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SMOKING AND PERIODONTAL DISEASE: A COMPREHENSIVE REVIEW

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Abstract
Periodontal disease is a kind of disease that affects tooth-supporting tissues. Whenever dental plaque accumulates in the gingival sulcus or supragingival area, it can cause inflammation of the gingiva, a condition known as (plaque-induced) gingivitis. If it (inflammation) spreads into underlying supporting tissues and causes destruction of connective tissue that leads to a periodontal pocket, which is called chronic periodontitis, it can be significantly influenced by various factors, such as systemic conditions, host responses, or environmental factors. Among these factors, tobacco use (smoking) is one of the environmental factors. It has various systemic effects, such as causing cancers, chronic obstructive pulmonary disease, cardiovascular disease, pregnancy problems, cataracts, osteoporosis, and periodontitis. Nicotine (the main ingredient of tobacco), which is rapidly absorbed through inhalation or diffusion across the buccal mucosa. About up to two hours after smoking, gingival fibroblasts can continue to absorb and release nicotine, which is found in saliva and gingival fluid, profoundly influencing the composition of the subgingival biofilm. Cotinine (a metabolite of nicotine) can increase collagenase activity, reduce fibroblast adhesion and proliferation, and decrease synthesis of fibronectin and collagen.
This article will discuss an overview of the available data in order to give dental health professionals a better understanding of the relationship between smoking and periodontal disease and the impact of smoking and its cessation on the pathogenesis and treatment of periodontal diseases.

INTRODUCTION

Periodontal disease is a kind of disease that affects tooth-supporting tissues (1). The periodontium (periodontal) tissue comprises the gingiva, alveolar bone, periodontal ligament, and cementum (2). The gingiva is keratinized epithelium of oral mucosa that covers the surrounding area of a tooth and alveolar bone (3). Alveolar bone is a specialized type of bone that occurs in the mandible and maxillae, which form sockets for teeth and make joints with cementum by periodontal ligament (4). Cementum is a part of tooth tissue that covers radicular dentin (5). Periodontal ligament is a dense, fibrous connective tissue located in the periodontal space between the tooth's cementum and the surrounding alveolar bone (6). Gingiva is divided into three following parts: marginal gingiva, which covers the tooth collar-like; attached gingiva that attaches with the tooth surrounding alveolar bone; and interdental gingiva located in the interdental area (7). The gingiva is attached to the tooth surface by the junctional epithelium (a part of

the gingival epithelium) at the cementoenamel junction area (8). A slight V-like depression located between free (marginal) gingiva and tooth, which is called a gingival sulcus (9). The gingival sulcus consists of three following parts: a soft tissue wall (formed by sulcular epithelium), a hard tissue wall (made by the associated tooth surface), and the base of the sulcus (formed by junctional epithelium) (10). Whenever dental plaque accumulates in the gingival sulcus or supragingival area, it causes inflammation of the gingiva, which is called (plaque induced) gingivitis (11). If improved oral hygiene is practiced, it can be eliminated; otherwise, it (inflammation) can spread into underlying supporting tissues (connective tissue attachment) and cause destruction of connective tissue attachment (alveolar bone, PDL, cementum) that leads to a periodontal pocket, which is called chronic periodontitis (12). Recent studies on periodontal diseases suggest that the clinical condition of plaque-induced gingival inflammation can be significantly influenced by various factors, such as systemic conditions, host responses, or environmental factors (13). Among these factors, tobacco use (smoking) is one of the environmental factors that profoundly influences the composition of the subgingival biofilm (14). It can suppress overt gingival inflammation, manifested as reduced angiogenesis and a compromised bleeding response to plaque, while simultaneously promoting periodontal tissue destruction compared to non-smokers. Tobacco smoke has a lot of (more than 3,800) dangerous and addicting chemicals, such as carbon monoxide, hydrogen cyanide, and reactive oxidizing radicals; among them, sixty are suspected to cause cancer (15). Numerous cancers, chronic obstructive pulmonary disease, cardiovascular disease, pregnancy problems, cataracts, osteoporosis, and periodontitis are just a few of the many illnesses and negative consequences that tobacco smoking brings about. Since periodontal disease is regarded as an opportunistic infection, the host's reaction to the difficulties posed by oral microbiota plays a significant role in how the illness develops (16). Cigarette smoking's extensive effects on the host and its connection to periodontal disease have garnered attention in the past ten to fifteen years (17).

The purpose of this review is to provide an overview of the available data in order to give dental health professionals a better understanding of the relationship between smoking and periodontal disease and the impact of smoking and its cessation on the pathogenesis and treatment of periodontal diseases.

Is there an actual connection between smoking and periodontal disease?

Studies considered that approximately three hundred people initiate smoking every day, while 2000 adolescents under the age of 18 begin smoking for the first time in the world (18). According to the World Health Organization report, approximately 1.1 billion people smoke tobacco worldwide (19). Earlier epidemiological studies considered that smoking significantly increases the chance of developing periodontal disorders (20). The tobacco users between the ages of 5 and 7 are 2.5–3.5 times more likely to experience severe destruction of tooth-supporting structures (periodontal attachment) (21). According to studies, after controlling for age, gender, socioeconomic status, and dental hygiene, it has been observed that, compared to non-smokers, tobacco users (smokers) experience greater attachment loss (22). These issues include gum recession, deeper pockets, increased probing depths, more teeth with furcation involvement, greater alveolar bone loss, and higher rates of tooth loss. Smoking has a dose-dependent effect on periodontium tissues (23). There is a significant correlation between the severity of periodontal disease and smoking duration and daily consumption amount (24). Acute necrotizing ulcerative gingivitis is more common in tobacco users (25).

PATHOGENETIC MECHANISMS OF PERIODONTAL LESIONS RELATED TO SMOKING

Whenever an imbalance occurred between the host's defense effectiveness to defend itself and the harmful bacteria in dental plaque, it caused the beginning and progression of periodontal damage (26). While research has focused on understanding how smoking worsens these issues, the exact mechanisms remain unclear, highlighting the need for further detailed studies (27). The majority of studies explained that mineralized plaque (accumulation) is more common in tobacco users than nonsmokers (28). However, all

tobacco users (smokers) are able to control their plaque just as effectively as nonsmokers; there does not appear to be a clear correlation between smoking and the pace of plaque production (29). High calcium concentrations in the smoker's saliva and plaque, as well as elevated pH and salivary flow, may account for heavy calculus (mineralized plaque) deposition (30). Although tobacco users brush as often as nonsmokers, they appear to devote less time and attention to brushing daily (31). Overall, smoking-related increases in calculus buildup do not significantly impact periodontal health (32). There are no noticeable differences in the subgingival microbiota between smokers and nonsmokers (33). Nicotine is a major contributing factor to the risk of periodontal disease in smokers, increasing their susceptibility to the disease rather than directly altering the microbiological environment (34). In the molecular phase of tobacco use, the main ingredient, nicotine, is rapidly absorbed through inhalation or diffusion across the buccal mucosa (35). It has various systemic effects. Cotinine (a metabolite of nicotine) is found in saliva and gingival fluid (36). According to certain investigations, up to two hours after smoking, gingival fibroblasts can continue to absorb and release nicotine (37). Nicotine has been associated with increased collagenase activity, reduced fibroblast adhesion and proliferation, and decreased synthesis of fibronectin and collagen (38). Smokers experience less noticeable gingival bleeding than nonsmokers because of nicotine's vasoactive effects, which cause an increased release of epinephrine and norepinephrine (39). This vasoconstrictive effect of nicotine on the gingival capillaries caused the prevention of efficient vascularization of the gingiva, thus lowering its resistance to infection. Nicotine can increase levels of inflammatory mediators (prostaglandin and interleukin 1-beta), which promote bone resorption through osteoclastic effects (40). Smoking may weaken the periodontal tissue's resistance to plaque-induced bacterial invasion (41). Polymorphonuclear leukocytes, which are crucial for periodontal defense, are reduced, changed, and impaired by tobacco smoke exposure (42). The periodontal disease may deteriorate as a result of these cells' impaired phagocytic adhesion, ingestion, and motility (43).

Smoking and gingival inflammation

Inflammation of the gingiva (gingivitis) progresses through various stages, initially affecting the gingiva and, if left untreated, changing to periodontitis (44). In the initial lesion, blood vessel levels are altered, and there is an increase in blood flow and capillary dilatation (45). Clinically, the gingiva becomes enlarged as a result of the early lesions (41). The cell population then undergoes modifications, with an increase in macrophages and lymphocytes (46). When some of the bacteria in tooth plaque remain and infiltrate the host tissue, developed lesions result (44). At this stage, perivascular buildup of chronic inflammatory cells is visible (47). Collagen loss in the impacted connective tissue occurs after the number of cells in the chronic inflammatory substance increases (48). Nevertheless, neither bone nor connective tissue attachment has been lost at this point (47). Typically, smoking does not cause noticeable gingival abnormalities (49). It has been considered that smoking reduces the clinical symptoms of gingivitis, and this effect is unaffected by plaque levels (48). Heavy smokers may have hyperkeratosis and grayish staining of the gingiva, which is caused by an increase in keratinized cells, and epithelium reveals hyperplastic, hyperkeratotic, and keratotic changes (50). A number of studies found that tobacco consumption was a contributing factor to ANUG and that the frequency of ANUG increased as tobacco usage increased (25). Both nicotine and the tar in the smoke had the potential to cause capillary contraction and gingivitis by directly irritating the gingiva (24).

Smoking and gingival bleeding

Gum bleeding on probing is increasingly frequently utilized in clinical examination as a way to identify active lesions in periodontal disease, and it is a significant early indicator of gingivitis (early lesion) (44). While smoking is known to cause peripheral vasoconstriction, vasodilatation may occur before this in certain individuals (39). The degree of tobacco smoke inhalation and the pace of nicotine absorption are likely to influence the effect generated in any given situation (33). Cigarette nicotine causes the sympathetic nervous system to release catecholamines and other neurotransmitters (40). Nicotine's vasoconstrictive effects could be the cause of the gingival blood flow reduction and smokers experience less gingival bleeding than non-smokers (43). Hence

smokers often show fewer visible signs of gingivitis, such as redness, bleeding, or discharge, compared to nonsmokers (41).

Smoking and periodontal inflammation

Even though oral bacterial infections are the direct cause of periodontitis, a variety of hereditary (genetic) and environmental factors influence the disease's course and intensity (26). In 1980, it was first observed that tobacco use could lead to a reduced inflammatory response (51). Subsequent studies have supported these findings and revealed a link between tobacco use and periodontal disease, and it is considered that tobacco use is the most significant behavioral risk factor for the occurrence and development of periodontitis (52). Decreasing of gingival bleeding response is the initial and most obvious consequence of smoking on periodontal tissues, which may lead to eluding clinical identification of disease and may even be recognized as something positive and healthy (43). However, the absence of bleeding or insufficient bleeding of the gingiva in tobacco users does not indicate healthy periodontium (53). Smoking causes the supporting periodontal tissues to be excessively destroyed, which leads to pocket formation, bone loss, and premature tooth loss compared to non-smokers, as demonstrated in a number of studies (54).

Smoking and periodontal treatment

Studies have revealed that tobacco use negatively affects all forms of periodontal treatment and also indicated that 90% of refractory periodontal disease cases occur in tobacco users (55). Numerous clinical studies considered how smokers and nonsmokers react to different forms of periodontal therapy, such as non-surgical and surgical treatments, in the field of periodontal disease (56). Compared to smokers, nonsmokers show significantly greater reductions in probing depths and bleeding on probing, as well as a significantly greater gain in clinical attachment following both non-surgical and surgical treatments, as well as observed in the treatment of furcation regions and after regenerative procedures (57). Additionally, recent studies suggest that adjunct use of local and systemic antibiotics may enhance the clinical outcomes of surgical and nonsurgical treatment such as scaling/root planing and guided tissue regeneration in smokers and may favor the removal of T. forsythensis and P. gingivalis in a larger proportion of sites than traditional mechanical therapy (58). Machion et al. considered that in smokers, combining local 10% doxycycline (anticollagenase) with the scaling and root planing in the treatment of chronic periodontitis may lead to better clinical treatment outcomes than mechanical therapy alone (59).

Benefits of Smoking Cessation on the Periodontium

Despite the fact that quitting smoking does not undo all the effects of smoking, tobacco use seems to have a reversible harmful effect on the subgingival environment and flora and decreases the rate of bone and attachment loss and the severity of disease (56).
The fact that ex-smokers react to both non-surgical and surgical treatment as well as implant treatment similarly to individuals who have never smoked is heartening (60).

Research Gaps

According to the available literature, 90% of refractory periodontal disease cases occur in tobacco users. Despite this, no research has been done yet to assess the effectiveness of quitting smoking as a treatment approach for instances that are not responding to treatment.

Conclusion

Periodontal disease is a kind of disease that affects tooth-supporting tissues, caused by dental plaque and can be significantly influenced by various factors, such as smoking (environmental factor). Tobacco use (smoking) reveals a link between tobacco use and periodontal disease, and it is considered that tobacco use is the most significant behavioral risk factor for the occurrence and development of periodontitis.
Nicotine, the primary component of smoke, found in saliva and gingival fluid, has the potential to cause capillary contraction in gingival tissue and significantly influence the composition of the subgingival biofilm. These effects of smoking (capillary contraction) could be the cause of the gingival blood flow reduction, and smokers experience less gingival bleeding than non-smokers, which may lead to eluding clinical identification of disease and may even be recognized as something positive and healthy that does not indicate healthy

periodontium. Smoking causes the supporting periodontal tissues to be excessively destroyed, which leads to pocket formation, bone loss, and premature tooth loss compared to non-smokers.

Studies have revealed that tobacco use negatively affects all forms of periodontal treatment and also indicated that 90% of refractory periodontal disease cases occur in tobacco users. Adjunct use of local and systemic antibiotics may enhance the clinical outcomes of surgical and nonsurgical treatment, such as scaling/root planing and guided tissue regeneration, in smokers and may favor the removal of T. forsythensis and P. gingivalis in a larger proportion of sites. Ex-smokers react to both non-surgical and surgical treatment as well as implant treatment similarly to individuals who have never smoked.

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