International Journal of Health Sciences and Research
www.ijhsr.org ISSN: 2249-9571

Review Article

Peri-Implantitis: An Upcoming Complication of Concern

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Received: 02/05/2016 *Revised: 01/06/2016* *Accepted: 08/06/2016*

ABSTRACT

Recent advances in the field of dental implants have led to widespread use of them as a treatment of choice for missing teeth hence increasing the incidences of peri-implantitis. Peri-implantitis is the site-specific destructive inflammatory process affecting the soft and hard tissues surrounding dental implants. The array of periodontal pathogens similar to those found in association with various forms of periodontal disease; include the etiology of this disease around an osseointegrated implant. Other factors which govern the etiology include 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 in the clinical parameters namely color of the gingiva, bleeding and probing depth of peri-implant pockets, suppuration. Degree of bone destruction can be evaluated using radiographs. Depending on the diagnosis, treatment will differ for a case of peri-implant mucositis or peri-implantitis. This review article gives a brief summarization etiopathogenesis, clinical features, diagnosis and the varied treatment modalities available in the management of peri-implantitis.

Key words: Peri-implantitis, Peri-mucositis, debridement, decontamination, Bone loss.

INTRODUCTION

The name peri-implant disease alludes to the pathological inflammatory changes taking 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 a localized inflammatory lesion involving soft and hard tissues around an osseointegrated implant leading to surrounding bone loss. [3] The overall frequency of peri-implantitis was reported to be 5% to 8% for selected implant systems. [4] An array of studies suggests that plaque bacteria particularly anaerobic may be one of the primary causative agents acting on peri-implant tissue health leading to peri-implantitis. [5] The microorganisms most commonly related to the failure of an implant are the Gram-negative anaerobes, like *Prevotella intermedia, Porphyromonas gingivalis, Aggregatibacter actinomycetemcomitans, Bacteroides forsythus, Treponema denticola, Prevotella nigrescens, Peptostreptococcus micros*, and *Fusobacterium nucleatum*. [6,7] Peri-implantitis can also be directly is related to inadequate distribution of the masticatory

forces on the tissues surrounding the implant, thus leading to loosening of the artificial supports and consequent inflammatory processes. [8] Failure of a dental implant is often related to failure in osseointegration in a nutshell the etiopathogenesis of peri-implantitis is complex and related to a variety of factors that influence the peri-implant environment viz., 1) Patient-related factors including systemic diseases (e.g., diabetes, osteoporosis) and prior dental history (periodontitis), 2) Social factors such as inadequate oral hygiene, smoking, and drug abuse, 3) Parafunctional habits (bruxism and malocclusion). Others include iatrogenic factors inclusive of faulty restorations, cement left following restoration cementation, and/or loose components can also play a valid role in the development of peri-implantitis.

Although restorations of end osseous implants have demonstrated a very high survival rate, [9] one study suggested that over a five-year period, 0 to 14.4% of dental implants demonstrated peri-implant inflammatory lesions associated with crestal bone loss. [4] Following prove to be leading signs of peri-implantitis

- Progressive increase in probing depth
- Bleeding on probing
- Clinical appearance of inflamed tissue (bleeding, swelling, colour change, suppuration, and plaque/calculus accumulation)
- Suppurations and exudation from peri-implant space
- Progressive loss of crestal bone on follow-up radiographs: loss of supporting bone beyond 0.2 mm annually, after the expected physiologic remodelling.

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. The optimal result of peri-implantitis treatment is regeneration of the lost implant supporting hard and soft tissues. [10]

The diagnosis of peri-implantitis requires careful distinction from peri-implant mucositis and primary failures to achieve tissue integration. The diagnostic parameters used for determining peri-implantitis include clinical indices, peri-implant probing using a rigid graduated plastic probe, bleeding on probing (BOP), suppuration, mobility, peri-implant radiography, and microbiology.

Management Modalities of Peri-Implantitis

The oral microflora seems to be a critically significant factor for the success or the failure of a dental implant. A protein layer called the salivary pellicle instantly covers the implant surface as soon as it is exposed to the oral cavity. This pellicle is colonized by oral microbes, forming a microbial biofilm. Many methods of treating peri-implantitis have been documented in the literature and most focus on removal of the contaminating agent from the implant surface. The long-term objectives are to stop the progression of the disease and maintain the implant site. Depending on the etiology of the problem, a categorical treatment is selected. Appropriate management of peri-implantitis often calls for referral to a periodontist.

Surface debridement composes the chief element for treating both periodontitis and peri-implantitis. However, the screw-shaped design of the implants, along with various surface modifications of titanium, may promote plaque accumulation making mechanical debridement on such surfaces difficult resulting in limited effect and incomplete removal of all clinging microorganisms. Therefore, adjunctive peri-implant therapies are put forth as vivid options to improve treatment of peri-implantitis. These consist of such mechanical debridement with systemic antibiotic treatment, [11] mechanical debridement with localized drug delivery and chlorhexidine oral rinses, [12] mechanical debridement combined with LASER decontamination, [13] surgical debridement, and more recently - Surgical

debridement with guided bone regeneration (GBR) for reparation of bony and soft-tissue defects. [14]

Local Debridement

Conventional metal hand and ultrasonic scalers tend to produce scratches on the implant surface hence roughening it and facilitating further plaque retention. [15] 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. 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. [16]

The study by Karring *et al*. [17] demonstrated that sub-mucosal debridement alone, using either an ultrasonic device or carbon fiber curettes, is insufficient for the decontamination of the surfaces of implants with peri-implant pockets ≥ 5 mm and exposed implant threads. So one can infer that mechanical or ultrasonic debridement alone may not be an adequate modality for the resolution of peri-implantitis.

Implant Surface Decontamination

Implant surface bacterial decontamination is essential in treating peri-implantitis infections. 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. [18] No significant differences between any of the methods used were revealed. Elimination of bacteria from different titanium surfaces was possible without modification of the implant surface according to an in vitro study featuring combined use of photosensitization by toluidine blue solution and soft laser irradiation. [19]

Photodynamic therapy is a non-invasive method that could be used to decrease microorganisms at peri-implantitis site. [20] 2% chlorhexidine or 3% hydrogen peroxide can be used as topical antiseptics.

The use of an erbium-doped: yttrium, aluminum, and garnet (Er:YAG) laser showed lower counts of *F. nucleatum* 1 month after therapy. [21] According to Schwarz *et al*., [22] 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*., [13] the efficacy of the Er:YAG laser appeared to be limited to a 6-month period, particularly for advanced peri-implantitis lesions.

Anti-Infective Therapy

The study by Schwarz *et al*. [22] 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 the clinical parameters at 6 months compared with baseline. A study by Renvert *et al*. [11] showed that the addition of antiseptic therapy to mechanical debridement does not provide added benefits in shallow peri-implant lesions where the mean probing pocket depth was <4 mm but seems to provide additional clinical improvements in deep peri-implant lesions with mean pocket probing depth >5 mm.

Local drug delivery devices can be opted for in patients suffering from localized peri-implant problems in the absence of other infections. 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 minocycline microspheres as an adjunct to mechanical therapy is favorable in the treatment of peri-implant lesions, but the treatment may have to be repeated. [23] The study by Renvert *et al*. [11] demonstrated that the adjunctive advantages deduced 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 and the exhibited bone loss was not more than three implant threads.

If the problem is generalized, specific microbiological information is collected and a systemic approach of antibiotic administration is preferred. 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 are required to be treated. In this case, the adjunctive use of systemic antibiotics may be considered. No clinical trials are available nowadays on the systemic administration of antibiotics for the treatment of peri-implantitis.

Provided that mechanical and aseptic protocols are followed prior to administering antibiotic therapy, it appears that shallow peri-implant infection may be successfully controlled using antibiotics. [1] But it remains questionable 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 circumscribed to implants placed in non-aesthetic sites. [24] Surgical flap helps in meticulous 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 exhibited considerably larger amounts of bone regeneration and re-osseointegration than those treated with the other three procedures. [16] However, membrane exposure is a frequent complication after such procedures. Exposure of porous e-PTFE membranes may lead to bacterial penetration and reinfection. [25]

No randomized controlled clinical trials are documented on the use of access flap surgery (open-flap debridement) alone for the treatment of peri-implantitis. A randomized comparative clinical trial by Romeo *et al*. [26,27] inferred that resective surgical procedures combined with implantoplasty could have a positive impact on the survival rates of rough-surfaced implants affected by peri-implantitis as well as on peri-implant clinical parameters. The study by Schwarz *et al*. [28] demonstrated that both nanocrystalline hydroxyapatite and guided bone regeneration provided clinically notable improvements in clinical parameters following 6 months of non-submerged healing. The 2-year results by Schwarz *et al*. [22] of the same clinical study yet again demonstrated that both treatment modalities were effectual in providing considerable reduction of pocket-probing depth and gain in clinical attachment level, but the application combination of natural bone mineral and collagen membrane seemed to exhibit greater improvements in those clinical parameters and, therefore purring forth a more predictable and amplified healing outcome. More data on various regenerative techniques for treating peri-implantitis have to be gathered to validate the above findings.

Explantation

In case of advanced bone loss and the implant cannot be salvaged, it has to be removed. If the treatment calls for removal of the implant, explantation trephines are available as per the implant system concerned. Keeping in mind that these

trephines have an external diameter of up to 1.5 mm greater than the diameter of the implant to be removed, explantation may be associated with significant bone removal including buccal or lingual bone cortices. Also damage to adjacent natural teeth is inevitable, where the inter-radicular space is limited. The second approach is to allow progression of bone loss from peri-implantitis to occur, resulting in adequate bone loss so as to allow yielding 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

The frequency of failures of late implants is relatively low thereby providing limited the number of longitudinal studies evaluating varied treatment protocols for peri-implantitis. One has to differentiate whether it is peri-implant mucositis or peri-implantitis. Accordingly the treatment protocol will differ depending on proper diagnosis. If there is no bone loss, as in the case of mucositis, a non-invasive approach is sought involving meticulous removal of bacterial plaque and calculi and incorporation of chemical plaque control using topical application of 0.12% chlorhexidine, every 8-12 h for 15 days; oral hygiene instructions are must. Prosthetic design should be altered as and when necessary, in order to facilitate proper hygiene, as well as to correct occlusal stress factors and abnormalities involved. Once this initial phase is completed, periodic check-up must be scheduled, gradually reducing the interval between maintenance visits. [29]

If peri-implantitis is diagnosed, the amount of bone lost determines the treatment protocol. If bone loss is at an incipient stage, treatment will be similar to that prescribed for peri-implant mucositis, with the addition of detoxification of the prosthetic abutments and systemic antibiotics. If bone loss is advanced or persists despite initial treatment, it will be mandatory to surgically debride the soft, peri-implant tissues affected by the chronic infection, disinfect the micro implant surface, and finally apply bone regeneration techniques attempting to recover the lost bone.

Until now, no consensus on a particular methodology has been established as a gold standard approach for the treatment of peri-implantitis. Therefore, so far the therapy of peri-implantitis constitutes (a) the nonsurgical phase, which includes mechanical debridement by ultrasonic or laser devices, either alone or in combination with antiseptics and/or antibiotics and (b) the surgical phase, comprising of either resective or regenerative techniques.

Although the existing evidence is inadequate, certain available data tend to pinpoint the following. Sub-gingival debridement alone may not be competent enough for the removal of complete bacterial load from the implant surfaces with peri-implant pockets ≥5 mm. [17] The use of the Er:YAG laser has shown to improve peri-implant clinical parameters within 6 months, but it remains ambiguous whether these effects can be maintained over time. [13] A comparative study between combination of minocycline and mechanical debridement with the combination of chlorhexidine and mechanical debridement revealed an improved treatment outcome, although to a limited extent, with the former combination, at least during a short-term period of 12 months. [11] Guided bone regeneration or the application of a bone substitute (nanocrystalline hydroxyapatite) appears to be effectual for the treatment of peri-implantitis lesions. [28]

A decisive answer for the best way of dealing with failing implants can be come upon only from long-term clinical randomized controlled trials. New treatment options need to be evaluated using such studies to identify predictable and successful treatment of peri-implantitis.

Regular maintenance program defines long-term success of an implant. During maintenance phase, peri-implant

tissue should be evaluated for inflammation. The status of bone around implants can be evaluated with the help of radiographs.

CONCLUSION

Implants have become an exceedingly popular treatment of choice in many cases of edentulism. Thus owing to the increasing number of implants being placed, peri-implantitis has become much more prevalent. Early detection, differentiation, diagnosis and treatment of peri-implant mucositis and peri-implantitis determine the prognosis of the affected implant. Albeit the currently available data dealing with varied treatment options of peri-implantitis is not comparable, an apparent improvement in the clinical picture comes forth with the use of anti-infective therapies, with regard to resolution of inflammation and healing of bone. Most studies suggest difficulty in establishing a healthy peri-implant tissue environment owing to persistent inflammation in a significant number of patients. This observation, in unison with the knowledge of the unquestionable role of periodontal pathogens in the etiology of peri-implantitis, implies the implementation of a combination of anti-infective therapy with any other modality for dealing with this problem.

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How to cite this article: Mani A, Dalvi A, Mani S et al. Peri-implantitis: an upcoming complication of concern. Int J Health Sci Res. 2016; 6(7):319-325.

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