Review Article

Dentistry Section

Peri-Implantitis: A Risk Factor In Implant Failure

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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. Implant failure has classically been attributed to bacterial infections, occlusal overload, surgical trauma, faulty or incorrect prosthetic design and/or improper surgical placement. The management of implant infection should be focused both on the infection control of the lesion, the detoxification of the implant surface and regeneration procedures. The treatment option can be surgical or non-surgical. It was observed that the non surgical treatment of peri-implantitis was unpredictable, while the use of chemical agents such as chlorhexidine had only limited clinical effects. Adjunctive local or systemic antibiotics were shown to reduce bleeding on probing and probing depths and some beneficial effects of laser therapy on peri-implantitis have been shown. Regenerative therapies can also be applied in conjunction with the anti-infective procedures. The purpose of this paper is to review the literature with regards to peri-implantitis as a risk factor in implant failure and also to identify the protocols which may assist in its diagnosis and management.

Key words: Peri-implantitis, Re-osseointegration, Implant failure

INTRODUCTION

The oral rehabilitation of partially or totally edentulous patients with dental implants has become a common practice over the last decade, with reliable long term results. The documented high survival rate of osseointegrated root form dental implants has led to their acceptance as a realistic treatment alternative. In spite of these successes, however, over a 5 year period, 0 to 14.4% of the dental implants demonstrated peri-implant inflammatory reactions which were associated with crestal bone loss that may eventually lead to the loss of an implant[1].

Peri-implantitis is defined as an inflammatory process which affects the tissues around an osseointegrated implant in function, resulting in the loss of the supporting bone, which is often associated with bleeding, suppuration, increased probing depth, mobility and radiographical bone loss. It has been shown that the inflammation is more pronounced and the inflammatory process goes deeper and faster around the dental implant than around the adjacent natural tooth [2].

It has been suggested that implants have a less effective natural tissue barrier than natural teeth and are less resistant to infection. The predictability of a stable soft tissue attachment has not been confirmed, and the permucosal seal may be just a circular fiber arrangement around the implant [3].

AETIOLOGY

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[4].

Studies have shown that the bacterial flora at the failing implant sites consist of gram-negative anaerobic bacteria including *Porphyromonas gingivalis*, *Prevotella intermedia* and *Actinobacillus actinomycetem comitans*, which resemble the pathogens in periodontal disease[5].

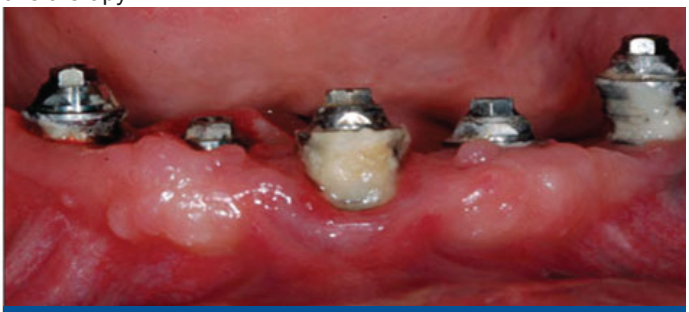
It has been demonstrated that the bacteria which are found in the implant sulcus in the successful implant cases, are basically the same flora as are found in the natural tooth sulcus in a state of health. The implants in partially edentulous patients appear to be at a greater risk of peri-implantitis than the implants in completely or fully edentulous patients. There are few qualitative differences in the microflora surrounding implants and the teeth in partially edentulous patients. However, there is a marked quantitative decrease in the number of periodontal pathogens around the implants in completely edentulous patients. It is possible that the natural teeth may serve as reservoirs for periodontal pathogens from which they may colonize the implants in the same mouth [6]. This reinforces the importance of rigorous oral hygiene programs in the implant patients.

Biomechanical factors such as an occlusal overload may play a significant role in the failure of the implant. The occlusal overload may result in progressive bone loss around the implant, thus leading to the failure of the implant. The implants which suffer from traumatic failure have subgingival microflora resembling that which is present in a state of periodontal health, with cocci and nonmotile rods as the predominant morphotypes i.e. *Streptococcus* and *Actimomyces* species as the predominant microflora[7].

The other aetiological factors are patient related factors that include systemic diseases e.g. diabetes mellitus, osteoporosis, etc; social factors- such as inadequate oral hygiene, smoking and drug abuse; para functional habits e.g. bruxism and iatrogenic factors such as lack of primary stability and premature loading during the healing period [8].

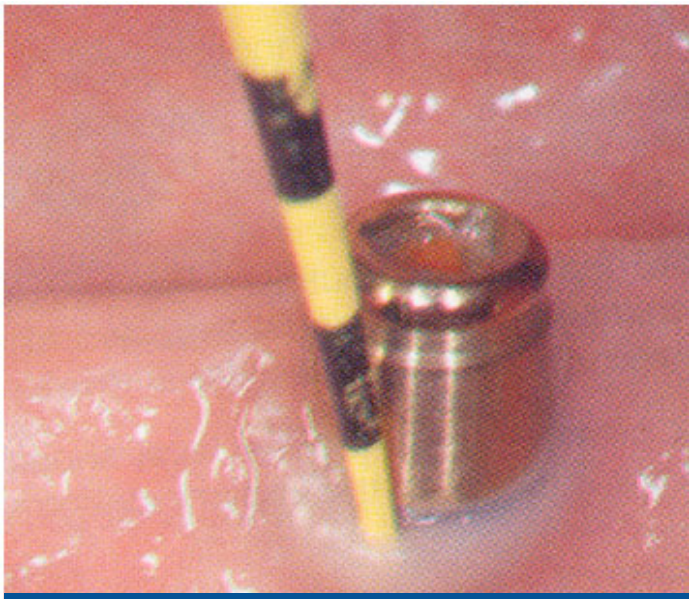
DIAGNOSIS

A number of clinical parameters which are used to evaluate periodontal conditions have also been used to assess the peri-implant conditions. Swelling, redness of the peri-implant marginal tissues, calculus build up and bleeding on probing are important signs of peri-implantitis [Table/Fig 1]. Suppuration is a clear indicator of the disease activity and indicates the need for anti-infective therapy.



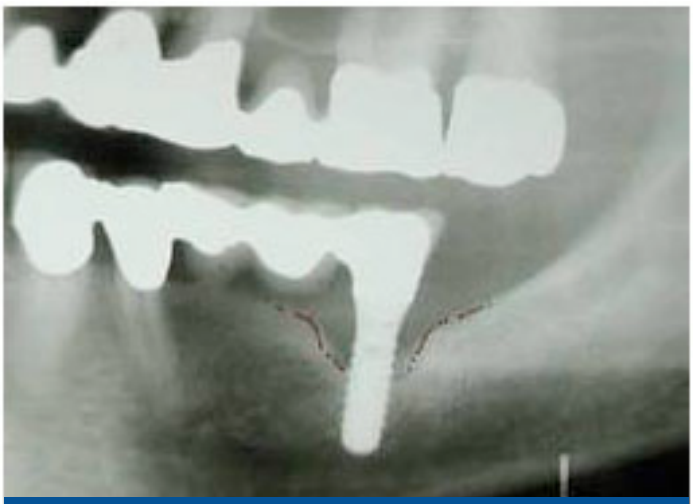
[Table/Fig 1]: Redness of peri-implant tissue and calculus build up

Probing the peri-implant sulcus with a blunt, straight plastic periodontal probe such as the automated probe or the TPS probe, allows the assessment of the following parameters: a) Peri-implant probing depth b) Bleeding on probing and c) Exudation and suppuration from the peri-implant space. Studies have shown that successful implants generally allow a probe penetration of approximately 3 mm to 4mm in the healthy peri-implant sulcus [Table/Fig 2].



[Table/Fig 2]: Plastic probe inserted around an implant abutment

Periapical intra oral radiographs reveal the peri-implant bone status as well as the marginal bone level. Progressive bone loss is a definite indicator of peri-implantitis, but it should not be confused with physiological bone remodeling around the implant during the first year of function[9] [Table/Fig 3].



[Table/Fig 3]: Bone loss around implant in function

The implant mobility serves to diagnose the final stage of osseodisintegration. For the interpretation of low degrees of mobility, an electronic device like periotest has been used.

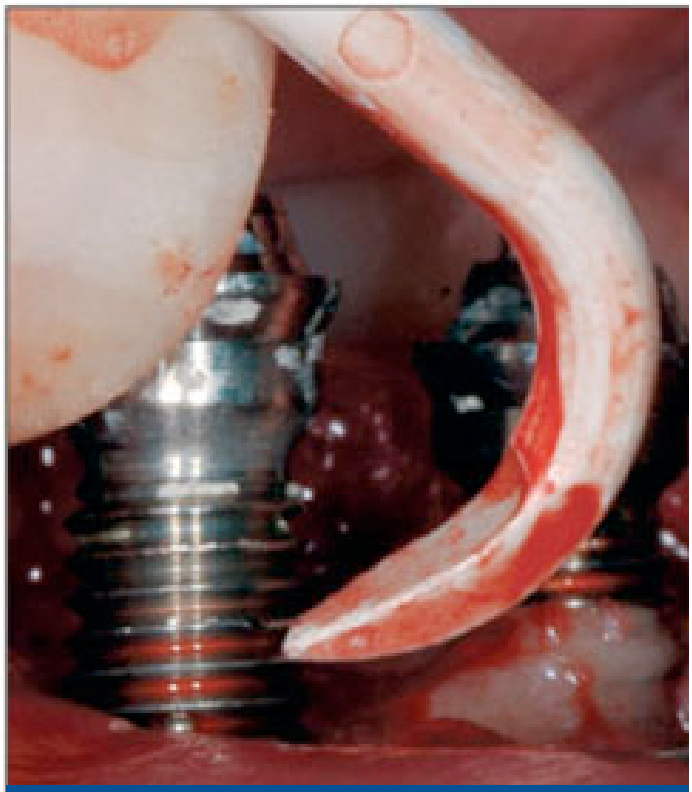
Bacterial cultures, DNA probes, polymerase chain reaction (PCR), monoclonal antibody and enzyme assays which are used to monitor the subgingival microflora can help to determine an elevated risk for peri-implantitis. It is a biologically sound and good medical practice to base the systemic antimicrobial therapy on appropriate microbiological data.

MANAGEMENT

If clinical and radiological evidences suggest that the peri-implant conditions are not stable and that advancing bone loss is occurring, this indicates the need for intervention. Peri-implantitis is managed by using specific treatment strategies, depending on the aetiology of the problem.

When biochemical forces are considered as the main aetiological factors; then the first phase involves an analysis of the fit of the prosthesis, the number and position of the implants, and an occlusal evaluation. Occlusal equilibration; improvement of the implant number and position, and changes in the prosthetic design can contribute to arrest the progression of the peri-implant tissue breakdown. The second phase includes a surgical technique to eliminate the deep peri-implant soft tissue pockets or to regenerate the bone around the implant.

When the main aetiological factor is bacterial infection; the first phase involves the control of the acute infection and the reduction of inflammation. This involves the local removal of the plaque deposits with plastic instruments (Implacare) [Table/Fig 4] and the polishing of all the accessible surfaces with pumice, the subgingival irrigation of all peri-implant pockets with 0.12% chlorhexidine; systemic antimicrobial therapy for 10 consecutive days; and improved patient compliance with oral hygiene until a healthy peri-implant site is established. This may be sufficient to re-establish gingival health or may need to be followed by a surgical approach in the second phase[10].



[Table/Fig 4]: Removal of plaque deposits with plastic scaler

The implants which are affected with peri-implantitis are contaminated with soft tissue cells, microorganisms and microbial byproducts. The defect must be debrided and the contaminated implant surface has to be treated to achieve the regeneration of new bone and for 're-osseointegration' to occur. Conventional hand and ultrasonic instruments are not suitable for the preparation and detoxification of the implant surface. Prophy jet, the use of a high pressure air powder abrasive (mixture of sodium bicarbonate and sterile water), has been advocated, as this removes the microbial deposits, does not alter the surface topography and has no adverse effect on cell adhesion. Various chemotherapeutic agents like contact with a supersaturated solution of critic acid (40% concentration; pH 1) for 30-60 seconds have been used for the preparation of the implant surfaces, as they have the highest potential for the removal of endotoxins from both the hydroxy-

apatite and the titanium implant surfaces. Soft laser irradiation has also been used for the elimination of the bacteria which are associated with peri-implantitis [11].

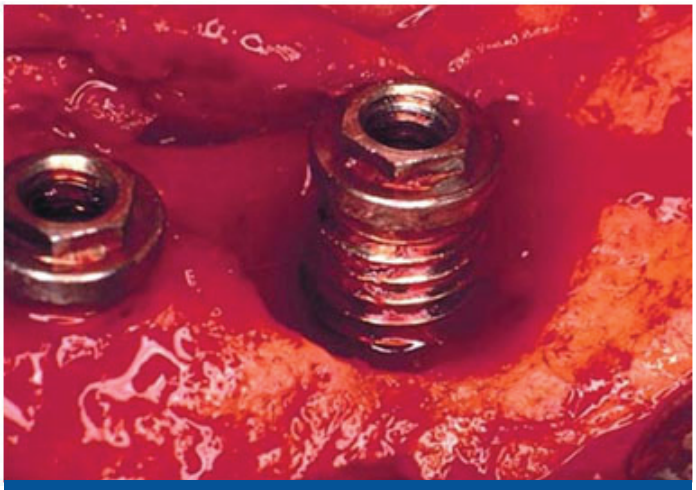
Additionally, the systemic administration of antibiotics that specifically target gram-negative anaerobic organisms has shown an alteration in the microbial composition and a sustained clinical improvement over a 1-year period [12].

Alternatively, a local delivery device, Actisite (fibers containing polymeric tetracycline HCl) has been tried and this resulted in significantly lower total anaerobic counts [13].

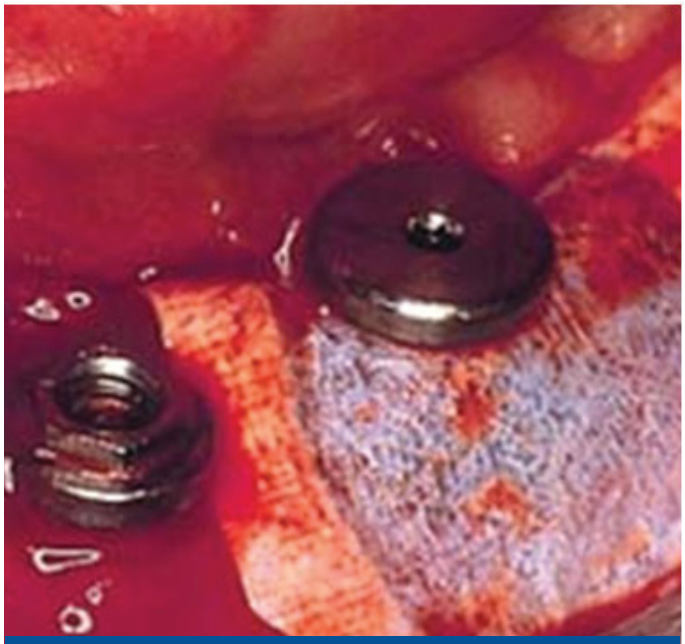
The type of osseous defects should be identified before deciding on the surgical treatment modality. If the defect is in the unaesthetic zone and is mainly of the horizontal type, the management can focus on the correction of the soft tissue portion of the peri-implant pocket. Standard techniques such as gingivectomy and apically displaced flaps are used in these situations to reduce the pocket and to improve the access for oral hygiene.

If the vertical (< 3mm) 1 to 2-wall defects are found, then the respective surgery can be used to reduce the pockets, to smoothen the rough implant surfaces, to correct the osseous architecture and to increase the area of the keratinized gingival [14]. To arrest the progression of the disease and to achieve a maintainable site for the patient, all implant surfaces that are smooth and clean coronal to the bone level are preferred. Therefore, the surface with threads or roughened topography such a hydroxyapatites, are indicated for alteration with high speed diamond burs and polishers to produce a smooth continuous surface [15]. Surface modifications are not performed during a regeneration surgery, where metal particles can interfere with the regeneration of bones.

Various bone graft techniques and guided bone regeneration (GBR); even in conjunction with platelet rich plasma (PRP), have been successfully used for the regeneration of lost bones in 3 wall or circumferential defects. It is advisable to remove the prosthe - sis at the time of regenerative surgery; nevertheless, perigingival regenerative therapy for one stage implants or for implants with non-retrievable prosthesis can also be done. A thorough preparation of the implant surface should be followed by an elaborate rinsing with saline solution. Roughening of the bone surface can be done by penetration with round burs to increase the accessibility to the osteogenic cells. The membranes which are placed should ensure the complete coverage and the isolation of the bony defect. The reflected flap should be closed primarily over the site with a mattress and interrupted sutures. The membrane should be left undisturbed for 4-6 weeks. Intra-oral autogenous bone grafts are the most preferred types of grafts for GBR therapies [16]. Other bone graft materials like demineralized freeze- dried bone and hydroxyapatite can also be used [Table/Fig 5 and 6].



[Table/Fig 5]: Circumferencial bone defect around implant



[Table/Fig 6]: Bone defect covered with gtr membrane

The long term success of any peri-implant treatment strategy requires a program of periodic maintenance, including subgingival plaque removal and instructions in proper hygiene.

CONCLUSION

Peri-implantitis is an inflammatory process affecting the tissues around an osseointegrated implant in function, resulting in the loss of the supporting bone. Micro organisms play a major role in this disease, particularly gram negative anaerobic bacteria. Several treatment modalities are presently being evaluated, but however, there is still insufficient evidence to support an ideal universal therapy for peri-implantitis. Therefore, it appears reasonable to attempt the interception of destructive peri-implantitis as early as possible and to stop its progression by the removal of bacterial deposits. Long term treatment modalities need to be assessed and there is a need for randomized controlled studies which evaluate the non surgical and surgical treatment of of peri-implantitis.

REFRENCES

[1] Bergbundh T et al. A Systematic review of the incidence of biological and technical complications in implant dentistry. J Clin Periodontol. 2002; 29: 197-212.

[2] Lindhe J, Berglundh T et al. Experimental breakdown of peri-implant and periodontal tissue – A study in the Beagle dog. Clin Oral Implant Res. 1992; 3: 9-16.

[3] Meffert RM. The soft tissue interface in dental implantology. J Dent Educ. 1988; 52: 810-811.

[4] Hydenrjik K, Majjer JA. Microbiota around root-form endosseous implants: A review. The Int J of Oral and Maxillofacial Implants. Nov/Dec 2002, Vol17(6).

[5] Mombelli A and Long NP. The diagnosis and Treatment of Peri-implantitis. Periodontal 2000. 1998; 17: 63-76.

[6] Aspe P, Allen RP et al. Microbiota and crevicular fluid collagenase activity in the osseo integrated dental implant sulcus: a comparison of sites in edentulous and partially edentulous patients. J Perio Res. 1989; 24: 96-105.

[7] Mombelli A, Mericske-sterm R. Microbiological features of stable osseointegrated implants used as abutments for over denture. Clin Imp Res. 1990; 1: 1-7.

[8] Quirynen M, Van Der Mei HC, Bollen CML, Schotte A, Marechal M, Doornbusch GI et al. An in vivo study of the influence of the surface roughness of implants in microbiology of supra- and subgingival plaque. J Dent Res. 1993; 72(9):1304-1309.

[9] Alberktsson T et al. The long term efficacy of currently used dental implants. A review and proposed criterion of

success. Int J Oral Maxillofac Implants. 1986; 1: 11-25.
[10] Meffert RM. Treatment of ailing and failing implants J Cliff Dent Assoc. 1992; 3: 162.
[11] Deppi H et al. Peri-implant care of ailing implants with Co2 laser. Int J Oral Maxillofac Implants. 2001; 16; 659-667.
[12] Roos-Jansaker C et al. Treatment of Perio-implant infections. A literature review. J Clin Periodontol. 2003; 30: 467-485.
[13] Mombelli A et al. Treatment of Peri-implantitis by local delivery of tetracycline. Clinical, microbiological and radiological results. Clin Oral Implant Res. 2001; 12: 287-294.
[14] Jovanoic SA. The management of peri-implant breakdown around functioning osseointegrated dental implants. J Periodontol. 1993; 64: 1176.
[15] Jovanoic SA, Spickermann H and Richter EJ. Bone regeneration on titanium dental implants with dehisced defect sites. A clinical study. Int J Oral Maxillofac Implants. 1992; 7: 233.
[16] Newman HG, Takei HH, Carranza. Clinical Periodontology. 9th Edition,1987 p. 936-940.

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