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PERI-IMPLANTITIS AND ITS MANAGEMENT: A REVIEW

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Abstract- peri-implantitis is a destructive inflammatory process affecting the soft and hard tissues surrounding dental implants. The soft tissues become inflamed whereas the alveolar bone (hard tissue), which surrounds the implant for the purposes of retention, is lost over time. The bone loss involved in peri-implantitis differentiates this condition from peri-mucositis, a reversible inflammatory reaction involving only the soft tissues around the implant. Present article discussed periimplantitis and its management.

Key word- Periimplantitis, Mucositis, Bleeding on probing, Implant placement.

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. Periimplant disease is a common finding among patients rehabilitated with dental implants. Two types of periimplant disease have been defined in consensus report: one is mucositis, identified as inflammation of the periimplant mucosa without periimplant bone loss; The other is periimplantitis, characterized by periimplant crestal bone loss in conjunction with bleeding on probing (BOP) and/or suppuration with or without deepening of the periodontal pockets.

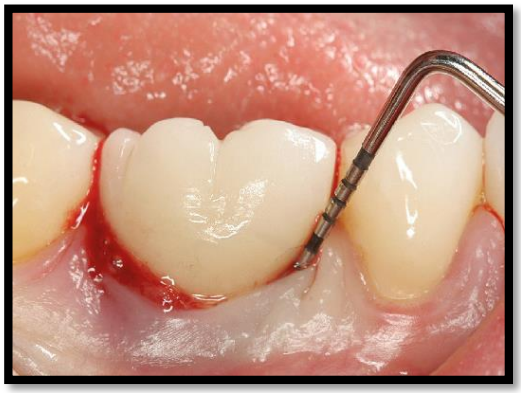

Fig 1 – Mucositis

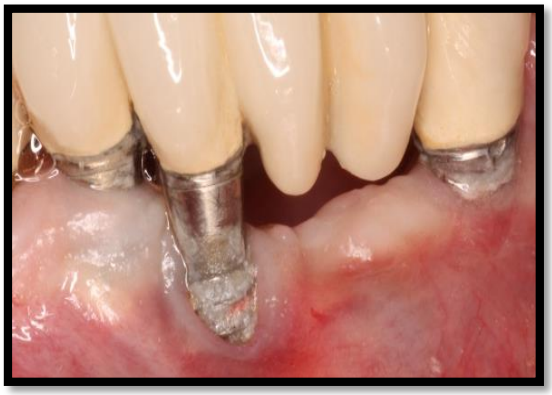

Fig 2- Periimplantitis

Peri-implant mucositis: A disease in which the presence of inflammation is confined to the mucosa surrounding a dental implant with no signs of loss of supporting bone

•**Peri-implantitis**: An inflammatory process around a dental implant which includes both soft tissue inflammation and loss of supporting bone.

DISCUSSION

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

•Class I defect – Intraosseous

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

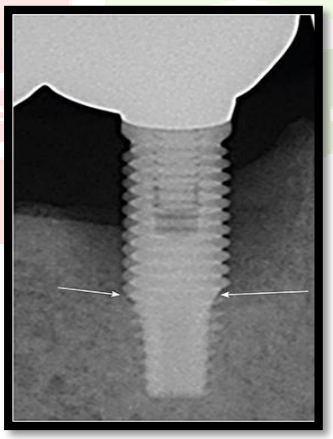

Fig 3 – Class 1

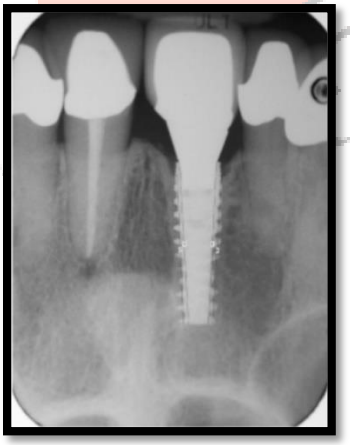

Fig 4- Class 2 defect

Spiekermann characterized peri-implant defect into the type of bone resorption pattern into 5 category.

1. Class I – Horizontal,
2. Class II – Hey-shaped
3. Class III a – Funnel shaped
4. Class III b – Gap-like
5. Class IV –circular form

Another system of classification exists amount of bone loss with shaped of defect associated

1. Class 1: Slight horizontal bone loss with minimal peri-implant defects
2. Class 2: Moderate horizontal bone loss with isolated vertical defects
3. Class 3: Moderate to advanced horizontal bone loss with broad, circular bony defects.
4. Class 4: Advanced horizontal bone loss with broad, circumferential vertical defects, as well as loss of the oral and/or vestibular bony wall

A new classification of bone defects adjacent to dental implants highlighting the defect anatomy in the progression of the regenerative process.

(1) **CLOSED DEFECTS** – It is characterized by the maintenance of intact surrounding bone walls.

(2) **OPEN DEFECTS** – it is the one which lack one or more bone walls.

Table 1- Classification of mucositis proposed by Ata-Ali

Staging	Definition
Stage A	Probing Depth 4 mm and Bleeding on probing and/or suppuration, with no signs of loss of supporting bone following initial bone remodeling during healing
Stage B	Probing Depth >4 mm and Bleeding on probing and/or suppuration, with no signs of loss of supporting bone following initial bone remodeling during healing

Table 2-Classification of peri-implantitis proposed by Ata-Ali

Staging	Definition
Stage I	Bleeding on probing and/or suppuration and bone loss≤3 mm beyond biological bone remodeling
Stage II	Bleeding on probing and/or suppuration and bone loss >3 mm and <5 mm beyond biological bone remodeling
Stage III	Bleeding on probing and/or suppuration and bone loss ≥5 mm beyond biological bone remodeling
Stage IV	Bleeding on probing and/or suppuration and bone loss ≥50% of the implant length beyond biological bone remodeling

Table 3-Classification of peri-implantitis proposed by Froum and Rosen

Staging	Definition
Early	PD ≥ 4 mm (bleeding and/or suppuration on probing[a]) Bone loss <25% of the implant length[b]
Moderate	PD ≥ 6 mm (bleeding and/or suppuration on probing[a]) Bone loss 25% to 50% of the implant length[b]
Advanced	PD ≥ 8 mm (bleeding and/or suppuration on probing[a]) Bone loss >50% of the implant length[b]

Zhang L etal demostrated classification of peri-implant bone defects (PIBDs) on the basis of their Panoramic radiographic shapes in patients with lower implant-supported overdentures.

1. Saucer-shaped defects
2. Wedge-shaped defects
3. Flat defects
4. Undercut defects
5. Slit-like defects
6. pathogenesis

Mucositis describes a bacteria-induced, reversible inflammatory process of the peri-implant soft tissue with reddening, swelling and bleeding on periodontal probing. Bleeding on probing (BOP) might be an indicator for peri-implant disease, but sufficient evidence according to the predictive value of BOP is still lacking. 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. On a microscopic and molecular level, striking differences between peri-implant tissue and intact periodontium can be determined. Due to the reduced vascularization and parallel orientation of the collagen fibres, peri-implant tissues are more susceptible for inflammatory disease than periodontal tissues. This phenomenon can be verified immunohistochemically through increased formation of inflammatory infiltrate, nitric oxide 1/3, VEGF, lymphocytes,

leukocytes and Ki- 67.Besides, in analogy to periodontitis the level of matrix-metalloproteinases (MMP), such as MMP-8, is increased up to 971% in peri-implant lesions.

ETIOLOGY AND EPIDEMIOLOGY

There are several reports on the prevalence of mucositis and peri-implantitis that differ between 5% and 63.4%. This enormous range is mainly based on varying study designs and population sizes with different risk profiles and statistic profiles.Zitzmann et al. quantified the incidence of the development of peri-implantitis in patients with a history of periodontitis almost six times higher than in patients with no history of periodontal inflammation.After 10 years, 10% to 50% of the dental implants showed signs of peri-implantitis. Based on the Consensus Report of the Sixth European Workshop in Periodontology, Lindhe & Meyle reported an incidence of mucositis of up to 80% and of peri-implantitis between 28% and 56%

However, the prevalence of peri-implant diseases, evaluated recently by Mombelli et al., revealed peri-implantitis in 20% of all implanted patients and in 10% of all inserted implants. Frequently, a spectrum of pathogenic germs can be detected such as Prevotella intermedia, Prevotella nigrescens, Streptococcus constellatus, Aggregatibacter actinomycetem-comitans, Porphyromonas gingivalis, Treponema denticola and Tannerella forsythia . Which is responsible for periimplantitis. 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. In particular, Staphylococcus aureus appears to play a predominant role for the development of a peri-implantitis. This bacterium shows an high affinity to titanium and has according to the results of Salvi et al. a high positive (80%) and negative (90%) predictive value

RISK FACTORS

The following factors or circumstances have been reported as risk factors for the development of peri-implantitis. Smoking with additional significantly higher risk of complications in the presence of an positive combined IL-1 genotype polymorphism.

HISTORY OF PERIODONTITIS

Lack of compliance and limited oral hygiene (including missing checkups). Systemic diseases (e.g. maladjusted diabetes mellitus, cardiovascular disease, immunosuppression). Iatrogenic causes (e.g. “cementitis”). 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 The presence of periodontitis or cigarette smoking increased the risk for peri-implantitis up to 4.7-fold as reported by Wallowy et al. study by Vervaeke et al. maxillary implants were at a sig- nificantly higher risk for peri-implant bone loss compared to mandibular implants. Bone augmented areas could not be determined as risk factors for implant failure or increased peri-implant disease. The so called “cementitis” may be regarded as the most

import- ant identifiable iatrogenic risk factor since its first description by Wilson et al. in 2009. Korsch et al. found that the removal of cement remnants led to a decrease of the inflammatory response by almost 60%

Systemic Risk Factors for the Development of Periimplant Diseases

1.**scleroderma,**
2.**Sjögren syndrome,**
3.**neuropsychiatric disorders/Parkinson disease,**
4.**lichen ruber planus/oral lichen planus,**
5.**human immunodeficiency viral infection,**
6.**ectodermal dysplasia,**
7.**long-term immunosuppression after organ transplantation,**
8.**cardiovascular disease,**
9.**Crohn disease,**

Medications

Patients with dental implants are often also being treated for chronic diseases such as asthma, depression, cancer, rheumatoid arthritis, chronic inflammatory diseases, and autoimmune diseases. Although some of these diseases may play a role in increasing the risk of implant failure, many of the medications used to treat these conditions have myriad effects on bone metabolism. However, one retrospective study demonstrated reduced bone-to-implant contact, bone area, and bone density around the implants in subjects who used NSAIDs perioperatively, whereas another demonstrated bone-sparing activity. Glucocorticoids, another line of treatment for chronic inflammatory conditions, also affect bone physiology.

Radiotherapy

Implants that are placed within 12 months of radiotherapy also seem to have a lower survival rate. The underlying pathology is a reduction in the vascularity, cellularity, and reparative capacity of bone

Prosthetic Risk factors

Occlusal overload- One of the major causes for the loss of implants is overload, which causes peri-implantitis. Occlusal overload can cause bone resorption around the osteointegrated implants. Occlusal trauma with peri-implantitis may accelerate bone destruction.

Residual cement-

The effect of flooding cement on peri-implantitis formation is similar to that of dental calculus in periodontal disease. The rough surface of the cement makes it difficult to remove microorganisms and this causes peri-implant mucositis initially and peri-implantitis resulting in bone loss later Noncleansable prosthesis- will causes inflamed

periimplant mucosa and formation is similar to that of dental calculus in periodontal disease. Excessive Deep pocket at implants with submucosa crown margins will causes inflamed periimplant mucosa

Implant design-

Design of an implant affects the tissues around the implant. Cause of crestal bone loss after implant surgery is bacterial accumulation that occurs in the gap between implant and abutment. Therefore; factors such as implant abutment, platform switching concept, and surface roughness can determine amount of bone loss.

Implant placement

In the long run, thickness of bone in buccal region of implants should be at least 2 mm in anterior region and at least 1 mm in posterior region in order to reduce soft and hard tissue loss. The fact that implants are placed in an excessive buccal position and loss of tissue enough to require application of pink porcelain is among other things that may affect peri-implant diseases.

Implant surafces-

The mechanical nature of the bone-implant interface is influenced by the nature of the implant surface. Different surface characteristics and coatings can greatly affect the longevity and function of a restoration. There are concerns that dissolved titanium could increase the risk of peri-implantitis. Genetic factor A study by some researchers found that the areas of marginal bone loss around the implant were very seriously increased in the concentration of blood microspheres, but showed that the expression of the vascular endothelial growth factor was low. For this reason, VEGF may play a protective role in marginal bone loss, that is, periimplantitis . Poor oral hygiene Individuals with poor oral hygiene are exposed to periodontal diseases are also a risk factor for periimplantitis. The most important risk factor for Peri-implantitis is poor plaque control. This may reflect a patient's inadequacy or reluctance to maintain optimal oral hygiene. Other obstacles may include prosthetic design, adjacent restoration contours, margins and broken restorative components Smoking The incidence of peri-implantitis in smokers is between 3.6 and 4.6 percent. A meta-analysis of 13 trials found that cigarette smoking increased the bone loss rate around implants by 0.164 mm/year. Baig and Rajan reported that in smokers significantly more marginal bone loss after placement and higher Peri-Implantitis percentages Alcohol consumption contents found in alcoholic beverages such as a mixture of toxic alcohol, nitrosamines, and ethanol can also cause osteoclasis, as well as inhibit bone stimulation. Studies conducted by some researchers have reported that drinking more than 10 grams of alcohol a day causes more peri-implantitis than cigarette smoking Parafunctional Habits (Bruxism – Malocclusion)

In 9 studies, a total of 761 patients were evaluated biological complications for 2511 implants. In 3 studies, implant failure was assessed according to marginal bone loss. As a result of the studies, there was a clear evidence in 3 studies to evaluate bruxism as a risk factor for implant failure; in the remaining 6, there was no relationship between bruxism and implant loss.

THERAPY

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 per-carbonate, povidone-iodine) may support the antimicrobial therapy.

Therapy of peri-implantitis

Treatment of peri-implantitis, both conservative (non- surgival) as well as surgical therapies can be applied. Thereby, the surgical treatments can be done using resective or regenerative approaches 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 Manual treatment Basic manual treatment can be provided by teflon-, carbon-, plastic- and titanium curettes due to the fact that therapy with conventional curettes is able to modify the implant surface and can roughen the surface, it has been recommended that the material of the tip should be softer than titanium

Drug therapy

Antiseptic rinses in relation to different parameters. Application of systemic and locally delivered antibiotics in relation to pocket depth or different parameters. Javed et al., summarizing nine studies, systemic and local antibiotic applications (e.g. tetracycline, doxycycline, amoxicillin, metronidazole minoxicycline hydrochloride, ciprofloxacin, sulfonamides + trimetho- prim) led to significant reductions of pocket depths in a period between one and six years Application of chlorhexidine resulted in the reduction of pocket depths, a higher implant adhesion and general weakening of inflammation measured by the level of the inflammatory markers IL-1 beta, VEGF and PGE-2 in various studies.

Laser therapy

By means of a bactericide mode of action, CO_2, Diode-, Er:YAG- (erbium-doped: yttrium-aluminum-garnet) and Er,Cr:YSGG- (erbium, chromium-doped: yttrium- scandium-gallium-garnet) lasers are used in the treatment of peri-implant diseases with increasing frequency.

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 photosen- sitizers (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

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 In 2004 it was modified and called AKUT-concept by Lang et al.

Resective therapy

In analogy to periodontitis, resective surgery has been shown to be effective in reduction of BOP, probing depths and clinical signs of inflammation. The basic principles include the elimination the periimplant osseous defect using ostectomy and osteoplasty as well as bacterial de-contamination. Additionally, smoothening and polishing of the supracrestal implant surface (implantoplasty) may be applied.

Regenerative approaches

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

Implantoplasty

Implantoplasty, a procedure that is done to smooth contaminated implant surfaces, has been used in the treatment of peri-implantitis. It reduces the implant diameter, which might compromise the implant's strength.

CONCLUSION

The risks of peri-implantitis, such as smoking, alcohol consumption, diabetes and unsure but genetic features, prosthetic factors, implant design and parafunctional habits, are all shown as biological and mechanical factors that can influence the patient's inflammatory reaction and infection response host response. Conclusions As a result; the main effect of Peri-implant diseases is microbial plaque. However, it may play a role in the etiology of peri-implantitis in biological and mechanical factors and may have an increased effect on plaque involvement. For this reason, proper design of implants preserve periodontal tissues around of implants from periimplantitis should be performed in conscious individuals who do not have any systemic discomfort with a healthy periodantal tissue under appropriate conditions. In addition, the patient should be informed about oral hygiene protocols and prosthesis cleaning and should be regularly checked at dental examinations and in dental education faculties.

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