

Smoking: A Sinful Act for Periodontal Health

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Abstract: Cigarette smoking has long been suspected to be associated with a variety of oral conditions including periodontal diseases. The role of tobacco smoking as a risk factor in the progression of the periodontal disease process has long been suspected. Tobacco smoking increases the overall risk for severe periodontal disease by 2 to 8 times compared to non-smokers by affecting the gingival vasculature, the humoral immune system, and the cellular immune and inflammatory systems, and have effects throughout the cytokine and adhesion molecule network. Dental health professionals have the opportunity to become engaged in tobacco cessation whereby the ill effects of not only oral cancer and periodontal diseases, but many additional adverse health effects, can be prevented.

Key Words: Smoking, Tobacco, Risk Factor, Periodontal disease

I. INTRODUCTION

“Chewing tobacco is tobacco’s body, smoke is its spirit and snuff is the tobacco’s heart”. The use of tobacco is dangerous to general health, as it is a common cause of addiction, preventable disorder, disability and death. The use of tobacco causes an increased risk of oral malignancy, periodontal disease, oral mucosal lesions and other detrimental conditions and it adversely affects the outcome of the periodontal treatment also. It has been significantly proved that tobacco causes fatal disabling disease including cancer, cardiovascular and respiratory diseases. It is also risk factor for COPD and LBW babies.

Tobacco can be devoured through the mouth in several shapes, shifted from smoking to smokeless tobacco chewing on itself or combined with betel nut. The harmful substances from smoking tobacco such as tobacco specific nitrosamines and benzopyrene along with nicotine are enter into the systemic circulation by firstly passing through oral cavity ^[1].

Tobacco Smoking is highly widespread and can be considered an endemic in both developed and developing country. Smoking tobacco has been firstly introduced as an additive habit in Europe. Currently, the most of the population smoke cigarettes. The number of cigarette smokers is gradually declining, but those who do smoke are smoking more. Smokers are three times more likely to get acute periodontitis than non smokers ^[2].

Periodontitis is an inflammatory disease of the supporting tissues of the teeth which is caused by particular microorganisms. It is characterized by progressive destruction of the periodontal ligament, alveolar bone, pocket formation, and recession. There are several factors that may affect the onset or progression of periodontitis by modifying the expression of periodontal disease ^[3]. Only cigarette smoking does not cause periodontal disease. Periodontal disease is caused by bacterial plaque that gets subgingivally and initiates a process by which the infection initiates bone loss around the teeth. Smokers have faster bone loss rate than in non smokers after progression of periodontal disease. This is because of the effect of nicotine on the circulatory system, which in turn decrease on the blood supply and also reduces the intake of oxygen by hemoglobin, thus in turn retards the body’s capability to fight the infection and bone resorption ^[4].

Smoking is the strongest of the variable risk factors for periodontal disease after dental plaque. Smokers may harbor a higher occurrence of probable periodontal pathogens, and smoking impairs various aspects of the innate and adaptive immune responses, including neutrophil function, antibody production, fibroblast activities, vascular factors and inflammatory mediator production ^[5]. There is also an impact of smoking tobacco on implant success and survival rates..

Advising patients to quit tobacco use is a dental professional responsibility, and the dentists may take an active role in nicotine replacement counseling ^[6]. It is encouraging that clinical studies suggest that patients who quit smoking have a similar response to that of nonsmokers to periodontal and implant procedures.

I. TOBACCO AND ITS DIFFERENT FORMS

Tobacco is a green leafy plant that is grown in warm environment. After leaves of the plant are picked up, these are wasted and used in smokeless and smoking forms. Smokeless tobacco refers to the use of unburned tobacco, in the form of chewing, spitting, dipping and snuff. Smoking tobacco is the act of burning dried leaves of the tobacco plant and inhaling the smoke.

Smoking tobacco: cigarette, Cigar, Cigarillo, Cherooot, Pipe, Hookah, Hookli, Bidi, Chillum, Chutta, Dhumti, Reverse Dhumti, Keeyo, Kreteks, Inhalation Snuff.

Constituents of Tobacco: Tobacco smoke is estimated to have over 4000 compounds many of which are pharmacologically active, toxic, mutagenic and carcinogenic ^[7]. In smoking tobacco 43 known carcinogens are found. A volatile Nnitroso compound of NNN (N-nitrosornicotine) was the first organic carcinogen isolated from smokeless tobacco.

Tobacco smoke consists of a gaseous phase which contains carbon monoxide, nitrogen, oxygen, and carbon dioxide as well as particulate phase containing nicotine, water and polycyclic aromatic hydrocarbons.

- According to Centre for Disease Control (CDC) and prevention, the smokers are classified as: ^[7]

Current smokers: Those that had smoked ≥ 100 cigarettes in their lifetime and smoked at the time of interview

Non-smokers: Those that had smoked ≥ 100 cigarettes in their lifetime

Former smoker: Those that had smoked ≥ 100 cigarettes in their lifetime, but were not currently smoking.

- According to the number of cigarettes smoke/day, smokers can be classified as:

Heavy smokers: Smoke ≥ 20 cigarettes/day

Light smokers: Smoke ≤ 19 cigarettes/day

II. ETIOPATHOGENESIS OF PERIODONTAL DISEASE PROGRESSION IN SMOKERS

Effect of smoking on microbiology

Smoking has important effects on oral bacteria, plaque development and accumulation. Smokers showed a higher prevalence of dental plaque and poorer oral hygiene than non-smokers in earlier studies and suggested that more severe periodontal disease in smokers might be because of greater accumulation of plaque [8]. Smoking tobacco could cause a lowering of the oxidation-reduction potential (Eh), and this could cause an increase in anaerobic plaque microorganism. There was a statistically significant increase in the proportion of Gram-positive to Gram-negative bacteria in 3-day-old plaque from smokers, when compared with the non-smokers [9]. Smokers were more positive for aggregatibacter actinomycetemcomitans, Porphyromonas gingivalis, and Tannerella forsythia than non smokers [10]. Current smokers displayed an increased risk of Treponema denticola in periodontal pockets [11].

Etiologic Factor	Effects of Smoking
Microbiology	Increased complexity of the microbiome and colonization of periodontal pockets by periodontal pathogens
Immune-inflammatory response	Altered neutrophil chemotaxis, phagocytosis, and oxidative burst \uparrow Tumor necrosis factor- α and prostaglandin E_2 in gingival crevicular fluid \uparrow Neutrophil collagenase and elastase in gingival crevicular fluid \uparrow Production of prostaglandin E_2 by monocytes in response to lipopolysaccharide
Physiology	\downarrow Gingival blood vessels with \uparrow inflammation \downarrow Gingival crevicular fluid flow and bleeding on probing with \uparrow inflammation \downarrow Subgingival temperature \uparrow Time needed to recover from local anesthesia

Fig:1 Effects of etiological factors in smokers [12]

Effect of smoking on Immunology

The immune response of the host to biofilm accumulation is really protective. Smoking exerts a major effect on immune –inflammatory response that results in an increase in an extent and severity of periodontal destruction. The toxicity of smoking appear to result from alterations in the immune-inflammatory response to bacterial challenge. Smoking altered the activities of neutrophils, lymphocytes and pro inflammatory mediators [13].

Effect of Smoking on fibroblast function

Gingival fibroblasts: There is evidence that gingival fibroblasts from smokers may be less susceptible to the cytotoxic effects of high levels of nicotine possibly because of the development of tolerance. There is reduction in cell viability and disruption to the microtubules, intermediate filaments and actin [14]. PDL fibroblasts Cell attachment was significantly less on root surfaces obtained from heavy smokers compared with non-smokers and healthy controls [15].

Effect of smoking on physiology

Earlier studies have shown that certain clinical signs of gingival inflammation are less pronounced in smokers than in nonsmokers due to alteration in vascular response of the gingival tissues [16]. The light smokers responded with a significant increase in blood flow which causes a gingival bleeding paralleling the changes but the heavy smokers showed no response, indicating a high level of tolerance. Smokers experienced less gingival bleeding than non-smokers [14].

III. EFFECTS OF SMOKING ON GINGIVITIS

Inflammation of the marginal gingival is a common condition and its extent and severity can be inconsistent. This condition known as gingivitis which can be modified by systemic and local influences and is plaque induced. It can be reversed if improved oral hygiene measures are introduced [17]. Smoking does not normally lead to remarkable gingival changes. In gingivitis reduction of clinical signs has been reported in smokers and this effect has been shown to be independent of plaque levels. Heavy smokers may have greyish discoloration and hyperkeratosis of the gingiva: an increased number of keratinized cells have been found in the gingiva of smokers than light smokers. Changes in the epithelium were described as keratotic, hyperkeratotic and hyperplastic [18]. Smoking has long been considered an etiologic factor in acute necrotizing ulcerative gingivitis (ANUG). Rowland in a series of studies determined that tobacco smoking was a factor in ANUG and that with the increase in the use of tobacco there was an increase in frequency of ANUG [19]. The tar in the smoke exerted a direct irritating effect on the gingiva giving rise to gingivitis, and that nicotine could cause contraction of the capillaries [20].

Periodontal Disease	Effects of Smoking
Gingivitis	↓ Gingival inflammation and bleeding on probing
Periodontitis	↑ Prevalence and severity of periodontal destruction ↑ Pocket depth, attachment loss, and bone loss ↑ Rate of periodontal destruction ↑ Prevalence of severe periodontitis ↑ Tooth loss ↑ Prevalence with increased number of cigarettes smoked per day ↓ Prevalence and severity with smoking cessation

Fig. 2: Effects of smoking on gingivitis and periodontitis ^[12]

IV. EFFECTS OF SMOKING ON PERIODONTITIS

Smoking is associated with excessive destruction of the supporting periodontal tissues (gingival recession), resulting in bone loss, pocket formation, and premature tooth loss. It is well documented in the literature that bone loss and attachment loss are significantly more pronounced in smokers compared to non-smokers ^[21]. Many authors investigated the relationship between cigarette smoking and the prevalence of periodontal pathogens using polymerase chain reaction techniques. In this study, which included equal numbers of smokers and non-smokers with generalised aggressive periodontitis, the investigators could discover no significant differences in the occurrence of any of the pathogenic species which included *Porphyromonas gingivalis*, *Prevotella intermedia*, *Tanarella forsythensis*, *Aggregatibacter actinomycetemcomitans* and *Tanarella denticola* ^[22]. Cigarette smokers had significantly greater probing depths and bone loss than non-smokers although no difference was found in relation to tooth mobility.

V. EFFECTS OF NON-SURGICAL PERIODONTAL THERAPY IN SMOKERS

Current smokers do not respond as well to periodontal therapy as non- smokers or former smokers. The majority of clinical research supports the observation that reduction in pocket depth is more effective in non- smokers than in smokers after non-surgical periodontal therapy, including oral hygiene instructions, scaling and root planing. Smokers respond less well to non-surgical therapy than nonsmoker ^[12]. Compared with non-smokers, smokers have less reduction of PD for the dentition as a whole. Compared with non smokers, the reduction in bleeding was less marked in smokers in spite of greater reduction of plaque index. Treated infra- bony defects are adversely affected in smokers c ompared with non-smokers. Smoking adversely affects treatment outcomes ^[23].

Table 1: Effect of smoking on response to nonsurgical therapy ^[5]

Treatment	Outcomes in smokers compared to non- smokers
Scaling and root planing; various forms of surgical periodontal therapy.	Smokers exhibit 50-75% as much improvement in clinical parameters.
Antimicrobial therapy	Adjunctive antimicrobial therapy brings the smokers response to that of non-smokers receiving scaling and root planning alone.
Sub- antimicrobial doxycycline therapy	Adjunctive sub-antimicrobial doxycycline treatment brings the smokers response to that of the non- smokers receiving scaling and root planning alone.
Site development and dental implant therapy	Smokers experience approximately twice the failure rate based on a variety of implant designs and surfaces.

VI. EFFECTS OF SURGICAL PERIODONTAL THERAPY IN SMOKERS

The less favourable response of the periodontal tissues is observed in surgical therapy in smokers ^[23]. Non-smokers showed more percentage of reduction in presurgical pocket depth than smokers. Open flap debridement surgery without regenerative or any grafting procedures is the most common surgical procedure used to assess the root and osseous surfaces. Smokers exhibit a less favourable post operative healing in terms of vertical and horizontal attachment gain. Smoking impairs healing of guided tissue regeneration treated infra-bony defect. Smokers respond less favourably to flap debridement surgery in terms of pocket depth reduction and attachment level gains especially in sites with deep pocket depth than non-smokers ^[24].

Smoking and implants

The increased risk of wound healing complications as well as the risk of peri-implantitis and increased implant failure rates is found in smokers ^[25]. Smoking shows more negatively impacts on implants placed in maxillary arch than in mandible. The percentage of maxillary implant failures among smokers (10.9%) was higher than that reported for non smokers or past smokers (6.4%) ^[5].

VII. SMOKING CESSATION

Dental treatment outcomes may be unpredictable for patients who continue to smoke and requires long term management. The financial burden may be increased when a more demanding treatment plan is required to achieve oral health. The most substantial decreases in management expenses may be achieved for patients who stop smoking at an early stage. Smoking cessation efforts are divided into two broad categories: pharmacological and behavioural. Pharmacological approaches currently include two general strategies: nicotine replacement and bupropion therapy. In April 2002, the National Institute for Clinical Excellence in the United Kingdom released guiding principle to health care professionals on the use of nicotine replacement

therapy (NRT) and non-NRT, such as bupropion (Zyban; Glaxo Wellcome, London, UK) for smoking cessation ^[26]. Nicotine replacement therapy reduces physical withdrawal symptoms. NRT products like Nicotine gum, transdermal patch, nasal spray, inhalers, sublingual tablets, and lozenges are available.

Concise advice for the patient to stop smoking may last for around 10 min.

A five-step program recommended by the Agency for HealthCare Research and Quality, which uses the five A's ^[27] –

1. Ask - Identify patients tobacco use status
2. Advice - On association between oral disease and smoking and the benefits of cessation.
3. Assess - Patients interest and readiness to participate in tobacco cessation programs
4. Assist - Use appropriate technique to assist patient in tobacco cessation
5. Arrange - Follow-up contacts with the patient

Smoking cessation is favorable to PDL treatment outcomes and periodontal health. Periodontal disease progression slows down in individuals who stop smoking. Smoking cessation restores the normal periodontal and microbial healing responses: the healing responses of ex smokers become similar to that of non smokers ^[28].

VIII. CONCLUSION

Tobacco smoking has general systemic effects, many of which may provide mechanisms for the increased susceptibility to periodontitis and the poorer response to treatment. As an environmental factor, smoking interacts with the host-microbial challenge. The host genetic and environmental interaction is of the highest importance. Smoking status and willingness to quit should be determined for all patients. Based upon the obvious broad health gains and the possible periodontal benefits derived from quitting the smoking habit, the skill to provide qualified smoking cessation counseling should be part of the armamentarium of every periodontal team.

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