Acta Clin Croat 2021; 60:406-414
doi: 10.20471/acc.2021.60.03.10

Original Scientific Paper

CLINICAL AND MICROBIOLOGICAL ASSESSMENT OF NON-SURGICAL TREATMENT OF CHRONIC PERIODONTITIS IN CONTROLLED AND UNCONTROLLED TYPE 2 DIABETIC PATIENTS

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SUMMARY – Chronic periodontitis is a common complication in diabetes. The aim of this study was to evaluate some clinical and microbiological parameters in controlled and uncontrolled type 2 diabetes mellitus (type 2 DM) patients compared to non-diabetic (NDM) individuals, as well as to assess the effect of non-surgical periodontal therapy on these parameters. The study was performed in 61 type 2 DM patients with periodontitis (group 1A: 29 patients having achieved good metabolic control, HbA1c <7%; group 1B: 32 patients with poor metabolic control, HbA1c ≥7%), and 31 NDM individuals suffering from periodontitis. Periodontal indices (plaque index, PI; gingival index, GI; probing pocket depth, PPD; and clinical attachment level, CAL) were measured and subgingival plaque samples were analyzed using polymerase chain reaction prior to treatment initiation and 3 months post-treatment. The results recorded on the majority of measured parameters indicated that differences in treatment success achieved in the three treatment groups were not statistically significant (ΔPI p=0.646; ΔGI p=0.303; and ΔCAL p=0.233). Likewise, comparison of the effectiveness in microorganism reduction revealed no significant differences between DM groups and NDM patients. Therefore, study results supported the hypothesis that periodontal therapy outcome was unaffected by the level of glycemic control in patients with diabetes.

Key words: Diabetes mellitus; Periodontal disease/therapy; Glycosylated hemoglobin (HbA1c); Polymerase chain reaction

Introduction

Diabetes prevalence is increasing globally, making it one of the most significant diseases affecting modern society. The current worldwide prevalence of this condition is estimated at approximately 425 million individuals, and is expected to reach 629 million by 2045[1]. Diabetes mellitus (DM) is an umbrella term pertaining to metabolic disorders characterized by hyperglycemia arising from defects in insulin secretion, action, or both. In diabetic individuals, chronic hyperglycemia can result in long-term damage, dysfunction, and failure in various organ systems[2].

A greater periodontitis prevalence and severity has been observed in people with diabetes relative to the general population[3]. The degree of metabolic control of diabetes is likely to influence patient susceptibility to periodontitis, as hyperglycemia is the primary cause of the characteristic complications of diabetes[4]. More-

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Received December 11, 2018, accepted March 12, 2020

over, some authors posit that glycemic control quality is related to periodontitis severity, as more severe periodontal disease is typically diagnosed in individuals with poor glycemic control relative to those with well-controlled diabetes[5,6]. In addition to the degree of periodontal tissue destruction, the success of periodontal disease treatment is affected by the degree of diabetes metabolic control[7,8]. Kaur *et al.*[7] report similar periodontal therapy success in patients with good metabolic control and systemically healthy individuals, while noting that poorer metabolic control typically results in a less optimal periodontal response.

Although it is believed that poor metabolic control increases the risks associated with the onset and progression of periodontal disease, this correlation was not observed in some studies[9,10]. Furthermore, findings yielded by several trials failed to link glycemic control in type 2 DM patients with patient response to non-surgical periodontal therapy[11-13].

Periodontal health is maintained by optimal host response to the bacterial challenge imposed by dental plaque. It is widely accepted that elevated glucose levels in gingival crevicular fluid in diabetic individuals could provide an altered source of nutrition for subgingival microorganisms and thus might favor growth of certain bacterial species[14]. Furthermore, the immune response to periodontal pathogens may be altered or impeded in diabetics, potentially leading to overgrowth of certain species[15]. Current evidence on the effects of type 2 DM on dental plaque microbiota is, however, inconsistent. Several authors report significant differences in the bacterial composition of dental plaque between individuals with and without type 2 DM[16-19]. Moreover, Silva-Boghossian *et al.*[16] report that, following non-surgical periodontal therapy, DM2 patients with inadequate metabolic control presented different microbiological profile relative to that of systemically healthy individuals. In contrast, the authors of several microbiological studies report the prevalence and quantity of subgingival bacteria in diabetic patients similar to that in individuals suffering from periodontal disease[20-23].

Considering the inconsistencies in the available data, the aim of the present study was to evaluate some clinical and microbiological parameters in controlled and uncontrolled type 2 DM patients compared to non-diabetic individuals, and to assess the effect of non-surgical periodontal therapy on these parameters.

Materials and Methods

Subjects

The diabetics that took part in this prospective experimental clinical study were selected among 150 type 2 DM patients. Following regular control examination by an endocrinologist, all type 2 DM patients were referred to the specialist of periodontics and 67 of these individuals were invited to take part in the study, as they met the following inclusion criteria: age 30-70 years, type 2 DM treated with oral antidiabetic agents, and clinically diagnosed chronic periodontitis. Periodontitis was defined as minimum ≥2 sites with clinical attachment level (CAL) ≥3 mm and ≥2 sites with probing depth (PD) ≥4 mm at different teeth or 1 site with PD ≥5 mm[24]. Exclusion criteria were insulin medication, smoking, use of antibiotics during the preceding three months, periodontal treatment within the previous six months, pregnancy, and evidence for systemic diseases other than diabetes deemed a risk factor for periodontitis. The initial sample of 67 patients was further reduced to 61, as diabetes treatment was modified in four individuals, and another two failed to attend their 3-month recall appointment.

Thus, the sample utilized in the analyses comprised of 61 patients diagnosed with type 2 DM, on which complete data were available at the end of the study period. This diabetic group was divided into 2 subgroups based on the level of glycosylated hemoglobin (HbA1c), as follows: subgroup 1A including 29 subjects (10 males and 19 females, mean age 60.5 years) with good metabolic control (HbA1c <7%), and subgroup 1B including 32 subjects (16 males and 16 females, mean age 58.3 years) with poor metabolic control (HbA1c ≥7%). The borderline glycosylated hemoglobin values adopted in this study to assess metabolic control of diabetes were those recommended by the American Diabetes Association[25].

Control patients (group 2) that were not diagnosed with DM but suffered from chronic periodontitis were recruited from a total of 98 patients referred to a specialist of periodontics. After applying the aforementioned study inclusion and exclusion criteria, 34 patients with chronic periodontitis were recruited for the study. However, as one patient did not attend the 3-month recall appointment and two patients were subsequently prescribed antibiotics and were thus excluded from the study, the final control group consist-

ed of 31 individuals (13 males and 18 females, mean age 57.4 years).

Glycated hemoglobin was determined in all patients. Venous blood samples were obtained in the morning, prior to periodontal examination.

All participants signed the informed consent form. The study protocol was approved by the local Ethics Committee.

Periodontal examination

Periodontal condition was evaluated using the following indices: plaque index (PI), according to the Silness and Löe method[26]; gingival index (GI), in line with the Löe and Silnes methodology[27]; probing pocket depth (PPD), distance from the gingival margin to the bottom of the pocket (in mm); and CAL, distance from the cementoenamel junction to the bottom of the pocket (in mm). These indices were recorded at four sites *per* tooth for all teeth (mid-buccal, mesio-buccal, mid-lingual, and disto-lingual) using a Michigan "O" probe with William's markings.

Subgingival plaque sample collection

The sampling site was isolated with cotton rolls before supragingival plaque was removed in preparation for sampling. Subgingival plaque sample was obtained from the deepest pocket in each patient using a sterile periodontal curette and placed into Eppendorf tube containing 1.5 mL saline solution. Plaque samples were stored at -80 °C until processing.

Polymerase chain reaction analysis

For polymerase chain reaction (PCR) analysis, the samples were dispersed by vortex for 60 s before being boiled for 10 minutes. PCR was performed on 25 μL samples containing PCR buffer, 0.2 μM of each primer, 0.2 mM of each dNTP, 0.5 U *Taq* DNA polymerase, and 3-5 μL of template DNA containing supernatant.

Amplification was performed in a DNA Thermal Cycler (Hybaid, Champaign, IL, USA), commencing with a 5-minute cycle at 94 °C, followed by 35 1-minute routine cycles at 94 °C, annealing temperatures adequate for each primer pair (1 min), 90 s extension at 72 °C, and final 5-minute extension at 72 °C. The amplicons were visualized on 8% native polyacrylamide gels stained with ethidium bromide using a UV transilluminator.

Periodontopathogens were detected by means of multiplex PCR, using the following primers: *Porphyromonas gingivalis* (Pg1: 5' CAA TAC TCG TAT CGC CCG TTA TTC 3')[28], *Aggregatibacter actinomycetemcomitans* (Aa1: 5' CAC TTA AAG GTC CGC CTA CGT GC 3')[28], *Tannerella forsythia* (Tf V530: 5' GTA GAG CTT ACA CTA TAT CGC AAA CTC CTA 3')[29], and *Prevotella intermedia* (Pi: 5' GTT GCG TGC ACT CAA GTC CGC C 3')[29].

For negative control, DNA sample was replaced by distilled water.

Periodontal treatment

Non-surgical periodontal therapy comprising scaling and root planing (SRP) was performed by using an ultrasonic device and Gracey curettes in one or two sessions lasting for approximately 1 hour each. Oral hygiene instructions for home care procedures were administered to study patients.

Clinical evaluation and periodontal treatment were performed by the same therapist. All periodontal parameters, subgingival plaque sample collection, and HbA1c values were measured at baseline and three months after therapy completion.

Statistical analysis

Statistical analyses were conducted using the SPSS 16.0 for Windows software (SPSS, Chicago, IL, USA). The means and standard deviations (SD) were calculated for patient characteristics and clinical parameters. Differences in the mean values among the groups were compared *via* ANOVA analysis and post-hoc test for multiple comparisons. The significance of differences between the mean values before and after periodontal therapy was tested by t-test. The percentages of bacterial prevalence were compared between patients with DM and controls using χ^2-test. McNemar test was applied to test changes in bacterial presence between the two time points. Statistical significance was defined at the 5% level.

Results

Basic study group characteristics are presented in Table 1. At baseline, both groups of patients with DM showed significantly higher PI (1A: 1.74; 1B: 1.88) and GI (1A: 1.62; 1B: 1.59) values compared to con-

Table 1. Basic characteristics and clinical periodontal parameters in subjects at baseline

	Group 1A (n=29)	Group 1B (n=32)	Group 2 (n=31)	p
Gender: male/female	10/19	16/16	13/18	0.471
Age (years)	60.45±6.78	58.25±6.71	57.42±7.33	0.226
Number of teeth	17.28±4.61^{2*}	17.00±5.12^{2*,Ans}	20.32±5.22	**0.018**
DM duration (years)	6.7±5.52	8.55±5.7		0.204
HbA1c (%)	6.15±0.45^{2***}	8.35±1.42^{2***,A***}	5.51±0.32	**0.000**
PI	1.74±0.48^{2**}	1.88±0.37^{2***,Ans}	1.32±0.51	**0.000**
GI	1.62±0.64^{2***}	1.59±0.58^{2***,Ans}	0.94±0.72	**0.000**
PPD (mm)	2.09±0.51	2.11±0.47	2.38±0.60	0.061
CAL (mm)	2.81±0.98	2.62±1.36	2.32±1.39	0.327

Values are expressed as mean±SD and number of patients; Group 1A = patients with well-controlled DM; Group 1B = patients with poorly controlled DM; Group 2 = non-diabetic patients; n = number of patients; DM = diabetes mellitus; HbA1c = glycated hemoglobin; PI = plaque index; GI = gingival index; PPD = probing pocket depth; CAL = clinical attachment level; *p<0.05; **p<0.01; ***p<0.001; ns p>0.05 (post hoc tests – multiple comparisons); ^{2}*vs*. group 2; ^{A}*vs*. group 1A

Table 2. Periodontal parameters and HbA1c values at baseline versus those obtained three months after completing periodontal therapy

	Group	Baseline	3 months	p^{b}	Change Δ (baseline – 3 months)	p^{c}
PI	1A 1B 2	1.74±0.48 1.88±0.37 1.32±0.51	1.14±0.46 1.31±0.37 0.66±0.49	<0.001	0.60±0.31 0.57±0.42 0.66±0.39	0.646
GI	1A 1B 2	1.62±0.64 1.59±0.58 0.94±0.72	0.86±0.37 0.95±0.46 0.37±0.45	<0.001	0.76±0.43 0.64±0.45 0.57±0.53	0.303
PPD (mm)	1A 1B 2	2.09±0.51 2.11±0.47 2.38±0.60	1.98±0.53 2.03±0.35 2.05±0.52	<0.01 >0.05 <0.001	0.11±0.16^{2***} 0.08±0.27^{2***,Ans} 0.34±0.23	**0.000**
CAL (mm)	1A 1B 2	2.81±0.98 2.62±1.36 2.32±1.39	2.57±1.01 2.39±1.27 1.98±1.31	<0.001	0.24±0.24 0.23±0.27 0.34±0.3	0.233
HbA1c (%)	1A 1B 2	6.15±0.45 8.35±1.42 5.51±0.32	6.54±1.15 7.9±1.45 5.46±0.27	>0.05	-0.39±1.14^{2ns} 0.45±1.26^{A*,2ns} 0.05±0.15	**0.032**

* p<0.05; *** p<0.001; ns p>0.05 comparison between groups (post hoc tests – multiple comparisons);^{2}*vs*. group 2; ^{A}*vs*. group 1A; Δ = changes in values from baseline to 3 months; ^{b}p value refers to longitudinal changes within each group (t-test); ^{c}p value refers to comparison of changes in parameters between treatment groups (ANOVA for all examined parameters except for HbA1c for which Welch analysis was used); PI = plaque index; GI = gingival index; PPD = probing pocket depth; CAL = clinical attachment level

trol group (PI=1.32; GI=0.94), while PPD and CAL yielded no statistically significant differences between patients with DM and controls. In addition, comparison of patients with well-controlled DM (1A) and those with poorly controlled DM (1B) revealed no statistically significant differences in any of the measured clinical parameters prior to treatment.

At 3-month assessment following therapy completion (Table 2), the values of all periodontal parameters examined in the study were at a lower level than at

Table 3. Percentage of patients that were positive for four periodontal pathogens studied at baseline and three months after completing periodontal therapy

	Group	Baseline	3 months	Change (baseline – 3 months)	p^a	p^b	p^c
Aa	1A 1B 2	14 (48.3) 14 (43.8) 22 (71.0)	9 (31.0) 4 (12.5) 11 (35.5)	5 (17.3) 10 (31.3) 11 (35.5)	0.070	0.063 0.002** 0.001**	0.262
Pg	1A 1B 2	21 (72.4) 20 (62.5) 27 (87.1)	15 (51.7) 15 (46.9) 17 (54.8)	6 (20.7) 5 (15.6) 10 (32.3)	0.082	0.031* 0.063 0.002**	0.275
Pi	1A 1B 2	25 (86.2) 24 (75.0) 24 (77.4)	21 (72.4) 14 (43.8) 19 (61.3)	4 (13.8) 10 (31.3) 5 (16.1)	0.529	0.125 0.002** 0.063	0.182
Tf	1A 1B 2	28 (96.6) 26 (81.3) 25 (80.6)	23 (79.3) 18 (56.3) 19 (61.3)	5 (17.2) 8 (25) 6 (19.4)	0.136	0.063 0.008** 0.031*	0.738

Data are presented as n (%); *p<0.05; **p<0.01; Aa = *Aggregatibacter actinomycetemcomitans*; Pg = *Porphyromonas gingivalis*; Pi = *Prevotella intermedia*; Tf = *Tannerella forsythia*; [a]no statistically significant differences were noted among three treatment groups at baseline (p>0.05; χ^2-test); [b]p value refers to longitudinal changes within each group (McNemar test); [c]no statistically significant differences were noted among three treatment groups in bacterial reduction (p>0.05; χ^2-test)

baseline in all three patient groups. All differences were statistically significant, with the exception of PPD in patients with poor metabolic control, which declined from 2.11 mm to 2.03 mm. Diabetic control (HbA1c values) did not change significantly during the study. In terms of treatment success, both diabetic groups showed a significantly lower PD reduction (1A: ΔPPD=0.11 mm; 1B: ΔPD=0.08 mm) compared to that achieved in control group (ΔPPD=0.34 mm). However, the reduction noted in other clinical parameters was not statistically significant among the three groups (ANOVA: ΔPI p=0.646; ΔGI p=0.303; ΔCAL p=0.233).

Microbiological results (Table 3) indicated that most of the patients were PCR positive for Pg, Pi and Tf at baseline, with no differences in the frequency of detection between the subgroups of patients with DM and control group. Aa was less prevalent in both diabetic groups (1A 48.3%; 1B 43.8%) compared to control group (71%); however, these differences were not statistically significant. Furthermore, no differences were recorded in the prevalence of any of the tested species between the groups with well and poorly controlled diabetes.

Three months after treatment completion, the number of patients positive for periodontal pathogens in all three study groups declined, and the reduction was statistically significant for Pg in the group of patients with well-controlled diabetes, for Aa, Pi and Tf in the group of patients with poorly controlled diabetes, and for Aa, Pg and Tf in control group. Comparison of the effectiveness in microorganism reduction revealed no statistically significant differences among the three treatment groups (χ^2-test: Aa: p=0.262; Pg: p=0.275; Pi: p=0.182; Tf: p=0.738).

Discussion

Initially, significantly higher PI and GI values were recorded in both groups of DM patients compared to the control group. The explanations for this finding can be numerous. Xerostomia and increased salivary glucose in patients with diabetes may be responsible for additional plaque formation. Furthermore, diabetes has been shown to promote alterations in immune cell phenotype and elevation of serum proinflammatory cytokine levels, which can explain exaggerated inflammatory host response[3]. This finding could be due to the less optimal oral self-care in type 2 DM patients compared to those in the control group. Specifically, diabetic patients were referred to our clinic by their endocrinologist for the purpose of the present investigation, whereas those in the control group sought treatment for an existing periodontal issue and were thus likely

aware of the need to maintain optimal oral hygiene. Commmiso *et al.*[30] found that the degree of oral hygiene was related to dental health awareness in patients with type 2 DM. They compared type 2 DM patients that periodically attended appointments with a dentist or dental hygienist and those that did not, and found that the former group had lower PI, less bleeding during tooth brushing, and less extensive gingivitis. Even though our diabetic patients and those in the control group were non-equivalent with respect to PI and GI, these differences were not deemed relevant for the assessment of non-surgical periodontal therapy outcomes, as the groups were not statistically significantly different according to CAL and PPD as the key determinants of the degree of destruction in the supporting dental apparatus. The authors of several earlier studies have also reported less favorable clinical periodontal parameters such as PI and bleeding on probing in patients diagnosed with type 2 DM compared with those in the systemically healthy group[16,23]. Additionally, in our study, both groups of DM patients had a significantly lower number of teeth compared to the control group. A high incidence of tooth loss in diabetic patients was confirmed by other authors[10,18]. Andjelski-Radicevic *et al.*[31] found the number of teeth present, in addition to patient age and level of oral hygiene, to be significantly affected by the duration of diabetes.

To determine the link between glycemic status and periodontal treatment outcome, patients with diabetes were stratified into groups with good (subgroup 1A) and poor (subgroup 1B) metabolic control based on the HbA1c levels. Comparison of treatment success in DM patients with good metabolic control, DM patients with poor metabolic control, and non-diabetic individuals revealed an equally good improvement in PI, GI and CAL level in all three groups. These results are in line with the findings reported by Dag *et al.*[11], who noted similar improvements in clinical periodontal conditions in patients with poorly controlled DM (median HbA1c, 9.96%), well-controlled DM (median HbA1c, 6.26%) and non-diabetic patients three months after non-surgical periodontal therapy. The only difference in our study pertained to PD, as both diabetic groups showed a significantly lower PD reduction compared to control group. This outcome could be attributed to low mean PD in patients with DM, as it is known that more severe baseline PPD is associated with greater improvements after non-surgical periodontal therapy[13].

Somewhat less successful treatment in DM groups relative to controls could potentially be attributed to metabolic control alterations in diabetic patients. It is considered that, as hyperglycemia affects immune functions and the microvasculature, it compromises the person's response to periodontal treatment[32]. For example, Kaur *et al.*[7] report a significantly higher percentage of sites with bleeding on probing, as well as a higher GI score at 3- and 6-month follow-up in patients with poor glycemic control despite having similar plaque levels as those with good glycemic control and non-diabetic individuals.

The results obtained in the present study, however, indicate that patients with poorly controlled diabetes achieved similar clinical periodontal status improvement after treatment as did those with well-controlled diabetes mellitus. Our results are in accordance with those reported by Santos *et al.*[33], who showed similar clinical responses three months after scaling and root planing in subjects with better and poorly controlled diabetes. However, in the aforementioned study, subjects with better controlled disease had a significantly lower CAL at 6-month follow-up, while our investigation did not include periodontal status assessment six months upon therapy completion. Longitudinal follow-up is needed to evaluate any differences in periodontal therapy outcomes between patients with good and poor glycemic control.

Analysis of microbiological data at baseline showed that the majority of diabetics and non-diabetics harbored *P. gingivalis, T. forsythia* and *P. intermedia*, with a similar prevalence of these periodontopathogens across the groups. *A. actinomycetemcomitans* was less frequently detected in both diabetic groups of patients compared to control group, although the difference was not statistically significant. The reason behind this finding could be a slightly lower PD in diabetics compared to the control group at baseline, since some studies showed the Aa percentage to increase with periodontal pocket depth[34]. Similar findings have been reported by other authors using the same[22,23] or different methodological approaches[20,21]. For example, using real-time quantitative PCR, Field *et al.*[21] demonstrated that *A. actinomycetemcomitans*, *Fusobacterium nucleatum* and *P. gingivalis* were present in similar amounts in individuals with periodontitis, irrespective of their DM status.

Similarly, based on the findings yielded by the checkerboard DNA-DNA hybridization method, Hintao *et al.*[20] report no significant differences in the prevalence and level of 17 subgingival species in the participants with and without DM.

The results reported here suggest that the prevalence of subgingival bacteria is unrelated to diabetic status of patients suffering from periodontal disease, thus challenging the assertions put forth by other authors[16-19]. This incongruence in findings could be attributed to the limited scope of our investigation, as we evaluated four bacterial species only (*P. gingivalis, T. forsythia*, *P. intermedia* and *A. actinomycetemcomitans*) in subgingival plaque. Casarin *et al.*[17] detected even lower percentages of two components of the 'red complex', *P. gingivalis* and *T. forsythia*, in DM patients with periodontal disease when compared to non-diabetic individuals. On the other hand, the higher percentages of *Capnocytophaga* spp., *Fusobacterium nucleatum*, *Veillonella parvula, Eikenella corrodens* and *Streptococcus mitis* were noted in diabetic subjects. It is noteworthy that comparisons across different studies are difficult due to variations in the clinical protocols and participant selection criteria utilized. Campus *et al.*[18] report that, in their study, subjects with diabetes had a higher degree of periodontal tissue destruction relative to those in control group, which was probably the cause of the higher prevalence of *P. gingivalis* in diabetic patients. On the other hand, in the study conducted by Ebersole *et al.*[19], focusing on populations with a high incidence of type 2 DM, greater prevalence of *P. gingivalis, Campylobacter* spp. and *A. actinomycetemcomitans* was noted in patients with diabetes compared to control group. However, these findings cannot be applied to the general population due to the participant selection criteria. Moreover, our findings revealed no significant differences in the prevalence of any of the tested species between diabetic patients irrespective of the degree of glycemic control. This finding is in line with the results of other studies indicating that glycemic control does not significantly influence the composition of the subgingival biofilm in diabetic individuals[14,35].

Periodontal diseases are caused by bacteria residing in subgingival biofilms. Empirical evidence indicates that limiting the quantity of periodontal pathogens is crucial for good clinical response to periodontal therapy[36]. Studies assessing the relationship between periodontal disease therapy and DM from microbiological point of view are limited. Consequently, there is no consensus on whether the subgingival microbiota in patients with DM is significantly affected by scaling and root planing[16,37]. Silva-Boghossian *et al.*[16] showed that, after non-surgical periodontal therapy, individuals with DM2 and inadequate metabolic control presented with a different microbiological profile compared to that of a control group. Even though reduction in a greater number of species was noted after therapy in systemically healthy individuals, the prevalence and extent of pathogenic species (*P. gingivalis, T. forsythia* and *P. intermedia*) significantly decreased in patients with type 2 DM, allowing good clinical response to be achieved. In the present study, upon therapy completion, a decrease in the number of patients affected by tested microorganisms was observed in all three treatment groups, with no significant differences in the effectiveness in microorganism reduction. These findings are in accordance with those reported by Da Cruz *et al.*[37]. When microbiological changes were evaluated three months after full-mouth scaling and root planing in type 2 DM patients and compared to those observed in non-diabetic patients, da Cruz *et al.*[37] report reduction in *A. actinomycetemcomitans, P. gingivalis* and *T. forsythia* at the sites with PPD ≥5 mm in both groups. However, the change was statistically significant only for *T. forsythia* in the non-diabetic group. On interpreting these findings, it should be noted that these authors used the PCR assay, as was done in the present study. PCR is a relatively simple, sensitive and rapid test for detection of bacterial DNA sequences; however, it does not permit quantitative determination of the pathogens identified[38]. Therefore, even though the reduction may be even more significant, the changes cannot be quantitatively evaluated. Further studies employing larger patient samples, and using quantitative PCR to reveal any potential differences in the microbiota of these individuals are thus required.

The present study indicated that DM patients might respond to non-surgical periodontal therapy similarly well to non-diabetic patients. There were no significant differences in the treatment outcomes among the groups according to most of the clinical parameters measured. Similar reductions in the prevalence of periodontal pathogenic bacteria were found in diabetic patients and non-diabetic individuals.

In conclusion, based on the results yielded by the present study, the periodontal therapy outcome in pa-

tients with diabetes does not seem to be significantly affected by the level of glycemic control.

Acknowledgment

This study was financially supported by Research Grant N° 175075 from the Ministry of Education and Science of Serbia.

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Sažetak

KLINIČKA I MIKROBIOLOŠKA PROCJENA NE-KIRURŠKOG LIJEČENJA KRONIČNOG PARODONTITISA KOD BOLESNIKA S KONTROLIRANOM I NEKONTROLIRANOM ŠEĆERNOM BOLEŠĆU TIP 2

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Kronična parodontopatija je jedna od vrlo čestih komplikacija dijabetesa melitusa (DM). Cilj istraživanja bio je usporediti kliničke i mikrobiološke parametre osoba s dobrom i lošom metaboličkom kontrolom DM tip 2 i onih koje ne boluju od DM (NDM) te procijeniti učinke bazične terapije parodontopatije na ove parametre. Ispitivanjem su obuhvaćene osobe s kroničnom parodontopatijom: 61 bolesnik s DM tip 2 (skupina 1A: 29 ispitanika s dobrom metaboličkom kontrolom, HbA1c <7%; skupina 1B: 32 ispitanika s lošom metaboličkom kontrolom, HbA1c ≥7%) i 31 NDM osoba. Mjerenja indeksa (plak indeks, PI; gingivalni indeks, GI; DPDž, dubina parodontnog džepa; i gubitak pričvrstka, GP) i mikrobiološka analiza subgingivalnih uzoraka plaka pomoću lančane reakcije polimeraze provedena su na početku istraživanja i 3 mjeseca nakon tretmana. Rezultati vezani za većinu kliničkih parametara pokazuju da nema statistički značajnih razlika u uspjehu terapije između tri ispitivane skupine (ΔPI p=0,646; ΔGI p=0,303; ΔCAL p=0,233). Također, nije utvrđena značajna razlika u uspješnosti eradikacije ispitivanih bakterijskih vrsta između skupina DM i NDM osoba. Navedeni rezultati idu u prilog tezi da razina metaboličke kontrole ne utječe značajno na uspjeh terapije parodontopatije kod osoba s DM.

Ključne riječi: *Dijabetes melitus; Parodontne bolesti/terapija; Glikozilirani hemoglobin (HbA1c); Lančana reakcija polimeraze*