

Association of periodontal status and metabolic control in periodontitis patients with diabetes mellitus in Hospital Pakar Universiti Sains Malaysia

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Abstract

Evidence establishes diabetes mellitus (DM) as a significant risk factor for periodontitis. Periodontitis with DM patients exhibit more pronounced periodontal loss, elevated clinical attachment levels (CAL), and deeper periodontal pockets. This study aims to determine the association between periodontal status and metabolic control indicators in periodontitis with type 2 DM patients. A retrospective record review study was conducted from 2015 to 2024 at Periodontal Clinic, Hospital Pakar Universiti Sains Malaysia. All data of periodontitis with Type 2 DM patients were recorded and analyzed using SPSS 29.0. Demographic data was analysed descriptively and Fisher's exact test was used to evaluate the association between periodontal status and metabolic control indicators. Forty two periodontitis patients were included with the mean (SD) age of 59.26 (9.97) years and male predominance (61.9%). Most patients were Malay (97.6%), and 28.6% had blood pressure of more than 140/90 mmHg. Periodontal status revealed 61.9% of patients were in stage 3 and 14.3% in stage 4 with grade B periodontitis was the most common (59.5%). Metabolic control indicators showed 64.3% of patients has HbA1c of $\geq 7.0\%$, 83.3% has fasting blood glucose of >6.0 mmol/L, and 54.8% with LDL of >2.6 mmol/L. Fisher's exact test analysis shows no significant association between periodontal severity and metabolic control indicators, including HbA1c ($p=0.513$), fasting blood glucose ($p=0.539$), and fasting lipid profiles ($p \geq 0.05$). The findings highlight a high prevalence of severe periodontitis among patients with poor glycemic control. However, no significant association of periodontal status and metabolic control indicators.

Keywords: *metabolic control indicators, periodontal severity, periodontitis, type 2 DM*

Introduction

Periodontitis is a chronic inflammatory disease that leads to the progressive destruction of the supporting structures of the teeth due to an immune response triggered by anaerobic Gram-negative microorganisms (Preshaw *et al.*, 2011). It is characterized by clinical attachment loss

(CAL), probing depth (PD), bleeding on probing (BOP), and radiographic evidence of bone loss (Tonetti *et al.*, 2018). The classification of periodontitis is based on staging and grading system. Staging reflects the disease severity and complexity of the management, while grading is an indicator of periodontitis progression and influenced by risk factors such as smoking and diabetes mellitus (Papapanou *et al.*, 2018). According

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to the National Oral Health Survey of Adults (NOHSA) conducted by the Ministry of Health Malaysia in 2020, 94.0% of Malaysian adults have periodontal disease with 48.5% of them having periodontitis. In fact, the prevalence of severe periodontitis was higher (18.2%), as compared to the global prevalence (Anuwar *et al.*, 2024). The prevalence of severe periodontitis has notably increased from 6.0% in 1990 to 17.8% in 2010 (Syakimah, 2013), highlighting the growing burden of the disease.

Diabetes mellitus (DM) is a metabolic disorder characterized by chronic hyperglycemia due to defects in insulin secretion, insulin action, or both (American Diabetes Association, 2009). Metabolic control indicators include glycated hemoglobin (HbA1c), fasting blood glucose (FBG), and lipid profiles. HbA1c reflects average blood glucose levels over the past two to three months, with levels above 7% indicating poor glycemic control (Eyth & Naik, 2022). Fasting blood glucose usually measured after an 8-hour fasting, should ideally remain below 5.6 mmol/L (Riley, 2023). Lipid profiles assess total cholesterol, triglycerides, low-density lipoprotein (LDL), and high-density lipoprotein (HDL) to evaluate cardiovascular risk in diabetic patients (Reddy *et al.*, 2022). DM is a well-established systemic risk factor for periodontitis. The prevalence of diabetes in Malaysia ranges between 7.3% and 23.8% (Akhtar *et al.*, 2022), with Type 2 diabetes mellitus (T2DM) being the most common form. Type 1 diabetes results from autoimmune destruction of pancreatic β -cells, leading to complete insulin deficiency (Sameer *et al.*, 2020), whereas Type 2 diabetes is characterized by insulin resistance and progressive β -cell dysfunction.

Epidemiological studies have consistently demonstrated that individuals with diabetes are approximately three times more susceptible to periodontitis. The US National Health and Nutrition Examination Survey (NHANES) III reported that adults with HbA1c levels exceeding 9.0% had a significantly higher prevalence of severe

periodontitis compared to non-diabetics, even after adjusting for age, sex, smoking, and socioeconomic status (Preshaw *et al.*, 2011). Similarly, studies on the Pima Indian population showed that individuals with T2DM had an approximately threefold increased risk of developing periodontitis compared to those without diabetes (Preshaw *et al.*, 2011).

Despite strong evidence supporting the link between diabetes and periodontitis, there is limited local data assessing the association between periodontal status and metabolic control in Malaysian periodontitis with T2DM patients. Given the increasing prevalence of diabetes and periodontitis in Malaysia, therefore, it is a need for targeted research for better understanding on how metabolic control parameters influence the periodontal health. This study aims to address this gap by analysing the association between periodontal severity and metabolic indicators among periodontitis with T2DM patients at Hospital Pakar Universiti Sains Malaysia.

Specifically, this study aims to evaluate the periodontal status of T2DM patients, assess metabolic control indicators such as HbA1c, FBG, lipid profiles, and elucidate how these metabolic parameters influence the periodontal disease severity. By bridging the existing knowledge gap, this research will contribute to better periodontal disease management strategies for diabetic patients in Malaysia.

Materials and Methods

A retrospective record review was conducted from 2015 to 2024 at the Periodontal Clinic, Hospital Pakar Universiti Sains Malaysia (HPUSM), Kubang Kerian, Kelantan. Patients' folders were retrieved from Record Unit, HPUSM. Data were collected from patient records that fulfilled the inclusion and exclusion criteria.

The study population comprised of periodontitis patients with DM in Kelantan. The target population included patients diagnosed with both periodontitis and

T2DM who attended the Periodontal Clinic, HPUSM for treatment. The source population consisted of patients from the target group who fulfilled the inclusion and exclusion criteria, who had undergone treatment at HPUSM between 2015 and 2024.

Sample size was calculated for each objective. The largest sample size was derived from objective 1, to evaluate the periodontal status of periodontitis with T2DM patients, using single proportion formula with parameters set at $Z=1.96$, $\Delta=10\%$, $\text{power}=0.95$ (Singh *et al.*, 2019), $n=38+10\%$ (drop out), yielding the total sample size of 42.

The sampling frame includes patients who met the study's inclusion and exclusion criteria. Patients eligible for the study include those were diagnosed with periodontitis with T2DM who had at least eight natural teeth, and had complete metabolic control status data including HbA1c, FBG, and lipid profiles results within the last six months from their periodontal clinic visit. Pregnant or lactating women, smokers, patients who had chronic liver disease, or were undergoing treatment that could potentially influence the study parameters such as antibiotics, immunosuppressants, antiepileptics, steroids, or non-steroidal anti-inflammatory drugs, were excluded from the study. This study was approved by Human Research Ethics Committee USM

(USM/JEPeM/KK/24040307: Dated 2/6/2024).

Data entry and analysis

All data collection and analysis were conducted using SPSS 29.0. Demographic data was analysed using Descriptive method and Fisher's exact test was applied to evaluate the association between periodontal status and metabolic control indicators in periodontitis with T2DM patients. A *p*-value of less than 0.05 was considered as statistically significant.

Results

A total of 42 periodontitis patients with T2DM were included in this study. Table 1 shows sociodemographic data of the patients. The mean age of participants was 59.2 years (SD = 9.97). Majority of them were male (61.9%), and the predominant ethnic group was Malay (97.6%). Only one participant (2.4%) was Indian and no participant from Chinese or other ethnic groups. Regarding the blood pressure, majority of participants (71.4%) had blood pressure less than 140/90 mmHg, whereas 28.6% had elevated blood pressure, which is more than 140/90 mmHg.

The severity of periodontitis among participants was classified into four stages. None of the patients were classified in stage 1. Most patients (61.9%) were in stage 3, while 23.8% and 14.3% were in stage 2 and stage 4 respectively (Table 2).

Table 1. Sociodemographic data of periodontitis patients with type 2 diabetes mellitus (n = 42).

Sociodemographic data		n (%)
	Age ^a	59.2 (9.97) ^a
Gender	Male	26 (61.9)
	Female	16 (38.1)
Race	Malay	41 (97.6)
	Chinese	0 (0)
	Indian	1 (2.4)
	Others	0 (0)
Blood pressure	<140/90mmHg	30 (71.4)
	>140/90mmHg	12 (28.6)

*a - Mean (SD)

Table 2. The periodontal severity in periodontitis with type 2 diabetes mellitus patients (n = 42).

Periodontal status	n (%)
Stage 1	0 (0)
Stage 2	10 (23.8)
Stage 3	26 (61.9)
Stage 4	6 (14.3)

Periodontal grading revealed that majority of patients (59.5%) were classified as grade B, while 35.7% and 4.8% of patients were in grade A and grade C respectively (Table 3).

Other parameters such as metabolic control indicators, which include HbA1c, fasting blood glucose, and fasting lipid profile, also were assessed (Table 4). Among participants, 64.3% had uncontrolled HbA1c levels ($\geq 7.0\%$), and 83.3% had high FBG levels (>6.0 mmol/L). For fasting lipid profiles, 28.6% of participants had high total

cholesterol (TC ≥ 5.2 mmol/L), 35.7% had elevated triglycerides (TG > 1.7 mmol/L), while 54.8% had high low-density lipoprotein (LDL > 2.6 mmol/L).

The association between periodontal severity and metabolic control indicators was determined and the results were shown in Table 5. No statistically significant association was found between periodontal severity and HbA1c ($p = 0.513$), FBG ($p = 0.539$), TC ($p = 0.296$), TG ($p = 0.487$), or LDL ($p = 0.230$) levels.

Table 3. The periodontal grading in periodontitis patients with type 2 diabetes mellitus (n = 42).

Periodontal grading	n (%)
Grade A	15 (35.7)
Grade B	25 (59.5)
Grade C	2 (4.8)

Table 4. The metabolic control indicators in periodontitis patients with type 2 diabetes mellitus (n = 42).

Metabolic Control Indicators	n (%)
HbA1c (%)	
• < 7.0 (control)	15 (35.7)
• ≥ 7.0 (uncontrolled)	27 (64.3)
Fasting blood glucose (mmol/L)	
• ≤ 6.0 (control)	7 (16.7)
• > 6.0 (uncontrolled)	35 (83.3)
Fasting lipid profile (mmol/L)	
• TC < 5.2 (control)	30 (71.4)
• ≥ 5.2 (uncontrolled)	12 (28.6)
• TG ≤ 1.7 (control)	27 (64.3)
• > 1.7 (uncontrolled)	15 (35.7)
• LDL ≤ 2.6 (control)	19 (45.2)
• > 2.6 (uncontrolled)	23 (54.8)

*HbA1c – glycated hemoglobin, TC - total cholesterol, TG – triglyceride, LDL- low density lipoprotein

Table 5. Association between periodontal severity and metabolic control indicators in periodontitis patients with type 2 diabetes mellitus (n=42).

Metabolic Control Indicators	Periodontal status		p-value
	Mild-Moderate (Stage 1 and 2) n (%)	Severe (Stage 3 and 4) n (%)	
HbA1c (%)			
• < 7.0 (control)	4 (26.7)	11 (73.3)	0.513
• ≥ 7.0 (uncontrolled)	6 (22.2)	21 (77.8)	
Fasting blood glucose (mmol/L)			
• ≤6.0 (control)	2 (28.6)	5 (71.4)	0.539
• > 6.0 (uncontrolled)	8 (22.9)	27 (77.1)	
Fasting lipid profile (mmol/L)			
• TC <5.2 (control)	6 (20)	24 (80)	0.296
• ≥5.2 (uncontrolled)	4 (33.3)	8 (66.7)	
• TG ≤1.7 (control)	7 (25.9)	20 (74.1)	0.487
• >1.7 (uncontrolled)	3 (20)	12 (80)	
• LDL ≤2.6 (control)	3 (16.7)	16 (83.3)	0.230
• >2.6 (uncontrolled)	7 (30.4)	16 (69.6)	

*Interpretation is based on Fisher's exact test, Significant p -value < 0.05, HbA1c (glycated hemoglobin), TC (total cholesterol), TG (triglyceride), LDL (low density lipoprotein).

Discussion

This study aimed to evaluate the relationship between metabolic control indicators such as HbA1c, fasting blood glucose, and fasting lipid profile as well as the severity of periodontitis in periodontitis with T2DM patients. Both periodontitis and T2DM are chronic conditions with a well-established bi-directional relationship, where diabetes is known to increase the prevalence and severity of periodontitis, while advanced periodontitis is linked to poor glycemic control (Valentim *et al.*, 2022). However, in contrary to this expected association, the findings of this study revealed no significant association, despite most participants demonstrated poor glycemic control and severe periodontitis.

In a cross-sectional study conducted in North India, Preshaw *et al.* (2011) assessed the prevalence of periodontal disease among 427 patients with T2DM. They reported that more than 95% of diabetic patients

exhibited some degree of periodontal destruction. They concluded that diabetes is a major risk factor for periodontitis, the susceptibility to periodontitis is increased by approximately threefold in people with diabetes. There is a clear relationship between degree of hyperglycemia and severity of periodontitis. Furthermore, a study conducted among Japanese individuals found that patients with T2DM were 1.17 times more likely to experience periodontal tissue destruction compared to healthy individuals (Morita *et al.*, (2011)). While periodontal status in non-diabetic individuals typically follows a normal distribution, the presence of diabetes shifts this distribution toward more severe stages of the disease (Battancs *et al.*, 2020). These findings are consistent with existing research indicating that diabetes accelerates periodontal tissue damage and contributes to disease progression. Consequently, it is reasonable to suggest that diabetes may play a role in both the onset and worsening of periodontitis (Battancs *et al.*, 2020). Given the well-established association between

diabetes and periodontitis, this hypothesis appears justified. However, the data from our study do not provide insight into the precise mechanism by which diabetes affects periodontal health, as no significant difference between periodontal severity and HbA1c as well as fasting blood glucose level.

The cross-sectional study conducted in South Jordan by Preshaw *et al.* in 2011 highlighted the well-established bidirectional relationship between diabetes and periodontitis. Diabetes not only elevates the risk and severity of periodontitis but also compromises the body's immune response, accelerating the progression of periodontitis. Conversely, periodontal inflammation can have a negative impact on glycemic control, creating a cyclical effect that further exacerbates both conditions (Preshaw *et al.*, 2011). This is proved by our data. In terms of the grading and progression of periodontitis, our results suggest that individuals with diabetes mellitus may have more progression in the severity of periodontitis as reflected by high percentage of individuals with grade B periodontitis (59.5%).

In our study, 64.3% of participants had uncontrolled HbA1c levels. Among those with controlled HbA1c level, 73.3% were diagnosed with stage 3 or 4 periodontitis, while 77.8% of participants with uncontrolled HbA1c levels fell into severe or advanced stages. However, this difference was not statistically significant ($p = 0.513$), suggesting that glycemic control status did not have a significant correlation with periodontitis severity in this population. Similarly, a study by Lim *et al.* (2007) explored the relationship between metabolic control and periodontitis severity in individuals with diabetes mellitus. Their findings indicated that poor metabolic control reflected by elevated HbA1c level, was associated with more severe periodontitis. Specifically, patients with poorly controlled diabetes exhibited greater clinical attachment loss and deeper probing pocket depths compared to those with better glycemic control. The discrepancy between our findings and those of Lim *et al.* (2007) might be due to differences in study design,

sample size, or population characteristics. Additional factors, such as the duration of diabetes, compliance to the treatment, presence of comorbidities, and variations in oral hygiene practices may also influence the observed relationship between glycemic control and periodontitis severity.

Although our study did not find a statistically significant association between HbA1c level and periodontal severity, the higher prevalence of advanced periodontal disease among participants with uncontrolled diabetes suggests potential clinical relevance. While some studies (Morita *et al.*, 2011; Kim *et al.*, 2013) have demonstrated a positive correlation between poor glycemic control and worsening periodontitis, the findings have not been consistent across all populations. Therefore, further research is necessary to better understand the complex relationship between HbA1c level and periodontal health, taking into account for potential confounding factors and differences in population characteristics.

Moreover, we could not find a significant association between periodontal status and fasting blood glucose level. In the study by Kim *et al.* (2013), the relationship between various diabetes-related factors and periodontal health was examined among individuals with T2DM. The study found that periodontal parameters, including the number of missing teeth and papillary bleeding index, were significantly influenced by fasting blood glucose level. Specifically, higher fasting blood glucose level was associated with worse periodontal health outcomes (Kim *et al.*, 2013).

By contrast, our study observed that 71.4% of participants had controlled fasting blood glucose levels, while 77.1% had uncontrolled fasting blood glucose levels. However, there was no statistically significant association between periodontal severity and fasting blood glucose levels ($p = 0.539$). This discrepancy may be attributed to differences in study design, population characteristics, or other confounding factors that were not accounted in our analysis. In addition to glycemic control, factors such as diet, physical activity, drug susceptibility,

and access to professional medical care also play a crucial role in the progression of periodontitis (Kiedrowicz *et al.*, 2015).

Additionally, a study by Bridges *et al.* (1996) also found no association between glycemic control levels and periodontal disease parameters, including probing depth and clinical attachment loss. Similarly, research conducted in Poland by Krajewski *et al.* (2008) reported no significant differences in periodontal status between individuals with well-controlled diabetes ($HbA1c \leq 7.5\%$) and those with poor glycemic control ($HbA1c > 7.5\%$) (Kiedrowicz *et al.*, 2015).

FBG is an important parameter for diagnosis of T2DM. Two readings of FBG more than 7.0 mmol/L is considered to confirm diagnosis of T2DM in asymptomatic individuals, while one reading of FBG more than 7.0 mmol/L is needed to confirm the diagnosis in symptomatic individuals (Clinical Practice Guideline Diabetes Mellitus, 2020). Understanding the potential mechanisms underlying the relationship between fasting blood glucose levels and periodontal health is crucial. Elevated fasting blood glucose levels can lead to the formation of advanced glycation end-products (AGEs), which initiate and propagate inflammatory responses, resulting in the degradation of periodontal tissues. Additionally, a hyperglycemic environment can impair the function of fibroblast, subsequently leading to increase susceptibility of collagen to degradation as well as hindering tissue repair and regeneration (Kim *et al.*, 2013). These pathophysiological processes may explain the association between poor glycemic control and periodontal disease progression.

Fasting lipid profile, a key metabolic parameter that may play a role in periodontal disease progression, was evaluated among the study groups. However, there was no statistically significant association between periodontal severity and fasting lipid profile, including total cholesterol ($p = 0.296$), triglycerides ($p = 0.487$), or LDL cholesterol ($p = 0.230$). Nevertheless, other studies (Lim *et al.*, 2007; Mirzaei *et al.*, 2022; Reddy *et al.*, 2022)

reported there was a relationship between lipid metabolism and periodontal disease, suggesting that lipid profiles may be altered in individuals with metabolic disorders. However, their direct impact on periodontal severity remains uncertain.

Lim *et al.* (2007) explored the effect of hyperlipidemia on periodontal inflammation and reported minimal influence on gingival inflammation severity. Their study suggested that the widespread use of statins among participants could have mitigated the expected effects of hyperlipidemia on periodontal outcomes. Additionally, they also observed that individuals with poor glycemic control ($HbA1c > 8\%$) had higher LDL cholesterol and triglyceride levels compared to those with better metabolic control. This finding reinforcing the relationship between diabetes, lipid metabolism, and inflammatory responses. They further proposed that elevated cholesterol levels could contribute to periodontal inflammation by triggering bacterial endotoxin-induced cytokine release, which alters lipid metabolism by increasing LDL cholesterol and reducing HDL cholesterol level.

In contrast to Lim *et al.*'s findings, our study did not establish a statistically significant correlation between lipid profiles and periodontal severity. This discrepancy may be attributed to differences in the study population, the use of lipid-lowering medications, or variations in glycemic control. Despite the lack of statistical significance, the trend of higher lipid levels in the uncontrolled metabolic group suggests that metabolic dysregulation may still influence the periodontitis progression. Additionally, a systematic review and meta-analysis conducted by Mirzaei *et al.* (2022) further supports the link between dyslipidemia and periodontal disease. Their study found that periodontitis increased the odds of dyslipidemia by 15.0% and the likelihood of low HDL by 32.0%. Furthermore, a significant positive association was identified between periodontitis indices and mean triglycerides, LDL, and total cholesterol levels, while HDL exhibited a protective effect against

periodontitis. These findings highlight the complex interplay between lipid metabolism and periodontal health.

One of the limitations of this study was the sample size, which was restricted to a specific geographic area, limiting the generalizability of the findings. Additionally, the retrospective nature of the record review posed a challenge, as only a limited number of patients met all the necessary inclusion criteria. Since our study did not find a significant statistical correlation, larger-scale studies may provide further insight into the potential impact of dyslipidemia on periodontal disease. Further research with controlled variables, such as statin use, might help to clarify this relationship. Other limitation was the lack of clarity regarding the smoking history of participants due to missing data. Similarly, incomplete weight records prevented the assessment of body mass index, an important parameter for its association with obesity and increased risk of insulin resistance. Future studies should consider incorporating these parameters as potential risk factors for diabetes mellitus.

Conclusion

Metabolic control indicators such as HbA1c, fasting blood glucose, and lipid profiles showed no significant correlation with periodontal severity in individuals with T2DM. In future study, there is a need for comprehensive approach to take into account additional factors such as systemic health conditions, oral hygiene practices, and lifestyle behaviors in managing periodontal health among diabetic patients. Emphasizing glycemic control, routine periodontal care, and patient education on oral health may play a crucial role in preventing diabetes-related complications and improving overall quality of life of the patients.

Future research should be conducted on a larger sample size with a diverse study design to establish a temporal relationship between periodontal health and T2DM. It is crucial to investigate why some individuals

with poor glycemic control do not develop periodontitis-related complications, while others with well-controlled diabetes exhibit both micro- and macrovascular complications along with more severe periodontitis.

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