Influence of Etiologic Factors in Peri-Implantitis: Literature Review and Case Report

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Peri-implantitis is a pathology that has been described in many clinical studies and case reports. However, it is still not clear how the roles of its etiologic agents work. This article is based on a review of the literature and a case report. It aims to offer data related to the factors that cause this pathology, and to analyze how these factors interact, leading to the contamination of the peri-implant tissue.

Key Words: dental implants, peri-implantitis, oral pathology

INTRODUCTION

With the advent of oral implantology in rehabilitation treatments, it became clear that the research conducted on peri-implant tissue pathologies and reactions plays an important role in the evolution of dentistry. With this in mind, implant behavior during osseointegration must still be studied to improve conditions for the successful installation of the prosthesis.

One of the important complications related to dental implant treatment is a pathology called peri-implantitis, a condition caused by peri-implant tissue alterations, including intense inflammation similar to what is found in cases of periodontitis. It is considered a rare occurrence and has been described in previous articles. The conclusion has been that the disease is primarily caused by plaque and biofilm, which can accumulate even on implant surfaces, covering both screws and healing abutments.[1,2]

The biological process that leads to implant loss is still poorly understood. Some patient-related factors such as gender, age, lack of hygiene, and presence of keratinized mucosa have been considered as possible etiologic factors. However, there is controversy in the literature about the importance of some of these events. Thus, the aim of this study is to clarify the importance of the role played by each one of the peri-implantitis etiologic factors in cases of this pathology, carried out by reviewing MEDLINE literature from 1988 to 2010, and illustrating it with a peri-implantitis case report.

LITERATURE REVIEW

It is clearly known that the oral pathology called peri-implantitis is one of the most important risk factors in cases of dental implant rehabilitation. It is an irreversible inflammation of the peri-implant tissue, usually associated with marginal bone loss, and differs from peri-implant mucositis cases, in which there is a reversible inflammation of the same tissue but with no associated bone loss.[3]

Although peri-implantitis has been related to cases with implant loss,[2,4,5] some clinical studies show different results for its frequency. Low rates have recently been found by Montes et al[2] in a clinical study about failing factors, evaluated after implant osseointegration. Nevertheless, in a previous similar study done 7 years earlier, a frequency that ranged between 5% and 10% had been encountered in cases of dental implant rehabilitation.[3]

Jung et al[6] evaluated implant survival rates in cases with implant-supported single crowns in a 5-year review. Interestingly, peri-implantitis and soft-tissue complications were observed by the authors

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in 9.7% of all cases reviewed. However, only 6.3% of the cases showed a marginal bone loss of 2 mm in the period under review.

The early biofilm formation on oral implants was analyzed quantitatively using different methods in a microbiologic study. Polymerase chain reaction results showed low levels for *Haemophilus actinomycetemcomitans* and *Porphyromonas gingivalis*, which are considered important etiologic factors in periodontal desease.[7] Despite this result, other studies found a close relation between the presence of these types of bacteria and peri-implantitis occurrence, supporting the theory that they play an important role in this kind of pathology.[8,9]

Other microbiologic studies evaluated partly edentulous patients and concluded that both the microbiology of the supra- and subgingival plaque on implant abutments and teeth surfaces are similar.[10,11] Endotoxins associated with gram-negative bacteria lead to tissue inflammation and favor the survival of different types of bacteria.[3]

The acquired pellicle, derived mainly from salivary glycoproteins, has been related to higher levels of *Streptococcus mitis, Streptococcus sanguis,* and *Streptococcus oralis* in the biofilm formed on oral implants. Other types of bacteria are also considered peri-implantitis etiologic factors, such as *Actinobacillus actinomycetemcomitans*, found also in edentulous patients with a previous history of periodontal disease,[4] and *Bacteroides forsythus*, which has been described as able to colonize the peri-implant sulcus 1 month after implant loading.[12]

There are some factors that affect peri-implantitis development. One of the most important is the lack of hygiene, which can increase bone loss and inflammation levels.[13–15] Overloading also shows similar effects in cases that already presented high levels of peri-implant tissue inflammation.[16] On the other hand, different levels of keratinized mucosa were evaluated and considered not as influential on the microbiota levels found on implant sites.[17]

The negative effects of tobacco use on implant success are clearly known. Smokers have higher failure rates and complications following grafts and implant-related surgical procedures. It has been proved that smoking also causes significantly greater marginal bone loss after implant placement and increases the incidence of peri-implantitis.[18]

Similar complications have been described in patients with diabetes or other systemic alterations. Xerostomia and poor healing of oral tissues are common factors presented by patients, which can worsen the peri-implant tissue conditions.[3,19]

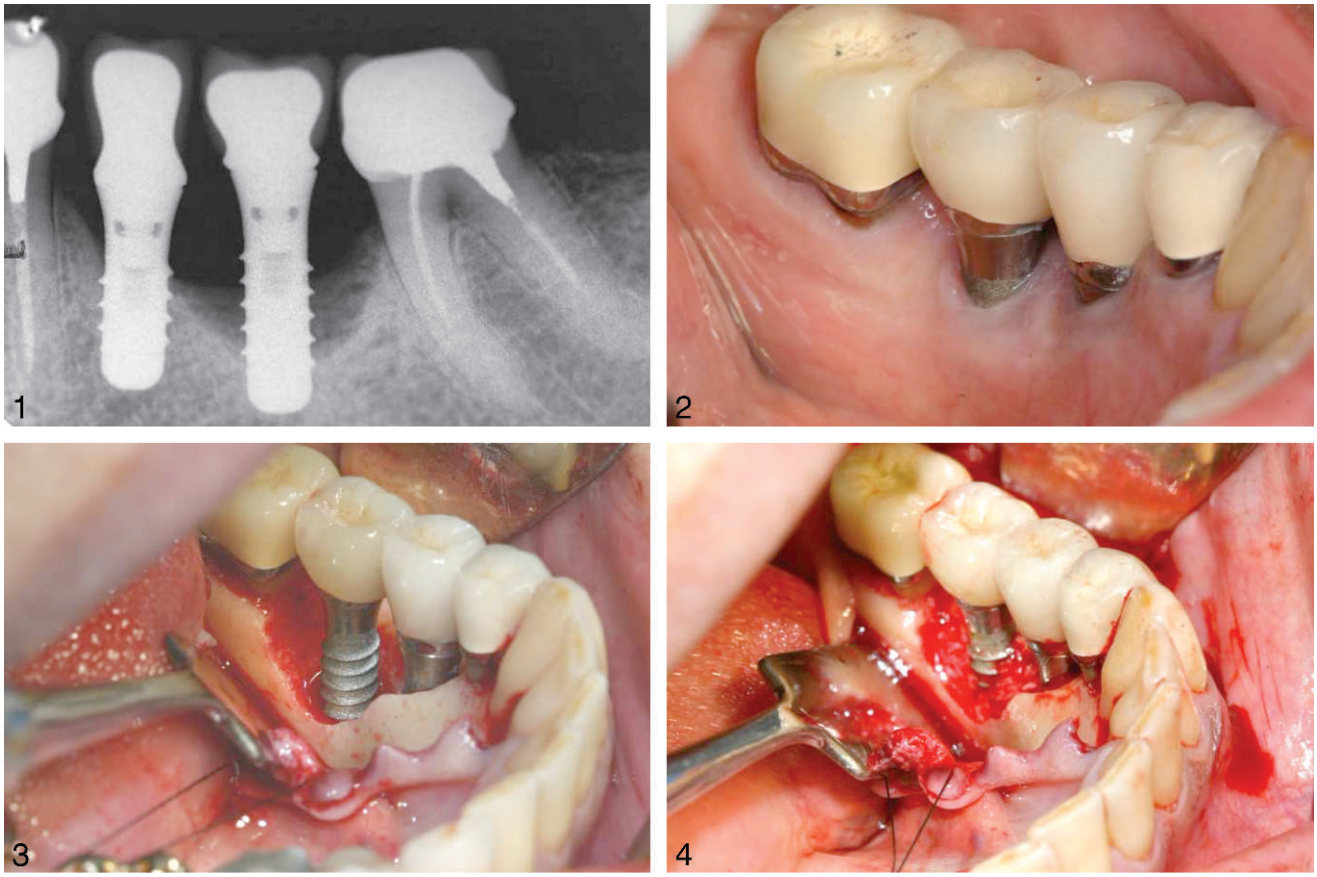
The issue of varying implant surfaces has gained importance with the development of oral implantology. Although surface treatments are intended to improve osseointegration, parameters such as surface roughness and chemical composition of the implant surface have been found to significantly affect plaque formation.[20] Nevertheless, it has been proven that physical modification of titanium implant surfaces, such as coating with TiN or ZrN, might reduce bacterial adherence and hence improve clinical results[20] or even show a different biofilm composition under in vivo conditions.[21]

Some genetic changes have also been correlated with peri-implantitis, and one of the main genes found altered in the literature is IL-1.[22] Some studies describe certain cofactors that suggest a direct linkage between the polymorphism of this gene and periimplantitis.[23,24] Another study suggests that alkaline phosphatase and elastase activity could be promising markers of bone loss around dental implants. In addition, the results confirmed the similarity of the inflammatory response of tissues surrounding implants and natural teeth.[25]

Case Report

A 50-year-old nonsmoking female patient, with no systemic condition, including no metabolic disorders, presented with 2 implants with single-tooth restorations of masticatory function at sites #18 and #19 for more than 10 years. A standard control periapical radiograph showed an extensive peri-implant lesion around an osseointegrated dental implant with a rough titanium plasma spray surface (TPS) that rehabilitated tooth #19 (Figure 1). At the clinical examination, there was a 1-mm lingual recession (Figure 2), with an initial 9-mm probing depth on the buccal side. Initially, antibiotic therapy was applied with amoxicillin 875 mg associated with metronidazole 400 mg, twice a day, for 10 days.

During the antibiotic therapy, chlorhexidine 0.12% intracrevicular local irrigation was performed every 3 days to improve site decontamination. Open flap procedure was performed, including complete defect debridement and implant surface



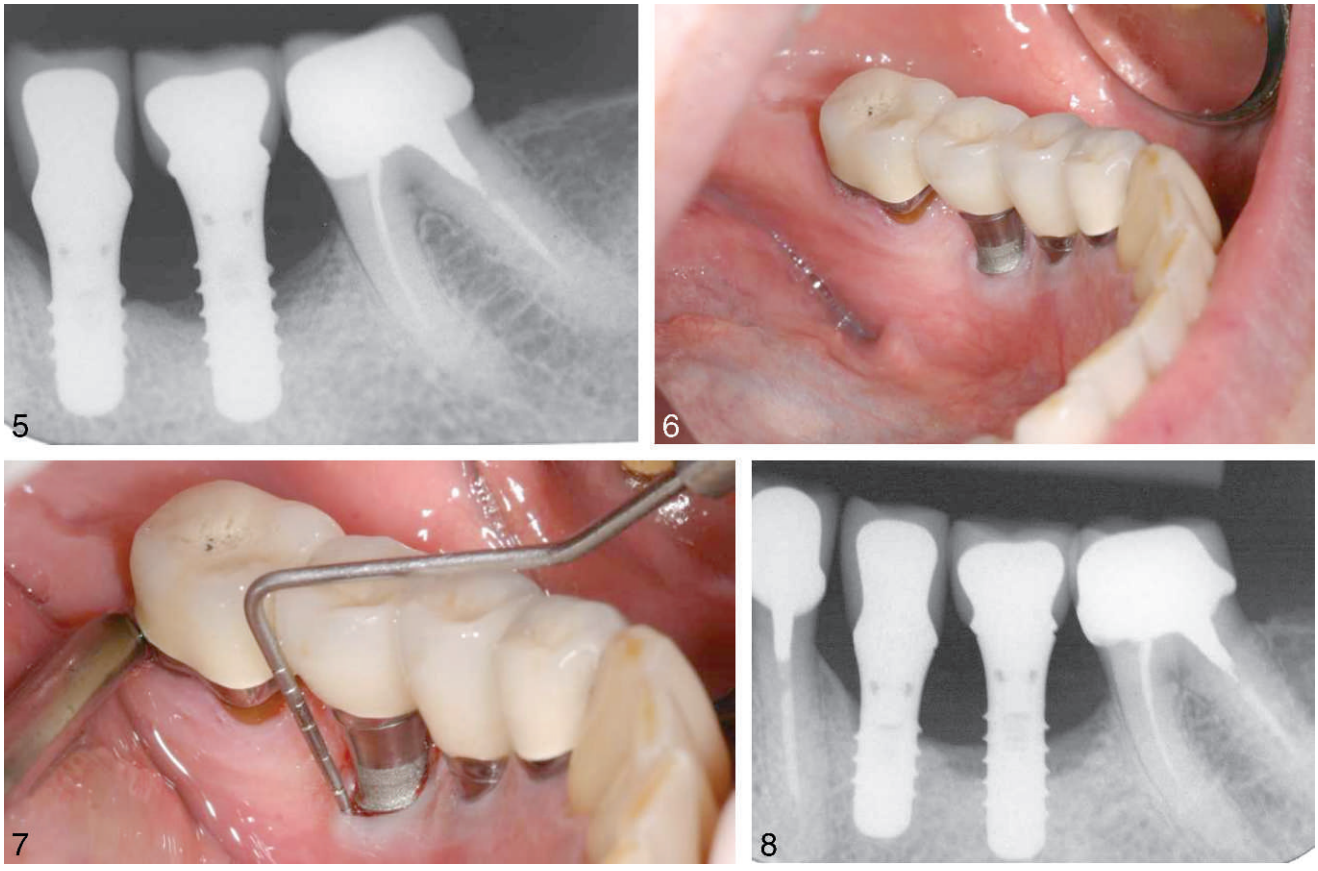
Figures 1–4. Figure 1. Initial periapical radiograph. **Figure 2.** Initial clinical aspect. **Figure 3.** Clinical aspect obtained after defect debridement and implant surface decontamination. **Figure 4.** Bone harvesting and defect-filling procedure.

decontamination with sponges soaked in chlorhexidine 0.12% for 5 minutes (Figure 3).

Mineralized autogenous bone chips were harvested from the same surgical site (Figure 4), and the 4-wall bone defect was completely filled (Figure 5) and covered with a resorbable collagen membrane. The wound was carefully closed with 5.0 nylon monofilament sutures. Healing was uneventful, leading to clinically healthy peri-implant soft tissues 6 weeks later. After 12 months of healing, a 3-mm soft-tissue recession resulted (Figure 6), having a probing depth of 4 mm with mild bleeding (Figure 7). The satisfactory peri-implant tissue condition was confirmed after a 5-year follow-up (Figure 8).

Discussion

The role played by bacteria in peri-implantitis etiology, as well as the influence of tissue conditions around the implant after surgery, are points of agreement between the articles cited by the present study.[1–7] As one of the conclusions, peri-implant probe depth is directly related to tissue inflammation frequency. The same occurs with implant mobility, a factor in itself able to indicate implant loss.[1,2,5] Other forms of bacterial plaque development, such as dental biofilm and deposition on healing screws, caused by poor dental hygiene, might pose a problem for maintaining peri-implant tissue health. Patients should be instructed to keep implant areas clean and healthy and also improve their dental hygiene methods.[7] The influence of dental implant surface roughness on peri-implantitis occurrence, as described in literature, is also supported by the case report described herein. Implants with rough TPS surface have been considered more susceptible to contamination by plaque, as compared with implants having other types of surface, such as sandblasted and acid-etched types.[26] Articles on peri-implantitis have also shown the acceptable bone volume gain, observed in the treatment results of the present case report,



Figures 5–8. Figure 5. Radiographic aspect obtained immediately after defect-filling procedure. **Figure 6.** Final clinical aspect. **Figure 7.** Final probing depth measurement. **Figure 8.** Final radiographic aspect obtained after a 5-year follow-up.

in cases that required debridement and decontamination of implant surface.[27,28]

Adjacent tooth lesions and previous periodontitis occurrence can be considered important causes of peri-implantitis. Thus, medical and dental case history should be analyzed before initiating treatment to avoid oral factors that cause peri-implantitis and also cases of medically compromised patients.[1,22]

Although most of the peri-implantitis cases occur in partially edentulous patients, some cases also occur in total edentulous patients. However, these patients usually present a less aggressive bacterial flora associated with the peri-implant tissue, indicating a lower inflammation risk.[4]

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