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CURRENT CONCEPT OF PERI-IMPLANTITIS- A REVIEW

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ABSTRACT

Peri-implant inflammations represent serious diseases after dental implant treatment, which affect both the surrounding hard and soft tissue. Due to prevalence rates up to 56%, peri-implantitis can lead to the loss of the implant without multilateral prevention and therapy concepts. Specific continuous check-ups with evaluation and elimination of risk factors (e.g. smoking, systemic diseases and periodontitis) are effective precautions. In addition to aspects of osseointegration, type and structure of the implant surface are of importance. For the treatment of peri-implant disease various conservative and surgical approaches are available. Mucositis and moderate forms of peri-implantitis can obviously be treated effectively using conservative methods. These include the utilization of different manual ablations, laser-supported systems as well as photodynamic therapy, which may be extended by local or systemic antibiotics. It is possible to regain osseointegration. In cases with advanced peri-implantitis surgical therapies are more effective than conservative approaches. Depending on the configuration of the defects, respective surgery can be carried out for elimination of peri-implant lesions, whereas regenerative therapies may be applicable for defect filling. The cumulative interceptive supportive therapy (CIST) protocol serves as guidance for the treatment of the peri-implantitis. The aim of this review is to provide an overview about current data and to give advices regarding diagnosis, prevention and treatment of peri-implant disease for practitioners.

KEYWORDS: Periodontal Disease, Mucositis, Peri-implantitis Therapy, Epidemiology, Etiology.

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.^[1] 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. The present review gives an updated overview on the pathogenesis, etiology, risk factors and prevention of peri-implantitis, but also on actual recommendations in treatment and therapy options.^[2]

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%.^[3] However, the prevalence of peri-implant diseases, evaluated recently by Mombelli et al., revealed periimplantitis in 20% of all implanted patients and in 10% of all inserted implants. Although this percentage has to be interpreted with caution because of the variability of the analyzed studies, it underlines the fact that bone remodelling processes often result in marginal bone loss during the first weeks after abutment connection which cannot be regarded as peri-implantitis. This led to the recommendation to take a radiograph after insertion of the supra structure and to consider it as a basis for any future assessment of peri-implant bone loss.^[3,4]

Frequently, a spectrum of pathogenic germs can be detected such as Prevotella intermedia, Prevotella nigrescens, Streptococcus constellatus, Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Treponema denticola and Tannerella forsythia. Rams et al. revealed 71.7% resistance to at least one antimicrobial substance in a group of 120 patients. 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 periimplantitis. 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. As another beneficial cause, smooth implant surfaces in comparison to rough surfaces can accelerate the periimplant inflammation.^[1,5]

Treatment

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. It is possible to reduce bleeding on probing scores by cleaning with piezoelectric scalers as well as with hand instruments, and no differences have been found between these methods concerning reduction of bleeding on probing, plaque index and probing depths after at least 6 months.^[6]

As to the above-mentioned methods, the efficacy of ultrasonic curettage seems to underly the use of air polishing systems. Persson et al. And Renvert et al. experienced significantly lower numbers of bacteria with partial reduction of plaque and bleeding scores after mechanical curettage, while Schwarz et al. reported 30%-40% less residual biofilm areas by using ultrasonic methods.^[7]

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.. The basis of this concept is a regular recall of the implanted patient and repeated assessment of plaque, bleeding, suppuration, pockets and radiological evidence of bone loss.^[8-11]

CONCLUSION

The ideal management of peri-implant infections should focus both on infection control of the lesion, detoxification of the implant surface, and regeneration of lost support. The most important part that should be kept in mind is that a healthy periodontal environment is absolutely necessary to achieve desirable treatment outcomes. Failure in controlling plaque is the most serious confounding factor that leads to inconsistencies in the results. Enormous efforts are needed to motivate patients to maintain their oral hygiene and follow instructions for which regular maintenance sessions need to be scheduled. Treatment options can be surgical and nonsurgical. To date, studies suggest that nonsurgical treatment of PI is unpredictable, and the use of chemical agents such as chlorhexidine has only limited effects on clinical and microbiological parameters. Adjunctive local or systemic antibiotics have shown to reduce bleeding on probing and probing depths in combination with mechanical debridement. Beneficial effects of laser therapy on PI have been shown, but this approach needs to be further evaluated. Implant surface bacterial debridement is essential in treating PI. Most studies suggest that establishing an adequate healthy periimplant tissue environment proved to be difficult since inflammation was still present in a significant number of patients. Future strategies include the development of surfaces that become antimicrobial in response to infection and improvements in the permucosal seal. Further research is still needed to identify strategies to prevent bacterial attachment and enhance normal cell/tissue attachment to implant surface.

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