ISSN No: 2455-7803

REVIEW ARTICLE

ETIO-PATHOGENESIS OF PERI-IMPLANTITIS- A COMPREHENSIVE REVIEW

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ABSTRACT:
Peri-implantitis represent serious diseases after dental implant treatment, which affect both the surrounding hard and soft tissue. Due to high prevalence rate peri-implantitis can lead to the loss of the implant without multilateral prevention and therapy concepts. Early identification of sign and symptoms associated with peri- implantitis is necessary for better prognosis. Complete knowledge of aetiology associated with peri- implantitis will help in preventing the pathology. Hence; in the present review, we aim to highlight the aetiology and pathogenesis of peri-implantitis.
Key words: Aetiology, Implant, Peri- implantitis.

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This article may be cited as: Gupta R, Luthra RP, Sharma A. Etio-Pathogenesis of Peri-implantitis: A comprehensive review. Int J Res Health Allied Sci 2017;3(3):51-53.

INTRODUCTION

Dental implants have become an indispensable established therapy in dentistry in order to replace missing teeth in different clinical situations. Success rates of 82,9% after 16 years follow-up have been reported. Under care and attention of indications, anatomical and intra-individual limiting factors, insertion of dental implants seems to represent a "safe" treatment option. Nevertheless, in the last decades increasing evidence raised on the presence of peri-implant inflammations representing one of the most frequent complications affecting both the surrounding soft and hard tissues which can lead to the loss of the implant. Therefore, strategies for prevention and treatment of peri-implant disease should be integrated in modern rehabilitation concepts in dentistry.[1]

PATHOGENESIS

Bacterial infections play the most important role in the failure of dental implants. Bacterial flora, which is associated with periodontitis and peri-implantitis, are found to be similar. The microorganisms most commonly related to the failure of an implant are the Gram-negative anaerobes, like *Prevotella intermedia, Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, Bacterioides forsythus, Treponema denticola, Prevotella nigrescens, Peptostreptococcus micros,* and *Fusobacterium nucleatum*. Healthy peri-implant tissue plays an important role as a biological barrier to some of the agents that cause peri-implant disease, and if that is destroyed, bacterial contamination spreads directly to the bone, leading to its rapid destruction. Excessive mechanical stress, poor design of the implant, and the corrosion that can occur when a non-noble metal structure is connected to a titanium implant are important factors in the onset and development of peri-implantitis. Other etiological factors include diabetes mellitus, osteoporosis, smoking, long-term treatment with corticoids, radiation, and chemotherapy.[2]

Inflammation leading to tissue destruction

Inflammation is a complex reaction of the body in response to an infectious agent, antigen challenge, or injury. An accumulation of microbes at the periimplant/mucosal margin is followed by a local inflammatory response. Within 10 to 20 days of plaque accumulation on teeth, clinical signs of inflammation can be seen. Even during early stages of inflammation, considerable tissue damage occurs. As reported in dogs, the collagen content of the inflammatory lesion in the gingival of teeth decreases by approximately PERI-IMPLANTITIS 665 30% after 28 days of undisturbed plaque accumulation. Thus, the cells in the inflammatory lesion cause considerable tissue damage in their effort to combat the invading microorganisms. Accumulation of plaque in the gingival crevice aggravates the inflammatory reaction over time, and consequently, irreversible tissue destruction occurs. Degradation of connective tissue is followed by epithelial migration and bone resorption, which marks the borderline between gingivitis/mucositis and periodontitis/peri-implantitis. The onset of peri-implant disease is caused by an imbalance between the bacterial load and the host defense. The microbiota responsible for the disease and the factors that can sustain and increase its detrimental potential will be discussed later in this chapter.[3-6]

The criteria for a correct diagnosis of peri-implant disease have been clearly defined by Heitz-Mayfield in a review edited in the context of the 6th EFP Consensus. According to this review, probing and radiographic assessment are the primary diagnostic means. Probing should be performed using a force of 0.25 N in order not to damage the peri-implant tissues and aims at assessing the presence of BOP, which indicates the presence of inflammation in the peri-implant mucosa. It is a predictor for the loss of tissue support. PD should be assessed regularly for the detection of BOP and possible suppuration, and to determine any increase in depth over time, which is usually associated with the loss of attachment and supporting bone.[7]

THE MICROBIOTA ASSOCIATED WITH PERI-IMPLANTITIS

The subgingival microbial flora of diseased implants has generally been considered to have quite common characteristics. An early study from Rams et al showed that, while the microbial population surrounding healthy implants had high rates of coccoid gram-positive cells and few spirochetes, there was an inversion of this tendency with increasing PD and gingival inflammation. In most of the

human studies assessing peri-implant microbiota, there is a consistently high incidence of Prevotellaceae (P. intermedia, P. buccae, P. oralis, P. melaninogenica, P. denticola, P. nigrescens), Porphyromonas gingivalis, Fusobacterium nucleatum, Tannerella forsythia, and Treponema denticola in diseased implants, as well as coccoid gram positive cells in healthy implants. These findings have been confirmed by experimental studies on ligature-induced peri-implantitis on animal models, in which the characteristic shift in the microbial flora has been confirmed. The finding of Aggregatibacter actinomycetemcomitans is inconstant with the findings of the other studies; this might be due to the individual composition of the oral microflora of the patients included in the studies. The colonization of the implant's surface by microbial species starts already 30 minutes after implant placement, and the bacteria load stays the same for the first week. Between the first and the twelfth week after surgery, the bacterial load becomes significantly higher for several species, among which are P. gingivalis, T. forsythia, and T. denticola. At 12 months, the bacterial load appears to be significantly higher for some species, in particular T. forsythia and, to a lesser extent, P. gingivalis.[8- 10]

According to the studies cited above, the bacterial composition of the peri-implant biofilm closely resembles that of the neighboring teeth, which implies that the microbial flora on natural teeth serve as the reservoir for the biofilm formation around implants. In the same way, the qualitative composition of the biofilm microflora in peri-implantitis resembles that of periodontitis, which explains why patients with active periodontal disease are at higher risk for peri-implantitis. In support of this theory, a study conducted by Kočar et al on a population of partially edentulous and fully edentulous patients found that the peri-implant and periodontal sulci of partially edentulous patients had no differences in the microflora, sharing the same periodontopathogenic species, but none of these bacteria were found in the peri-implant sulci or the alveolar gingiva of completely edentulous patients. Moreover, a few studies on humans have shown the presence of non-periodontal microbial species, such as Pseudomonas aeruginosa, Candida albicans, Staphylococcus aureus, and Staphylococcus Warneri, around diseased implants. In particular, previous authors found, in two patients out of 33, a complete absence of periodontal microorganisms commonly found in periodontitis.[11, 12]

However, all the above-mentioned studies have the limitation of using culture-dependent or molecular methods to detect bacteria around implants. The culture-dependent methods initially used are time consuming and limited only to the cultivable species. Molecular methods, such as PCR or DNA-DNA hybridization, are faster, but have the disadvantage of a need to pre-select DNA probes for the specific bacterial taxa investigated, thus creating a sort of bias. In fact, most of the knowledge of peri-implant microbiota derives from periodontitis. In recent years, the latest sequencing technologies, such as the 16S rRNA sequencing, have been able to overcome the limitations of both the methods mentioned above and have introduced a new concept of the "microbiome," intended to refer to the full collection of genes of all the microbes in a community. These culture-independent metagenomic methods are potentially able to identify previously undetected and uncultivable bacteria, as well as different strains of known bacteria, allowing the analysis of genetic material harvested directly from the oral microbial environment. The Human Oral Microbiome Database (HOMD) includes 619 taxa in 13 phyla, as follows: Actinobacteria, Bacteroidetes, Chlamydiae, Chloroflexi, Euryarchaeota, Firmicutes, Fusobacteria, Proteobacteria, Spirochaetes, SR1, Synergistetes, Tenericutes, and TM7. Fifty-four percent are officially named, 14% unnamed (but cultivated) and 32% are known only as uncultivated phylotypes. There are only a few studies thus far that have used metagenomic methods of investigation, but the results are interesting and pave the way to a paradigm shift in the understanding of peri-implantitis disease. Previous authors found a unique microbial population in peri-implant sulci (both healthy and diseased) compared to periodontal-associated biofilm, with lower levels of Prevotella, non-mutans Streptococcus, Lactobacillus, Selenomonas, Leptotrichia, and Actinomyces, and higher levels of Peptococcus, Mycoplasma, Eubacterium, Campylobacter, Butyrivibrio, S. mutans, and Treponema. In a subsequent study, they also concluded that local proximity of teeth to the implant is not sufficient to determine colonization, in contrast with the previous theory based on culture-based and molecular methods. Sixty percent of subjects analyzed in the aforementioned study shared less than 50% of all species between their periodontal and peri-implant biofilms, and 85% of individuals shared less than 8% of abundant species between tooth and implant.[13]

DIAGNOSIS

From a clinical standpoint, signs that determine the presence of periimplant mucositis include bleeding on probing and/or suppuration, which are usually associated with the following: Probing depths≤4 mm; swelling and redness of the marginal tissues, which may or may not be manifest; and no pain. However, when similar parameters are present with detectable bone loss following the initial bone remodeling after implant placement, a clinical diagnosis of periimplantitis is made only if the probing depth is ≥5 mm, confirmed by radiologic evidence of bone loss. A baseline standardized radiograph has to be recorded at the time of delivery of the suprastructure. Periodic radiographs will help in assessing the marginal bone changes. Bone loss of <1.5 mm in the first year of functional implant and subsequently <0.2 mm per year is considered acceptable, but additional bone loss in the presence of clinical changes is considered pathologic.[14]

The type of osseous defect that forms around an implant is determined by the type of bone that existed before bone loss. Thin bone housing leads to the complete loss of bone around the implant. Thick bone housing results in crater-like defects. Typical moat-like bone defects are formed around the implants, and are strictly demarcated. As perfect osseointegration is maintained apically to the defect, bone destruction can progress without any notable signs of implant mobility. Mobility therefore indicates complete loss of osseointegration and is a sign of total failure. Bone resorption can also be caused by the deep insertion of an implant or the placement of implants too close to each other; such a situation could be misdiagnosed as periimplantitis.[15]

CONCLUSION

With the increased number of implants being placed, it has become incumbent on the part of the dentist to educate and motivate the patient for a regular follow-up and to insist on adherence to cumulative interceptive supportive therapy (CIST).

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