Review Article

Periodontal disease and smoking: An overview

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ABSTRACT

The role of tobacco smoking as a causative factor in the development of the periodontal disease has long been debated and recently a large number of papers have been published in the literature regarding this symbiosis. Smoking also gives an encouraging environment for microbes in the mouth such as *Porphyromonas gingivalis*, *Prevotella intermedia*, and *Aggregatibacter actinomycetemcomitans* because the by-products of smoking prevent the mechanisms that limit the growth of harmful bacteria in the oral cavity. Thus, smoking promotes early stages of periodontal disease. Smoking in the form of cigars and pipes have similar deleterious effects that cigarettes do on oral health. Much of the literature has also showed that smokers affected with periodontitis respond less favorably to periodontal treatment be it nonsurgical and surgical. In this paper, we have reviewed the effects of smoking on various aspects of the periodontal disease process.

Key words: Oral health, periodontal disease, smoking, tobacco

INTRODUCTION

Periodontal disease is among the main contributors to the global burden of oral disease. However, somewhat limited attention has been given to periodontal disease in many countries by providers of oral health care and public health administrators.[1] Since last decade dentists and dental researches have become more alert over the serious role of smoking on incidence and severity of periodontal disease, thus naming smoking as a risk factor for periodontal disease.[2] According to previous researches, the life expectancy of people who smoke is decreased by 14 years depending on the duration of time they smoke.[3] Smoking not only deteriorates the body's immune system and causes bad breath but it increases the risk of periodontal disease by 2–7-fold. The effects that smoking tobacco has on the periodontal tissues depends on frequency and duration of smoking in a subject.[4] Periodontal diseases due to smoking show more predilection for males than females. There are more than 4000 chemicals in a cigarette that slow down the healing process during periodontal treatment which includes formaldehyde, carbon monoxide, ammonia, and arsenic. Smoking creates a favorable environment for bacteria in the mouth like *Porphyromonas gingivalis*, *Prevotella intermedia*, and *Aggregatibacter actinomycetemcomitans* as by-products of smoking inhibit the mechanisms of preventing the growth of bacteria in the oral cavity.[5] Thus, smoking can encourage early stages of periodontal lesions. Smoking also decreases the periodontal response to treatment and causes refractory disease.[6]

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EFFECTS OF SMOKING

Smoking and plaque

It has been reported that smokers have a poor level of oral hygiene when compared to nonsmokers.[7-9] The tooth brushing effectiveness of smokers is much less, and calcium concentration in the dental plaque of smokers has been found to be significantly higher than in nonsmokers, which suggests a direct influence on the rate of calculus

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formation and deterioration of oral hygiene.[10] It is well known that smokers have higher plaque index scores than nonsmokers. Males have significantly more plaque than females and in both genders, smokers have almost twice the percentage of marginal line with an adherent plaque as nonsmokers.[11] Recent researches have suggested that plaque is more adherent to the tooth and less freely removed from the teeth of smokers due to the deposition of tars from smoke.[12]

Cigarette smoking also causes a lowering of the oxidation-reduction potential (Eh), which causes an increase in anaerobic plaque bacteria. According to previous studies, Eh values in gingiva drops significantly after smoking one cigarette.[13] Furthermore, tobacco smoke contains phenols and cyanides which can account for antibacterial and toxic properties. Smokers harbor significantly higher levels of these and are at significantly greater risk of infection with *Bacteroides forsythus* than nonsmokers.[14] *P. gingivalis* is also more likely to subgingivally infect smokers than nonsmokers. However, the relative risk for infection due to this bacterium is significantly higher.[15,16] It has been found that three species of Gram-negative bacteria, *Branhamella catarrhalis*, *Neisseria perflava*, and *Neisseria sicca* are more susceptible to cigarette smoke than three species of Gram-positive bacteria *Streptococcus mitis*, *Streptococcus salivarius*, and *Streptococcus sanguinis*.[17]

Effects of smoking on exocrine glands

A marked increase in parotid flow rate has been reported following smoking a cigarette; however, regular smoking presented no significant alteration in salivary secretion. Though in the novice smokers, smoking noticeably increases the flow rate of saliva.[18] Calculus formation is found more abundant in smokers which may be due to an increase in salivary flow rates. It has been previously reported that there is an increased calcium concentration in fresh saliva following smoking.[19] The effect of nicotine is by an initial increase in salivary and bronchial secretions that are followed by inhibition of the secretions. The initial salivation created by smoking is possibly caused by smoke irritation and the acidity due to cigarette smoke. This change of the salivary flow or microflora may be accountable for the increased calculus prevalent in smokers.[20] The vasoconstriction of peripheral blood vessels which is caused by smoking can also affect the periodontal tissue as smokers have less overt signs of gingivitis than nonsmokers and clinical signs of gingival inflammation such as redness, bleeding, and exudation are not as apparent in smokers.

Effects of smoking on polymorphonuclear leukocytes

The polymorphonuclear leukocytes (PMNs) are the primary line of defence in an inflammatory response.[21] Decreased efficiency of PMNs may result in degranulation of these cells and release of more lysosomal enzymes, which contributes to the severity of periodontal inflammation. Studies suggest that smokers have higher blood PMN counts than nonsmokers.[22] Decreased salivary immunoglobulin A has also been found more in smokers as compared to nonsmokers. Both humoral and cellular immune responses occur as a result of continued exposure to cigarette smoking. There is an acute depression of immune response initially, followed by stimulation (weeks to months), and finally depression of immune system sets in.[23]

Effects of smoking on lymphocytes

Nicotine affects T lymphocytes by increasing the number, decreasing proliferation, decreasing their response to antigen, also decreasing T helper cells, and increasing T suppressor cells.[24] Smokers have a greater severity of gingival inflammation than nonsmokers, which is attributed to vasoconstriction of gingival vessels and heavier keratinization of gingiva in smokers. The reason suggested was a gingival inflammatory response to accumulating plaque might be suppressed under the influence of smoking (reduced capacity to mount and maintain an effective defence reaction).[25]

Acute necrotizing ulcerative gingivitis and smoking

Smoking has long been considered an etiologic factor in acute necrotizing ulcerative gingivitis (ANUG). Tar in the smoke has a direct irritating effect on gingiva giving rise to gingivitis and nicotine causes contraction of capillaries, thus interfering with the nutrition of the gingiva which consequently becomes less resistant to infection.[26] Smoking activates the release of epinephrine and stimulates contraction of peripheral vessels, reducing blood flow to the gingiva. Lesion of ANUG present within the avascular epithelial tissue is entirely dependent upon diffusion from the connective tissue for its oxygen and nutrient supply. Severe reduction of blood flow induced by sepsis, stress, and smoking may cause loss of vitality to the most vulnerable regions of the gingival epithelium leading to the onset of ANUG.[27]

Pocket depth, attachment loss, and alveolar bone loss in smokers

Cigarette smokers were seen to have significantly greater probing depths and bone loss than nonsmokers with an increased tooth mobility.[28] It affects periodontium primarily by interfering with the body response mechanisms rather than by adding to local destructive factors. Besides the presence of nicotine in the gingival crevicular fluid, it has also been found on the root surfaces of periodontally diseased teeth in smokers. It affects the fibroblasts by increasing the production of collagen but impairs its secretion.[29] Nicotine changes the cell structure of the fibroblasts. It is possible that a similar disturbance in fibroblast attachment occurs in people who smoke making them more susceptible to periodontal damage.[30]

Smoking and its effects on periodontal healing

Healing as a result of conventional scaling and root planning is seen clinically as a reduction in pocket depth and is the result of a reduction in inflammation which causes tissue shrinkage or reduced inflammatory swelling and also an improved tissue tone. This improved tissue form is more resistant to pocket probing forces and is detected clinically as an increase in clinical attachment.[31] The tissue shrinkage may result in a recession which produces reduced probing depths. It has been hypothesized that in smokers much of inflammatory tissue swelling before the treatment may be absent and thus part of the posttherapy tissue change may not contribute as much to posttreatment pocket depth reduction in smokers as compared to nonsmokers.[32] Smokers could therefore have deeper pockets after therapy than nonsmokers, and these pockets harbor quantitatively and qualitatively more pathogenic bacteria than shallower pockets. It also causes reduction of fibroblasts, PMNs, and epithelial cell function, reduced host defense response and reduced vascularity of the affected site. These tissue differences in smokers following initial short-term healing after therapy may partly explain the differences in treatment response. Differences in the healing process in smokers and nonsmokers in long-term may be related to cellular and tissue differences in smokers. Fibroblasts play an important role in periodontal healing response, poorly functioning fibroblasts do not produce collagen fibers as efficiently, and thus gingival tissue support and adaptation will be impaired or at least slowed. Poor tissue form will often result in greater microbial plaque retention around teeth.[33] The brief overview of tobacco induced periodontal changes are shown in Table 1. [34,35]

Table 1: Tobacco induced periodontal tissue changes

Changes with use of tobacco	Effects
Increased vasoconstriction	Paler tissue color
Decreased blood flow	Oxygen depletion
Thickened fibrotic consistency, minimal erythema relative to extent of disease	Compromised immune response
Fewer and impaired PMNs reduced IgG antibody	
Increased collagenase production	Gingival recession
Increased TNF-α, and PGE2 in GCF	
Decreased GCF flow	Increased inflammation
	Bleeding on probing
Reduction of bone mineral and impaired fibroblast formation	Increased tooth loss
	Greater probing depth, bone and attachment loss, furcation invasion
	Increased rate of periodontal destruction
	Increased colonization of shallow periodontal pockets by periodontal pathogens
Decreased lymphocyte proliferation	Impaired wound healing
Altered neutrophil chemotaxis, phagocytosis and oxidative burst	
Increased neutrophil collagenase and elastase in GCF	

GCF: Gingival crevicular fluid, PMNs: Polymorphonuclear leukocytes, IgG: Immunoglobulin G, TNF-α: Tumor necrosis factor alpha, PGE2: Prostaglandin E2

CONCLUSION AND PERSPECTIVE

Smoking results in a number of changes within the periodontium which lead to the progression of periodontal disease. Dentists have an important role in creating awareness among the public regarding the detrimental effect of smoking on oral health and health in general.

Proper history regarding the use of tobacco in any form or habits should be taken or should be uncovered during the oral examination. The patient should be strongly advised to quit tobacco by pointing out risks involved and positive benefits gained after quitting of this habit. In the present scenario, tobacco is a major killer, thus a major drive against tobacco shall be started, and a public health dentist shall be a model role player in this. The day to day health camps organized at the institutional level or at personal level shall have a mandatory part against smoking and tobacco.

Though a lot has come up through the regulatory approach put by the government and the acting agencies against tobacco, however, a proper reinforcement method is important for the eradication of this evil called "tobacco."

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Conflicts of interest

There are no conflicts of interest.

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