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Review Article

The Role of Etiologic Factors Causing Peri-Implantitis; A Current Update

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Abstract

In recent years, dental implant applications have become more frequent in order to treat both aesthetic and functional disorders caused by tooth loss. Dental implants are frequently used in fixed and partial implant supported prostheses. Despite the high success rates, some of the implants are also failing. Peri-implantitis is bone loss due to infections around the implant. Peri-implant disease progress quietly without pain and is often diagnosed when marginal bone loss emerge.

Risk factors for peri-implantitis include periodontitis, dental plaque, bad oral hygiene, smoking, alcohol consumption and diabetes mellitus. Radiographically, marginal bone loss is evaluated on periapical films, bleeding and pocket depth on probing is evaluated in clinical examination. For the prevention of such risk factors proper design of implants preserve periodontal tissues' around of implants from peri-implantitis' destructive effects should be performed in conscious individuals who do not have any systemic discomfort with a healthy periodantal tissue under appropriate conditions. In addition, the patient should be informed about oral hygiene protocols and prosthesis cleaning and should be regularly checked at certain sites.

The purpose of this literature is to provide current information about the risk factors of peri-implantitis

Keywords: Peri-Implantitis; Dental Implants; Risk Factor; Prevalence

Introduction

In recent years, dental implant applications have become more frequent in order to treat both aesthetic and functional disorders caused by tooth loss. However, even if the implants can retain their presence in the mouth for a long time, the majority of the implants experience implant-related diseases [1-3]. In the literature review, peri-implant diseases are divided into two groups: Peri-implant mucositis and peri-implantitis. Both of them are associated with an inflammatory reaction in the peri-implant tissues. Peri-implant mucositis is limited to soft tissues encircling a dental implant that does not contain a supporting bone [4] while periimplantitis was defined as the inflammatory reactions associated with supportive bone loss around a functioning implant in the first European applied course of Periodontology in 1994. This definition has been refined at various reconciliation sessions, promoting a progressive bone loss pattern following reshaping of the initial bone [5]. Peri-implantitis occurs from incompatibility between host defense and increased bacterial [6]. In recent reconciliation meetings, periimplantitis has been associated with multifactorial etiology with the inclusion of implant-related factors (biological, biomechanical), clinician factors and patient factors (systemic disease), periimplant pathology [7]. Peri-implant disease improve quietly without pain and is often diagnosed when marginal bone loss emerge. Trigger determinants for periimplant bone loss generally split in two main categories: biological factors and biomechanical factors [8]. Biological failures include progressive bone loss, bacterial infections, microbial plaque subsidence and sensory corruptions [9,10]. Biological complications are divided into two groups as early biological failures and late implant failures; early failures are not applied to appropriate aseptic measures of the surgical implant [10,11], late complications are typically infections caused by periimplantitis and bacterial plaque [6,12]. Periimplantitis due to biomechanical factors are: Prosthesis-related Factors (Occlusal overload, residual cement, inadequate prosthetic placement), inappropriate abutment angle and bruxism are the cause of exceeded forces due to parafunctional habits [13,14].

The diagnosis of peri-implantitis involves the use of deepening depth measurement, hemorrhage bleeding values and radiographic evaluation. The increase in pocket depth is an indicator of attachment and bone loss. Intraoral radiographs are used to assess marginal bone loss. Interproximal bone loss on intraoral periapical radiographs taken with a parallel technique should be assessed at a determined reference point [15].

In this study, it is intended to evaluate the etiologic factors and frequency that cause fairy implantation in approximately 60 articles.

Etiologic Factors

Peri-implantitis is a consequence of impaired balance between bacterial and host response following failure of osseointegration of the implant with bone [16]. Reviewrs [17] suggested that poor oral hygiene, smoking, periodontitis history, diabetes mellitus, genetics, alcohol consumption, related factors for prosthetic and implant surface characteristics may be potential risk factors for developing peri-implantitis.

Poor oral hygiene

Individuals with poor oral hygiene are exposed to periodontal diseases are also a risk factor for periimplantitis. The most important risk factor for Peri-implantitis is poor plaque control. This may reflect a patient's inadequacy or reluctance to maintain optimal oral hygiene. Other obstacles may include prosthetic design, adjacent restoration contours, margins and broken restorative components [18].

Prospective works have shown that individuals with generalized aggressive periodontitis are more sensitive to peri-implantitis [19].

Smoking

Smoking has been associated with a long duration of periimplantitis scores and continues to be reported in literature as a potential risk factor for the survival of osseointegrated implants. At the literature to assess whether treated periodontitis and smoking cues could be considered as risk factors for adverse outcomes in dental implants, either alone or in combination. Cigarette was accepted as an important risk for periimplantitis formation in 3 of 4 systematic complements [17,20]. Although the majority of studies report high implant survival rates ranging from 80% to 96% in smokers, the implant survival rate is statistically lower than in those who do not use cigarettes in most studies. The literature, as mentioned above, presented highly controversial reports on the effect of cigarette on periimplantitis, despite the evidence. The incidence of peri-implantitis in smokers is between 3.6 and 4.6 percent. A meta-analysis of 13 trials found that cigarette smoking increased the bone loss rate around implants by 0.164 mm/year. Baig and Rajan reported that in smokers significantly more marginal bone loss after placement and higher Peri-Implantitis percentages [20,21].

Periodontitis history

The incidence of implant failure was significantly higher in partial edentulous patients than in total edentulous patients [22]. Renvert., et al. reported that although peri-implantitis is not associated with partial edentulous or total edentulous, periodontal disease is a critical adventure for peri-implantitis [23]. İndividuals with a history of chronic periodontitis show a higher prevalence of peri-implantitis. It also represent that individuals with periodontitis are inclined to peri-implantitis when marginal bone loss around the peri implant is ≥ 2 mm [24]. Similarly, Renvert., *et al*. have found that treated periodontal patients have greater risk for peri-implantitis infections than people who have not had periodontal disease before [25]. In the periodontal space are probably deposits of microorganisms that colonize the implant surfaces, since the pathogenic flora in perimplantitis is similar to those found in periodontitis [26,27].

Diabetes mellitus

As the periodontitis is more common in diabetic patients, glycemic control is also related with peri-implant disease [28]. Although the role of distinct physiological mediators in pathogenesis is not fully understood, evidence suggests that proinflammatory gene expression in peri-implantitis regions is affected by glycemic control [29]. Ferreira., *et al.* exhibited patients with diabetes mellitus are more tendency to develop peri-implant than non-diabetic patients also the risk of diabetes and the increased risk of peri-implantitis were statistically related [30]. In diabetics, poor metabolic control has been shown to provide a more favorable environment for infection and loss of implants [30].

Related Factors for Prosthetics

Although there are no published randomized clinical trials that demonstrates that the direct-crown design is linked to peri-implantitis, it has long been established that insufficient subgingival margins of the crown alter microflora and cause inflammation around the natural teeth. To minimize the likelihood of peri-implant disease, it is envisaged that the same principles will be applied when the prosthetic design of dental implants are considered [31,32].

Occlusal overload

One of the major causes for the loss of implants is overload, which causes peri-implantitis. The factors related with occlusal overload are probably related to location of the implant, the significant deviation of the implantation force axis from the long axis of the implant, and the incompatibility of the implant diameter/length, probably due to an excessively large prosthesis in the posterior region [10,33]. Occlusal overload can cause bone resorption around the osteointegrated implants. Occlusal trauma with peri-implantitis may accelerate bone destruction. After a while, the anaerobic environment formed in the periimplantitis region changes the rates of normal bacteria. Therefore, a microbial flora that changes with excessive occlusal loading causes an increase in marginal bone resorption. Occlusal corrections should be made to stop progressive bone destruction. Occlusal loading controlled by bone density may improve bone loss due to periimplantitis [34-36].

Residual cement

The effect of flooding cement on peri-implantitis formation is similar to that of dental calculus in periodontal disease. The rough surface of the cement makes it difficult to remove microorganisms and this causes peri-implant mucositis initially and peri-implantitis resulting in bone loss later [37]. The remaining cement after cementation of the prosthesis is related with clinical and radiographic findings of peri-implantitis. Another cause of peri-implant diseases is peri-implant tissues have different morphology from the natural teeth [38]. The periodontal ligaments around the natural teeth are more resistant to occlusal forces due to their viscoelastic structure. However, relation of the implant with the surrounding bone causes occlusal forces to be transmitted to the surrounding bone. In addition, absence of periodontal ligaments around the implant and elongation of the connective fibers parallel to the implant surface reduce the pressure resistance of the peri-implant tissues. Cause of force applied during the cementation, the cement is pushed into the deep tissues and the cementum does not burst of the sulcus, making it difficult to clear the cementum surplus. Linkevicius., et al. stated that the cement can no longer be detected in the radiograph. Wadhwai noted that radiographic imaging of the cement after implant prosthesis cementation is poor [3,37,39,40].

Factors associated with implants

Implant design

Design of an implant affects the tissues around the implant. Cause of crestal bone loss after implant surgery is bacterial accumulation that occurs in the gap between implant and abutment. Therefore; factors such as implant abutment, platform switching concept, and surface roughness can determine amount of bone loss. Comparison of standard platform implants, it has been reported that there are less bone loss in implants when the design is platform switch. However, in some studies it is stated that there is no meaningful difference in bone loss between two platform designs [20,41].

The biocompatibility of titanium is attributed to its surface properties. Also, it is stated that surface free energy and especially surface roughness is a significant effect on plaque formation. Exposing of rough implant surfaces into the oral cavity provokes an environment that leads to plaque build-up [42]. Roughening of surface improves environment for adhesion and cleaning of these surfaces are difficult. This causes growing plaque rapidly and irreversibly. For this reason, it is recommended that implants have an average surface roughness of 0.2 micrometers. In case implants groove with rough surface are exposed; these surfaces need to be smoothened and polished to prevent plaque buildup [20].

Implant placement

In the long run, thickness of bone in buccal region of implants should be at least 2 mm in anterior region and at least 1 mm in posterior region in order to reduce soft and hard tissue loss. The fact that implants are placed in an excessive buccal position and loss of tissue enough to require application of pink porcelain is among other things that may affect peri-implant diseases. Trullenque-Eriksson and Guisado-Moya. stated that one of the most major factors affecting implant success is quality of bone in region where implant is placed [43].

Parafunctional Habits (Bruxism – Malocclusion)

Bruxism is disorder of a chewing system, which is expressed by stress, anxiety and tense situations, or rubbing, creaking and tightening teeth during normal activity and sleep [44].

ligament is an essential part to make a relation between natural tooth and bone. The most important characteristic of periodontal ligament is shock absorption. There is no periodontal ligament between implant and bone. For this reason, all the loads on implant are transmitted to bone directly, which may impair the relationship between bone and implant [44-46].

In 9 studies, a total of 761 patients were evaluated biological complications for 2511 implants. In 3 studies, implant failure was assessed according to marginal bone loss. As a result of the studies, there was a clear evidence in 3 studies to evaluate bruxism as a risk factor for implant failure; in the remaining 6, there was no relationship between bruxism and implant loss. As a result, the presence of bruxism is likely to be a risk factor for mechanical complications in implant periphery, but it is unlikely to be a potential risk factor for biological complications [47,48].

Alcohol consumption

Alcohol consumption can cause periimplantitis as well as indirectly caused by periodontitis. One of the reasons for the lack of vitamin K is the frequent consumption of alcohol. In healthy individuals, prothrombin production is normal, but alcohol can break the prothrombin repletion and thus reduce it, thus affecting the coagulation mechanisms [49]. Furthermore, contents found in alcoholic beverages such as a mixture of toxic alcohol, nitrosamines, and ethanol can also cause osteoclasis, as well as inhibit bone stimulation. Studies conducted by some researchers have reported that drinking more than 10 grams of alcohol a day causes more peri-implantitis than cigarette smoking [50,51].

Genetic factor

A study by some researchers found that the areas of marginal bone loss around the implant were very seriously increased in the concentration of blood microspheres, but showed that the expression of the vascular endothelial growth factor was low. For this reason, VEGF may play a protective role in marginal bone loss, that is, periimplantitis [39]. A genetic disorder characterized by interleukin 1 gene polymorphism is suggested as a risk factor for peri-implantitis. A study showed that the IL-1 genotype was a risk factor for peri-implant diseases and a synergistic effect between IL-1 genotype and cigarette. In the other two studies, they concluded that there was no relationship [52].

Prevalence of Peri-implantitis

Despite the success of implants and their high survival rate, the number of individuals with peri-implantation is increasing [4,53]. However, since the standardized diagnosis criterion is not used in the diagnosis of peri-implantitis, the prevalence values also show differences in the study. In a study conducted by Ferreira., *et al.* [30] 578 implants in 212 patients, the peri-implantitis prevalence was found to be 8.9%. Koldsland., *et al.* [53] reported that the prevalence of peri-implantitis in the study of 109 patients in an average of 8.4 years was between 11% and 47%. Mir-Mari., *et al.* [54] they found that 16% of patients had peri-implantitis in a study in which they examined implants that were functioning for an average of 6 years. Renvert., *et al.* [55] Reported the incidence of peri-implantitis as 14.9%. Zitzmann., et al. [56] Reported that between 28% and 56% of peri-implantitis was observed. Atieh., *et al.* [57] Reported that peri implantitis was 19%. Konstantinidis and colleagues [58] found that the rate of peri-implantitis for 5 years was 13.3% for patients. Daubert and colleagues [4] found that periimplantitis rate was 26% on patient basis in 2015. Schwarz and colleagues [59] reported a periimplantitis rate of 7.6% in their study of 512 implants. Cavalli., *et al.* [60] a total of 336 implants in 69 patients with fixed implant prosthesis restoration were followed for an average of 5 years under supportive therapy every year for the first 2 years after every 6 months and found a prevalence of peri-implantitis around the implants was 3.81%. Constantinidis., *et al.* [58] in their study of 597 implants in an average of 5.5 years in 186 patients, they found prevalence of peri-implantitis as 12.9%. In a study of 200 average age 12.2 ± 52.81 53.52 ± 36.76 months in average 60% of the total 655 tissue around the implant within the patient is determined as function of peri-implantitisl in Turkey [61]. They reported that the likelihood of peri-implantitis was 3.2 times higher in individuals aged 60 years and older, and 3 times higher in implants with 5 years and longer function [61]. This suggests that the knowledge of dentists to prevent peri-implantitis should be increased. However, it is also necessary for patients to undergo regular dental checkups within the period of supportive periodontal treatment and to ensure maximum oral hygiene levels.

Discussion

In this article, the effect of etiologic factors causing periimplantitis is evaluated. Peri-implantitis is the most used criterion for assessing the success of osseointegrated implants. Implants are considered to be successful in the presence of marginal bone loss of lesser than 1 mm in the 12 month and less than 0.2 mm in the following years. Long-term peri-implant bone loss; periodontal disease history, excessive occlusal loads, systemic factors and poor oral hygiene are considered. However, short-term peri-implant diseases, such as a few months, occur in simulated cases of implant restorations where the peri-implant tissue cement can not be completely cleaned. Furthermore, age can be considered a risk indicator since many systemic diseases are chronic and more common in elderly individuals and can directly or indirectly impair implant health. Ferreira., *et al.* reviewed 212 partial toothless study groups rehabilitated with osseointegrated implants. The authors concluded that for elderly individuals (> 45 years) the prevalence of peri-implantitis was slightly higher. Age has also been described as a contributing factor to the development of peri-implantitis when associated with periodontitis and cardiovascular disease history [62,63].

As indicated, the access to dental implant sites is limited by oral cleaning devices such as brushes or dental floss, which causes inadequate oral hygiene and insufficient plaque control and peri-implantation.

One of the major causes of tooth loss in adults is the loss of bone at an advanced level due to periodontitis [64]. It is reasonable to suppose that patients with periimplantitis are also patients with a periodontal disease story. Various studies have been reported on the passage of periodontal pathogens from the teeth into the implants [65]. Renvert., et al. reported that although peri-implantitis is not associated with partial edentulous or total edentulous, periodontitis is a critical risk for peri-implantitis [23]. Patients with a history of chronic periodontitis show a higher prevalence of peri-implantitis. However, the literature appear to existing no controversy over the negative impact of poor periodontal conditions on implant success although it is also known that auxiliary periodontal programs can increase the rate of success for dental implants even in patients with periodontal disease history except there is no history of aggressive periodontitis [17].

In addition they emphasized that cigarette smoking negatively affects bone mineral density and wound healing [66]. In some studies they have come to the conclusion that cigarette has created a negative effect on osseointegration [67]. Although some researchers have previously found a correlation between smoking and peri-implantitis prevalence as a result of their work, it has recently been reported that there is controversial evidence of the negative effect of smoking on peri-implantitis prevalence [2,17].

Although the relationship between peri-implantitis and platform switching has not been investigated, it has been shown that platform-key implants lead to a lower crestal bone loss in proportion to normal implants. Cappiello and colleagues showed that bone loss around implant was 1.3 to 2.1 mm for control implants and 0.6 to 1.2 mm for the switching platform implant for 12 months [19].

Moreover, many studies have shown that systemic diseases are a risk factor for the development of peri-implantitis. Implant survival rates have been reported in people with diabetes mellitus. On the other hand, well-controlled diabetes has also been shown to be a contraindication to implant therapy. For this reason, it is arguable that patient awareness and glycemic control should be taken into consideration when implant therapy is needed to achieve a definite conclusion that the prevalence of peri-implantitis is higher for individuals with diabetes mellitus. Ferreira., et al. exhibited patients with diabetes mellitus are more tendency to develop peri-implant than non-diabetic patients. Genetic characteristics may be a risk factor for dental implant therapy. However, since no consensus on a systematic oversight is verified, this relationship should be investigated in future studies [68,69].

Occlusal overloading on the implant can lead to marginal bone loss [70]. Microfracture inflammatory phenomena cause bone defect without involvement. However, marginal bone loss has not been detected in implants with occlusal load in experimental studies performed by Hurzeler and colleagues on other living beings other than humans [71]. Miyata., *et al.* also showed that occlusal load with a height of 100um in other living beings did not provoke bone loss in implants with healthy marginal gingiva. Bone loss was significant after induction of inflammation [72]. Despite the absence of periodontal inflammation earlier, 180um or more peri-implant appeared early in bone resorption. This indicates that occlusal overload can impair the health balance of periodontal tissues and reduce the magnitude of overload required to provoke bone loss in previous gingival inflammation. In the compilation we made, peripheral overload was the main factor associated with peri-implant [73].

Conclusions

The risks of peri-implantitis, such as smoking, alcohol consumption, diabetes and unsure but genetic features, prosthetic factors, implant design and parafunctional habits, are all shown as biological and mechanical factors that can influence the patient's inflammatory reaction and infection response host response.

As a result; the main effect of Peri-implant diseases is microbial plaque. However, it may play a role in the etiology of peri-implantitis in biological and mechanical factors and may have an increased effect on plaque involvement. For this reason, proper design of implants preserve periodontal tissues around of implants from peri-implantitis should be performed in conscious individuals who do not have any systemic discomfort with a healthy periodantal tissue under appropriate conditions. In addition, the patient should be informed about oral hygiene protocols and prosthesis cleaning and should be regularly checked at dental examinations and in dental education faculties.

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Volume 2 Issue 8 August 2018