

TREATMENT MODALITIES FOR PERI- IMPLANTITIS: A REVIEW OF LITERATURE

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Abstract: *Peri-implantitis is an infectious disease which causes an inflammatory process in soft tissues followed by bone loss around an osseointegrated implant. Its etiology depends on the status of the tissue surrounding the implant, implant design, degree of roughness, external morphology, and excessive mechanical load in presence of the most important factor, bacteria such as spirochetes and mobile forms of Gram-negative anaerobes. Diagnosis is made by assessing the changes of color in the gingiva, bleeding and probing depth of peri-implant pockets, suppuration, X-ray, and gradual loss of bone height around the tooth. The management of implant infection should be focused on the control of infection, the detoxification of the implant surface, and regeneration of the alveolar bone. This review of literature gives an overview of aetiopathogenesis, clinical features, and diagnosis of periimplantitis along with various treatment options in its management.*

Key words : Peri-implant disease, Peri-implant infection.

I. Introduction

Dental implants have in the recent years revolutionized prosthetic rehabilitation of edentulous and partially edentulous arches in patients all over the world primarily due to introduction of advanced imaging techniques and improvements in the designs which makes not only the diagnosis and treatment planning but also its execution easier than ever before. But despite high success rates, Implants are still subject to failures mainly due to biological complications which occur when bacterial plaque accumulates around an implant, causing inflammatory changes in the tissues surrounding it [1,2]. Peri-implantitis is hence an inflammatory process which results in loss of supporting bone around an Osseointegrated implant in function.

II. Definition

American Academy of Periodontology and the European Federation of Periodontology outlined Peri-Implantitis as “a plaque-associated pathological condition occurring in tissues around Dental Implants, characterized by inflammation in the peri-implant mucosa and resulting progressive loss of supporting bone” [3,4]. If this inflammation is restricted to the soft tissues, the condition is termed as peri-implant mucositis however once it spreads deeper to the underlying alveolar bone, it is known as peri-implantitis [5,6]

Radiographically, peri-implantitis has been defined as a clear radiographic threshold of more than 2 mm of marginal bone loss beyond biological peri implant bone remodelling along with presence of bleeding on probing (BOP), and/or suppuration [7]. Dental implant failure is often related to failure in osseointegration. A dental implant is considered to be a failure if it is lost, mobile, or shows peri-implant bone loss of greater than 1.0 mm in the first year and greater than 0.2 mm a year after [8].

III. Etiopathogenesis

Gram-negative anaerobes, like *Prevotella intermedia*, *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, *Bacterioides forsythus*, *Treponema denticola*, *Prevotella nigrescens*, *Peptostreptococcus micros*, and *Fusobacterium nucleatum* have been found to be the main causative organisms of this infection [9, 10, 11]. Excessive mechanical stress due to inadequate distribution of the chewing pressure on the tissues surrounding the implant, loosening of the artificial supports, infection of the surrounding tissues, and consequent inflammatory processes, poor design of the implant, and corrosion of a non-noble metal structure connected to a titanium implant are considered as the most important factors in the onset and development of peri-implantitis [12]. Other aggravating factors include diabetes mellitus, osteoporosis, smoking, parafunctional habits, longterm treatment with corticosteroids, radiation, and chemotherapy. The healthy peri-implant tissue plays a vital role and acts as a biological barrier to some of the agents that cause peri-implant disease, and if that barrier is destroyed, bacterial contamination spreads directly to the bone, leading to its rapid destruction [8].

IV. Prevalence

The overall frequency of peri-implantitis was reported to be 5% to 8% for selected implant systems [13] but with an increasing number of implants being placed annually, its prevalence is expected to increase proportionally. Peri-implantitis is predicted to affect 63.4% of all patients and 30.7% of all functional Implants [14,15]. Without regular maintenance program and reduced oral hygiene awareness, after a 5-year period of loading, 25% patients were found to have

periimplantitis in a recent study. Tissue-level implants had lower values of periimplantitis prevalence and crestal bone loss [16].

V. Diagnosis

The peri-implantitis has to be diagnosed clinically and radiographically from peri-implant mucositis. The diagnostic parameters used for assessing peri-implantitis include clinical indices, peri-implant probing using a rigid plastic probe, bleeding on probing (BOP), increased probing depth, suppuration, mobility, peri-implant radiography, and microbiology.

Clinical features of peri-implantitis are bleeding or suppuration on probing, increased peri-implant probing depth, mucosal recession, possible swelling or hyperplasia of the peri-implant tissues. Pain is unusual, but if present, is usually associated with an acute infection [17].

Radiographic features include marginal bone loss with vertical destruction of the crestal bone associated with the formation of a periimplant pocket. The defect is usually saucer shaped with osseointegration of the apical part of the fixture. The prevalence of periimplant pathology has been shown to be positively correlated to the duration for which the implant has been in function [18].

Peri-implantitis progresses faster around rough implants when compared with machined-surface implants and there is a variation in progression rate among the various micro-structures [19]. There are remarkable differences in the size of the residual inflammatory infiltrate and in the distance of the infiltrate to the bone among implants with similar surface roughness but different microstructural design. Albouny and co-workers in their study found that, the least residual inflammatory infiltrate which was also located furthest from the bone was observed around implants with a machined surface [20].

VI. Management of Peri-implantitis lesions

Maintenance and treatment of peri-implantitis must be based on a strong evidence-based approach as its aetiopathogenesis has still not been completely deciphered making its treatment planning slightly complicated [21]. If an implant surface is exposed to the oral cavity, it becomes immediately covered by the salivary pellicle and is colonized by oral microorganisms, forming a microbial biofilm. This microflora plays a crucial role in the success or the failure of that dental implant. Therapeutic strategies developed over the years for managing periimplantitis have been modelled largely on the evidence available for treating periodontitis where surface debridement has always been considered as the basic protocol. However, the screw-shaped design of the implants, combined with various surface modifications of titanium facilitates greater chances of plaque accumulation along with decreased chances of removal by regular oral hygiene measures, resulting in bacterial biofilm formation around them. Similarly,

mechanical debridement of such surfaces may also have a limited effect and can result in the inadequate removal of all adhering microorganisms. Therefore, adjunctive peri-implant therapies, such as antibiotics, antiseptics, ultrasonic and laser treatments, have been proposed to improve the non-surgical treatment options of peri-implantitis. Regenerative procedures using bone graft substitutes in combination with a membrane have been proposed to treat bone defects in advanced cases [22, 23].

Local debridement:

The presence of implant threads and/or surface roughness has been found to compromise the access for proper surface debridement due to which ultrasonic scalers with a non-metallic tip or resin/carbon fiber cures have been suggested for this purpose by Berglundh and coworkers [24]. The implants have thus been recommended to be cleaned by instruments softer than titanium, such as polishing with a rubber cup and paste, floss, interdental brushes, or plastic scaling instruments. These have been shown not to roughen the implant surface unlike metal and ultrasonic scalers [25]. However, in the case of cleaning of surfaces of implants with peri-implant pockets ≥ 5 mm with exposed implant threads, Karring et al suggested that sub-mucosal debridement accomplished by utilizing either an ultrasonic device or carbon fiber cures, is not sufficient to ensure complete decontamination precluding the use of this step alone for treating peri-implantitis [26].

Antibiotic therapy:

Schwarz et al found statistically significant improvements in bleeding on probing, peri-implant probing pocket depth, and clinical attachment level at 6 months compared with baseline peri-implant infection when they combined mechanical debridement with plastic cures and antiseptic (0.2% chlorhexidine) therapy [27] whereas Renvert et al showed that the addition of antiseptic therapy to mechanical debridement does not provide adjunctive benefits in shallow periimplant lesions where the mean probing pocket depth was <4 mm [28]. Both studies recommended addition of antiseptic therapy to mechanical debridement only in deep peri-implant lesions with mean pocket probing depth >5 mm to provide additional clinical improvements. Local application of antibiotics by the insertion of tetracycline fibers for 10 days has been recommended by Lang et al to provide a sustained high dose of the antimicrobial agent precisely into the affected site for several days [29]. Minocycline microspheres placed intra sulcular has also been assessed by Renvert et al who demonstrated that the adjunctive benefits derived from using it in combination with mechanical debridement tend to be greater than those achieved when an antiseptic (chlorhexidine) is used in its place. This study also proved that improvements in peri-implant probing depths obtained by the adjunctive use of

minocycline can be maintained during a short-term period of 12 months and the exhibited bone loss was not more than three implant threads [30].

Local antibiotic delivery in addition to mechanical debridement and irrigation with an antimicrobial agent was considered an effective option for treating peri implantitis lesions by Mombelli and co-workers [31]. Development of bacterial resistance was also deemed unlikely even in the event of repeated applications [32].

Although inadequate clinical trials have undervalued the use of systemic administration of antibiotics for treating peri-implantitis, in cases where it is associated with persisting periodontal disease, when generalized infective foci are detected in the oral cavity along with signs of systemic involvement or immune compromise, adjunctive use of systemic antibiotics has been suggested. Lang et al recommended culture sensitivity and following antibiotics to be administered systemically: 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 [29]. Before antibiotics administration, mechanical and antiseptic protocols have to be followed meticulously and studies have shown that shallow periimplant infection may be successfully controlled using antibiotics [33]. The drug to be administered is selected according to the composition of the subgingival microbial component, oral distribution patterns of potential pathogens are also important in deciding whether an antimicrobial agent should be administered locally or systemically.

Implant surface decontamination:

Implant surface decontamination using different techniques like airpowder abrasive followed by citric acid application, air-powder abrasive alone, gauze soaked in saline followed by citric acid application, and gauze soaked in 0.1% chlorhexidine and saline alternately evaluated for clinical parameters, radiography (including quantitative digital subtraction radiography), histology, and stereology failed to reveal any significant differences between any of the methods used [34]. Nevertheless, a combined mechanical and chemical removal of biofilm has been recommended by most experts [35]. Photodynamic therapy has also been recommended for reducing microorganisms in peri-implantitis using 2% chlorhexidine or 3% hydrogen peroxide as adjunctive topical antiseptics [36].

Laser Dis-infection:

Er:YAG laser used to treat peri-implantitis showed lower counts of *F. nucleatum* 1 month after therapy and when used along with combination of mechanical debridement/chlorhexidine was found to be equally efficacious in significantly improving peri-implant probing pocket depth and clinical attachment level at 6 months after therapy. Er:YAG laser when used as monotherapy provided a

significantly higher reduction of bleeding on probing compared with the adjunctive application of chlorhexidine [37]. In another study, the efficacy of the Er:YAG laser appeared to be limited to a 6-month period, particularly for advanced peri-implantitis lesions. It was further suggested that treatment with the Er:YAG laser only may not be effective for achieving a stable therapy of peri-implantitis and that additional therapeutic measures, such as supplementary use of the Er:YAG laser and/or subsequent osseous regenerative procedures, might be required [38].

Surgical technique:

Surgical techniques for treating peri-implantitis are used depending on multiple factors including patient general health, oral hygiene, and type of bony defects, implant surface, postoperative maintenance program, and other factors. For supra crestal bone defects (horizontal bone loss) with exposed threads in aesthetically non-demanding areas, non-augmentative surgical modalities involving reduction or elimination of pathological peri implant pockets, apical positioning of a mucosal flap, recontouring bone with or without implantoplasty are indicated based on patient needs and satisfaction. In case of modifications of implant surfaces, the rough design should be removed and polished taking care of the remaining titanium particles [39].

Peri-implantitis treatment using surgical intervention has been considered slightly tricky over the years due to increased chances of bone loss occurring post surgically due to which it is generally confined to implants placed in non-aesthetic sites [40]. Many studies have proved that clinical parameters like pocket depth (PD) and Bleeding on probing (BOP) improved after access apical surgery only [41, 42]. Surgical flap raised for debridement and decontamination of the affected implant followed by membrane-covered autogenous bone grafting showed significantly larger amounts of bone regeneration and re osseo-integration than those treated with other surgical procedures in a study by Schou et al [43]. However, exposure of porous e-PTFE membranes resulting in bacterial penetration and subsequent reinfection was found to be the most frequent complication after such procedures [44]

Schwarz et al in a clinical study proved that application of a combination of natural bone mineral and collagen membrane led to greater improvements in clinical parameters and was associated with a more predictable and enhanced healing outcome. They also demonstrated that nano crystalline hydroxyapatite bone graft and guided bone regeneration provided clinically significant improvements in clinical parameters following 6 months of non-submerged healing treatment. These modalities were also efficacious in providing clinically significant reductions of pocket-probing depth and gains in clinical attachment level albeit lesser than the earlier group [45]. Resective surgical procedures

coupled with implantoplasty were found to have a positive influence on the survival rates of roughsurfaced implants affected by peri-implantitis. They were also found to influence peri-implant clinical parameters like pocket-probing depth, suppuration, and sulcus bleeding. In a classical study by Romeo et al, Implantoplasty (diamond/Arkansas burs + silicone polishers) as an adjunct to open flap debridement with bone recontouring and apical flap repositioning resulted in better BOP and PD scores, but higher mean mucosal recessions compared with the control group, where persistent active signs of peri implant inflammation recurred in all patients after 24 months [46].

Resective surgery in combination with implantoplasty has also been associated with superior clinical and radiographic treatment outcomes compared with resective treatment alone. In particular, implantoplasty, performed with diamond or carbide burs and metal polishing instruments with irrigation, positively influenced implant survival rates (100% test, 87.5% control group), significantly reduced peri implant pocket depths, reduced bleeding scores and were associated with stable interproximal bone levels at the 3-year followup. Although significant radiographic bone loss was detected, the gingival recession index was significantly higher when implant surface modification was performed. Therefore, this type of surgical technique can be of benefit primarily in the non-esthetic areas, and alternative treatments should be developed for the esthetic zone [47].

Implantoplasty is performed at the aspects of the implant, where due to defect anatomy only a limited potential for bone regeneration and/or re-osseo integration after healing can be expected. Studies even though have reported successful clinical and radiographic outcomes after surgical treatment of peri-implantitis combined with implantoplasty raised concerns about issues like perforation of the implant body, destruction of the implant-abutment connection, overheating of the implant during grinding causing thermal damage to the surrounding bone, or induction of mucosal staining and/or increased risk for late inflammatory reactions due to titanium particle deposition, generated from the grinding procedure, appear as reasonable concerns. Further, reduction of the implant diameter at its coronal aspect, occasionally also involving the implant collar, may compromise implant strength and lead to an increased rate of late mechanical complications, for example, implant collar deformation, loosening of the supra-structure, fixation-screw fracture, and implant fracture; this may, in turn, lead to recurrent peri-implant biological complications and/or require explantation [48, 49]

Adjunctive benefits, derived from the addition of resective surgical treatment consisting of apically repositioned flap, bone recontouring, and surface debridement with 0.12% CHX + 0.05% CPC showed a greater immediate suppression of anaerobic bacteria on the implant surface than a placebo solution, but did not lead to superior clinical results or differences in mean marginal bone loss at 12 months of follow-up [50].

In a recent study by Marco AB et al, patients diagnosed with periimplantitis, treated with a combined resective-implantoplasty therapy and with minimum 2-year follow-up were screened for progressive marginal bone loss, bleeding on probing and suppuration. Clinically and radiographically, implant mobility, and implant fracture were considered to assess peri-implant bone stability of the treated implants.

Results showed that over the 2 to 6-year follow-up period, the disease resolution rate was 83% to 87%. 13% implants were lost or removed due to continuous marginal bone loss and osseointegration failure. BOP was absent in 89.3%, suppuration was resolved in all cases, and no pain or implant fracture was reported [51].

When role of systemic antibiotics in combination with open flap debridement was assessed one study showed no clinical benefits using adjunctive systemic azithromycin [52] and another study stated no potential benefits of systemic antibiotics over 3 years [53]. But another study by Carcuac et al proved that clinical parameters improved significantly with the use of systemic antimicrobials [54].

Adjunctive use of 980-nm diode laser following mechanical open flap debridement failed to reveal any significant clinical improvements in mean BOP and PD scores at the 6-month follow-up [55].

A recent study evaluated the use of recombinant human bone morphogenetic protein (rhBMP-2) in experimental peri-implantitis sites [56]. Within the newly formed bone, re-osseointegration averaged 40% in rhBMP-21 defects as compared with control defects.

Analysis of various studies related to peri-implantitis treatment revealed that oral hygiene instructions and its importance must be stressed very strongly to patients before and after the treatment. Nonsurgical subgingival mechanical debridement in conjunction with local antibacterial such as chlorhexidine digluconate or locally delivered antibiotics is effective in reducing soft-tissue inflammation and should be the first step in successful treatment [57]. In a 5-year follow-up observational study, healthy peri implant tissue conditions in patients with high oral hygiene standard (maintenance every 6 months) were maintained for most patients after resective peri implant surgery [58]. It was found that disease progression occurred only in 10% of the treated implants, revealing that risk factors that includes presence of residual pockets after surgery, smoking, poor oral hygiene, untreated periodontal disease, and systemic disease like diabetes may modify both the initial and longterm outcome of the treatment [59]. If attainable, it is strongly suggested to remove the supra structure during surgical procedure and could be adapted for higher cleansing ability.

Explantation:

If there is advanced bone loss and also the implant cannot be saved, it has to be removed. Explantation is mostly associated with significant bone removal including buccal or lingual bone cortices, and damage to adjacent natural teeth where the inter-radicular space is limited. Different explantation techniques like bur-forceps, neo bur-elevatorforceps, trephine drill, high torque wrench, and scalpel-forceps have been proposed by experts. Studies have found that the posterior maxilla was the most frequent site of implant removal, longer implants were more frequently removed and majority of implants were removed after 1 year in function. The bur forceps, neo bur elevator forceps and scalpel forceps techniques were found to be the most efficient with 100% success. The neo bur elevator forceps technique enabled safe insertion of a new implant in the same explantation site. The high torque wrench technique was found to be the most elegant technique with the highest predictability for insertion of another implant [60]. 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.

VII. Conclusion

Treatment for peri implantitis is aimed at reduction of the anaerobic bacteria by mechanical therapies and other adjuncts to improve clinical tissue condition. Treatment modalities like apically repositioned flap, chemical surface decontamination, and implantoplasty with or without bone augmentation and guided tissue regeneration have been identified as supplements to mechanical debridement. However, histologic evidence of true re osseo integration has not been obtained till date. Main obstacle for bone regrowth in infected implants is incomplete surface decontamination and its interception as early as possible by removing bacterial deposits to stop progression of the disease has been advocated by most of the experts. Prognosis of peri implantitis depends upon its early detection and treatment. An overall clinical improvement appears to emerge with the use of mechanical, surgical and antiinfective therapies, in terms of resolution of inflammation and bone healing.

VIII. References

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