Review Article

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Peri-Implantitis: A Systemic Review

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ABSTRACT

Background: Peri-implant inflammations represent serious diseases after dental implant treatment, which affect both the surrounding hard and soft tissue. Specific continuous check-ups with evaluation and elimination of risk factors 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. In cases with advanced peri-implantitis surgical therapies are more effective than conservative approaches. Depending on the configuration of the defects, resective 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: Mucositis, Peri-implantitis, Periodontal disease.

INTRODUCTION

Implant based dental rehabilitation technique has come to offer steadfast result hence it has become a cardinal entrenched therapy in order to restore missing natural teeth in regular clinical practice. van Velzen et al. has reported 91.6 % of success rate for dental implant and shows 7 % of peri-implantitis after 10 years follow up. Dental implant has majority of success rate in long term however failure does occur. Peri-implant disease which is commenced by bacteria have two subtypes

(1) Peri-implant mucositis and
(2) Peri-implantitis.

Peri-implant mucositis is the reversible inflammatory process of the soft tissue surrounding the peri-implant, which is followed by reddening, swelling and bleeding on probing. Peri-implantitis or Periimplantitis is characterized as an inflammatory reaction that affects the hard and soft tissue, which results in loss of supporting bone and pocket formation surrounding the functioning osseointegrated implant. Peri-implantitis has been put under three categories depending on the pocket depth and bone loss.[1]

Classification of peri-implantitis (Froum and Rosen 2012)

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 <25–50 % of the implant length[b]

Severe PD ≥8 mm (bleeding and/or suppuration on probing)[a]

Bone loss >50 % of the implant length

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[a]Noted one two or more aspects of the implants

[b]Measured on radiographs from time of definitive prosthesis loading to current radiograph. If not available, the earliest available radiograph following loading should be used.[2]

DEFINITION UND PATHOGENESIS

In analogy to gingivitis and periodontitis affecting the periodontium of natural teeth, an inflammation and destruction of soft and hard tissues surrounding dental implants is termed as mucositis and peri-implantitis. Thereby, transitions are often fluent and not clinically clearly separable. Mucositis describes a bacteria-induced, reversible inflammatory process of the peri-implant soft tissue with reddening, swelling and bleeding on periodontal probing. These are typical signs, but they are sometimes not clearly visible. Furthermore, 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. Bleeding on probing, bone loss and deep probing depths may have other reasons than inflammation, e.g. too deep insertion of the implant. Moreover, type and shape oft the implant, connection type, abutment and suprastructure material and the type of prosthetic suprastructure affect the peri-implant soft and hard tissues.[3]

Depending on the configuration of the bony defect, Schwarz et al. distinguished between an intraosseous class I defect and a supra-alveolar class II defect in the crestal implant insertion area. Spiekermann characterized the type of bone resorption into horizontal (class I), key-shaped (class II), funnel- and gap-like (class III a, b) as well as horizontal-circular (class IV) forms. However, it is not possible to conclude progression and prognosis criteria from these classifications.

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. The latter can be used for disgnostic purposes.[4]

CLASSIFICATION

Branemark in 1952 described titanium as a biocompatible material that “fuses” to bone and till date titanium is the gold standard material for of implants globally. Osseointegrated dental implants are predictable treatment option for replacement of missing teeth, around the world. Success rates of more than 90% and predictability of dental implants is well published in literature, but still complications and failures occurs. There are number of classifications of periimplant defects quoted in medical literature with their Pros and cons but till date there is no uniformly accepted classification. Various system of classification proposed in literature described below.

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.

This Classification informs about only two classes. No clinical and radiological interpretation is evident. Spiekermann 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

This classification describes 5 different patterns of bone resoption around implant. No clinical criteria and treatment protocol for each class is given in it. Another system of classification

exists amount of bone loss with shaped of defect associated

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

This classification reveals horizontal bony defect along with other types of bony defects around implant. No clinical picture, treatment modality and prognosis is highlighted.

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.

The classification could be a useful tool for planning the correct regenerative procedure for each type of defect but classification lacks clinical and radiological assessment statistically.[5]

Peri-implantitis is classified into[18]:

2.1. Early Peri-implantitis

PD ≥ 4 mm, Bleeding and/or suppuration on probing, Bone loss <25% of the implant length.

2.2. Moderate Peri-implantitis

PD ≥ 6 mm, Bleeding and/or suppuration on probing, Bone loss ranging from 25% to 50% of the implant length.

2.3. Advanced Peri-implantitis

PD ≥ 8 mm, Bleeding and/or suppuration on probing, Bone loss >50% of the implant length.

This classification provides a standarised method about clinical and radiographic status of implant but it does not share any information of information related to management of periimplantitis and prognosis.[6]

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.[5]

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%.[6]

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. Although this percentage has to be interpreted with caution because of the variability of the analyzed studies, it underlines the fact that bone remodeling 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 suprastructure and to consider it as a basis for any future assessment of peri-implant bone loss.[7]

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 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. As another beneficial cause, smooth implant surfaces in comparison to rough surfaces can accelerate the peri-implant inflammation.[8]

RISK FACTORS AND PREVENTION

Implant loss may occur as "early implant loss" up to one year after implant insertion and "delayed implant loss" with a time period of more than one year after implant insertion. 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.

Studies indicate smoking as the greatest identifiable and most often cited risk factor for peri-implant disease followed by a history of periodontitis. Both are related to higher prevalences of peri-implantitis. The presence of periodontitis or cigarette smoking increased the risk for peri-implantitis up to 4.7-fold as reported by Wallowy et al. Moreover, smoking has been shown to be a predictor for implant failure. In a recent meta-analysis smoking increased the annual rate of bone loss by 0.16 mm/year and represented the main systemic risk factor. The extent of osseointegration as well as the oral hygiene around dental implants was found to be reduced among smokers. It is commonly accepted that the outcome of almost all intraoral therapeutic parameters are negatively affected by smoking although not in all previous studies a positive correlation between peri-implantitis and tobacco smoking could be found. Evidence of predictors for implant success such as gender or age could not be found but for the jaw of treatment (maxillary versus mandibular implants). In a study by Vervaeke et al. maxillary implants were at a significantly 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.[7, 8]

Across an observation period of 10 years in a group of patients with periodontitis, the previously eliminated bacterial strains of Aggregatibacter actinomycetemcomitans and Porphyromonas gingivalis could again be detected in the oral mucosa. Prevotella intermedia was, however, continuously evident. This indicates a niche survival of bacteria after tooth extraction with recurrence of the same microflora after a short period of time. In particular, attention should be paid to the remaining teeth with periodontitis as a potential source of infection. Therefore, the type of dentition (edentulous versus partially edentulous) may influence the colonization of peri-implant tissues with periodontal pathogens.[9]

The impact of keratinized gingiva around dental implants has been controversially discussed, but most studies emphasize the importance of an adequate zone of keratinized tissue surrounding implants. The so called "cementitis" may be regarded as the most important identifiable iatrogenic risk factor since its first description by Wilson et al. in 2009. The latter group revealed that residual dental cement in a group of patients with clinical or radiographic signs of peri-implant disease was present in 81% of the sites. After its removal, clinical signs were absent in 74% of the affected sites. Korsch et al. found that the removal of cement remnants led to a decrease of the inflammatory response by almost 60%.[46] Linkevizius et al. examined the manifestation of peri-implantitis in a group of patients with present cement remnants. In those who had a history of periodontitis, peri-implantitis was found in 100% of the patients, whereas cement remnants in patients with no previous periodontal disease ended up in 65% peri-implantitis manifestations. Another preventive arrangement regarding antibacterial

precautions are internal connections with inward located microgap, which should be preferred.[10]

Peri-implant probing is recommended to be carried out carefully with a minimal probing force. However, the so-called platform switch (abutment is located horizontally between implant and crown) can complicate probing and, thus, hide the true extension of peri-implantitis. Nevertheless, studies have indicated that platform switch might be an important protective factor against peri-implant disease.

Implant loss can be differentiated on the basis of the following additional factors:

- Overloading of the implant,
- Faults in material and techniques,
- Poor bone quality at the implant area,
- Systemic diseases and drug therapies, which inhibit bone modulations according to "Wolff's law" (bone density and strength increase with stress - and vice versa).[11]

Thus, implants of more than 10 mm length in square thread design show higher success rates than shorter implant lengths or shapes without thread or buttress thread. Also rough implant surfaces of more than 2 microns seem to feature better osseointegration than smooth (<0.5 microns) or moderate surfaces (1–2 microns).

Development of strengths in the temporomandibular joint of more than 1300 Newton may shift the implants in the first few months of healing up to 100 microns by presence of sagittal forces acting from an average of 50 Newton. These average reference forces even increase to 87 Newton when articulation angles up to 60° in horizontal axis.[12]

In addition to patient training sessions for optimal oral hygiene, preventive strategies such as professional tooth and implant cleaning as well as individually continuous peri-implant examinations (probing status) should be considered in order to prevent peri-implant diseases.[10]

DIAGNOSIS

According to the included articles, the time of prosthesis placement should be chosen to establish baseline criteria at both clinical and radiographic levels. Recorded baseline data should be used as a reference from which the development of peri-implant disease can be recognized and followed in subsequent examinations. The following parameters are suggested to be used for the diagnosis of peri-implantitis:[12]

Pain

Only one paper suggested using pain as a diagnostic parameter. Pain should not be associated with the implant after healing. Percussion and forces up to 500 g may be used clinically to evaluate implant pain or discomfort. Usually, pain from the implant body does not occur unless the implant is mobile and surrounded by inflamed tissues or has rigid fixation but impinges on a nerve. Pain during function places the implant in the failure category.

Mobility

Five articles suggested evaluating implant mobility. All authors agreed that implant mobility indicates the final stage of peri-implant disease, characterized by complete loss of the direct bone-to-implant interface.

Probing

Probing around implants should be repeated over time. The specified PD varies between ≥ 4 mm and > 5 mm. According to different authors, different thresholds are referred to as peri-implantitis: > 6 mm PD; ≥ 4 mm initial peri-implantitis, ≥ 6 mm moderate peri-implantitis, and ≥ 8 mm severe peri-implantitis; < 4 mm pockets indicate soft-tissue inflammation and > 4 mm soft-tissue pockets.[13]

Bone loss

Conventional periapical radiographs are recommended. Panoramic radiographs (PRs) might also be used for peri-implantitis diagnosis. However, three-dimensional radiographs, whereby not only mesial and distal but also buccal and lingual/palatinal bone walls could be evaluated, are superior. Bone-loss thresholds to diagnose peri-implantitis suggested by the authors differ: The implant success index by Kadhazahed et al. reported

that ≤ 2 mm (≤ 20%) of bone loss indicated the initiation of hard-tissue breakdown, 2 - 4 mm (< 40%) indicated hard-tissue breakdown, and > 40% indicated severe bone loss. Ata-Ali et al. [26] suggested using different stages of peri-implantitis based on the amount of marginal bone loss beyond biological bone remodelling (Stage I: ≤ 3 mm; Stage II: > 3 mm but < 5 mm; Stage III: ≥ 5 mm; Stage IV: ≥ 50% of the implant length).[14]

Other clinical parameters

The paper by the American Academy of Periodontology suggested that bacterial culturing, inflammatory markers, and genetics may be useful in the diagnosis of peri-implantiti.[14]

TREATMENT

In light of the aforementioned evidence and given the continuously increasing number of implants placed in everyday clinical practice, it is reasonable to anticipate an increasing prevalence of peri-implantitis, which underlines the necessity for a predictable therapy. Peri-imlantitis and its causes is still poorly understood. The decision process for peri-implantitis maintenance and treatment should be a rational and evidence-based approach.[15]

The oral microflora seems to be a defining factor for the success or the failure of a dental implant. As soon as an implant surface is exposed to the oral cavity, it becomes immediately covered by a protein layer – the salivary pellicle – and is colonized by oral microorganisms, forming a microbial biofilm Therapeutic strategies proposed for managing peri-implant diseases appear to be largely based on either the evidence available for treating periodontitis or on clinical empirical values but not on particular scientific findings. Surface debridements constitute the basic element for treating both periodontitis and peri-implantitis. However, the screw-shaped design of the implants, combined with various surface modifications of titanium, may facilitate plaque accumulation, resulting in bacterial biofilm formation. Mechanical debridement on such surfaces may have a limited effect and can certainly not result in the complete removal of all adhering microorganisms. Therefore, adjunctive peri-implant therapies, such as antibiotics, antiseptics, and ultrasonic and laser treatments, have been proposed to improve the non-surgical treatment options of peri-implant mucositis and peri-implantitis. Regenerative procedures using a bone graft substitute in combination with a membrane have been proposed to treat bone defects in advanced cases of peri-implantitis.[14]

Local debridement

The implant should be cleaned by instruments softer than titanium, such as polishing with a rubber cup and paste, floss, interdental brushes, or using plastic scaling instruments. These have been shown not to roughen the implant surface unlike metal and ultrasonic scalers. Although implant surface damage can almost be prevented by using either ultrasonic scalers with a nonmetallic tip or resin/carbon fiber curettes, the presence of implant threads and/or implant surface roughness may compromise the access for cleaning.

The study by Karring et al. demonstrated that sub-mucosal debridement alone, accomplished by utilizing either an ultrasonic device or carbon fiber curettes, is not sufficient for the decontamination of the surfaces of implants with peri-implant pockets ≥ 5 mm and exposed implant threads. So it seems reasonable to suggest that mechanical or ultrasonic debridement alone may not be an adequate modality for the resolution of peri-implantitis.[16]

Implant surface decontamination

Four implant surface decontamination methods were compared in a monkey model: (1) air-powder abrasive technique followed by citric acid application, (2) air-powder abrasive technique, (3) gauze soaked in saline followed by citric acid application, and (4) gauze soaked alternately in 0.1% chlorhexidine and saline. Clinical parameters, radiography (including quantitative digital subtraction radiography), histology, and stereology did not reveal significant differences between any of the methods used. Findings from an in vitro study combining photosensitization by toludine blue solution and soft laser irradiation have indicated that elimination of bacteria from different titanium surfaces without modification of the implant surface was possible.[17]

Photodynamic therapy is a non-invasive method that could be used to reduce microorganisms in peri-implantitis. 2% chlorhexidine or 3% hydrogen peroxide can be used as topical antiseptics. Decontamination of affected implants with titanium plasma-sprayed or sandblasted/acid-etched surfaces may most easily and effectively be achieved by applying gauze soaked alternately in chlorhexidine and saline.[18]

The non-surgical treatment of peri-implantitis lesions using an erbium-doped:yttrium, aluminum, and garnet (Er:YAG) laser showed lower counts of F. nucleatum 1 month after therapy.[67] According to Schwarz et al., the Er:YAG laser and the combination of mechanical debridement/chlorhexidine are equally efficacious at 6 months after therapy in significantly improving peri-implant probing pocket depth and clinical attachment level, but the use of the Er:YAG laser provides a significantly higher reduction of bleeding on probing compared with the adjunctive application of chlorhexidine. However, in a subsequent study by Schwarz et al., the efficacy of the Er:YAG laser appeared to be limited to a 6-month period, particularly for advanced peri-implantitis lesions. It was further suggested that a single course of treatment with the Er:YAG laser may not be adequate for achieving a stable therapy of peri-implantitis and that additional therapeutic measures, such as supplementary use of the Er:YAG laser and/or subsequent osseous regenerative procedures, might be required.[19]

Anti-infective therapy

Specific microbial information regarding the presence of putative pathogens is indispensible to make a meaningful decision regarding systemic or local antibiotic therapy. Although the composition of the subgingival microbial component is important for the choice of the drug, oral distribution patterns of potential pathogens are also important in deciding whether an antimicrobial agent should be administered locally or systemically. To accomplish this task, clinician needs to look at the periodontal condition of the residual teeth.[20]

The study by Schwarz et al. demonstrated that the treatment of peri-implant infection by mechanical debridement with plastic curettes combined with antiseptic (0.2% chlorhexidine) therapy may lead to statistically significant improvements in bleeding on probing, peri-implant probing pocket depth, and clinical attachment level at 6 months compared with baseline. A study by Renvert et al. showed that the addition of antiseptic therapy to mechanical debridement does not provide adjunctive benefits in shallow peri-implant lesions where the mean probing pocket depth was <4 mm. Thus, it seems that the addition of antiseptic therapy to mechanical debridement does not provide adjunctive benefits in shallow peri-implant lesions with mean pocket probing depth <4 mm but seems to provide additional clinical improvements in deep peri-implant lesions with mean pocket probing depth >5 mm.[21]

Patients suffering from localized peri-implant problems in the absence of other infections may be candidates for treatment by local drug-delivery devices. Local application of antibiotics by the insertion of tetracycline fibers for 10 days can provide a sustained high dose of the antimicrobial agent precisely into the affected site for several days. The use of minoccline microspheres as an adjunct to mechanical therapy is beneficial in the treatment of peri-implant lesions, but the treatment may have to be repeated.[22]

If the problem is generalized, specific microbiological information is collected and antibiotics are administered systemically. Lang et al. suggest the following antibiotic regimes: systemic ornidazole 500 mg bd for 10 days or metronidazole 250 mg td for 10 days or a once daily combination of metronidazole 500 mg and amoxicillin 375 mg for 10 days. If peri-implantitis is associated with persisting periodontal disease, then both conditions need to be treated. In this case, the adjunctive use of systemic antibiotics may be considered. There are no clinical trials available nowadays on the systemic administration of antibiotics for the therapy of peri-implantitis.[18]

Provided that mechanical and antiseptic protocols are followed prior to administering antibiotic therapy, it appears that shallow peri-implant infection may be successfully controlled using antibioics. But it is still open to question whether deeper peri-implant lesions can be

adequately treated non-surgically by a combination of a local antibiotic and mechanical debridement.[17]

Surgical technique

Surgical resection is generally confined to implants placed in non-aesthetic sites. Surgical flap helps in comprehensive debridement and decontamination of the affected implant. Surgical therapy was carried out, using: (1) autogenous bone grafts covered by membranes, (2) autogenous bone grafts alone, (3) membranes alone, and (4) a control access flap procedure showed that defects treated with membrane-covered autogenous bone demonstrated significantly larger amounts of bone regeneration and reosseointegration than those treated with the other three procedures. However, membrane exposure is a frequent complication after such procedures. Exposure of porous e-PTFE membranes may result in bacterial penetration and lead to infection.[23]

To date, no randomized controlled clinical trials are available on the use of access flap surgery (open-flap debridement) alone for the therapy of periimplantitis. A randomized comparative clinical trial by Romeo et al. concluded that resective surgical procedures coupled with implantoplasty could have a positive influence on the survival rates of rough-surfaced implants affected by peri-implantitis as well as on peri-implant clinical parameters, such as pocket-probing depth, suppuration, and sulcus bleeding. The study by Schwarz et al. demonstrated that both nanocrystalline hydroxyapatite and guided bone regeneration provided clinically significant improvements in clinical parameters following 6 months of non-submerged healing. The 2-year results by Schwarz et al. of the same clinical study once more demonstrated that both treatment modalities were efficacious in providing clinically significant reductions of pocket-probing depth and gains in clinical attachment level, but the application of the combination of natural bone mineral and collagen membrane seemed to correlate with greater improvements in those clinical parameters and, hence, was associated with a more predictable and enhanced healing outcome. Unfortunately, the relatively small sample size of the study (22 patients) did not allow a reliable statistical comparison of the efficacy of the two therapeutic procedures. In general, more data on various regenerative techniques for treating peri-implantitis have to be accumulated.[24]

Explantation

If there is advanced bone loss and the implant cannot be saved, it has to be removed. If a decision has been made to remove the implant, explantation trephines are available to suit the implant system concerned. It should be noted that these trephines have an external diameter of up to 1.5 mm greater than the diameter of the implant to be removed. Thus, explantation may be associated with significant bone removal including buccal or lingual bone cortices, and damage to adjacent natural teeth where the inter-radicular space is limited. An alternative approach is to allow progressive bone loss from peri-implantitis to occur, resulting in sufficient bone loss to allow for the removal of the implant with extraction forceps. Implants may be removed by forceps when there is less than 3 to 4 mm of residual bone support.[18]

SUMMARY

Due to the lack of prospective randomized long-term follow-up studies lots of approaches but no “ideal peri-implantitis therapy” have been described. Therefore, prevention is the most important instrument based on appropriate treatment planning, an atraumatic approach for implant insertion and continuous check-up intervals with professional teeth and implant cleaning. Above all, attention should be paid to risk factors such as smoking and active or previous periodontitis.

In non-surgical therapy, combinations of mechanical cleaning with curettes and air polishing systems are recommendable. Adjuvant antiseptic rinses and local or systemic antibiotics are effective for short-term bacteria eradication; laser and photodynamic therapy are additional treatment options. However, results for long-term benefits for these methods are missing.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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