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Smoking and Periodontal Treatment Outcome

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Abstract

Smoking has harmful consequences for the general health and the oral cavity. The consequences of smoking on the general health have been sufficiently documented, though the list of diseases linked to smoking continues to become longer. The health consequences causally linked to smoking include cancers and chronic diseases. Periodontitis belongs to the diseases linked to smoking. For smokers, inhaling chemical compounds that originate from tobacco burning is a severe health hazard. Chronic nicotine consumption develops nicotine addiction. Moreover, secondhand and thirdhand smoking entails risks. The first aim of the present study was to review the diseases that are nowadays linked to smoking. The second aim of this study was to thoroughly review the literature related to the impact of smoking on the periodontal health, periodontal disease and periodontal treatment outcome. Smoking affects the inflammatory and immune response. Smoking is a risk factor for periodontitis. It negatively affects the presence and severity of periodontitis as well as the outcome of periodontal treatment. Smokers respond less favorably than non-smokers to non-surgical and surgical periodontal treatment. In terms of periodontal maintenance, disease recurrence and progression are common in smokers. The risk for progression of periodontitis is higher for smokers. It seems that smokers have reduced compliance with scheduled supportive periodontal treatment visits. The negative impact of smoking on the periodontium indicates that in terms of the periodontium smokers might benefit from smoking cessation.

Keywords: Periodontitis; Periodontal treatment; Periodontal treatment outcome; Periodontal disease progression; Smoking; Tobacco; Nicotine; Diseases linked to smoking

Introduction

Cigarette smoking has been reduced since several decades ago. However, the epidemic of smoking continues. The quantity of nicotine consumption varies widely among people of different countries, country regions, races, ethnicities, educational levels and socioeconomic status. The initiation of smoking is usually early in life, which entails more prolonged exposure to nicotine and health risks.

Significant part of the risks of smoking originates from inhaling chemical compounds. These compounds are either contained in tobacco or are a result of tobacco burning. A great variety of chemicals are used to produce the cigarettes in the market. There are more than 7,000 chemicals and chemical compounds in tobacco smoke. Among them, there are numerous toxic or carcinogenic constituents. Smoke composition has been changed since several decades ago aiming at decreasing tar and nicotine smoke yields. Numerous physical processes and chemical reactions happen in the burning zone of a cigarette. There are liquid droplets in the cigarette smoke, which are suspended within a mixture of gases and semi-volatile compounds. During smoking, there are two kinds of smoke that differ in composition and properties. Specifically, the mainstream smoke is inhaled by the smoker and the side stream smoke is released into the environment from the lit end of the cigarette [1,2]. Nicotine, carbon monoxide, reactive oxidant substances (ROS), and acrolein are among the most significant cigarette smoke toxins [3,4].

Nicotine has psychoactive and addictive properties [3-5]. Addiction is most of the problem with nicotine [6]. Nicotine is the main addictive cigarette smoke constituent, though there are other chemicals that contribute directly or indirectly to the addictive effect of nicotine. Most smokers are addicted to nicotine. It has been suggested that the development of nicotine addiction is easier for children and adolescents than for adults [6]. For nicotine addicted smokers, nicotine absence often leads to nicotine craving and withdrawal symptoms [5]. With cigarette smoking, nicotine absorption from the lung and thereafter nicotine delivery to the brain are very rapid [5,7]. It is transported to brain within seconds. Brain delivery of nicotine results in the release of dopamine, which creates a heightened sense of alertness and contentment [5]. Nowadays, several cigarettes are more addictive than in the past. This is partly due to chemical compounds allowing nicotine to reach the brain more quickly [6].

There are several types of active nicotine consumption, such as cigarette smoking, smokeless tobacco chewing, water pipes and e-cigarettes [8]. Trends in tobacco use change and many people tend to use multiple tobacco products, particularly young people.

The consequences of smoking on the general health have been sufficiently documented, though the list of diseases linked to smoking continues to become longer. Therefore, the first aim of the present study was to review the diseases that are nowadays linked to smoking. The second and main aim of this study was to thoroughly review the literature related to the impact of smoking on the periodontal health, periodontal disease and periodontal treatment outcome.

The Impact of Smoking on the General Health

Based on the 2014 United States Surgeon General's Report on the health consequences of smoking [6], cigarette smoking has been causally linked to diseases of nearly all organs of the body, to overall diminished health status and to harm to the fetus. The health consequences causally linked to smoking include cancers and chronic diseases. Additionally, cigarette smoking impairs immune function. Chronic nicotine use activates multiple biological pathways through which smoking increases risk for disease. Cancers in the oropharynx, larynx, esophagus, trachea, bronchus, lung, stomach, liver, pancreas, kidney, ureter, cervix, bladder and colorectal as well as acute myeloid leukemia have been causally linked to smoking. Stroke, blindness, cataracts, age-related macular degeneration, aortic aneurysm, early abdominal aortic atherosclerosis in young adults, coronary heart disease, pneumonia, chronic obstructive pulmonary disease, asthma, atherosclerotic peripheral vascular disease, tuberculosis, diabetes, rheumatoid arthritis and periodontitis are among the chronic diseases that have been causally linked to smoking. Furthermore, female reduced fertility and male erectile disfunction have been causally linked to smoking. For women, the increased smoking prevalence seen as compared to the past led to significantly increased disease risks from smoking. Nowadays, women and men have equal risks from smoking to present lung cancer, chronic obstructive pulmonary disease, and cardiovascular diseases. Smoking during pregnancy negatively affects both the mother and the fetus. Orofacial cleft of the fetus has been causally linked to maternal smoking. There is sufficient evidence to infer that nicotine exposure during fetal development has lasting adverse consequences for brain development. There is sufficient evidence to infer that maternal smoking is related to preterm delivery and stillbirth. Concerning adolescents, the existing evidence suggests that nicotine exposure during adolescence may have lasting adverse consequences for brain development.

Based on the World Health Organization (WHO) estimate, one-third of the world's adult population is exposed involuntarily to cigarette smoke [9]. The main form of passive smoking is secondhand smoking. Secondhand smoking has been causally linked to cancer, respiratory, and cardiovascular diseases, and to adverse effects on the health of infants and children. Specifically, secondhand smoking has been causally linked to nasal irritation, stroke, lung cancer and coronary heart disease for the adults and to middle ear disease, respiratory symptoms, impaired lung function, lower respiratory illness, sudden infant death syndrome for the infants, children and adolescents. Thirdhand smoke, another form of passive smoking, is residual tobacco smoke contamination that remains after the cigarette is extinguished in the environment [10]. It consists of a mixture of volatile compounds and particulate matter that may be deposited or adsorbed on surfaces, including clothing, furniture, and upholstery [11]. With thirdhand smoke, toxic tobacco compounds, such as nitrosamines, remain in the environment [12]. Information on thirdhand smoke is very limited [12], especially on the possible health hazards for children [10]. It has been suggested that emphasizing that thirdhand smoke harms the health of children may be an important element in encouraging home smoking bans [10].

Smoking and Periodontal Tissues

There is convincing cross-sectional evidence that smoking correlates inversely with deteriorating periodontal health and that this association may be dose-dependent [13-19]. Smoking is related to more severe periodontal condition [20].

The effect of smoking on the subgingival microflora was explored early in the literature [21-23] with varying results. Later, Apatzidou et al., found in periodontitis similar subgingival microflora for smokers and non-smokers [24]. The impact of smoking on the periodontium is not mainly mediated by changes in the microflora. Immunological changes in the host play a critical role in periodontal disease occurrence and progression [25]. Smoking affects the inflammatory and immune responses, including neutrophil function, antibody production, fibroblast activities and inflammatory mediator production, as well as the microvasculature. The impact of smoking on innate immunity ranges greatly from topical to systemic changes [3]. Smoking can directly affect the ability of the host to control the infection [25]. In periodontitis, smokers have a rather suppressed inflammatory response and an altered host antibody response to antigenic challenge than non-smokers [24].

Smoking increases total white blood cell count in the systemic circulation, affects neutrophils more than all white blood cells [26,27] and elicits the most destructive neutrophil actions [28]. It induces systemic neutrophilia [7], decreases neutrophil chemotaxis [29] and phagocytosis [30-33] with a dose-dependent effect [34] and increases the circulating neutrophil elastase and metalloproteinases (MMPs) [35,36], though it does not affect gingival crevicular neutrophil number [37].

Smoking activates inflammatory cells [38], increasing the systemic levels of various inflammatory markers, such as C-reactive protein, fibrinogen, interleukin-6 and haptoglobin [39]. It impairs humoral immune response [25]. The total leucocyte count was highest among heavy smokers (>10 cigarettes/day) and significantly higher compared with non-smokers irrespective of periodontitis presence or absence, and was reflected in increased neutrophil numbers but not in lymphocyte or monocyte numbers [38]. It decreases the levels of IgG, especially of IgG2[40-42]. IgG2 levels are higher for non-smokers than smokers [43]. Smoking upregulates the expression of pro-inflammatory cytokines, such as interleukin-1 (IL-1) [44]. IL-1 genotype-positive smokers are more susceptible to severe chronic periodontitis [45]. The risk of severe clinical attachment level (CAL) loss was 4.5 times higher in IL-1 genotype-positive smokers compared to genotype-negative non-smokers. Among IL-1 genotype-negative individuals, smokers had 2.4 times higher chance of severe CAL loss than non-smokers. There is gene-environmental interaction between smoking and IL-1 genetic polymorphism [46].

The decrease in several pro-inflammatory cytokines, chemokines and T-cell regulators (interleukin-7 and interleukin-15) seen in smokers indicates the immunosuppressant effect of smoking contributing to an increased susceptibility to periodontitis [47]. Smoking suppresses certain Th1 responses, while it facilitates the generation of Th2 inflammation [48-52]. Tumor necrosis factor-alpha (TNFα) significantly differed between smokers and non-smokers [53]. Reactive oxidant substances of cigarette smoke activate epithelial cell intracellular signaling cascades that lead to inflammatory gene activation, such as IL-8 and TNFα [54,55]. The secretion of these inflammatory mediators promotes chronic immune cell recruitment and inflammation. Smoking activates oral cells so that chronic inflammation is enhanced [56]. Moreover, it might dysregulate innate immune responses in the oral cavity by modifying local Toll-like receptor (TLR) expression, distribution and activation, which permits the development of chronic inflammation [57,58]. Furthermore, smoking impairs the vasculature of the periodontium and the gingival blood flow [7].

The Impact of Smoking on the Periodontal Treatment Outcome

In smokers, clinical improvement with periodontal treatment has been documented following various therapeutic approaches. However, both extent and predictability of clinical improvement were significantly reduced with respect to non-smokers, even after corrections of oral hygiene levels [59-66]. Smokers respond less favorably than non-smokers to non-surgical [64,67] and surgical periodontal treatment [28,68-74].

In the short-term, both non-surgical and surgical approaches are less efficient in smokers than non-smokers [61,75]. Concerning the short-term response to non-surgical periodontal treatment, at 6 to 8 weeks smokers presented 0.7 mm less probing depth (PD) reduction and 0.4 mm less CAL gain than non-smokers [76] and at 6 months smokers had 0.9 mm less PD and 0.6 mm less CAL improvement at periodontitis sites (PD>5 mm, CAL>3 mm) than non-smokers [77]. Palmer and Soory stated that in non-surgical treatment, smoking is associated with poorer PD and CAL improvement (approximately 0.5 mm less) and that in most studies smokers have lower bleeding level at baseline, and following treatment there is similar bleeding reduction for smokers and non-smokers [78]. In a systematic review on smoking effect on non-surgical treatment, Labriola et al. found that for all sites the PD reduction was 0.13 mm greater in non-smokers than smokers (6 studies). CAL gain and bleeding reduction did not differ between smokers and non-smokers [69]. A longitudinal study over 6 years demonstrated a continuously inferior treatment outcome in smokers. Specifically, less PD and CAL improvement and greater horizontal CAL loss were found in smokers than non-smokers [62]. Mdala et al. in a two-year study showed that past and present smoking significantly increased PD and that current smokers were at higher risk of CAL loss [79].

An increased need for surgical treatment could be expected for smokers [80], since smoking has a negative impact on PD reduction after non-surgical treatment [62,75]. Kotsakis et al. [81] in a systematic review and meta-analysis on the impact of smoking on the clinical outcomes following periodontal flap surgical procedures found improved treatment effect among non-smokers compared with smokers. PD reduction in smokers and non-smokers ranged from 0.76 to 2.05 mm and 1.27 to 2.40 mm, respectively. CAL gain in smokers and non-smokers ranged from 0.29 to 1.6 mm and 0.09 to 1.2 mm, respectively. Meta-analysis demonstrated for non-smokers an increased PD and CAL improvement of 0.39 and 0.35 mm, respectively. They concluded that smokers could be candidates for periodontal flap surgical procedures. Though, the magnitude of the therapeutic effect is compromised in smokers compared with non-smokers. Therefore, smokers should be encouraged to abstain from smoking and should be thoroughly informed preoperatively of substantial reduction in clinical outcomes.

Concerning endosseous defects, a Consensus Report from the American Academy of Periodontology (AAP) Regeneration Workshop in 2015 concluded that the outcomes of periodontal regeneration are negatively affected by smoking [82]. Patel et al. in their systematic review and meta-analysis studied the effect of smoking on periodontal bone regeneration in endosseous defects [83]. Six of the 10 studies concluded that smoking significantly negatively influenced post-treatment bone gain or fill [84-87]. Ehmke et al. reported mean bone gain of 0.2 mm in smokers compared to 2.2 mm in non-smokers after the use of bio absorbable membrane [87]. Heden reported bone gain of 2.6 mm in smokers compared to 3.3 mm in non-smokers after the use of Emdogain (EMD) [85]. Two of the 10 studies reported no significant difference in bone gain between smokers and non-smokers after EMD [88] or beta-tricalcium phosphate (b-TCP) and recombinant human platelet-derived growth factor [89]. The meta-analysis by Patel et al. showed a standardized mean difference of -2.05 (95% CI: -2.64 to -1.47) in probing bone level change for smokers and non-smokers after regenerative treatment [83]. When the long-term stability of CAL following guided tissue regeneration (GTR) and conventional therapy was evaluated, it was found that most patients with further CAL loss were non-compliant and smokers, while most patients with stable periodontal conditions were compliant and non-smokers [90].

Smoking adversely affects GTR outcomes for root coverage purposes in terms of recession reduction, root coverage, CAL gain and probing-bone improvement [91]. Concerning smoking effect on the outcomes achieved by root-coverage procedures, a meta-analysis by Chambrone et al. showed that the use of subepithelial connective-tissue (CT) graft was less effective in smokers than non-smokers in reducing root exposure and improving CAL. There was greater chance of achieving complete root coverage in non-smokers (27 to 80%) than smokers (0 to 25%). After CT graft, smokers had fewer sites exhibiting complete root coverage than did non-smokers [72]. It has been reported that the percentage (%) of mean root coverage at 6 months after CT graft varied according to the level of cotinine in saliva (84.2% for 10- to 40-ng/mL cotinine levels and 76. % for cotinine levels >40 ng/mL) and that higher cotinine levels were negatively associated with root coverage [92]. Recurrence of gingival recession after root coverage procedures was more evident for smokers than non-smokers [93,94]. Concerning the smoking effect on the outcomes achieved by coronally advanced flap for root-coverage, Chambrone et al. [72] in their meta-analysis found similar results between smokers and non-smokers. Though, there were only two studies available for analysis [94,95] that differed in design. It should be emphasized that in one [94] of them the long-term (two-year) healing response was affected by smoking.

Smoking and Periodontal Maintenance

Periodontal treatment leads to clinical improvement both in smokers and non-smokers, though disease recurrence and progression is common in smokers. Cortellini et al. in 1996 showed that in the long-term most patients who lost CAL after active treatment were non-compliant and smokers [90]. Kaldhal et al. in 1996 reported deterioration of the periodontal condition during periodontal maintenance for heavy smokers [96]. Tonetti et al. in 1998 found that during periodontal maintenance, the prevalence of bleeding residual pockets was significantly increased for smokers as compared with never smokers and former smokers [97]. Ah et al. showed that self-reported smokers exhibited less CAL and PD improvement over each of 6 years of maintenance compared with self-reported non-smokers, though these differences were small (<0.5 mm) and their clinical importance was questioned [62]. Furthermore, the same research group found that, over 7 years of regular maintenance, self-reported light and heavy smokers exhibited less CAL and PD improvement compared with non-smokers or former smokers. The clinical importance of these differences was limited, since they were small for the entire 7-year maintenance period (<1 mm) [98]. In a relatively recent study smoking had a negative periodontal effect at 12 months of maintenance. It affected the presence of sites with bleeding residual (PD ≥ 5 mm) pockets. In smokers, bleeding on probing (BOP) at 3

months after active treatment was a strong site-specific predictor for residual pockets with BOP at 12 months of maintenance [99].

Data reveals that following a strict periodontal maintenance care program is imperative for smokers. Specifically, Papantonopoulos, in well-maintained advanced periodontitis patients showed absence of significant difference in PD and radiographically imaged bone loss over a 5 to 8-year period between smokers and non-smokers [68]. Fisher et al. in a 3-year study of well-maintained chronic periodontitis patients failed to demonstrate any statistically significant differences in disease progression (CAL and PD), inflammatory indices (PI and BOP) and tooth loss between current smokers and validated current non-smokers. Hence, in this small study of highly motivated individuals receiving optimal care in a hospital clinic, regular maintenance treatment seemed equally successful in preventing progressive periodontal destruction in current smokers and current non-smokers with chronic periodontitis [100].

In McGuire and Nunn's study heavy smoking was significantly related to tooth loss in patients under periodontal maintenance for 14 years. Most of these patients were compliant with maintenance visits, though presenting fair oral hygiene [101]. In terms of tooth loss due to periodontitis during maintenance, it has been found that smokers (≥ 10 cigarettes per day) might have almost five times greater risk of tooth loss even while complying with strict periodontal maintenance [102].

In terms of aggressive periodontitis, smoking was the main significant risk factor for disease recurrence during maintenance (defined as the occurrence of PD ≥ 5 mm at ≥ 30% of the teeth) [103]. Furthermore, nearly significant correlations were reported between smoking and tooth loss in aggressive periodontitis [104].

The influence of smoking on compliance with scheduled supportive periodontal treatment (SPT) visits has not been thoroughly explored, since there is only limited information on this [80,105]. Ramseier et al. found that smokers tend to return less likely for scheduled SPT visits than non-smokers or former smokers. Even after adjusting for possible confounders, such as gender, age, disease severity or active treatment duration, the effect of smoking remained significant [106]. Their finding on reduced compliance of smokers with scheduled SPT visits agrees with previous findings on higher frequency of smokers in the non-compliant group [80,105].

Smokers had greater risk for further CAL loss (odds ratios of 2.3 to 4.8 for light to heavy smokers, respectively) [107] and for further alveolar crest height loss (3.2 to 7.3 for light to heavy smokers, respectively) [108]. Smoking is a risk factor for further progression of periodontitis during maintenance. The strong relation between smoking and periodontitis progression should be taken under consideration for the risk assessment of disease progression at the patient level in periodontal maintenance. During maintenance, clinicians should recall smokers more frequently than non-smokers [20] and they should maintain their efforts to verify that smokers keep their scheduled SPT appointments [106].

The harmful effect of smoking on the periodontium demonstrates that non-smokers should not start the smoking habit and that smokers should quit smoking. Dentists may play an important role in the smoking cessation effort by advising and motivating all patients who smoke to quit smoking and supporting the attempting to quit patients in achieving their goal [109].

Conclusion

Smoking is harmful for the general health and the oral cavity. The list of cancers and chronic diseases causally linked to smoking is long and keeps increasing. In terms of the periodontium, smoking negatively affects the presence and severity of periodontal disease and the outcome of periodontal treatment. Both for smokers and non-smokers, there is clinical improvement with periodontal treatment. Though, smokers respond less favorably than non-smokers to non-surgical periodontal treatment, to flap surgical procedures, to periodontal regeneration in endosseous defects and to root coverage procedures. Periodontal disease recurrence and progression is common in smokers. Smokers present higher risk of periodontal disease progression. Therefore, they should be recalled more frequently, and the dentist should verify that they keep their scheduled periodontal maintenance appointments. The negative impact of smoking on the periodontium indicates that in terms of the periodontium smokers might benefit from smoking cessation.

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