EAO
POSITION PAPER

Current Concepts for the Treatment of Peri-implant Disease

Ausra Ramanauskaite, Dr med dent, PhD
Frank Schwarz, Prof Dr med dent
Department of Oral Surgery and Implantology, Goethe University Frankfurt, Frankfurt, Germany.

Peri-implant diseases are defined as bacterial plaque-induced inflammatory conditions affecting implant-surrounding tissues and are classified as peri-implant mucositis and peri-implantitis. Peri-implant mucositis is characterized by an inflammatory lesion that resides in the soft tissue compartment, whereas at peri-implantitis sites the lesions also feature progressive loss of implant-supporting bone. Inflammation resolution and disease progression arrestment are the main therapeutic endpoints of the treatment of peri-implant diseases. The present position paper displays the current evidence and clinical recommendations of the European Association for Osseointegration for the treatment of peri-implant diseases. Mechanical biofilm removal along with the reinforcement of patient-administered oral hygiene is considered the standard treatment for managing peri-implant mucositis. It is recommended to assess the outcomes of peri-implant mucositis treatment 2 to 3 months after therapy, and repeated intervention should be considered in the absence of treatment success. Peri-implantitis treatment should follow a stepwise treatment approach, starting with nonsurgical treatment followed by surgical intervention, if that is not sufficient. Surgical peri-implantitis therapies include nonreconstructive, reconstructive, and combined treatment modalities. Implantoplasty may be advocated for the treatment of supracrestal peri-implant defects, whereas reconstructive therapy is indicated at peri-implantitis sites featuring intraosseous defects with a depth ≥ 3 mm. Adjunctive reconstructive measures may be beneficial in enhancing radiographic defect fill and maintaining postoperative soft tissue levels, which may have a great impact in esthetic cases. The adjunctive use of systemic antibiotics during surgical therapy does not seem to improve the clinical outcomes. Regular supportive peri-implant therapy with biofilm removal should be an integral part of the treatment protocol for peri-implant diseases. In the presence of advanced bone loss around implants that do not play a strategic role in masticatory function, implant removal may be considered immediately. *Int J Prosthodont 2024;37:124–134. doi: 10.11607/ijp.8750*

During the 2017 World Workshop on the Classification of Periodontal Diseases and Conditions, peri-implant diseases were defined as inflammatory lesions affecting the tissues surrounding functioning dental implants and were further classified as peri-implant mucositis and peri-implantitis.[1] Peri-implant mucositis is an initial stage of the disease that is restricted in the soft tissue compartment. This stage clinically features bleeding on probing (BOP) or suppuration (Sup) with or without increased peri-implant probing depth (PD) compared to previous examinations and also the absence of bone loss beyond the crestal-bone-level changes resulting from the initial bone remodeling.[2,3] If untreated, peri-implant mucositis may convert into peri-implantitis lesions, which are mainly characterized by the loss of supporting bone.[4] The progression of peri-implantitis lesions is characterized by a nonlinear, accelerating pattern that in the absence of therapy may ultimately lead to implant loss.[5] Recent cross-sectional studies have revealed the high prevalence of peri-implant diseases, highlighting their common clinical appraisal in daily clinical practice.[6–8]

Correspondence to:
Dr Ausra Ramanauskaite
ramanauskaite@med.uni-frankfurt.de

Submitted May 31, 2023;
accepted November 9, 2023.

Although peri-implant diseases are initially caused by a bacterial challenge, numerous factors such as history of chronic periodontitis, poor plaque control skills, absence of regular maintenance care after implant placement, and prosthesis overcontouring impeding access to oral hygiene procedures were reported to increase the risk of developing peri-implant diseases.[2,3,9,10] Furthermore, there is evidence linking peri-implantitis to other factors such as submucosal cement remnants, improper implant positioning, and lack of keratinized mucosa.[3,11]

The endpoint of peri-implant mucositis therapy is resolution of peri-implant mucosal inflammation, which is determined by the absence of ≤ 1 site with BOP.[12] Nonsurgical therapeutic approaches in conjunction with oral hygiene reinforcement were shown to be effective in reducing signs of inflammation, but complete resolution could not always be achieved.[2,13] In addition to the resolution of inflammation at peri-implantitis sites, the treatment also seeks to arrest further marginal bone loss.[14] In contrast to peri-implant mucositis, various nonsurgical peri-implantitis therapeutic measures demonstrated limited predictability in reliably implementing the aforementioned outcomes, indicating the need for surgical interventions in most cases.[15]

The aim of this position paper is to provide an update on the concepts for the treatment of peri-implant disease based on the currently available evidence.

DIAGNOSIS OF PERI-IMPLANT DISEASES

Following implant-supported restoration placement, all patients should be enrolled into regular maintenance therapy to monitor peri-implant tissue health.[12] The maintenance program should include repeated probing of peri-implant tissues to identify clinical signs of inflammation and interventions for primary prevention of peri-implant disease, such as professional supra- and submarginal plaque biofilm removal and oral hygiene reinforcement.[12] A recall interval of 6 months may be considered, but the frequency of maintenance appointments should be tailored to every patient according to their risk profile.[16]

Based on the recent clinical recommendations, the diagnosis of peri-implant diseases should be based on the following parameters.[1,12,17] *(a)* Peri-implant mucositis: presence of ≥ 1 BOP spot or presence of a line of bleeding or profuse BOP at any location or Sup on gentle probing in the absence of bone loss beyond crestal-bone-level changes resulting from initial bone remodeling. *(b)* Peri-implantitis: presence of BOP/Sup upon gentle probing, increased PD values compared to previous examination, and presence of bone loss beyond crestal-bone-level changes resulting from initial bone remodeling.

In the absence of previous examination or baseline data, the diagnosis of peri-implantitis requires a combination of the following criteria:

- Presence of BOP or Sup on gentle probing
- PD values ≥ 6 mm
- Bone levels ≥ 3 mm apical to the most coronal portion of the intraosseous part of the implant

NONSURGICAL TREATMENT OF PERI-IMPLANT MUCOSITIS

Based on evidence derived from experimental clinical studies, peri-implant mucositis is a treatable condition and can be resolved by implementing meticulous control of the peri-implant biofilm.[18–20] However, if left undiagnosed or untreated, peri-implant mucositis may progress to peri-implantitis. Therefore, peri-implant mucositis is considered a precursor for peri-implantitis.[4] Conversion of peri-implant mucositis to peri-implantitis was clinically evaluated in one retrospective observational study with 80 patients initially diagnosed with peri-implant mucositis.[21] Over 5 years, 43% of the patients not adhering to the preventive maintenance therapy (nonmaintenance group) were diagnosed with peri-implantitis; whereas, in the group of patients following the preventive maintenance, the incidence of peri-implantitis was considerably lower (maintenance group; 18% of the patients). Clinical parameters (eg, BOP, PD, and the presence of periodontitis) were associated with a higher risk of developing peri-implantitis.[21]

Given that peri-implant mucositis precedes peri-implantitis, the treatment of peri-implant mucositis lesions is considered a primary preventive intervention for peri-implantitis.[4] The aim of peri-implant mucositis therapy is the resolution of peri-implant mucosal inflammation clinically defined as ≤ 1 point of BOP and absence of Sup.[12] Conventional nonsurgical mechanical therapy in conjunction with oral hygiene reinforcement is the standard treatment for peri-implant mucositis[2,13] (Fig 1). This treatment results in an average of 0.5 to 1.0 mm PD reduction and 15% to 40% decrease in BOP values.[13] To increase the efficacy of mechanical subgingival instrumentation, numerous alternative and adjunctive measures have been suggested to facilitate the resolution of peri-implant mucosal inflammation. In particular, as documented in numerous controlled clinical trials, the use of alternative mechanical and physical measures for subgingival-biofilm removal—including air polishing with glycine powder (Fig 2), chitosan brushes, ultrasonic devices and CO_2 lasers—provided no beneficial effects in terms of BOP or PD reduction over the 3- to 12-month period compared to subgingival mechanical debridement alone.[22–26] Likewise, similar treatment outcomes, depicted by comparable BOP and PD changes, could be obtained at peri-implant mucositis sites treated with adjunctive local applications of various local antiseptics—including chlorhexidine gel (CHX 0.12%), a full-mouth disinfection concept using

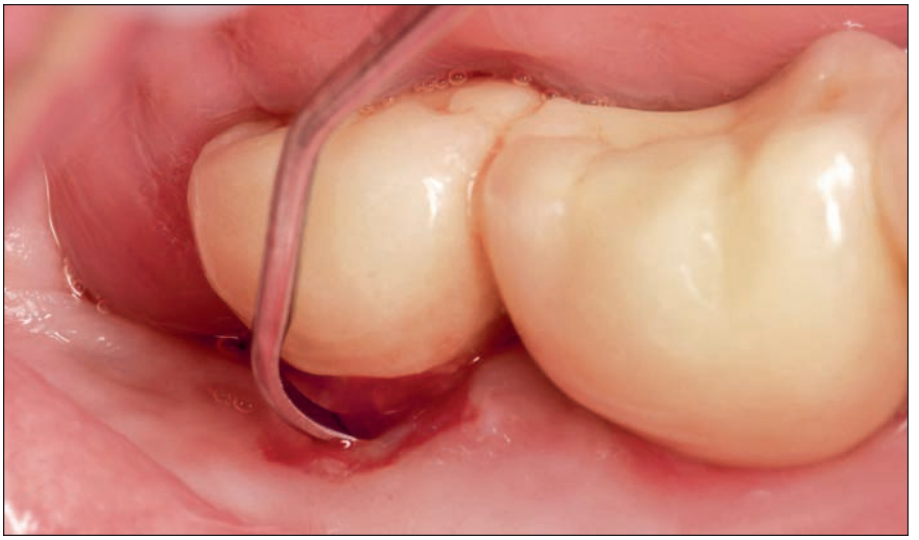

Fig 1 Conventional peri-implantitis treatment using titanium curettes for subgingival debridement.

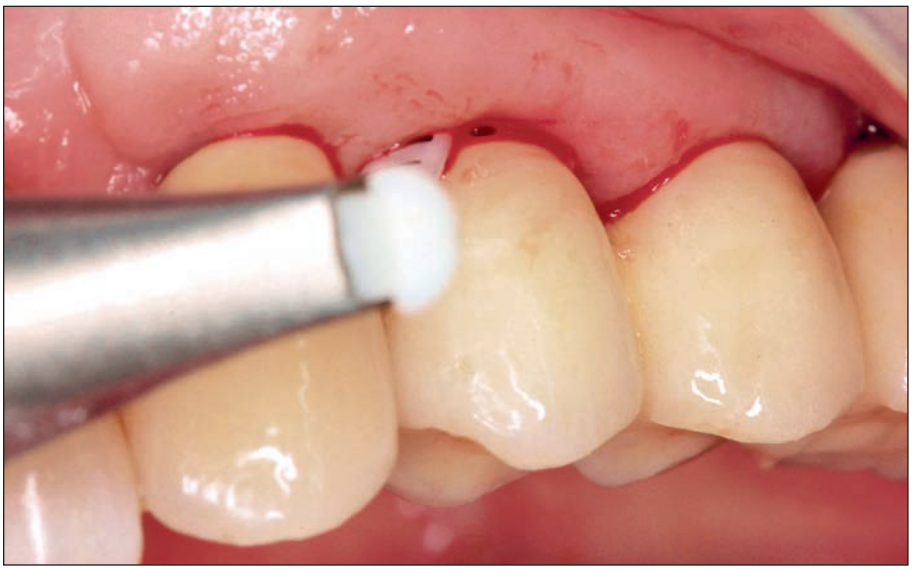

Fig 2 Air polishing with glycine powder as an alternative measure for nonsurgical peri-implantitis treatment.

CHX gel and mouth rinse, or applications of sodium hypochlorite—and those only treated with mechanical debridement.[27–30]

Adjunctive prescription of probiotics for peri-implant mucositis treatment have been tested in two RCTs, one of which pointed to higher BOP reduction 3 months after the use of probiotics for 30 days, whereas another RCT after 4 to 5 months failed to detect any benefit of probiotics administered for 15 days.[31,32] As reported in two RCTs, the adjunctive administration of systemic antibiotics (azithromycin) did not have any beneficial effect on the changes in BOP and PD values over the follow-up period of 3 to 6 months, thus not supporting the rationale for prescription of systemic antibiotics for peri-implant mucositis treatment.[33,34] Furthermore, the adjunctive use of home care mouth rinse has been assessed in 3 RCTs, one of which suggested higher BOP reduction in the patients adjunctively using oral irrigation with 0.06% CHX mouth rinse.[35–37]

Although peri-implant mucositis is considered a reversible condition, irrespective of the therapeutic approach, complete disease resolution cannot be achieved in all cases. It is recommended that the outcomes of peri-implant mucositis treatment be assessed after 2 to 3 months, and in the presence of ≥ 2 BOP sites, ≥ 1 sites with profuse BOP, or presence of Sup, a repeated intervention should be considered.[12]

NONSURGICAL TREATMENT OF PERI-IMPLANTITIS

Peri-implantitis treatment should follow a stepwise treatment approach, starting with nonsurgical treatment and following with surgical intervention if it's not sufficient.[12]

The purpose of nonsurgical peri-implantitis therapy is to control peri-implant biofilm and resolve inflammation.[12] Nonsurgical treatment by mechanical submarginal debridement alone usually provides clinical improvements in reduced bleeding tendency (20% to 50%) and, in some cases, PD reduction (≤ 1 mm).[13] To improve the outcomes of nonsurgical peri-implantitis therapy, numerous studies have assessed the efficacy of various alternative and adjunctive measures for subgingival-biofilm removal.

Based on previous RCTs' findings, within the 6- to 12-month period, alternative measures used for the submarginal instrumentation, (eg, Er:YAG and Er,Cr:YSGG lasers and air polishing with glycine powder) compared to mechanical debridement with curettes, lead to significant improvements in BOP reduction; whereas, similar outcomes were obtained in terms of the PD changes.[38–41] Alternative use of ultrasonic devices or adjunctive use of a diode laser failed to show any benefit in terms of BOP or PD changes; whereas, after 6 months, the adjunctive use of antimicrobial photodynamic therapy (aPDT) was associated with greater improvement in PD and bleeding (sulcus bleeding index) values.[42–44]

Previous clinical studies with a follow-up period ranging from 6 to 12 months investigated the efficacy of local antimicrobials used along with nonsurgical mechanical debridement at peri-implantitis sites, including application of local antibiotics (single or repeated application of minocycline microspheres), CHX 1.0% gel (single or repeated), repeated applications of CHX-containing chips, or single subgingival application of desiccant material.[45–50] A greater decrease in BOP values could be achieved following the repeated applications of local antibiotics,[46] and higher improvements in PD values were obtained at peri-implantitis sites additionally treated with repeated application of CHX chips or single application of local antibiotics or desiccant material.[49,50]

The efficacy of the administration of systemic antibiotics in conjunction with mechanical nonsurgical peri-implantitis therapy has been investigated in four RCTs. Three of them indicated the benefits of systemic antibiotics (ie, a combination of metronidazole and amoxicillin 500 mg, metronidazole, or azithromycin 500 mg 3 days before treatment) that were observed 12 months after treatment in terms of higher reductions in BOP and PD values.[50–53] On the other hand, one RTC denied the advantages of systems antibiotics (amoxicillin and metronidazole) by reporting on similar clinical outcomes (ie, BOP and PD changes) yielded 3 months after the treatment in the test and control groups.[54] Despite the potential added effect of systemic antibiotics—particularly at initially deep sites (PD > 6 mm), considering their benefits vs harm, systemic antibiotics should not be routinely prescribed as an adjunctive measure for nonsurgical peri-implantitis therapy.[55]

According to recent recommendations for nonsurgical supra- and submarginal instrumentation in patients with peri-implantitis, curettes or sonic or ultrasonic devices may be used.[12] The current clinical data does not support the beneficial use of alternative or adjunctive measures, including lasers, air polishing, local antimicrobials or desiccant antiseptics, or local and systemic antibiotics for the nonsurgical treatment of peri-implantitis.[12]

It is recommended to assess the outcomes of nonsurgical peri-implantitis treatment after 6 to 12 weeks.[12] The end point of nonsurgical peri-implantitis should be residual PD ≤ 5 mm with no BOP at more than one point and no Sup.[12] If the end points of the nonsurgical therapy have not been achieved, surgical intervention should be considered.

Although previous consensus conferences suggested nonsurgical treatment as an initial therapeutic step prior to the surgical intervention, one recent RCT failed to identify any clinical benefits of the submarginal instrumentation performed 6 weeks before the surgical treatment of peri-implantitis.[56] The latter findings call for more and larger clinical trials to justify the need of a nonsurgical treatment prior to surgical intervention.

SURGICAL TREATMENT OF PERI-IMPLANTITIS

Surgical peri-implantitis treatment approaches can be categorized into three modalities: nonreconstructive (open flap debridement or pocket elimination procedures), reconstructive therapy, and a combined surgical approach (ie, implantoplasty and reconstructive therapy).[57]

Nonreconstructive Therapy

Nonreconstructive surgical approaches include the open flap debridement and pocket elimination procedures (ie, resective therapy).[58] Open flap debridement is a basic surgical treatment modality that includes the repositioning of the soft tissue flaps upon the decontamination of the implant surface at the presurgical level.[59,60] Surgical pocket elimination procedures involve the elimination or reduction of pathologic peri-implant pockets by means of apical positioning of the soft tissue flap with or without osseous recontouring.[61,62]

To resolve peri-implant tissue inflammation, decontamination of the implant surface is a crucial step of the surgical treatment protocol.[57] Previous clinical studies assessed the efficacy of various implant surface decontamination approaches along with the surgical nonreconstructive peri-implantitis treatment. Based on the results of the RCTs with a follow-up period ranging from 6 months to 3 years, use of aPDT, diode laser, air polishing with erythritol powder, or local irrigation

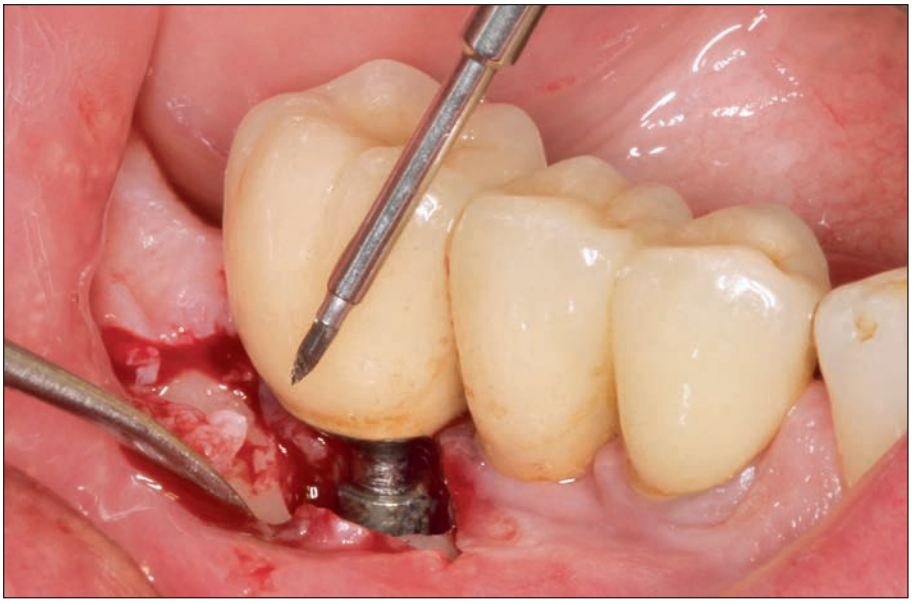

Fig 3 Titanium brush for the implant surface decontamination following surgical peri-implantitis treatment.

antimicrobials (ie, CHX with or without cetylpiridinium chloride) in conjunction with mechanical debridement failed to improve treatment outcomes compared to mechanical debridement alone.[61,63–67] On the other hand, the use of titanium brushes for the mechanical debridement of the implant surface resulted in greater PD reduction and stable marginal bone levels compared to the implant sites cleaned with either plastic curettes or air-polishing with glycine powder.[68] According to the findings of the recent systematic reviews, no alternative or adjunctive physical, mechanical, or chemical decontamination measures could be superior to standard debridement procedures (mechanical debridement with or without saline).[69,70] Nonetheless, based on a limited clinical evidence, titanium brushes may be beneficial in reducing signs of inflammation[70] (Fig 3).

With respect to the efficacy of local antimicrobials, the repeated application of local antibiotics (ie, minocycline ointment) after 6 months resulted in greater PD reduction, whereas similar BOP changes were achieved at test and control sites (ie, mechanical debridement alone).[71] As demonstrated in two RCTs with follow-up periods of 1 and 3 years, prescription of systemic antibiotics failed to improve the outcomes of nonreconstructive peri-implantitis treatment.[60,61] In corroboration with these results, one recent systematic review concluded that the adjunctive use of the currently tested systemic antimicrobials during surgical nonreconstructive therapy, compared to surgical therapy alone, does not seem to improve the clinical efficacy.[72]

Outcomes of Nonreconstructive Therapy

The estimation of one recent meta-analysis revealed pronounced clinical improvements after 1 to 5 years following nonreconstructive peri-implantitis therapy, depicted by a considerable decrease in PD (standardized mean effect (SME): 2.2 mm, CI: 1.8, 2.7 mm), BOP (SME: 27.0%; CI: 19.8, 34.2 mm) values, and marginal bone level gain (SME: 0.2 mm, CI: –0.0, 0.5 mm).[58] Treatment success, defined as an absence of PD ≥ 5 mm with concomitant BOP/Sup and no progressive bone loss after 5 years, was documented in 53% of the implants, and 63% of the patients enrolled in regular supportive therapy.[73] Regarding the postoperative soft tissue-level changes, open flap debridement and pocket elimination surgical procedures were followed by a considerable soft tissue recession (weighted mean effect [WME] = 0.95 mm, CI: –0.20, 2.1 mm; and WME = 1.22 mm, CI: 0.71, 1.73 mm, respectively).[74]

Disease recurrence was, however, frequently reported 5 years after the treatment (32.1% to 43.8% of implants).[58] Treatment outcomes were shown to be negatively influenced by the presence of initial PD > 8 mm, presence of Sup, extensive bone loss (> 7 mm), residual PDs ≥ 6 mm 1 year after surgery, and modified implant surfaces.[75–77]

Implantoplasty

Implantoplasty—the mechanical modification of an implant, including thread removal and surface smoothening—has been proposed during surgical peri-implantitis treatment.[78,79] The procedure is intended to reduce the roughness of the exposed rough implant parts in the presence of supracrestal defects, thus reducing the surfaces' affinity for plaque accumulation and subsequently decreasing the risk of reinfection.[80]

The benefits of implantoplasty were reported in one 3-year RCT, indicating a considerably higher extent of

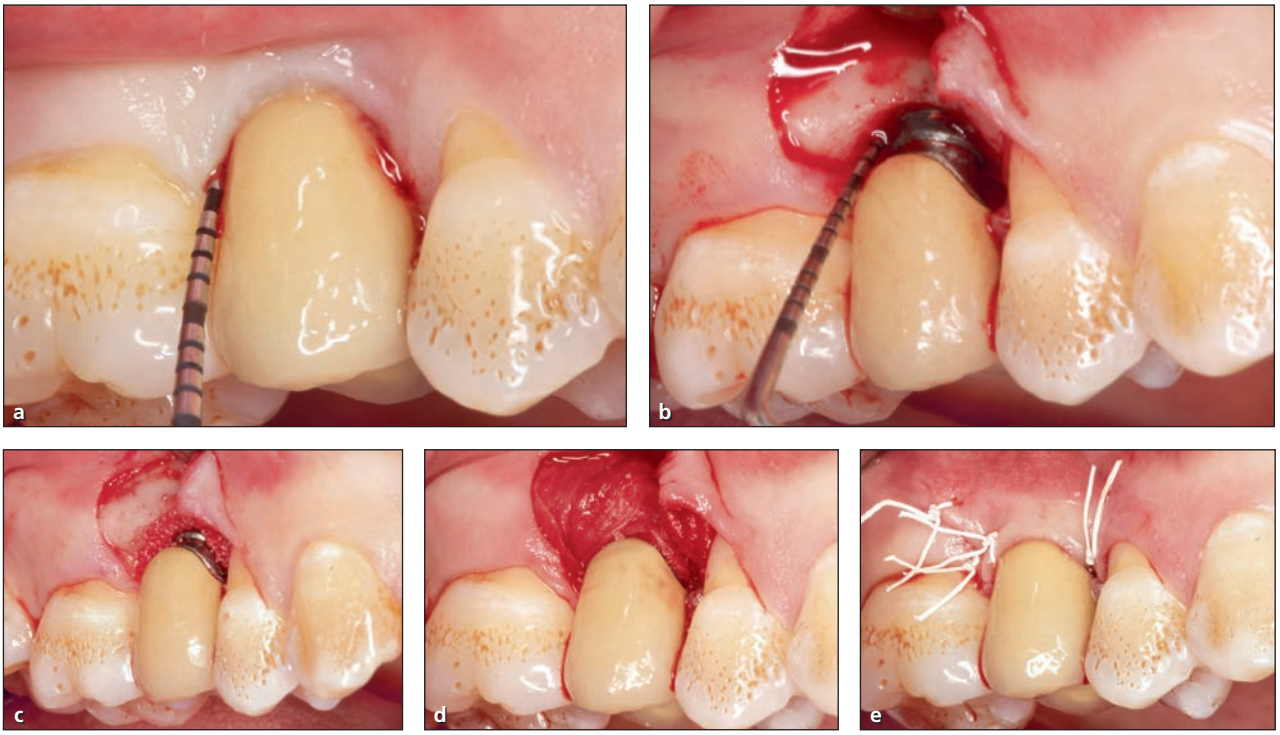

Fig 4 Clinical case illustrating reconstructive peri-implantitis treatment at implant 015. *(a)* Preoperative intraoral view showing a PD value of 5 mm along with BOP. *(b)* Intraoperative view showing intrabony defect 3 mm in depth. *(c)* Intrabony defect homogenously filled with a bovine mineral embedded in a collagen matrix (Bio-Oss Collagen). *(d)* Treated area covered with a native collagen membrane (Bio-Guide). *(e)* Suturing.

PD reduction, greater decrease of mucosal-inflammation, and stable marginal-bone levels at implant sites adjunctively treated with implantoplasty compared to those treated with open flap debridement alone.[81,82] This report is in line with the results of one recent meta-analysis, which pointed out significant PD reduction following implantoplasty (WME = –1.11 mm; P = .02) and revealed no negative effect of the procedure on postoperative soft tissue recession (WME = –0.02 mm; P = .95).[15] Moreover, the probability of treatment success (defined as implant being in function) 6 and 24 months after surgical peri-implantitis treatment employing implantoplasty amounted to 97.5% and 94.7% of the implants, respectively.[83]

The summary of the currently available preclinical in vivo and clinical evidence could not relate implantoplasty with any remarkable mechanical or biologic complications in the short to medium term.[79] Nonetheless, as suggested by the recent experimental data, although implantoplasty does not alter the biomechanical properties of implants with a standard diameter (4.1 to 4.7 mm), implants with reduced diameters (3.3 to 3.75 mm) after implantoplasty have shown reduced resistance to bending forces and in turn increased risk of an implant fracture.[84,85] Therefore, one should take caution when planning to perform implantoplasty at implants with reduced diameters.

Reconstructive Therapy

In addition to the resolution of inflammation, reconstructive peri-implantitis therapy is intended to regenerate the bone defect, achieve osteointegration, and limit postoperative soft tissue recession.[86] Reconstructive measures may be considered at peri-implantitis sites featuring intraosseous defects with a depth ≥ 3 mm[12] (Fig 4).

Comparative clinical studies investigating the efficacy of various implant surface decontamination methods in conjunction with reconstructive peri-implantitis therapy have found no benefits provided by the application of a CO_2 laser (ie, in terms of BOP, PD changes), whereas improved radiographic defect fill could be observed at implant sites adjunctively treated with ozone therapy.[87,88]

The restoration of peri-implant defects may be performed using bone filler particles alone or in a combination with a barrier membrane by employing a guided bone regeneration principle (GBR). One recent network meta-analysis was designed to determine the efficacy of different reconstructive protocols for treating peri-implantitis related bone defects.[89] Based on the established networks, the GBR approach employing xenogeneic bone substitutes led to higher reduction in BOP and PD values, improved radiographic bone levels, and less soft tissue recession compared to the GBR protocol implementing autogenous bone.[89] Furthermore, as shown by the previous 4-year clinical study,

greater improvement in BOP and PD values were obtained at peri-implantitis sites filled with xenogeneic bone substitute particles compared to those treated with synthetic bone filler.[90] Given these findings, it appears that from the clinical perspective, reconstructive treatment protocols implementing xenogeneic bone substitutes may lead to enhanced therapeutic endpoints compared to GBR employing autogenous or synthetic bone alone.

Considering the potential added benefit of the adjunctive use of a barrier membrane compared to only using a bone substitute, based on three comparative clinical studies, a resorbable barrier membrane applied over allogenic or xenogeneic bone substitute materials failed to improve therapeutic outcomes compared to the application of a bone filler alone.[91–93] In fact, the implant sites treated with an adjunctive barrier membrane were more frequently associated with postoperative complications, such as such soft tissue dehiscence and membrane or bone filler exposure.[93] One recent network meta-analysis likewise failed to show any benefits of a barrier membrane applied over xenogeneic bone substitutes (particulated or collagen embedded) in terms of PD and BOP changes (WME = 0.3 mm; $P = .689$ and WME = 2.2%, $P = .865$, respectively).[94] Furthermore, the comparison of a concentrated growth-factor membrane with a collagen membrane along with xenogeneic bone filler resulted in higher PD reduction, whereas changes in BOP and radiographic defect fill were comparable between the treatment groups.[95]

So far, there are no comparative clinical studies assessing the influence of systemic antibiotics on the clinical or radiographic outcomes of reconstructive peri-implantitis treatment. However, in the case of reconstructive procedures, systemic antibiotics may not be used to improve the therapeutic outcomes but to reduce the risk of postoperative infection of the delicate grafting material. Nonetheless, also from this perspective, the current clinical data is scarce.

Outcomes of Reconstructive Therapy

In accordance with the recent clinical recommendation, the results of reconstructive peri-implantitis treatment should be based on a composite outcome, including parameters such as PD, BOP, Sup, soft tissue recession, and radiographic bone fill.[86] Considering the similar success criteria based on the combination of clinical and radiographic outcomes, at 5 to 7 years after reconstructive peri-implantitis therapy, treatment success was achieved in 51.1% and 58.3% of implants with smooth and moderately rough surfaces whereas at implants with modified surfaces, treatment success was documented in 14.3% of the implants after 7 years.[92–96] With respect to the postoperative soft tissue level changes, reconstructive approaches yielded significantly lower soft tissue mucosal recession compared to open flap debridement (WDE = –1.35 mm, $P = .038$).[74]

One relevant factor influencing the outcomes of the reconstructive peri-implantitis therapy is the morphology of peri-implant defects. Specifically, greater PD reduction and radiographic defect fill following reconstructive peri-implantitis therapy using particulated bone filler in conjunction with a barrier membrane was obtained in peri-implantitis cases featuring circumferential-type defects (ie, four-wall defects) compared to those lacking buccal or lingual walls (ie, three-wall defects).[97,98] Likewise, four-wall defects showed 6.0 to 7.0 times greater odds ratios of a successful treatment outcome (ie, absence of additional bone loss and PD ≤ 5 mm) compared to three- and two-wall defects, respectively.[95] Moreover, deeper intrabody defects resulted in greater radiographic defect fill.[98] Contradictory findings were reported in one 5-year prospective clinical study, in which defect morphology did not affect the outcomes of reconstructive peri-implantitis therapy implemented using xenogeneic bone filler particles embedded in a collagen matrix.[99] When interpreting those contradiction, it is important to underline differences in the nature of the bone substitute materials used in the aforementioned studies, because particulated bone substitutes may be less prone to obtaining the stability in the noncontained parts of the defect compared to the volume-stabilized material.

Reconstructive vs Nonreconstructive Therapy

Based on a recent systematic review and meta-analysis, at 12 months, reconstructive peri-implantitis therapy resulted in improved radiographic marginal bone levels (WME = –0.75 mm, $P = .022$) compared to open flap debridement, whereas no significant difference was observed in the extent of PD reduction (WME = –0.38 mm; $P = .325$).[94] The results of another systematic review and meta-analysis in addition to the improved radiographic defect restoration (WME = 56.46%, $P = .01$ and WME = –1.47 mm, $P = .01$, respectively) revealed greater PD reduction (WME = 0.63, $P = .01$) and less postoperative soft tissue recession (WME = 0.63 mm, $P = .01$) following implementation of adjunctive reconstructive compared to the open flap debridement surgery.[15] However, the extent in the BOP changes were comparable between the reconstructive and nonreconstructive treatment approaches (WME = 11.11%, $P = .11$).[15] In corroboration with these results, the outcomes of a recent multicenter RCT revealed a reduced extent of postoperative soft tissue recession following reconstructive therapy, which may have a great impact in esthetic cases, while changes in BOP were comparable between the test and control (ie, open flap debridement) groups.[100]

In this context, it is important to note that the aforementioned meta-analyses and RCT comparing the

efficacy of reconstructive therapy to open flap debridement induced a wide range of defect configurations, which, as previously discussed, might greatly influence the reported outcomes.

Combined Therapy

Combined peri-implantitis surgical approaches have been advocated for the peri-implantitis sites featuring a combined defect configuration (ie, horizontal [noncontained] and vertical [contained] bone loss patterns).[101] This surgical treatment includes the use of implantoplasty at the noncontained parts of the defects and reconstructive therapy employed at the contained defect compartments.[101]

Three previous RCTs assessed the efficacy of various implant surface decontamination approaches in conjunction with the combined surgical approach.[102–104] One RCT revealed the beneficial effects of the adjunctive use of titanium brushes along with the mechanical debridement and H_2O_2 (3%) irrigation in reducing the deepest PD values.[103] Two remaining RCTs investigated the efficacy of an Er:YAG laser, one of which pointed to significantly higher PD reduction after 6 months following adjunctive use of Er:YAG, whereas another 7-year RCT denied the benefits of an Er:YAG laser by reporting similar BOP and PD changes compared to the conventional decontamination (ie, mechanical debridement and saline-soaked cotton gauze).[69,104] Within 1 to 7 years, the reported treatment success, defined as either the absence of BOP or the absence of PD ≥ 5 mm with no BOP, Sup, and no additional bone loss was achieved in 60% and 23% to 66.7% of the implants, respectively.

Assessment of Surgical Peri-implantitis Treatment Outcomes and Supportive Peri-implant Therapy

As documented by clinical studies, presence of plaque at the implant sites and poor or lack of adherence to supportive care are the main factors associated with the recurrence of inflammation at the treated implant.[76,105] Therefore, surgical treatment of peri-implantitis should be followed by supportive therapy provided every 3 to 4 months for the first 12 months, commencing 3 months after surgery.[12] Thereafter, the frequency of supportive therapy appointments should be tailored individually according to patient-, implant-, and restoration-based risk factors.

The protocol for supportive therapy should include assessment of peri-implant tissue health, reinforcement of individually performed oral hygiene, and professional plaque removal. So far, no specific protocol could be recommended for the professional mechanical plaque removal in patients treated for peri-implantitis.[12] However, such tools as titanium or stainless steel curettes, ultrasonic instruments, rubber cup or brushes, air-polishing device with glycine or erythritol powder may be used alone or in combination.

It is recommended to assess the clinical outcomes of surgical peri-implantitis treatment 6 months after the treatment by gently probing peri-implant tissues.[12] The disease resolution is defined by the combination criteria, including ≤ 1 point of BOP, absence of Sup, PD ≤ 5 mm, and absence of progressive bone loss compared to pretreatment bone levels.[12] If those endpoints cannot be obtained, re-treatment (nonsurgical or surgical) should be considered. In cases showing acute signs of recurrent peri-implant tissue inflammation (ie, profuse BOP, Sup), particularly advanced peri-implantitis lesions (bone loss > two thirds of implant length), implant removal may be indicated. At dental implants with advanced bone loss that do not play a strategic role in masticatory function, implant removal may be immediately considered. Indications for explantation also include the presence of clinical signs indicating a loss of osseointegration, complex implant designs (eg, hollow-cylinder implants), technical complications (eg, implant fracture), or complex infections affecting adjacent anatomical structures (eg, maxillary sinus, inferior alveolar nerve).[106]

In this context, it should be noted that the high recurrence rates of peri-implantitis following the surgical treatment irrespective of the treatment modality generate additional costs for the patients, of which the patients should be aware prior to the start of the treatment. Although implant loss is related to considerably greater costs compared to the peri-implantitis treatment, the need for repeated interventions after surgical peri-implantitis treatment and the risk of reinfection leading to implant loss need to be discussed with the patient.[107] On the other hand, in the decision to "treat or remove," the gradually decreasing implant survival rates following the second or third attempt of reimplantation at the sites of previously failed implants sites should be considered.[108]

CLINICAL RECOMMENDATIONS

Mechanical biofilm removal along with the reinforcement of patient administered oral hygiene is considered standard case treatment for peri-implant mucositis. The outcomes of peri-implant mucositis treatment should be assessed 2 to 3 months after therapy, and in the absence of treatment success, a repeated intervention may be considered.

Peri-implantitis treatment should follow a stepwise treatment approach, starting with nonsurgical treatment and followed by surgical intervention if the treatment is not sufficient.

Assessment of the outcomes of nonsurgical peri-implantitis treatment after 6 to 12 weeks is recommended. If the end points of the nonsurgical therapy (ie, PD ≤ 5 mm

with ≤ 1 point with BOP and no Sup) have not been achieved, surgical intervention should be considered.

Surgical peri-implantitis therapies include nonreconstructive, reconstructive, and combined treatment. Implantoplasty may be advocated for the treatment of supracrestal peri-implant defects, whereas reconstructive therapy is indicated at peri-implantitis sites featuring intraosseous defects with a depth ≥ 3 mm. Adjunctive reconstructive measures may be beneficial in enhancing radiographic defect fill and maintaining postoperative soft tissue levels, which may have a great impact in esthetically demanding areas.

Outcomes of surgical peri-implantitis therapy should be assessed 6 months after treatment. If the end points (ie, ≤ 1 point with BOP, absence of Sup, PD ≤ 5 mm and absence of progressive bone loss) could not be obtained, re-treatment or, in some cases, implant removal should be considered.

Ultimately, the currently available evidence for the treatment of peri-implant disease recommends the following: *(1)* The adjunctive use of systemic antibiotics during surgical therapy does not seem to improve clinical efficacy. *(2)* Regular supportive peri-implant therapy with biofilm removal should be an integral part of the treatment protocol of peri-implant diseases. *(3)* In the presence of advanced bone loss at implants that do not play a strategic role in masticatory function, implant removal may be immediately considered.

ACKNOWLEDGMENTS

The authors declare no conflicts of interest related to this study.

REFERENCES

1. Berglundh T, Armitage G, Araujo MG, et al. Peri-implant diseases and conditions: Consensus report of workgroup 4 of the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions. J Clin Periodontol 2018;45(suppl 20):s286–s291.
2. Heitz-Mayfield LJA, Salvi GE. Peri-implant mucositis. J Clin Periodontol 2018;45 (suppl 20):s237–s245.
3. Schwarz F, Derks J, Monje A, Wang HL. Peri-implantitis. J Periodontol 2018;89(suppl 1):s267–s290.
4. Jepsen S, Berglundh T, Genco R, et al. Primary prevention of peri-implantitis: Managing peri-implant mucositis. J Clin Periodontol 2015;42(suppl 16):s152–s157.
5. Derks J, Schaller D, Håkansson J, Wennström JL, Tomasi C, Berglundh T. Peri-implantitis—Onset and pattern of progression. J Clin Periodontol 2016;43:383–388.
6. Obreja K, Ramanauskaite A, Begic A, et al. The prevalence of peri-implant diseases around subcrestally placed implants: A cross-sectional study. Clin Oral Implants Res 2021;32:702–710.
7. Vignoletti F, Di Domenico GL, Di Martino M, Montero E, de Sanctis M. Prevalence and risk indicators of peri-implantitis in a sample of university-based dental patients in Italy: A cross-sectional study. J Clin Periodontol 2019;46:597–605.
8. Romandini M, Lima C, Pedrinaci I, Araoz A, Soldini MC, Sanz M. Prevalence and risk/protective indicators of peri-implant diseases: A university-representative cross-sectional study. Clin Oral Implants Res 2021;32:112–122.
9. Schwarz F, Alcoforado G, Guerrero A, et al. Peri-implantitis: Summary and consensus statements of group 3. The 6th EAO Consensus Conference 2021. Clin Oral Implants Res 2021;32(suppl 21):s245–s253.
10. Roccuzzo A, Imber JC, Marruganti C, Salvi GE, Ramieri G, Roccuzzo M. Clinical outcomes of dental implants in patients with and without history of periodontitis: A 20-year prospective study. J Clin Periodontol 2022;49:1346–1356.
11. Ramanauskaite A, Schwarz F, Sader R. Influence of width of keratinized tissue on the prevalence of peri-implant diseases: A systematic review and meta-analysis. Clin Oral Implants Res 2022;33(suppl 23):s8–s31.
12. Herrera D, Berglundh T, Schwarz F, et al. Prevention and treatment of peri-implant diseases—The EFP S3 level clinical practice guideline. J Clin Periodontol 2023;50(suppl 26):s4–s76.
13. Renvert S, Hirooka H, Polyzois I, Kelekis-Cholakis A, Wang HL. Diagnosis and non-surgical treatment of peri-implant diseases and maintenance care of patients with dental implants—Consensus report of working group 3. Int Dent J 2019;69(suppl 2):s12–s17.
14. Sanz M, Chapple IL. Clinical research on peri-implant diseases: Consensus report of Working Group 4. J Clin Periodontol 2012;39(suppl 12):s202–s206.
15. Ramanauskaite A, Fretwurst T, Schwarz F. Efficacy of alternative or adjunctive measures to conventional non-surgical and surgical treatment of peri-implant mucositis and peri-implantitis: A systematic review and meta-analysis. Int J Implant Dent 2021;7:112.
16. Monje A, Aranda L, Diaz KT, et al. Impact of maintenance therapy for the prevention of peri-implant diseases: A systematic review and meta-analysis. J Dent Res 2016;95:372–379.
17. Tonetti MS, Sanz M, Avila-Ortiz G, et al. Relevant domains, core outcome sets and measurements for implant dentistry clinical trials: The implant dentistry core outcome set and measurement (ID-COSM) international consensus report. J Clin Periodontol 2023;50(suppl 25):s5–s21.
18. Salvi GE, Aglietta M, Eick S, Sculean A, Lang NP, Ramseier CA. Reversibility of experimental peri-implant mucositis compared with experimental gingivitis in humans. Clin Oral Implants Res 2012;23:182–190.
19. Pontoriero R, Tonelli MP, Carnevale G, Mombelli A, Nyman SR, Lang NP. Experimentally induced peri-implant mucositis. A clinical study in humans. Clin Oral Implants Res 1994;5:254–259.
20. Schwarz F, Mihatovic I, Golubovic V, Eick S, Iglhaut T, Becker J. Experimental peri-implant mucositis at different implant surfaces. J Clin Periodontol 2014;41:513–520.
21. Costa FO, Takenaka-Martinez S, Cota LO, Ferreira SD, Silva GL, Costa JE. Peri-implant disease in subjects with and without preventive maintenance: A 5-year follow-up. J Clin Periodontol 2012;39:173–181.
22. Ji YJ, Tang ZH, Wang R, Cao J, Cao CF, Jin LJ. Effect of glycine powder air-polishing as an adjunct in the treatment of peri-implant mucositis: A pilot clinical trial. Clin Oral Implants Res 2014;25:683–689.
23. De Siena F, Corbella S, Taschieri S, Del Fabbro M, Francetti L. Adjunctive glycine powder air-polishing for the treatment of peri-implant mucositis: An observational clinical trial. Int J Dent Hyg 2015;13:170–176.
24. Riben-Grundstrom C, Norderyd O, André U, Renvert S. Treatment of peri-implant mucositis using a glycine powder air-polishing or ultrasonic device: A randomized clinical trial. J Clin Periodontol 2015;42:462–469.
25. Wohlfahrt JC, Aass AM, Koldsland OC. Treatment of peri-implant mucositis with a chitosan brush—A pilot randomized clinical trial. Int J Dent Hyg 2019;17:170–176.
26. Aimetti M, Mariani GM, Ferrarotti F, Ercoli E, Liu CC, Romano F. Adjunctive efficacy of diode laser in the treatment of peri-implant mucositis with mechanical therapy: A randomized clinical trial. Clin Oral Implants Res 2019;30:429–438.
27. Iorio-Siciliano V, Blasi A, Stratul SI, et al. Anti-infective therapy of peri-implant mucositis with adjunctive delivery of a sodium hypochlorite gel: A 6-month randomized triple-blind controlled clinical trial. Clin Oral Investig 2020;24:1971–1979.
28. Menezes KM, Fernandes-Costa AN, Silva-Neto RD, Calderon PS, Gurgel BC. Efficacy of 0.12% chlorhexidine gluconate for non-surgical treatment of peri-implant mucositis. J Periodontol 2016;87:1305–1313.
29. Porras R, Anderson GB, Caffesse R, Narendran S, Trejo PM. Clinical response to 2 different therapeutic regimens to treat peri-implant mucositis. J Periodontol 2002;73:1118–1125.
30. Thöne-Mühling M, Swierkot K, Nonnenmacher C, Mutters R, Flores-de-Jacoby L, Mengel R. Comparison of two full-mouth approaches in the treatment of peri-implant mucositis: A pilot study. Clin Oral Implants Res 2010;21:504–512.

31. Galofré M, Palao D, Vicario M, Nart J, Violant D. Clinical and microbiological evaluation of the effect of Lactobacillus reuteri in the treatment of mucositis and peri-implantitis: A triple-blind randomized clinical trial. J Periodontal Res 2018;53:378–390.
32. Peña M, Barallat L, Vilarrasa J, Vicario M, Violant D, Nart J. Evaluation of the effect of probiotics in the treatment of peri-implant mucositis: A triple-blind randomized clinical trial. Clin Oral Investig 2019;23:1673–1683.
33. Hallström H, Persson GR, Lindgren S, Olofsson M, Renvert S. Systemic antibiotics and debridement of peri-implant mucositis. A randomized clinical trial. J Clin Periodontol 2012;39:574–581.
34. Deeb MA, Alsahhaf A, Mubaraki SA, Alhamoudi N, Al-Aali KA, Abduljabbar T. Clinical and microbiological outcomes of photodynamic and systemic antimicrobial therapy in smokers with peri-implant inflammation. Photodiagnosis Photodyn Ther 2020;29:101587.
35. Bunk D, Eisenburger M, Häckl S, Eberhard J, Stiesch M, Grischke J. The effect of adjuvant oral irrigation on self-administered oral care in the management of peri-implant mucositis: A randomized controlled clinical trial. Clin Oral Implants Res 2020;31:946–958.
36. Pulcini A, Bollaín J, Sanz-Sánchez I, et al. Clinical effects of the adjunctive use of a 0.03% chlorhexidine and 0.05% cetylpyridinium chloride mouth rinse in the management of peri-implant diseases: A randomized clinical trial. J Clin Periodontol 2019;46:342–353.
37. Philip J, Laine ML, Wismeijer D. Adjunctive effect of mouthrinse on treatment of peri-implant mucositis using mechanical debridement: A randomized clinical trial. J Clin Periodontol. 2020;47:883–891.
38. Abduljabbar T, Javed F, Kellesarian SV, Vohra F, Romanos GE. Effect of Nd:YAG laser-assisted non-surgical mechanical debridement on clinical and radiographic peri-implant inflammatory parameters in patients with peri-implant disease. J Photochem Photobiol B 2017;168:16–19.
39. Schwarz F, Sculean A, Rothamel D, Schwenzer K, Georg T, Becker J. Clinical evaluation of an Er:YAG laser for nonsurgical treatment of peri-implantitis: A pilot study. Clin Oral Implants Res 2005;16:44–52.
40. Sahm N, Becker J, Santel T, Schwarz F. Non-surgical treatment of peri-implantitis using an air-abrasive device or mechanical debridement and local application of chlorhexidine: A prospective, randomized, controlled clinical study. J Clin Periodontol 2011;38:872–878.
41. John G, Sahm N, Becker J, Schwarz F. Nonsurgical treatment of peri-implantitis using an air-abrasive device or mechanical debridement and local application of chlorhexidine. Twelve-month follow-up of a prospective, randomized, controlled clinical study. Clin Oral Investig 2015;19:1807–1814.
42. Roccuzzo A, Klossner S, Stähli A, et al. Non-surgical mechanical therapy of peri-implantitis with or without repeated adjunctive diode laser application. A 6-month double-blinded randomized clinical trial. Clin Oral Implants Res 2022;33:900–912.
43. Renvert S, Samuelsson E, Lindahl C, Persson GR. Mechanical non-surgical treatment of peri-implantitis: A double-blind randomized longitudinal clinical study. I: Clinical results. J Clin Periodontol 2009;36:604–609.
44. Wang H, Li W, Zhang D, Li W, Wang Z. Adjunctive photodynamic therapy improves the outcomes of peri-implantitis: A randomized controlled trial. Aust Dent J 2019;64:256–262.
45. Renvert S, Lessem J, Dahlén G, Lindahl C, Svensson M. Topical minocycline microspheres versus topical chlorhexidine gel as an adjunct to mechanical debridement of incipient peri-implant infections: A randomized clinical trial. J Clin Periodontol 2006;33:362–369.
46. Renvert S, Lessem J, Dahlén G, Renvert H, Lindahl C. Mechanical and repeated antimicrobial therapy using a local drug delivery system in the treatment of peri-implantitis: A randomized clinical trial. J Periodontol 2008;79:836–844.
47. Bassetti M, Schär D, Wicki B, et al. Anti-infective therapy of peri-implantitis with adjunctive local drug delivery or photodynamic therapy: 12-month outcomes of a randomized controlled clinical trial. Clin Oral Implants Res 2014;25:279–287.
48. Machtei EE, Frankenthal S, Levi G, et al. Treatment of peri-implantitis using multiple applications of chlorhexidine chips: A double-blind, randomized multi-centre clinical trial. J Clin Periodontol 2012;39:1198–1205.
49. Machtei EE, Romanos G, Kang P, et al. Repeated delivery of chlorhexidine chips for the treatment of peri-implantitis: A multicenter, randomized, comparative clinical trial. J Periodontol 2021;92:11–20.
50. Merli M, Bernardelli F, Giulianelli E, et al. Short-term comparison of two non-surgical treatment modalities of peri-implantitis: Clinical and microbiological outcomes in a two-factorial randomized controlled trial. J Clin Periodontol 2020;47:1268–1280.
51. Shibli JA, Ferrari DS, Siroma RS, Figueiredo LC, Faveri M, Feres M. Microbiological and clinical effects of adjunctive systemic metronidazole and amoxicillin in the non-surgical treatment of peri-implantitis: 1 year follow-up. Braz Oral Res 2019;33(suppl 1):e080.
52. Blanco C, Pico A, Dopico J, Gándara P, Blanco J, Liñares A. Adjunctive benefits of systemic metronidazole on non-surgical treatment of peri-implantitis. A randomized placebo-controlled clinical trial. J Clin Periodontol 2022;49:15–27.
53. Gomi K, Matsushima Y, Ujiie Y, et al. Full-mouth scaling and root planing combined with azithromycin to treat peri-implantitis. Aust Dent J 2015;60:503–510.
54. De Waal YCM, Vangsted TE, Van Winkelhoff AJ. Systemic antibiotic therapy as an adjunct to non-surgical peri-implantitis treatment: A single-blind RCT. J Clin Periodontol 2021;48:996–1006.
55. Liñares A, Sanz-Sánchez I, Dopico J, Molina A, Blanco J, Montero E. Efficacy of adjunctive measures in the non-surgical treatment of peri-implantitis: A systematic review. J Clin Periodontol 2023;50(suppl 26):s224–s243.
56. Romandini M, Laforí A, Pedrinaci I, et al. Effect of sub-marginal instrumentation before surgical treatment of peri-implantitis: A multi-centre randomized clinical trial. J Clin Periodontol 2022;49:1334–1345.
57. Schwarz F, Jepsen S, Obreja K, Galarraga-Vinueza ME, Ramanauskaite A. Surgical therapy of peri-implantitis. Periodontol 2000 2022;88:145–181.
58. Karlsson K, Trullenque-Eriksson A, Tomasi C, Derks J. Efficacy of access flap and pocket elimination procedures in the management of peri-implantitis: A systematic review and meta-analysis. J Clin Periodontol 2022;50(suppl 26):s244–s284.
59. Heitz-Mayfield LJA, Salvi GE, Mombelli A, Faddy M, Lang NP. Anti-infective surgical therapy of peri-implantitis. A 12-month prospective clinical study. Clin Oral Implants Res 2012;23:205–210.
60. Hallström H, Persson GR, Lindgren S, Renvert S. Open flap debridement of peri-implantitis with or without adjunctive systemic antibiotics: A randomized clinical trial. J Clin Periodontol 2017;44:1285–1293.
61. Carcuac O, Derks J, Abrahamsson I, Wennström JL, Petzold M, Berglundh T. Surgical treatment of peri-implantitis: 3-year results from a randomized controlled clinical trial. J Clin Periodontol 2017;44:1294–1303.
62. Berglundh T, Wennström JL, Lindhe J. Long-term outcome of surgical treatment of peri-implantitis. A 2-11-year retrospective study. Clin Oral Implants Res 2018;29:404–410.
63. Albaker AM, ArRejaie AS, Alrabiah M, et al. Effect of antimicrobial photodynamic therapy in open flap debridement in the treatment of peri-implantitis: A randomized controlled trial. Photodiagnosis Photodyn Ther 2018;23:71–74.
64. Papadopoulos CA, Vouros I, Menexes G, Konstantinidis A. The utilization of a diode laser in the surgical treatment of peri-implantitis. A randomized clinical trial. Clin Oral Investig 2015;19:1851–1860.
65. de Waal YC, Raghoebar GM, Meijer HJ, Winkel EG, van Winkelhoff AJ. Implant decontamination with 2% chlorhexidine during surgical peri-implantitis treatment: A randomized, double-blind, controlled trial. Clin Oral Implants Res 2015;26:1015–1023.
66. de Waal YC, Raghoebar GM, Huddleston Slater JJ, Meijer HJ, Winkel EG, van Winkelhoff AJ. Implant decontamination during surgical peri-implantitis treatment: A randomized, double-blind, placebo-controlled trial. J Clin Periodontol 2013;40:186–195.
67. Hentenaar DFM, De Waal YCM, Stewart RE, Van Winkelhoff AJ, Meijer HJA, Raghoebar GM. Erythritol airpolishing in the non-surgical treatment of peri-implantitis: A randomized controlled trial. Clin Oral Implants Res 2021;32:840–852.
68. Toma S, Brecx MC, Lasserre JF. Clinical evaluation of three surgical modalities in the treatment of peri-implantitis: A randomized controlled clinical trial. J Clin Med 2019;8:966.
69. Wilensky A, Shapira L, Limones A, Martin C. The efficacy of implant surface decontamination using chemicals during surgical treatment of peri-implantitis: A systematic review and meta-analysis. J Clin Periodontol 2023;50(suppl 26):s336–s358.
70. Ramanauskaite A, Schwarz F, Cafferata EA, Sahrmann P. Photo/mechanical and physical implant surface decontamination approaches in conjunction with surgical peri-implantitis treatment: A systematic review. J Clin Periodontol 2023;50(suppl 26):s317–s335.
71. Cha JK, Lee JS, Kim CS. Surgical therapy of peri-implantitis with local minocycline: A 6-month randomized controlled clinical trial. J Dent Res 2019;98:288–295.

72. Teughels W, Seyssens L, Christiaens V, Temmerman A, Castro AB, Cosyn J. Adjunctive locally and systemically delivered antimicrobials during surgical treatment of peri-implantitis: A systematic review. J Clin Periodontol 2023;50(suppl 26):s359–s372.
73. Heitz-Mayfield LJA, Salvi GE, Mombelli A, et al. Supportive peri-implant therapy following anti-infective surgical peri-implantitis treatment: 5-year survival and success. Clin Oral Implants Res 2018;29:1–6.
74. Sanz-Martín I, Cha JK, Sanz-Sánchez I, Figuero E, Herrera D, Sanz M. Changes in peri-implant soft tissue levels following surgical treatment of peri-implantitis: A systematic review and meta-analysis. Clin Oral Implants Res 2021;32(suppl 21):s230–s244.
75. Koldsland OC, Wohlfahrt JC, Aass AM. Surgical treatment of peri-implantitis: Prognostic indicators of short-term results. J Clin Periodontol 2018;45:100–113.
76. de Waal YC, Raghoebar GM, Meijer HJ, Winkel EG, van Winkelhoff AJ. Prognostic indicators for surgical peri-implantitis treatment. Clin Oral Implants Res 2016;27:1485–1491.
77. Carcuac O, Derks J, Abrahamsson I, Wennström JL, Berglundh T. Risk for recurrence of disease following surgical therapy of peri-implantitis-A prospective longitudinal study. Clin Oral Implants Res 2020;31:1072–1077.
78. Stuani VT, Kim DM, Nagai M, Chen CY, Sant'Ana ACP. Effectiveness and surface changes of different decontamination protocols at smooth and minimally rough titanium surfaces. J Periodontol 2021;92:704–715.
79. Stavropoulos A, Bertl K, Eren S, Gotfredsen K. Mechanical and biological complications after implantoplasty—A systematic review. Clin Oral Implants Res 2019;30:833–848.
80. Ramanauskaite A, Cafferata EA, Begic A, Schwarz F. Surgical interventions for the treatment of peri-implantitis. Clin Implant Dent Relat Res 2023;25:682–695.
81. Romeo E, Ghisolfi M, Murgolo N, Chiapasco M, Lops D, Vogel G. Therapy of peri-implantitis with resective surgery. A 3-year clinical trial on rough screw-shaped oral implants. Part I: Clinical outcome. Clin Oral Implants Res 2005;16:9–18.
82. Romeo E, Lops D, Chiapasco M, Ghisolfi M, Vogel G. Therapy of peri-implantitis with resective surgery. A 3-year clinical trial on rough screw-shaped oral implants. Part II: Radiographic outcome. Clin Oral Implants Res 2007;18:179–187.
83. Esteves Lima RP, Abreu LG, Belém FV, Pereira GHM, Brant RA, Costa FO. Is implantoplasty efficacious at treating peri-implantitis? A systematic review and meta-analysis. J Oral Maxillofac Surg 2021;79:2270–2279.
84. Bertl K, Isidor F, von Steyern PV, Stavropoulos A. Does implantoplasty affect the failure strength of narrow and regular diameter implants? A laboratory study. Clin Oral Investig 2021;25:2203–2211.
85. Chan HL, Oh WS, Ong HS, et al. Impact of implantoplasty on strength of the implant-abutment complex. Int J Oral Maxillofac Implants 2013;28:1530–1535.
86. Jepsen S, Schwarz F, Cordaro L, et al. Regeneration of alveolar ridge defects. Consensus report of group 4 of the 15th European Workshop on Periodontology on Bone Regeneration. J Clin Periodontol 2019;46(suppl 21):s277–s286.
87. Deppe H, Horch HH, Neff A. Conventional versus CO2 laser-assisted treatment of peri-implant defects with the concomitant use of pure-phase beta-tricalcium phosphate: A 5-year clinical report. Int J Oral Maxillofac Implants 2007;22:79–86.
88. Isler SC, Unsal B, Soysal F, Ozcan G, Peker E, Karaca IR. The effects of ozone therapy as an adjunct to the surgical treatment of peri-implantitis. J Periodontal Implant Sci 2018;48:136–151.
89. Ramanauskaite A, Becker K, Caferatta EA, Schwarz F. Clinical efficacy of guided bone regeneration in peri-implantitis defects. A network meta-analysis. Periodontology 2000 2023;93:236–253.
90. Schwarz F, Sahm N, Bieling K, Becker J. Surgical regenerative treatment of peri-implantitis lesions using a nanocrystalline hydroxyapatite or a natural bone mineral in combination with a collagen membrane: A four-year clinical follow-up report. J Clin Periodontol 2009;36:807–814.
91. Monje A, Pons R, Vilarrasa J, Nart J, Wang HL. Significance of barrier membrane on the reconstructive therapy of peri-implantitis: A randomized controlled trial. J Periodontol 2023;94:323–335.
92. Roos-Jansåker AM, Persson GR, Lindahl C, Renvert S. Surgical treatment of peri-implantitis using a bone substitute with or without a resorbable membrane: A 5-year follow-up. J Clin Periodontol 2014;41:1108–1114.
93. Regidor E, Ortiz-Vigón A, Romandini M, Dionigi C, Derks J, Sanz M. The adjunctive effect of a resorbable membrane to a xenogeneic bone replacement graft in the reconstructive surgical therapy of peri-implantitis: A randomized clinical trial. J Clin Periodontol 2023;50:765–783.
94. Donos N, Calciolari E, Ghuman M, Baccini M, Sousa V, Nibali L. The efficacy of bone reconstructive therapies in the management of peri-implantitis. A systematic review and meta-analysis. J Clin Periodontol 2023;50(suppl 26):s285–s316.
95. Isler SC, Soysal F, Ceyhanlı T, Bakırarar B, Unsal B. Efficacy of concentrated growth factor versus collagen membrane in reconstructive surgical therapy of peri-implantitis: 3-year results of a randomized clinical trial. Clin Oral Investig 2022;26:5247–5260.
96. Roccuzzo M, Pittoni D, Roccuzzo A, Charrier L, Dalmasso P. Surgical treatment of peri-implantitis intrabony lesions by means of deproteinized bovine bone mineral with 10% collagen: 7-year-results. Clin Oral Implants Res 2017;28:1577–1583.
97. Schwarz F, Sahm N, Schwarz K, Becker J. Impact of defect configuration on the clinical outcome following surgical regenerative therapy of peri-implantitis. J Clin Periodontol 2010;37:449–455.
98. Aghazadeh A, Persson RG, Renvert S. Impact of bone defect morphology on the outcome of reconstructive treatment of peri-implantitis. Int J Implant Dent 2020;6:33.
99. Roccuzzo M, Mirra D, Pittoni D, Ramieri G, Roccuzzo A. Reconstructive treatment of peri-implantitis infrabony defects of various configurations: 5-year survival and success. Clin Oral Implants Res 2021;32:1209–1217.
100. Derks J, Ortiz-Vigón A, Guerrero A, et al. Reconstructive surgical therapy of peri-implantitis: A multicenter randomized controlled clinical trial. Clin Oral Implants Res 2022;33:921–944.
101. Monje A, Schwarz F. Principles of combined surgical therapy for the management of peri-implantitis. Clin Adv Periodontics 2022;12:57–63.
102. Wang CW, Ashnagar S, Gianfilippo RD, Arnett M, Kinney J, Wang HL. Laser-assisted regenerative surgical therapy for peri-implantitis: A randomized controlled clinical trial. J Periodontol 2021;92:378–388.
103. de Tapia B, Valles C, Ribeiro-Amaral T, et al. The adjunctive effect of a titanium brush in implant surface decontamination at peri-implantitis surgical regenerative interventions: A randomized controlled clinical trial. J Clin Periodontol 2019;46:586–596.
104. Schwarz F, John G, Schmucker A, Sahm N, Becker J. Combined surgical therapy of advanced peri-implantitis evaluating two methods of surface decontamination: A 7-year follow-up observation. J Clin Periodontol 2017;44:337–342.
105. Roccuzzo M, Bonino L, Dalmasso P, Aglietta M. Long-term results of a three arms prospective cohort study on implants in periodontally compromised patients: 10-year data around sandblasted and acid-etched (SLA) surface. Clin Oral Implants Res 2014;25:1105-–1112.
106. Schwarz F, Ramanauskaite A. It is all about peri-implant tissue health. Periodontol 2000 2022;88:9–12.
107. Karlsson K, Derks J, Wennström JL, Petzold M, Berglundh T. Health economic aspects of implant-supported restorative therapy. Clin Oral Implants Res 2022;33:221–230.
108. Agari K, Le B. Successive reimplantation of dental implants into sites of previous failure. J Oral Maxillofac Surg 2020;78:375–385.