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Correlation Between Periodontal Status and Lipid Profile Among Type 2 Diabetic Patients

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Abstract

Background and Objectives: Type 2 diabetes mellitus is often accompanied by various complications, including periodontal disease and dyslipidemia. This study aimed to investigate the correlation between periodontal status and lipid profile among patients with type 2 diabetes mellitus.

Methods: This case-control study was conducted from June to December 2022 at the Diabetes and Endocrinology Center in Sulaimani, Kurdistan. 384 participants were selected using simple random sampling and divided into uncontrolled (140 participants) and controlled diabetes (244 participants) groups. Clinical periodontal examinations and blood investigations were conducted to measure periodontal parameters (bleeding on probing, plaque index, probing depth, clinical attachment loss) and lipid profiles (total cholesterol, high and low-density lipoprotein, and triglycerides).

Results: The study identified significant differences in periodontal parameters between uncontrolled and controlled groups, including clinical attachment loss (4.3 ± 8.0 mm vs. 1.7 ± 2.2 mm), visible plaque index (44.8 ± 16.4% vs. 28.7 ± 13.4%), bleeding index (26.0 ± 11.4% vs. 17.6 ± 12.0%), and average pocket depth (3.5 ± 2.0 vs. 1.5 ± 2.4) ($P \leq 0.001$). The study results indicated a significant and direct correlation between higher levels of total cholesterol, low-density lipoprotein, and triglycerides with periodontal disease, enhancing the risk of disease onset. Conversely, high-density lipoprotein was identified as a protective factor against periodontal disease ($P < 0.05$).

Conclusion: The study findings elucidate that elevated serum levels of total cholesterol, low-density lipoprotein, and triglycerides are significantly and directly correlated with periodontal disease, thereby increasing the susceptibility to this condition. Conversely, high-density lipoprotein serves as a protective agent against the disease.

Keywords: Cholesterol, Diabetes Mellitus, High-Density Lipoprotein cholesterol, Low-Density Lipoprotein cholesterol

Introduction

Type 2 diabetes, formerly referred to as adult-onset diabetes,[1] is a kind of diabetes mellitus characterised by hyperglycemia, insulin resistance, and a relative deficiency of insulin [2]. Around 90% of all diabetic patients are affected by this kind of diabetes, making it the most common diagnosis.[3] It is anticipated that the prevalence of diabetes will increase from 10.5% in the year 2021 to 11.3% in the year 2030 and 12.2% in the year 2040.[4] It is known to have a multitude of systemic complications, including cardiovascular diseases, nephropathy, neuropathy, and retinopathy.[5] Periodontal disease (PD), which is one of these consequences, has arisen as a serious worry owing to the fact that it has a double-sided interaction with diabetes.[6] Additionally, studies show a strong association between PD and atherosclerotic cardiovascular disease and diabetes.[7, 8]

The PD is a chronic inflammatory condition that occurs in a specific area. It is caused by the buildup of a harmful dental plaque biofilm above and below the gum line. This leads to an imbalance of microbes, resulting in a long-lasting and damaging inflammatory response that does not resolve independently.[9] Around the world, the prevalence of PD varies from 20% to 50%. However, it is anticipated that this number will rise.[10] According to the findings of many studies, the relationship between periodontitis and T2DM is a two-way street.[6] According to research by Wu et al. patients with T2DM have a 34% increased likelihood of developing periodontitis. Furthermore, severe periodontitis increases the likelihood of developing T2DM by 53%.[11]

Periodontitis is strongly associated with systemic inflammatory reactions, which are intensified in patients with diabetes. The presence of inflammation may impact how lipids are processed in the body, resulting in dyslipidemia. Increased TC, LDL, and TG values and decreasing HDL levels indicate dyslipidemia.[12]

While significant research has been conducted on the relationship between diabetes and PD, the existing literature is deficient in addressing the precise impact of lipid profiles on the periodontal status of individuals with T2DM. Furthermore, these conflicting findings need further research in this domain. Therefore, the current study aimed to evaluate the periodontal conditions of patients presenting with dyslipidemia and T2DM.

Patients and Methods

This case-control study was conducted from June 2022 to December 2022 at the Diabetes and Endocrinology Center in Sulaimani, Kurdistan,

The sample size estimate was determined based on the 50% incidence of PD (prevalence ratio) observed in previous research.[13] The confidence range used in the sample size computation was 90%, with an absolute error or precision of 5%. The estimates showed that the minimum sample size was 135 participants. Still, to increase the study's accuracy, 384 participants (140 uncontrolled diabetic patients and 244 controlled diabetic patients) were included in the current study. The participants were chosen using simple random selection and inclusion/exclusion criteria.

Patients in the age group of 18 to 75 years who had been diagnosed with diabetes based on the criteria provided by the World Health Organization (WHO),[13] in the last two years, those who had been visiting the mentioned center and had given their informed consent were included in the study. Exclusion criteria included individuals who had undergone antibiotic therapy within the previous month, smokers, patients with debilitating diseases affecting oral health, pregnant women, and those who had received periodontal therapy within the past six months.

Data collection involved both clinical periodontal examinations and blood investigations. The diagnostic criteria for T2DM included classic symptoms of hyperglycemia (polyuria, polydipsia, and unexplained weight loss) along with specific glucose levels. These glucose levels include a fasting plasma glucose (FPG) level of ≥ 126 mg/dL (7.0 mmol/L), a random blood sugar (RBS) of ≥ 200 mg/dL (11.1 mmol/L), or a 2-hour glucose level (OGTT) of ≥ 200 mg/dL (11.1 mmol/L). Additionally, a hemoglobin A1c (HbA1c) level of ≥ 6.5% (48 mmol/mol) is also considered diagnostic for T2DM.[14]

The periodontal parameters measured included Bleeding on Probing (BI), Plaque Index (PI), Probing Depth (PD), and Clinical Attachment Loss (CAL).[14] A standardized periodontal probe (Hu-Friedy, Chicago, IL, USA) was used for these measurements. Blood samples were collected to measure Glycosylated Hemoglobin (HbA1c) serum levels and lipid profiles including TC, HDL, LDL, and TG. Blood analyses were conducted using an automated analyzer (Cobas® b 101, Roche, Switzerland).

This study protocol was reviewed and approved by the Kurdistan Higher Council of Medical Specialties Ethical Committee, approval number [1207], on June 2, 2022. All participants provided verbal and written agreement before their involvement in the study. Individuals were guaranteed secrecy and anonymity, and their participation was optional, allowing them to quit at any point without facing any consequences.

Version 25.0 of SPSS software (IBM Corp., Armonk, NY, USA) was used to analyze the data. The clinical parameters and demographic features were compiled using descriptive statistics. The Chi-square test was used to compare categorical variables. The means of continuous variables in the case and control (CG) groups were compared using the Mann-Whitney U test. A Spearman's correlation coefficient was used to evaluate the association between periodontal characteristics and lipid profiles. Statistical significance was considered to be achieved when the p-values were below 0.05.

Results

Table (1) presents the demographic characteristics of patients in both the uncontrolled (UG) and controlled groups (CG). Most patients in both groups were 45 to 64 years old. Specifically, 112 (80%) in the UG and 164

(67.2%) in the CG were within this age range, indicating a statistically significant difference (P < 0.007). A notable disparity in sex distribution was observed, with 128 (91.4%) females in the UG and 193 (79%) females in the CG (P < 0.002).

Table (1): The demographic characteristics of participants

Variables		Uncontrolled	Controlled	Total	P-value *
Age	36 - 44 Years	8 (7.7%)	39 (16%)	47 (12.2%)	0.007
	45 - 64 Years	112 (80%)	164 (67.2%)	276 (71.9%)	
	> 64 Years	20 (12.3%)	41 (16.8%)	61 (15.9%)	
Sex	Male	12 (8.6%)	51 (21%)	63 (16.5%)	0.002
	Female	128 (91.4%)	193 (79%)	321 (83.5%)	
Total		140 (100%)	244 (100%)	384 (100%)	

* Performed by Chi-square test

Significant variations were observed in the study of periodontal parameters between the two groups. The mean Clinical Attachment Loss (CAL) was 4.3 ± 8.0 mm in the UG and 1.7 ± 2.2 mm in the CG (P ≤ 0.001). The Visible Plaque Index (VPI) was 44.8 ± 16.4% in the UG and 28.7 ± 13.4% in the CG, indicating significant variation (P ≤ 0.001). Furthermore, the Bleeding Index (BI) was 26.0 ± 11.4% in the UG and 17.6 ± 12.0% in the CG (P ≤ 0.001); while the average pocket depth was 3.5 ± 2.0 mm in the UG versus 1.5 ± 2.4 mm in the CG, both showing significant differences (P ≤ 0.001) Table (2).

Table (2): Periodontal Parameters for the uncontrolled and controlled Group

Periodontal parameters	Uncontrolled (n = 140)		Controlled (n = 244)		P-value*
	Mean ± SD	Median	Mean ± SD	Median	
CAL (mm)	4.3 ± 8.0	3	1.7 ± 2.2	0	< 0.001
VPI %	44.8 ± 16.4	46	28.7 ± 13.4	28	< 0.001
BI %	26.0 ± 11.4	27.5	17.6 ± 12.0	14	< 0.001
Avg. Pocket Depth (mm)	3.5 ± 2.0	4	1.5 ± 2.4	0	< 0.001

* Performed by Mann-Whitney test

Table (3) shows the lipid profile values for both groups. The mean TC level was 186.7 ± 38.5 mg/dl in the UG and 167.7 ± 40.7 mg/dl in the CG, highlighting a significant difference (P ≤ 0.001). LDL cholesterol levels were 111.1 ± 34.5 mg/dl in the UG and 95.1 ± 29.7 mg/dl in the CG, showing significant differences (P ≤ 0.001). HDL cholesterol levels were 45.6 ± 11.1 mg/dl in the UG and 45.8 ± 7.8 mg/dl in CG. Triglyceride (TG) levels were significantly different, at 237.9 ± 192.2 mg/dl in the UG and 165.9 ± 120.5 mg/dl in the CG (P ≤ 0.001). The mean HbA1c was 9.4 ± 1.5 mg/dl in the UG and 6.1 ± 0.4 mg/dl in the CG, indicating a significant discrepancy (P ≤ 0.001).

Table (3): The Lipid Profile for the Case and CG

Lipid Profile	Uncontrolled (n = 140)		Controlled (n = 244)		P-value *
	Mean ± SD	Median	Mean ± SD	Median	
T. Cholesterol (mg/dl)	186.7 ± 38.5	185	167.7 ± 40.7	156.5	< 0.001
LDL (mg/dl)	111.1 ± 34.5	105	95.1 ± 29.7	85	< 0.001
HDL (mg/dl)	45.6 ± 11.1	44	45.8 ± 7.8	45	0.09
Triglyceride (mg/dl)	237.9 ± 192.2	196	165.9 ± 120.5	144	< 0.001
HbA1c %	9.4 ± 1.5	9.2	6.1 ± 0.4	6.1	< 0.001

Table (4) presents the correlation of lipid profile components and periodontal parameters across all patients. The TC showed no significant correlation with CAL, VPI, or BI, yet displayed a weak, positive, and significant correlation with average pocket depth (r = 0.12, P ≤ 0.02). LDL showed no significant correlation with CAL but had a weak, positive, and significant correlation with VPI (r = 0.14, P ≤ 0.007), BI (r = 0.15, P ≤ 0.002), and average pocket depth (r = 0.2, P ≤ 0.001). HDL had no significant correlations with VPI or BI but had a weak, indirect, and significant correlation with CAL (r = -0.19, P ≤ 0.001) and with average pocket depth (r = -0.24, P ≤ 0.001). Triglyceride demonstrated a weak, positive, and significant correlation with CAL, VPI, BI, and average pocket depth (r = 0.1, P ≤ 0.05).

Table (4): linear correlation between each component of lipid profile and periodontal parameters for all patients

lipid profile and periodontal parameters Total participants (n = 384)		Spearman Correlation coefficient (Rho)	P-value
T. Cholesterol (mg/dl)	CAL (mm)	-0.043	0.40
	VPI %	0.09	0.08
	BI %	0.09	0.08
	Avg. Pocket Depth (mm)	0.12	0.02
LDL (mg/dl)	CAL (mm)	0.04	0.5
	VPI %	0.14	0.007
	BI %	0.154	0.002
	Avg. Pocket Depth (mm)	0.2	< 0.001
HDL (mg/dl)	CAL (mm)	-0.196	< 0.001
	VPI %	0.039	0.45
	BI %	-0.08	0.11
	Avg. Pocket Depth (mm)	-0.241	< 0.001
Triglyceride (mg/dl)	CAL (mm)	0.16	0.002
	VPI %	0.12	0.02
	BI %	0.159	0.002
	Avg. Pocket Depth (mm)	0.18	0.001

Table (5) shows the correlation of each component of the lipid profile and periodontal parameters. The analysis of lipid profiles among the UG revealed that LDL did not correlate significantly with other lipid components. The TC showed a weak, indirect, and significant correlation with CAL (r= -0.16, p≤0.05). HDL did not correlate significantly with CAL but displayed a weak, indirect, and significant correlation with the VPI (r= -0.19, p≤0.02), the BI (r= -0.24, p≤0.004), and an average pocket depth indicating a moderate, indirect, and significant correlation (r= -0.34, p≤0.001). Triglyceride showed no significant correlation with CAL or the VPI, yet exhibited a weak, direct, and meaningful relationship with the BI and average pocket depth (r= 0.10, p≤0.05). In the CG, TG did not display significant correlations with other lipid profile components. LDL correlated weakly and directly only with average pocket depth (r= 0.14, p≤0.02). HDL showed no significant relationship with the BI but had a weak, indirect, and significant correlation with CAL (r= -0.25, p≤0.001), a weak, direct, and significant relationship with the BI (r= 0.15, p≤0.02), and a weak, indirect, and meaningful correlation with average pocket depth (r= -0.16, p≤0.01). Lastly, TG had a weak, indirect, and noteworthy correlation only with the VPI (r= -0.16, p≤0.002) Table (5).

Table (5): Linear correlation between each component of lipid profile and periodontal parameters for Uncontrolled and Controlled group

lipid profile and periodontal parameters		Spearman Correlation coefficient (Rho)	
		Uncontrolled (P value)	Controlled (P value)
T. Cholesterol (mg/dl)	CAL (mm)	-0.166 (0.050)	-0.098 (0.13)
	VPI %	0.065 (0.45)	-0.114 (0.08)
	BI %	-0.053 (0.54)	0.009 (0.89)
	Avg. Pocket Depth (mm)	-0.144 (0.09)	0.111 (0.08)
LDL (mg/dl)	CAL (mm)	-0.075 (0.38)	-0.02 (0.73)
	VPI %	0.11 (0.22)	-0.05 (0.42)
	BI %	0.03 (0.77)	0.096 (0.14)

	Avg. Pocket Depth (mm)	-0.004 (0.96)	0.144 (0.02)
HDL (mg/dl)	CAL (mm)	-0.09 (0.31)	-0.256 (< 0.001)
	VPI %	-0.198 (0.02)	0.15 (0.02)
	BI %	-0.241 (0.004)	0.02 (0.770)
	Avg. Pocket Depth (mm)	-0.34 (< 0.001)	-0.162 (0.01)
Triglyceride (mg/dl)	CAL (mm)	-0.05 (0.56)	0.08 (0.23)
	VPI %	0.16 (0.06)	-0.196 (0.002)
	BI %	0.197 (0.020)	-0.09 (0.150)
	Avg. Pocket Depth (mm)	0.17 (0.04)	0.02 (0.79)

Discussion

This study examined the periodontal status of patients suffering from dyslipidemia and T2DM. Periodontal parameters and lipid profiles were notably higher in dyslipidemic and diabetic patients compared to a CG. The results revealed a significant direct correlation between TC and average pocket depth, LDL and VPI percentage, BI percentage, and average pocket depth, as well as TG and CAL, index percentage, BI percentage, and average pocket depth. As each lipid profile component increased, so did the periodontal degradation in these patients. Additionally, an inverse and significant relationship was observed between HDL and periodontal parameters, indicating decreased periodontal degradation with higher HDL levels.

Research by Mirzaei et al. explored the relationship between lipid profiles and periodontal health. According to the research, dyslipidemia raises the chance of PD. Although there is no statistically noteworthy direct relationship between lipid profiles and periodontal parameters, the average lipid profiles were more significant in patients than in healthy persons. Such findings suggest a heightened susceptibility to periodontal disease in individuals with dyslipidemia.[15] In a study by Bitencourt et al., lipid profiles, including TC, LDL, HDL, and TG, were assessed using the National Cholesterol Education Program standards. Periodontal evaluation followed the NHANES III examination protocol. Findings indicated that dyslipidemia significantly impacted the increase of PD and was also mediated through HbA1c and obesity.[12]

Evidence suggests a bidirectional and reciprocal relationship between PD and dyslipidemia.[16] Through systemic inflammation, PD can elevate pro-inflammatory cytokines, altering blood lipid levels and inducing abnormal lipid metabolism.[17] Conversely, lipid disorders, by disrupting immune regulation and increasing oxidative stress, heighten the risk of PD.[18] Periodontal disease, characterized as a localized inflammation caused by oral microorganisms, also involves the release of pro-inflammatory cytokines, linking it to systemic diseases such as dyslipidemia, cardiovascular diseases, atherosclerosis, and T2DM.[6, 19]

The findings showed a significant and direct relationship between TC and LDL levels and periodontal parameters in the subjects examined. Similarly, a study by Tran et al., demonstrated that elevated cholesterol could potentially contribute to PD, with changes in gene expression resulting from inflammatory responses in periodontal cells leading to its pathogenesis.[20] Research by Ayoobi et al. investigated the association between dyslipidemia, diabetes, and PD. The study revealed that high lipid profiles, specifically increased TC and LDL levels, together with diabetes, boost the likelihood of developing PD.[21] The current study also revealed a relationship between TG levels and periodontal parameters. In this context, research by Kim et al. found that high TG levels were associated with PD and increased the risk of developing this condition.[22]

Examining the relationship between HDL and periodontal parameters indicated that HDL has a protective role against PD, with higher levels as a protective factor. Research by Chen et al. consistently showed that HDL reduces the risk of PD, whereas TC, LDL, and TG increase this risk.[23] It was also determined that T2DM is associated with PD and increases the risk of its onset. The study by Dewake et al. also indicated that diabetes could heighten the risk of PD.[9] In contrast, a study in Brazil by Valentim et al. did not demonstrate this relationship, which may be attributed to differences in methodology and sample size compared to the current study.[24]

Periodontal disease and elevated blood lipid levels should be considered significant health conditions that require more attention. Given the established relationship in this study, it is crucial to recognize that high blood lipid levels and diabetes are key risk factors for cardiovascular and atherosclerotic diseases. Thus, further detailed research is necessary to elucidate these relationships' precise nature and underlying mechanisms.

Ultimately, more focus is needed on preventive and therapeutic policies concerning oral health and the management of lipid levels to enhance overall community health.

Conclusions

The study results confirmed that elevated serum levels of TC, LDL, and TG are significantly and directly associated with PD, increasing the risk of its development. HDL serves as a protective factor, reducing the risk of periodontitis in both controlled and uncontrolled T2DM. Additionally, a positive association between T2DM and PD was established, with diabetes increasing the risk of the disease.

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