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Review Article

Risk factors of Peri-implantitis: A comprehensive review

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ABSTRACT:
Peri-implant diseases are inflammatory in origin and affect the tissues around dental implants. They are of two types: peri-implant mucositis and peri-implantitis. In peri-implant mucositis, there is no loss of marginal bone as inflammation is restricted to the peri-implant tissue whereas peri-implant mucositis is reversible by managing it in earlier stages and eliminating the causes [1,2]. Literature postulates numerous risk factors of for peri-implant pathologies. In the present review, we aim to summarize risk factors pertaining to peri-implantitis.
Key words: Peri-implantitis, Peri-implant mucositis, dental implants.

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INTRODUCTION

The dental implant has transfigured oral rehabilitation and has become an important part of routine management in prosthetic rehabilitation. The advancement in implant design, materials used, and surgical protocols has increased over time. As estimated, ninety percent of patients who received an implant did not face any problem with their ability of chewing and were completely satisfied with their convenience methods of plaque control at the implant sites [3]. Complications due to peri-implant diseases are recurrent even after long-term survival rates. The loss of the implants and their prostheses are proven to be the most severe long-term complications [4].

Peri-implant diseases are inflammatory in origin and it affects the tissues around the implants. They are of two types: peri-implant mucositis and peri-implantitis. In peri-implant mucositis, there is no loss of marginal bone as inflammation is restricted to the peri-implant tissue and is reversible by managing it by eliminating the causes [1]. Peri-implantitis is an inflammation of the peri-implant mucosa with the marginal bone loss[2]. The prevalence rate was high in both the types according to a systematic review and meta-analysis by Lee et al 2017 [5]. The prevalence of implant-based peri-implant mucositis was 29.50% and the prevalence of subject-based peri-implant mucositis was 46.80%. Whereas, the prevalence rate of implant-based and subject-based peri-implantitis was estimated as 9.25% and 19.84%. [5]

DEFINITION

As comparing to gingivitis and periodontitis which affects the periodontium of natural teeth, the terms mucositis and peri-implantitis is stated as an inflammation of the soft and hard tissues and destruction of the same surrounding the dental implants. Thereby, transitions are not clinically clearly distinguishable. Mucositis is an inflammatory process of the peri-implant soft tissue with bacterial invasion resulting in redness, swelling and bleeding on probing [1,6]. These clinical features are sometimes not clearly visible. Moreover, bleeding on probing (BOP) might be considered as an indicator for peri-implant disease, but according to the predictive value of BOP there is no evidence available to prove[2,6–9].

In comparison to mucositis, peri-implantitis is described as a progressive, irreversible pathology of implant-surrounding hard and soft tissues with bone resorption, enhanced pocket and pus formation[8]. Bleeding on probing, bone loss and deep probing depths can also occur but they can be due to other factors and not only because of inflammation, e.g. implant insertion position [8]. The peri-implant soft and hard tissues can be affected by implant type and shape, type of connection, abutment material and the

type and the material used in prosthetic suprastructure [6]. Human saliva markers such as osteocalcin, tartrate-resistant acid phosphatase (TRAP), dickkopf-related protein-1 (DKK-1), osteoprotegerin (OPG) and cathepsin K (CatK) alone cannot differentiate peri-implantitis from other inflammatory periodontal processes [7].

PERIODONTAL DISEASE AND MICROBIOLOGICAL ASPECTS

The diagnosis of periodontal pathology is considered as the most researched factor associated with peri-implantitis. This is because of the similarities in the subgingival microbiota between the diseased teeth and implants [10]. Certain periodontal pathogens which are present submucosally has been associated with peri-implantitis[11]. Current studies suggest that peri-implantitis is associated with a specific microbiota which resembles that of periodontal lesions but all other microorganisms are not commonly related to periodontitis [12]. However, it is well-understood that peri-implantitis has a rich diversity of microbes, and the deepened peri-implant pockets manifests significant microbial alterations and greater levels of dysbiosis [10–13].

The adjacent teeth suffering from periodontitis may be considered as a predictor of future peri-implantitis. Periodontal pathologies have been strongly associated with peri-implantitis [14]. The risk of developing peri-implantitis is twice in periodontally compromised patients as compared with periodontally healthy individuals [11]. A long-term cohort study showed that subjects with Generalized Aggressive Periodontitis are five times more at a risk of implant failure, and fourteen times more susceptible to peri-implantitis as compared to the healthy population[15]. The risk of peri-implantitis can be lowered by treating the periodontal diseases properly prior to the implant placement which has proven to be beneficial, periodontal treatment and referrals to periodontists when necessary are considered an important initial part of the whole treatment plan[10–13].

LACK OF MAINTENANCE THERAPY

To lower the risk of peri-implant biological complications, supportive therapy has been proven to be beneficial with a minimum recall period of six months. Maintenance programs should be modified according to the specific needs of the individuals and sensitivity to both periodontal and peri-implant pathologies. Risk assessment factors are- the BOP percentage, the prevalence of active residual pockets, level of oral hygiene, habit of smoking and the presence of systemic or genetic conditions. Four annual visits are recommended in high-risk profile individuals as for the prevention and early detection of peri-implantitis [12]. Unfortunately, the least compliant patients are the ones with greater needs. The extent and severity of periodontal lesions as well as the patient's smoking habits affect cohesion to maintenance programs. Therefore, the importance of regular supportive therapy for the prevention of peri-implantitis should be informed to the patients by their clinicians[12,16,17].

SMOKING INCLUDING CIGARETTES, WATER PIPES, SMOKELESS TOBACCO, VAPING AND CANNABIS

Smoking has a negative impact on periodontal health and which is well known. It affects innate and adaptive immune responses, impairs the defense mechanisms of host and its response to microbial challenges [18]. Cigarette smoking is detrimental to periodontal treatment as it affects wound healing. Smoking causes alterations in microbial flora and increases the oxidative stress and inflammatory burden [19]. It significantly affects colonization of implants with periodontal pathogens such as *Porphyromonasgingivalis* (Pg) and *Fusobacterium nucleatum*. Cigarettes are not only harmful to smokers but also the exposure to environmental smoke increases the risk of developing periodontal complications by approximately thirty percent[20].

According to studies, smoking is a proven risk predictor for peri-implantitis. Smokers are considered to be two times more at risk of developing peri-implantitis in comparison to the non-smokers; Moreover, there is a dose-dependent relationship between smoking and tissue destruction as smoking increases the severity of peri-implantitis lesions [21]. Smoking cessation has favorable effects on both incidence and progression of the disease and leads to the positive impact on periodontal health [18–20]

In addition to marketing of cigarettes, the popularity of non-cigarette tobacco products has been remarkably rising. Nowadays, a popular form of smoking tobacco among adolescents and adults is water pipes which is also known as shisha, hookah or narjilah. It contains high levels of nicotine, a multitude of carcinogens and heavy metals but still widely used due to its recreational value [22]. Water pipes emits variety of pollutants by heating tobacco and are smoked for hours in social settings. Several studies have already confirmed a link between water pipe smoking and periodontal disease. Furthermore, water pipe smokers are at a more risk of developing periodontitis as compared with cigarette smokers but harmful effects were strongly associated with the frequency and amount of daily use [22]. In comparison to cigarette smoking, water pipe smoking is a possible risk predictor for peri-implant disease [21].

In cases such as absence of adequate oral hygiene measures, smokeless tobacco is considered as another factor associated with periodontal disease [18]. Its most common use is in India and Southeast Asia. On both periodontal and peri-implant tissues, the adverse effects of smokeless tobacco are equivalent with those of cigarette smoking. Cigarette smokers and smokeless tobacco users have deeper probing depths

and increased degrees of peri-implant bone loss as compared to the non-tobacco users [18,21].

The latest trend among individuals of all ages is electronic cigarettes (e-cigarettes) or vaping. They are widely miscalculated as harmless recreational products which increases their popularity and also because of lack of evidence on long-term health effects. Nevertheless, of nicotine content, e-cigarettes increase oxidative/carbonyl stress and causes pro-inflammatory responses [22]. It also has harmful effects on endothelial cells, fibroblasts and concomitant dysregulation in periodontal repair. The link of vaping with periodontal attachment loss and marginal bone resorption has been proven. A recent cross-sectional study has compared vaping patients with the non-smokers and demonstrated the deepened probing depths of peri-implant and higher marginal bone loss in vaping patients [23]. The data available is not conclusive so further research is required on the extent and severity of peri-implant complications and also on the impact of vaping on general health [24,25]

Among all the "illegal" smokable substances, Cannabis is one of the most commonly used drugs worldwide. After its recent legalization in several countries, it should be taken under more consideration and relationship between cannabis and peri-implantitis should be evaluated [26]. Studies have already proved higher prevalence and severity of periodontitis in cannabis users. However, a comparison study known as animal study evaluated bone loss on the periodontitis affected teeth by giving exposure to the cannabis and no effect can be seen on a periodontally healthy teeth [26]. It shows that cannabis only aggravates periodontitis-associated bone loss. Since prevalence rate of periodontitis is high in adults so this explains the higher impact of cannabis on older individuals as compared with adolescents as shown by the clinical studies [26,27]. Despite studies supports the link between cannabis use and periodontitis but the evidence regarding peri-implantitis is still not clear [26, 27].

SYSTEMIC CONDITIONS

Certain systemic diseases have an influence on periodontal health which is known from a longer duration. Studies have proved that diabetes mellitus is one of the most researched condition due to its high prevalence rate [28,29]. It affects 415 million adults and the number for future reference is estimated to be 640 million in 2040 [30]. The major effect is on insulin secretion, its function or both which leads to disruption of glycemic levels. This results in variety of complications such as neuropathological, visual, microvascular and kidney related severities [27,31,32].

The process of insulin-induced osteoblastic matrix synthesis is affected by the metabolic changes in diabetic. In this process, the differentiation of osteoblastic cells and hormones regulate calcium metabolism and the homeostasis of minerals in bone tissue and bone matrix [32]. The above stated process plays a major role in the osseointegration of dental implants. Not only the metabolic changes but the high glucose level shows the negative effect on the healing and defence mechanisms of the host. This also leads to the disbalance in the homeostasis of collagen in the extracellular matrix and the function of neutrophils is also affected [31, 33]. According to the study by Ferreira et al 2006, the prevalence rate of peri-implantitis in diabetic patients was approximately nine percent [34]. However, subjects with severity of diabetes and periodontitis have higher chances of developing peri-implantitis. Appropriate maintenance schedule is recommended in patients with controlled diabetes and undergoing implant surgery[31, 35]. The progression and severity of periodontitis is affected by rise in glycemic levels. This association has affected several vascular and cellular responses leading to enhanced tissue destruction and impaired healing response. Similar mechanisms can be seen in peri-implant tissues due to hyperglycaemia which results in higher susceptibility to peri-implantitis in those individuals [36]. Severe cases of diabetics are at more risk of developing peri-implantitis with clinical profile of deeper peri-implant pockets and increased marginal bone loss as compared with the normal glycemic level subjects. Both the smokers and severe cases of diabetic subjects are considered to be at higher chances of developing peri-implantitis. On the other hand, as comparing non-smokers with poor glycaemic control and the individuals with the normal glycaemic levels, the former ones are approximately three times at higher risk of developing peri-implantitis. Hence, on comparison of hyperglycaemia and diabetes, hyperglycaemia, is a risk predictor for peri-implantitis and not the diabetes [37].

Obesity is the another highly prevalent cause with detrimental effects on periodontal health. It is defined as abnormal or excessive accumulation of body fat with increased risk on general health. This is a major issue which leads to the physiological changes in the body, including diabetes mellitus and coronary heart disease [36]. It is a generalized hyper-inflammatory state causing an alteration in immune responses. Therefore, increases the production of pro-inflammatory cytokines which has an adverse effect on periodontal tissues and alveolar bone [37]. The data collected and analysed shows that obesity is a risk predictor for peri-implantitis. Obese patients show the clinical profile of increased bleeding, deeper depths of peri-implant probing and increased marginal bone loss as compared with the subjects of normal body weight. There is a well-established relationship between the severity of inflammation of peri-implant and the level of obesity [36]

Few studies have examined the relationship between cardiovascular diseases and peri-implantitis and most of them showed more chances of developing peri-implantitis and bone loss in subjects with cardiac anomalies [38,39]. But the results are questionable

[40]. Larger studies with adequate data availability should be done for showing an impact of these conditions on the success of implants.There are no evidences available on the effect of autoimmune diseases on peri-implantitis but questions are still raised. Studies have been done on assuring the links of peri-implantitis with rheumatoid arthritis and Sjogren's syndrome and nothing have been proved so far [41,42]. The link between autoimmune diseases and peri-implantitis is yet to be proved so further data and research is required so far.

Osteoporosis could not be linked to peri-implantitis. Medication related to osteoporosis as anti-resorptive medications, including bisphosphonates (BP) and hormone replacement therapy (HRT) are gaining attention. A study has showed increase in marginal bone loss and implant thread exposure with subjects on taking BP [43]. But a recent study showed that there is no negative effect of low-dose BP on peri-implant bone levels [44]. On the other hand, marginal bone levels are compromised by HRT. The negative influence of medication-related to osteonecrosis on hard tissues of the peri-implant should not be underrated [43,44].

GENETIC FACTORS

A clear link between genetic predisposition to peri-implantitis with specific risk predictors is yet to be determined. The most researched genetic factor is Interleukin-1 polymorphism (IL-1). There is involvement of this gene cluster in the encoding of two main pro-inflammatory cytokines, IL-1α and IL-1β, as well as the antagonist such as anti-inflammatory IL-1 receptor. The severity of peri-implantitis is associated with the increased levels of both IL-1α and IL-1β [45,46]. The studies have shown the arguable results while analyzing the link between IL-1 and peri-implantitis [46-48]. A recent study showed that subjects with IL-1 polymorphisms were 2–2.47 times higher at risk of developing peri-implantitis [48]. The synergistic effect of smoking on individuals with IL-1 polymorphisms have also raised various questions [48].

Another pro-inflammatory cytokine associated with inflammation of peri-implant and destruction of bone is tumour necrosis factor-alpha (TNF-α). No correlation has been developed even after several studies [47–49].

OCCLUSAL OVERLOAD AND PARA-FUNCTIONAL HABITS

Occlusal overload of implant-supported dentures and the mechanism which causes marginal bone loss still requires detailed research. Some studies have shown that while exceeding a certain threshold in overloading an implant leads to marginal bone loss. Moreover, peri-implantitis-affected sites shows increase in marginal bone loss under similar overload as compared to those with mucositis [50]. Overloaded implants and ligature-induced peri-implantitis shows the difference in patterns of bone resorption. The effect of overloading on peri-implant bone levels is highlighted by positioning of implant, poorly designed prosthetic reconstructions, inadequate bone quantity or inappropriate quality [51]. Para-functional habits also increase marginal bone loss due to elevated occlusal forces. On the contrary, several animal studies in the absence of inflammation have shown insignificant effect of overload on bone levels. The controlled clinical trials with experimental periodontitis models have been done but still controversial. Even the results of intentionally overloaded implants are questionable [50–52].

The diagnosis of occlusal overload and para-functional habits can be done by attrition or prosthetic reconstructions. The increase in prevalence rate of peri-implantitis more than twice is associated with the presence of wear facets on implant-supported dentures [53]. It has been demonstrated by the case investigations that occlusal adjustment may result in peri-implant bone repair [54]. A risk predictor for peri-implant bone loss is occlusal overload and it has an aggravating influence on peri-implantitis-associated bone loss [53,54].

To increase the resistance of dental implants, a larger surface contact with the bone can be provided by placing longer and wider implants. Modern simple advancements of radiographic science has provided tools for evaluation of the amount location of bone better implant surgery planning [45,55]. Dentist preference is also a factor which influences the selection of the implant type, size, or locations. This may vary from dentist to another according to their experience and specialty [56].

IMPLANT SURFACE

Different types of implant surfaces are currently available. A surface area is increased by rough implant surface which causes high bone to implant contact. Peri-implantitis is associated with increased implant surface roughness. Previous studies indicate the better reliability of machined surface implants [57]. Hybrid implants can possibly minimize the risk of peri-implantitis as they have a machine collared [58]. Few retrospective studies have compared the anodized implant surfaces with the non-anodized and shows that anodized implant surfaces are more prone to peri-implantitis [59-61]. The limited data is available for indicating that the surface roughness is the cause of crestal bone loss. Various implant therapeutic trends also influence the type of implants and its surface characteristics [57]. Therefore, it is pretty clear from a history of periodontitis and smoking. Hence, they are considered as the greater risk predictors than characteristics of implant surface in peri-implantitis [40].

CONCLUSION

Peri-implantitis is a common problem which results in tissue destruction and loss of implant. The two major

roles in its initiation and development are played by plaque accumulation and formation of biofilm. Sufficient clinical evidence about the role of prosthetic factors such as residual cement and overloading as they may result in peri-implantitis. The risk of the onset of peri-implantitis is lowered by the routine supportive therapy and proper planning. The extent of disease should be the basis for the selection of patient, treatment plan and good radiographic evaluation. Periodontal therapy and maintenance programs should be the basis of any treatment plan including implant therapy.

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