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REVIEWS

PERI-IMPLANTITIS

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ABSTRACT

Peri-implantitis is a biological complication that can occur after a dental implant is placed. It encompasses inflammatory reactions around the implant, such as peri-implant mucositis and peri-implantitis itself. As dental implants become more widely used, complications associated with them are also becoming more common. Several risk factors can contribute to the development of peri-implantitis. These include placing an implant when there is an existing periodontal infection in the remaining teeth, as well as when there is inadequate width of attached gingiva. Poor oral hygiene is another significant risk factor. Additional factors include the presence of an endodontic infection near where the implant is placed, improper implant positioning in terms of prosthetic and/or biological alignment, the implant design itself, incorrect abutment choice, and unremoved cement in cement-retained prosthetic devices. Systemic and genetic factors can also play a role in modifying the risk of peri-implantitis. Peri-implant inflammatory diseases are diagnosed through both clinical and paraclinical testing methods. This involves assessing the risks associated with the implant, such as whether plaque is present, whether there is bleeding on probing, edema, erythema, increased probing depths, exudation, bone loss around the implant, and implant mobility. The treatment for peri-implant inflammation involves CIST therapy.

Keywords: *peri-implantitis, CIST therapy, risk factors, complications in implantology, biological complications*

INTRODUCTION

Minimizing the risk of biological complications around implants begins at the start of implant treatment (1). Proper planning to position the implant in a way that is both prosthetically and biologically optimal helps prevent peri-implantitis (1). It's crucial to have a hygiene phase to address periodontitis and eliminate harmful microorganisms (2). Encouraging patients to maintain excellent personal oral hygiene is vital (3). Managing chronic diseases, using replacement therapies, and quitting smoking all enhance the chances of successful implant outcomes (4). Using screw-retained prosthetic designs and choosing hybrid or conical implant platforms tend to produce better results (4). Implants with an SLA/SLA-active surface, combined with placing implants in adequate bone volume or conducting appropriate bone regeneration procedures, can also reduce the likelihood of peri-implantitis development (4).

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AIM

The goal of this study is to mention the risk factors that contribute to peri-implantitis, enabling dentists to prevent potential biological complications in cases with placed implant/s.

MATERIALS AND METHODS

This article is based on information, which was conducted across various scientific databases like PubMed, Google Scholar, Scopus, and ScienceDirect

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to gather the necessary information. The keywords used for this search included: peri-implantitis, peri-mucositis, dental implant complications, and risk factors.

RESULTS

The classification system for peri-implant diseases established during the 2017 World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions defines peri-implant health as the absence of inflammation in peri-implant soft tissues, such as erythema, edema, or excessive bleeding upon probing, alongside the absence of further bone resorption after successful osseointegration (4). Implantology complications are categorized into intraoperative and postoperative types, with postoperative complications further divided into early and late phases (5). Early and intraoperative complications are collectively referred to as perioperative (5). Late postoperative complications can be classified as biological, technical, or aesthetic in nature, when the biological complications primarily include peri-implant inflammatory conditions, such as peri-implant mucositis and peri-implantitis (5). Peri-implant mucositis is a reversible inflammatory response in the peri-implant soft tissues caused by plaque accumulation, characterized by clinical signs such as bleeding on probing, erythema and edema, in contrast to peri-implantitis, which is a more severe, plaque-induced condition involving inflammation and progressive bone loss around the implant (6). Notably, peri-implant mucositis is often a precursor to peri-implantitis (7).

The concept of osseointegration was initially introduced by Brånemark as a direct connection between vital bone and the implant without the formation of fibrous tissue, observable under an electron microscope. However, today, osseointegration is defined clinically as a rigid, asymptomatic fixation of the implant, maintained under functional loading conditions (8,9).

Peri-implantitis can be developed only in implants which where primary osseointegrated, and it results as loss of peri-implant bone tissue (10).

Successful implant treatment and the prevention of peri-implantitis depend on a combination of factors, including appropriate planning, implant design and material properties, bone quality, surgical technique, and prosthetic considerations (11). Additionally, ensuring proper healing and adhering to loading protocols are critical, as well as, particularly untreated periodontitis, which is significant risk factor for peri-implantitis due to the similarities in the microbial profile of periodontal and peri-implant infections (11–13). However, peri-implant infections generally exhibit a lower microbial load, likely due to surgical interventions and direct exposure of bone tissue, the weaker tissue barrier surrounding implants compared to natural teeth also increases susceptibility to infection (14,15).

Microorganisms commonly implicated in peri-implant infections include: *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tannerella forsythia*, *Aggregatibacter actinomycetemcomitans*, and others(16). The initial bacterial colonization begins with the formation of a salivary pellicle on the implant surface, which differs biochemically from the pellicle on natural teeth. Despite these differences, microbiological studies reveal limited distinctions between the microbial communities of implants and natural teeth; for example, in patients with periodontitis, *P. gingivalis* and *F. nucleatum* are more present than in patients with peri-implantitis. However, certain Gram-positive bacteria associated with peri-implantitis demonstrate higher resistance to beta-lactam antibiotics (17–21).

Inflammatory responses around implants differ significantly from those in natural teeth, with peri-implant tissues showing a greater predominance of neutrophils, macrophages, and a larger inflammatory zone (22–25). An interesting fact is that a *Porphyromonas gingivalis* infection causes greater bone loss around implants than around natural teeth (26). The inflammatory cascade is triggered by bacterial endotoxins, leading to peri-implant mucositis and potentially progressing to peri-implantitis (27). Biomarkers like sclerostin, RANK, RANKL, and OPG are actively involved in the transition from mucositis to peri-implantitis, with OPG serving as a protective inhibitor of osteoclast maturation (28–30). Various bacterial virulence factors, including lipopolysaccharides and enzymes like cathepsin K, stimulate osteoclastogenesis, further exacerbating bone loss (31). Key proinflammatory cytokines, such as interleukins (IL-1, IL-6, IL-17), tumor necrosis factor-alpha

(TNF-α), and colony-stimulating factor-1, play pivotal roles in bone resorption and osteolysis and the further progression from peri-mucositis to peri-implantitis (32–34). Loss of implant/s is associated with bacteria commonly found in patents with periodontitis and/or periodontal abscess (35).

Surface modifications, such as SLA coatings, and placing and implant next to a tooth with good endodontic treatment (without endodontic pathology), when needed, reduce the risk of peri-implantitis (36,37).

The macroscopic topography is also important: in implants with moderately rough surface, compared to implant with rough surface, less bone resorption of the supporting bone is observed (38). Mechanical factors, such as the connection of the prosthetic superstructure to the implant, emergence profile (>30 degrees), insufficient keratinized mucosa (<2 mm), and not well performed guided bone regeneration can also contribute to peri-implant disease development (39–43). Smoking, uncontrolled diabetes, certain medications (methotrexate, proton pump inhibitors, etc.), genetic predispositions, and poor oral hygiene significantly increase the risk of peri-implant disease (44–48). For instance, smoking accelerates marginal bone loss, while systemic conditions like diabetes impair healing and increase infection susceptibility (44,45). Additionally, avoiding excessive loading during the healing phase is essential for maintaining osseointegration (49). Peri-implantitis is diagnosed based on clinical and radiographic findings, including bleeding on probing, the presence of plaque, inflammation (erythema, edema, pain), increased probing depth, suppuration, and radiographic evidence of bone defects around the implant (50). In advanced cases, implant mobility may be observed (50).

Cumulative interceptive supportive therapy (CIST) provides a structured approach for managing peri-implant diseases based on the severity of clinical signs (51). Preventive strategies include careful case selection, rigorous oral hygiene maintenance, and the use of prosthetic designs (screw-retained prosthetic constructions) that minimize plaque retention (41,43).

By addressing these multifactorial risks and employing evidence-based management strategies, clinicians can significantly enhance the long-term success of implant therapy (50,51).

DISCUSSION

Peri-implant health is assumed when there are no signs of inflammation. The earliest indication of inflammation around peri-implant tissues is bleeding during probing (52). Peri-implant mucositis is an inflammatory condition that can be reversed (53). If the patient neglects personal oral hygiene and misses preventive check-ups, the inflammation can progress to peri-implantitis (54). In its advanced stage, peri-implantitis can result in the loss of the implant (54). Removing periodontal pathogens helps prevent biological complications around implants and also aids in maintaining natural teeth (55).

CONCLUSION

Adherence to the rules mentioned so far will minimize the risk of biological complication when placing dental implants. It is extremely important, in addition to the dentist's precise work and the correct personal oral hygiene of the patient.

The plaque retained for a long time on the prosthetic construction leads to the first manifestations of peri-implant inflammation, affecting the gingiva around the implant, with subsequent involvement of the underlying structures, leading, in the final stage, to peri-implant bone resorption.

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