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Review Article

Peri-Implantitis - A Bird's Eye View

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ABSTRACT

As dental implants are more widely used, so associated problems have also become more common.Peri-implantitis is an inflammatory process affecting the soft and hard tissues surrounding an implant? This disease is associated with loss of supporting bone, bleeding on probing, and occasionally suppuration. . However, in order to prevent such an inflammatory changes around dental implants, the following measures can be considered: periodontal health in the remaining dentition, the avoidance of deepened peri-implant pockets, and the use of a relatively smooth abutment and implant surface. Many methods of treating peri-implantitis have been documented in the literature and most focus on removal of the contaminating agent from the implant surface.

Key words: Dental implants; peri-implantitis; periodontitis

INTRODUCTION

Oral implants have enjoyed high clinical success rates over the last decade, with an explosion of numerous surfaces and designs of implants all claiming to have superior quality over another. It should be recognized however, that clinical complications or failures do occur and as such, a challenge is posed to the clinician in terms of initial diagnosis of peri-implant diseases and subsequent management.

Peri-implantitis is an implant-related condition which is increasingly being noticed in the clinical setting, contributing to a significant proportion of implant failures. As modern dentistry moves forward in leaps and bounds, the focus so far has been on the design of implants, both on a macro- and microscopic scale to improve and ensure success. Implant failure due to peri-implantitis however, is a multifactorial disease process most likely more attributed to the interaction of certain host factors e.g. microbiology, genetic susceptibility and host modifying factors. [1] The mechanism of interaction between these factors is still unknown, yet animal and human studies implicate each factor as playing a crucial role. Additionally, diagnosis and management of a peri-implant disease poses a major challenge to the clinician and relies on a rational and evidence based clinical approach.

According to the Consensus Report of the Sixth European Workshop on Periodontology Peri-implant mucositis and peri-implantitis are infectious diseases. Peri-implant mucositis describes aninflammatory lesion that resides in the mucosa, while peri-implantitis also affects the supporting bone. [2]

The studies on prevalence have presented that Peri-implant mucositis occurred in 80% of the subjects and in50% of the implant sites. Peri-implantitis was identified in 28% and at least 56% of subjects and in 12% and 43% of implantsites, respectively. [3] Whereas one more study yielded a substantial variance in prevalence of peri implant disease from 11.3% to 47.1% and said that peri-implant inflammation was a frequent finding with and without peri-implant bone loss. [4]

ETIOLOGY:

Peri-implant infections are generally classified as peri-implant mucositis and peri-implantitis depending on the severity. Peri-implant mucositis is defined as a reversible inflammatory reaction in the soft tissues surrounding an implant. Peri-implantitis is an inflammatory reaction with loss of supporting bone in the tissues surrounding an implant.

As soon as an implant is exposed to the oral cavity plaque will form on its surface. The process may be identical to that seen on teeth, with the formation of pellicle and subsequent microbial colonization. In edentulous patients colonization of the peri-implant sulcus originates from the microflora found in saliva.

A comparison of residual periodontal pockets and periimplant sulci found that the same bacteria colonized both. If periodontal pathogens were identified in pockets prior to implant placement they were also detected at implant sites three months after exposure to the oral environment.

The microbiota associated with healthy peri-implant tissues closely resembles that of the flora associated with gingival health. The organisms associated with mucositis are very similar to that of gingivitis and, unsurprisingly, that of peri-implantitis is very similar to that seen in periodontitis.

Using dark-field microscopy to analyze plaque samples collected the percentage of coccoid cells, motile rods and spirochetes from the periimplant mucositis sites was very similar to that from the gingivitis sites. Interestingly, the inflammatory infiltrate was of equal size to adjacent control teeth and to implants when *de novo* plaque formation was studied in a beagle dog model. [5]

In 1987 Mombelli et.al reported that in the unsuccessful sites, a substantially different distribution of morphotypes was found microscopically compared with both healthy sites in the same patients and in the successful patients. Spirochetes were not found in any of the successful cases and in only 2 samples of the healthy sites of the healthy sites of the unsuccessful patients, but all but one failing site in these patients harbored spirochetes. Furthermore, failing sites harbored significantly elevated numbers of motile rods fusiform bacteria (Fig 1).

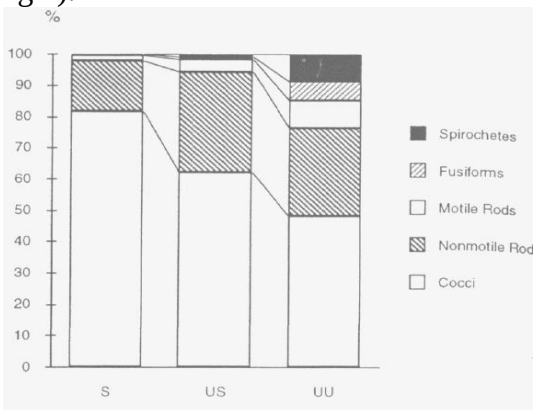

Fig. 1. Distribution of bacterial morphocytes as seen in the dark field microscope. S: datafrom patients with only successfully implants. US: data from the healthiest sites in patients with unsuccessful implants. UU: data from sites with peri-implantitis.

The total counts of colony-forming units, determined by anaerobic culture, were significantly higher in the failing sites than in the healthy sites.41% of the cultivated organisms were gram-negative anaerobic rods in the samples of the failing sites (Fig. 2). This number was significantly higher than that of the successful sites, where the group of facultative cocci was predominating. Failing sites harbored significantly elevated numbers of *Provetala intermedia* and *Fusobacterium species*. On the other hand, the proportions of *streptococci* and *Actinomyces species.* was reduced .*Provetala gingivalis* was not found in any of the samples investigated in this study, neither culturally nor by indirect immunofluorescence.

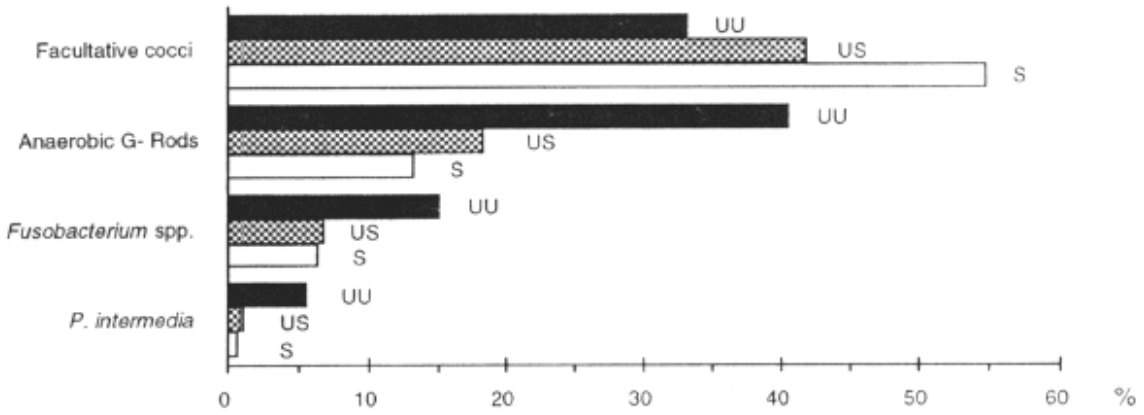

Fig.2. Mean proportion of facultative cocci, anaerobic gram negative rods, Fusobacterium species. and Provetala intermedia in the anaerobically cultivable microflora.
S: data from patients with only successful implants. US: data from the healthiest sites in patients with unsuccessful implants.
UU: data from sites with peri-implantitis.

Meffert R.M. 1996 showed that implants present for three to four years had more bacteria present than did implantitis in place for one to two years, and that there were more *P.gingivalis* and *Provetala intermedia* in the partially edentulous case than in the fully edentulous case.

IMPLANT FAILURE:

Failure sometimes happens in implant therapy. Such failures occur due to complications that may include a number of causes including prosthesis instability, implant mobility, occlusal trauma, fractured components, pain, inflammation, infection and neuropathy. [6]

Implant failures can be broadly divided into two categories:

a) Early failures or

b) Late failures.

Early failures may be related to an inability to establish a close bone-to-implant interface and may occur before or after loading. It is suggested that a number of factors are relevant to early failures, such as; premature loading, biocompatibility, surgical trauma or an impaired host healing response.

Late failures refer to a disruption of an already osseointegrated relationship between the mineralized bone and implant. Causative factors of late failures include; over-loading and chronic bacterial infection (peri-implantitis) but the major cause of late failures could be attributed to peri-implant infections. It was noted that patients with good oral hygiene tended to keep implants longer.

Peri-implantitis lesions are often asymptomatic and usually detected at routine recall appointment. The following signs and symptoms are typical for peri-implantitis lesions:

- Radiological evidence for vertical destruction of the crestal bone. The defect is usually saucer shaped and there is osseointegration of the apical part of the fixture;
- Vertical bone destruction associated with the formation of a peri-implant pocket;
- Bleeding and suppuration on probing; possible swelling of the peri-implant tissues and hyperplasia; and
- Pain is an unusual feature, which, if present, is usually associated with an acute infection.

The incidence of peri-implantitis is quite rare, ranging from 2-10%, an estimation of the prevalence of peri-implantitis is difficult and depends upon the criteria used to separate health from disease. A mean crestal bone loss of 0.9- 1.6mm is expected during the first post-surgical year and then a yearly loss of 0.02-0.15mm subsequently. [7]

It is apparent that periodontitis = peri-implantitis in etiology and therapy.

1. The bacteria are the same (black pigmented bacteriodes and others)
2. The infective process is the same.....progressing from gingivitis or soft tissue involvement to the osseous structures
3. The osseous defect topography is the same....crater or cup like defect at crest around the implant fixture, progressing apically
4. The response of the soft tissue around implants and teeth is the same when exposed to dental plaque..... When home care is instituted and effective, the tissues respond.
5. The response to therapy is the same, applied to implants and teeth.....after teeth / implants are detoxified and osseous defects grafted, repair will usually take place.

In fact, the implant is more subject to breakdown than the natural tooth.

1. There is no periodontal or peri-implant ligament allowing for shock absorbing or stress absorbing.
2. There is no connective tissue attachment.
3. The design of the superstructure on dental implants renders it less conducive to optimum home care *(Ronald M. Meffert D.D.S 1994).*

DIAGNOSIS: [7, 8, 9]

The diagnosis of peri-implantitis needs careful differentiation from peri-implant mucositis, primary failures to achieve tissue integration and problems lacking an inflammatory component. This includes

- Ruling out unusual anatomical features,
- Unusual tissue morphology,
- Hperplastic responses and
- Exposure of parts of the implant due to recession or surgical trauma.

Given the similarity between periodontal and implant diseases the diagnostic parameters used for assessing peri-implantitis are the same as one would use for assessing periodontitis.

The parameters include

- Clinical indices,
- Peri-implant probing,
- Bleeding on probing (BOP),
- Suppuration,
- Mobility,

- Peri-implant radiography and
- Microbiology.

Clinical signs of peri-implantitis may not always be evident. Standardized radiographs are suggested one year after fixture placement and every alternate year thereafter.

Clinical indices:

Swelling and redness of the peri-implant mucosa have been reported from peri-implant infections. There are difficulties in using indices developed for periodontal disease, perhaps due to the different structure of the tissues around implants. The soft tissue layer immediately adjacent to an implant is a less vascular, less cellular, highly collagenous scar tissue compared to normal gingival tissue. In addition, texture and color may depend on appearance before implantation and properties of the implant surface. The amount of plaque around an implant should always be evaluated.

Peri-implant probing: [1, 2]

Probing is essential for diagnosis ofperi-implant diseases.The soft tissue cuff around an implant in a canine model has been shown to be about 3-3.5mm regardless of system and the connective tissue attachment of 1-1.5mm. Therefore, generally successful implants allow the probe to penetrate approximately 3mm.The exception here is deeply submerged implants. However, when placing implants one should, ideally, try not to create deep pockets as those over 5mm are ideal niches for putative periodontopathogens and may be confused for peri-implantitis. There is no scientific evidence to suggest that periodontal probing affects the integrity of an implant, but it should be noted that a metal probe may damage the implant surface. A rigid plastic probe is ideal. Probing the peri-implant sulcus with a blunt, straight periodontal probe allows for assessment of peri-implant probing depth, distance between the soft tissue margin and a reference point on the implant for measuring hyperplasia or recession, bleeding and suppuration.

Lang *et al*. investigating the effect of different mucosal conditions around implants confirmed the excellent sealing effect of the soft tissue collar in health and peri-implant mucositis and reported relatively uninhibited penetration to the alveolar crest in peri-implantitis lesions. Probing around oral implants should be considered a reliable and sensitive parameter for the long term monitoring of peri-implant mucosal tissues.

Bleeding on probing:

Bleeding on probing indicates thepresence of inflammation in theperi-implant mucosa.It may be used asa predictor for loss of tissue support.It has been shown that it is not a reliable predictor for progression of periodontal disease. Instead its absence is a much better predictor for stability. In the absence of any evidence to the contrary, it would seem reasonable to extend this concept to implants. [2]

Suppuration:

The probing depth, the presence ofbleeding on probing and suppuration should be assessed regularly for the diagnosis of peri-implant diseases. [2] Neutrophils are present whenever disease is present. High numbers have been linked with inflammation of the peri-implant tissues, suggesting that suppuration maybe a sign of peri-implantitis.

Mobility:

Implant mobility is an indication of lack of osseointegration, but it is of no use in diagnosing early implant disease, rather it shows the final stages of de-integration. Initially the bone loss associated with peri-implantitis is observed to be marginal and results in the formation of infrabony defects. The apical portion of the implant will be fully integrated, so an increase in mobility will not be evident. Complete loss of

osseointegration would be reflected in a sudden increase in implant mobility.

Peri-implant radiography:

Conventional radiography has been widely applied to evaluate the bony structures adjacent to implants over long periods of time. However, minor changes in bone morphology may not be noticed until they reach a significant size. Radiographic evidence of bone to implant contact does not indicate osseointegration. Digital subtraction radiography can increase the sensitivity significantly and has been successfully applied. [2]

MAINTENANCE, CARE AND TREATMENT:

There are a number of steps at time of placement and restoration that can improve the long-term prognosis of fixtures. Patient motivation and oral hygiene are paramount. Periodontal health should be achieved prior to proceeding with implant therapy. Restorations should be cleansable with well-fitting margins. In addition, as much of the mucosal tissue as possible should be preserved in its original position.

A maintenance program should be undertaken after successful implant therapy. This should be tailored to the individual and include regular recalls to provide optimal disease prevention. The recall visit is similar to that for a periodontal patient in maintenance in that each visit includes examination, re-evaluation, diagnosis, motivation, and treatment of infected sites.

Before a patient is enrolled in a maintenance program one should ensure that baseline data has been established. Probing pocket depths and mucosal margins position are both noted and radiographic crestal bone levels are established.

The decision process for peri-implantitis maintenance and treatment should be a rational and evidence–based approach.

The first question is 'Are there peri-implant pockets greater than 3mm?' One should also assess presence/absence of plaque and bleeding. If the answer is in the negative to all three, then no therapy is required, the length of recall appointment may be increased and radiographs taken every other year.

The presence of plaque or bleeding indicates insufficient oral hygiene. The patient's oral hygiene should be checked and proper plaque control measures introduced/re-in forced. [10, 11, 12] The implant should be cleaned by instruments softer than titanium, such as polishing with a rubber cup and paste, floss, interdental brushes or using plastic scaling instruments. These have been shown not to roughen the implant surface unlike metal and ultrasonic scalers.

If there are pockets over 3mm the next question is *'Is there bone loss?'* Where there is bone loss there may be peri-implantitis. No bone loss may reflect a primary failure of the implant to integrate, submerged placement of the fixture, or unfavorable tissue morphology. If there is no bone loss, one should assess plaque and bleeding. An absence of both indicates no therapy is required. The presence of one or both indicates a need for oral hygiene instruction, local debridement and perhaps surgical resection to reduce the depth of the peri-implant pocket. Surgical resection is generally confined to implants placed in nonaesthetic sites. Probing depths of 4 or 5mm may be caused by tissue swelling and can often be corrected by improvement of peri-implant plaque control. [13, 14, 15] The presence of pus or pockets greater than 5mm indicates that additional measures may be required, including application of antiseptics, such as 2 per cent chlorhexidine or 3 per cent w/v hydrogen peroxide. In addition, local or systemic antibiotics may be considered. The decision for local or systemic antibiotics depends on the

distribution patterns of these pathogens, the periodontal conditions of the rest of the teeth and whether the implant problem is localized. [16, 17] Obviously a localized implant problem can be treated by local drug therapy.

Lang *et al*. suggest the following antibiotic regimes: systemic ornidazole 500mg bid for 10 days or metronidazole 250mg tid for 10 days or a once daily combination of metronidazole 500mg and amoxicillin 375mg for 10 days. Local application of antibiotics consisted of the insertion of 25 per cent tetracycline fibers for 10 days. Provided that mechanical and antiseptic protocols are followed prior to administering antibiotic therapy, it appears that periimplant infection may be successfully controlled using antibiotics.

When there is bone loss, the next question is *'How extensive is it?'* and can be divided into mild, moderate or severe. Mild bone loss may be treated by cleaning the implants, surgical resection, and topical antiseptic treatment, local or systemic antibiotics. Moderate bone loss indicates the same treatment for mild, but open debridement should be considered. This surgical approach is associated with recession with possible exposure of the neck of the implant fixture and consequent aesthetic problems. Bone grafting may be considered to fill the infrabony component of the periimplant bone defect. Lastly, advanced bone loss may indicate cleaning the implant, oral hygiene instruction, local and/or systemic antibiotic delivery, open debridement or explantation. If a decision has been made to remove the implant, explantation trephines are available to suit the implant system concerned. It should be noted that these trephines have an external diameter of up to 1.5mm greater than the diameter of the implant to be removed. Thus explantation may be associated with significant bone removal including buccal or lingual bone cortices, and damage to adjacent natural teeth where the inter-radicular space is limited. An alternative approach is to allow progressive bone loss from peri-implantitis to occur, resulting in sufficient bone loss to allow removal of the implant with extraction forceps.

Incomplete surface decontamination seems to be a major problem in implant maintenance. Titanium screw thread makes scaling difficult and the presence of the periopathogenic bacteria is associated with a poor response to guided tissue or bone regeneration. As a result, there is little evidence of true re-osseointegration in humans. [18, 19] However, there is early experimental evidence to suggest that re-osseointegration may be possible following appropriate decontamination procedures of sand-blasted and acid-etched implant surfaces.

If an implant does not respond to treatment, the evidence suggests that rather than trying to save the failing implant, it would be better to remove it and place another fixture once the site has healed.

CONCLUSION

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 remains obscure and undoubtedly further long term studies are necessary for clarification. Limited scientific evidence is available to endorse or recommend a specific modality for treatment and it seems that like periodontal disease, one regime may be successful in one patient and not another. New treatment modalities need to be evaluated using long-term randomized-

controlled studies to identify predictable and successful treatment of peri-implantitis.

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