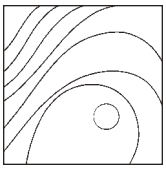
Guidelines for the Diagnosis and Treatment of Peri-implant Diseases



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Although some risk factors of peri-implant disease are well defined, the lack of efficient and predictable approaches to treat peri-implantitis has created difficulty in the management of those complications. The aim of this review was to evaluate the reliability of the diagnosis methods and to provide a set of guidelines to treat peri-implant diseases. A search of PubMed and a hand search of articles related to peri-implant diseases were conducted up to August 2013. A summary of the current methods for the diagnosis of peri-implantitis, its potential risk factors, and a flow chart to guide the clinical management of these conditions are presented.
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Implant therapy is a predominant and useful armamentarium for patients with missing teeth. An increased number of procedures with no clear etiology of complications has led to an increase in cases of peri-implant disease. Peri-implant diseases may occur in two forms, peri-implant mucositis and peri-implantitis.[1] *Peri-implant mucositis* is an inflammatory lesion that resides in the soft tissue surrounding a dental implant without signs of bone loss following the initial bone remodeling. In contrast, *peri-implantitis* also affects the supporting bone, causing progressive bone loss beyond the normal biologic remodeling.[2] Peri-implantitis appears in combination with marginal bone loss (MBL) greater than 3 mm, bleeding on probing (BOP) or purulence, or both.[3] It has been estimated that 12% to 43% of implants have bone loss in combination with BOP,[4] with a prevalence of approximately 10% for implant-supported single crowns.[5]

Different cross-sectional studies have investigated potential risk indicators for peri-implant diseases, including poor oral hygiene, smoking, pre- or coexisting periodontitis,

diabetes, genetic traits, alcohol consumption, and/or absence of keratinized mucosa and implant surface. However, there is not enough evidence to establish these factors as the true risk factors.[6] Acknowledging that, it is important to remember that an appropriate implant therapy must always include an appropriate preventive approach to avoid future complications, accompanied by professional maintenance protocols and the patient's personal oral hygiene procedures.

Implant survival, defined as the retention of the implant at the site, should not be the main goal of the clinician's practice. Instead, success should be the goal of treatment. *Implant success* has classically been defined as a crestal bone loss lower than 1.5 mm during the first year after loading and 0.2 mm annually thereafter.[7] A more recent definition describes implant success (or a healthy implant) as the ideal clinical condition for implants serving as prosthetic abutments for at least 12 months after prosthetic loading in the absence of pain, mobility, radiographic bone loss beyond 2 mm from initial surgery, and exudates around the implant.[8] However, those definitions do not include esthetic success,[9] patient satisfaction in terms of discomfort and paresthesia, satisfaction with appearance, and ability to chew/taste.[10] Even though esthetics and patient satisfaction have to be considered in the treatment plan, they are not strictly the aim of this review.

Diagnosis of peri-implant diseases requires the measurement of probing depth (PD), BOP, and clinical attachment level (CAL) and a radiographic examination to further analyze the characteristic and extent of bone loss, if present.[6] Different treatment strategies, often combined, have been proposed, including mechanical debridement, pharmaceutical therapy, and surgical procedures (eg, open-flap debridement, smoothing the implant surface, and "decontamination" or "detoxification" of the implant surface followed by resective and/or bone regenerative procedures to correct the anatomical conditions for improving plaque control and for eliminating the pathologic peri-implant pockets).[11] Although nonsurgical treatments are recommended for mucositis lesions, their use has been proven not to be effective in peri-implantitis lesions.[12] In such scenarios, open-flap debridement, including mechanical or chemical methods for decontamination,[13] and guided bone regeneration (GBR) are recommended.[14,15]

The lack of agreement on etiology and a more precise disease classification leads clinicians to propose random therapies. Hence, the aim of this review was to analyze the diagnosis and risk indicators for peri-implant diseases and to propose a clinically based guide for the management of these conditions.

Diagnosis of peri-implant diseases

Periodontal implant complications can be biologic, technical, and/or esthetic in origin. Prognosis, once disease is already present, will be determined by the ability to restore those characteristics.

Clinical parameters

In general, based on longitudinal clinical studies, the time of prosthesis placement should be chosen to establish baseline criteria both at a clinical and radiographic level representing homeostasis.[1] It is evident that recorded baseline data will be the reference from which the development of peri-implant disease can be recognized and followed in subsequent examinations. Changes from implant placement to prosthesis delivery also must be considered. However, changes in that early phase should not be considered to be peri-implant disease but rather physiologic remodeling or early implant failure, neither of which are the purpose of this review. During follow-up visits, if changes in the clinical parameters indicate pathology/disease (BOP, increased CAL/PD), the clinician is encouraged to take new radiographs to evaluate possible bone loss and treat the case accordingly, depending on the criteria that will be discussed below.

Implant mobility

If the mobility is due to abutment loosening, occlusion should be checked and adjusted. Mobility of an implant clearly indicates the complete lack of osseointegration and that the implant should be removed.[6] Lack of clinical detection of implant mobility is a condition often

used to describe implant integration.[8] However, that does not mean an absolute absence of mobility, since a healthy implant may move up to 75 µm (which cannot be clinically detected).[16]

Probing depth

A reliable reference point such as the implant shoulder or the implant-abutment junction should be used to determine the CAL/PD, and these measurements should be repeated over time. This reference point to measure CAL can change by implant design, system, and placement level. So, PD is most widely accepted. On the other hand, probing around an implant also may be influenced by different parameters, such as the quality of the histologic soft tissue seal, the type and surface of the implant and suprastructure,[17] and probing force (0.2 to 0.3 N).[18–21] No data exist regarding how probe material (metal versus plastic) or design influence peri-implant probing.[6]

Generally, implant PD ranges from 3 to 4 mm and this can vary between implant systems, esthetic placement depths, bone levels to adjacent teeth, healing time, surgical protocol (one or two stages), and loading protocol.[22,23] Although these studies did not include concepts such as platform switching, which may lead to lower measurements, it is widely accepted that the soft tissue thickness down to the crest of the bone is a constant in a healthy tissue with no more than 2 mm of radiographic bone loss from the implant platform. A baseline PD measurement after the initial soft tissue healing after loading should be established and monitored since an increase in PD over time has clearly been associated with attachment and bone loss in experimental peri-implantitis studies.[24,25] A 6-mm peri-implant pocket has been suggested as the cut-off indicative of peri-implant disease if accompanied by other signs (eg, radiographic radiolucencies, purulent exudate, bleeding) and/or symptoms (eg, discomfort, pain).[26] Sulcus depths greater than 5 to 6 mm around implants have a greater incidence of anaerobic bacteria and may require intervention in the presence of inflammation or exudate.[8] Therefore, PD should not be considered as an absolute and isolated diagnostic tool.

Bleeding on probing

Presence of BOP is a useful parameter for the diagnosis of mucosal inflammation. Lang et al demonstrated that healthy peri-implant sites had an absence of BOP, while there was increased BOP at mucositis (67%) and peri-implantitis (91%) sites.[18] Furthermore, BOP is considered a valuable parameter for diagnosing peri-implant disease with a high negative predictive value. Moreover, it also has a positive predictive value of 100%.[27] Therefore, absence of BOP is a good indicator of stable peri-implant conditions.[6] In contrast, presence of BOP clearly suggests a diagnosis of peri-implant mucositis. Hence, the clinician must evaluate other clinical parameters, such as PD, exudates, and radiographic data, to rule out the presence of peri-implantitis.[1]

Suppuration

The presence of pus in the peri-implant sulcus, either spontaneously or after probing, indicates the presence of an infection and/or inflammatory lesion.[26,28] This is a common finding in peri-implantitis sites.[1]

Radiographic evaluation

It is assumed that initial bone loss due to adaptive remodeling and creation of biologic width at the marginal level of an implant is normal. Platform switching may reduce/eliminate that MBL.[29] It is required, however, to periodically evaluate (every 6 months to 1 year) both clinical and radiographic data from a fixed reference point (eg, implant shoulder or implant-abutment junction) to the interproximal bone level.[6] Although bone loss in buccal and lingual sites is difficult to detect radiographically, standardized and high-quality radiographs always should be performed for the early detection of peri-implantitis.[8]

Several different radiographic techniques can be used to evaluate the peri-implant structures, such as intraoral radiography, panoramic tomography, computed tomography (CT), cone beam computed tomography (CBCT), and volume imaging.[30]

Conventional radiography

Conventional radiography includes intraoral and panoramic techniques. Both techniques are widely used for peri-implant diagnosis and both are reliable to assess bone levels around dental implants.[31] Although panoramic radiographs offer a view of both jaws, intraoral radiographs provide less magnification/distortion, a more detailed picture, and higher resolution.[32] Nonetheless, both methods underestimate the MBL, are unable to monitor facial and lingual bone levels, and have low sensitivity in the detection of early bone loss.[33] Hence, a thorough clinical examination is mandatory for complete diagnosis.

Subtraction radiography

Subtraction radiography allows the clinician to digitally compare periapical intraoral[34] and panoramic radiographs[35] from different time points in order to obtain information on MBL and minor variations in bone density that would be more challenging to detect with conventional radiographic analysis.

Computed tomography

Due to the limitations of conventional radiography, the use of three-dimensional (3D) images to diagnose the peri-implant surrounding architecture has increased during the past few years. The accuracy and quality of such techniques have been documented,[36] which make them a promising, useful technology for assessing bone levels on implants. However, the application of these new technologies needs to be further studied. In addition to the possibility of measuring the bone defects in three planes, CT and digital volume tomography (DVT) showed minimal deviation compared to direct peri-implant defect measurements, whereas the DVT scans were found to offer the best imaging quality with less scattering, which may help to overcome this technical limitation.[36]

Risk indicators

Oral hygiene

Poor oral hygiene and peri-implant alveolar bone loss have been correlated with an increased odds ratio (OR) of 14.3 (95% confidence interval [CI]: 2.0 to 4.1).[37,38] So, in all cases, plaque detection and oral hygiene instructions should be performed at every follow-up visit, including teeth cleaning, as needed.

History of periodontitis

Patients with a history of periodontitis are more susceptible to peri-implant diseases[39] and peri-implant MBL.[40] Therefore, before placing dental implants, the existing periodontitis condition must be adequately treated. If not, those infected sites (pocket probing depth [PPD] > 5 mm) may act as reservoirs of pathogens to colonize implant surfaces, which represent a significant risk for the development of peri-implantitis and implant loss.[41] However, it is important to mention that, despite similarities in clinical features, etiology, and host response to biofilms,[42] histopathologic differences exist between peri-implantitis and periodontitis lesions.[43]

Smoking and alcohol consumption

The negative effect of cigarette smoking on the peri-implant tissues is well documented, not only for peri-implant mucositis but also for MBL and peri-implantitis.[40,44–46]

Regarding alcohol consumption, it has been reported that peri-implant MBL can be significantly related to a daily consumption of > 10 g of alcohol. In that study, MBL was even greater in patients taking alcohol compared with tobacco users.[40]

Prosthetic rehabilitation

Peri-implant disease also may be initiated by prosthodontic/iatrogenic factors such as cement remnants, inadequate restoration-abutments seating, overcontoured restorations, presence of cantilevers, and technical complications.[1] Implant malpositioning, other than causing esthetic complications, also may increase the risk of peri-implant disease development. Clinicians must consider on an individual basis the influence malpositioning has on whether the patient is experiencing a true peri-implant disease or whether the symptoms are an expected consequence of the

malposition (eg, fenestration, dehiscences). When the implant malposition is so severe that the implant cannot be adequately restored, implant removal and placement of a new one in a position closer to ideal should be considered as the best long-term treatment option.

Occlusal overloading seems to play a role as a cofactor in the initiation and progression of peri-implant diseases.[47] Miyata et al in a monkey study found that traumatic occlusion plays a role in bone breakdown around the implant. The authors proposed an excessive height threshold of the superstructures at which peri-implant tissue breakdown may start (approximately 180 µm), even when there was no inflammation in the peri-implant tissue.[48] On the contrary, a recent study in Labrador dogs concluded that, in the presence of peri-implant mucosal health, excessive occlusal load did not result in loss of osseointegration or MBL when compared with non-loaded implants. However, no force measurements were performed in this study.[49]

Other indicators of occlusal overloading such as lost or loosening screws, prosthesis detachment, porcelain fractures, etc, help the clinician in identifying the presence of a problem before the supporting bone is affected.

Implant surface

Many different implant surfaces and their new modifications are commercially available.[50,51] Interestingly, according to recent evidence, there is not enough data to ensure that implant surface characteristics can have a significant effect on the initiation of peri-implantitis.[52] However, rough surfaces tend to accumulate more biofilm and are more difficult to clean. Therefore, it has been suggested that, once exposed to the oral environment, rough surfaces are more likely to develop peri-implantitis. They are also more susceptible to disease progression than smooth or minimally rough surfaces.[53,54]

Keratinized mucosa

Regarding the role of keratinized mucosa (KM) or attached mucosa (AM) in maintaining healthy tissues around dental implants, no clear evidence is yet available. Some studies have reported no association between the absence of an adequate width of KM (ie, ≥ 2 mm) or AM (ie, ≥ 1 mm) and peri-implant diseases.[28] However, evidence for the opposite also is available.[55–57] For instance, the 3rd EAO Consensus Conference (European Association for Osseointegration) concluded that evidence in support of the need for keratinized tissues around implants to maintain health and tissue stability is limited,[58] similarly to a more recent systematic review that was unable to draw a definitive conclusion.[59] Nonetheless, it seems to be well established that the absence of adequate KM promotes higher gingival inflammation and plaque accumulation, especially in posterior implants,[60] which would lead to more susceptibility to tissue inflammation, and, therefore, increase the risk for peri-implant disease. Similar results have been reported in a 5-year multicenter study.[61]

Biologic markers

In recent years, microbiologic tests and peri-implant crevicular fluid and saliva analyses to detect different biologic markers (eg, cytokines, enzymes, matrix metalloproteinases) have been introduced. They could enhance the prognostic characteristics of other parameters, orient for an early diagnosis, and, therefore, allow for prompt treatment of peri-implant infections.[27,62] However, even though current evidence shows promising results, prospective longitudinal studies are needed, and, consequently, the search for markers predicting peri-implant diseases continues.[6]

Treatment of peri-implant diseases

Continuing with the proposed diagnosis flow chart (Fig 1), several clinical and radiographic parameters must be considered before proposing any treatment. Every one of them will provide valuable information about the etiology, risk factors, and severity of the disease and, therefore, will guide clinicians in the decision process to provide the most reliable treatment. No single parameter (except mobility) is intended to substitute the others; all have to be studied as key constituents of the whole process.

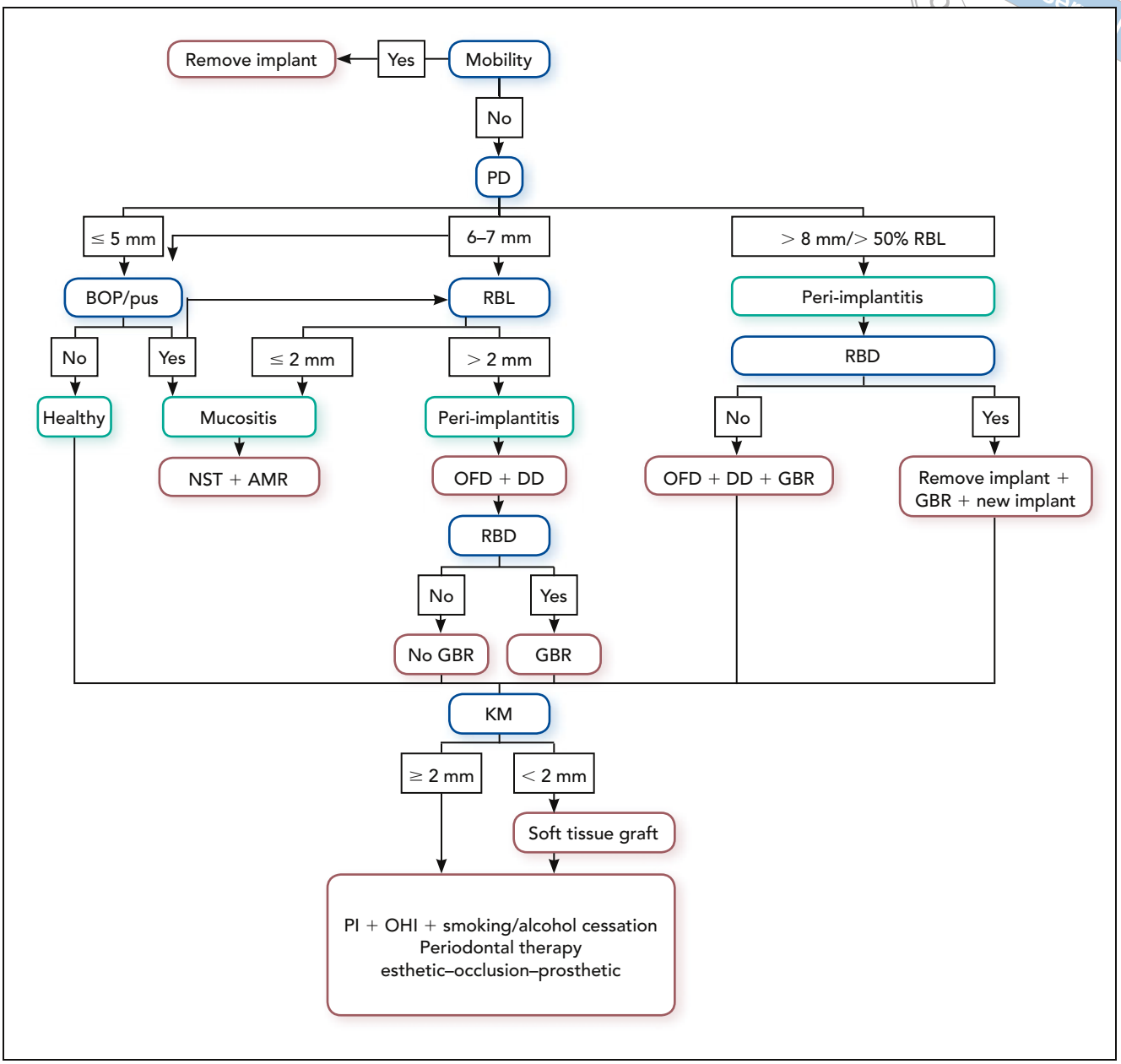

Fig 1 *Guidelines for the treatment of peri-implant diseases. PD = probing depth; BOP = bleeding on probing; NST = nonsurgical therapy; AMR = antimicrobial mouthrinse; RBL = radiographic bone loss; OFD = open-flap debridement; DD = detoxification/decontamination; RBD = retentive bone defect; GBR = guided bone regeneration; KM = keratinized mucosa; PI = Plaque Index; OHI = oral hygiene instructions.*

First, a clinically detectable mobile implant after prosthesis delivery must be removed, because there is no chance for the implant to be osseointegrated again. It is very important to be sure that the mobility is due to the implant itself and not to the prosthesis or abutments. Any coexisting periodontitis lesions must be treated before or simultaneously to the peri-implant lesions. In all cases, plaque detection and oral hygiene instructions should be discussed with the patient. Similarly, smoking and/or alcohol consumption cessation counseling is always recommended. Occlusal overloading, premature contacts, and other

prosthodontic issues, such as inadequate restoration-abutment seating, should be analyzed and corrected at every follow-up visit.

According to the International Congress of Oral Implantologists (ICOI) Pisa Consensus Conference on "Implant Success, Survival, and Failure," *optimum health* is defined as an implant with neither BOP nor radiographic bone changes higher than 2 mm from baseline, no mobility, and no pain upon function.[8] However, sometimes a healthy implant might be a treatment failure if it does not allow for a functional and esthetic reconstruction, in which case it should not be considered to be a successful treatment. Different approaches can be designed to deal with such cases, from changes in prosthetic design to use of pink porcelain, soft tissue strategies, and even implant removal.

Diagnosis of mucositis, considering positive BOP with increased or normal PD but no radiographic bone changes, leads to a conservative treatment. Peri-implant mucositis is usually a reversible condition that can be successfully treated with nonsurgical therapy and the adjunctive use of antimicrobial mouthrinses.[12,63] Food impaction, cement retention, overcontoured restorations, and any kind of irritative agents should be explored and removed.

When PD lower than 5 mm and positive BOP (with or without purulent exudates) are detected, a radiographic examination is also required. Presence of radiographic bone defects can hardly be detectable at this point. So, the initial therapy should be for a mucositis problem followed by close monitoring. If radiographic bone loss is obvious (≥ 2 mm), an open-flap debridement is needed. This procedure should always be accompanied by a proper surface decontamination and detoxification.[13] Such decontamination/detoxification can be performed by either mechanical or chemical methods. Its aims are to remove all bacteria deposits, facilitate soft tissue re-accommodation, and limit and minimize future plaque deposition that would reinitiate the disease episode. Grafting procedures at this time may be indicated in cases of retentive bone defects while bone recontouring is indicated in irregular bone defects. If prosthesis replacement is required, prosthesis removal, a closed bone-grafting procedure, and delivery of the new prosthesis after 6 months is the choice when the crest of the bone is near the implant shoulder.

Special consideration should be given to cases of implants with a PD ≥ 8 mm or more than 50% of the implant length. According to a recent systematic review, reosseointegration is unpredictable and is not achieved for the entire previously contaminated implant surface.[64] Therefore, if a deep circumferential defect is present, although GBR could be performed, extraction of the problematic implant followed by GBR to reconstruct the lost bone and placement of a new implant in the regenerated bone is a good, safer, and more predictable alternative. Vertical bone defects are a risk that may be overcome by early implant explantation. Otherwise, clinical scenarios with vertical bone defects can be more challenging to treat. In contrast, cases with nonretentive bone defects should be treated by open-flap debridement followed by mechanical and/or chemical decontamination/detoxification. Prosthesis removal followed by a closed bone-grafting procedure and delivery of the new prosthesis after 6 months can benefit the GBR procedure.

According to the literature, no single method of surface decontamination is superior.[13,65] Similarly, regenerative procedures such as bone graft techniques, with or without the use of barrier membranes, result in various degrees of success, although these techniques are not aimed to resolve the disease but to restore the osseous defect.[65]

In any of the afore-mentioned cases, if KM is deficient (< 2 mm), a corrective soft tissue grafting procedure could be performed. Free gingival grafts,[66] allografts,[67] or collagen membranes[68] can be used.

Finally, we should remark once again that every implant patient, healthy or not, should be carefully instructed in oral hygiene techniques, counseled on the risk factors for peri-implant disease development, such as use of tobacco and alcohol or the presence of coexisting or previously treated periodontitis, and strongly encouraged to adhere closely to the maintenance regimen. Of course, this also requires from clinicians a careful peri-implant diagnosis, treatment planning, and maintenance protocols, all of which are key to achieving successful implant therapy.

Conclusions

With the limitations of lacking an agreement on etiology and a more precise disease classification, a flowchart based on current evidence and daily clinical practice is proposed for the treatment of peri-implant diseases. Based on three PD ranges (≤ 5 mm, 6 to 7 mm, and ≥ 8 mm), BOP, and radiographic bone loss (ie, ≤ 2 mm; > 2 mm), different diagnoses are depicted and treatment modalities proposed. These include nonsurgical therapy, open-flap debridement, decontamination/detoxification, and GBR. In some cases, such as those with implant mobility, extreme malpositioning, or extensive but retentive bone loss, implant removal is recommended. Bone defect shape and KM should also be considered and corrected. Plaque Index, oral hygiene, periodontal status, smoking/alcohol intake, esthetics, occlusion, and the prosthesis must be checked and corrected, if needed.

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