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PERI – IMPLANTITIS: A REVIEW

Dental Science

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ABSTRACT

Peri-implantitis is described as a destructive inflammatory disease affecting the soft and hard tissues around osseointegrated implants, leading to the formation of a peri-implant pocket and loss of supporting bone. There is various treatment approaches are available for peri-implant disease (conservative & surgical therapy). Peri implant mucositis and moderate case of peri-implantitis can be treated by conservative approach. Treatments include different manual techniques, laser-assisted systems as well as photodynamic therapy, which can be extended by local or systemic antibiotics. It is possible to regain osseointegration. In cases of moderate to severe peri-implantitis cases surgical approach are more effective than conservative therapy. This review aimed to evaluate the etiologies & pathogenesis, risk factors, diagnosis & treatment of peri-implantitis.

KEYWORDS

Peri-implantitis, mucositis, conservative & surgical therapy, osseointegration

INTRODUCTION:

A dental implant is a surgical component that provides support to the dental prosthesis. The basis for modern dental implant is a biologic process called osseointegration (Branemark) form an intimate bond to bond. The hard & soft tissues surrounding an osseointegrated implant show similarities to the periodontium around natural dentition[1]. The gingiva around dental implant is called peri implant mucosa, & consists of well keratinized oral epithelium, sulcular epithelium, & junctional epithelium with underlying connective tissue. Between implant surface & epithelial cells are hemidesmosomes & basal lamina[2]. The most significant difference between natural teeth & implant is that implants lack the periodontal ligament. The collagen fibres are unattached & parallel to the implant surface rather than in functional contact from bone to the cementum.

Micro – organisms in the oral cavity colonise the surface of the tooth or an implant & form a bio film & microbial challenge include inflammatory reaction in the surrounding tissues. At teeth, inflammation in the gingival is termed gingivitis, while the term periodontitis addition to gingival inflammation also includes loss of supporting tissues. The corresponding conditions at implants are peri-implant mucositis & peri-implantitis.

Although the prevalence of peri-implantitis is 5-8%, every affected implant represents a threat to the congivety of associated prosthetic replacement. Therefore detection & treatment of rarely pathogenic changes during regular recall maintenance visits prevent peri-implant soft-tissue inflammation & progressive bone loss.

Etiologies & pathogenesis

Shortly after the implants are placed, salivary glycoprotein will adhere to the exposed titanium surface with associated microbiological colonisation[3-10] .the formation of bio film plays a key role in initiation & progression of peri-implant disease[3,3,7,9-12]. Peri –implant conditions have been associated with gram-ve anaerobic bacteria similar to patients with severe chronic periodontitis[3,4,11,12]

Peri-implant mucositis does not necessarily progress to peri-implantitis. The 'epithelial scaling' around implants is similar in function to that around implants & is also similar in function to that around teeth[13]. It is also concluded that there is no evidence to suggest that any structural difference with natural teeth & implants would significantly alter the host response to bacterial challenge[4,5,14,15.]

Furthermore, evidence are there to suggest that peri-implant mucositis are reversible when effectively treated[4,11]. Elimination of the biofilm from the implant surface is prime-objective when treating peri-implant mucositis.

Peri-implantitis & periodontitis lesion from human biopsies have many features in common[16]. The connective tissue adjacent to pocket epithelium is infiltrated by inflammatory cells, with B-lymphocytic & plasma cells bring the most dominating cell types. In case of peri-implantitis & periodontitis pro-inflammatory cytokines such as IL-1,6,8,12 & TNF-α are uncontrolled[17,18].

Experiments that allowed undisturbed dental plaque formation on implants & teeth in humans[19] & in dogs[20] demonstrated more advanced inflammatory cell infiltration in peri-implant mucosa. The results of clinical & radiographic studies suggest that tissue destruction is more pronounced & size of inflammatory cell infiltrate is more, approaching crystal bone in peri-implantitis.the increased susceptibility of bone loss around implants may be related to absences of inserting collagen fibres in to implants[21].

A comparison of periodontitis & peri-implantitis not self-limiting' process existing in the tissues around- natural teeth that resulted in a protected connective tissue capsule of supra-crestal gingival fibres of the tooth that separated the lesion from the alveolar bone. They[22] also noted that 'such a self-limiting' did not occur in peri-implant tissue & the lesion extended to the bony crest.

On experimentally induced peri-implantitis it was noted that after the removal of ligature there was spontaneous & continuous progression of disease with additional bone loss[19,23,24]. All implants appeared to be susceptible to peri-implantitis[23,24].The primary objective of treating peri-implantitis is the elimination of the biofilm from the implant surface.

Risk factors:

Previous periodontal disease.

Systematic reviews have 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[25].

Poor plaque control / inability to clean

It may be related to implant positioning & meeting patient expectations for aesthetics, phonetics & function. Prosthetic design can also preclude clinical evaluation with probing & adequate home-care

procedures[26].

Residual cement.

Inflammations & disease related to dental cements maybe related to its roughness, which may cause inflammation; its surface topography may provide a positive environment for bacterial attachment.

Smoking

Systematic reviews have concluded that there is an increased risk for peri-implantitis in smokers with odds ratio ranging from 3.6-4.6. Cohort & cross sectional studies frequently have linked smoking to higher implant failures. One Study[27] reported that 78% of implants in smokers had the diagnosis of peri-implantitis. While non-smokers it was 64%. A cross-sectional study demonstrated that smokers had an odds ratio of 3.8 of developing peri-implant mucositis & an odds ratio of 31.6 of developing peri-implantitis[28].

Genetic factors

Association between IL-1 gene & peri-implantitis remains to be determined since conflicting results exist[29].

Diabetics

Conclusive evidence does not allow for a definitive conclusion that diabetic patients have a higher incidence of peri-implantitis. Additional perspective cohort studies are needed to clarify the association between diabetics & peri-implantitis[30].

Occlusal overload

Implants are considered less tolerable to non-axial occlusal load compared to teeth because of lack of periodontal ligament. Finite element studies suggest that occlusal load is concentrated at implant marginal bone[31]. A recent systematic review[32] suggested that occlusal overload was positively associated with peri-implant marginal bone loss. However poor oral hygiene was still the key causative factor. The role of occlusal overload on peri-implanitis requires further investigations with more precise definition of occlusal overload.

Potential emerging risk factors.

They include rheumatoid arthritis with concomitant connective tissue disease, increased time of loading & alcohol consumption[33].

Diagnosis:

Suppuration has been recognised as one of the diagnostic criteria for peri-implant diseases[34]. The clinician must use a combination of probing data overtime, inflammatory status of mucosa "bleeding on light probing" radiographic changes in bone levels overtime[35] & possibly bacterial &/or peri-implant cervicular fluid sample data to arrive at an accurate diagnosis of peri-implantitis.

Probing, bleeding & suppuration:

Initial probing of implant can be done once the final restorations have been installed with a force of 0.25N[36]. Probing may have to be done with prosthesis removed as it may obviate probing along a parallel axis to the implant[37].Increasing probing depth & bleeding are indications need to perform additional radiographic examination[38,39]. The presence of exudates indicates pathological changes & the necessity for further evaluation & treatment.

Secondary diagnosis.

These include bacterial culturing, inflammatory markers & genetic diagnostics for diagnosis of peri-implant diseases.

Treatment of peri-implantitis.

The management of peri-implantitis is focused on infection & bacterial controls. The treatments proposed for peri-implant disease are based on evidence gained from treatment of periodontitis. Both surgical & non surgical techniques have been developed for treatment of peri-implantitis.

Non surgical techniques

The treatment of peri-implantitis in case of incipient bone loss involves the limitation of local irritants with or without surface decontamination, systemic antibiotics, addition adjunctive therapy agents or devices (Machtei 2014)[40]

Mechanical treatment

Karring et al (2005)[41] compared the treatment results obtained with vector ultrasound system & with carbon fibre curettes. After 6 months of follow-up. No significant differences were found between the 2 techniques & neither proved sufficient to treat peri-implantitis. Presson et al (2010)[42]did similar studies with titanium curettes & with ultrasonic devices & both methods failed to eliminate or reduce bacterial counts in peri-implantitis.

A study conducted by sahm et al (2011)[43] compared mechanical debridement using carbon curettes & antiseptic therapy (chlorhexidine digluconate (MDA)) with amino acid glycine powder (air-abrasive device (AAD)).after 6 months of follow-up treatment of both study groups resulted in limited clinical attachment level & bleeding was reduced in AAD groups compared to MDA groups.

Mechanical treatment associated to antibiotics.

Studies by Renvert (2006-08)[44] et al compared minocycline microspheres & chlorhexidine gel debridement. After 1 year of treatment both study groups showed improvement in plaque index, pocket depth, & bleeding without improvement in terms of micro-biota.

Schar et al (2013)[45] examined the benefit of photodynamic therapy over minocycline microspheres. In both the groups' significant reductions in mucosal inflammation was observed up to 6 months.

Machtci et al (2012)[46] evaluated & compared the matrix chip (matrix C) with that of chlorhexidine chips (Perio C) in 60 patients with probing depth 6-10 mm & bone loss > 2mm. the result yielded after 6 months of repeated treatment shows probing depth reduction was greater in Perio C compared to Matrix C. clinical attachment gain for both groups were significant.

Laser Assisted Peri Implantitis Procedure (LAPIP)

Giannini et al (2006)[47] investigated the efficacy of pulsed (Nd:YAG) in achieving bacterial ablation while preserving the surface properties of titanium implants. It was found, by light and atomic force microscopy, that Nd:YAG laser, when used with proper working parameters, was able to bring about a consistent microbial ablation of both aerobic and anaerobic species, without damaging the titanium surface Caccianiga et al (2013)[48] evaluate the bactericidal potential of photodynamic therapy by using a new high level laser irradiation protocol associated with hydrogen peroxide in peri-implantitis.it was found, Photodynamic therapy using HLLT appears to be a good adjunct to surgical treatment of peri-implantitis.

Pai et al (2014)[49] evaluated the added clinical benefits of laser as an adjunct in the treatment of peri-implantitis. It was found; the Irradiation with a diode soft laser had positive biostimulating effects, which might be used in treatment of peri-implantitis and osseointegration of dental implants.

Surgical techniques.

Surgical techniques can be divided into respective & regenerative surgery. There techniques can be used according to the type of bony defect, whereas Schwarz et al (2014)[50] have demonstrated that combined surgical procedure is effective in controlling advanced peri-implant lesion.

Aghazadeh et al (2012)[51] concluded that respective surgical procedures coupled with bovine derived xenografts & placement of collagen membrane have more radiographic evidence of bony defect filled as compared to autogenous bone graft.

The 2 year results by Schwarz et al (2008)[52] demonstrated that both nanocrystaline hydroxyapatite & application of combination of natural

bone mineral & collagen membrane were efficacious in providing clinical significant reduction of pocket probing depth & gain in clinical attachment level, but in the 4 year study by Schwarz et al (2009)[53] application of the combination of natural bone mineral & collagen membrane were more efficacious in clinical improvement as compared to nanocrystaline hydroxyapatite. But the 6 months of Schwarz (2006)[54] study concluded that application of nanocrystaline hydroxyapatite & guided tissue regeneration showed significant improvement in clinical parameters.

Wohlfahrt et al (2012)[55] evaluated the 12 months outcome by adding porous titanium granules (PTG) together with open flap procedure & in conjugation with mechanical debridement of implant surface for decontamination with 24% EDTA gel followed by antibiotics 3 days prior to surgery & for 7 days after the surgery. Both the treatment demonstrated significant improvement in probing pocket depth but the reconstruction with PTG resulted in better radiographic peri-implant defect fill.

Romen et al (2007)[56] have compared the efficacy of respective surgery with that of implantoplasty. The results obtained after 3 years of therapy demonstrated that the marginal bone loss was significantly lower after implantoplasty.

Schwaz et al (2011[57], 2012[58]) in 2 studies of advanced peri-implantitis evaluated & compared the efficacy of Er:YAG laser (ERL) surface decontamination/debridement (DD) with that of plastic curettes & cotton pellets (CPS) socked in sterile saline & both procedures were followed by an implantoplasty at the exposed implant surface & were augmented with a natural bone mineral & were covered with a collagen membrane. After 24 months of treatment CPS group yield significant reduction in bleeding on probing & the radiographic bone fill at intrabony defect were same in both groups but the clinical attachment values were not significantly different in both groups.

Study by waal et al (2013)[59] demonstrated that the adjunctive benefits derived from the addition of respective surgical treatment consisting of apically repositional flap, bone recontouring & surface debridement & with 0.12% CHX + 0.05% CPC to a placebo solution tend to be greater immediate suppression of anaerobic bacteria on the implant surface than a placebo solution, But does not lead to superior clinical results.

Current status

Until now, no particular treatment protocol has been shown effective. Disease resolution is satisfactory by surgical treatment. Peri-implant mucositis can be treated by non-surgical treatment (schar et al 2013[45]). If peri-implantitis is diagnosed then the treatment protocol depends on intraosseous defects. If bony defect is minimum, then implantoplasty can improve the bony defect. (Romeo et al. 2007)[56] .

Non-surgical treatment could improve significant clinical parameters but pathogens are not reduced. Treatment standard of peri-implantitis can be improved by decreasing the bacterial pathogen hence it is effective if respective surgery is followed in the incipient case of peri-implantitis as well.

In the advanced peri-implantitis combined treatment of respective & regenerative surgical procedure followed surface decontamination /debridement reduce bacterial count but there was no superior improvement in clinical parameters hence GBR (Aghazodeh et al 2012)[51] or the application of bone substitute (Schwarz et al .2009)[53] (Nanocrystaline hydroxyapatite) can be efficacious for treatment of peri-implantitis. The majority of surgical protocols include pre-operative & post-operative chlorhexidine rinse. Maintenance phase after surface is also important which include oral hygiene instructions & surface bio film removal.

While the current available evidence does not allow any firm specific recommendations for non-surgical or surgical therapy of peri-implantitis, the following elements of therapy seems to be beneficial.

A pre-treatment phase

Oral hygiene instructions & counselling for smoking cessation.
Assessment of the prosthesis for access for plaque control.
Prosthesis removal & adjustment if required.
Non-surgical debridement with or without antimicrobials.

*Surgical access (*when resolution of peri-implantitis is not achieved with non-surgical treatment)
Full thickness muco-periosteal flap to allow through cleaning of the contaminated implant surfaces.
Stabilization of intra-osseous peri-implant defect with a bone substitute/bone graft/bioactive substance without or with a resorbable barrier membrane.

Post-operative anti-infective protocol.

Peri or postoperative systemic antibiotics
Chlorhexidine rinses during the healing period.

Maintenance care

3-6 month maintenances, including oral hygiene instruction & supra mucosal bio film removal.

Future prospects

The basis for any treatment is to know all the possible cause & etiology of the specific disease & then to determine its prevalence. The exact etiopathogenesis of the disease condition have not been established, & the exact nature of hoe the diagnosis of peri-implant conditions have been based on the presence of bleeding on probing, probing depth & suppuration. Diagnostic aids helpful in disfiguring the clinical condition include various chemokines that can be used as biomarkers could give an indication of the clinical condition in a non-invasive manner. Genetic factor could also be studied via the availability of genetic diagnosis for diagnosis of peri-implant diseases have now evolved from a traditional elimination of local irritants with or without systemic antibiotics to additional adjunt therapy agents like local drug delivery system & the use of laser as in application of PDT & use of ER;YAG laser. Surgical techniques have showed improved clinical outcome. Various bone graft along with membranes have been used in the treatment of peri-implantitis with successful clinical outcomes. The use of tissue engineered products can be the future in regenerative therapy of peri-implantitis disease conditions.

Conclusion:

Routine monitoring of dental implants as a part of comprehensive periodontal evaluation & maintenance is essential.

It is suggested to:
Identify risk factors associated with developing peri-implant diseases
Establish radiographic baseline at the time of implant placement.
Establish clinical & radiographic baseline at final prosthesis insertion.
Employ methods that monitor implant health & determine inflammatory complications as part of an ongoing periodontal maintance program.
Establish an early diagnosis & intervention which will contribute to more effective management of peri-implant disease.
Complete osseointegration is difficult to achieve. Even though the different treatment modalities cannot be comparable, however the outcome of surgical treatment of peri-implantitis is good. Surgical procedures for peri-implantitis in human have shown positive results but long-term study is needed to achieve the reliability of the treatment.

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