REVIEW

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The influence of smoking on the periodontal biome. A review.

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Abstract

Periodontal disease is believed to be an opportunistic infection due to the interaction of bacterial plaque and the patient's response that may be influenced by environmental, genetic, and other risk factors. In addition to the fact that smoking is considered a risk factor in many systemic diseases, it has also been associated with the initiation of periodontal disease. Smoking is one of the modifiable risk factors and has a significant influence on the development, progress, and results of the treatment of periodontal disease. The current state in the field of study suggests that smoking aids the colonization of periodontal microorganisms, accelerating the onset of periodontal disease. Biological modifications in pathogens, such as Porphyromonas gingivalis, along with poor immune response, influence the variations of subgingival flora in smoking patients. Only with an individual approach can the risk factors of each patient be identified and satisfactory results obtained. The aim of this paper is to present a comprehensive review of the influence of smoking on periodontal microbiome and the importance of adopting the appropriate treatment method according to the influence of this risk factor on healing.

Keywords: smoking, periodontal disease, dental plaque, bleeding, teeth.

Introduction

Periodontitis is a group of inflammatory conditions that affect the components of the periodontal complex: gingiva, periodontal ligaments, and alveolar bone. It affects more than 90% of the population globally, being one of the most prevalent conditions in adults [1]. Besides tooth decay, periodontal disease is the main cause of tooth loss in adults [2].

A review of the current literature was commenced in order to debate the influence of smoking on the periodontal biome. Electronic searches were performed in PubMed, Embase, and Scopus in order to identify and include articles regarding this subject using the keywords "smoking", "dental plaque", "tobacco" and "periodontal disease". Manual searches of published articles and related reviews were performed as well for completing the research necessary in writing this paper. Only original prospective longitudinal observational studies up to April 2022 that investigated the association between smoking and the onset and progression of periodontal disease were included in this research. Articles that did not meet the inclusion citeria were not considered for our paper. Seventy-three articles were identified in our research, of which, 33 were included in the writing of this review.

The initial symptoms are pre-existing erythema, hypertension, and gingival bleeding. However, gingivitis does not affect the supporting structures of the teeth and is reversible. When gingivitis is not correctly treated, it progresses to periodontitis [3].

Despite all the campaigns carried out to raise awareness of the harmful effects of smoking on general health, it still remains a widespread addiction around the world. The number of smokers exceeds 1.1 billion worldwide, killing about 8 million people yearly. Smoking is a risk factor in the onset of various conditions such as cardiovascular, lung, and periodontal disease, as well as cancer. [4].

Smoking is considered, after dental plaque, an important risk factor in the occurrence of periodontal disease, influencing its prevalence, progression, severity, and response to treatment. Epidemiological studies have shown a considerably higher risk of periodontal disease for smoking patients. This is linked to the duration and frequency of smoking [5]. In addition, smoking has a negative influence on

surgical and non-surgical treatments, with tissues having a much slower healing rate [6].

Given the harmful effects of tobacco on periodontal health, it is important to comprehend the basic mechanisms by which smoking affects the normal structure of periodontal tissues. It was accepted that both the host's response and the periodontal microflora play essential roles in the onset, severity, and evolution of periodontal disease. Numerous studies discuss the consequences of smoking on the host's response, demonstrating that smoking increases the risk of infection and susceptibility of the host by inducing immune dysfunction [7]. However, further research into the effects of smoking on the periodontal complex is needed, with some mechanisms still being unclear.

Smoking and subgingival microflora

The complex mixture generated by burning tobacco consists of more than five thousand chemicals with mutagenic, cytotoxic, carcinogenic, and antigenic properties [8]. Nicotine is the best known constituent. It is considered to have an important contribution to the development of addiction. Thus, nicotine and its main metabolite called cotinine have been utilized to research the influence of smoking on periodontal microbiome. Smoking acts locally by depositing nicotine, combustion products and hydrocarbons on dental and root surfaces, facilitating the formation of dental plaque [9, 10].

Pathogens found in smokers

The bacterial etiology of periodontal disease has been the main focus, over the last decades, with numerous suppositions being postulated.

The main pathogen involved in the onset and evolution of periodontal disease was considered to be Porphyromonas gingivalis. As recent studies have shown, Porphyromonas gingivalis has an increased impact on the oral microbiota, being able to influence the host's immune response through various pathways, eventually inducing periodontal disease [11, 12]. Streptoccocus gordonii plays a key role in initiating the formation of dental biofilm allowing its attachment to dental surfaces, and generation of mature film. Recent studies have shown that tobacco can increase the growth of Candida albicans, all these effects stimulating the subsequent attachment of pathogenic microorganisms, the formation of dental tartar and the progression of periodontal disease in smoking patients. The main pathogens found in the crevicular fluid of smokers are presented in Table 1 [13].

Table 1. Main pathogens found in smoker's crevicular fluid

YEAR	RESEARCHER	PATHOGENS
1917	Meyer	Spirochetes, Fusiforms, Streptococci
1956	McDonald	Mixed anaerobic bacteria
1988	Holt	Porphyromonas gingivalis, Treponema denticola, Tanerella forsithensia
2009	Zeller	Fusobacterium nucleatum, Filifactor alocis
2009	Cogo	Actinobacillus actinomycetemcomitans
2010	Tymkiw	Candida albicans
2011	Bagaitkar	Streptococcus gordonii
2014	Guglielmetti	Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, Treponema denticola, Tanerella forsithensia
2017	Karasneh	Treponema amylovorun
2022	Xu	Dialister, Selenomonas, Leptotrichia

The impact of nicotine on the periodontal defense system

The defense systems of the periodontal complex are represented by the epithelial barrier, saliva, immune cells and crevicular fluid. All of the above have a significant role in the conservation of the periodontal tissue against bacterial invasion and destruction. The first line of defense against bacterial aggression and harmful environmental stimuli is constituted by epithelial cells. Despite the fact that numerous studies have been performed on the effects of tobacco on the host cell, these have still not been fully elucidated. Porphyromonas gingivalis has been found to induce low levels of IL-8 and IL-1β on epithelial cells, leading to increased neutrophil levels. Exposure to tobacco reduces the proinflammatory burden of cytokines, which may promote invasion and survival of Porphyromonas gingingivalis [14].

One hypothesis for increasing periodontal changes in smokers is that periodontal pockets tend to be more anaerobic compared to non-smokers. However, studies have failed to show a significant difference in subgingival flora in smokers and non-smokers. Some researchers have suggested that smoking can influence the host's response in two ways: smoking could alter the host response by neutralizing the infection and can alter the host's response by destroying surrounding healthy tissues.

Numerous studies have shown that the effect of smoking on the periodontium may involve both processes. Smokers tend to have a low number of T-helper lymphocytes, which are important cells for the immune system with a role in regulating cell-mediated immunity and activating B lymphocytes [15]. Several studies have shown that tobacco has a detrimental effect on neutrophil function. Smoking has been shown to affect phagocytosis and chemotaxis of both oral and peripheral neutrophils. Neutrophils can be found in inflammatory lesions, especially acute lesions, and are chemically attracted to a process called chemotaxis. Once the neutrophils have reached the site of the lesion, they absorb and destroy microorganisms and can neutralize other harmful substances. The low number of neutrophils has been shown to contribute to severe periodontal destruction [16].

Smoking also causes decreased blood flow and damage to tissue revascularization, resulting in delayed healing. Nicotine generates contraction of vascular endothelial cells, making gingivitis less clinically evident furthermore it reduces crevicular fluid and immune cells. Untreated, gingivitis easily passes into the periodontitis stage [17].

While tobacco cigarette smoking has been proven to be a risk factor for periodontitis, limited information is available regarding e-cigarettes, a new alternative to smoking that has been branded as less harmful. Recent studies have proved that e-cigarette smoking increases oxidative stress, inflammatory responses, change in pulmonary cellular behavior, and stimulates DNA injury. Moreover, in vitro studies demonstrate that the flavoring agents that are combined with the aerosol of e-cigarettes have been shown to enhance DNA injury and the increase of several inflammatory proteins such as cyclooxygenase and prostaglandin E2 in gingival cells. [18, 19]

Chewable tobacco products such as pan, guthka, mawa, khaini, zarda, and quimam are popular. Long-term studies are required to be performed in such patients to evaluate the effects of tobacco on periodontal tissues and also to determine response to non-surgical therapy. [20]

The effects of smoking on alveolar bone

Smoking alters the metabolism of the alveolar bone and induces bone loss. Nicotine has been shown to significantly reduce trabecular bone volume, trabecular thickness, bone mineralization and can cause bone destruction, bleeding and even necrosis. Tobacco also reduces angiogenesis, affects bone remodeling during orthodontic tooth movement, and delays the pairing process of collagen in the bone matrix.

Osteoclasts and osteoblasts play a vital role in bone remodeling. Tooth loss is mainly the

result of bone resorption, which indicates increased activity of osteoclasts. The formation of osteoclasts in periodontal tissue is performed in several stages led by osteoclast genesis, which supports cells such as periodontal ligament cells CD4 + T cells, and inflammatory cytokines that induce osteoclast genesis [21]. Nicotine can also inhibit osteoblast differentiation. Furthermore, nicotine can speed up the metabolic rate of the bone matrix, can alter the migration and adhesion of osteoclasts and can generate osteoblast apoptosis, thus destroying the balance of bone resorption and apposition [22].

The effects of smoking on periodontal treatment

As a result of smokers having different clinical expression of periodontal disease compared to non-smokers, there is no surprise that they react differently to treatment.

Although clinical parameters, such as probing, bleeding index, and periodontal pocket depth, improved after non-surgical and/ or surgical treatment, a higher prevalence of periodontal pathogens was observed in smokers. It has also been found that smokers are more vulnerable to the restoration of a microbial subgingival plaque after scaling and root planning [23, 19].

Smoking endangers numerous aspects of innate and adaptive immune mechanisms. The general impression is that smoking affects the protective response and stimulates the inflammatory response, hence accelerating the evolution of periodontal disease. Both in vivo and in vitro studies have shown that smoking affects phagocytosis of neutrophils in periodontitis, which leads to inadequate elimination of microorganisms and increases bacterial colonization. Smoking has been demonstrated to have an inverse correlation with the level of G immunoglobulin (IgG) antibodies specific to certain periodontal agents. Low levels of IgG antibody can affect the host's immune response and may have a protective effect on periodontal microorganisms.

Discussion

A study conducted in New York on 1361 participants, aged between 20 and 74 years, showed that severe bone loss is more common in smoking patients than in non-smokers [24].

Another study in Sweden, with 540 subjects aged between 20 and 70 years, concluded that smoking, high bacterial plaque index, and old age are important risk factors for periodontal disease [25].

A longitudinal study of 273 Swedes over a 10-year period found that the risk associated with tooth loss was 78% for subjects who smoked more than 15 cigarettes a day [26].

Smoking was found to be the most important factor affecting the course of periodontal disease in a study of 499 Finnish men [27].

A study was conducted in India involving 400 men (200 smokers, 200 non-smokers) between the ages of 18 and 65, proving that periodontal disease showed statistically significant changes in smokers, with diminished gingival bleeding and deeper periodontal pockets. [28].

Some studies found a significant difference in the subgingival microbial flora present in smokers compared to non-smokers [29, 30], while others did not find statistically significant differences in the occurrence of periodontal disease-associated bacteria between smokers and non-smokers [31, 32, 33].

Conclusion

In conclusion, smoking is a dominant risk factor associated with the evolution of periodontal disease. Smoking can create a pathogenic subgingival microbiota in the periodontal complex, can reduce the host's resistance against gingivitis, and can aggravate the condition of the periodontium by turning gingivitis into periodontitis. The subgingival biome responds poorly to periodontal treatment in smokers, with a significant improvement in periodontal status with smoking cessation.

The treatment of patients with periodontal disease should be focused on understanding the relationship between environmental and genetic factors. An individual approach is required, tailored to each clinical case, to identify the patient's risks and to obtain satisfactory results.

Although much research has been done on the effects of smoking in periodontal disease, further research is needed to provide serious evidence about the underlying mechanisms.

Conflict of interest: None to declare.

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