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PHYSIOPATHOLOGICAL MECHANISMS IN THE RELATIONSHIP BETWEEN SMOKING AND PERIODONTAL DISEASE. REVIEW.

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ABSTRACT

The systemic and oral effects caused by the use of passive or active tobacco in general and oral health are considerably high throughout the world. The negative effects of smoking habits on health depend on the dose and type of compounds of cigarettes, as well as the frequency and timing of exposure. Smoking is the main modifiable risk factor for periodontitis. Clinical studies are unanimous in demonstrating that smokers have increased susceptibility to periodontitis and greater severity and progression of periodontal disease compared to non-smokers. Paradoxically, the signs and symptoms of gingival inflammation are less pronounced in smokers than in non-smokers.

Keywords: *smoking, periodontal status, smoking cessation*

INTRODUCTION

Many developed and developing countries face an epidemic of diseases caused by smoking and tobacco use. Tobacco smoking promotes an increased risk for the development of cardiovascular diseases (eg ischemic and peripheral cardiac disease and stroke), lung cancer and chronic obstructive pulmonary disease. Despite numerous initiatives, government tobacco control policies and well-known smoking-related risks, tobacco consumption remains widespread.

The World Health Organization estimates that the number of smokers worldwide is over 1 billion and is expected to increase to 1.7 billion by 2025 [1]. Based on these trends, around 10 million people are expected to die annually through tobacco use by 70% of such deaths in low- and middle-income countries by 2030 [2].

The direct link between smoking, lung disease, cancer, cardiovascular disorders and adverse effects in pregnancy, such as spontaneous abortion and low birth weight, have been extensively studied. Over the past three decades, there has been an increasing awareness of the impact of smoking on periodontal disease. Smoking is considered the major environmental risk factor in the prevalence, extent and severity of periodontal disease.

Periodontitis is the result of complex interdependencies between infectious agents and host factors [3]. Among the risk factors we count systemic predisposing conditions, such as diabetes, infectious diseases [4, 5, 6], inflammatory diseases [7], nutritional deficiencies [8], a series of systemic drugs [9], to which local risk factor can exert their influence [10]. The environmental, acquired and genetic risk factors change the expression of the disease and therefore may affect the onset

or progression of periodontitis. Among the environmental risk factors, smoking has been shown to be associated with increased prevalence and the severity of periodontal disease.

For many years, smoking has been associated with lung disease, cancer, cardiovascular disease and adverse pregnancy outcomes such as spontaneous abortion and low birth weight. In the last two decades, it has also been recognized that smoking is associated with periodontal disease.

It is well established that tobacco use is one of the most important modifiable risk factors in the incidence and progression of periodontal disease. In addition, tobacco consumption has a negative adverse effect on the whole spectrum of periodontal treatment approaches, from mechanical debridement, local and systemic antimicrobial therapy to periodontal surgery, including regenerative procedures and dental implants.

Effects of nicotine on periodontal tissues

While nicotine is the main psychoactive component and dependence on it is the main reason why people often obey large doses over many years, we must appreciate that tobacco smoke contains thousands of different compounds. Many of these substances are directly harmful / poisonous to living organisms and cells and nicotine can be unfairly accused of most of these properties. Moreover, it is very important to appreciate that most of the harmful effects of tobacco products will result from systemic exposure by absorption into the lungs rather than by local absorption into the oral cavity. A regular smoker is exposed to these compounds several times a day for several minutes at a time.

Although there is increasing evidence of the harmful effects of passive smoking, periodontal literature is generally limited to active smoking. Many smokers develop their habit in the years of adolescence and continue throughout their lives. No other medication is given as frequently or over a period of time as smoking. This should highlight the fact that harmful effects on periodontitis are derived from chronic long-term exposure and have no relation to the effects that can be measured in a single exposure [11].

Cotinine, a nicotine metabolite, can be measured in serum / plasma and saliva and is a better measure of exposure to tobacco smoke because it has a half-life longer than nicotine (18 hours versus 1-2 hours). Smokers would expect to have serum cotinine levels above 14 ng / ml, and this could be as high as 1000 ng / ml. The rest of plasma nicotine levels are much lower (5-50 ng / ml) and are maintained by the individual to satisfy their nicotine desire. Because nicotine is so rapidly absorbed from the lungs and transport to the brain is rapid, peak levels can be measured in the brain. It is important to understand these variations with respect to the levels tested in in-vitro experiments.

Some early studies suggest that smokers had less gingival bleeding than non-smokers [12]. This observation was confirmed in a comparative study of 10 chronic smokers (at least 20 cigarettes a day) and 10 non-smokers who had similar periods of periodontitis. Bleeding was demonstrated by Bergstrom & Bostrom [13]. Gingival bleeding was lower for 130 smokers than 113 non-smokers with similar periodontal levels of destruction.

Tobacco smoke contains carbon monoxide that can be detected in smoker breathing and can be used to assess compliance in smoking programs [11]. The oxygen saturation of haemoglobin is impaired and attempts have been made to measure this in the gingival tissue of smokers and non-smokers.

Hanioka and colleagues [14] showed variable results. In healthy gums, smokers appear to have a lower saturation of oxygen, determined by reflective spectrophotometry. The same group also examined the oxygen tension in the bags of 34 non-smokers and 27 smokers with mild to moderate periodontitis. They showed that the oxygen tension in the pocket was significantly lower in smokers (average 21.9 mm Hg) compared to non-smokers (mean 33.4 mm Hg [p <0.0001]). This could have an impact on the microflora in the pocket.

Vascularization was also examined in histological and immunocytochemical studies. In a very limited study of a three-smoked and four non-smoked histological section, Mirbod and colleagues [15] found that there is a large proportion of small pots compared to large vessels in smokers compared to non-smokers, but there are no differences in vascular density. The region chosen for study was connective tissue under the external gingival epithelium, which was therefore removed from the purulent wall and inflammatory lesion.

Orientation and location of the samples were not described. A more comprehensive histological comparison of smokers and non-smokers was presented by Rezavandi and colleagues [16], who labelled the vessels by immunohistochemical staining of factor von Willebrand, ICAM-1 and E-Selectin. They reported that a significantly greater number of vessels were seen in inflamed tissues of non-smokers than smokers (p <0.05).

Baab & Öberg [17] were the first researchers to question the vasoconstrictive action of nicotine (from cigarette smoking) on gingival tissues. In a study of Doppler laser (LDF) flow in 12 smokers, they showed that the gingival blood flow increased by about 25% during smoking, was maintained for 5 minutes and then gradually decreased to baseline. This has been associated with an increase in heart rate and systolic and diastolic blood pressure.

They confirmed that blood flow to the forearm skin decreased slightly, demonstrating the differences in response between peripheral skin and head and neck responses. It was interesting to note that 3 of their subjects felt a slight defeat after smoking, suggesting that the inhalation dose was higher than usual.

Animal studies have shown that local administration of nicotine has a negative impact on bone healing, which may be related to inhibited expression of various growth factors and delayed revascularization [18]. These findings could help explain the low response of treatment to surgical periodontal procedures, especially in tissue regeneration. This means that tobacco smoke can have a masked effect on gingival symptoms of inflammation, which could give smokers a false sense of gingival care [13]. Smoking stimulates the expression of proinflammatory cytokines, such as interleukin-1, which contributes to increased tissue lesions and alveolar bone resorption [19].

Effects of smoking on etiology and pathogenesis of periodontal disease

Increased prevalence and severity of periodontal disease associated with smoking suggests that host-aggression interactions normally observed in chronic periodontitis are altered, resulting in more aggressive periodontal destruction. This imbalance between bacterial challenge and host response may be due to changes in subgingival plaque composition, increase in the number and / or virulence of pathogens, changes in host response to bacterial challenge or a combination of the two.

Physiopathology

Clinical signs of inflammation are less pronounced in smokers than non-smokers. These observations may be due to changes in inflammation response in smokers or due to alteration in the vascular response of gingival tissues.

Although no significant differences in vascular density of healthy gums between smokers and non-smokers were observed, microcirculation response to plaque accumulation appears to be modified in smokers compared to non-smokers. Developing inflammation, increased flow of gingival fluid, bleeding in the sound and gums were lower in smokers than in non-smokers.

In addition, oxygen concentration in healthy gingival tissues appears to be lower in smokers than non-smokers, although this state is reversed in the presence of moderate inflammation [20]. Subgingival temperatures are lower for smokers than for non-smokers, and recovery from vasoconstriction caused by local anesthetic administration lasts longer for smokers. These aggregated data suggest that there are significant changes in microvascularization of smokers compared to non-smokers and that these changes lead to lower blood flow and decreased clinical signs of inflammation when the disease progresses.

Microbiology

Several studies explored the changes that may occur in the subgingival plaque as a result of smoking with contradictory and inconclusive results. In a study of 142 patients with chronic periodontitis, the plate samples in deep pockets (> 6 mm) showed no differences in the number of *Aggregatibacter actinomyceterncomitans, Porphyromonas gingivalis* and *Prevotella intermedia* [21]. In a similar study in 615 patients, the prevalence of *A. actinomyceterncomitans, P. gingivalis, P. intermedia* and *Eikenella corrodens* was not significantly different between smokers and non-smokers.

Of particular interest was the observation that smokers did not respond to mechanical therapy, such as non-smokers, and that this was associated with the increase in levels of T. *forsythia, A. actinomyceterncomitans* and *P. gingivalis* remaining in pockets after smoking group therapy versus non-smokers [22].

Another study took samples of subgingival bacterial plaques in 272 adult subjects, including 50 smokers, 98 smokers and 124 non-smokers [20]. Using DNA DNA hybridization technology to scan 29 different subgingival species, it was found that members of the orange and red complexes including *Eikenella nodatum, Fusobacterium nucleatum ss., P. intermedia, Peptostreptococcus micros, Prevotella nigrescens*, T. *forsytenis, P. gingivalis* and *Treponema denticola* were significantly more prevalent among current smokers than non-smokers and former smokers.

Interestingly, the increased prevalence of these periodontal pathogens was due to increased colonization of superficial sites (bag depth <4 mm), with no differences between smokers, ex-smokers and non-smokers in bags deeper than 4 mm [20]. In addition, these pathogenic bacteria were more common in the upper maxilla than the mandible.

There are also reports of larger proportions and / or prevalence of exogenous or commensal flora in moderate or deep depths of smoker exploration, indicating an adverse effect of smoking on host response. This concept is also supported by the persistence of periodontal bacteria in smokers after root scaling and planing [23]. These data suggest that smokers have a greater colonization of periodontal pathogens than non-smokers or former smokers and that this colonization can lead to an increased prevalence of periodontal disease.

Immunology

The immune response of the host to plaque build-up is essentially protective. In periodontal and gingival health, there is a balance between bacterial platelet challenge and immune response inside the gum tissues without loss of periodontal support. In contrast, periodontitis appears to be associated with a change in host-aggression balance that can be initiated by alterations in the bacterial composition of the subgingival plaque, changes in the immune response or a combination of the two elements.

Smoking has a major effect on the immune response protecting elements, leading to an increase in the degree and severity of periodontal damage. The harmful effects of smoking seem to be partly due to inhibition of the immune response to bacterial challenge [24]. Neutrophils are an important component of the host's response to bacterial infection, and changes in the number or function of neutrophils can lead to local and / or systemic infections. Critical functions of neutrophils include chemotaxis (directed from blood flow to the site of infection), phagocytosis (internalization of foreign particles such as bacteria) and killing using oxidative and non-oxidative mechanisms.

Neutrophils, obtained from the peripheral blood or saliva of smokers or exposed in vitro to whole tobacco smoke or nicotine, have been shown to exhibit functional changes in chemotaxis, phagocytosis and oxidative burst. Smoking has been shown to impair oral chemotaxis and phagocytosis, and in vitro studies on the effects of tobacco products on neutrophils have shown deleterious effects on cellular movement and oxidative breakdown [25].

However, it should be noted that most studies did not report major differences in the number of neutrophils in the pocket [26]. However, in pulmonary exposed tissues there is an increased number of neutrophils in the tissue itself. In addition, smokers have an increased number of T cells in periodontal tissues [19], another host cell response using the integrin / selectin system to migrate into tissues. These series of observations indicate that neutrophils in smokers can be stimulated to migrate to periodontal tissues, but once they reach the tissues, continued exposure to smoke can affect their progress in the periodontal pocket.

However, neutrophils in tissues would still be exposed to an acute intermittent action of tobacco substances that could penetrate into superficial tissue and trigger destructive processes. In addition, it has been reported that production of antibodies essential to phagocytosis and killing of bacteria, especially IgG2 levels in periodontal pathogens, is reduced in smokers to non-smokers with periodontitis [27].

In contrast, high levels of TNF-α have been demonstrated in gingival crevicular fluid of smokers, as well as high levels of PGEg, neutrophil elastase and matrix metalloproteinase. In vitro studies have also shown that nicotine exposure increases PGE2 secretion by monocytes in response to LPS. These data suggest that smoking may affect the neutrophil resonance to periodontal infection, but may also increase the release of tissue destructive enzymes.

It is interesting to know that for patients who use tobacco as a smoke product, such as cigarettes, cigars and pipes, there are two levels of exposure to tobacco that are encountered; these may be called "chronic" and "acute" smoke exposures. In the smoker, there are low "chronic" levels of tobacco products in serum, saliva, gingival fluid and inside cells, and in the extracellular matrix of periodontal tissue itself. These low concentrations of tobacco may have one

type of effect on host response [19]. However, during smoking, concentrations of tobacco products, several hundred to thousands times larger, are found in saliva, gingival fluid, and periodontal tissues.

These much higher concentrations may have different effects on the cells and other elements of the host response compared to long-term chronic levels. Such higher concentrations have been shown to affect the attachment of fibroblasts and the synthesis of collagen, which may in turn reduce the response to periodontal disease in smokers [19].

Given that smokers are two to eight times more likely to have periodontitis than non-smokers, quitting smoking should be an important treatment for periodontitis patients. This may be useful in patient education and may encourage patients who intend to stop.

Dental professionals can offer advice on giving up smoking to patients. Therefore, close collaboration between dentists / periodontologists and doctors in the treatment of smokers is recommended.

Benefits of quitting smoking on the results of periodontal treatment

There is a central question: are there significant long-term benefits of cessation of tobacco use? Over the past three decades, several lines of evidence clearly show the beneficial effects of giving up tobacco to periodontal tissues, the progression of periodontal disease and the results of periodontal therapy. A line of evidence comes from transversal studies that compare the periodontal condition between current smokers, former smokers and non-smokers. The results of these studies clearly demonstrate that smokers have a higher proportion of deep sites and higher clinical attachment losses than non-smokers [28]. However, ex-smokers present probing depths, clinical loss of attachment and alveolar bone loss between the clinical values reported for smokers and non-smokers [29].

These findings imply that in former smokers there may be a reduction in the rate of periodontal destruction or even repair and regeneration of the periodontal tissues after the cessation of tobacco. A second line of evidence on the benefits of tobacco withdrawal is based on longitudinal studies of smokers, former smokers and non-smokers. These studies demonstrate that the rate of progression of periodontal destruction over observation periods over several years, measured by probing depth, attachment loss or alveolar levels, is similar between smokers and non-smokers and is significantly lower in both groups than the progression of the observed disease to current smokers who continue to smoke [30]. In addition, the risk of tooth loss in former smokers is approaching the low risk of tooth loss observed in non-smokers, as the period of renunciation increases [31].

A third line of evidence on the benefits of smoking cessation derives from studies focusing on the clinical response of smokers, smokers and current non-smokers to specific periodontal therapies. These published studies have highlighted clinical improvements obtained from basic non-invasive periodontal procedures such as supra- and subgingival debridement as well as advanced surgical periodontal procedures such as open flap debridement, grafting and regeneration procedures, periodontal plastic surgery and preparation of the implant site [32]. Although many studies have shown clinically measurable healing deficiency responses to the whole range of periodontal procedures in current smokers compared to non-smokers, most studies suggest that healing responses to ex-smokers are comparable to non-smokers.

Although the harmful effects of tobacco use and the potential benefits of tobacco withdrawal are well established

and can serve as motivational tools for dental practitioners in counselling and motivating patients, a frequent question among smokers is whether the reduction in the amount of tobacco used, the number of cigarettes smoked per day), instead of giving up completely, can bring some benefits. Some published transversal studies present a dose-dependent association between tobacco consumption and the status of periodontal disease, with a disease severity increasing with the number of cigarettes smoked per day [33]. Similarly, several years of exposure to cigarette smoke (packs per day, 9 years of smoking) were associated with a worse periodontal disease.

However, reducing the level of smoking, for example by reducing the number of cigarettes smoked per day, can bring questionable benefits. In some studies, moderate and chronic smokers have shown a severity of comparable periodontal disease [34]. When the number of cigarettes consumed daily is low, levels of nicotine and other tobacco substances in blood and urine are not necessarily reduced proportionally [35]. This may be due to the need for smokers to maintain the same usual levels of nicotine and thus to compensate for the smoke of each cigarette.

Therefore, the general objective of addressing the issue of tobacco consumption and periodontal disease should not be a reduction in tobacco intake, but an effective program to end long-term tobacco consumption. In view of these considerations, dentists may be in a unique position to help their patients in a smoking cessation program. In particular, some patients may visit the dentist at a more frequent maintenance or recall interval, such as from 3 to 6 months, and therefore have the opportunity to receive educational and personal repetitions about the effects of tobacco use on their oral health, as well as training, feedback and reinforcement in a cessation program.

CONCLUSIONS

Current immune-inflammatory mechanisms, which could accurately explain the severity and progression of periodontitis to tobacco users, have not been fully elucidated. However, it seems that oxidative stress and changes in immune-inflammatory systems play an important role in the pathogenesis of smoking-associated periodontitis. In vitro studies are generally agreed that smoking and its compounds have deleterious stimuli for periodontal cell function.

Local and circulating levels of immune-inflammatory and mediator cells in periodontitis smokers are usually contradictory and inconclusive, highlighting the complexity of the host's response to smoking-related periodontitis.

Although adverse effects of smoking on health may persist for many years, such effects may be reversible after smoking cessation. Therefore, quitting smoking seems to be a relevant approach to reducing the risk of periodontitis and improving the response to periodontal therapies in smokers.

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