

Role of Smoking in Periodontal Disease among Diabetic Patients

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ABSTRACT

Objective: The objective of the study was to analyse the effect of smoking on periodontal disease in diabetic patients.

Methods: One hundred and fifty patients participated in the study. Fifty patients with Type 1 diabetes mellitus (DM) and periodontal disease were the first group; 50 patients with Type 2 DM and periodontal disease were the second group, and 50 non-diabetic patients with periodontal disease were the third group. After anamnesis was taken, the variable in the analysis was smoker/non-smoker. The status of the oral hygiene and periodontal condition was recorded using Plaque index, Supragingival calculus index, Subgingival calculus index, Gingival index, Periodontal Disease Index and Community Periodontal Index of Treatment Needs, by the periodontologist.

Results: Except Gingival index, mean values of all investigated indices were higher in smokers in all investigated groups.

Conclusion: Periodontal disease is more advanced in diabetic smokers compared to diabetic non-smokers. It can be concluded that smoking negatively affects the course of diabetic periodontal disease and increases the risk of attachment loss. Diabetic smokers are at high risk for poor periodontal prognosis, and they should be included in careful periodontal treatment.

Keywords: Diabetes mellitus, periodontal disease, smoking

Papel del Hábito de Fumar en la Enfermedad Periodontal entre los Pacientes Diabéticos

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RESUMEN

Objetivo: El objetivo del estudio fue analizar el efecto del hábito de fumar en la enfermedad periodontal en los pacientes diabéticos.

Métodos: Ciento cincuenta pacientes participaron en el estudio. Cincuenta pacientes con diabetes mellitus (DM) Tipo 1 y enfermedad periodontal, constituyeron el primer grupo; 50 pacientes con DM Tipo 2 y enfermedad periodontal, constituyeron el segundo grupo; y 50 pacientes no diabéticos con la enfermedad periodontal, constituyeron el tercer grupo. Después de hecha la anamnesis, la variable en el análisis fue fumador/no fumador. Se registró el estado de la higiene oral y la condición periodontal mediante el índice de placa, el índice de cálculo supragingival, el índice de cálculo de subgingival, el índice gingival, el índice de la enfermedad periodontal y el índice periodontal comunitario del tratamiento de las necesidades, por parte del periodontólogo.

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Resultados: Excepto el índice gingival, los valores promedios de todos los índices fueron más altos en los fumadores en todos los grupos investigados.

Conclusión: La enfermedad periodontal está más avanzada en fumadores diabéticos, en comparación con los no fumadores diabéticos. Puede concluirse que fumar afecta negativamente el curso de la enfermedad periodontal diabética y aumenta el riesgo de pérdida de inserción periodontal. Los fumadores diabéticos presentan un alto riesgo de prognosis periodontal pobre, y deben recibir tratamiento periodontal cuidadoso.

Palabras claves: Diabetes mellitus, enfermedad periodontal, hábito de fumar

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INTRODUCTION

Diabetes mellitus (DM) is a chronic disease frequently associated with many risk factors (1–3). It is a syndrome of abnormal carbohydrate, fat and protein metabolism due to the decreased insulin secretion or/and disturbed insulin activity. There are four general categories of DM: Type 1 which usually occurs in childhood or adolescence, in 5–10% of diabetic patients, results from an insulin deficiency which occurs due to genetically predisposed autoimmune destruction of β -cells and large number of environmental factors; Type 2 occurs in more than 90% of diabetic patients and results from insulin resistance and an insulin secretion defect; gestational is a condition of abnormal glucose tolerance during pregnancy, the most frequent metabolic disorder during pregnancy (1 to 14% pregnancies), occurs due to inadequate insulin secretion and production of placental hormones that block its action; and some specific forms of diabetes mellitus (rare forms of DM which cannot be classified in the above mentioned categories) occur due to genetically predisposed defect in β -cell function caused by genetic defect in insulin action, diseases of the exocrine pancreas (such as cystic fibrosis), the use of drugs or chemical agents (eg in the treatment of AIDS or after organ transplantation). Diabetes mellitus develops in people of all ages and races, and prevalence has increased dramatically over the past several decades (4). Owing to the increasing longevity of the population and the growing prevalence of DM, as well as the increased effectiveness of diagnostic and therapeutic protocols, dental practitioners treat patients with this disease frequently. Also, dental professionals can play an important role in diagnosing DM (5).

Smoking is a risk factor for periodontal disease and hence an important confounding factor (6). Diabetic smokers have deeper periodontal pockets and more attachment loss (AL) compared to never-smokers or diabetic patients who are non-smokers (7, 8). Moore *et al* (9) observed that severe periodontal disease is associated with smoking in patients with Type 1 DM. Taylor *et al* (10) reported that severe periodontal disease and smoking are associated with poor glycaemic control. It was observed that diabetic patients have disorders in phagocytosis, chemotaxis, and the synthesis of collagen, delayed wound healing and increased death of neutrophils. Also, it was observed that smoking has a negative effect on neu-

trophils and collagen (11). Simultaneous effects of these factors may partially explain the influence of poor metabolic control and smoking on AL (12). In addition, smoking favours the growth of microbes in shallow periodontal pockets (12). It is also possible that smoking may have a modifying effect on the association between metabolic control and periodontal disease and this statement is yet to be clarified in future investigations.

The aim of this study is to analyse the effect of smoking on periodontal disease in diabetic patients.

SUBJECTS AND METHODS

A cohort of 150 patients participated in the study. Fifty patients with Type 1 DM and periodontal disease were the first group, 50 patients with Type 2 DM and periodontal disease were the second group, and 50 non-diabetic patients with periodontal disease were the third (control) group. All participants were fully informed before completing their written consent document. The Ethics Committee of the Medical Faculty, University of Niš, approved the study protocol (evidential number 01-2800-7).

The diabetic patients were treated in the endocrine clinic of the Medical Faculty, University of Niš, as outpatients during their routine diabetic review appointments. Glycosylated haemoglobin (HbA_{1C}) was measured to assess glycaemic control. Patients were referred to the dental clinic of the Medical Faculty, University of Niš. The control group was recruited from outpatients attending the Department of Oral Medicine and Periodontology in the same clinic for their routine dental appointment. After anamnesis was taken, patients who received antibiotic and corticosteroid therapy, or had acute systemic illness, haemorrhagic disorders, autoimmune diseases or who were pregnant were excluded from the study. Data related to smoking were obtained from the self-administered questionnaire, where the average number of cigarettes smoked daily was inquired. The variable in the analysis was smoker/non-smoker.

Patients were examined by a periodontologist and the status of oral hygiene and the periodontal condition were recorded for each individual participating in the study. Measurements were obtained with the tip of the periodontal probe inserted into the pocket with constant probing force. PI (Plaque index), Izk (Supragingival calculus index), Ikon (Sub-

gingival calculus index), Gi (Gingival index – Loe and Silness), PDI (Periodontal Disease Index) and CPITN (Community Periodontal Index of Treatment Needs) were measured around all present teeth (13–15).

Entry and tabulation of results were performed using MS Excel programme, and calculations were made using SPSS, version 15.0. Pearson's χ^2 test was a nonparametric test for attribute comparison of numerical parameters frequency. Student's *t*-test for independent samples was performed to test statistically significant differences in the mean values of the two groups.

RESULTS

The mean age of patients in the first group was 25.54 ± 3.65 years, in the second 62.57 ± 8.57 years, and in the third was 45.68 ± 8.91 years. In the first group, there were 23 males (46%), in the second 26 males (52%), and in the third 25 males (50%). The mean HbA_{1C} measured in the first group was $9.87 \pm 0.32\%$, and in the second group $8.70 \pm 0.45\%$. The normal level for HbA_{1C} in healthy subjects ranged between 3.3% and 5.2%. In the first group, 15 (30%) patients, in the second group, 13 (26%) patients and in the third group, 19 (38%) patients were smokers (Table 1).

Table 1: The data obtained of all participants files (gender, age, DM duration, HbA_{1C}, smoking)

Groups	Group I	Group II	Group III
Gender			
male	23 (46%)	26 (52%)	25 (50%)
female	27 (54%)	24 (48%)	25 (50%)
Age (years)	25.54 ± 3.65	62.57 ± 8.57	45.68 ± 8.91
DM duration (years)	9.01 ± 1.22	14.68 ± 3.43	/
HbA _{1C} (%)	9.87 ± 0.32	8.70 ± 0.45	3.3%–5.2%
Smoker	15 (30%)	13 (26%)	19 (38%)
Non-smoker	35 (70%)	37 (74%)	31 (62%)

DM – diabetes mellitus

Index values in the first, second and third groups in relation to smoking are shown in Table 2. Except Gi, mean values of all investigated indices were higher in smokers in all

investigated groups. Gingival index had statistically higher values in non-smoker diabetic patients compared to smoker diabetic patients. In the third control group, Gi was minimally higher in non-smokers, with no statistical significance (Table 2).

DISCUSSION

Epidemiological studies have shown that DM is a risk factor for periodontal disease (16, 17). It is believed that people with Type 2 DM are three times more likely to have periodontal disease compared to healthy controls. If smoking as a risk factor is considered, the risk for developing periodontal disease and loss of supporting alveolar bone is 20 times higher. According to the literature, smoker-diabetic patients have deeper periodontal pockets and greater attachment loss compared to healthy non-smokers (18) or non-smoker-diabetic patients (19).

In the present study, it was observed that smokers had a poorer level of oral hygiene. Plaque index, Izk and Ikon were higher in smokers than in non-smokers in all investigated groups. Literature data reveal that smoking and poor metabolic control relate to neglect of self-care among diabetic patients (17, 20). Also, in the present study, PDI and CPITN were higher in smokers than in non-smokers. Similar findings were found in the literature which indicate that the periodontal health of smokers is poorer compared to non-smokers (21). The combined effect of “poor metabolic control and smoking” clearly increases the risk of AL (22). Haffajee and Socransky (23) noted that smokers generally had more AL and probing depths of 5–9 mm compared to non-smokers (22).

Gingival index had higher value in smoker–diabetic patients than in non-smoker-diabetic patients. In the control group, Gi was minimally higher in non-smokers, with no statistical significance. These findings are consistent with statements from the literature that highlight the influence of tobacco nicotine on the degree of keratinization of the gingival tissue and decrease of Gi index (24). Many epidemiological and clinical studies noticed the reduction of gingival inflammation and lower values of Gi index during periodontal disease, in smok-

Table 2: Index values (X \pm SD) in relation to smoking in all investigated groups

Groups	Smoking	N	PI	Izk	Ikon	Gi	PDI	CPITN
I	Smoker	15	$2.67 \pm 0.49^{*ab*}$	$2.33 \pm 0.49^{**ab**}$	$2.67 \pm 0.49^{a**b*}$	$1.83 \pm 0.38^{b***}$	$5.13 \pm 0.35^{**}$	$3.17 \pm 0.48^{b**}$
	Non-smoker	35	$2.17 \pm 0.66^{b***}$	$1.80 \pm 0.63^{b***}$	$2.54 \pm 0.56^{ab***}$	$1.93 \pm 0.26^{b***}$	4.77 ± 0.55	$2.92 \pm 0.54^{b***}$
II	Smoker	13	$2.14 \pm 0.71^{c**}$	1.62 ± 0.77	1.77 ± 1.01	$1.85 \pm 0.38^{c***}$	4.85 ± 0.69	3.05 ± 0.58
	Non-smoker	37	2.08 ± 0.76	$1.57 \pm 0.60^{c*}$	$1.65 \pm 0.75^{c**}$	$1.70 \pm 0.46^{c***}$	4.78 ± 0.71	$2.91 \pm 0.51^{c***}$
III	Smoker	19	$2.21 \pm 0.71^{**}$	$1.84 \pm 0.50^{***}$	$1.89 \pm 1.15^{***}$	1.23 ± 0.43	$4.63 \pm 0.50^{**}$	2.73 ± 0.44
	Non-smoker	31	1.65 ± 0.71	1.16 ± 0.73	1.06 ± 1.00	1.16 ± 0.50	3.84 ± 1.19	$2.12 \pm 0.80^{**}$

a – I vs II gr, b – I vs III gr, c – II vs III gr, * – $p < 0.05$, ** – $p < 0.01$, *** – $p < 0.001$

PI = Plaque Index, Izlc = Supragingival Calculus Index, Ikon = Subgingival Calculus Index, Gi = Gingival Index, PDI = Periodontal Disease Index, CPITN = Community Periodontal Index of Treatment needs

ers than in non-smokers (21, 25, 26). Suppression mechanisms through which smoking affects gingival bleeding are not yet fully clarified (21). It is assumed that nicotine has a short vasoconstrictive effect on the oral mucosa and a more complex effect on gingival vascularization and its cellular metabolism (25, 26).

Diabetes mellitus has not been found to induce alterations in periodontal microbes, but smoking has been found to favour the growth of periodontal microbes in shallow periodontal pockets (22, 27, 28). Thus, changes in the number and composition of periodontal pathogens may partly explain the results found in this study. Unfortunately, periodontal pathogens were not analysed in the study.

Diabetes mellitus involves defects in phagocytosis, chemotaxis and the activity of neutrophils, impaired collagen production and degradation, and impaired wound healing (29). Smoking has also been found to have parallel effects on neutrophils and collagen (30, 31). The simultaneous effect of these biologically deleterious factors may partly explain the combined effect of “poor metabolic control and smoking” on AL found in this study.

CONCLUSION

Periodontal disease is more advanced in smoker-diabetic patients compared to diabetic patients who do not smoke. It can be concluded that smoking negatively affects the course of diabetic periodontal disease and increases the risk of attachment loss.

Smoker-diabetic patients are at a high risk for poor periodontal prognosis, and they should be included in regular periodontal assessment and treatment.

REFERENCES

- Lorenzo C, Haffner SM. Performance characteristics of the new definition of diabetes. *Diabetes Care* 2010; **33**: 335–7.
- Craig ME, Hattersley A, Donaghue KC. Definition, epidemiology and classification of diabetes in children and adolescents. *Pediatr Diabetes* 2009; **10** (Suppl 12): 3–12.
- Dodds MW, Yeh C-K, Johnson DA. Salivary alterations in type 2 (non-insulin-dependent) diabetes mellitus and hypertension. *Comm Dent Oral Epidemiol* 2000; **28**: 373–81.
- Shaw JE, Sicree RA, Zimmet PZ. Global estimates of the prevalence of diabetes for 2010 and 2030. *Diabetes Res Clin Pract* 2010; **87**: 4–14.
- Lalla RV, D'Ambrosio JA. Dental management considerations for the patient with diabetes mellitus. *JADA* 2001; **132**: 1425–32.
- Obeid P, Bercy P. Effects of smoking on periodontal health: a review. *Advances in Therapy* 2000; **17**: 230–7.
- Klokkevold PR, Han TJ. How do smoking, diabetes, and periodontitis affect outcomes of implant treatment? *Int J Oral Maxillofac Implants* 2007; **22**: 173–202.
- Anner R, Grossmann Y, Anner Y, Levin L. Smoking, diabetes mellitus, periodontitis, and supportive periodontal treatment as factors associated with dental implant survival: A long-term retrospective evaluation of patients followed for up to 10 years. *Implant Dentistry* 2010; **19**: 57–64.
- Moore PA, Weyant RJ, Mongelluzzo MB, Myers DE, Rossie K, Guggenheimer J et al. Type I diabetes mellitus and oral health: assesment of periodontal disease. *J Periodontol* 1999; **70**: 409–17.
- Taylor GW, Burt BA, Becker MP, Genco RJ, Schlossman M, Knowler WC et al. Severe periodontitis and risk for poor glycemic control in patients with non insulin-dependent diabetes mellitus. *J Periodontol* 1996; **67**: 1085–93.
- Sørensen LT, Toft BG, Rygaard J, Ladelunde S, Paddon M, James T et al. Effect of smoking, smoking cessation, and nicotine patch on wound dimension, vitamin C, and systemic markers of collagen metabolism. *Surgery* 2010; **148**: 982–99.
- Syrjälä A-MH, Ylöstalo P, Niskanen MC, Knuutila MLE. Role of smoking and HbA1c level in periodontitis among insulin-dependent diabetic patients. *J Clin Periodontol* 2003; **30**: 871–5.
- Löe H. The Gingival index, the Plaque Index and the Retention Index systems. *J Periodontol* 1967; **38**: 610–6.
- Ramfjord SP. The Periodontal Disease Index (PDI). *J Periodontol* 1967; **38**: 602–10.
- Ainamo J, Barmes D, Beagrie G, Cutress T, Martin J, Sardo-Infirri J. Development of the World Health Organization (WHO) community periodontal index of treatment needs (CPITN). *Int Dent J* 1982; **32**: 281–91.
- Choi YH, McKeown RE, Mayer-Davis EJ, Liese AD, SongKB, Merchant AT. Association between periodontitis and impaired fasting glucose and diabetes. *Diabetes Care* 2011; **34**: 381–6.
- Toljamo M, Hentinen M. Adherence to self-care and glycaemic control among people with insulin-dependent diabetes mellitus. *J Advan Nurs* 2001; **34**: 780–6.
- Hujoel PP. Destructive periodontal disease and tobacco and cannabis smoking. *JAMA* 2008; **299**: 574–5.
- Corraini P, Baelum V, Pannuti CM, Pustiglioni AN, Romito GA, Pustiglioni FE. Periodontal attachment loss in an untreated isolated population of Brazil. *J Periodontol* 2008; **79**: 610–20.
- Habashneha RA, Khaderb Y, Hammada MM, Almuradia M. Knowledge and awareness about diabetes and periodontal health among Jordanians. *J Diabetes Complications* 2010; **24**: 409–14.
- Nair P, Sutherland G, Palmer RM, Wilson RF, Scott DA. Gingival bleeding on probing increases after quitting smoking. *J Clin Periodontol* 2003; **30**: 435–7.
- Lamster IB, Lalla E, Borgnakke WS, Taylor GW. The relationship between oral health and diabetes mellitus. *J Am Dent Assoc* 2008; **139**: 19–24.
- Haffajee AD, Socransky SS. Relationship of cigarette smoking to attachment level profiles. *J Clin Periodontol* 2001; **28**: 283–95.
- Ahmed GH, Ali Ebnoof SOM, Hussein MOM, Gbreel AYA. Oral epithelial atypical changes in apparently healthy oral mucosa exposed to smoking, alcohol, peppers and hot meals, using the AgNOR and Papanicolaou staining techniques. *Diagnostic Cytopathology* 2010; **38**: 489–95.
- Meekin TN, Wilson RF, Scott DA, Ide M, Palmer RM. Laser Doppler flowmeter measurement of relative gingival and forehead skin blood flow in light and heavy smokers during and after smoking. *J Clin Periodontol* 2000; **27**: 236–42.
- Amarasena N, Ekanayaka ANI, Hearh L, Miyazaki H. Association between smoking, betel chewing and gingival bleeding in rural Sri Lanka. *J Clin Periodontol* 2003; **30**: 403–8.
- Kubota M, Tanno-Nakanishi M, Yamada S, Okuda K, Ishihara K. Effect of smoking on subgingival microflora of patients with periodontitis in Japan. *BMC Oral Health* 2011; **11**: 1–6.
- Eggert FM, McLeod MH, Flowerdew G. Effects of smoking and treatment status on periodontal bacteria: evidence that smoking influences control of periodontal bacteria at the mucosal surface of the gingival crevice. *J Periodontol* 2001; **72**: 1210–20.
- Abiko Y, Selimovic D. The mechanism of protracted wound healing on oral mucosa in diabetes. Review. *Bosn J Basic Med Sci* 2010; **10**: 186–91.
- Lindblad SS, Mydel P, Jonsson IM, Senior RM, Tarkowski A, Bokarewa M. Smoking and nicotine exposure delay development of collagen-induced arthritis in mice. *Arthritis Res Ther* 2009; **11**: R88.
- Ryder MI. The influence of smoking on host responses in periodontal infections. *Periodontol* 2000, 2007; **43**: 267–77.