
Review Article

Risk Indicators for Implant Therapy on Periodontitis

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Abstract: Periodontitis is one of the most common chronic inflammatory disease triggered by microbial dysbiosis and is considered to be the major cause of losing tooth in the adult. With the development of oral implantology, dental implant has become widely accepted alternative method for replacing poor prognosis teeth in patients with periodontitis. However, over the years, researchers have been concerned about the prognosis and clinical survival of patients with periodontitis after implant treatment. Studies have shown that individuals with previous tooth loss caused by periodontitis have an increased risk of loss implants, peri-implant marginal bone loss, and peri-implantitis. The aim of the present review was to evaluate the risk indicators for implant therapy on periodontitis.

Keywords: Periodontitis, Implant Therapy, Risk Indicators

1. Introduction

Periodontitis is one of the most common chronic inflammatory disease and is considered to be the major cause of losing tooth in the adult [1, 2]. Although the periodontitis can be triggered by microbial dysbiosis, the progression of periodontal disease and bone loss depends on the host inflammatory response [3-6]. With the development of oral implantology, implant therapy has become widely accepted alternative method for patients with periodontitis. Implant prostheses has advantage of not only good for retentive force, masticatory efficiency, better aesthetics, but also comfortable sense. Meantime, the use of dental implants for replacement of poor prognosis and missing teeth is also a significant option in the rehabilitation of periodontitis patients. However, several studies have shown that the success rate of implant therapy in patients with periodontitis is lower than that of periodontal

health patients [7-9]. In 2008, the 6th European Conference on Periodontology showed that patients with a history of periodontitis have a higher risk of implant survival [10]. In this review, risk indicators for implant therapy on periodontitis will be evaluated.

2. Dental Implant Survival in Patients with Periodontitis

High success rates of oral implantology have been demonstrated in many reports [11-15]. However, a series of risk indicators such as a history of periodontitis, poor oral hygiene cigarette smoking and diabetes may influence the prognosis and the long-term outcomes of dental implant [10]. For the past few years, lots of researches had certified that failure rates of implant therapy were higher in patients with periodontitis than in periodontally healthy patients. A

retrospective 5-year study from Hardt and his fellows found that the corresponding failure rate was 3.3% for the Non-periodontitis and 8.0% for the periodontitis patients [7]. Karoussis also compared the failure, success and complication rates between patients having lost their teeth due to periodontitis or other reasons from a 10-year prospective cohort study, which found that the failure rate was 9.5% in patient with a history of chronic periodontitis, while it was only 3.5% in patients without a history of periodontitis. And the survival rate for the group with a past history of chronic periodontitis was 90.5%, while for the group with no past history of periodontitis was 96.5% [8]. In the another 10-year prospective cohort study, it also found that the implant survival rate was 96.6%, 92.8% and 90% for the implants placed in periodontally healthy patients, in patients treated for moderate periodontitis periodontally compromised patients and in patients treated for severe periodontitis periodontally compromised patients respectively [16]. It is interesting that subjects treated for periodontitis can be rehabilitated successfully with osseointegrated implants. It can also receive good survival rate and success rate in a short time after implant restoration [17]. It was observed that survival functions by periodontal status seem to be similar to healthy periodontal patients. Until around 50 months, the hazard for implant failure was eight times greater for the severe chronic periodontal patients than healthy periodontal patients [18]. Therefore, periodontitis is the high risk indicator which affects long-term survival rate of implant. In a systematic review, Ong *et al.* concluded that there is some evidence that patients treated for periodontitis may experience higher bone loss and peri-implantitis than non-periodontitis patients, which affects long-term success rate of implant [19, 20]. Wu *et al.* used a 5-year retrospective study to show that factors such as implant length, site, and application of guided bone regeneration did not have an impact on the long-term success of the implants [21].

3. Peri-Implantitis

Peri-implantitis, where there are clinical signs of inflammatory response in the soft and hard tissue around the implant after osseointegration, may lead to complete failure of implants with loss of supporting bone [22]. Many researches had showed that patients with history of periodontitis had 4 to 10 times more chance of developing peri-implant disease than patients with healthy periodontal tissues [8, 9, 23-26]. In addition, 2.2% (3/13) implants placed in the periodontally untreated group presented peri-implantitis during the observation period, while 23.6% (13/55) placed in the periodontally treated group were found to be positive for peri-implantitis [23]. Therefore, it is suggested that individuals with a history of periodontitis that are treated with implant prosthesis have an increased risk to develop peri-implant disease. Some studies have identified similarities between periodontitis and peri-implantitis [22, 27, 28]. There is a correlation between them.

3.1. Periodontopathic Bacteria on Peri-Implantitis

Some scholars believe that infection by periodontopathic bacteria is a major cause of peri-implantitis [22]. They found that bacterial colonization occurs within 30 minutes after implant placement, then a climax community is reached within 2 weeks in periodontitis patients [29]. The community present at several months (6 ~ 12 months) heralds a stable community exists in the peri-implant crevice. Even though in the fully edentulous patients, the periodontopathic bacterial would not disappear immediately after tooth extraction. Fernandes *et al.* have even suggested that periodontal pathogens can persist in the oral cavity of edentulous subjects who have suffered periodontal disease for up to 1 year after extraction of all teeth and in the absence of other hard surfaces in the mouth [30]. Hence, some authors concluded that a greater abundance of periodontopathogens in implants placed in patients who previously had periodontitis [31-33]. Several studies continue to detect the source of periodontopathogens, and show that the periodontal pathogens *Actinobacillus actinomycetemcomitans* (Aa), *Prevotella intermedia* (Pi), *Porphyromonas gingivalis* (Pg), *Treponema denticola* (Td), *Tannerella forsythia* (Tf) and *Fusobacterium nucleatum* (Fn) can be found in the areas of the peri-implant disease [22, 33-35]. And the detection rates of for these periodontal pathogens in the subgingival samples from the implant sites were 28.5%, 61.9%, 33.3%, 23.8%, 47.6%, and 76.1%, respectively. The detection rates of these microorganisms were similar with natural teeth. In the subgingival samples from the natural teeth, the detection rates of for these periodontal pathogens were 36.5%, 47.6%, 39.7%, 28.6%, 34.9%, and 68.3%, respectively [22]. Further studies have shown that implants are quickly colonized by indigenous periodontal pathogens in partially dentate patients harboring periodontal lesions. The periodontal pathogens from periodontal pocket can divert and adhere to the implants continuously [7, 22, 36]. Therefore, it is believed that the adjacent teeth are the major source of the periodontal bacteria colonizing at an implant sulcus. Moreover, the detection of periodontal bacteria from the gingival crevices of occluding and contralateral teeth was not associated with colonization of implant sulci by these microorganisms [22].

Studies in humans and animals have demonstrated that a *de novo* biofilm formed on the surface of the implant initiates a host response involving the establishment of an inflammatory lesion in the peri-implant mucosa (peri-implant mucositis) [37-39]. This lesion is initially located in the connective tissue immediately lateral to the barrier epithelium, and in many respects, is similar to that which develops in the gingiva when plaque forms on adjacent tooth surfaces. In the continued presence of a sub marginal biofilm, the lesion in the marginal mucosa around implants may occasionally spread in an “apical” direction to involve the hard tissue, compromise osseointegration, cause varying degrees of marginal bone loss (peri-implantitis), and eventually cause the loss of the implant [40].

3.2. Immunological Factors on Peri-Implantitis

Not only periodontal pathogens are capable of causing disease. Local immune response, which is determined by the interaction between molecules and cells, can promote greater protection or susceptibility to several inflammatory and infectious diseases such as peri-implant disease [24]. In periodontitis, a localized inflammatory response to bacterial infection activates the innate immune system, resulting in the release of a range of cytokines and other mediators and propagation of inflammation through the gingival tissues [41, 42].

However, is there a similar mechanism in dental implants? The relationship between biochemical markers of inflammation and clinical parameters around implants has attracted the attention of scholars. In the presence of periodontal pathogens, the immune response may trigger a higher or lower levels of cytokine production and interfere with its physiological role [43]. In chronic periodontitis, the pro-inflammatory immune response is highly correlated with bone loss.

Benefit from the current advance in human genome sequencing, and machine learning algorithm, the contribution of host inflammatory response to periodontitis has been further emphasized in a recent clinical study.

By clustering analysis of gingival tissue transcriptomes and clinical parameters in 120 patients, Kebschull et al. divided patients with periodontitis into two groups according to their gene expression characteristics, and found that there was significant difference in whole-mouth periodontal destruction, and subgingival microbial burden [44]. This finding translates different host inflammatory response (gene expression signatures) in pathologic gingival tissues into phenotypic differences and may provide hints for future classification of periodontitis. Therefore, even in patients with edentulous jaws and those under maintenance of periodontal and peri-implant health that are considered to be inactive infection, their intrinsic pattern of individual inflammation may also trigger peri-implant bone loss, as it occurs in periodontitis [24].

Despite the similarities in clinical features and etiology of peri-implantitis and periodontitis, the resistance and repair capacity are remarkable difference between lesions present in the periodontium and in the peri-implant tissue in histopathology [24, 45]. When comparing cytokine production, the implant sulcus showed a higher inflammatory state than periodontium with a bacterial challenge that seems to be similar [46].

The apical extension of inflammatory cell infiltration is more pronounced in peri-implantitis than in periodontitis, and in most cases is located at the top of the pocket epithelium. In both types of lesions, plasma cells and lymphocytes predominate in cells, whereas in implant inflammation, the proportion of neutrophils and macrophages is high. The apical extension of the inflammatory cell infiltration is more pronounced in peri-implantitis than in periodontitis and in most cases is located at the top of the pocket epithelium. Plasma cells and lymphocytes dominate in two types of

lesions, whereas neutrophil granulocytes and macrophages occurred in larger proportions in peri-implantitis [45]. Subsequently, histological comparison studies reveal that peri-implantitis lesions contained significantly higher densities of CD138, CD68, and MPO positive cells, at least twice as large as periodontitis lesions. These lesions extended apical of the pocket epithelium and were not "encapsulated" by non-infiltrated connective tissue, which presented with significantly denser vasculature than periodontitis [47]. It's demonstrate that periodontitis and peri-implantitis lesions primarily differ in wound cell-to-cell adhesion, healing, complement activation and other innate immune responses by a meta-transcriptomic comparison of the two lesions [48]. The studies above show that the peri-implant mucosa seems to be less capable of resolving lesions in comparison with the gingiva of natural teeth.

4. Bone Loss Around the Implant

Partial non-dental patients with periodontitis can successfully repair with osteosynthesis implants. However, the long-term study show that the bone and attachment loss at the implants are higher than in periodontally healthy subjects [17]. Furthermore, a retrospective 5-year study show that 64% of the periodontally compromised patients have a mean bone loss at the implants of >2 mm, while only 24% on the periodontally healthy patients [7]. Karoussis et al used a 10-year prospective cohort study to compare bone loss in implants placed in periodontitis patients and periodontally health patients after 10 years. This study showed that 15.1% of severe periodontitis patients, 11.2% of moderate teeth periodontitis patients, 4.7% of periodontal health bone loss \geq 3mm [8]. Implant bone loss in patients with periodontitis is higher than that in periodontally health patients.

Physiological bone resorption exists in the implant under normal conditions. In 1986, Albrektsson et al. proposed criteria for the definition of implant success. Bone resorption of 1 mm during the first year and of 0.2 mm/year after the first year of loading was defined as physiologic bone remodeling [49]. It is normal for the implant to have a bone loss of less than 2.8 mm after 10 years of implant placement. However, in a 10-year prospective cohort study, the long-term cohort study found that individual bone loss ranged from 2.7 - 4.0 mm in patients with periodontitis [17]. Among them, the bone loss rate of implants in patients with periodontitis was 2.07 mm after the first year of implantation, and 1.3 mm in the subsequent 9 years, while, bone loss of periodontal healthy patients subjects was only 1.13 mm in the first year after insertion of the superstructure and 0.11 mm in the subsequent 9 years [17]. According to the physiological bone resorption criteria described above, it is considered pathological bone resorption when the bone loss is \geq 3 mm. Matarasso et al. compared the 10-year rate of bone loss around the implant between patients with impaired implants and periodontally health and found that there was a significant difference in bone resorption rates between them [50]. According to the Nobel Biocare dental implant system, the mean bone loss in patients

with periodontal lesions and periodontally health were 3.34 ± 0.41 mm and 2.41 ± 0.41 mm, respectively. Furthermore, according to the Straumann Dental Implant System, the average bone loss was 3.38 ± 0.35 mm and 2.54 ± 0.36 mm in periodontally compromised patients and periodontally health patients. The number of sites with bone loss ≥ 3 mm was more than four times higher in periodontally compromised patients compared with periodontally healthy patients for both implant types [50].

In addition, the bone mass at the site of implantation in patients with periodontitis and periodontal health needs to be assessed. For bone mass, the proportion of patients with periodontitis (Grade D) was higher than that of non-periodontitis (20% vs. 0%), and patients with Grade A or B (20% vs. 48%) periodontitis [7]. Periodontal patients often have bone loss, which may increase the difficulty of implant placement and the unpredictability of long-term outcomes. In patients with periodontal disease, bone loss around the implant is more likely to occur, which will affect long-term success rates and is a potential risk of implant failure.

5. Conclusion

In patients with periodontitis, the risk of implant failure may increase the pathological features of periodontitis. Therefore, the use of dental implants to repair periodontitis patients is more challenging. First, although periodontal treatment may successfully control periodontal infection, it does not improve the host immune response. Second, patients with periodontal disease often have bone loss, which may affect the choice of implant diameter and location. Third, it may lead to the development of infections around the implant, that is, the periodontal microflora transplanted from the teeth to the implant. Finally, it is generally believed that patients who maintain poor periodontal lesions have a higher risk of periodontal treatment and recurrence of periodontal disease.

For the above analysis, the use of dental implants to repair periodontitis patients and the greater risk of implant loss. Therefore, periodontal treatment should be performed before implant surgery. For example, it is essential to perform procedures such as removing areas of microbial retention and supra and subgingival scaling, and provide oral hygiene guidance. Furthermore, it may be advisable to perform procedures with the least stress possible to the bone and gingival tissues, and to have a wider zone of attached gingiva around implants in patients with severe periodontitis. It has been shown that it is possible to reduce or eliminate any rehabilitation with implant-supported dentures that retain plaque or occlusal overload. Finally, these patients need to maintain a rigorous maintenance program and shorten the assessment interval to increase the success rate of dental implants.

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Conflicts of Interest

The authors have no conflicts of interest to declare.

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