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Review Article

PERI-IMPLANTITIS: ETIOLOGY, DIAGNOSIS AND MANAGEMENT

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ABSTRACT

Dental implants are considered to be effective and predictable treatment options for replacing missing teeth, with good long-term success rates. Peri-implantitis is one of the complications of implant treatment, which may lead to implant failure. The etiology of peri-implantitis is multifactorial, with microorganisms playing a major role in its development. An early diagnosis and intervention should be established for effective management of peri-implantitis. Even though different treatment strategies may have been employed, some with successful outcomes, long-term evaluation to assess the validity and reliability of the treatment techniques may be needed.

Keywords: Peri-Implantitis, Peri-Implant Mucositis, Peri-Implant Disease, Decontamination, Implantoplasty

INTRODUCTION

Endosseous dental implants have successfully been used during the last decades as a treatment modality for the replacement of missing teeth in partially or completely edentulous patients[1]. It has become increasingly possible to manage a broad range of clinical dilemmas with dental implants due to their high level of predictability and applicability for a variety of treatment options. While in many cases dental implants have been reported to achieve long-term success, they are not immune from complications associated with improper treatment planning, surgical and prosthetic execution, material failure, and maintenance[2].

Peri-implant diseases present in two forms – peri-implant mucositis and peri-implantitis. Both of these are characterized by an inflammatory reaction in the tissues surrounding an implant[3,4]. Peri-implant mucositis has been described as a disease in which the presence of inflammation is confined to the soft tissues surrounding a dental implant with no signs of loss of supporting bone following initial bone remodelling during healing. Peri-implantitis has been characterized by an inflammatory process around an implant, which includes both soft tissue inflammation and progressive loss of supporting bone beyond biological bone remodeling[2, 5].

Distinct differences in the incidence and prevalence of peri-implantitis have been reported in the literature[2]. Lack of a specific clinical and radiographic definition of peri-implantitis makes it difficult to determine the exact prevalence of the disease. The important concept is that this disease is prevalent and longer the implants have been in place, the number of implants affected increases[6]. According to the Consensus Report of the Sixth European Workshop on Periodontology, peri-implant mucositis occurs in about 80% of the patients and in 50% of implants, while peri-implantitis is observed in between 28% and 56% of the patients, and in 12–43% of the implants[4]. Koldsland et al evaluated the prevalence of peri-implantitis by applying different diagnostic thresholds and observed that its prevalence varied from approximately 11% to 47% of subjects depending on the threshold used and concluded that peri-implant inflammation was a frequent finding with and without peri-implant bone loss[7].

ETIOLOGY

The etiopathogenesis of peri-implantitis is complex and related to a variety of factors that affect the peri-implant environment[8].

The formation of a biofilm on the implant surface plays a significant role in the initiation and progression of peri-implant diseases and is essential for the development of infections around dental implants[2, 3, 9]. Peri-implantitis, like periodontitis, occurs primarily as a result of an overwhelming bacterial insult and subsequent host immune response. Animal and human studies have found that the bacterial species associated with periodontitis and peri-implantitis are similar, mainly Gram-negative anaerobes including Aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, Tannerella forsythia, Peptostreptococcus micros, Campylobacter rectus and Fusobacterium species. Moreover,

Staphylococcus aureus may also be an important pathogen in the initiation of peri-implantitis [2, 10].

Studies have shown that peri-implantitis and periodontitis lesions from human biopsies have many features in common. The connective tissue adjacent to the pocket epithelium is infiltrated by inflammatory cells, with B-lymphocytes and plasma cells being the most dominating cell types. Basically, similar markers are upregulated between periimplantitis and periodontitis, including proinflammatory cytokines such as interleukin-1 (IL-1), IL-6, IL-8, IL-12, and tumor necrosis factor- alpha (TNF-α).[2,11]

However, the rate of disease progression and the severity of inflammatory signs for peri-implantitis may differ from those of periodontitis. The clinical and radiographic signs of tissue destruction were more pronounced and the size of inflammatory cell infiltrate in the connective tissue was larger, approaching the crestal bone in peri-implantitis as compared to periodontitis. The increased susceptibility for bone loss around implants may be related to the absence of inserting collagen fibers into the implant as is the case with a tooth[2]. A recent comparison of periodontitis and peri-implantitis noted a "self-limiting" process existing in the tissues around natural teeth that resulted in a protective connective tissue capsule of the supracrestal gingival fibers of the tooth that separated the lesion from the alveolar bone. Such process did not occur in peri-implant tissues and the lesion extended to the bony crest, which was different than the periodontitis lesions. Also, spontaneous continuous progression of the disease occurs with additional bone loss. All implants appear to be susceptible to peri-implantitis. Hence, the primary objective for treating peri-implantitis is the elimination of the biofilm from the implant surface[2,12,13].

Risk Factors

A number of risk factors have been identified that may lead to the establishment and progression of peri-implantitis. These include the following:

1. **Previous Periodontal Disease**
   It has been indicated that although the implant survival rate may not be affected by the periodontal history, peri-implantitis was a more frequent finding in patients with a history of periodontitis[14, 15].
   Karoussis et al compared the failure, success, and complication rates of patients who lost their teeth due to periodontitis or other reasons. The group with a history of chronic periodontitis had a significantly higher incidence of peri-implantitis (28.6%) than the group with no history of periodontitis (5.8%) [16].

2. **Poor Plaque Control/Inability to Clean**
   Implant prosthesis design can obviate the patient's ability to mechanically clean the site with brushes, interdental brush, and floss. This can be related to implant positioning and meeting patient expectations for esthetics, phonetics, and function. Moreover, prosthesis design can also preclude clinical evaluation with probing and adequate home-care procedures[2, 17]. These concerns must be factored in the prosthetic decisions to facilitate daily oral hygiene. While the prosthesis suprastructure, if screw retained, can be removed to facilitate evaluation, the same cannot be said for patient home care. It is important for the dental clinicians to educate the patient in proper plaque control and to ensure the establishment of regular periodontal maintenance. This will help to assess the adequacy of plaque removal efforts and to intervene as early as possible if problems are detected[2].

3. **Residual Cement**
   A growing area of concern has been the incomplete removal of cement left in the subgingival space around dental implants[18]. After the cementation of crowns on implants, it is quite plausible for cement to be left behind because of implant positioning and the subsequent suprastructure design, which may hamper mechanical non-surgical therapy efforts to access the subgingival space[19]. Moreover, many of the commonly used cements are undetectable radiographically. How dental cement causes inflammation and disease may be related to its roughness which, unto itself, may cause inflammation; however, its surface topography may provide a positive environment for bacterial attachment[2].

4. **Smoking**
   It has been established that there is an increased risk for peri-implantitis in smokers, with odds ratios ranging from 3.6 to 4.6. Moreover, cohort studies and cross-sectional studies frequently have linked smoking to higher implant failures[2, 20, 21]. Klokkevold et al[20] reported that 78% of the implants in smokers had the diagnosis of peri-implantitis, while for non-smokers it was only 64%. More recently, a cross-sectional study demonstrated that smokers had an odds ratio of 3.8 of developing peri-implant mucositis and an odds ratio of 31.6 of developing peri-implantitis[22].

5. **Genetic Factors**
   Genetic variations have been cited as a risk factor for peri-implantitis. However, the association between IL-1 gene polymorphism and peri-implantitis remains to be determined since conflicting results exist. A systematic review with 27 relevant articles found no consensus among the studies reviewed. If certain cofactors are present, IL-1 polymorphism alone cannot be considered a risk factor for bone loss[2]. Another study on IL-1RN gene polymorphism concluded that it is associated with peri-implantitis and may represent a risk factor[23]. Future studies in this area are certainly needed to determine the role of genetic susceptibility and which genetic markers, if any, may provide a clue as to patient susceptibility[2].

6. **Diabetes**
   Current evidence does not allow a definitive conclusion that diabetic patients have a higher incidence of peri-implantitis. Diabetic control is an important factor when assessing the relationship. High blood glucose level can impact tissue repair and host defense mechanisms, as diabetic control affects neutrophil function. As a result, diabetes can disrupt collagen homeostasis in the extracellular matrix and is associated with neutrophil dysfunction and imbalance of immune system. Thus, the tissue repair ability and defensive mechanisms of

diabetic patients to the insult of dental plaque are impaired. Additional prospective cohort studies are needed to clarify the association between diabetes and peri-implantitis[2, 24].

7. **Occlusal Overload**
A positive association of occlusal overload and peri-implant marginal bone loss has been suggested. Differences in the magnitude, duration, direction, and frequency of the applied occlusal load and the tolerance threshold of the host are the underlying reasons for the conflicting observations obtained through the clinical studies on occlusal overload. Possible mechanisms of why occlusal overload can lead to peri-implantitis are conceivable. Implants are considered less tolerable to non-axial occlusal load compared to teeth because of a lack of a periodontal ligament. Finite element studies have suggested that the occlusal load is concentrated at the implant marginal bone. Bone remodels in response to the strain. Excessive stress can cause microfracture within bone and eventual bone loss[2, 25].

8. **Potential Emerging Risk Factors**
Research efforts have explored some additional areas that may impact the development and pathogenesis of peri-implantitis. These include rheumatoid arthritis with concomitant connective tissue disease, increased time of loading and alcohol consumption. Further study will determine the appropriateness of their inclusion[2].
It is imperative for clinicians to not only identify these risk factors but also to manage them to the best of their ability. This could include stabilising periodontal infections before implant therapy, providing ongoing maintenance care, delivering smoking cessation advice and also designing prosthesis that would allow easy oral hygiene practices[26].

DIAGNOSIS OF PERI-IMPLANTITIS
Peri-implant diagnostic procedures can serve several functions[3]:
1. Screening for peri-implant disease or for factors increasing the risk to develop an undesirable condition,
2. differential diagnosis of peri-implantitis and peri-implant mucositis,
3. treatment planning and
4. evaluation of therapy and monitoring

The early detection of the peri-implant diseases, namely peri-implant mucositis and peri-implantitis, is essential as the treatment of peri-implantitis is not predictable and at times, complex and difficult to perform. Peri-implantitis lesions are often asymptomatic and usually detected at routine recall appointments. The diagnosis of peri-implantitis should be based on the following considerations:

Probing, Bleeding, Suppuration
Initial probing of the implant should be done once the final restoration has been installed. This can be done with a traditional periodontal probe using light force (0.25N) because of the delicate and unique anatomy of the peri-implant mucosa[2]. Probing depth should be recorded, and defined as the depth of probe penetration from the base of the implant sulcus to the crest of the mucosa. Similar to assessing natural teeth,

the level of the crestal soft tissue can be measured using a fixed reference point on the restoration and should be noted as the clinical attachment level. A change in these parameters over time may be more important than the initial findings as implants may be placed more apically to achieve optimal esthetics, resulting in deeper soft tissue probing depths. It must also be remembered that probing may have to be done with the prosthesis removed as it may obviate probing along a parallel axis to the implant. Gentle probing resulting in bleeding suggests the presence of soft tissue inflammation. Increasing probing depth and bleeding are indicators for the need to perform an additional radiographic examination. The presence of suppuration/exudate indicates pathological changes and the necessity for further evaluation and treatment. In peri-implantitis, a bony defect develops around single or multiple implants and most often extends the full circumference of the implant[2, 4, 27, 28].

Radiographs
Periapical radiographs of the implant following placement and then following the prosthesis installation should function as the baseline by which all future radiographs are to be compared. These radiographs should be perpendicular to the implant body to show a clear demarcation between the threads of the implant[2, 3]. Other radiographs such as CBCT may be considered depending on the location of progressive attachment loss. CBCT images have been utilized to aid in evaluating the extent of facial, lingual, and proximal bony lesions around implants. In peri-implantitis, the radiographic appearance is often in the shape of a saucer or rounded beaker and the lesion most often extends the full circumference of the implant[2, 29].Froum & Rosen[30] proposed a classification for peri-implantitis based on the severity of the disease using a combination of bleeding on probing and/or suppuration, probing depth (PD), and extent of radiographic bone loss around the implant (Table I).

Table I: Classification of Peri-implantitis (Froum & Rosen, 2012[30])

EARLY	PD ≥ 4 mm (bleeding and/or suppuration on probing) Bone loss < 25% of the implant length
MODERATE	PD ≥ 6 mm (bleeding and/or suppuration on probing) Bone loss 25% to 50% of the implant length
ADVANCED	PD ≥ 8 mm (bleeding and/or suppuration on probing) Bone loss > 50% of the implant length

Mobility
While mobility of implants is found only in very advanced cases of bone loss primarily in situations where the integration has been completely lost, mobility of the restoration and/or abutment should be routinely checked as these can indicate loose or broken components. A loose implant-supported prosthesis may contribute to the accumulation of plaque, which may lead to the development of peri-implant mucositis and/or peri-implantitis, & as such, this should be corrected[2,4].

Secondary Diagnostics
Bacterial culturing, inflammatory markers, genetic diagnostics may be useful in diagnosis of peri-implant diseases[2].
Thus, it should be borne in mind that there is no single diagnostic tool that can, with certainty, establish a diagnosis of

peri-implantitis. Therefore, the clinician must use a combination of probing data over time, inflammatory status of the mucosa, bleeding on light probing, radiographic changes in bone levels over time, and possibly bacterial and/or PICF (peri-implant crevicular fluid) sample data to arrive at an accurate diagnosis of peri-implantitis[2, 30, 31].

It is important to understand that peri-implant lesions may develop after several years. Therefore, a function time exceeding 5 years for implants may be required to detect destructive peri-implantitis sites. Regular check-up visits and life-long supportive therapy is an absolute necessity for the implant patient[32].

MANAGEMENT OF PERI-IMPLANTITIS

Given that the presence of micro-organisms is a key factor for the development of peri-implantitis, etiological management must seek to reduce the microbial load, eliminate inflammation of the peri-implant mucosa and decontaminate the implant surface in order to preserve supporting bone and then, if possible, bring about regeneration of the lost bone[33].

The principal objectives of the treatment of peri-implantitis are to reduce bacterial colonization of the surface of the implant, mechanically eliminate the bacterial microbiota, and introduce an ecology capable of suppressing the subgingival anaerobic flora. Both surgical and nonsurgical techniques have been developed to this effect[34].

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 calculus 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 be given 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[35].

For management of peri-implantitis, both non-surgical as well as surgical approaches can be employed.

I)   Non-Surgical Approach
i)   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 non-metallic tip or resin/carbon fiber curettes, the presence of implant threads and/or implant surface roughness may compromise the access for cleaning[35, 36].

Karring et al[37] 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 resolution of peri-implantitis.

ii)   Anti-infective therapy/Chemical Decontamination
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[35].

Chemical decontamination of dental implant surfaces involves the localized use of citric acid, chlorhexidine, ethylene diamine tetraacetic acid (EDTA), hydrogen peroxide, tetracycline, minocycline, saline and saline soaked cotton pellet, or 35% phosphoric acid gel, in combination with mechanical debridement for eliminating hard and soft deposits. Comparisons of the decontaminating efficacy of these chemical agents have been made mainly by means of in vitro studies on different types of implant surface[6, 33].

Reviews made by various authors conclude that 40% citric acid with pH 1 for 30-60 seconds has proved the most effective agent for the reduction of bacterial growth on hydroxyapatite (HA) surfaces, although clinical application at a more acidic pH could affect the peri-implant tissues and if the time of application is prolonged this can affect the union between the HA and the implant body. Chlorhexidine has been seen to be ineffective on HA surfaces. Machined titanium decontaminates more effectively than other surface types, with topical applications of tetracycline as the antibiotic of choice[33, 38,39]. However, a review by Claffey et al[40], of a total of 43 experimental and clinical studies (13 of them performed on human subjects), which evaluated different decontamination protocols using sterile saline solution, chlorhexidine, citric acid and hydrogen peroxide, failed to show that any one method was more effective than the others. The use of 35% phosphoric acid gel for treating peri-implant mucositis would appear to achieve microbial reduction but further studies, both in vitro and in humans are needed to determine its efficacy for decontaminating implant surfaces[39].

Most in vivo studies use empirical combinations of chemical agents and mechanical procedures with or without systemic antibiotic treatment.

Renvert et al[41] demonstrated 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 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 beneficial in the treatment of peri-implant lesions, but the treatment may have to be repeated[35]. The study by Renvert et al[42]

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[35].

Systemic administration of antibiotics may be needed for the therapy of peri-implantitis. The recommended antibiotic treatments are amoxicillin, amoxicillin plus clavulanic acid, amoxicillin plus metronidazole, or erythromycin plus tetracycline, with duration of 7-10 days[34]. Lang et al[43] 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 and for this, the adjunctive use of systemic antibiotics may be considered[35].

iii) Air Powder Abrasive System

Air powder abrasive system (AP) features the use of an abrasive powder, generally sodium bicarbonate, and sodium hydrocarbonate, or amino acid glycine, propelled by a stream of compressed air to remove biofilm or extrinsic stains from teeth. This instrument applies a mix of water, air, and powder at pressures of 65 to 100 pounds per square inch (psi) and has been demonstrated in in vitro and in vivo studies to be effective in cleaning the previously contaminated implant surfaces[6].

Tastepe et al[44] analyzed 27 articles, including 19 in vitro studies, 3 in vivo studies, and 4 human studies that dealt with the efficacy of this approach in cleaning the implant surface as well as the clinical response to implants treated using this method. They concluded that the cleaning efficiency evaluated by the removal of bacterial endotoxin ranged from 84% to 98% and the removal of the bacteria biofilm was up to 100% in in vitro studies. This approach has not been shown to alter the physical structure of some implant surfaces. However it has been shown that particles of the powder can stay attached to the implant surface after cleaning. In addition, when this approach is used on machined surfaced implants, alterations of the surface topography can occur and large amounts of powder particles attaching to the implant surface have been seen in in vitro studies[6].

Froum et al[8] proposed a surgical protocol for detoxification of implant surfaces in humans that included AP. They reported significant bone fill and general clinical improvement up to 7.5 years after the use of AP 60 seconds followed by a solution of tetracycline application, followed by a second application of AP for 60 seconds and finally rinsing with 0.12% chlorhexidine for 30 seconds before bone grafting in 38 patients with 51 implants affected by peri-implantitis.

Thus, air powder abrasive can contribute to the detoxification of the implant surface and can improve the clinical outcomes when used in combination with surgical regenerative procedures. However, adverse effects like subcutaneous emphysema have been reported with the use of air abrasive around teeth and around implants. While this complication might not occur if the tip if the instrument is cautiously used at a 45∘ angle to the implant, this approach could not be routinely recommended based on the available literature[6].

iv) Laser decontamination

Laser therapy is another therapeutic option for decontaminating both implant surfaces and peri-implant tissues. Laser decontamination is based on its thermal effect, which denatures proteins and causes cellular necrosis.[33]

Diode, CO2, and erbium-doped yttrium, aluminum, and garnet (Er:YAG) lasers are suitable for implant irradiation because of their hemostatic properties, selective elimination of calculus and bactericidal effects, which achieve complete or almost complete elimination of bacteria from titanium surfaces, providing they are used within the appropriate parameters for each surface type[33]. Also, the degree of energy absorption by titanium is low, and there is no significant temperature increase of the implant body[45]. Whereas electron microscopy has revealed extensive melting of the titanium surface with neodymium-doped:yttrium, aluminum, and garnet (Nd:YAG) laser application. Er:YAG and CO2 lasers exhibit bactericidal effects on implant surfaces in vitro without this drawback. In contrast to diode and CO2 lasers, the ability of Er:YAG laser to effectively ablate calculus from titanium surfaces has been demonstrated[45, 46].

GaAlAs laser has been shown to be one of the safest as it does not alter implant surfaces, regardless of the strength at which it is applied. However, not all studies of the exclusive use of laser techniques have obtained complete surface decontamination[33]. In a literature review by Subramani, laser combined with chlorhexidine or saline solution was found to achieve greater percentage of re-osseointegration[33, 39].

v) Photodynamic Therapy (PDT)

This technique is based on the application of photosensitive dyes activated by a light with a specific wavelength to kill bacteria. It includes three basic elements: visible harmless light, nontoxic photosensitizer, and oxygen. The oxygen is transformed into ions and radicals that are highly reactive and kill the microorganisms. The main photosensitizers found in the literature are hematoporphyrin derivatives (620–650 nm), phenothiazine, like toluidine blue and methylene blue (620–700 nm), cyanine (600–805 nm), phytotherapic agents (550–700 nm), and hytalocyanines (660–700 nm) [6].

PDT appears to be more efficient for eliminating bacteria from implant surfaces than laser irradiation alone. A comparison between four groups (G1: without decontamination; G2: decontamination using chlorhexidine; G3: PDT= laser + methylene blue dye; G4: laser alone) with the use of GaAlAs laser (660nm, 30mW) there were significant differences between G1 and the other groups, and between Group 4 and Groups G2 and G3, The best results were achieved by G2 and G3, without statistically significant difference between these two groups[33,47].

An application of toluidine blue with soft laser irradiation has been shown to significantly reduce the presence of Aggregatibacter Actinomycetemcomitans, P gingivalis and P intermedia on different implant surfaces, reduce bleeding on probing and inflammation, but more long-term clinical studies are needed to confirm its effectiveness[33, 47].

Some factors can influence the PDT effectiveness of surface decontamination including light absorption by the bacteria, wavelength of the laser, time of laser exposure, area to be stained, and the organic matrix of the biofilm[6].

One negative aspect is that currently the dyes do not differentiate between bacteria and host cells; therefore, this could adversely affect the surrounding tissues. One possible advantage of PDT over conventional antibiotic therapy is that this is a topical treatment where only the affected sites requiring antimicrobial treatment receive the dye and illumination limiting the adverse effect seen with systemic antibiotics. Also, there is no evidence of resistance development in the target bacteria after PDT [6, 48].

II) Surgical Approach

When bone loss is advanced or persists despite the initial treatment provided, surgical debridement of the peri-implant soft tissues is required, due to the chronic infection, with decontamination of the implant surface and the application of bone regeneration techniques to restore the lost bone[34, 38]. The surgical treatment can be divided into resection procedures and regenerative techniques, depending on the morphology and type of bone defect[34].

i) Resection techniques

Resection techniques are used when there are moderate (< 3 mm) horizontal suprabony defects or vestibular dehiscences in a non-aesthetically compromised region. These procedures include ostectomy or osteoplasty, with the raising of an apical repositioning flap and implantoplasty[34, 38]. The objectives of resective surgery are to reduce pocket depth and secure adequate soft tissue morphology, in order to facilitate adequate hygiene and peri-implant health[34].

The resection technique comprises the following steps[34]: 1) removal of the supragingival bacterial plaque; 2) surgical access; 3) removal of granulation tissue and detoxification of the implant surface; 4) correction of bone architecture; 5) modification of implant surface roughness; 6) and implementation of plaque control.

Implantoplasty

When a titanium implant surface has been exposed to the oral cavity and contaminated with bacteria, implantoplasty may be indicated so as to completely flatten/smoothen the exposed part of the implant threads using rotary instruments[49]. Diamond stones with adequate cooling can be used to grind away plasma-spray coatings or threads on the implant surface, with final polishing accomplished using rubber disks[50].

This technique aims to reduce the roughness of the titanium surface to decrease plaque accumulation since it has been demonstrated that rough surfaces accumulate more plaque than smooth or moderately rough surfaces[51].

Romeo et al[52] compared resective surgery plus implantoplasty with resective surgery alone for the treatment of 17 patients with 35 implants with per-implantitis and a 3-year follow-up period. This study demonstrated that implantoplasty improves the survival rate (100% versus 77.6%) and prevented further significant marginal bone loss. This approach significantly improved probing depths (PD), clinical attachment levels (CAL), and bleeding (BOP) compared to resective surgery. However the marginal recession was increased in the implantoplasty group. Implantoplasty has also been combined

with regenerative surgery and sub-epithelial connective tissue graft (SCTG). Schwarz et al[53], published a 6-month follow up of 10 cases treated with implantoplasty, surface decontamination with saline soaked cotton pellets and xenograft plus collagen membrane, and SCTG and showed a significant reduction in PD, CAL, and soft tissue recession.

One of the major disadvantages of the implantoplasty technique is the increased postoperative recession of the marginal tissues and exposure of the abutment and implant surface which negatively affects the esthetics and increases food impaction. In most situations reattachment of bone to previously toxic implant surfaces is the desirable outcome. Therefore, smoothening of the exposed implant surface as monotherapy is not the optimal approach in many clinical situations[6].

ii) Regenerative Surgery

Regenerative surgery is used when the implant is decisive for prosthetic preservation, or when esthetic considerations are involved[34, 38].

Regenerative therapy requires prior decontamination of the implant surface. A wide array of bone grafting materials like autogenous bone, demineralised freeze dried allogenic bone, bovine inorganic bone and hydroxyapatite, in combination with resorbable or nonresorbable membranes, using the concept of guided bone regeneration (GBR) have been used successfully over the years for the treatment of peri-implantitis[34, 38].

Membranes are applied to stabilize the blood clot and to prevent growth of connective tissue and epithelium into the peri-implant bone defect during surgical therapy[54]. Various grafting materials have been combined with membranes to maintain the space created under the membrane and to serve as an osteoconductive scaffold to promote bone regeneration. Even though, this regenerative approach is preferable however, membrane exposure is a frequent post-surgical complication, which may result in bacterial penetration leading to infection[36, 54]. Satisfactory results may occasionally be obtained despite membrane exposure if plaque control is optimal. However, based upon observations in humans, immediate removal of exposed membranes used as a part of the surgical treatment of peri-implantitis is recommended to avoid impeding bone regeneration[36].

Leonhardt et al[55] evaluated the effect of systemic antimicrobial therapy (amoxicillin and metronidazole) together with surgical (open flap) procedure and in conjunction with mechanical debridement of the implant surface for decontamination. The treatment was successful in 58% of the implants treated during follow-up period of five years.

Heitz-Mayfield et al[56] showed that an antimicrobial protocol with surgical flap access was able to stop the progression of peri-implantitis in 90 percent of cases for the short term (one year), but bleeding on probing persisted in nearly 50 percent of those cases.

Schwarz et al[57] evaluated and compared the efficacy of two bone regenerative procedures for the treatment of moderate intrabony peri-implantitis lesions that included a greater than 6 mm probing depth and an intrabony component of 3 mm as detected on radiographs. The defects were randomly treated either with a surgical debridement and filled with

nanocrystalline hydroxyapatite, or surgical debridement and filled with bovine-derived xenograft (Bio-Oss®) combined with a bioresorbable porcine-derived collagen membrane (Bio-Gide®). After two years, the study showed that the combination of bovine bone mineral and the collagen membrane seemed to yield greater improvements in clinical parameters. Schwarz et al[58] found regenerative surgical treatment to be effective over two years, resulting in cessation of peri-implant bone loss and a reduction of bleeding on probing from 80 percent to 34 percent. Froum et al[8] also demonstrated the effectiveness of surgical regeneration where peri-implantitis was arrested with reduced bleeding on probing over three to seven years.

Unfortunately, not all peri-implantitis lesions are favourable to regeneration. For implants with thin facial and lingual walls, peri-implantitis typically does not produce a crater-form defect with four walls. In some of these cases, the defect will present as a complete loss of the surrounding bony walls leaving regeneration as an unpredictable treatment choice[59].

Aljateeli et al[60] proposed a decision tree based upon the defect morphology. If the defect had sufficient walls (two or greater), regeneration was attempted, but if there was zero or one wall, an apically positioned flap (APF) was suggested with implantoplasty.

Charalampakis et al[61] evaluated the longevity and incidence of relapse of multiple different treatments on peri-implantitis lesions, in which relapse was observed in over half of the cases. Smoking and early disease development, were associated with higher rates of relapse and surgical therapy with lower rates of relapse. This means peri-implantitis is not just hard to treat, but treated cases must be watched closely as relapse is common.

iii) 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 insufficient 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[35].

Cumulative Interceptive Supportive Therapy (CIST)[3]

This is a method for implant maintenance and therapy of peri-implantitis. The principle of this method is to detect peri-implant infections as early as possible and to intercept the problems with appropriate therapy. The basis for this system is a regular recall of the implant patient and the repeated assessment of the following key parameters around each implant: the presence of plaque, the bleeding tendency of the peri-implant tissues, suppuration, the presence of peri-implant pockets and radiological evidence of bone loss. Optimally an implant should yield negative results for all of these

parameters. In this case no therapy is needed and one may consider increasing the length of the recall interval.

If plaque and/or an increased tendency of the peri-implant tissues to bleed is detected, then the implants are mechanically cleaned using a rubber cup and polishing paste. Oral hygiene practices should be checked, and the proper plaque control technique should be instructed and reinforced (A).

In the presence of pus, or if first signs of peri-implant tissue destruction are detected (pockets in the range of 4 to 5 mm and slight bone loss) regimen A should be combined with the application of a local antiseptic (B).

If the peri-implant sulcus allows more than 5 mm of penetration of a periodontal probe then a radiograph is taken. If there is clear evidence of bone loss, then a microbiological sample is taken. Should there be evidence of an anaerobic flora, the patient is given treatments A and B, and, in addition, is placed on systemic antimicrobial therapy (C)

If the bone destruction has advanced considerably, surgical intervention to correct the tissue morphology or to apply guided bone regeneration techniques may be necessary (D). Such treatment would, however, only be given in addition to the other measures (A, B and C).

The goal of this cumulative treatment approach is to intercept peri-implant tissue destruction as early as possible and to avoid explantation (E) due to loss of osseointegration.

CONCLUSION

Dental implants have become a superior treatment option for replacement of missing teeth. With the increasing number of implants being placed, peri-implantitis has become much more prevalent. It has been suggested that, the earlier the diagnosis and intervention of peri-implantitis, the better the treatment outcome. The management of peri-implantitis involves various treatment modalities, including non-surgical and surgical techniques with different protocols. However, long-term controlled studies are needed to validate which treatment modality may be optimal, given the different clinical scenarios. Routine monitoring of dental implants as a part of a comprehensive periodontal evaluation and maintenance is essential.

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