Original Article

Glycemic control in patients with type 1 diabetes mellitus affects periodontal health but not salivary status: An observational study

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Abstract:

Aim: The aim of this observational study was to assess the periodontal and salivary status of patients with type 1 diabetes mellitus (T1DM). **Materials and Methods:** Thirty patients were divided into a test group (DM1G – T1DM, $n = 15$) and a control group (CG – normoglycemic, $n = 15$). Periodontal clinical parameters were evaluated using probing depth (PD), bleeding on probing (BOP), clinical attachment level (CAL), and plaque index (PI). Salivary tests assessed pH, buffering capacity, salivary glucose, and peroxidase activity. Capillary blood glucose was recorded for all patients, and glycated hemoglobin (HbA1c) was measured only for patients with T1DM. Statistical analysis was performed using Student's *t*-test and Pearson's correlation coefficient ($P < 0.05$). **Results:** The results showed significantly higher PI and BOP values in DM1G ($P < 0.01$). Positive correlations were found between HbA1c, PI, and BOP ($P < 0.01$). Capillary blood glucose levels differed between groups ($P < 0.01$). Positive correlations between capillary blood glucose, PD, and CAL were found only in the CG ($P < 0.01$). No significant differences were observed between the groups for salivary parameters ($P > 0.05$). **Conclusions:** Patients with T1DM exhibit poorer periodontal conditions compared to normoglycemic individuals, although no significant differences were found in salivary parameters between the two groups.

Keywords:

Diabetes mellitus, periodontal disease, saliva

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INTRODUCTION

Diabetes mellitus (DM) is a group of clinically and genetically heterogeneous metabolic disorders characterized by chronic elevation of blood glucose levels.[1] Type 1 DM (T1DM) accounts for approximately 5% to 10% of all DM cases. T1DM results from the destruction of beta-pancreatic cells (autoimmune or idiopathic), leading to a consequent deficiency in insulin secretion. Although T1DM predominantly develops in children or adolescents (ages 10–14), some studies report that 15% to 30% of new cases occur in patients older than 30 years.[2] There has been a notable increase in the incidence of T1DM, particularly among children under 5 years of age.[3] In Bauru, a city in the interior of Brazil, the incidence of T1DM has been classified as high or very high over the past decade, with a 9.6-fold increase in incidence observed between 1987 and 2002.[4]

The three primary symptoms of DM are polyuria, polydipsia, and polyphagia. In addition, the chronic state of hyperglycemia can lead to microvascular complications such as nephropathy, retinopathy, neuropathy, as well as macrovascular complications such as coronary artery disease, cerebrovascular disease, and peripheral vascular disease.[5] Individuals with poorly controlled diabetes may present with oral alterations, including reduced salivary flow, variations in saliva composition, taste disorders, burning mouth syndrome, frequent oral infections, delayed wound healing, caries, coated tongue, and halitosis.[6]

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Alterations in oral biofilm are among the factors responsible for the development of caries and periodontal disease.[7] It is well established in the literature that periodontal disease is more prevalent and severe in patients with DM. Therefore, periodontal disease is considered the sixth complication of DM.[8] Metabolic alterations in periodontal tissues may reduce the resistance of patients with diabetes to infection. These alterations influence the initiation, development, and progression of periodontal disease.[9] The impact of DM on the periodontium is associated with both macro- and microvascular complications.[10] Hyperglycemia affects periodontal disease by altering the immune response to bacteria, increasing the presence of hyper-responsive macrophages, and promoting the production of advanced glycation end-products.[1,10] Conversely, the inflammatory status of periodontal disease impairs metabolic control, increases insulin resistance, and raises the risk of other complications associated with diabetes.[1,11]

There are a few studies comparing periodontal status of patients with T1DM and those with normoglycemia, which have found inferior periodontal conditions in the T1DM group.[12] In contrast, another investigation encountered no difference in periodontal and microbiological parameters between patients with T1DM and those with normoglycemia.[13]

Studies comparing salivary conditions and periodontal disease in patients with T1DM are scarce. One study observed a higher prevalence and severity of periodontal disease in patients with T1DM.[14] A correlation was found between gingival index and levels of total protein, albumin, lysozyme, and lactoferrin in whole saliva for both patients with and without diabetes. Another study reported significant alterations in the saliva of patients with T1DM, including changes in potassium, total protein, amylase, and secretory IgA levels. In addition, patients with diabetes exhibited significantly greater probing depths (PDs).[15] Salivary alterations, such as lower pH, buffering capacity, and peroxidase activity have been described in both newly diagnosed and long-term patients with T1DM.[16] Moreover, periodontal clinical parameters such as plaque index (PI), gingival index, and mean PD were higher in long-term T1DM patients, indicating that glycemic status of patients with DM affects periodontal health.[16] None of the aforementioned studies conducted a complete periodontal evaluation, including PD, clinical attachment level (CAL), and bleeding on probing (BOP) at six sites per tooth.

Therefore, the aim of the present study was to evaluate the periodontal and salivary conditions of patients with T1DM and correlate these conditions with glycemic status.

MATERIALS AND METHODS

This cross-sectional observational study was approved by the Ethics Committee in Human Research (#640.249) and registered on Clinical Trials.gov (NCT02935868).

The participants were divided into two groups: type 1 diabetes mellitus group (DM1G) and control group (CG).

Patients with T1DM were recruited from 2013 to 2015 at periodontology clinics.

A total of 22 patients were examined and sampled according to the inclusion and exclusion criteria. The inclusion criteria was a diagnosis of T1DM by an endocrinologist, age of DM1G between 18 and 45 years, and the presence of at least one tooth per quadrant. The exclusion criteria were a diagnosis of type 2 DM, edentulous patients, and the presence of other systemic diseases.

The DM1G comprised 15 patients, whereas the CG comprised 15 healthy volunteers within the same age range as the DM1G group. The inclusion criteria were age between 18 and 35 years and the presence of at least one tooth per quadrant. The exclusion criteria were the presence of systemic diseases and periodontal diseases.

A full-mouth periodontal clinical examination was conducted by a calibrated specialist ($\kappa = 0.82$). Periodontal parameters were evaluated using PD, CAL, BOP, and PI at six sites per tooth. Subsequently, the prevalence of gingivitis and periodontitis was categorized according to a previous study.[17] Chronic periodontitis was categorized as follows:

- Mild periodontitis: Gingival inflammation and BOP, periodontal pocket depth ≤4 mm, and attachment loss of 1–2 mm
- Moderate periodontitis: Gingival inflammation and BOP, presence of pus, periodontal pocket depth ≤6 mm, attachment loss of 3–4 mm, and possible slight tooth mobility
- Severe periodontitis: Obvious inflammation or occurrence of periodontal abscess, periodontal pocket depth >6 mm, attachment loss ≥5 mm, and one tooth mobility.[18]

Capillary blood glucose was measured with a glucose meter in the morning, before the dental examination. Glycated hemoglobin (HbA1c) levels were recorded for the DM1G.

For the salivary analysis, stimulated saliva was collected between 8:00 am and 10:00 am. Patients were instructed to chew a piece of Parafilm (Bemis NA, Wisconsin, USA) and to spit the saliva into sterile tubes for 10 min. The saliva was kept on ice until it was transported to the laboratory for pH and buffering capacity tests. Subsequently, the samples were stored at-80°C.

Salivary pH was measured using a pH meter, and buffering capacity was evaluated. A pH of 6–7 was considered normal.[19] Buffering capacity was classified as high (≥5.6), medium (4.1–5.5), or low (≤4).[20]

For the analysis of salivary glucose and peroxidase activity, the samples were thawed at room temperature and centrifuged at 5000 rpm for 10 min to collect only the supernatant. Salivary glucose was quantified using a colorimetric test (Glucose Liquicolor – *In vitro* Diagnostica – Itabira, Brazil) in a spectrophotometer. Results were presented in mg/dL after conversion. The activity of salivary peroxidases was evaluated according to previous protocols.[21]

Statistical analysis was performed using the Student's *t*-test for comparisons between groups and Pearson's correlation coefficient for examining relationships between all variables ($P < 0.05$).

RESULTS

A total of 37 patients were recruited for the study. Three patients were excluded due to age, three patients declined to participate, and one missed the appointments. Demographic data are presented in Table 1. The mean age was similar between both groups. The mean HbA1c (10.39%) indicated poor metabolic control in the DM1G. Capillary blood glucose was significantly higher in the DM1G ($P < 0.05$).

Regarding periodontal clinical parameters, patients with T1DM exhibited a significantly higher percentage of BOP and PI [Table 2].

Statistical tests were used to evaluate the correlations between HbA1c, capillary blood glucose, and periodontal clinical parameters. Positive correlations were observed between PD and CAL for both groups; PD and PI only for DM1G; PD and BOP/CAL and BOP for the CG; and PI and BOP for both groups [Table 3]. For the DM1G, strong positive correlations were found between HbA1c and BOP, as well as HbA1c and PI. Regarding capillary blood glucose, only the CG showed a strong correlation with CAL and PD [Table 3].

All tested salivary parameters were similar for both groups [Table 4]. In general, all patients presented high buffering capacity.

There was a weak correlation between PD and CAL, as well as salivary glucose in the DM1G [Table 5].

For the periodontal clinical examination, 2448 sites were evaluated in the DM1G, and 2466 sites were examined in the CG. These sites were categorized as healthy, mild periodontitis, moderate periodontitis, and severe periodontitis.[18] The DM1G comprised 68% healthy subjects and 21% with mild periodontitis, whereas the CG had 78% and 20%, respectively. The DM1G presented a higher percentage of moderate (6%) and severe (5%) periodontal disease compared to the CG (2% moderate and no severe).

DISCUSSION

The present study demonstrated poorer periodontal conditions in patients with T1DM compared to those without diabetes, with higher PI and BOP. Moreover, positive correlations were observed between salivary glucose, PD, and CAL. No differences were detected in salivary pH, buffering capacity, or peroxidase activity.

The positive correlation between PI, BOP, and HbA1c is consistent with previous reports suggesting that the glycemic status of patients with DM affects periodontal health.[16]

Previous studies have reported the dependence of severity of periodontal disease of severity of periodontal disease on the time lapsed from diagnosis of T1DM.[22] In the present study, only 5% of patients presented severe periodontal disease, possibly due to their young age. The mean time since diabetes diagnosis was 11.8 years, and the mean age was 27.7 years. This mean age is considered below the typical onset age for chronic periodontal disease in the general population (35 years old).

Table 1: Demographic data

	DM1G	CG
Age (years), mean±SD	27.73±8.51	26.02±3.94
Women (%)	53.33	66.67
Men (%)	46.67	33.33
Duration of T1DM diagnosis (years), mean±SD	11.8±6.37	-
HbA1c (%), mean±SD	10.39±2.12	-
Capillary blood glucose (mg/dL)	206.6±94.54*	92.13±8.31*

*Student's *t*-test=*P*<0.05. DM1G – Type 1 diabetes mellitus group and CG - normoglycaemic patients group. SD – Standard deviation; HbA1c – Glycated hemoglobin; DM1G – Type 1 diabetes mellitus group; CG – Control group; T1DM – Type 1 diabetes mellitus

Table 2: Periodontal clinical parameters

	PD (mm) mean±SD	CAL (mm), mean±SD	BOP (%), mean±SD	PI (%), mean±SD
DM1G	2.18±0.66	0.79±1.15	36.31±15.83*	78.93±23.12*
CG	1.82±0.35	0.31±0.25	17.84±11.86*	55.87±18.03*

**P*<0.05 between groups (Student's *t*-test). Comparison between DM1G – type 1 diabetes mellitus group and CG - normoglycaemic patients group. SD – Standard deviation; DM1G – Type 1 diabetes mellitus group; CG – Control group; T1DM – Type 1 diabetes mellitus; CAL – Clinical Attachment Level; PD – Probing depth; BOP – Bleeding on probing; PI – Plaque index; *P* – Probability value

Table 3: Pearson's correlation test (*r*) for periodontal clinical parameters, glycated hemoglobin and capillary blood glucose

	DM1G (*r*)	Correlation power	CG (*r*)	Correlation power
PD and CAL	0.8816*	Very strong	0.8901*	Very strong
PD and BOP	0.5079	-	0.5514*	Moderate
PD and PI	0.5162*	Moderate	0.4515	-
CAL and BOP	0.2771	-	0.6239*	Strong
BOP and PI	0.8595*	Very strong	0.7079*	Strong
BOP and HbA1c	0.7743*	Strong	-	-
PI and HbA1c	0.7004*	Strong	-	-
PD and blood glucose	0.1034	-	0.6427*	Strong
CAL and blood glucose	0.0645	-	0.6149*	Strong

*Statistically significant correlation (*P*<0.05). DM1G – Type 1 diabetes mellitus group and CG - normoglycaemic patients group. DM1G – Type 1 diabetes mellitus group; CG – Control group; CAL – Clinical attachment level; PD – Probing depth; BOP – Bleeding on probing; PI – Plaque index; HbA1c – Glycated hemoglobin; Blood glucose – Capillary blood glucose; *P* – Probability value

Table 4: Saliva analysis

	pH	Buffering capacity	Salivary glycemia (mg/dL)	Salivary peroxidases activity (U/mg)
DM1G	5.00±0.56	6.69±0.28	19.52±23.69	0.629±0.68
CG	5.41±0.63	6.48±0.59	6.99±17.09	0.748±0.89

Comparison between DM1G – Type 1 diabetes mellitus group and CG - normoglycaemic patients group. Student's *t*-test=*P*>0.05. Mean±SD. U/mg – Enzymatic specific activity unit; DM1G – Type 1 diabetes mellitus group; CG – Control group; SD – Standard deviation; *P* – Probability value

Since correlations between the time since diabetes diagnosis and the extent of periodontal disease in young patients may exist, this relationship may not be observed in some studies.[10,13] Age is considered a predisposing factor in the relationship between DM and periodontal disease. With advancing age, all factors involved in both diseases become more pronounced and can be more strongly associated.[11] In the present study, we intentionally

Table 5: Correlation between salivary glucose and periodontal parameters in Type 1 diabetes mellitus group and control group

	DM1G	CG	Correlation power
Salivary glucose and PD	0.6278*	0.2338	Weak
Salivary glucose and CAL	0.6711*	0.1239	Very weak
Salivary glucose and BOP	0.2021	0.2671	-
Salivary glucose and PI	0.2882	-0.0174	-

*Pearson's correlation, *P*<0.05. CAL – Clinical attachment level; PD – Probing depth; BOP – Bleeding on probing; PI – Plaque index; DM1G – Type 1 diabetes mellitus group; CG – Control group (normoglycaemic patients); *P* – Probability value

selected a younger sample to determine whether type 1 diabetes would contribute to worse periodontal conditions, and the study demonstrated poorer periodontal conditions in these patients.

The mean HbA1c in the test group was 10.39%, indicating poor metabolic control. Only 13.33% of the DM1G presented good metabolic control, with HbA1c ≤7%. A correlation was observed between HbA1c, PI, and BOP, suggesting that patients with poor metabolic control have a higher prevalence of gingival inflammation. These results indicate that glycemic levels may contribute to poorer periodontal status, consistent with previous studies that demonstrated higher periodontal disease severity, PD, and CAL in patients with DM.[22] Conversely, some studies found no relationship between glycemic control and periodontal health. This discrepancy may be attributed to the younger age of patients in those studies, who generally have a lower prevalence of periodontal disease.[23]

Positive correlations between capillary blood glucose and periodontal parameters were observed only in the CG, suggesting that lower glycemic values are associated with better periodontal health. However, some reports indicate higher glucose levels in patients with periodontal disease despite having normoglycemia. In this context, periodontal disease may be a risk factor for DM, as inflammatory cytokines associated with periodontal disease can lead to insulin resistance.[11]

In the present study, the DM1G exhibited 18.4% and 23% higher BOP and PI, respectively, compared to the CG. These results are consistent with other studies reporting higher gingival bleeding in patients with poor glycemic control.[1] Other periodontal parameters, such as PD and CAL, were similar between the groups, with differences in CAL being <0.5 mm. A systematic review reported a mean CAL difference of 1 mm between patients with type 2 diabetes and those with normoglycemia, and no significant differences found between patients with T1DM and those with normoglycemia.[24] The authors attributed these results in patients with T1DM to their young age, noting that there may not be sufficient time for severe periodontal disease to develop in younger patients.[24] Although differences in mean CAL were not significant, when patients were stratified by the severity of periodontal disease, those with T1DM presented a higher prevalence and severity of the disease. In the CG, 78% were healthy and 22% had mild periodontitis. In the DM1G, 68% were healthy, 21% presented mild periodontitis, 6% had moderate periodontitis, and 5% had severe periodontitis.

Saliva plays an important role in maintaining oral health and may be affected by systemic diseases and medications.[16] Patients with DM may present alterations in saliva composition and secretion, although the results are contradictory.[25] There is a significant association between DM, caries, periodontal disease incidence, and a reduction in salivary pH.[26] While some studies report lower salivary pH[16,25] and altered buffering capacity[16] in patients with T1DM, others have found no differences.[27]

The majority of studies have reported higher glucose concentrations in the saliva of patients with DM, with positive correlations to other salivary components.[28,29] In contrast, our results demonstrated no differences in salivary glucose between the groups and no correlation with capillary blood glucose or HbA1c. One study supports our findings, showing no relationship between salivary glucose and capillary blood glucose.[30] These contradictory findings may be explained by differences in the mean age of patients and variations in saliva collection methods (stimulated, nonstimulated, whole saliva, or specific glands).

A positive correlation between periodontal parameters (PD and CAL) and salivary glucose was found in patients with T1DM. Although causation cannot be established, this finding may suggest a relationship between high concentrations of glucose in saliva and periodontal disease. High glucose levels in the saliva of patients with DM can contribute to an increased susceptibility to infections, such as oral candidiasis and caries.[31] Xerostomia and reduced salivary flow are consequences of hyperglycemia and are considered predisposing factors for periodontal disease.[16,31]

The salivary peroxidase system is one of the most important nonimmunologic defense mechanisms in saliva. This enzyme regulates microorganisms in the oral cavity, prevents the accumulation of toxic byproducts of hydrogen peroxide, possesses antimicrobial properties, and inactivates cariogenic and mutagenic compounds.[32] Salivary peroxidase activity is increased in patients with gingivitis.[33] In relation to patients with T1DM, some studies showed higher enzymatic activity in saliva,[34] whereas others have not detected it,[16] as was the case in our study.

The present study has some limitations, such as a small sample size, which may limit the generalizability of the findings, and the collection of whole saliva rather than saliva from individual glands. However, investigations into the correlations between all variables, such as periodontal clinical parameters (six sites per tooth/whole mouth), capillary blood glucose, HbA1c and salivary status, are scarce. Despite these limitations, this study demonstrated a relationship between poor glycemic control and periodontal disease. In addition, low levels of salivary glucose were associated with periodontal health. Future research involving an older cohort of patients with T1DM, a larger sample size, the isolation of confounding factors, and different methodologies in salivary analysis may confirm the relationship between these conditions and potentially identify new correlations with saliva components.

CONCLUSION

Patients with T1DM exhibited poorer metabolic control and worse periodontal status. A positive relationship was found between higher levels of salivary glucose and periodontal disease. The salivary condition was similar in patients with and without diabetes.

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Conflicts of interest
There are no conflicts of interest.

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