Comment

Revisiting peri-implant diseases in order to rethink the future of compromised dental implants: Considerations, perspectives, treatment, and prognosis

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Once present, peri-implantitis is difficult to fully eliminate. Surgical interventions show some promising results in fighting the disease, but for the time being, prevention remains the strongest tool.

Introduction

Peri-implant disease (PID) is a global term for biological responses to local aggression on tissues around dental implants. Similar to gingivitis and periodontitis, peri-implant mucositis (PIM) and peri-implantitis (PI) were first coined in 1993 at the 1st European Workshop on Periodontology in a consensus report.[1] Peri-implant disease encapsulates all inflammatory and immune system-mediated responses around the tissues of the osseointegrated implant. Peri-implant mucositis refers to the reversible inflammation of these tissues. Peri-implantitis presents the same inflammatory status involving soft and hard tissues, which may progress to severe bone loss in its advanced phases.[1,2] At the 2017 World Workshop on the Classification of Periodontal and Peri-Implant Diseases and Conditions,[2] the definition of PI was updated to a plaque (polymicrobial)-associated disease that occurs around the osseointegrated implant.[1–4]

Peri-implant health, peri-implant mucositis and peri-implantitis

The expected clinical scenario in a healthy peri-implant location is an erythema-free area with no bleeding on probing (BoP) and no edema or suppuration. The main assessment tools are visual/clinical observation and palpation. If erythema, edema or pus are present, a periodontal/peri-implant probe should be used to assess the probing depth (PD).[2]

Peri-implant mucositis is clinically characterized by erythema, edema and/or suppuration. After gentle probing, bleeding is often observed on the implant sulcus. An increased PD is mostly due to tissue swelling, as mucositis does not present with bone loss. Plaque/biofilm is the main etiological factor for PIM, as observed in animal and human experimental studies.[2] Even though there are some human studies evidencing that mucositis can resolve after treatment, some systematic reviews claim that total disease resolution is not predictable, and PIM progression to PI tends to occur.[5–7]

Peri-implantitis is believed to be the progression of PIM, showing the same clinical characteristics, but greater PD (>6 mm) and radiographic bone loss (RBL). Bone loss is usually directly related to the progression and severity of the disease. Poor biofilm control by the patient and the lack of compliance with follow-up recommendations have been shown to be risk-increasing behaviors.[6–10] Some other factors may play a role in the development of PI, such as smoking habits and diabetes[2]; occlusal overload, prosthodontic rehabilitation characteristics, the presence or absence of keratinized mucosa, titanium (Ti) particles, abutment micromovements, biocorrosion, and cement remnants seem to play some role in the progression of the disease, but high-quality studies fail to accurately determine the effects of these factors on PI progression.[2,5,11–13]

Prevalence and pathogenesis

Since dental implants are reported to have high survival rates (90–95% at >5 years),[14–17] it is only natural that we are observing an upward trend in their usage for the replacement of missing teeth. With regard to the above, it is logical to assume that the rate of complications will follow a similar trend. It has been clinically confirmed, with an increase in PI cases over the last 25 years.[1,2,5] Recent studies estimate a prevalence of approx. 20% for PI, which can rise up to around 47% if the incidence of PIM is considered.[17,18]

Over the last decade, some new theories have arisen about the true pathogenesis of PID. The main theory assumes that a foreign body reaction (FBR) always occurs around the osseointegrated implant, causing chronic low-grade inflammation. With the aggravation of this latent inflammation, peri-implant bone loss will be the ultimate result.[19] The main difference between the current, widely accepted theory and the FBR theory is the relevance of dental plaque accumulation and bacterial implication in the pathogenesis of the disease. The determination of one or another factor could dictate a paradigm shift in the treatment options for PI. There is still a lack of scientific evidence for FBR to be a unidirectional cause of PI, and it seems that plaque accumulation should not be underestimated.[20]

Treatment lines for peri-implant disease

According to the current knowledge about PI, the progression of the disease is plaque-dependent. Thus, treatment should focus on controlling and eliminating plaque.[1,5] With that being said, PI therapy comprises some initial steps, i.e., infection control and non-surgical interventions by the removal of plaque, through sub- and supragingival debridement, and finally follow-up evaluation. This treatment approach has been proven to be insufficient for treating true PI lesions, although it is successfully applied in the case of PIM.[2,5]

For true PI lesions, another stage is required, namely the surgical therapy phase. Surgical treatment encompasses flap elevation, implant surface detoxification/decontamination, and pocket/granulation tissue elimination if required. If it is justified by the type of the existing bone defect, a regenerative approach with the use of the available biomaterials and a membrane for guided bone regeneration (GBR) can also be applied.[5,11]

Several adjunctive therapies have been thoroughly studied, such as laser mono/combined application, the usage of antibiotics (locally or systemically), and alternative decontamination methods (implantoplasty, air polishing, the use of an ultrasonic apparatus and Ti brushes, and electrolytic decontamination with GalvoSurge®).[5]

It has been widely recognized that PI disease prevention is the best available choice, since treatment options are still not totally predictable and reliable. The best actions for preventing the development of PID, as per the latest guidelines,[11] are: (1) the proper evaluation of soft and hard tissues that will receive the future dental implant (the presence or absence of keratinized mucosa, the width of the available mucosa, the presence or absence of any gingival/bony defect, and bone availability for implant placement); (2) proper three-dimensional (3D) implant placement; (3) the proper planning and execution of the prosthodontic piece (allowing adequate cleanability for the patient); and (4) the proper establishment of a follow-up schedule for each patient, taking into consideration particular risk factors.

Adherence to oral health instructions and periodic follow-up appointments for supportive peri-implant care are believed to be key factors in maintaining peri-implant health and preventing the development of any PID.[1,2,11]

Treatment outcomes for peri-implant disease

An umbrella review published in 2022 by Martins et al. evaluated 9 systematic review articles encompassing 59 unique randomized controlled trials (RCTs).[5] The study

found that in treating PI lesions, non-surgical approaches had limited effects and could not stop the evolution of PI. Some clinical parameters might be improved, i.e., BoP and, to a lesser degree, PD. Non-surgical options were mostly recommended for treating PI within the first-stage intervention or treating PIM more efficiently. The greater the PD, the more limited the effects of non-surgical interventions appear to be. Combining non-surgical therapies with adjunctive methods (i.e., lasers and local antibiotic/antiseptic therapy) offered better clinical results, although some methods were controversial. Abrasive polishing with glycine powder, erbium-doped yttrium aluminum garnet (Er:YAG) laser application, debridement with an ultrasonic apparatus or curettes, and local antibiotics/antiseptics worked better when used in conjunction. Yet, none of them reduced the bacterial load at the implant surface enough to avoid the development of PI.[5]

Surgical techniques seem to be the best option to treat PI and hinder the development of the disease.[1,2,5,11] Resective interventions may improve clinical parameters and, to some extent, diminish the effects of inflammation (lower BoP and sulcus/pocket PD). Normally, resective techniques by themselves result in some kind of soft tissue/peri-implant tissue loss. Thus, a regenerative procedure may be recommended.[5]

Regenerative surgical techniques yield generally positive results, showing better clinical and radiographic outcomes in most high-quality studies.[21] Predicting the magnitude of improvement with any surgical technique is still difficult and disease recurrence is not uncommon. Patient-related outcome measurements are also rarely reported.[5,11] In the available literature, no specific material (i.e., membranes, bone substitutes or bioactive agents) is superior to another. No clear advantage with regard to clinical outcomes was found when comparing resective only and regenerative procedures.[5,11] Implantoplasty was the most effective implant surface decontamination method, but other concerns, such as Ti particles scattering during the procedure, still need to be investigated. The most recent European Federation of Periodontology (EFP) guidelines[11] as well as another study[1] corroborate the aforementioned findings. Table 1 summarizes the prevention, treatment and prognostic issues regarding PID.

Conclusions and considerations

As modern-day oral rehabilitation protocols for partially and fully edentulous patients rely more and more on implantology, preventing biological complications is a key factor for the success and longevity of implants. Preventing the development of PID seems to be the best path to avoid having to deal with the most serious version of the disease, PI. With the current knowledge, several steps can be taken toward the prevention of PID: (1) preoperative evaluation, especially examining the gingival and bone characteristics of the area; (2) the proper planning of implant placement in the correct 3D position (reverse

Table 1. Peri-implant disease (PID) – prevention, treatment, follow-up, and prognosis

PID	Prevention	Treatment	Follow-up	Prognosis
PIM/PI	(1) proper evaluation of soft and hard tissues that will receive the future dental implant (the presence or absence of keratinized mucosa, the width of the available mucosa, the presence or absence of any gingival/bony defect, and bone availability for implant placement); (2) proper 3D implant placement; (3) proper planning and execution of the prosthodontic piece (allowing adequate cleanability for the patient); (4) proper establishment of a follow-up schedule for each patient, considering all risk factors	–	–	extremely favorable
PIM	–	non-surgical procedures: abrasive polishing with glycine powder, Er:YAG laser application, debridement with an ultrasonic apparatus or curettes, and local antibiotics/antiseptics	moderate/severe PIM: at 3 months mild PIM: at 6 months	favorable
PI	–	non-surgical procedures: first-stage intervention non-surgical and surgical procedures (combination): abrasive polishing with glycine powder, Er:YAG laser application, debridement with an ultrasonic apparatus or curettes, and local antibiotics/antiseptics implantoplasty, Ti brushes and electrolytic decontamination with GalvoSurge®, and GBR	moderate/severe PI: at 6 weeks–3 months mild PI: at 3–6 months	poor/favorable

PIM – peri-implant mucositis; PI – peri-implantitis; 3D – three-dimensional; Er:YAG laser – erbium-doped yttrium aluminum garnet laser;Ti – titanium; GBR – guided bone regeneration.

planning); (3) correct prosthodontic planning, allowing the cleanability of the rehabilitated area; and (4) evaluating the patient comprehensively, taking into consideration their hygiene habits, as well as systemic factors, to correctly define a supportive care schedule for the maintenance of peri-implant health.

The available data on the actual treatment of PI are reliable and already provide rough guidelines for dealing with possible complications. Yet, due to the scarcity of high-quality evidence in the literature, knowledge on PI treatment still has to be expanded. In the last few years, we have witnessed great advancement with regard to understanding the physiopathology and progression pattern of the disease, but fully eliminating it still seems like a distant aspiration. Patient-related outcome measurements are also lacking in most of the available studies. Some promising decontamination methods are starting to be investigated, as well as extra surface treatment to reduce plaque accumulation on implants and abutments. The role of the potential release of Ti particles is also a point of interest, and further studies are required.

To conclude, once present, peri-implantitis is difficult to fully eliminate. Surgical interventions show some promising results in fighting the disease, but for the time being, prevention remains the strongest tool.

Take-home message

(1) Prevention is the key to the maintenance of peri-implant health. We suggest keeping the following schedule of periodontal follow-ups: 6 months for healthy individuals; 3–6 months for PIM cases (depending on the level of mucositis); and 3 months for PI cases.

(2) In cases of PI, full elimination still seems impossible; thus, depending on the implant length being compromised, prolonging the life of the implant is extremely questionable, which can result in implant removal.

(3) Surgical interventions for PI present better results than non-surgical activities; therefore, prognosis for the implant (depending on the level of involvement) may be unpredictable.

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