Effect of Scaling and Root Surface Debridement on Lipid Profile in Type-2 Diabetes Patients with Chronic Periodontitis

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Abstract

Objective: Periodontitis is the most common inflammatory conditions affecting the periodontal tissues and considered as one of the complications of type 2 diabetes mellitus. The study aimed to evaluate the effects of scaling and root surface debridement on clinical periodontal parameters and serum lipid profile levels in chronic periodontitis and type 2 diabetes patients.

Methods: The study enrolled on 45 patients, 15 systemically healthy patients with chronic periodontitis, 15 well controlled (glycated hemoglobin <8%) and 15 poorly controlled (HbA1c ≥8%) type 2 diabetic patients with chronic periodontitis. Intra-oral clinical parameters (plaque index, gingival index, probing pocket depth, and clinical attachment loss) and blood samples were collected to test high density lipoprotein (HDL), low density lipoprotein (LDL), total cholesterol (TC) and triglyceride (TG) for all patients at baseline and after 30 days of periodontal therapy. One-way ANOVA used to compare between groups.

Results: The study groups showed significant reduction in clinical periodontal parameters after 30 days of periodontal therapy ($P < 0.000$), while no significant differences were seen in serum lipid profile ($P > 0.05$). Non-significant differences were also seen between the 3 groups in relation to clinical periodontal parameters and serum lipid profiles after periodontal therapy $P > 0.05$.

Conclusions: Scaling and root surface debridement reduce clinical parameters of periodontitis in all study groups, however, no significant reduction of lipid profile were exhibited.

Keywords: *Periodontitis, Type 2 diabetes, Periodontal treatment, Lipid profile.*

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Introduction

Periodontitis is a chronic infectious disease affects the tissue that surround and support the teeth namely gingiva, cementum, periodontal ligament and alveolar bone, it arise from host-bacterial interaction[1] and has been recognized as one of the 6th complication of diabetes mellitus (DM)[2].

Diabetes mellitus is a common metabolic disorder characterized by absolute or relative deficiencies in insulin secretion and/or insulin action associated with chronic hyperglycemia and disturbances of carbohydrate, lipid and protein[2]. DM occurs as a result of a complex interaction of genetic, environmental factors and lifestyle choices[3], and the two metabolic defects which contribute to the development of type 2 diabetes are relative insulin insufficiency and insulin resistance[2].

A bi-directional relationship has been found between diabetes and periodontitis, within this relationship it is well accepted that people with diabetes are more likely to have or develop periodontitis[4] by three folds compared to diabetes-free controls[5].

Jaramillo et al (2013)[6] found an association between high blood lipoproteins and alterations in periodontal conditions. They reported that patients with mild or moderate hyperlipidemia have higher levels of periodontal parameters compared with normolipidemic individual[6], however, these results are conflicting with several studies showing no association[7,8].

The ability of periodontal diseases in altering the lipid metabolism leading to hyperlipidemia in DM is conflicting. Studies have shown that type II DM is positively correlated with hyperlipidemia and periodontitis does not show a definite association[9] , however, other studies have shown a positive association between periodontitis and hyperlipidemia[10,11].

It has been shown that scaling and root planing in subjects with DM reduce clinical parameters of periodontal disease such as probing pocket depth, bleeding on probing and clinical attachment loss[12], another study reported that serum lipid profile has been also improved by the removal of the infectious and inflammatory burden of periodontal disease through periodontal therapy[13].

The present study aimed to evaluate the effect of scaling and root surface debridement on clinical periodontal parameters: plaque index (PI), gingival index (GI), probing pocket depth (PPD), and clinical attachment loss (CAL) as well as serum lipid profile: (TC), (LDL), (HDL) and (TG) in type 2 diabetes patients with chronic periodontitis (controlled and uncontrolled) and in systemically healthy patients with chronic periodontitis after 30 days of periodontal therapy.

Patients and methods

Setting and time of study

The present study was carried out in diabetes center (Shaheed Layla Qassim), Medical center (Shaheed Nafee Akree), BIO clinical laboratory, and periodontic department /College of Dentistry/ Hawler Medical University in Erbil city. The study conducted during the period of 5th January to 27Th April 2018.

Subjects

This prospective study was conducted on 45 subjects of both sexes. The subjects were divided into three groups; first group consist of 15 systemically healthy patients with chronic periodontitis (chronic periodontitis without diabetes), second group consist of 15 patients with well-controlled type 2 diabetes with chronic periodontitis (fast blood sugar test FBS >125, and glycated hemoglobin 1 assay HbA1c <8%)[18], and the third group consist of 15 patients with poorly- controlled type 2 diabetes with chronic periodontitis (FBS >125, HbA1c ≥8%)[14]. The criteria for chronic periodontitis was define by PPD ≥ 4 mm or CAL ≥ 3mm[15], in two or more different sites of at least two teeth in each quadrant, and all participants should have ≥ 20 teeth in the oral cavity. Patients with a medical history of any systemic disease other than type 2 diabetes mellitus, type 2 diabetes patients with concomitant diseases or condition affecting the lipid levels (hypothyroidism, thiazides or lipostatic drugs), smoking, alcoholic, pregnant, menopause and lactating women, medication other than hypoglycemic agents and patients underwent periodontal treatment in the last 6 months were excluded from the study. The study was reviewed and approved by institutional ethical committee of the college of dentistry/ Hawler medical university and informed consent was signed by all participants before conduction of the study.

Clinical periodontal examination and treatment

All participants underwent a full-mouth periodontal examination by double blinded clinicians at the base line before treatment and after 1 month of periodontal therapy including: measurement the thickness of plaque according to PI[16] for four surfaces of all the examined teeth and given a score from 0-3, measuring the extent and severity of gingival inflammation according to GI[17] inspection by naked eyes and by gentle probing through using periodontal probe for four gingival

surfaces of all examined teeth and given a score from 0-3, probing pocket depth was assessed by insert calibrated periodontal probe (UNC-15 probe) from gingival margin to the base of pocket at four surfaces of each tooth, no pressure was used and the probe was allowed to fall by its own weight[18], and clinical attachment level was assessed by measured the distance from cementoenamel junction (CEJ) to the base of the pocket[18] by a UNC-15 probe. When CEJ was obliterated by the gingival margin, CAL was measured indirectly by subtracting the distance from the gingival margin to the CEJ from PPD, and in case when there was a gingival recession, loss of attachment was measured by adding the distance from gingival margin to the CEJ to PPD[19]. Periodontal parameters measured under supervision of second examiner, then all participants underwent non-surgical periodontal treatment that consist of motivation, oral hygiene instruction, scaling (supra and sub gingival), root surface debridement by ultrasonic scalers and gracy curettes and followed up after 30 days.

Blood sample collection

Fasting venous blood samples (7 ml) were collected from all participants at baseline before treatment and after 30 days of periodontal treatment. For diabetic patients the samples stored in two tubes, a plain tube containing no anticoagulant for serum lipid profile, and ethylene diamine tetraacetic acid containing tube (EDTA containing tube) for estimation of FBS and HbA1c. The samples were transferred immediately to the laboratory to evaluate the following serum parameters: FBS test, HbA1c, and lipid profile (HDL, LDL, TC and TG).

Statistical analysis

Paired T-test was used to compare clinical and laboratory variables before and after treatment and one-way ANOVA was used to compare tested variables in three studied groups. P value <0.05 was regarded as statistically significant. All analyses were performed by SPSS software package (version 22; SPSS Inc., Chicago, IL, USA).

Results

A total of 45 patients (23 females and 22 males) with the mean age of 47.98± 6.84 years were enrolled in the study. For female and male, the mean ages were 47.82±7.02 and 48.09±6.82 years respectively. Also the mean levels of HbA1c in well controlled and poorly controlled diabetes before periodontal therapy were 6.94±0.63% and 9.95±1.37%, respectively.

Comparison of clinical periodontal parameters

The results showed that the mean values of PI, GI, PPD and CAL in chronic periodontitis group at the baseline before treatment were 1.65±0.49, 1.74±0.43, 4.96±0.44mm and 5.33±1.16 mm respectively, then their mean values were significantly reduced into 0.79±0.18, 1.07±0.15, 3.91±0.37mm and 4.39±1.02 mm respectively, after 30 days of periodontal therapy (P<0.0001 using t-test), while the mean values of PI, GI, PPD and CAL in uncontrolled diabetic group at base line were 1.86 ± 0.41, 1.76 ± 0.45, 4.96 ± 0.54mm and 5.25 ± 0.84mm respectively, and significantly reduced to 0.95 ± 0.29, 1.07 ± 0.12, 3.80 ± 0.36mm, 4.15 ± 0.69mm respectively, after 30 days of periodontal therapy (P< 0.0001 using t-test). Furthermore, in the controlled diabetic group the mean values of PI, GI, PPD and CAL at the baseline before treatment were 1.56± 0.55, 1.53 ± 0.41, 4.74 ± 0.43mm, 5.58 ± 0.93mm respectively, and significantly reduced into 0.77 ± 0.28, 0.96 ± 0.18, 3.72 ± 0.53, 4.47 ± 0.83 respectively, after 30 days of periodontal therapy (P< 0.0001 using t-test) as shown in Table 1. Finally, there were no statistically significant differences of clinical parameters between the three studied main groups (ANOVA test, P>0.05) as shown in Table. 2.

Comparison of serum lipid profile (HDL, LDL, TC and TG).

Intra group comparison showed that the mean values for HDL, LDL, TC and TG for the chronic periodontitis group at baseline before treatment were 33.86±6.98, 100.78±40.87, 164.93±42.30 and 173.53±90.44 mg/dl and after 30 days of therapy, their mean values were 35.61±6.81, 88.18±26.90, 147.72±30.30 and 148.16±68.54 mg/dl respectively, with no statistically significant differences when compared to the baseline before treatment (t-test, P> 0.05). For uncontrolled diabetic group before treatment the mean values of HDL, LDL, TC and TG were 43.93±11.49, 94.40±39.95, 171.86±40.21 and 216.53±113.24 mg/dl and after 30 days of periodontal therapy, their mean values were 43.20±11.06, 97.16±42.34, 167.46±56.16 and 184.0±130.76 mg/dl respectively, with no significant differences with base line before treatment (P> 0.05). For controlled diabetic group the mean values of HDL, LDL, TC and TG at base line before treatment were 44.33±7.16, 102.79±42.50, 165.06±51.40 and 169.93±88.90 mg/dl, respectively, and after treatment their mean values were 41.20±10.43, 91.96±40.16, 161.73±48.99 and 169.80±85.81 mg/dl, respectively, also with no significant differences with the base line before treatment as seen in Table 3.

Table 1: Intra group comparison of PI, GI, PPD, and CAL before and after periodontal therapy (N =15 for each group).

Index	Groups	Times	Mean ± SD	Paired-T-test	P-Value
PI	Chronic periodontitis	Before	1.65 ± 0.49	8.59	0.000**
		After	0.79 ± 0.18		
	Uncontrolled diabetic	Before	1.86 ± 0.41	10.85	0.000**
		After	0.95 ± 0.29		
	Controlled diabetic	Before	1.56 ± 0.55	8.19	0.000**
		After	0.77 ± 0.28		
GI	Chronic periodontitis	Before	1.74 ± 0.43	7.43	0.000**
		After	1.07 ± 0.15		
	Uncontrolled diabetic	Before	1.76 ± 0.45	7.21	0.000**
		After	1.07 ± 0.12		
	Controlled diabetic	Before	1.53 ± 0.41	7.43	0.000**
		After	0.96 ± 0.18		
PPD (mm)	Chronic periodontitis	Before	4.96 ± 0.44	12.55	0.000**
		After	3.91 ± 0.37		
	Uncontrolled diabetic	Before	4.96 ± 0.54	11.46	0.000**
		After	3.80 ± 0.36		
	Controlled diabetic	Before	4.74 ± 0.43	15.60	0.000**
		After	3.72 ± 0.53		
CAL (mm)	Chronic periodontitis	Before	5.33 ± 1.16	13.93	0.000**
		After	4.39 ± 1.02		
	Uncontrolled diabetic	Before	5.25 ± 0.84	8.35	0.000**
		After	4.15 ± 0.69		
	Controlled diabetic	Before	5.58 ± 0.93	15.40	0.000**
		After	4.47 ± 0.83		

** highly significant

Inter group comparison before and after periodontal treatment showed that there were no statistically significant differences in lipid profile of all studied three groups except the level of HDL at baseline before treatment (ANOVA test, P = 0.003) as shown in Figure 1 and 2.

Table 2: Inter group comparison of clinical parameters before and after periodontal treatment (N =15 for each group).

Index	Times	Groups	Mean ±Std. Deviation	F-test	P-Value (Sig.)
PI	Before therapy	Periodontitis	1.65 ±0.49	1.498	0.23 (NS)
		Uncontrolled	1.86±0.41		
		Controlled	1.56±0.55		
	After therapy	Periodontitis	0.79±0.18	2.165	0.12 (NS)
		Uncontrolled	0.95±0.29		
		Controlled	0.77 ±0.28		
GI	Before therapy	Periodontitis	1.74±0.43	1.245	0.29 (NS)
		Uncontrolled	1.76±0.45		
		Controlled	1.53±0.41		
	After therapy	Periodontitis	1.07±0.15	2.668	0.08 (NS)
		Uncontrolled	1.07±0.12		
		Controlled	0.96±0.18		
PPD (mm)	Before therapy	Periodontitis	4.96±0.44	1.069	0.35 (NS)
		Uncontrolled	4.96±0.54		
		Controlled	4.74±0.43		
	After therapy	Periodontitis	3.91±0.37	0.760	0.47 (NS)
		Uncontrolled	3.80±0.36		
		Controlled	3.72±0.53		
CAL (mm)	Before therapy	Periodontitis	5.33±1.16	0.463	0.63 (NS)
		Uncontrolled	5.25±0.84		
		Controlled	5.58±0.93		
	After therapy	Periodontitis	4.39±1.02	0.563	0.57 (NS)
		Uncontrolled	4.15±0.69		
		Controlled	4.47±0.83		

NS= non-significant

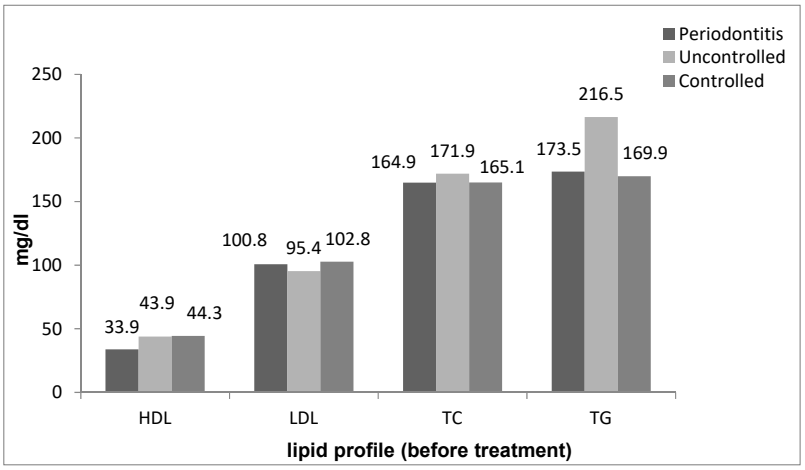

Figure 1. The mean levels of HDL, LDL, TC and TG in three main groups before periodontal therapy.

Table 3: Intra group comparison of HDL, LDL, TC and TR before and after periodontal treatment (N=15 for each group).

Variables (Mg/dl)	Groups	Times	Mean ± Std, Deviation	Paired T-test	P-Value (Sig.)
HDL	Chronic periodontitis	Before	33.86 ± 6.98	-1.85	0.08 (NS)
		After	35.61 ± 6.81		
	Uncontrolled diabetic	Before	43.93 ± 11.49	0.33	0.74 (NS)
		After	43.2 ± 11.06		
	Controlled diabetic	Before	44.33 ± 7.16	1.73	0.10 (NS)
		After	41.2 ± 10.43		
LDL	Chronic periodontitis	Before	100.78 ± 40.87	1.56	0.14 (NS)
		After	88.18 ± 26.90		
	Uncontrolled diabetic	Before	95.4 ± 39.95	-0.19	0.84 (NS)
		After	97.16 ± 42.34		
	Controlled diabetic	Before	102.79 ± 42.50	1.01	0.32 (NS)
		After	91.96 ± 40.16		
TC	Chronic periodontitis	Before	164.93 ± 42.30	1.72	0.10 (NS)
		After	147.72 ± 30.30		
	Uncontrolled diabetic	Before	171.86 ± 40.21	0.45	0.65 (NS)
		After	167.46 ± 56.16		
	Controlled diabetic	Before	165.06 ± 51.40	0.24	0.81 (NS)
		After	161.73 ± 48.99		
TG	Chronic periodontitis	Before	173.53 ± 90.44	1.46	0.16 (NS)
		After	148.16 ± 68.54		
	Uncontrolled diabetic	Before	216.53 ± 113.24	1.43	0.17 (NS)
		After	184 ± 130.76		
	Controlled diabetic	Before	169.93 ± 88.90	0.009	0.99 (NS)
		After	169.8 ± 85.81		

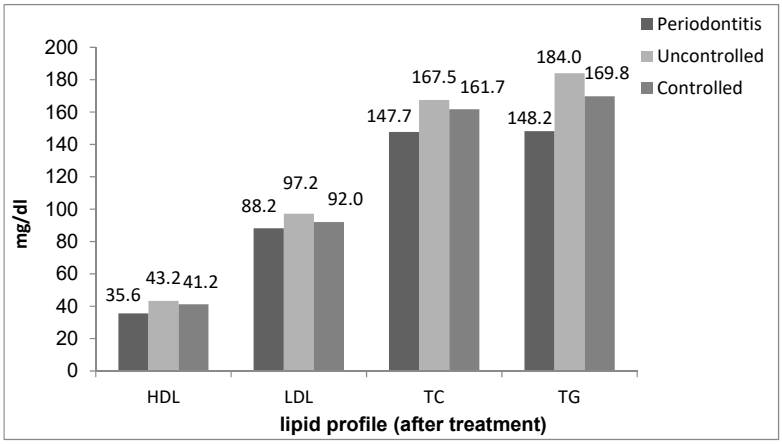

Figure 2: The mean levels of HDL, LDL, TC and TG in three main groups after periodontal therapy.

Discussion

The relationship between diabetes and periodontal diseases has been explored in several studies and it was widely accepted that the prevalent and severity of periodontal disease increased in subjects with diabetes compared to non diabetes[20-22]. A study suggested that some diabetes-induced metabolic alterations serve to diminish host resistance to periodontal breakdown and that these diabetes- induced alterations of host response may not be preventable or reversible with glycemic control[22]. The present study showed that scaling and root planing resulted in reduction of inflammatory reaction and healing of periodontal tissue throughout significant reduction of clinical periodontal parameters in chronic periodontitis, controlled, and uncontrolled diabetes patient groups after 1month of periodontal therapy. The reduction in PI, GI, PPD and CAL values may be attributed to that periodontal therapy decreases the intraoral bacterial bio-burden and reduces periodontitis-induced bacteremia/endotoxemia[23] .

These findings are similar to the results obtained from other studies[24-27]. A study reported significant improvement in the clinical periodontal parameters (PI, GI, PPD and CAL) after one and three months of periodontal therapy for both type 2 diabetes with chronic periodontitis (controlled and poorly controlled) and systemically healthy patients with chronic periodontitis[24], another study also reported significant reduction in clinical parameters (PI, GI, PPD and gingival crevicular fluid volume) after 3 months of non-surgical periodontal therapy by scaling and root planing (SRP) and sub gingival debridement for chronic periodontitis with or without type 2 diabetes[25]. Also in the same line with our results a study reported high significant reduction in the mean values of clinical parameters (GI, PPD, bleeding on probing, gingival recession and CAL) after 1 and 3 months of non-surgical periodontal treatment for type 2 diabetes[26], another study evaluate the effect of periodontal therapy on clinical parameters (PI, PPD, CAL and gingival bleeding index) in patients with type 2 diabetes mellitus and systemically healthy individuals after 3 months of non-surgical periodontal therapy and reported that periodontal therapy was effective in improving most clinical parameters[27].

For chronic periodontitis patients, a study reported reduction in the clinical parameters (PI, GI, PPD and gain of clinical attachment level) after two month of non-surgical periodontal therapy[28]. Another study concluded that the improvement in clinical periodontal parameters after 2 months of therapy for systemically healthy patients with chronic periodontitis may be attributed to that scaling and root planing accompanied by reduction of inflammatory infiltrate and lying down of new collagen[29].

For serum lipid profile, the present study showed non-significant differences in the mean concentration of TC, HDL, LDL and TG after 30 days of treatment, and this may be due to that hyperlipidemia are highly influenced by environmental variables like eating, life style, physical activity habits and socioeconomic conditions of the patients which may be interfere with our study since they are variables that are not included in our study and difficult to be control. Another limitation could be the limited numbers of the samples or the time of follow up was not sufficient to determine the comparison between base line and after periodontal therapy treatment. These results are in the same line with other studies[26,29], a study reported no change in serum lipid profile concentrations after 1 and 3 months of periodontal treatment in control chronic periodontitis group that received SRP alone while significant improvement in lipid profile (TC, LDL and HDL) of the test group (type 2 diabetes with chronic periodontitis) that received SRP with tetracycline fiber, and concluded that locally delivered tetracycline fiber is an effective treatment modality when used as an adjunct with SRP in patient with type 2 diabetes[26], another study conducted on 65 systemically healthy patients with chronic sever periodontitis divided into: untreated control group (group with no treatment), standard periodontal treatment receiving group (received SRP alone) and intensive periodontal treatment group (received SRP with local delivery of minocycline-HCL therapy) and reported that there were no significant differences in serum lipid levels of TC and LDL after 2 months of treatment[29].

In contrast to our results[13-31], a study demonstrated that periodontal therapy to 20 patients with both hyperlipidemia and chronic periodontitis by SRP could reduce the serum lipid levels of TG and TC, and increase HDL with no effect on LDL after 3 month of treatment[30], another study conducted on patients with chronic periodontitis and hypercholesterolemia revealed reduction in the serum lipid levels of TC and LDL after 3 months of SRP[31], also another study assayed plasma lipid concentration in chronic periodontitis patients with hyperlipidemia at baseline before and after 2 and 6 months of periodontal therapy and found that periodontal treatment significantly changed the plasma levels of TG and HDL, and reduced TC and LDL, and concluded that the lipid profile reduction in systemically healthy patients with chronic periodontitis might be due to the reduction of circulating proinflammatory cytokines Il-1 beta and TNF-alpha[13]. However, in this study the levels of lipid profile have been examined in patients with diabetes and this might be explain the

disagreement as diabetes have found to have an effect on lipid profile[32].

Conclusion

Scaling and root surface debridement have shown improvement in clinical parameters in all studied groups, whereas, no significant reduction of lipid profile in all groups were noticed. More studies with large samples size, long duration, and diet or physical exercise controlled will be required to assess the effect of periodontal therapy on lipid profile in chronic periodontitis with and without type 2 diabetes.

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