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Peri-implantitis: A new dimensional Perspective

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ABSTRACT
The anatomy of the periodontal tissues of the tooth differs from that of a dental implant. The most significant contrast is in the absence of a periodontal tissue between the bone and an implant and its absence of a connective tissue attachment at the collar. These anatomical alterations may influence the inflammatory response of each to bacterial plaque. Similarity has been demonstrated between the bacterial plaque on implants and teeth. Still, the significance of the difference between plaques at healthy and diseased sites on implants has not been determined. The consequence of periodontitis is the loss of fibrous tissue attached to the tooth and the loss of supporting bone. A similar loss of supporting bone adjacent to the dental implant has also been observed. Healing and regeneration after periodontitis are better understood, but regeneration around implants remains controversial. The evidence for a distinct entity, 'peri-implantitis', and its treatments are discussed here.
Keywords: Peri-implantitis, Osseointegration, Tooth loss; Microbiology

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INTRODUCTION

By the end of this decade, it has been estimated that more than 2 million implants will be placed yearly. With the increasing awareness about dental implants among the general population and failures of tooth rehabilitative treatments like root canals, crowns, and bridges, the number of implants placed orally should rise further. The placement of implants is a standard protocol in the rehabilitation of the utterly edentulous mouth. The prosthetic abutments of dental implants, like their natural predecessor, penetrate the oral cavity of a mouth. The biofilm accumulation on the exposed implant surface happens in the same manner as in the natural teeth. The bacteria in the biofilm cause the peri-implant tissue to follow the same response as in periodontal tissues of the susceptible host [1-3]. Previous studies have only shown destructive pathology and lesions around the implants [4-6]. Peri-implants are diagnosed when aggressive pathology crosses the bone resorption threshold after an implant's Osseointegration [7]. Mainly the reasons include bacterial ingress (plaque theory, traumatic load loading theory), or both [8-10].

The pathology starts because the ingress of microorganisms causing inflammation of the areas surrounding the implant, which quickly reverses [11]. But, if not treated for a substantial quantity of time, the inflammation involves the bone, causing bone loss. For this reason, implants affected by Peri-implantitis may demonstrate little clinical mobility until the final stage of Peri-implantitis is reached. Among the microorganisms believed to cause Peri-implantitis, *Prevotella intermedia*, *Porphyromonas gingivalis,* and *Fusobacterium* sp. are currently the subject of numerous microbiologic studies [12]. It has long been thought that the organisms that cause periodontitis and peri-implantitis are the same, mostly in partially edentulous patients [13]. In contrast to plaque-induced Peri-implantitis, Peri implants caused by excessive traumatic load begin with microscopic fractures of the bone and can occur without any significant markers of inflammation in the starting phase [14].

Although it may be necessary that the aetiology and diagnosis of Peri-implantitis is the subject of scientific research, it is even more essential to find an optimum treatment. Ideally, treatment of peri-implantitis aims to restore conditions favourable to Osseointegration. To achieve this goal, the causes of Peri-implantitis must be eliminated, and then the restoration of the original peri-implant condition must be attempted. Conservative, Respective, and regenerative treatment measures have been described, depending on the kind and dimensions of the defective bone [15]. During the initial stage of Peri-implantitis, attempts must

be made to reduce the swelling by way of excellent oral hygiene carried out by the patient and simultaneous treatment with antibacterial and anti-inflammatory substances (e.g., chlorhexidine). Respective and regenerative treatment methods for advanced bone resorption require surgical intervention. The treatment aims to decontaminate the implant surface, remove granulation tissue, and level the bone defect (respective treatment) or restore the lost bone following surface decontamination using different methods (regenerative treatment). Few experimental studies [16] have examined the value of different treatment measures, and clinical experiences were published mainly as case reports [17]. Only two clinical studies reviewed the treatment results of a larger group of patients [18].

Tissues around the Implants:

The tissue around the complete Osseointegrated implant is like a natural tooth in functional and anatomical features. This biological barrier of 3-4mm" thus formed with regards to the central region of the integrated implant [18].

An inflammatory exudate is formed in the peri-implant region by the connective tissue due to plaque accumulation due to the ingress of the microorganisms on the surface of the titanium [18]. The infiltration is a localised host reaction to microbial ingress and spreads upward when the time for bacterial collection is extended. The fixture-tissue mucosal sheath is close to the gingiva on all sides of the teeth. The histochemical examination has shown the presence of inflammatory infiltrate, which results in practical genesis.

Periodontitis and tooth loss

Examinations done by epidemiologists have demonstrated that age affects periodontitis, with limited numbers of patients developing more aggressive forms of the latter. Studies have also reported that a low percentage of the adult community had severe periodontitis, which was not influenced by mouth-cleansing protocols [19].

Periodontitis has also been studied along with various other reasons for tooth loss for many years, and it has been found that Caries is the most common cause of tooth loss. In higher generations, however, tooth loss prevalence due to dental caries and periodontitis is almost the same. Therefore, it is safe to assume that those who got their implants done after 40 also exhibit an implant loss of 30-40% due to these conditions.

Microbiology of peri-implantitis

The role of Trans- mucosal abutment is to provide a surface for bacterial and microbial biofilms. In a distinct similarity to the crevice of the gingiva present on all sides, the peri-implant mucosa has also been found to be strongly associated with the implant. In patients with few teeth, the associated microfilm [20]. A Previous presence of pathological microorganisms is an element that can affect the prognosis of soft tissues surrounding implants in partially dentate patients [21] evaluated the subgingival flora by using phase contrast microscopy. The examiners found that the subgingival microflora on all sides of implants was associated with high amounts of microorganisms compared with teeth in the same mouth. Another study also used phase contrast microscopy to establish the prevalence of periodontal pathogens in partially dentate and edentulous patients with a history of periodontal disease [22]. The microbiological content was the same around teeth, and dental implants of similar pocket depth, which may identify the pockets around teeth serve as a haven for periodontal pathogens. This assessment was concurred upon in several studies on partially edentulous subjects [23]. After just one month after the placement of implants, periodontal pathogens were observed around the implants of partially dentate subjects [24]. Failures of Osseo integrated implants due to infection result from complicated peri-implant microbial organisms like advanced periodontitis. In entirely edentulous patients, we do not *find Actinobacillus Actinomycete mcomitans* and *Porphyromonas gingivalis* as frequently associated with periimplantitis as in dentulous subjects [4]. It was reported by Danser et al. that after complete extraction in patients with advanced periodontitis, *P. gingivalis* could no more be detected on the mucosal surface of edentulous patients [24]. Also, it was noted that *Actinomycete mcomitans* and *P. gingivalis* could not be found in the peri-implant pockets in the same patients after the insertion of implants [25].

Other bacterial species and gram-negative anaerobic rods are associated with peri-implant infections [26]. Organisms like *Staphylococcus*, *Enterococcus,* and *Candida*, which are not much associated with periodontitis, have also been associated with peri-implantitis [27]. Information taken from partially dentate persons with previous exposure to periodontal disease has shown no indication of any association between periodontitis and loss of attachment at implants after 36 months of function [28]. It has also been seen that periodontal pathogens are isolated from healthy periodontium and at sites with no pathology. Therefore, it is safe to conclude that microbes found in peri-implantitis are associated with periodontitis [29].

Periodontitis-Peri-implants relation

One of the most common reasons for adult tooth loss is periodontitis. Most patients receiving dental treatment are associated with a previous history of periodontitis. When planning to restore lost teeth with implants, it is essential to co-relate that the patient had been previously associated with periodontitis to the extent that will interfere with the Osseointegration and conservation of implants.

Studies have shown that the implant protocol-based treatment of periodontitis patients resulted in the loss of implants and implant-based fixtures [5,6]. Implant losses in some patients suggest that systemic conditions or other host_factors are primarily responsible for implant losses [30]; however, these losses are similar in patients associated with periodontitis and partially dentate patients [31].

Peri-implantitis: A Review

Peri-implantitis results in the degradation of associated hard tissues surrounding a working implant [32]. It is mostly “a site-specific infection yielding many features in common with chronic adult periodontitis" or "an inflammatory, bacterial-driven destruction of the implant-supporting apparatus” [4, 33]. It has been proved that microbes have a predominant role in causing Peri-implantitis, as has been proved by many clinical findings.

Mucositis is predominantly present in animals and humans [34]. During peri-implantitis, a complex microbiota is developed, like that observed in periodontal infections [4]. Peri-implantitis causes tissue breakdown due to plaque-retentive ligatures sub-marginally, finally causing a shift in the microbiota [35].

Rosenberg et al. [36] divided the implant patients into two groups infection and trauma. In the trauma group, it was found that patients felt no pain or discharges, and the microbiologic findings were mostly like that of healthy implant sites. In the infected group, microbial flora closely resembling the one found in periodontitis was found.

The histopathologic investigation of Peri-implantitis has been primarily done in monkeys and dogs [37]. In these studies, plaque formation was allowed to develop while ligatures were placed in a submarginal position around the neck of the implants. The ligatures were removed when the inflammatory response in the peri-implant tissues had resulted in an increased amount of bone loss. When Histological data was collected, it showed the presence of large inflammatory lesions in the surrounding peri-implant tissues extending to the alveolar bone. It was also pointed out by Lindhe et al. [38] that peri-implant tissues, when compared to periodontal tissues, have a reduced ability to heal advanced, plaque-related pathologies. Although minimal studies on peri-implant tissues at failed implant sites in humans are available, they show the presence of inflammation in the peri-implant tissue [39]. In contrast, other studies report that inflammatory cells were completely missing [40]. In a recent histopathologic study of Peri-implantitis [41], it was observed that soft tissue samples had large inflammatory cell infiltrates that extended to the apex of the pocket epithelium. Pathologies have been mostly filled up to about 60% by inflammatory cells, with plasma cells being the most predominant among them. The investigators also observed an unrestrained amount of polymorph nuclear cells in the connective tissues. Similar observations were also made in a study on the immunohistochemical characteristics of human peri-implantitis lesions [42]. This observation is also like findings of polymorph nuclear cells in a human crevicular fluid where implants have been placed and per-implants have occurred [43].

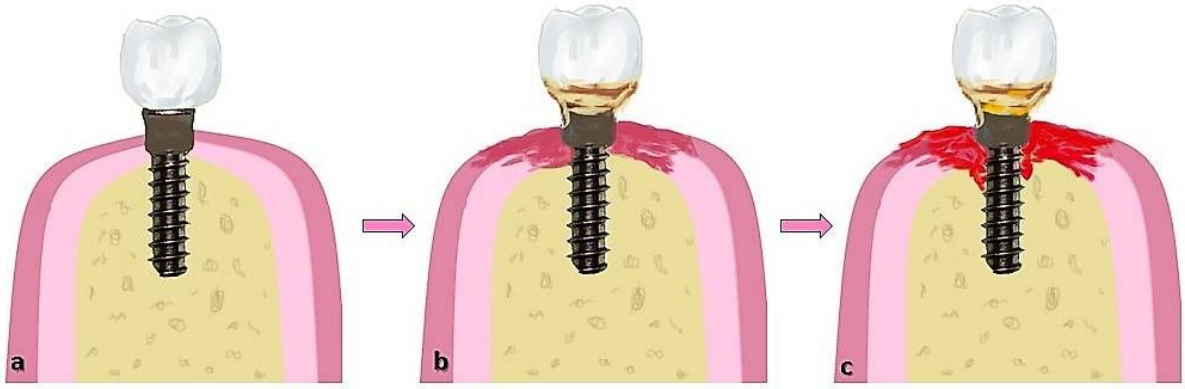

Figure 1. The sequence of development of peri-implantitis

Smoking

Many epidemiological surveys have exhibited the damaging results of smoking on the subject's periodontal state [44]. Its effect is one of the main factors for periodontal condition deterioration and has recently been confirmed [45]. Recent studies have been reminiscent of smokers' threefold rise in the risk of developing periodontitis [46]. There is a close connection between implant failures and smoking which has been

proven many times by several previous clinical studies [47]. In an analysis done by Bain and Moy [48] about the placement of 2194 implants in 540 subjects, it was reported that the most significant number of implant losses were associated with smokers. Gorman et al. [49] discovered implant losses were twice as prevalent in smokers as in nonsmokers at second-stage surgery. So, it can be safely deduced that smoking has a derogatory influence on implant longevity, most evident during the initial Osseointegration period of the implant. Smoking causes extensive marginal bone loss, primarily including more significant marginal bone loss of implants to restore edentulous mandibles [50]. Haas et al. [51] compared the link between smoking and peri-implantitis in 107 smokers to 314 nonsmokers. Smokers had more liability towards inflammation, deeper probing pocket depth, and more roentgenographic bone loss around implants than nonsmokers. Smoking affects Peri-implant tissue more in the maxilla than in the mandible.

Clinical aspects of peri-implantitis

Peri-implantitis and its associated inflammations are not found on routine and recall appointments. A Clinician should always stress methodical, clinical probing around teeth and implants during these checkup appointments. Sometimes it is difficult to assess the peri-implantitis over interfering bridgeworks over the implants properly. Still, this problem can be addressed by probing isolated areas around the implants as much as the prosthesis allows. Clinical findings can then be correlated with radiographs of the affected areas, and a probable diagnosis may be put forward. In peri-implantitis, the lesions occur around one or several implants. The roentgenographic aspect may resemble that of a disc, and the pathology mainly incorporates the wide margin of the implant.

The signs of peri-implantitis begin to show after many years, and the associated tissue loss is comparatively time-consuming. Approximately five years are required to appreciate and assess the destruction caused by peri-implantitis entirely. A proper maintenance protocol that includes supportive therapy and recall checkups is necessary for long-term implant survival.

Peri-implantitis treatment protocols

Several studies and evidence, basic treatment protocols of calculus & plaque control of the areas and flap access, if required, should be performed. It is important to educate about the essentiality of complete and thorough cleaning procedures, and the patient should be meticulously advised on the correct utilisation aids. Training of the patient must be done under professional supervision.

When a systematic review of the different treatments of peri-implantitis was done, it showed that many different treatment protocols were used [52]. All studies varied in the dosage protocol, test length, and time of the beginning of antibiotic therapy. 5-year results of peri-implantitis treatment in humans were shown by Leonhardt [53], in which implants with bone loss and suppuration from the sulci were included. Collection of Subgingival bacterial specimens were done for each individual and cultured. Implants were surgically exposed to the infection site. A susceptibility test was done, and systemic antibiotics were administered to control the target bacteria.

The developed treatment protocol showed satisfactory results during a follow-up study in only a few treated cases. Despite repeated treatment of Per-implants, it was found that 40% of the bone was lost in the advanced lesion of the implant region. New evidence points towards the support needed for the treatment of peri-implantitis infection. Why peri-implantitis causes excessive bone loss in some but relatively minor bone damage in others is relatively less understood. Wennstrom et al. [55], also found that patients who underwent periodontal therapy and were restored with implants showed slight bone loss but more severe in the maxilla than mandible over five years. Also, it was found that bone loss in smokers was more than that found in nonsmokers. It was found by Airila Manson et al. [56], in a study spanning many years, found that marginal bone loss was most pronounced in the maxillary molar area and especially pronounced in people with a smoking habit over time. More longitudinal studies are required to conclude how to treat peri-implantitis and complications arising thereof.

CONCLUSION

Peri-implantitis is most likely to occur in patients with a previous history of periodontitis. Although many different types of preventive treatment protocols have been shown, their advantageous clinical effects in humans are yet to be established.

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