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Effect of Cigarette Smoking on Periodontal Status of Diabetic Patients

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Abstract

Background: Periodontitis is an inflammatory disease of the tooth-supporting structures that can lead to periodontal destruction and tooth loss. It is also a common complication of diabetes mellitus (DM) and tobacco smoking. In this regard, this study aimed to assess the effect of smoking on periodontal disease in diabetic patients.

Methods: This case-control study was conducted on 80 diabetic patients who were referred to the clinics of the Department of Periodontics of Ardabil University of Medical Sciences from October 2015 to April 2016. Participants were enrolled in this study in four groups (n=20). Groups 1 and 2 included smoker diabetic patients and 20 non-smoker diabetics, respectively. In addition, groups 3 and 4 served as the control groups and included healthy smoker and non-smoker individuals, respectively. The plaque index (PI), clinical probing depth (CPD), clinical attachment level (CAL), and bleeding on probing (BOP) were measured in the four groups.

Results: The four groups were significantly different regarding the PI and CPD ($P<0.05$). The mean PI was higher in group 1 compared to groups 2 and 3. The highest mean CAL was recorded in group 1. Finally, non-diabetic smokers experienced the lowest mean BOP compared with other groups.

Conclusion: DM and tobacco smoking are the known major risk factors for periodontal disease, and the interaction effect of the two factors can aggravate the periodontal status in diabetic patients. Thus, dentists can take an important step in the healthcare system by encouraging their patients to control their DM and quit smoking.

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Background

Diabetes mellitus (DM) has been recognized as a common health dilemma worldwide. DM changes the cellular microenvironment and causes extensive complications in various organs by affecting the blood vessels, referred to as angiopathy. The microangiopathic process in the oral cavity can accelerate the destruction of periodontal tissues and increase the risk of tooth loss. Periodontitis is currently the sixth most common complication of DM thus DM patients are at the risk of periodontal disease (1-3).

Periodontal disease is a common disorder that arises from the chronic infection of gingiva and dental supportive structures (4,5). Periodontitis can cause the degradation of collagen fibers in the periodontal ligament, resulting in gingival recession and alveolar bone loss. It is a high-prevalence disorder affecting approximately 20%-50% of pregnant women (6,7).

Chronic periodontitis can damage the periodontal ligament and result in alveolar bone loss at the site of involvement. In the case of no treatment, periodontitis can lead to tooth mobility due to the loss of supporting structures and subsequent tooth loss (8-10). It can cause bacteremia, which subsequently activates the host response pathways and leads to vascular changes and atherosclerosis in many organs. Recent studies have suggested that periodontitis can induce a biological burst of endotoxins and inflammatory cytokines that initiate and exacerbate angiogenesis and thromboembolic events (1,11,12).

Smoking, systemic disease, steroids, anti-epileptic drugs, cancer drugs, contraceptive pills, pregnancy, severely crooked teeth, and ill-fitting bridges are risk factors for periodontal diseases (13,14).

Tobacco smoking enhances the plaque accumulation and overgrowth of bacteria in periodontal pockets, inhibiting

Highlights

- The results showed that the PI was significantly different between the groups
- ANOVA was used to compare the CPD and its results demonstrated that the difference in this respect was statistically significant between the groups
- The difference in CAL was statistically significant between the groups.
- Smoker non-diabetic individuals had the lowest mean BOP while the highest BOP was found in non-smoker diabetic patients

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the antimicrobial function of neutrophils and monocytes, resulting in alterations in the normal microbial flora of the oral cavity and a reduction in the serum immunoglobulin G level. On the other hand, both DM and smoking can independently cause the formation and deposition of advanced glycation end products in the periodontal tissue. These products interfere with the chemotactic and phagocytic function of polymorphonuclear cells and commence an inflammatory process through the secretion of cytokines and destruction of the periodontal tissue (15-18).

Considering the scarcity of studies on the effects of smoking on the periodontal status of diabetic patients, this study sought to evaluate the effect of smoking on the periodontal status in diabetic patients.

Materials and Methods

This case-control study was performed on 80 diabetic patients who were referred to the clinics of the Department of Periodontics of Ardabil University of Medical Sciences from October 2015 to April 2016.

Inclusion and Exclusion Criteria

The inclusion criteria were having diabetes and periodontal disease. Pregnancy, breastfeeding, history of periodontal therapy in the past 6 months, intake of corticosteroids or antibiotics, systemic disorders other than DM, and hemorrhagic disorders were the exclusion criteria. Patients with less than 20 teeth and alcoholics were also excluded from the study.

Study Design

Patients were categorized into four age- and gender-matched groups. Groups 1 and 2 consisted of 20 smoker and 20 non-smoker diabetic patients, respectively. Groups 3 and 4 served as the control groups and included 20 healthy smoker and non-smoker individuals, respectively. The glycated hemoglobin (HbA_1C) level > 6.5% was considered as the diagnostic cutoff point for DM (1). Patients were thoroughly examined and enrolled in the study. Age, gender, and smoking status of all individuals were recorded in previously prepared forms, and those who smoked more than one cigarette per day at least in the past year were considered as smokers.

Patients underwent clinical periodontal examinations including measurement of the plaque index (PI), clinical probing depth (CPD), clinical attachment level (CAL), and bleeding on probing (BOP). The PI was measured according to the Silness-Löe PI. Therefore, scores 0-3 were assigned to the absence of microbial plaque, a thin film of microbial plaque along the free gingival margin, moderate accumulation of plaque in the sulcus, and a large amount of plaques in the sulcus or pocket along the free gingival margin, respectively.

CPD was measured and recorded for all available teeth (except for the second and third molars) in four regions (i.e., mesiobuccal, midbuccal, distobuccal, and midpalatal/lingual) using a Williams probe according to the standard method. The probe was inserted into the periodontal pocket parallel to the longitudinal axis of the tooth. The applied force to the probe was limited such that the probe did not pass through the junctional epithelium (a force equal to 0.75 N).

To evaluate the CAL, the distance from the cementoenamel junction to the base of the periodontal pocket in the buccal surface was measured and recorded using a Williams probe for all available teeth except for the second and third molars.

BOP was measured according to the Ainamo and Bay gingival bleeding index. The presence/absence of bleeding was evaluated by the gentle probing of the gingival crevice using a Williams probe. The occurrence of bleeding within 10 seconds following probing was considered as a positive bleeding index. In each patient, the BOP was computed for all teeth. Then, the number of positive values was divided by that of all teeth to calculate the mean BOP.

Statistical Analysis

In the present study, SPSS software (version 24) was used to analyze the data. The mean and standard deviation, as well as frequency and percentage were applied to describe quantitative and qualitative variables, respectively. The data normality was analyzed using the Kolmogorov-Smirnov test. Statistical analysis was carried out using ANOVA and Tukey's HSD post hoc tests, and a P value lower than 0.05 was considered statistically significant.

Results

This study evaluated 80 patients who were divided into four groups of smoker diabetic patients (n=20), non-smoker diabetics (n=20), healthy smoker (n=20), and non-smoker individuals (n=20). The study population included 52 (65%) males and 28 (35%) females and their mean age was 45.01±13.82. Table 1 presents the mean age and gender frequency in the four groups. There was no significant difference between the groups regarding gender (P<0.05). The mean of the HbA1C level was 9.21 ± 0.32 and 8.89 ± 0.74 mmol/mol, respectively. The means of the PI, PD, CAL, and BOP in the four groups are provided in Table 2.

Table 3 summarizes data of the pairwise comparisons of the groups for different clinical periodontal parameters. The results showed that the PI significantly differed between the groups (P<0.05) such that the mean PI was higher in group 1 compared to groups 2 and 3. Although pairwise comparisons between the groups using Tukey's HSD post-hoc test revealed that the difference in the PI was not statistically significant between groups 2 and 3 (P=0.99), this difference was significant between other groups (P<0.05). Moreover, CAL in the smoker diabetics

Table 1. Demographic Information of Subjects in the Four Groups

Variables		Smoker Diabetic		Non-smoker Diabetics		Healthy Smoker		Non-smoker Individuals	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age		55.75	2.07	51.75	2.36	39.71	2.50	32.85	2.41
		No.	%	No.	%	No.	%	No.	%
Gender	Male	17	85	7	35	18	90	10	50
	Female	3	15	13	65	2	10	10	50

Note. SD: Standard deviation.

Table 2. Clinical Periodontal Findings in the Four Groups

Variables	Smoker Diabetic		Non-smoker Diabetics		Healthy Smoker		Non-smoker Individuals	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Plaque index	2.47	0.09	2	0.1	1.97	0.13	1.28	0.09
Pocket depth (mm)	2.15	0.09	2.03	0.12	1.84	0.11	1.6	0.07
Clinical attachment level (mm)	3.36	0.12	2.62	1.67	1.25	0.14	0.69	0.07
Bleeding on probing	0.92	0.1	1.20	0.17	0.91	0.2	1	0.08

Note. SD: Standard deviation.

Table 3. Pairwise Comparisons of the Groups for Different Clinical Periodontal Parameters

Groups	*P* Value			
	Plaque Index	Pocket Depth	Clinical Attachment Level	Bleeding on Probing
Group I vs. II	0.01	0.84	0.001	0.27
Group I vs. III	0.009	0.16	<0.001	0.99
Group I vs. IV	<0.001	0.002	<0.001	0.95
Group II vs. III	0.99	0.57	<0.001	0.24
Group II vs. IV	<0.001	0.02	<0.001	0.57
Group III vs. IV	<0.001	0.33	0.02	0.93

Note. Group I: Smoker diabetics; Group Ii: Non-smoker diabetics; Group Iii: Smokers without diabetes mellitus; Group Iv: Non-smokers without diabetes mellitus.

was significantly different from non-smoker diabetics, healthy smokers, and healthy non-smokers. The highest mean CAL was recorded in smoker diabetics. Finally, no significant difference was observed between the four groups regarding BOP, and smoker non-diabetic individuals had the lowest mean BOP.

Discussion

In summary, our findings demonstrated that the means of PI and CAL were higher in smoker diabetic patients compared to non-smoker diabetics, healthy smoker, and non-smoker individuals. Healthy smokers and non-smoker individuals represented no difference in terms of the PI. The highest means of CAL and PD were recorded in smoker diabetic patients. No significant difference was observed between the four groups regarding BOP. Smoker non-diabetic individuals had the lowest mean BOP.

Periodontal disease, most importantly periodontitis, can affect all populations regardless of individuals' gender, race, and ethnicity. Tobacco smoking and DM are both considered as etiologic factors for the development of periodontitis. However, the interaction effect of DM and tobacco smoking on periodontal status has not been confirmed yet. It is assumed that both hyperglycemic state and tobacco smoking contribute to the formation and accumulation of advanced glycation end products in periodontal tissues, which compromise periodontal health.

According to the current results, the mean PI was higher in smokers and diabetic patients in comparison with non-smoker individuals, which can be attributed to the role of DM in advanced glycation end product production, along with poor oral hygiene in smokers. Similar to our study, Kanmaz et al found higher mean PD and CAL values in the smoker group compared to non-smoker individuals (19). Based on the literature, higher PI values were reported among smokers and diabetic patients compared with healthy non-smoker individuals. In general, periodontal disease is attributed to both poor oral hygiene and the socioeconomic status of smokers (20-22). However, some other studies failed to find a statistically significant relationship between tobacco smoking or DM

with periodontal disease (23,24). The controversy in the literature in this regard may be due to the difference in study populations, study methods, or other confounding variables.

Similarly, Hodge et al showed that periodontal inflammatory conditions are worse among diabetic smokers compared to non-smokers (25). Both hyperglycemia and habitual smoking increase the formation of advanced glycation end products in the periodontal tissues (26). This explains similar periodontal inflammatory parameters among smokers and non-smokers with diabetes.

Based on the results of the pairwise comparisons of CPD, there was no statistically significant relationship between CPD and cigarette smoking although the difference in CPD was statistically significant between diabetic and non-diabetic individuals, which is in line with the findings of several other studies (20,22-24). Meanwhile, the smoking habit has been shown to affect the CPD according to some studies (20,24). According to Javed et al, tobacco smoking plays a role in periodontitis by increasing periodontal pocket pathogens, decreasing the serum level of immunoglobulin G (especially IgG_2) while increasing pro-inflammatory cytokines such as tumor necrosis factor-alpha and interleukin-6 (20).

It is not well understood why habitual smoking increases periodontal inflammation. Nonetheless, it is possibly due to inducing endothelial dysfunction owing to smoking, which may lead to an inflammatory response in the vascular walls mediated by pro-inflammatory cytokines and adhesion molecules (27). Lower serum levels of immunoglobulin G have been observed in smokers compared to non-smokers, which predisposes them to develop a periodontal disease (20).

The evaluation of CAL as another predictor of periodontitis revealed a statistically significant difference between the four groups, indicating the significant effects of tobacco smoking, DM, and their interaction on increasing the CAL. This relationship may be attributed to the poor oral hygiene and adverse effects of DM on collagen formation and neutrophil function.

The comparative analysis of BOP among the four study groups demonstrated that the percentage of positive BOP was lower in smoker individuals. Although this difference was not statistically significant, it showed undeniable clinical differences between smoker and non-smoker individuals, which corroborates with the results of other studies (24,25). Likewise, this difference could be attributed to the effect of nicotine on blood vessels, decreasing the peripheral perfusion while increasing the gingival epithelium thickness in smokers (16,20).

Considering the vasoconstrictive effect of nicotine on gingival blood vessels, it is possible that suppressed BOP is neglected in smokers until tooth mobility becomes evident. Therefore, providing educational services on the effects of smoking on oral health is necessary for the general public.

Conclusions

According to the results of the present study, DM and cigarette smoking are known as risk factors for periodontal disease. Additionally, a combination of these two factors aggravates the periodontal status of patients. Thus, dentists can take an important step in the healthcare system by encouraging their patients to control their DM and quit smoking.

Conflict of Interest Disclosures

The authors declare that they have no conflict of interests.

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Ethical Statement

The proposal of the current research was approved by the Ethics Committee of Ardabil University of Medical Sciences in Ardabil, Iran. All the procedures were conducted under the Declaration of Helsinki guidelines (10). In this regard, the participants were informed about the study objective, and written informed consent was obtained from all the participants. The patients voluntarily participated in this study and were assured of data confidentiality, and could withdraw from the study at any time.

Authors' Contribution

IF, SP, SH, SM and ZM contributed to study design, data collection, data analysis, results interpretation, and manuscript preparation.

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