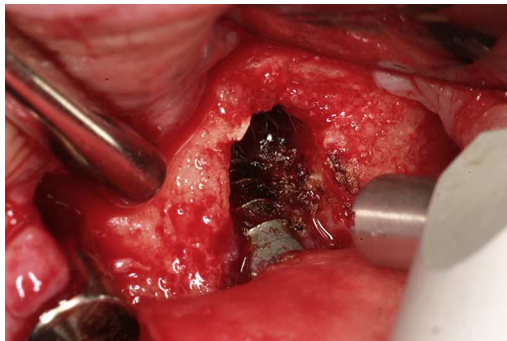


Fig. 8. Decontamination of the implant surface using a CO₂ laser.

implants may be difficult to remove. A variety of implant surface decontamination/detoxification techniques have been proposed; all of which aim to facilitate healing and render the implant surface biocompatible.

Mechanical decontamination

Implantoplasty (modification in implant surface topography) when used in combination with resective surgery has been reported to significantly reduce the clinical parameters of peri-implantitis, such as PD, sulcus bleeding, and suppuration.¹³⁷ Romeo and colleagues^{137,138} showed 100% survival rate in implants decontaminated with implantoplasty compared with controls (implants decontaminated without implantoplasty). In addition, mechanical debridement of peri-implant granulation tissue using Teflon curettes and abrasive sodium carbonate air powder reduces inflammation and pathogenic microbes within 3 months of treatment.^{139,140} According to Persson and colleagues,¹⁴¹ cotton pellets soaked in normal saline may be sufficient to decontaminate microrough implant surfaces.

Chemical decontamination

The most common chemical agents used for detoxification of implant surfaces are 10% hydrogen peroxide (H_2O_2) and CHX.^{137,138} However, epithelial cell growth has been reported to be higher on H_2O_2 -treated implant surfaces as compared with CHX-treated surfaces.¹⁴² It has been shown in vitro that rubbing a cotton pellet soaked with 3% H_2O_2 for 1 minute significantly decreases the microbial counts on implant surfaces as compared with untreated implant surfaces.¹⁴³

Citric acid (CA) also has been used for the detoxification of the implant surfaces. In an in vitro study,⁸⁹ application of CA (pH1 with cotton pellet for 1 minute) was shown to significantly reduce microbial counts on Ti alloy grit-blasted surfaces compared with untreated controls. In another study, CA-soaked cotton pellets were rubbed on Ti cylindrical units contaminated with *P gingivalis* endotoxin for 1 minute and for 2 minutes on machined, Ti plasma-sprayed, and hydroxyapatite (HA)-coated implants. One-minute application of CA caused reduction in endotoxin by 85.8%, 27.0%, and 86.8% for machined, Ti plasma-sprayed, and HA-coated implants, respectively; whereas, 2-minute CA application caused reductions in endotoxin by 90.0%, 36.4%, and 92.1% for machined, Ti plasma-sprayed, and HA-coated implants, respectively. It can therefore be concluded that CA is a useful decontamination agent for machined and HA-coated implant surfaces as compared with Ti plasma-sprayed implant surfaces.¹⁴⁴

Tetracycline is a bactericidal antibiotic that inhibits protein synthesis. In an experimental study by Hall and colleagues,¹⁴⁵ chronic bony defects induced around implants were cleaned with tetracycline. After 4 months of follow-up, 1.77 mm of reosseointegration was achieved.¹⁴⁵ Case-reports^{146–148} also have shown that application of 50 mg/mL tetracycline for 5 minutes on implant surfaces after mechanical decontamination and followed by guided bone regeneration arrests the lesion and facilitates bone refill in the defect.

Use of lasers

To our knowledge from indexed literature, there is a lack of consensus on the type and settings of lasers that are most favorable for bacterial decontamination of titanium implant surfaces. Various lasers including Erbium-doped yttrium-aluminum-garnet (Er:YAG) and CO_2 lasers have been used for implant surface decontamination.^{149–151} The Er:YAG laser has been shown to be effective in removing the oral biofilm from implant surfaces without jeopardizing the implant surface.¹⁵² Likewise, the CO_2 laser also has been reported to be safe and it does not hinder osteoblastic attachment to implant surfaces. However, a major risk while using CO_2 and Er:YAG lasers is the

temperature increase above the critical threshold (10°C) after 10 seconds of continuous irradiation. The Nd:YAG laser is contraindicated for the decontamination of implant surfaces because it produces morphologic changes in the implant surfaces, such as melting, cracks, and crater formation due to overheating.¹⁵³

Antibiotics in the Treatment of Peri-implant Diseases

It is known that bacteria associated with peri-implantitis are similar to those related to periodontal disease.^{38–41} Antibiotics when used as adjuncts in the treatment of peri-implant diseases have been reported to decrease microbial and peri-implant inflammatory parameters over a period of 12 months.¹⁵⁴ It is tempting to hypothesize that administration of local or systemic antibiotics with traditional peri-implantitis therapeutic regimens eliminates bacteria to a greater extent than when such treatments are performed alone. Several studies^{20,154–159} have shown that traditional treatments of peri-implantitis when used with adjunct antibiotic therapy reduce gingival bleeding, suppuration, and PD. However, in most of these studies,^{20,154–157,159} there was no placebo or control group. Khoury and Buchmann¹⁰⁵ tested the efficacy of 3 surgical approaches in the management of peri-implant diseases. In this study,¹⁰⁵ all patients were administered with antibiotics 4 weeks preoperatively for 1 week and later starting 1 day and finishing 7 days postoperatively. The results showed that the outcomes of the 3 surgical techniques (with preoperative and postoperative antibiotic cover) were comparable.¹⁰⁵ Moreover, results from a systematic review reported that the significance of using antibiotics as adjuncts to conventional peri-implantitis therapies remains debatable.²³

Because there is heterogeneity of studies, which have used experimental combinations of different decontamination methods, it is exigent to establish a single standardized protocol for implant decontamination for the treatment of peri-implantitis. However, further information regarding implant surface decontamination is published elsewhere.¹⁶⁰

PROPOSED GUIDELINES FOR THE MANAGEMENT OF PERI-IMPLANTITIS

Authors of the present review propose the following sequential guidelines for the management of peri-implantitis: (1) elevation of a full-thickness mucoperiosteal flap; (2) mechanical debridement of implant surface(s) using hand instruments (such as curettes) followed by decontamination using CO₂ laser (2-Watt, pulsed-mode or continuous wave); (3) guided bone regeneration using particular graft material and resorbable membrane; and (4) closure of the defect using resorbable sutures.

SUMMARY

There seems to be a lack of consensus over the treatment protocol that would yield the best outcomes in terms of management of peri-implant diseases in humans. Regardless of which therapeutic treatment protocol is adopted for the management of peri-implant diseases, a proficient diagnosis, patient compliance (plaque-control), control of possible risk factors, and skillful decontamination of implant surfaces influence the overall outcome of the treatment.

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