

Comparison of Periodontal Conditions Between Smokers and Nonsmokers

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Received 2015 February 22; Revised 2015 July 12; Accepted 2015 July 15.

Abstract

Background: For the last decades, dental researchers have believed that smoking is a major risk factor for periodontal disease, affecting the prevalence, extent, and severity of disease. In addition, smoking adversely affects the clinical outcome of nonsurgical and surgical therapy. This study aims to evaluate the effects of smoking on oral health. In addition, due to the lack of studies that have simultaneously compared the periodontal condition in healthy smokers and smokers with periodontal disorders with healthy nonsmokers and nonsmokers with periodontal disorders, we assessed the periodontal condition in these four groups.

Objectives: Assess the periodontal condition in healthy smokers and smokers with periodontal disorders and compare these conditions with nonsmokers.

Patients and Methods: This historical cohort study included four groups: healthy smokers, smokers with periodontal disorders, healthy nonsmokers and nonsmokers with periodontal disorders. Each group consisted of 20 men with an age range of 20 - 30 years, according to the group specifications. The parameters assessed in this study included: plaque control record (PCR), bleeding on probing (BOP), probing depths, clinical attachment level (CAL), gingival color, and gingival consistency.

Results: There was not a significant difference in the prevalence of isolated microorganisms between the smokers and nonsmokers. However, the cigarette smoking group had adverse effect on other periodontal indices including PCR, CAL, and BOP. The Mean PCR and CAL were significantly higher in the two smoker subgroups than the nonsmokers ($P < 0.05$). Regarding gingival color, red and bluish-red colors were observed more in those with periodontal disease compared to healthy individuals, regardless of cigarette smoking ($P = 0.000$). Also, the firm gingival consistency was more frequent in healthy subjects and a spongy pattern was detected more in subjects with periodontal disease, regardless of smoking ($P = 0.000$). The BOP percentage in smokers was significantly lower than in nonsmokers ($P = 0.000$).

Conclusions: Cigarette smoking increased some periodontal indexes including PCR and CAL, and reduced BOP ($P < 0.05$).

Keywords: Smoker, Nonsmoker, Periodontal Conditions, Plaque Control Record, Bleeding on Probing, Clinical Attachment Level, Probing Depths

1. Background

The pathological nature of periodontal disease is certainly based on inflammatory responses to pathogens and destructive materials (1, 2). The outcome of these responses is usually manifested by irreversible destruction of the connective tissue attachments leading to periodontal pocket formation and eventual loss of alveolar bone (1-3).

For the last decades, dental researchers have believed that smoking is the major risk factor for periodontal disease, affecting the prevalence, extent, and severity of disease. In addition, smoking adversely affects the clinical outcome of nonsurgical and surgical therapy (1, 4). It has been shown that smoking is a main risk factor for periodontal diseases, after setting of potential confounding

factors, for example, age, oral hygiene, gender, and socioeconomic status (5).

The harmful effects of smoking on oral and dental systems seem to be related to its different compounds including noxious substances and even carcinogens (6-9). Moreover, some components, such as nicotine, are quickly absorbed by the lungs and reach the brain within 10 to 19 seconds. Nicotine is highly addictive (10). It causes a rise in blood pressure, increased heart and respiratory rates, and peripheral vasoconstriction. This leads to contraction of oral capillaries affecting periodontal tissue and gingival blood flow (6). Smoking is the major risk factor for periodontitis, and it affects the prevalence, extent, and severity of disease. In addition, smoking has an adverse impact on the clinical outcome of nonsurgical and surgical therapy, as well as the long-term success of implant placement (10).

In addition, nicotine can concentrate in the periodontium and result in the impairment of the functional activity of inflammatory active cells, predisposing the patient to bacterial infection (11, 12).

Controlled clinical studies have demonstrated that, in human models of experimental gingivitis, the development of inflammation in response to plaque accumulation is reduced in smokers as compared with nonsmokers (10). In addition, cross-sectional studies have consistently demonstrated that smokers present with less gingival inflammation than nonsmokers (13). These data suggest that smokers have a decreased expression of clinical inflammation in the presence of plaque accumulation as compared with nonsmokers (10). In contrast, pocket depth, attachment loss, and alveolar bone loss are more prevalent and severe in patients who smoke as compared with nonsmokers (4).

Besides the pointed destructive effects of smoking on periodontal tissues, it has been epidemiologically indicated that smokers have poorer oral hygiene and less favorable tooth brushing habits (14, 15). These habits can lead to greater plaque accumulation in smokers than in nonsmokers (16-18).

2. Objectives

Recent studies on the impact of tobacco on periodontal health mainly concern cigarette smoking. To further evaluate the effects of cigarette smoking on oral health, and due to the lack of studies that simultaneously compared the periodontal condition in healthy smokers, smokers with periodontal disorders, healthy nonsmokers and nonsmokers with periodontal disorders, we assessed the periodontal condition in these four groups.

3. Patients and Methods

This historical cohort study included four groups: healthy smokers, smokers with periodontal disorders, healthy nonsmokers and nonsmokers with periodontal disorders. Each group had 20 men with an age range of 20-30 years, according to the specifications of each group. Smoker groups experienced cigarette smoking for at least three months and had no history of smoking other substances. None of the smokers used antibiotics or mouthwash within the three months prior to the study. Baseline information was collected by interviewing and oral examination. For checking gingival status, a Williams periodontal probe and dental mirror were used, and all gingival surfaces were assessed. All measurement was performed using the same Williams probe and graded from 1 to 10 mm.

The study method was approved by the ethical committee of the Hamadan University of Medical Sciences and informed consent was obtained from all participants.

The parameters assessed in this study were: plaque control record (PCR), bleeding on probing (BOP), probing depths, clinical attachment level (CAL), gingival color, and gingival consistency. For assessment of plaque control record (PCR), following the consumption of a disclosing tablet and appearance of plaques, the four dental surfaces of buccal, lingual, mesial, and distal were evaluated. PCR was calculated by the following formula:

$$\text{PCR}(\%) = \frac{\text{number of colored surfaces}}{\text{number of all teeth} \times 4} \times 100 \quad (1)$$

Gingival bleeding was also assessed using the gingival bleeding index (GBI) using the following formula:

$$\text{GBI}(\%) = \frac{\text{number of sites with bleeding}}{\text{number of all teeth} \times 4} \times 100 \quad (2)$$

For assessing clinical attachment level, the selected reference was CEJ. The distance between pocket depth and CEJ was determined in six medial, mesial, and distal sites of the buccal and lingual surfaces using the Williams periodontal probe.

For assessing gingival color, the dominant gingival color (pale pink, dark pink, red, bluish-red) on the buccal surface of the teeth was considered, and gingival consistency was considered to be: firm, spongy and fibrotic.

Based on oral examination, the deepest pocket with bleeding and maximum clinical attachment loss was selected for microbiological testing. After elimination of supragingival plaque sediments, sampling sites were isolated with cotton rolls and were gently air-dried. Finally, sampling was performed using a sterile paper point and transferred to the culture medium. The transitional environment for absolute anaerobic bacteria was thioglycolate 0.18%. Meanwhile, a direct smear was prepared from probing to evaluate *Spirochetes*. Biochemical tests were used to diagnose different isolates, and smears were prepared from suspected colonies for gram staining.

Results were presented as mean \pm SD for quantitative variables and were summarized by absolute frequencies and percentages for different variables. Different variables were compared using a chi-square test or Fisher's exact test when more than 20% of the cells, with the expected count of less than five, were observed. Quantitative variables were also compared with a t-test. Statistical significance was determined as a $P < 0.05$. All statistical analysis was performed using SPSS software.

4. Results

Dental examination data were available for all participants and none of them refused periodontal examination.

There was not a significant difference in the prevalence of isolated microorganisms between the smokers and non-smokers ($P = 0.669$) (Table 1). However, cigarette smoking had adverse effects on other periodontal indices including PCR, CAL and BOP. The mean PCR and CAL were significantly higher in the two smoker groups than in the non-smokers ($P < 0.05$). Considering gingival color, the red and bluish-red colors were observed more in participants with periodontal disease (smoker, nonsmokers) compared to healthy individuals (smoker, nonsmokers) ($P = 0.000$), regardless of cigarette smoking. Also, with regard to gingival consistency, a firm pattern was more frequent in healthy subjects (no periodontal disease) ($P = 0.000$) and a spongy pattern was detected more in participants with periodontal disease ($P = 0.000$), regardless of smoking. The BOP percentage in smokers was significantly lower than non-smokers ($P = 0.000$). In total, cigarette smoking induced an increase of some periodontal indexes, including PCR and CAL, and a reduction of BOP.

5. Discussion

In the present study, assessing periodontal and gingival features in smokers in comparison with nonsmokers, regardless of the presence of periodontal disorders, showed significant changes in some periodontal indices including PCR, CAL and BOP in smokers. Interestingly, we showed that the parameters of PCR and CAL increased following cigarette smoking ($P < 0.05$). We also found a higher incidence of plaque formation in smokers, indicated by the CAL index. Similar to our study, MacGregor (17, 18) evaluated the area of stained plaque, and the ratio of gingival margin in touch with plaque in smokers and non-smokers who were matched for age and sex. In both sexes, smokers had significantly more plaque than nonsmokers, and there was a correlation between increased plaque accumulation with the increase of smoking. Also, Torrungruang et al. (19) showed that current smokers had a higher percentage of sites with plaque. However contrarily, Feldman et al. (20) found significantly less plaque in smokers than in nonsmokers. Furthermore, similar to our observation, in a study by Albandar et al. (3), cigarette smokers had a higher and more severe extent of attachment loss and gingival recession than nonsmokers.

It has been demonstrated that smokers have a diminished response to periodontal therapy and show approximately half as much improvement in probing depths and clinical attachment levels, following non-surgical and various surgical modalities of therapy (4). The increased prevalence and severity of periodontal destruction associated with smoking suggests that the host-bacterial interaction normally seen in chronic periodontitis is altered,

resulting in more extensive periodontal breakdown (10). This imbalance between bacterial challenge and host response may be caused by changes in the composition of the sub-gingival plaque, with an increase in the number and virulence of pathogenic organisms changing the host response to the bacterial challenge, or a combination of both. Studies have failed to demonstrate a difference in the rate of plaque accumulation of smokers as compared with nonsmokers (10). This suggests that, if an alteration in the microbial challenge in smokers exists, it results from a qualitative rather than a quantitative alteration in the plaque (10). Of particular interest was the observation that smokers do not respond to mechanical therapy as well as nonsmokers do; this is associated with increased levels of *T. forsythia*, *A. actinomycetemcomitans*, and *P. Gingivalis* remaining in the pockets after therapy in the smoking group as compared with nonsmokers (10).

Smoking, through alteration of the oxidation-reduction potential in periodontal pockets, provides a suitable environment for anaerobic microorganisms, would perform the deposits of a more pathogenic plaque (10). Nonetheless, in vivo proof for an altered composition of plaque is meager. Based on the findings of poorer hygiene status in smokers than nonsmokers, more plaque formation in the former group seems to be predictable.

We also revealed lower BOP in smokers than nonsmokers ($P = 0.000$). It was seen also, in the Bergstrom, et al study (21), that the clinical signs of inflammation, such as gingival bleeding, are less pronounced in smokers than in nonsmokers. This was observed probably due to alteration in the vascular response of the gingival tissue, such as vasoconstriction of gingival vessels, as well as to the higher keratinization of the gingivae in smokers. Palmer et al (22), using a laser Doppler technique, showed no significant difference in blood flow of the periodontal tissues between smokers and nonsmokers. Smokers have also been associated with reduced permeability of peripheral blood vessels (23). The BOP index is now widely used in clinical examination as a means for determining active sites in periodontal disease. It has been theorized that nicotine from cigarettes actuates the sympathetic system to produce catecholamine that causes vasoconstriction (24). The vascular contraction actions of nicotine may be the cause of the decreased gingival blood flow (8).

Interestingly, smoking had no significant effects on prevalence of periodontal microbial isolates compared with nonsmokers ($P > 0.05$). Tobacco smoke receptacle phenols and cyanides, which can agent for antibacterial and toxic properties (8). However, there are other studies that show a higher relative risk for infection with a variety of bacteria (24). In this context, more assessment of the effects of smoking on susceptibility to bacterial infections

Table 1. Periodontal and Gingival Patterns in Smokers and Nonsmokers^a

Index	Healthy Smoker	Patient Smoker	Healthy Nonsmoker	Patient Nonsmoker	P Value ^b
PCR	32.665	74.322	40.335	81.893	< 0.001
CAL					
Anterosuperior region	1.472	2.431	1.023	2.106	< 0.001
Anteroinferior region	1.465	2.848	0.875	2.105	< 0.001
Gingival color					
Pale pink	15 (75.0)	1 (5.0)	17 (85.0)	0	< 0.001
Dark pink	5 (25.0)	1 (5.0)	2 (10.0)	1 (5.0)	0.258
Red	0	12 (60.0)	1 (5.0)	8 (40.0)	0.002
Bluish-red	0	6 (30.0)	0	11 (55.0)	0.001
Gingival consistency					
Firm	17 (85.0)	3 (15.0)	19 (95.0)	0	< 0.001
Spongy	5 (25.0)	17 (85.0)	0	20 (100)	< 0.001
Fibrotic	3 (15.0)	0	1 (5.0)	0	0.140
Microorganisms					
<i>A. Comitans</i>	5 (25.0)	5 (25.0)	2 (10.0)	5 (25.0)	0.705
<i>P. Gingivalis</i>	6 (30.0)	8 (40.0)	6 (30.0)	6 (30.0)	0.953
<i>Spirochetes</i>	5 (25.0)	13 (65.0)	3 (15.0)	8 (40.0)	0.136
<i>P. Intermedia</i>	4 (20.0)	6 (30.0)	7 (35.0)	6 (30.0)	0.882
BOP					
0 - 0	2 (10.0)	0	0	0	0.133
10 - 20	10 (50.0)	2 (10.0)	2 (10.0)	0	0.005
20 - 40	8 (40.0)	12 (60.0)	17 (85.0)	0	0.004
40 - 60	0	5 (25.0)	1 (5.0)	12 (60.0)	0.002
60 - 80	0	1 (5.0)	0	7 (35.0)	0.003
80 - 00	0	0	0	1 (5.0)	0.408

^aN = 20.^bP value of t-test for comparison of two groups (smokers and nonsmokers).

should be considered.

Acknowledgments

We thank the research center of the school of dentistry who cooperated with us in doing this dissertation work.

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