original article

Peri-implantitis: Critical and current overview of etiological factors, clinical/radiographic diagnosis and prognosis. A literature review

Abstract / Introduction: *Peri-implantitis has been more frequently diagnosed; however, the best treatment method has not yet been established. It is paramount to understand its etiology, as well as clinical and radiographic diagnosis to prevent, treat and control peri-implant infections and, as a result, achieve implant success.* Objective: *This literature review aims to address the etiology, diagnosis and prognosis of peri-implantitis in a critical and up-to-date manner. It is based on articles retrieved from PubMed and Science Direct databases.* Results: *Peri-implantitis is an infectious/inflammatory process affecting the peri-implant tissues around dental implants and of multifactorial etiology. Some clinical/radiographic, diagnostic methods are essential to assess the occurrence and progression of peri-implantitis. Despite existing in high numbers, scientific studies are divergent and inconclusive with regard to ideal treatment and prognosis.* Conclusion: *Peri-implantitis is a disease that must be diagnosed at an early stage. Regular monitoring of patients is important for treatment and implant success.* Keywords: *Peri-implantitis. Clinical diagnosis. Dental prosthesis and implants.*

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INTRODUCTION

With the advent of Implantodontics, rehabilitation procedures have been associated with a number of clinical complications. This is due to the fact that the population is having greater access to this type of treatment and a higher number of professionals are using implants to rehabilitate patients' masticatory, speech and esthetic function.[1]

Failures associated with osseointegrated implants are grouped into early and late. Early failures are related to the preparation of the site of implant installation; bacterial contamination; lack of primary mechanical bicortical stability; and early loading. Late failures are associated with excessive loading and/or infection which is also known as peri-implant mucositis and peri-implantitis.[2]

Peri-implantitis is an infectious/inflammatory process that affects hard and soft tissues around osseointegrated implants and results in loss of bone support.[3] Peri-implant mucositis, however, is the inflammation of soft tissues around implants, but without bone loss.[4]

The aim of the present study was to conduct a literature review using Pubmed and Science Direct to retrieve studies about the etiologic factors, diagnosis and prognosis of peri-implantitis.

LITERATURE REVIEW

Etiological factors

Recent literature reveals a high prevalence of peri-implant mucositis affecting 80% of subjects and 50% of implant sites, whereas peri-implantitis affects 28-56% of subjects and 12-43% of implant sites.[5]

The role microorganisms play in the etiopathogenesis of periodontal disease has been well documented since the first study about experimental gingivitis in humans.[6] In this study, gingival tissue presented inflammatory signs (bleeding, redness, swelling and loss of gingival contour) after suppression of oral hygiene habits. The role bacterial biofilms play as a source of damage was also demonstrated for peri-implant tissues with no statistically significant differences between teeth and implants.[7]

The primary etiological factor of peri-implantitis is the presence of bacterial plaque, but other factors associated with the host should also be highlighted. Similarly to the etiological factors of periodontal disease, acquired and congenital risk factors also negatively influence the onset and progression of peri-implant diseases.[8]

Poor oral hygiene, previous history of active periodontal disease, smoking, poorly controlled diabetes, alcohol consumption and genetic factors are also involved in the etiology of peri-implantitis.[8]

Patients with high susceptibility to periodontitis may have increased risk of developing peri-implantitis. There is evidence that periodontal pockets can serve as reservoirs of pathogens, which are similar between periodontal disease and peri-implantitis, thereby leading to contamination of peri-implant tissues.[9]

A systematic review that assessed implants that had been 5-14 years in function revealed an increased incidence of peri-implantitis in patients with periodontal disease, with 2-2.6% in patients without periodontitis, and 16-29% in patients with a history of periodontal disease.[10] In partially edentulous patients with treated periodontal disease, the risk of peri-implant diseases was higher when compared to total edentulous patients.[11]

Some local etiologic factors, such as patient's periodontal biotype, should also be evaluated, since the quality/quantity of keratinized gingiva is essential for peri-implant homeostasis.[12]

When compared to periodontal tissues, the peri-implant mucosa has increased structural fragility in terms of sources of vascularization, composition of connective tissue and number of fibroblasts.[13]

Patient's smoking habit is also negatively correlated to peri-implant damage. Studies demonstrated greater marginal peri-implant bone loss and failure rates in smokers compared to non-smokers.[14,15] Smokers with periodontal disease together also presented low rates of implant survival and higher rates of marginal bone loss around them.[16]

Another clinical factor to be considered is the occlusal loading over implants. Bone turnover is a dynamic process that occurs to improve bone quality and quantity, including bone near the implant surface.[17] These changes are easily noticed after healing, and occur after the application of occlusal loading on the implant.[18] The type of bone tissue response is determined by the direction, frequency and magnitude of the occlusal loading.[19]

Whenever occlusal stability occurs (proper distribution of occlusal loading over the implant), there is an increase in osseointegration without evidence of marginal bone loss.[20] However, occlusal interference and parafunctional activities may lead to biological mechanical complications, such as peri-implantitis and/or loss or fracture of abutment screw or implant.[21]

Clinical and radiographic diagnosis

Proper diagnosis is key to maintenance and restoration of peri-implant health. Clinical diagnosis includes parameters related to probing pocket depth (PPD) (the distance from the edge of peri-implant tissue to the bottom of peri-implant sulcus/pocket) and clinical attachment level (CAL) (the distance from the implant platform or connection area to the bottom of peri-implant sulcus/pocket).[22]

Bleeding on probing should also be investigated, since this parameter represents histological inflammatory tissue changes. The presence of bleeding associated with peri-implant bone loss and increase in PPD are the main indicators of implant failure.[23]

Implant mobility might be assessed, but clinically-detected mobility demonstrates total loss of osseointegration. Some studies used Periotest,[24] a diagnostic method that can detect small changes in the bone-implant interface. However, the alveolar bone has some flexibility, which can lead to bias in the diagnostic assessment.

Occlusal examination should also be performed to detect potential implant failure and overloading. Correct protrusion and laterality should be restored.[12]

Radiographic examination allows clinicians to assess the relationship between implants and prosthetic components. In addition, it is essential for further evaluation of clinical parameters. The ideal would be to use periapical radiographs standardized by the parallelism technique. Standardization allows potential bone changes to be assessed, within certain limitations, overtime.[8,22,23]

It is essential to differentiate a biological process (saucerization) from a pathological one (peri-implantitis).[8,22,23] Saucerization is a biological process of peri-implant cervical bone remodeling starting in the first year after implant placement. It ranges from 0.4 mm to 1.6 mm within the first year and then approximately 0.1 mm/year.[14]

Prognosis

Prognosis for peri-implantitis treatment remains uncertain and relies on a number of factors related to the therapy of choice and the host's response. This is partially due to the fact that no ideal treatment modality with predictable results has yet been established. A number of therapies has been proposed, for instance, mechanical debridement associated with antiseptics in cases of peri-implant mucositis. As for peri-implantitis, some authors also suggest the use of systemic antibiotics,[25,26] as well as resection or regeneration surgery. However, the major therapeutic challenge in the treatment of peri-implantitis is the decontamination of the implant surface. This difficulty is related to the macroscopic (presence of threads) and microscopic implant structures (surface treatment).[26-29] In the presence of mobility and peri-implant radiolucency, however, it is advisable that the implant be removed.[30]

Before making any therapeutic decisions, the clinician must assess the characteristics of the peri-implant lesion (bone defects), availability of material and equipment (cleaning the implant surface, regeneration, membranes and bone grafts), his own clinical experience and the cost-benefit relationship.[31]

DISCUSSION

Peri-implant mucosa and periodontium have some similarities in terms of development and maturation[32] of bacterial biofilms, as proved by an experimental study about gingivitis around implants.[7] Nevertheless, when exposed to chronic bacterial aggression, these tissues tend to behave in different ways. The peri-implant mucosa is less efficient in encapsulating

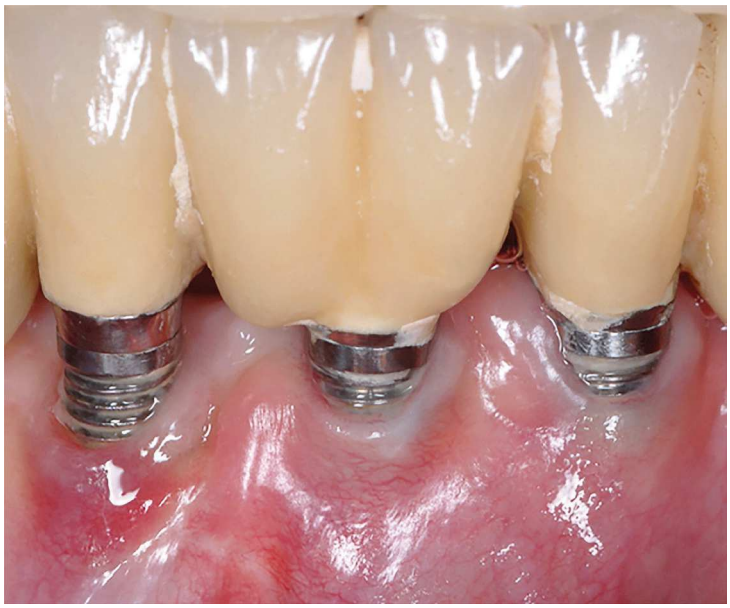

Figure 1. Clinical aspect of peri-implantitis with implant thread exposure and absence of peri-implant mucosa of good quality.

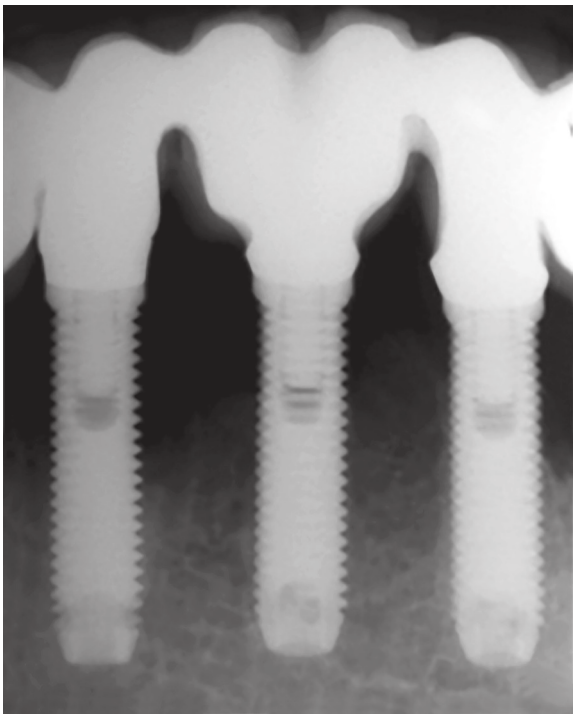

Figure 2. Radiographic examination showing bone loss around implants.

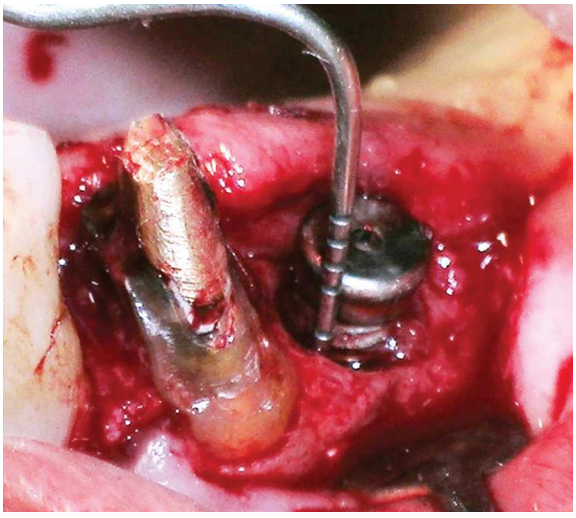

Figure 3. Transsurgical probing of bone defect around implant.

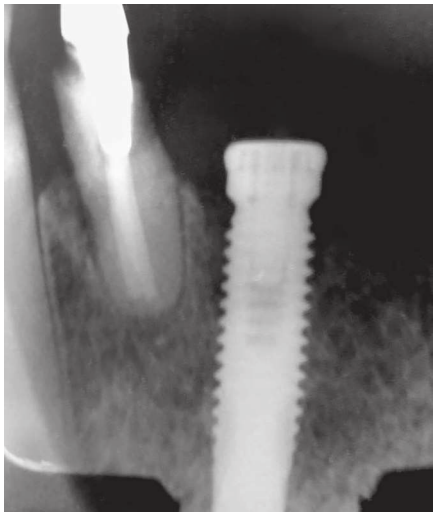

Figure 4. Radiographic examination showing bone loss around implant.

the inflammatory lesion, thus favoring it to evolve to peri-implantitis.[32,33]

The presence and quality of keratinized mucosa around implants is also paramount. Keratinized mucosa is responsible for determining the biological width around teeth and the union between tissues and teeth, which determines a dentogingival junction capable of maintaining marginal periodontium homeostasis.[34] Around implants, sites with keratinized mucosa width of 2 mm, when compared to sites with 4 mm, presented marginal osseous crest resorption so as to allow biological width to reestablish.[13] The quantity and quality of soft tissues around implants is paramount for peri-implant homeostasis, mainly when the vulnerability of peri-implant structures is considered in comparison to the periodontium. For this reason, gingival grafting procedures around implants are essential to enhance the protection of peri-implant structures.[34]

Rehabilitation of a patient with history of periodontitis demands detailed treatment planning for implant success.[25] It is imperative that periodontal disease be treated before any surgical procedure, and that the patient undergoes a maintenance program. These measures can assure longevity of implants.[8] The presence of a long epithelial junction favors reestablishment of periodontal pockets with a pathogenic subgingival microbiota that, in turn, favors the colonization of peri-implant sites. Resection and regeneration surgeries might be needed before implant installation with the objective of obtaining a shallow gingival sulcus.[35]

Patients with a history of periodontitis and residual pockets with pocket depth >5 mm present a significant higher risk to develop peri-implantitis and implant loss at the end of active periodontal therapy.[36] Implant placement in patients with periodontal disease is not contraindicated, but the higher incidence of peri-implantitis may hinder implant longevity. Periodontal infection control, proper oral hygiene and maintenance of periodontal therapy are essential for long-term success.[36]

Another important etiologic factor to be considered is the smoking habit, which produces the same negative effects in periodontal as well as peri-implant diseases. The harmful effects caused by the toxic substances of cigarettes lead to destructive mechanisms that act directly to tissues and affect the host immune-inflammatory responses. The vascular alterations promoted by nicotine, such as peripheral vasoconstriction, threaten one's metabolism and local defenses. In addition, reduced vascularization at the peri-implant site plays a major role in favoring peri-implant disease.[37] Nevertheless, smoking is not an absolute contraindication for implant surgery, since the presence of an isolated risk factor is not enough to cause unfavorable results and peri-implantitis is of multifactorial etiology.[9] Temporary or complete cessation of the smoking habit reverses the effects of smoking on bone healing around implants. It is the clinician's responsibility to warn patients about the benefits of smoking cessation.[38]

Factors related to prosthesis, occlusion, antagonist dentition and bruxism may also be associated with peri-implantitis and, therefore, deserve careful consideration.[39,40] Any signs of occlusal disharmony, premature contact or occlusal interference must be identified and corrected to prevent occlusal implant overload.[23]

Therefore, knowledge of the multifactorial etiology of peri-implantitis and its

early diagnosis is paramount for implant success. The success rate might be assessed taking the following clinical and radiographic characteristics into account: lack of mobility, painful symptoms, PPD > 5 mm, bleeding on probing, chronic radiolucency around implants (after one year in function, bone loss should not exceed 0.2 mm/year). This is due to the saucerization phenomenon, especially in external hexagon implants, which aims at reconstituting the biological width around implants.[41]

Clinical diagnosis by means of probing is a simple, low-cost method used to assess the peri-implant tissue insertion. Nevertheless, some variables such as emergence profile of the prosthesis, juxtaposition of tissues around implants, and roughness of implant might hinder examination. The clinical attachment level might yield the same values of the radiographic dimension of peri-implant bone.[8,23,25]

Perhaps the fact that implants do not present mobility, even with peri-implant bone loss, is a positive factor that favors marginal homeostasis, as it allows greater control against microbial aggression in the peri-implant sulcus. Teeth with mobility seem to have an increase in the production of crevicular fluid, which predisposes them to plaque accumulation and consequent greater damage to periodontal tissues.[34]

Periapical radiographic evaluation carried out by means of the paralleling technique might be used, but without standardization, it impairs comparison between different periods of time. In addition, conventional radiographic examination requires a change of at least 30% in order to allow mineral mass to be detected.[40,41]

An alternative would be the indication of cone beam computed tomography with three-dimensional images of the bone around the implant; however, should that be the case,

clinicians must consider the high costs and radiological exposure of this examination.[25]

The greatest difficulty in defining a prognosis for cases of peri-implantitis lies in the lack of a "gold standard" treatment. Some systematic reviews demonstrated a trend towards better clinical and radiographic results when using surgical approaches (compared to non-surgical), hydroxyapatite jet, systemic antibiotics, implantoplasty and bone grafting.[46] Depending on the degree of peri-implant involvement, heroic attempts are not indicated, being more reasonable to reconsider a new surgical/prosthetic planning.

The relationship between clinicians and patients is key to implant treatment success. Clinicians should assess patients individually, considering local and systemic etiological factors and also warning them about the importance of maintaining periodontal/peri-implant health.

Patients should be aware about the importance of their compliance, mainly in terms of plaque control and return to appointments.

CONCLUSION

Based on this literature review, it is reasonable to conclude that knowledge of local and systemic etiologic factors of peri-implantitis associated with diagnostic methods are paramount for early detection of peri-implant changes. Despite significant failure concerning peri-implant diagnosis and absence of protocols for peri-implantitis treatment, individualized patient assessment, as well as monitoring and control of patient prove essential for the longevity of dental implants. Thus, dental implants should not be considered a definitive treatment approach. The best prognostic for dental implants is attributed to the prevention of peri-implant changes.

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