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Review Article

Management of peri-implantitis

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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, degree of roughness, external morphology, and excessive mechanical load. The microorganisms most commonly associated with implant failure are spirochetes and mobile forms of Gram-negative anaerobes, unless the origin is the result of simple mechanical overload. Diagnosis is based on changes of color 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 various treatment options in the management of peri-implantitis. The article also gives a brief description of the etiopathogenesis, clinical features, and diagnosis of peri-implantitis.

Key Words: Dental implant, peri-implantitis, peri-implant mucositis

INTRODUCTION

The name peri-implant disease refers to the pathological inflammatory changes that take place in the tissue surrounding a load-bearing implant.[1] Two entities are described within the concept of peri-implant disease: peri-implant mucositis and peri-implantitis. Peri-implant mucositis is defined as a reversible inflammatory reaction in the soft tissues surrounding an implant.[2] Peri-implantitis is an inflammatory reaction with loss of supporting bone in the tissues surrounding an implant.[3] The overall frequency of peri-implantitis was reported to be 5% to 8% for selected implant systems.[4] An increasing number of studies suggests that anaerobic plaque bacteria may have an adverse effect on peri-implant tissue health leading to peri-implantitis.[5] Peri-implantis can also be directly is related to inadequate distribution of the chewing pressure on the tissues surrounding the implant, thus leading to loosening of the artificial supports, infection of the surrounding tissues, and consequently inflammatory processes.[6] Failure of a dental implant is often related to failure in osseointegration. A dental implant is considered to be a failure if it is lost, mobile, or shows peri-implant bone loss of greater than 1.0 mm in the first year and greater than 0.2 mm a year after. Peri-implantitis can result in bone loss around the implant and eventual loss of the implant. The optimal result of peri-implantitis treatment is regeneration of the lost implant supporting hard and soft tissues.[7]

Bacterial infections play the most important role in the failure of dental implants. Bacterial flora, which are associated with periodontitis and peri-implantitis, are found to be similar.[8] The microorganisms most commonly related to the failure of an implant are the Gram-negative anaerobes, like *Prevotella intermedia, Porphyromonas gingivalis, Aggregatibacter*

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actinomycetemcomitans, Bacterioides forsythus, Treponema denticola, Prevotella nigrescens, Peptostreptococcus micros, and *Fusobacterium nucleatum*.[8,9] Healthy peri-implant tissue plays an important role as a biological barrier to some of the agents that cause peri-implant disease, and if that is destroyed, bacterial contamination spreads directly to the bone, leading to its rapid destruction. Excessive mechanical stress, poor design of the implant, and the corrosion that can occur when a non-noble metal structure is connected to a titanium implant are important factors in the onset and development of peri-implantitis. Other etiological factors include diabetes mellitus, osteoporosis, smoking, long-term treatment with corticoids, radiation, and chemotherapy.

The following signs and symptoms are typical for peri-implantitis lesions: radiological evidence for vertical destruction of the crestal bone. The defect is usually saucer shaped and there is osseointegration of the apical part of the fixture; vertical bone destruction associated with the formation of a peri-implant pocket; bleeding and suppuration on probing; possible swelling of the peri-implant tissues, and hyperplasia. Pain is an unusual feature, which, if present, is usually associated with an acute infection. The diagnosis of peri-implantitis needs careful differentiation from peri-implant mucositis, primary failures to achieve tissue integration, and problems lacking an inflammatory component. The diagnostic parameters used for assessing peri-implantitis include clinical indices, peri-implant probing using a rigid plastic probe, bleeding on probing (BOP), suppuration, mobility, peri-implant radiography, and microbiology.

MANAGEMENT MODALITIES OF PERI-IMPLANTITIS

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.[1] The decision process for peri-implantitis maintenance and treatment should be a rational and evidence-based approach.[10]

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.

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.[11] 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.[12]

The study by Karring *et al*.[13] 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.

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.[14] 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.[15]

Photodynamic therapy is a non-invasive method that could be used to reduce microorganisms in peri-implantitis.[16] 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.[12]

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.[17] According to Schwarz *et al*.,[18] 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*.,[19] 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.

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.

The study by Schwarz *et al*.[18] 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*.[20] 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.

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[5] 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.[21] The study by Renvert *et al*.[20] demonstrated that the adjunctive benefits derived from the addition of an antibiotic minocycline to mechanical debridement tend to be greater, although to a limited extent, than those achieved by the combined use of an antiseptic (chlorhexidine) and mechanical debridement. The improvements in peri-implant probing depths obtained by the adjunctive use of minocycline can be maintained during a short-term period of 12 months. In the study by Renvert *et al*.,[20] the exhibited bone loss was not more than three implant threads.

If the problem is generalized, specific microbiological information is collected and antibiotics are administered systemically. Lang *et al*.[5] 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.

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.[1] 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.

Surgical technique

Surgical resection is generally confined to implants placed in non-aesthetic sites.[22] 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.[12] 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*.[24,25] 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*.[26] 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*.[27] 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.

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.

DISCUSSION

Owing to the fact that the frequency of late implant failures is relatively low, the number of longitudinal studies evaluating different treatment protocols for peri-implantitis is limited. Furthermore, ethical considerations often disallow the incorporation of proper placebo control in such trials. The treatment protocol will differ depending on whether it is peri-implant mucositis or peri-implantitis. If there is no bone loss, i.e. in the case of mucositis, bacterial plaque and calculi should be removed and chemical plaque control is achieved with 0.12% chlorhexidine applied topically, every 8-12 h for 15 days; the patient must give oral hygiene instructions. Prosthetic design should also be checked and modified if necessary, in order to correct design defects that impede proper

hygiene, as well as to correct biomechanical stress factors involved. Once this initial phase is completed, periodic check-up must be scheduled, gradually reducing the interval between maintenance visits.[28]

If peri-implantitis is diagnosed, treatment will depend on the amount of bone lost and the esthetic impact of the implant in question. If bone loss is at an incipient stage, treatment will be identical to that prescribed for peri-imlpant mucositis, with the addition of decontamination of the prosthetic abutments and antibiotics. If bone loss is advanced or persists despite initial treatment, it will be necessary to surgically debride the soft, peri-implant tissues affected by the chronic infection, decontaminate the microimplant surface, and finally apply bone regeneration techniques aimed at recovering the lost bone.

Until now, no methodology has been established as a gold standard approach for the treatment of peri-implantitis. So the therapy of peri-implantitis comprises (a) the nonsurgical phase, which includes debridement by mechanical means, ultrasonic, or laser devices, either alone or combined with antiseptic and/or antibiotic agents and (b) the surgical phase, utilizing either resective or regenerative techniques.

The available randomized controlled and/or comparative clinical trials on peri-implantitis treatment are limited in number and have short follow-up periods and small sample sizes, thereby exhibiting a high risk of bias. It is still dubious which therapeutic strategies are the most efficacious for the treatment of peri-implantitis lesions according to their morphology, extent, and severity. However, this does not suggest that currently implemented treatment modalities may not provide beneficial outcomes in clinical practice.

Despite the less than adequate level of existing evidence, certain data tend to indicate the following. Sub-mucosal debridement alone may not be adequate for the removal of bacterial load from the surfaces of implants with peri-implant pockets ≥5 mm.[13] The use of the Er:YAG laser can improve peri-implant clinical parameters within 6 months, but it remains unclear whether these effects can be maintained over time.[19] The combination of minocycline and mechanical debridement appears to provide an improved treatment outcome, although to a limited extent, compared with the combination of chlorhexidine and mechanical debridement, at least during a short-term period of 12 months.[20] Guided bone regeneration or the application of a bone substitute (nanocrystalline hydroxyapatite) can be efficacious for the treatment of peri-implantitis lesions.[26]

Only long-term clinical randomized controlled trials can give a definitive answer as to the best way of dealing with failing implants.

Long-term success of an implant depends on regular maintenance program. During maintenance phase, peri-implant tissue should be evaluated for inflammation. Radiographs will give the status of bone around implants. These programs help in the long-term success of an implant.

CONCLUSION

Prognosis of the affected implant will be contingent upon early detection and treatment of peri-implant mucositis and peri-implantitis. Even though the studies dealing with different treatment modalities of peri-implantitis are not comparable, an overall picture of some clinical improvement emerges with the use of anti-infective therapies, in terms of resolution of inflammation and bone healing. This observation, coupled with our knowledge of the indisputable role of periodontal pathogens in the etiology of peri-implantitis, indicates that some form of anti-infective therapy must be coupled with any other strategy for dealing with this problem.

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