REVIEW ARTICLE

Peri-implantitis 360^0

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ABSTRACT

Peri-implant diseases are inflammatory conditions that affect the tissues around implant fixtures. Peri-implant mucositis is seen to respond well to non-surgical treatment because the inflammatory reaction is confined to the soft tissues. Peri-implantitis that is considered a late complication, has less predictable treatment outcome because of the bone resorption that takes place around a functioning implant. The purpose of this review is to understand the etiopathogenesis, diagnosis and treatment of peri-implant diseases, as well as the prevention and maintenance of peri-implant infection in osseointegrated implants, which will be useful in order to achieve long-term success of dental implants.

KEYWORDS: Dental implants, Osseointegration, Peri-implantitis

INTRODUCTION

The introduction of Dental Implants has created a paradigm shift in the orodental rehabilitation of partially and fully edentulous patients.The long-term success of dental implants is dependent upon maintenance program with regular follow-up. Even though dental implants have demonstrated significant success in a majority of cases, they too are not immune from complications associated with improper treatment planning, surgical and prosthetic execution and maintenance.

PERI-IMPLANT DISEASE AND IMPLANT FAILURE

The term peri-implant disease refers to the pathological inflammatory changes that take place in the tissue surrounding a functional implant.[1] Peri-implant disease includes two entities: Peri-implant mucositis that corresponds to gingivitis and Peri-implantitis that corresponds to periodontitis. 'Peri-implant mucositis' is defined as a reversible inflammatory reaction in the soft tissue surrounding a functioning implant and 'Peri-implantitis' is defined as an inflammatory reaction with loss of supporting bone in the tissues surrounding a functioning implant.[2] The term Peri-implantitis first appeared in the literature in 1987 in a study by Mombelli et al. describing peri-implantitis as a site-specific infection which yields many features in common with chronic periodontitis.[3] 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 seen in the range of 28%-56% of the patients, and 12–43% of the implants.[4] A dental implant is considered to be a failure if it is shows peri-implant bone loss of greater than 1.0 mm in the first year and greater than 0.2 mm thereafter.[5]

Implant failures may be described as early or late. An early failure follows shortly after placement and osseointegration are never achieved which is due to premature loading, surgical trauma, or impaired healing response. A late failure, occurs as a result of peri-implantitis and/or excessive mechanical stress in a successfully osseointegrated implant.[6]

CLINICAL FEATURES

Signs that determine the presence of peri-implant mucositis include bleeding on probing and/or suppuration, with a probing depths of ≥4 mm and no evidence of radiographic bone loss, whereas in peri-implantitis radiological evidence for vertical destruction of the crestal bone is observed. The defect is usually saucer shaped and there is osseointegration of the apical part of the fixture. A radiographic observation of bone resorption mesial and distal to the implant of ≥3 threads will confirm a diagnosis of peri-implantitis.[7]

ETIOPATHOGENESIS

Etiology of Peri-implantitis can be attributed to 3 main factors which include bacterial infections, biomechanical factors and patient-related factors:

Bacterial flora associated with periodontitis and peri-implantitis, are found to be similar, consisting of gram-negative anaerobic bacteria which includes *Porphyromonas gingivalis, Prevotella intermedia, Aggregatibacter Actinomycetemcomitans, Tennerella forsythia, Treponema denticola, Prevotella nigrescens, Peptostreptococcus micros, and Fusobacterium nucleatum*.[8] The implants in partially edentulous patients appear to be at a greater risk of peri-implantitis than the implants in completely edentulous patients because of the qualitative differences in the microflora surrounding implants and teeth in partially edentulous patients. However, there is a marked quantitative decrease in the

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number of periodontal pathogens around the implants in completely edentulous patients. It is possible that in a partially edentulous patient, the natural teeth may serve as reservoirs for periodontal pathogens to colonize the implants in the same mouth.[9]

Biomechanical factors include excessive mechanical stress caused by occlusal overload due to malocclusion, para-functional habits i.e. bruxism, prosthetic related factors include improper crown-to-implant length, poorly positioned implant, crowns, poor alignment of components. Patient-related factors include systemic diseases e.g. diabetes mellitus, osteoporosis, etc. Social factors such as poor oral hygiene/maintenance, smoking and drug abuse.[10]

The formation of a biofilm on the implant surface plays a significant role in the initiation and progression of peri-implant diseases.[1] Peri-implantitis, like periodontitis, occurs primarily as a result of change in the microflora and an altered host immune response. The connective tissue adjacent to the pocket epithelium is infiltrated by inflammatory cells, mainly B-lymphocytes and plasma cells. Markers that are upregulated in peri-implantitis and periodontitis, including pro-inflammatory cytokines such as IL-1, IL-6, IL-8, IL-12, and tumor necrosis factor alpha (TNFα).[11, 12] Although sharing similarities with periodontitis in both the bacterial initiators and key immune components to those insults, the rate of disease progression and the severity of inflammatory signs for peri-implantitis may be different. The clinical and radiographic signs of tissue destruction are more pronounced and the size of inflammatory cell infiltrate in the connective tissue is 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 in the case of a tooth.[13]

DIAGNOSIS

The early detection of these two diseases, peri-implant mucositis and peri-implantitis, is essential. While both are inflammatory lesions around a dental implant, the latter, peri-implantitis, includes loss of bone. While probing a dental implant can certainly aid in detecting bleeding and determine changes in probing depth over time, it may not be able to establish bone loss without the use of periapical radiographs to establish the extent and pattern of bone loss. Moreover, it should be recognized that not all peri-implantitis lesions may be detectable or verified with radiographs. Unlike periodontitis, many peri-implantitis lesions can occur on the facial and lingual aspects of dental implants and may, therefore, be "masked" with routine periapical dental radiographs.[14]

Diagnostic considerations for the early detection of peri-implantitis are as follows:

Clinical assessment: A visual assessment alone will reveal an inflamed gingival cuff. Probing the peri-implant sulcus allows the assessment of peri-implant probing depth, bleeding on probing and exudation/ suppuration from the peri-implant space. Probing is a valuable diagnostic tool and may be undertaken circumferentially around an implant fixture. A plastic (TPS or WHO 621) probe has greater flexibility than a metal counterpart, may cause less damage to the implant surface, and is potentially more accurate as it can adapt more readily to the abutment-suprastructure junction. A probing force of around 0.15–0.2N is recommended.[15] Bleeding following gentle probing is the characteristic sign for peri-implant mucositis and suppuration following probing is indicative of peri-implantitis.[16] The presence of bleeding on gentle probing (BOP) indicates mucosal inflammation and is a valuable diagnostic tool, which is seen in 91% of peri-implantitis sites.[17] Bleeding on probing also has a high prognostic value, since the absence of BOP is strongly indicative of peri-implant tissue stability and thus essential for monitoring peri-implant health. 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.[14]

Mobility within a year of placing an implant and/or its suprastructure is more likely to be a sign of lack of osseointegration, rather than an early sign of peri-implantitis. The only reliable method for assessing mobility (other than for a single tooth implant) is to dismantle the suprastructure so that the implant fixture(s) can be assessed independently. Late mobility of a successfully integrated implant, however, may be a sign of advanced or progressing peri-implantitis.[16]

Radiographic assessment: 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. 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.[14]

CLASSIFICATION OF PERI-IMPLANTITIS

Froum & Rosen, 2012[18] proposed a classification for peri-implantitis based on the severity of the disease which includes bleeding on probing and/or suppuration, probing depth (PD), and extent of radiographic bone loss around the implant. (Table 1)

Bacterial culturing, inflammatory markers, genetic diagnostics may be useful in the diagnosis of peri-implant diseases. No single diagnostic tool can, with certainty, establish a diagnosis of peri-implantitis. Therefore, 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 is required to arrive at an accurate diagnosis of peri-implantitis.[18] 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 supportive therapy is an absolute necessity for the implant patient.[19]

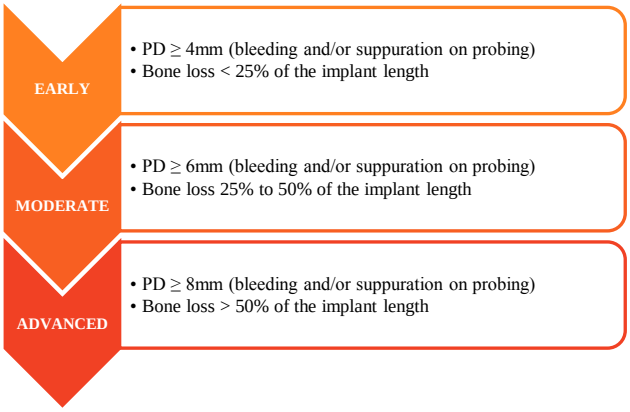

Table 1: Classification of Peri-implantitis (Froum & Rosen, 2012).

MANAGEMENT OF PERI-IMPLANTITIS

The principal objectives of the treatment of peri-implantitis are to reduce bacterial colonization on the surface of the implant, and introduce an ecology capable of suppressing the subgingival anaerobic flora. Both surgical and nonsurgical techniques have been developed to this effect.[20]

Nonsurgical approach

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 hours for 15 days; along with oral hygiene instructions. The 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.[21]

Local Debridement: The implant should be cleaned using plastic scaling instruments, floss, interdental brushes, and polishing should be done with a rubber cup and paste. 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.[22]

Karring et al.[23] demonstrated that sub-mucosal debridement alone, accomplished by using either an ultrasonic device (Vector® system) or carbon fiber curettes, is not sufficient for the decontamination of the implant surfaces with peri-implant pockets ≥5 mm and exposed implant threads. The same results were obtained by Renvert et al.[24] and Persson et al.[25] This suggest that mechanical or ultrasonic debridement alone may not be an adequate modality for the resolution of peri-implantitis.

***Anti-infective therapy/Chemical Decontamination*:** Chemical decontamination of dental implant surfaces involves the use of citric acid, chlorhexidine, ethylene diamine tetra acetic 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 based on in vitro studies on different types of implant surface.[26]

Several authors have concluded that 40% citric acid with pH 1 for 30-60 seconds has proven to be 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.[26-28]

Patients suffering from localized peri-implant problems in the absence of other infections may be treated using local drug-delivery devices. Local application of antibiotics by the insertion of tetracycline fibers for 10 days[29] 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.[30] The study by Renvert et al.[31] demonstrated that the 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 combination of an antiseptic (chlorhexidine) and mechanical debridement. The improvements in peri-implant probing depths obtained by the adjunctive use of minocycline can be maintained for upto 12 months. Büchter et al.[32] examined the benefits of adding 8.5% doxycycline to mechanical scaling – improvements being observed in pocket depth and bleeding in the group in which the antibiotic was added.

Most in vivo studies use empirical combinations of chemical agents and mechanical procedures with or without systemic antibiotic treatment. If peri-implantitis is associated with persisting periodontal disease, then both conditions need to be treated with adjunctive systemic antibiotics. The recommended antibiotic

treatments are amoxicillin, amoxicillin plus clavulanic acid, amoxicillin plus metronidazole, or erythromycin plus tetracycline, with duration of 7-10 days.[20] Lang et al.[29] suggested systemic ornidazole 500 mg bd for 10 days or metronidazole 250 mg td for 10 days or a combination of metronidazole 500 mg and amoxicillin 375 mg once daily for 10 days.

Air Powder Abrasive System: Air powder abrasive system (AP) includes 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 in-vitro and in-vivo studies have demonstrated this to be effective in cleaning the previously contaminated implant surfaces.

Tastepe et al.[33] 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 and 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 does not 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 vitro studies have demonstrated that when this approach is used on machined surfaced implants, alterations of the surface topography can occur with large amounts of powder particles attaching to the implant surface. 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.

Laser decontamination: Laser therapy is another therapeutic option for decontaminating both implant surfaces and peri-implant tissues. Laser decontamination based on its thermal effect denatures proteins and causes cellular necrosis. Diode, CO_2, 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, provided they are used within the appropriate parameters for each surface type.[34]

Photodynamic Therapy (PDT): In this technique, photosensitive dyes activated by a light with a specific wavelength is used to kill bacteria. PDT appears to be more efficient for eliminating bacteria from implant surfaces than laser irradiation alone. An application of toluidine blue with soft laser irradiation on different implant surfaces, has been shown to significantly reduce the presence of Aggregatibacter Actinomycetemcomitans, Porphyromonas gingivalis and Prevotella intermedia, reduce bleeding on probing and inflammation, but more long-term clinical studies are needed to confirm its effectiveness.[34, 35]

Surgical Approach

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

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 an apically repositioned flap and implantoplasty. 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. The resection technique comprises the following steps: 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.[20, 27]

Implantoplasty: Implantoplasty may be carried out to flatten/smoothen the exposed part of the implant threads using rotary instruments.[36] 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.[37] Diamond stones with adequate cooling can be used to smoothen threads on the implant surface, with final polishing accomplished using rubber disks.[38]

Romeo et al.[39] compared resective surgery plus implantoplasty with resective surgery alone for the treatment of 17 patients with 35 implants with peri-implantitis and a 3-year follow-up period. This study demonstrated that implantoplasty improved 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. 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.

Regenerative Surgery: Regenerative surgery is used when the implant is decisive for prosthetic preservation, or when esthetics is a concern. Regenerative therapy requires prior decontamination of the implant surface. A

wide array of bone grafting materials like autogenous bone, demineralized freeze dried allogenic bone, bovine inorganic bone and hydroxyapatite, in combination with resorbable or non-resorbable membranes, using the concept of guided bone regeneration (GBR) have been used successfully over the years for the treatment of periimplantitis.[20, 36]

Membranes are applied to stabilize the blood clot and to prevent the growth of connective tissue and epithelium into the peri-implant bone defect during surgical therapy. Various grafting materials along with membranes have been used for this purpose and to serve as an osteoconductive scaffold to promote bone regeneration. Even though, this regenerative approach is preferable, membrane exposure is a frequent post-surgical complication, which may result in bacterial penetration leading to infection.[40]

Schwarz et al.[41] found that a regenerative surgical treatment can result in cessation of peri-implant bone loss and a reduction in bleeding on probing from 80 percent to 34 percent, over a period of 2 years. Froum et al.[42] also demonstrated the effectiveness of surgical regeneration where peri-implantitis was arrested with reduced bleeding on probing over three to seven years.

Explantation: If there is advanced bone loss and 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 cause 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.[43]

PREVENTION AND MAINTENANCE

Following certain steps at the time of placement and restoration can improve the long-term prognosis of implant fixtures. Patient motivation and oral hygiene are paramount. Periodontal health should be achieved prior to proceeding with implant therapy. Proper treatment planning prior to implant surgery is necessary by selecting the number, diameter and location of the implants depending upon the bone type & type of prosthesis. Restorations should be cleanable with well-fitting margins. In addition, as much of the mucosal tissue as possible should be preserved in its original position.

Platform switching (the concept introduced in literature by Lazzara and Porter),[44] limits the circumferential bone loss around dental implants by using smaller-diameter abutment on a larger-diameter implant collar; this connection shifts the perimeter of the IAJ (Implant Abutment Junction) inward toward the central axis of the implant. It also provides increased biomechanical support and improved esthetics.

A maintenance program should be undertaken after successful implant therapy. This should include regular recalls to provide optimal disease prevention. Baseline data of probing pocket depths, mucosal margins position, radiographic crestal bone levels should be established, followed by repeated assessment of the presence of plaque, bleeding tendency of the peri-implant tissues, suppuration, presence of peri-implant pockets and radiological evidence of bone loss.

Nevertheless, a systematic approach for monitoring tissues around implants in the prevention and treatment of peri-implant disease has been recommended by Mombelli and Lang.[1] This systematic protocol, referred to as Cumulative Interceptive Supportive Therapy (CIST), contains four cumulative treatment modalities (A-D). (Table 2) Each step of the procedures is used in a sequential manner with increasing antibacterial intervention, combined with surgical resective/ regenerative treatment (A+B+C+D). The principle of this method is to detect peri-implant infections as early as possible and to intercept the problems with appropriate therapy.

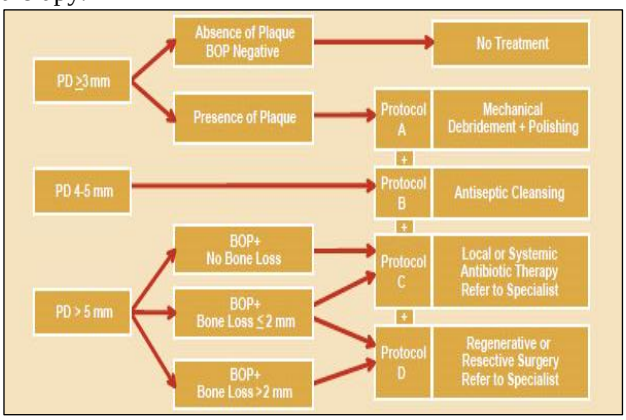

Table 2: Cumulative Interceptive Supportive Therapy (CIST) by Mombelli and Lang.

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.

CONCLUSION

With the increase in the acceptance of dental implants as a treatment modality for patients with missing teeth, peri-implant diseases are likely to become more prevalent. Hence, routine monitoring of dental implants as a part of a comprehensive periodontal evaluation and maintenance is essential.Early diagnosis is vital for better management of the disease and is useful for a favorable prognosis. 'Prevention' of peri-implant disease is the key to a successful Implant. In conclusion, *"Peri-implantitis is not a disease you would want to treat…but rather one you should prevent."*

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