REVIEW ARTICLE

Peri-implant Diseases: Pathogenesis and Treatment

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ABSTRACT

As the field of dental implantology has expanded in last several years and an increasing number of dental implants being placed, peri-implant diseases have now come to the forefront of implant research. The purpose of this article is to review available information on etiopathogenesis and treatment of peri-implant diseases. The results of animal research and human studies have been analysed. With the apparent increase in the incidence of affected cases, long-term follow-ups of treated cases would seem to be a realistic avenue for more information. This may assist in establishing the predictability, magnitude and stability of peri-implantitis treatment procedures.

Keywords: Peri-implantitis, Peri-implant mucositis, Etiology, Treatment modalities.

INTRODUCTION

For the last 25 years, dental implants have evolved into a predictable technology for replacing teeth. Despite the predictable treatment results, with most failures occurring during initial healing and the first year of loading complications do arise during maintenance and retention of implants.[1,2] The tissues supporting osseointegrated dental implants are susceptible to disease that may lead to implant loss.[3,4] Implants, like teeth, are susceptible to bacterial plaque accumulation and calculus formation. In fact, because of the lack of connective fiber insertion and decreased vascular supply around implant, there may be greater susceptibility to plaque induced inflammation.[5]

Therefore, the aim of this article is to review the types of implant associated diseases and current status of various treatment modalities of peri-implant diseases (Fig. 1).

PERI-IMPLANT DISEASES

The inflammatory lesions that develop in the tissues around implants are collectively recognized as peri-implant diseases.[6]

Types

Peri-implant disease includes two entities:

a. Peri-implant mucositis
b. Peri-implantitis

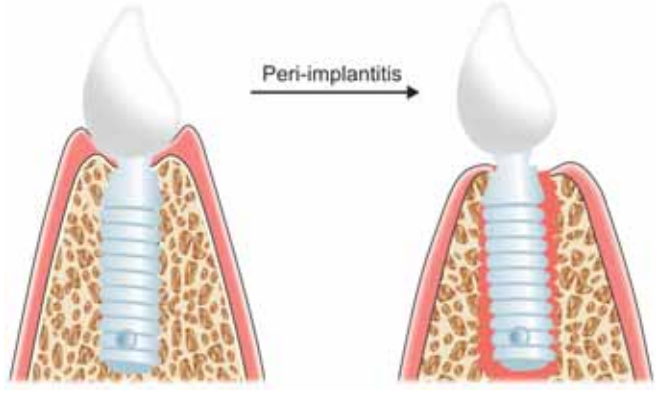

Fig. 1: Schematic illustration of healthy periodontium and peri-implantitis lesion

Definitions of the two peri-implant disease entities were proposed in a consensus report from the first European Workshop on Periodontology (EWOP) (Albrektsson and Isidor, 1994).[7]

a. *Peri-implant mucositis*: According to Albrektsson and Isidor (1994), peri-implant mucositis has been defined as a reversible inflammatory change of the peri-implant soft tissues without bone loss.
b. *Peri-implantitis*: Defined as inflammatory reactions associated with loss of supporting bone around an implant in function.

Peri-implantitis begins at the coronal portion of the implant, while the most apical portion of the implant maintains an osseointegrated status.[5,7-9] This means that implant is not clinically mobile until the late stages, when bone loss has progressed to involve the complete implant surface.[10]

PREVALENCE

There is limited data available related to the prevalence of peri-implant disease. The prevalence of peri-implant mucositis has been reported in the range of 8 to 44%,[1] while frequency of peri-implantitis has been reported in the range of 1 to 19%.[2] Meta-analysis of failures of implants after first year of loading has shown that peri-implantitis accounts for 10 to 50% of the failures.[1,2] In a recent study, Koldsland et al (2010)[11] found the prevalence of peri-implantitis at different levels of severity to be 11.3 to 47.1%.

According to Roos-Jansåker,[12] clinical signs of peri-implantitis are crestal bone loss, deepening of the peri-implant pocket, bleeding and/or pus after probing, swelling and redness of varying degrees, recession and mobility.

Etiology

The main predisposing factor in the etiology of peri-implant diseases is microbial colonization. Plaque formation on oral implants has been studied using scanning electron microscopy and results showed that plaque formation patterns identified on implants are similar to those observed on teeth.[13] The subgingival microflora associated with implant failures due to infection are the same as those associated with adult periodontitis.

Subgingival staphylococci (*S. aureus* and *S. epidermidis*) have been isolated from about 50% of gingivitis and periodontitis patients and from 55% of a small sample of ailing or failing implants.[14] A notable finding was that staphylococci were seen in 15% of cases with peri-implantitis but only in 1.2% of periodontitis and 0.06% in gingivitis. As the difference was a statistically significant, the authors concluded that staphylococci play a role in some implant failures. Recently, microbiological DNA-probe analysis revealed that patients with peri-implantitis harbored high levels of periodontal pathogens like *Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, Prevotella intermedia, Bacteroides forsythus and Treponema denticola*.[15] Also, subgingival spirochete levels in healthy implants have been reported to be low or even zero as compared to that of failing implants.

Etiopathogenesis of Peri-implant Mucositis

Plaque formation around implants and their host response in beagle dogs has been histologically studied. It was found that the host response to the plaque formation on teeth and implants was similar. Several investigators have studied the local defense mechanisms of the peri-implant soft tissue seal and compared them to those in dento-gingival unit. The production of inflammatory mediators and the expression of cytokines appear to be very similar in these two soft tissue types. The experimental model of gingivitis, originally described by Löe et al (1965),[16] represents the ultimate proof for a cause-and-effect relationship between bacterial plaque accumulation and developing gingivitis. The same cause-and-effect relationship was demonstrated in peri-implant mucosa model as well (Fig. 2).

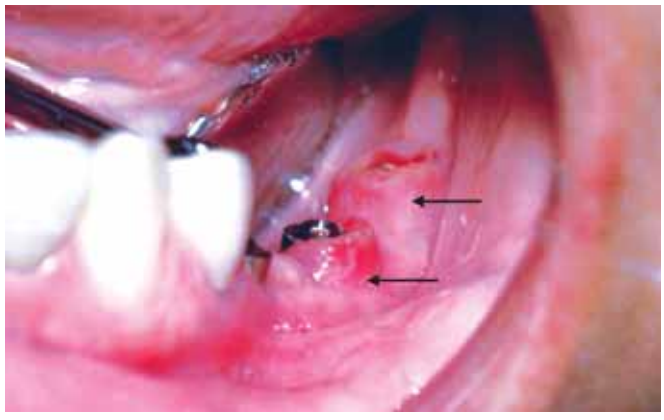

Fig. 2: Clinical view of peri-implant mucositis

Etiopathogenesis of Peri-implantitis

The marginal inflammatory tissue reactions in implants are similar to those encountered in gingivitis and periodontitis of teeth. Pathogenesis of gingivitis and periodontitis has been described recently,[17] as soon as plaque accumulation begins; neutrophils are recruited to the periodontal pocket or the gingival crevice because of the chemotactic peptides, released by the bacteria. Furthermore, as bacteria damage the epithelial cells, they cause epithelial cells to release cytokines that further attract leukocytes (predominantly neutrophils) to the crevice. The neutrophils within the crevice can phagocytose and digest bacteria and therefore, remove these bacteria from the pocket. If the neutrophil becomes overloaded with bacteria, it degranulates. This causes tissue damage from toxic enzymes that are released from the neutrophils. In conclusion, if there is an overload of microbial plaque, then the neutrophils and the barrier of epithelial cells will not be sufficient to control the infection. In such instances, the gingival tissue will become inflamed. If inflammation extends from the marginal gingiva into the supporting periodontal tissues, this result in bone destruction and loss of attachment, a process termed periodontitis. The factors involved in bone destruction in periodontal disease are bacterial and host mediated. Bacterial plaque products induce the differentiation of bone progenitor cells into osteoclasts and stimulate gingival cells to release mediators that have the same effect. Several host factors released by inflammatory cells are capable of inducing bone resorption *in vitro* and can play a role in periodontal disease. These include host produced prostaglandins and some cytokines (IL-1, TNF-α etc.) (Fig. 3).[18]

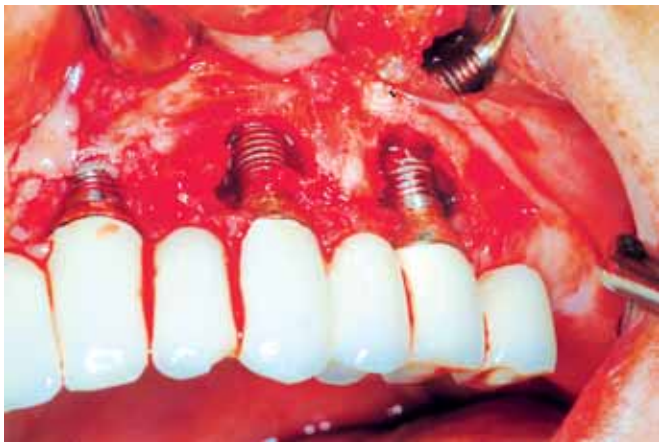

Fig. 3: Clinical view of peri-implantitis lesion

Plaque accumulation on the implant surface initiates the subepithelial connective tissue infiltration by large number of inflammatory cells and the epithelium appears ulcerated and loosely adherent. As the plaque front migrates apically, the clinical and radiographic signs of tissue destruction are seen around both implant and teeth; however, the size of the soft tissue inflammatory lesion and the bone loss is larger around implants (Fig. 4).[5,8] In addition, the implant lesions extend into the supracrestal connective tissue and approximate/populate the bone marrow while the lesions associated with teeth do not. These studies suggest that plaque-associated soft tissue inflammation around implants may have more serious implications than marginal inflammation around teeth with a periodontal ligament. Subgingival bacterial flora associated with clinically inflamed implant sites is different from that around healthy implants. These microbial shifts are comparable to those occurring around natural teeth, and the bacterial flora in chronic periodontitis and peri-implantitis have some similarities.[19] A subepithelial inflammatory response occurs and may play a role in continuing the inflammatory changes that cause the progressive breakdown. The potential periodontal pathogens were shown to be less prevalent in the implant sulci of the totally edentulous mouth. This might indicate a higher susceptibility for peri-implantitis in the partially edentulous mouth. It should be noted that the cause of peri-implant disease is multifactorial and there are many other factors that may contribute this process: biomechanical factors, traumatic surgical techniques, inadequate amount of host bone and compromised host response (Fig. 3).[20,21]

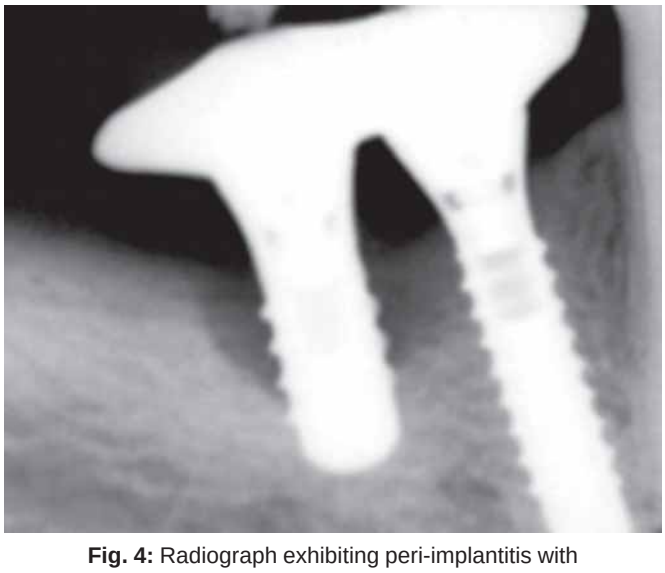

Fig. 4: Radiograph exhibiting peri-implantitis with crater shaped defects

TREATMENT MODALITIES FOR PERI-IMPLANTITIS

Mombelli (2002)[22] described five considerations in the treatment of peri-implantitis:

1. Removal of bacterial plaque residing in the peri-implant pocket.
2. Decontamination and conditioning of the implant surface.
3. Reduction of sites that cannot be maintained by home plaque control.
4. The establishment of an efficient plaque control regimen.
5. Regeneration of bone.

Ideally, the implant undergoes reosseointegration. Conservative, resective and regenerative treatment measures have been described,[23,24] depending on the type and size of the bone defect.

The treatment modalities of peri implantitis are:[25]

1. Nonsurgical treatment/Closed debridement
2. Surgical treatment/Open debridement
3. Regenerative treatment

Therapies proposed for the management of peri-implant diseases appear to be largely based on the evidence available for treatment of periodontitis. There are limited data in the literature on nonsurgical treatment of peri-implantitis. Those that are available suggest that little benefit can be expected (Figs 4 and 5).

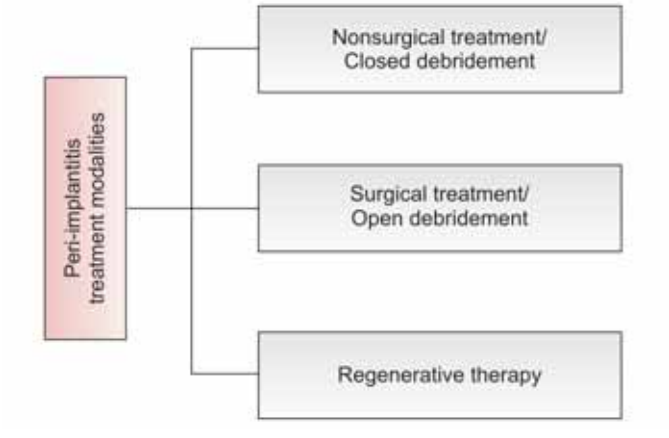

Fig. 5: Peri-implantitis treatment modalities

Nonsurgical Treatment/Closed Debridement

Mechanical Therapy Alone

It has been observed that mechanical nonsurgical therapy could be effective in the treatment of peri-implant mucositis lesions only.[26]

The screw shaped designs, rough surfaces of the implants together with various degrees of surface modifications may facilitate biofilm formation, if exposed to the oral environment. Hence, surface debridement constitutes the basic element for treatment of peri-implant mucositis and peri-implantitis. However, the design of the suprastructure may hinder effective mechanical treatment of the infected implants.

Various instruments and systems have been used for mechanical debridement:

a. Vector system (carbon fiber tip combined with aerosol spray with hydroxylapatite), sandblasting units.
b. Carbon fiber, acrylic, plastic curettes, scalers and plastic brushes.

Out of these, only sandblasting resulted in successful decontamination of all surfaces. It was observed that mechanical nonsurgical therapy could be effective in the treatment of peri-implant mucositis lesions. Mechanical non-surgical therapy alone was shown insufficient to treat peri-implantitis lesions. Only minor changes in bleeding tendency were reported and probing depths either did not improve or worsened.[27] Reduction of the bacterial load to a level allowing healing is difficult to accomplish with mechanical means alone. Therefore, adjunctive therapies like antibiotics, antiseptics and laser treatments have been proposed in order to improve the nonsurgical treatment options of peri-implant mucositis and peri-implantitis.

Mechanical Therapy with an Adjunct of Systemic Antimicrobials

The studies performed so far include both local antimicrobial irrigation and administration of antimicrobials systemically. Improvements were noted for measurements, such as probing depths, gingival index and microbiological parameters. These case reports do not allow definite conclusions on the effects of systemic antimicrobials alone. The systemic antibiotics which have been used are: Amoxicillin, Metronidazole, Tetracycline, Clindamycin, Erythromycin, Ciprofloxacin, Ornidazole.

Out of which metronidazole and combination of amoxicillin and metronidazole were most common choice.

Mechanical Therapy with an Adjunct of Local Antimicrobials

Treatment alternatives that have been investigated include chlorhexidine, citric acid, stannous fluoride, tetracycline, hydrogen peroxide, polymixin B.[28]

Studies have shown that:

- Treatment of peri-implantitis with chlorhexidine is a viable modality that results in a decrease in bacterial growth.[28]
- The application of citric acid at a pH of 1 on the implant surface reduces bacterial growth while also promoting cellular growth and attachment.[28]
- Stannous fluoride was found to cause an apparent increase in the endotoxin level.[28]
- Tetracycline, H_2O_2 and Polymixin B were found to be more effective than controls in decreasing bacterial levels in peri-implantitis.[28]

In a case series, it has been suggested that the adjunctive use of either chlorhexidine gel or minocycline microspheres would improve both clinical and microbiological parameters following treatment.[29]

Renvert et al[26] found benefit to the adjunctive use of minocycline containing microspheres. Although, all studies on local antimicrobial agents as an adjunct to mechanical treatment demonstrated mean improvements of bleeding on probing (BOP) and probing depths, this therapy failed to resolve the lesion in all cases.

Laser Therapy

The outcome data on laser therapy is incomplete and do not show benefits as compared with conventional mechanical therapy.[26] Schwarz[30] compared YAG laser versus manual debridement and chlorhexidine irrigation/gel after 6 months and found that there were no statistically significant difference for changes in attachment loss, probing depth and recession.

Schwarz et al[31] in a controlled study (parallel design) compared implant scaling (plastic curette), chlorhexidine (0.2%) irrigation, and chlorhexidine gel in pocket vs Er.YAG laser. After 1 year, the authors reported that there were no statistically significant differences for attachment loss, pocket depth and recession between the two treatment groups.

Photodynamic Therapy

Photodynamic therapy (PDT) is a process whereby a photo reactive drug, capable of binding to the targeted cell, is exposed to the infected site and subsequently excited to a higher energy state by laser light. This excitation results in release of cytotoxic reactive oxygen species leading to rapid cell death. Numerous PDT dyes have been studied to determine the efficacy of destroying periodontal pathogens. Toluidine blue O, Chlorine e6, Erythrosine, Photofrin, Methylene blue, Azulene and BLC 1010 are some of the photosensitizers currently under investigation for this use.[32]

Surgical Treatment/Open Debridement

Open Debridement Alone

To date, no randomized controlled clinical trials are available on the use of access flap surgery (open flap debridement) alone for the therapy of peri-implantitis. There is only one report of a case by Zablotsky (1992),[33] where one implant was treated by using open surgical debridement, osteoplasty and apical flap positioning and subsequent soft tissue healing. Overall, surgical treatment was shown to give a better outcome. On the other hand, Hayek et al[34] found little microbiological benefit to access surgery and irrigation with chlorehexidine over closed scaling and photodynamic therapy.

Surface Decontamination

Following surgical exposure of the contaminated implant surface, mechanical, chemical, or photodynamic measures or combinations of all three can be used to eliminate infection, resolve inflammation and render the surface conducive to bone regeneration and reosseointegration.

Various methods have been advocated with no definitive gold standard, e.g. air powder abrasion, saline wash, citric acid treatment, laser therapy, peroxide treatment, ultrasonic and manual debridement and application of topical medication. Various options have associated advantages, such as elimination of infection, resolution of inflammation and rendering the surface conducive to bone regeneration and reosseointegration. Disadvantages include damage to the implant surface with mechanical or laser therapy and the inability to remove all the remaining and potentially damaging cell components when using chemical treatment alone.[35]

Schwarz et al[36] in animal study compared surgical debridement using the Er:YAG laser, an ultrasonic scaler and plastic curettes with local application of metronidazole gel as methods for implant surface decontamination. They found within the open debridement group that ultrasonic cleaning clinically resulted in higher clinical attachment level gains than ultrasonic and plastic curettes with metronidazole gel. However, histologically, laser therapy resulted in the greatest degree of reosseointegration (44.8%) with ultrasonic and plastic curette/metronidazole gel debridement.

Persson et al[29] concluded that neither chemical nor mechanical debridement methods resulted in an implant surface capable of facilitating any noteworthy level of reosseointegration despite resolution of the inflammatory process.

In conclusion, resolution of peri-implantitis can be achieved with the various treatment methods of surface decontamination. However, evidence indicates that this open debridement in combination with surface decontamination does not achieve substantial reosseointegration despite new bone regeneration in some of the defects. Furthermore, this reosseointegration occurred with all of the three decontamination procedures i.e., citric acid, saline and hydrogen peroxide. Additional studies are needed to verify this.

Regenerative Treatment

Autogenous bone, allograft, xenograft materials, nonresorbable and resorbable membranes have been used to generate bone around implants. In general, better results have been reported with these methods compared with surgical debridement and surface decontamination alone. However, contradictory results pose a problem when attempting to specify an optimal treatment protocol.[42]

Bone Grafts and Bone Graft Substitutes

A wide selection of different grafting materials, like autogenous bone, bovine inorganic bone, demineralized freeze dried allogenic bone and hydroxyappatite have been used over the years for the treatment of peri-implantitis. The reports by Behneke et al (1997)[37] include multiple cases treated with autogenous bone grafts and with observation intervals extending up to 3 years, notable reductions of probing depths coupled with significant radiographic bone fill were reported.

Barrier Membranes

Studies indicate that using the concept of guided tissue regeneration is a predictable way of treating osseous defects in peri-implantitis as well as improving soft tissue conditions. Nonresorbable expanded polytetrafluoroethylene (ePTFE) membranes have been used clinically and experimentally to generate bone around exposed implant threads and after placing implants into extraction sockets. The treatment of peri-implantitis with nonresorbable ePTFE membrane results in bone fill of the defects and improved soft tissue conditions. But, as these membranes require second surgery after 6 to 8 months, need for development of bioresorbable devices was felt. Hurzeler[38] clinically evaluated collagen membrane (Bioguide) to treat peri-implant bony defects but encountered with membrane exposure.

Combination of Grafting Materials and Barrier Membranes

The majority of the studies identified on treatment of peri-implantitis comprised those in which combination of grafts and barrier membranes were used. The submerged approach

was used in half of these studies in order to allow for undisturbed healing and to reduce the risk of infection. From the results, it is evident that this submerged approach was not always successful in practice as the most common complication was membrane exposure.

A study by Schwarz et al[36] evaluated the healing of intrabony peri-implant defects following application of a nanocrystalline HA (NHA) paste or Bio-Oss® in combination with Bio-guide®. Clinical parameters were recorded at baseline and after six months. Results showed that both treatments resulted in improved clinical conditions. Recently, a study by Roos-Jansåker et al[39] compared two surgical techniques using a bone substitute with or without the use of a resorbable membrane. The nonsubmerged approach was used. No significant differences were observed between the groups. It can be concluded that placement of membranes in addition to bone grafting does not provide any adjunctive effect.

Regeneration Promotion

You et al[40] found a significant difference in terms of the amount of reosseointegration within the limits of the three most coronal threads when platelet enriched fibrin glue was used in addition to autogenous bone (50.1%) compared with autogenous bone alone (19.3%) or debridement (6.5%).

Other Treatment Modalities

Apically Repositioned Flap Surgery and Implant Surface Modification

A comparative study by Romeo et al[41] demonstrated that modification of the implants surface (implantoplasty) could be of value when treating peri-implantitis and should be considered as an adjunct to resective surgery.

Laser Decontamination as an Adjunct to Surgical Therapies

A study by Dortbudak et al[42] attempted to examine whether lethal photosensitization is effective in *in vivo*. The method of evaluation was by measuring the levels of *Aggregatibacter actinomycetemcomitans*, *porphyromonas gingivalis* and *prevotella intermedia* before and after application of the dye and after the laser application. Although, complete elimination of the bacteria was not achieved, the treatment resulted in significant reduction in all three groups of bacteria. In a case report by Dörtbudak O et al (2000),[42] a soft laser was used to decontaminate the implant surface before filling the defect with autogenous bone and covering it with an ePTFE membrane. The short-term results of this study corroborate the efficacy of the applied treatment method in prolonging the service time of dental implants involved with peri-implantitis. Recently, in a study by Deppe et al,[43] a conventional decontamination technique was compared with a CO_2 laser-assisted treatment. Based on the results of this study, authors concluded that, short-term CO_2 laser treatment may have a beneficial effect on the treatment of peri-implantitis. No such effect was observed in the long-term.

From the studies and case reports above, it can be concluded that treatment of peri-implantitis lesions with lasers as an adjunct to conventional treatment, may lead to better clinical results than conventional treatment alone. However, further long-term studies are still required in this field before firm recommendations can be made.

The cumulative interceptive supportive therapy (CIST)—a protocol for the monitoring of healthy implants and the interception of peri-implant diseases was suggested by Lang et al.[44]

This protocol relies on probing depth, bleeding on probing and radiographic evidence of bone loss. As each parameter becomes more severe, more complex treatment is introduced, with each subsequent treatment incorporating that of the previous. For example, according to this protocol, if a PPD of 6 mm is displayed, positive for BOP and greater than 2 mm bone loss, combination therapy of A + B + C + D can instituted in Figure 6.

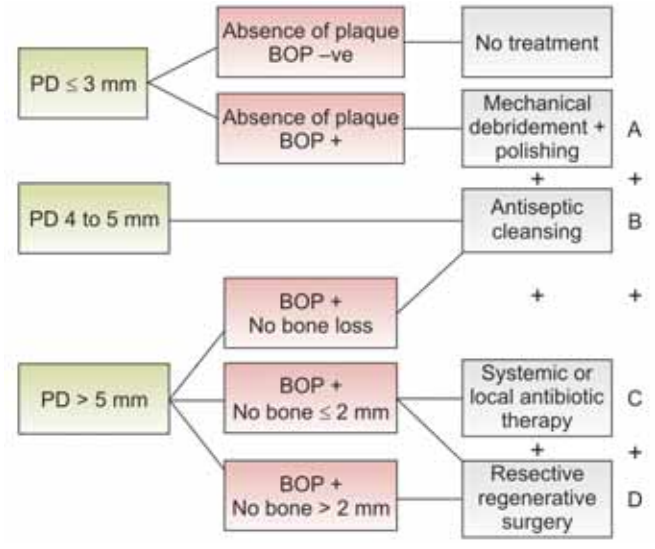

Fig. 6: Cumulative interceptive supportive therapy protocol

SUMMARY

Peri-implantitis is arguably one of the most significant risk factors associated with late implant failures. Despite a projected increase in the incidence of peri-implantitis, the clinician is faced with a difficult decision-making process from beginning to end. It must be recognized that peri-

implantitis is a multifactorial disease process, which may include factors, such as host immune response and susceptibility, microbiology, host modifying factors and local environment. The relevance, contribution and impact of other factors, such as implant surfaces, smoking, history of chronic periodontitis and occlusal loading remain obscure and undoubtedly further long-term studies are necessary for clarification. Further research is necessary to determine the most cost-efficient, safe and effective treatment of peri-implantitis. Until additional information is available, the clinician should make a clinical judgment based on the individual case using a rational and evidence-based approach.

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