PERI-IMPLANTITIS: A REVIEW

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ABSTRACT

Peri-implantitis is a site-specific infectious disease that causes an inflammatory process in soft tissues and bone loss around an osseointegrated implant in function. The etiology of the implant infection is conditioned by the status of the tissue surrounding the implant, implant design, plaque, biofilm and excessive mechanical load. The microorganisms most commonly associated with implant failure are Gram-negative anaerobes, unless the origin is the result of simple mechanical overload. Diagnosis is based on changes of colour in the gingiva, bleeding and probing depth of peri-implant pockets, suppuration, X-ray, and gradual loss of bone height around the tooth. Treatment will differ depending upon whether it is a case of peri-implant mucositis or peri-implantitis. The management of implant infection should be focused on the control of infection, the detoxification of the implant surface, and regeneration of the alveolar bone. This review article deals with the etiopathogenesis, classification, risk factors, diagnosis, clinical features and various treatment options of peri-implantitis and the management of peri-implantitis.

KEYWORDS - Peri-implantitis, Implant failure, Bone loss, Soft tissue

INTRODUCTION

Peri-implantitis is defined as an inflammatory process affecting the tissues around an osseointegrated implant in function, resulting in loss of supporting bone (1st European Workshop on Periodontology).[1] Periimplant mucositis is defined as "reversible inflammatory changes of the peri-implant soft tissues without any bone loss. The term "biofilm" describes the relatively indefinable microbial community associated with a tooth surface or any other hard, non shedding material.

Dental implants, like natural teeth, offer a non shedding surface for the accumulation and growth of biofilm, which provides biological insurance to a microbial population. Biofilm is also one of the causative agents of peri-implantitis. In contrast to mucositis, peri-implantitis is a progressive and irreversible disease of implant-surrounding hard and soft tissues and is accompanied with bone resorption, decreased osseointegration, increased pocket formation and purulence.

CLASSIFICATION

Schwarz et al.[2] classified peri implant defect depending on the configuration of the bony defect as:

I.] Class I defect – Intraosseous

II.] Class II defect – Supra-alveolar in the crestal implant insertion area.

Spiekerman et al.[3] characterized peri-implant defect into the type of bone resorption pattern into 5 category.

Class I – Horizontal

Class II – Hey-shaped

Class III a – Funnel shaped

Class III b – Gap-like

Class IV – Horizontal-circular form

Retrograde implantitis[4]

A clinically symptomatic periapical lesion that develops within the first few after implant insertion while the coronal portion of the implant sustains a normal bone to the implant interface.

Class I – Mild, Extends < 25% of the implant length from implant apex.

Class II – Moderate, Extends 25–50% of the implant length from implant apex.

Class III – Severe, >50% of the implant length from implant apex.

ETIOLOGY

Plaque

Dental plaque has been recognized universally as the main etiological agent of both periodontal and peri-implant diseases. Various authors have further identified other secondary and additive factors in the etiology of peri-implantitis. Within the dense bacterial population, efficient horizontal transfer of resistance and virulence genes takes place. A cause-and-effect relationship between biofilm formation on teeth and gingivitis as well as on implants and peri-implant mucositis can be demonstrated in humans.[15] Peri-implantitis is a poly-microbial anaerobic infection. However, in contrast to periodontitis, peri-implantitis lesions harbor bacteria that are not part of the typical periodontopathic microbiota.[16]

Microflora

Some studies have reported the microflora associated with peri-implant disease to be different from those of periodontal disease, which is mainly because of sporadic high numbers of Peptostreptococci and staphylococci (*Peptostreptococcus micros*, *Staphylococcus aureus*, *Staphylococcus epidermidis*) isolated from diseased sites of peri implant-tissue.[7]

On clinically stable implants, *Staphylococcus-sanguis* and *Staphylococcus mitis* are the most predominant organisms, while motile rods, spirochetes, fusiforms, and filaments are infrequently found; *Aggregatibacter actino-mycetemcomitans*, and *Porphyromonas gingivalis* are seldom detected; and *Prevotella intermedia* and *Prevotella nigrescens* are more common.[8]

The periimplant flora in edentulous patients is comparable with the flora colonizing the oral soft tissues of denture-wearing edentulous patients without implants and the subgingival flora of periodontally healthy dentate patients.[9]

In partially edentulous patients, the total number of peri-implant microorganisms and the proportion of motile rods, spirochetes, and cocci are greater than in edentulous patients, the flora seeming to depend on the periodontal flora of the remaining dentition.

RISK FACTORS

The following factors or circumstances have been reported as risk factors for the development of peri-implantitis.[6]

• Smoking with additional significantly higher risk of complications in the presence of a positive combined IL-1 genotype polymorphism

• History of periodontitis

• Lack of compliance and limited oral hygiene (including missing checkups)

• Systemic diseases

• Iatrogenic causes

• Soft tissue defects or poor-quality soft tissue at the area of implantation (e.g. lack of keratinized gingiva).

• History of one or more failures of implants

DIAGNOSIS OF DISEASES

From a clinical standpoint, signs that determine the presence of peri-implant 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 remodelling after implant placement, a clinical diagnosis of peri-implantitis is made only if the probing depth is ≥5 mm, confirmed by radiologic evidence of bone loss.

MANAGEMENT

Therapy of mucositis

One of the main aims of peri-implant therapy is to detoxify the contaminated implant surface. In the presence of peri-implant mucositis, non-surgical methods are appropriate and sufficient for detoxification. These include mechanical implant cleaning with titanium or plastic-curettes, ultrasonics or air polishing. Moreover, photodynamic therapy as well as local antiseptic medication (chlorhexidinglukonate, hydrogen peroxide, sodium percarbonate, povidone-iodine) may support the antimicrobial therapy.

Therapy of peri-implantitis

For the treatment of peri-implantitis, both conservative (non-surgical) as well as surgical therapies can be applied. Thereby, the surgical treatments can be done using resective or regenerative approaches.[10]

Conservative therapy

In addition to medication and manual treatment (e.g. with curettes, ultrasonic and air polishing systems) innovative techniques such as laser-supported and photodynamic therapy methods are recently described as conservative therapy options.

Drug therapy

Local or systemic antibiotics are an additional therapy option. In combination with other conservative or surgical treatments it results in more efficient reductions of clinical peri-implantitis symptoms.[11] Just administration of antibiotics is not a complete treatment option.

Photodynamic Therapy

Photodynamic therapy generates reactive oxygen species by multiplicity with help of a high-energy single-frequency light (e.g. diode lasers) in combination with photosensitizers (e.g. toluidine blue). In a wave length range of 580 to 1400 nm and toluidine blue-concentrations between 10 and 50 ug/ml, photodynamic therapy generates bactericide effects against aerobic and anaerobic bacteria.[5] Reducing the bacterial load helps in treating peri-implantitis and perimucocitis.

Surgical Therapy

The surgical therapy combines the concepts of the already mentioned non-surgical therapy with those of resective and/or regenerative procedures. The indication for the appropriate treatment strategy has been demonstrated in patient studies leading to the development of the "cumulative interceptive supportive therapy (CIST)" concept.[12]

Resective therapy

In analogy to periodontitis, resective surgery has been shown to be effective in reduction of bleeding on probing, probing depths and clinical signs of inflammation. The basic principles include the elimination the peri-implant osseous defect using

ostectomy and osteoplasty as well as bacterial decontamination. Additionally, smoothening and polishing of the supracrestal implant surface (implantoplasty) may be applied.

Regnerative therapy

Resective surgical therapy may result in re-osseointegration in only minor superficial defects. From functional, esthetic and long-time-survival point of views, full regeneration and re-osseointegration is aspired. In animal models it was possible to regenerate experimentally induced defects using various graft materials and/or resorbable membranes following the principles of guided bone regeneration (GBR).

For the purpose of bone regeneration, various approaches have been described with various success rates. There is a tendency that xenograft materials in combination with a resorbable membranes might have advantages in terms of re-osseointegration. Nevertheless, because of the lack of prospective randomized clinical studies there is no evident data concerning the long-time stability of such "defect fillings."

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). The main reasons for needing to maintain a strict protocol are: The increase in plaque retention zones around implants when compared to the tooth because of the presence of rough surfaces and suprastructures; difficulty in maintenance by the patient; and decreased resistance of peri-implant tissue against infection. Hence, the early detection of peri-implant diseases becomes imperative for the prevention and treatment of the same.

The primary causative factor for peri-implant disease is dental plaque, but many other contributing factors amplify the effect of plaque that must be identified early and kept under control.

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