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ABOUT COVER

Editorial Board Member of *World Journal of Clinical Cases*, Moises Rodriguez-Gonzalez, MD, Adjunct Professor, Senior Researcher, Department of Pediatric Cardiology, Hospital Universitario Puerta del Mar, Cadiz 11009, Spain. doctormoisesrodriguez@gmail.com

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ORIGINAL ARTICLE

Observational Study

Correlation of periodontal inflamed surface area with glycemic status in controlled and uncontrolled type 2 diabetes mellitus

Krishna Anil, Rosamma Joseph Vadakkekuttical, Chandni Radhakrishnan, Fairoz Cheriyalingal Parambath

ORCID number: Krishna Anil 0000-0001-7743-215X; Rosamma Joseph Vadakkekuttical 0000-0002-1324-8385; Chandni Radhakrishnan 0000-0002-0732-0651; Fairoz Cheriyalingal Parambath 0000-0002-4040-2683.

Author contributions: Vadakkekuttical RJ has proposed the article's conception; Vadakkekuttical RJ and Anil K has contributed to data analysis; Vadakkekuttical RJ, Radhakrishnan C and Parambath FC has contributed to intellectual content; all authors did the study design, data interpretation and approved the final version to be published.

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Krishna Anil, Rosamma Joseph Vadakkekuttical, Department of Periodontics, Government Dental College (Affiliated to Kerala University of Health Sciences), Calicut 673008, Kerala, India

Chandni Radhakrishnan, Department of Medicine, Government Medical College (Affiliated to Kerala University of Health Sciences), Calicut 673008, Kerala, India

Fairoz Cheriyalingal Parambath, Department of Microbiology, Government Medical College (Affiliated to Kerala University of Health Sciences), Calicut 673008, Kerala, India

Corresponding author: Rosamma Joseph Vadakkekuttical, MDS, Professor, Department of Periodontics, Government Dental College (Affiliated to Kerala University of Health Sciences), Govt. Medical College Campus, Calicut 673008, Kerala, India. drrosammajoseph@gmail.com

Abstract

BACKGROUND

The bidirectional link between periodontitis and diabetes mellitus (DM) has been established. Periodontitis causes systemic inflammatory burden through inflammatory mediators. The currently utilized tools [clinical attachment loss (CAL) and probing pocket depth (PPD)] are linear measurements, that do not exactly quantify the inflammatory burden of periodontitis. Periodontal inflamed surface area (PISA) quantifies the surface area of bleeding pocket epithelium and estimates the inflammatory burden. Studies relating to the periodontal status of diabetic patients with and without microvascular complications are scarce. This study assessed the proportion of periodontitis and correlation of PISA with glycemic status in controlled, uncontrolled type 2 DM (T2DM) with and without microvascular complications.

AIM

To assess the proportion of periodontitis and correlation of PISA with glycemic status in controlled, and uncontrolled T2DM with and without microvascular complications.

METHODS

This study comprised 180 T2DM patients. Based on glycated hemoglobin (HbA1c) levels, they were grouped into: (1) Controlled T2DMgroup: (HbA1c ≤ 7%); (2) Uncontrolled T2DM group: (HbA1c > 7%) without microvascular complications; and (3) Uncontrolled T2DM group: (HbA1c > 7%) with microvascular complic-

additional data are available.

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ations. Each group comprised 60 patients. All patients were assessed for periodontal parameters (Bleeding on Probing, PPD, CAL, Oral hygiene index simplified and PISA), and systemic parameters (HbA1c, fasting plasma glucose and post prandial plasma glucose).

RESULTS
The proportion of periodontitis among controlled T2DM group, uncontrolled T2DM group without microvascular complications, uncontrolled T2DM group with micro-vascular complications was 75%, 93.4% and 96.6% respectively. Extent and severity of periodontitis were high in the uncontrolled T2DM group. A significant positive correlation was found between PISA and HbA1c among all patients (r = 0.393, P < 0.001). The dose–response relationship between PISA and HbA1c was observed. An increase of PISA with 168 mm^2 was associated with a 1.0% increase of HbA1c.

CONCLUSION
High proportion and severity of periodontitis, and increased inflamed surface area in uncontrolled T2DM may have contributed to the poor glycemic control and microvascular complications.

Key Words: Type 2 diabetes mellitus; Periodontitis; Periodontal inflamed surface area; Glycated Hb; Diabetes

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Core Tip: Poor glycemic control and diabetic complications result in severe periodontal destruction. Periodontitis causes systemic inflammatory burden through inflammatory mediators and affects glycemic control. Periodontal inflamed surface area (PISA) estimates periodontal inflammatory burden. This cross-sectional study assessed the proportion and severity of periodontitis and evaluated the correlation between PISA and glycated hemoglobin (HbA1c) in controlled, and uncontrolled type 2 diabetes mellitus (T2DM) with and without microvascular complications. There was a significant positive correlation between PISA and HbA1c. High proportion and severity of periodontitis, and increased inflamed surface area in uncontrolled T2DM may have contributed to poor glycemic control and microvascular complications.

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INTRODUCTION

Diabetes mellitus (DM) is a chronic disease of metabolic dysregulation characterized by hyperglycemia due to defects in insulin secretion, insulin action, or both. Type 2 DM (T2DM) constitutes about 90% to 95% of all DM cases[1]. It results from insulin resistance rather than from the total absence of insulin production. Periodontitis is an immunoinflammatory disease that affects the supporting tissues of the teeth. It is caused by a complex interplay between specific Gram-negative microorganisms, their byproducts, and the host-tissue response[2]. This results in progressive destruction of the periodontal ligament, alveolar bone and cementum[3]. Although it is initiated and maintained by a specific anaerobic or facultative Gram-negative bacterial infection, the onset and progression of the disease is a result of the inflammatory host response[4,5]. The inflammatory response to the presence of subgingival biofilm is characterized by the local production of various inflammatory mediators such as cytokines, prostanoids and enzymes like matrix metalloproteinases (MMPs). Dysregulated immune response results in an imbalance between the proportions of pro- and anti-inflammatory cytokines and results in periodontal tissue destruction.

The relationship between periodontitis and DM is bidirectional[5-7]. DM increases the risk and severity of periodontitis. Hyperglycemia and advanced glycation end-products (AGEs) affect collagen stability, vascular integrity, and cell functions (leukocytes, fibroblasts, and osteoclasts). AGEs aggregate macrophage and monocyte receptors and stimulate the release of proinflammatory cytokines and alteration in the RANKL/OPG ratio. This provokes an increase in the susceptibility to periodontal diseases. The biological model for the plausibility of periodontitis as a risk factor for diabetes showed that periodontitis causes a systemic inflammatory burden. It results from the entry of periodontopathogens and other coaggregating microorganisms and their virulence factors into the systemic circulation. The increased production of inflammatory cytokines in periodontitis aggravates insulin resistance, thereby affecting glycemic control and diabetic complications[6,7]. The more significant the amount of inflamed periodontal tissue, the greater the chance of periodontitis eliciting bacteremia and inflammatory response. The currently utilized tools [clinical attachment loss (CAL) and probing pocket depth (PPD)] are linear measurements that do not exactly quantify the inflammatory burden of periodontitis. Periodontal inflamed surface area (PISA) quantifies the surface area of the bleeding pocket epithelium and estimates the inflammatory burden. Studies relating to the periodontal status of diabetic patients with and without microvascular complications are scarce. This study assessed the proportion of periodontitis and correlation of PISA with glycemic status in controlled, uncontrolled T2DM with and without microvascular complications.

MATERIALS AND METHODS

This cross-sectional study was conducted by the Department of Periodontics, Government Dental College, Calicut, in collaboration with the Department of Internal Medicine & Department of Microbiology, Government Medical College, Calicut, Kerala, India. The Institutional Ethics Committee Government Dental College Calicut (IEC No. 83/2016/DCC dated 29-11-16) approved this study, and it was registered under the Clinical Trial Registry of India (CTRI/2017/10/010217). Informed consent was obtained from the patients, and the study was conducted following the Helsinki Declaration of 1975, as revised in 2013. The duration of the study was 12 mo. T2DM patients in the age group between 30 and 60 years and with a minimum of 20 teeth were included in this study. Patients with known systemic diseases and conditions, pregnant and lactating mothers, patients with an acute condition that contraindicates a periodontal examination, patients who received systemic antibiotic therapy within the past 6 mo, patients who received periodontal therapy (scaling and root planing or surgery) within the past year were excluded from the study. In this study, 180 T2DM patients were selected from the Diabetic Clinic of the Department of Internal Medicine and divided into three groups (60 in each group) based on their glycated hemoglobin (HbA1c) levels: (1) Group I: controlled T2DM group: (HbA1c ≤ 7%); (2) Group II: uncontrolled T2DM group: (HbA1c > 7%) without microvascular complications; and (3) Group III: uncontrolled T2DM group: (HbA1c > 7%) with microvascular complications.

Patients were evaluated using a detailed questionnaire about their sociodemographic characteristics, medical history, oral hygiene practice, history of DM and drug allergy. HbA1c, fasting plasma glucose (FPG) and postprandial plasma glucose (PPPG) levels were also assessed. The oral and periodontal examination included PPD, CAL, bleeding on probing (BOP), Oral hygiene index-simplified (OHI-S) and periodontal inflamed surface area (PISA). All periodontal examinations were done by a single trained examiner. There were no sources of bias in this study.

The periodontal status was measured by probing pocket depth, gingival recession, and CAL in millimeters at six sites on each tooth using a William's graduated periodontal probe. The periodontal status was then recorded as no/mild periodontitis, moderate periodontitis, and severe periodontitis based on the criteria proposed by the CDC working group for use in population-based surveillance of periodontitis (CDC 2012 update)[8].

PISA was calculated with a Microsoft Excel spreadsheet available on the website: www.parsprototo.info. After filling CAL, gingival recession (GR) and BOP on six sites per tooth in this spreadsheet, mean CAL and GR for each tooth was calculated. Linear mean CAL and GR were translated into the periodontal epithelial surface area (PESA) for each specific tooth. The PESA measured in mm^2 for a particular tooth consists of the root surface area of that tooth, which is covered with pocket epithelium. PISA for a

particular tooth was estimated by multiplying PESA for a specific tooth with the proportion of sites with BOP. The Full-mouth PISA value (mm^2) of each participant was calculated by using the sum of all individual PISAs around the individual tooth.

Statistical analysis

mean ± SD was calculated for quantitative variables, and frequency was calculated for qualitative variables. An independent *t*-test was used to compare the quantitative variables between controlled T2DM and uncontrolled T2DM groups. Quantitative data (age, BOP, PPD, CAL, OHI-S, HbA1c, FPG and PPPG) between groups were analyzed by analysis of variance. The χ^2 test analyzed qualitative data such as gender, socioeconomic status, and the proportion and severity of periodontitis. Correlation between PISA and HbA1c, CAL and HbA1c was done by Pearson correlation test. The multivariate linear regression model was used to analyze the relationship between PISA and HbA1c.

RESULTS

Mean age, gender distribution, and socioeconomic status showed no significant difference among the groups (Table 1). A significant difference was observed among these groups in terms of duration of diabetes, HbA1c, FPG and PPPG. The difference in BOP score, DI-S score, CI-S score, OHI-S score, mean PPD, mean CAL, and PISA showed significant differences among the groups ($P < 0.001$). The uncontrolled T2DM group with microvascular complications attained the highest values for all these parameters (Table 2). Bonferroni *post hoc* adjustment showed no significant difference between the uncontrolled T2DM without microvascular complications and uncontrolled type 2 DM with microvascular complications groups for mean PPD, CAL and PISA ($P = 1.00$) (Table 3).

The proportion of periodontitis among the study patients was 88.6%. The proportion of periodontitis among the controlled T2DM group, uncontrolled T2DM group without microvascular complications, and uncontrolled T2DM group with microvascular complications was 75%, 93.4%, and 96.6%, respectively. The difference in the proportion of periodontitis among these groups was significant ($P < 0.001$) (Table 2).

There was a significant difference in the severity of periodontal diseases among these groups ($P < 0.001$). The proportion of severe periodontitis among Group I, II and III was 30%, 76.7% and 73.3%, respectively (Figure 1). The uncontrolled T2DM group with microvascular complications showed the highest percentage of sites with CAL ≥ 6 mm. The highest percentage of sites with CAL ≤ 3 mm was observed in the controlled T2DM group. Percentage of sites with CAL ≤ 3 mm, 4–5 mm and ≥ 6 mm showed a significant difference among these groups ($P < 0.001$) (Table 4).

The multivariate linear regression model with the dependent variable PISA showed that age, duration of diabetes and HbA1C were significantly associated with PISA. A dose–response relationship between PISA and HbA1c was observed. An increase of PISA with 168 mm^2 was associated with a 1.0% increase of HbA1c (Table 5). A significant positive correlation existed between the mean CAL and HbA1c in all patients ($r = 0.451$, $P < 0.001$) (Figure 2). A positive correlation was observed between PISA and HbA1c in all patients ($r = 0.393$, $P < 0.001$) (Figure 3).

DISCUSSION

A high proportion of periodontitis (88.6%) was observed in T2DM patients. The proportion of periodontitis among the uncontrolled T2DM group without microvascular complications and the uncontrolled T2DM group with microvascular complications was high compared to the controlled T2DM group. Several studies[9-13] have reported a higher prevalence of periodontitis in a poorly controlled group than in controlled T2DM. In this study, the mean overall OHI-S score of the controlled T2DM group came under the fair category, whereas the uncontrolled T2DM group with and without microvascular complications came under the poor category. Metabolic control of diabetes may be an important variable in the progression and aggravation of periodontal diseases. Conflicting reports are also available in the literature regarding glycemic control of diabetes and periodontitis. Sandberg *et al*[14]and Chuang *et al*[15] have opined that there is no relationship between metabolic control of diabetes and periodontitis.

Table 1 Distribution of sociodemographic characteristics of patients

Sociodemographic characteristics		Group I	Group II	Group III	*P* value
Age, yr (mean ± SD)		48.83 ± 7.01	49.75 ± 5.99	50.87 ± 5.98	0.22
Gender (%)	Male	23 (38.33)	21 (35.00)	13 (21.67)	0.12
	Female	37 (61.67)	39 (65.00)	47 (78.33)	
Socio-economic Status (%)	APL	18 (30.00)	25 (41.67)	13 (21.67)	0.06
	BPL	42 (70.00)	35 (58.33)	47 (78.33)	

mean ± SD calculated for age for group I, group II and group III. APL: Above poverty line; BPL: Below poverty line.

Table 2 Comparison of glycemic status, oral hygiene status (Debris index–simplified, Calculus index–simplified, Oral hygiene index–simplified), bleeding on probing (% of site), probing pocket depth, clinical attachment loss, periodontal inflamed surface area and percentage of periodontitis among groups

Variables	Group I	Group II	Group III	*P* value[a]
Duration of DM	8.95 ± 6.86	9.49 ± 6.873	13.67 ± 5.70	< 0.001
HbA1c	6.73 ± 0.25	8.87 ± 1.203	9.40 ± 1.54	< 0.001
FPG	117.25 ± 26.05	170.12 ± 57.86	173.67 ± 66.11	< 0.001
PPPG	176.55 ± 59.63	234.62 ± 79.34	242.17 ± 73.16	< 0.001
BOP (% of site)	55.31 ± 26.22	75.85 ± 22.72	79.14 ± 19.47	< 0.001
DI-S	1.08 ± 0.73	1.55 ± 0.78	1.6 ± 0.66	< 0.001
CI-S	1.31 ± 0.67	1.84 ± 0.71	1.94 ± 0.65	< 0.001
OHI-S	2.37 ± 1.35	3.39 ± 1.41	3.5 ± 1.14	< 0.001
PPD	2.59 ± 0.67	3.36 ± 0.77	3.43 ± 0.75	< 0.001
CAL	2.88 ± 0.77	3.81 ± 0.96	3.96 ± 0.92	< 0.001
PISA	852.22 ± 586.77	1506.5 ± 805.76	1530.05 ± 690.24	< 0.001
Periodontitis (%)	75	93.4	96.6	< 0.001

[a]$P < 0.001$. mean ± SD. A significant difference among three groups for systemic and periodontal variables. DM: Diabetes mellitus; HbA1c: Glycated hemoglobin A1c; FPG: Fasting plasma glucose; PPBS: Postprandial plasma glucose, BOP: Bleeding on probing; DI-S: Debris index–simplified; CI-S: Calculus index–simplified; OHI-S: Oral hygiene index–simplified; PPD: Probing pocket depth; CAL: Clinical attachment loss; PISA: Periodontal inflamed surface area.

In this study, a significant difference was obtained in mean PPD and mean CAL between these groups. The uncontrolled T2DM group with microvascular complications had the highest score. Hyperglycemia may have contributed to the increased PPD and CAL in this study. The PPD and CAL obtained in this study are in accordance with the studies of Taylor *et al*[11], Campus *et al*[13] and Tervonen *et al*[16]. In contrast to this, Bridges *et al*[17]in 1996 did not find any association between glycemic control of diabetes and periodontal parameters.

A significant difference was observed in the percentage of sites with CAL ≤ 3, CAL 4-5 mm and CAL ≥ 6 mm among these groups. The highest percentage of sites with CAL ≤ 3 mm was observed in Group I. Controlled glycemic status may have contributed to the maintenance of periodontal health in this group. The extent and severity of periodontitis were more in the uncontrolled T2DM group as compared to the controlled group. Uncontrolled T2DM group with microvascular complications showed the highest percentage of sites with CAL ≥ 6 mm. This observation is in accordance with Ternoven *et al*[16], who have reported that the percentage of sites with CAL ≥ 5 mm was significantly higher in poorly controlled T2DM than in a moderately controlled and controlled group. There are several studies that have reported a higher proportion of severe periodontitis in poorly controlled diabetic patients[16,18,19]. Although plaque is the main etiological agent for periodontitis, hyperglycemia and host immune responses to bacterial challenge also play an active

Table 3 Bonferroni *post hoc* analysis of probing pocket depth, clinical attachment loss and periodontal inflamed surface area among the study groups

Dependent variable	Group	Group	Mean difference	SE	Significant	95% confidence interval	
						Upper bound	Lower bound
Mean PPD	I	II	0.76778	0.1330	0.000	1.089	0.4463
		III	0.83550	0.1330	0.000	1.157	0.5140
	II	III	0.06772	0.1330	1.00	0.3892	0.2538
Mean CAL	I	II	0.93488	0.1618	0.000	1.326	0.5438
		III	1.08697	0.1618	0.000	1.478	0.6959
	II	III	0.15208	0.1618	1.00	0.5432	0.2390
Mean PISA	I	II	654.278	127.80	0.000	963.166	345.391
		III	677.836	127.80	0.000	986.723	368.948
	II	III	23.557	127.80	1.00	332.444	285.330

Group I: Well-controlled T2DM; Group II: Uncontrolled T2DM group without microvascular complications; Group III: Uncontrolled T2DM group with microvascular complications. There was no significant difference between uncontrolled T2DM without microvascular complications group and uncontrolled T2DM group with microvascular complications for mean probing pocket depth, clinical attachment loss, and periodontal inflamed surface area (P = 1.00). SE: Standard error; PPD: Probing pocket depth; CAL: Clinical attachment loss; PISA: Periodontal inflamed surface area; T2DM: Type 2 diabetes mellitus.

Table 4 Percentage of sites with clinical attachment loss ≤ 3 mm, 4-5 mm and ≥ 6 mm among well-controlled type 2 diabetes mellitus group, uncontrolled type 2 diabetes mellitus group without microvascular complications and uncontrolled type 2 diabetes mellitus group with microvascular complications

Variable, CAL	Group I (% of site)	Group II (% of site)	Group III (% of site)	*P* value[a]
CAL ≤ 3 mm	75.07	47.64	43.67	< 0.001
CAL 4-5 mm	22.16	37.53	38.43	< 0.001
CAL ≥ 6 mm	2.77	14.83	17.90	< 0.001

[a]P < 0.001. A significant difference in percentage of sites with CAL ≤ 3 mm, 4-5 mm and ≥ 6 mm among well-controlled T2DM group, uncontrolled T2DM group without microvascular complications and uncontrolled T2DM group with microvascular complications. CAL: Clinical attachment loss; T2DM: Type 2 diabetes mellitus.

role in the progression of periodontitis in patients with DM. Since a bidirectional relationship exists between periodontitis and diabetes, the systemic inflammation associated with periodontal diseases may contribute to the worsening glycemic control in patients with DM. Nonsurgical and surgical periodontal therapy improves glycemic status in DM and prediabetes[20,21]. Mammen *et al*[22] 2016 found a reduction in insulin resistance and an improvement in insulin sensitivity in patients with DM with chronic periodontitis after nonsurgical periodontal therapy.

Clinically meaningful description of periodontitis should include the proportion of sites with bleeding on probing along with CAL. The percentage of sites with bleeding on probing showed a significant difference among the study groups. The uncontrolled T2DM group with microvascular complications had a higher percentage of BOP. This is in accordance with the study by Emrich *et al*[9] in 1991 and Campus *et al*[13] in 2005. Ervasti *et al*[23] in 1985 found that patients with poorly controlled diabetes had higher gingival bleeding scores than those with good or moderate glycemic control but failed to find any correlation between diabetic complications and gingival bleeding. Hyperglycemia either directly or through AGE formation causes functional and structural modifications of cells. This will affect tissue hemostasis leading to a reduction in host resistance, which is reflected in gingiva as increased bleeding even with mild provocation[24].

Periodontitis may cause an inflammatory burden by the production of local inflammatory mediators entering the systemic circulation. A major disadvantage of the studies published on the relationship between periodontitis and systemic disease is the

Table 5 Regression analysis

Model 1	Unstandardized coefficients		Standardized coefficients	*t*	*P* value
	B	SE	β		
Constant	1558.557	1067.486		1.460	0.146
Age, yr	18.249	8.546	0.152	2.135	0.034[a]
Gender	89.509	141.724	0.055	0.632	0.529
Occupation	13.393	25.231	0.042	0.531	0.596
Socio-economic status	101.568	113.792	0.062	0.893	0.373
Smoking	173.619	145.772	0.094	1.191	0.235
Chew tobacco	171.018	272.268	0.044	0.628	0.531
Duration of diabetes	23.708	8.867	0.211	2.674	0.008[a]
Total dose of insulin	0.399	1.942	0.016	0.205	0.838
HbA1C	167.690	35.502	0.355	4.723	0.000[a]

[a]$P < 0.05$. Dependent variable: Periodontal inflamed surface area (PISA). The multivariate linear regression model with dependent variable PISA showed that age, duration of diabetes, and glycated hemoglobin were significantly associated with PISA. B: Unstandardized coefficients; SE: Standard error; β: Standardized coefficients; HbA1c: Glycated hemoglobin A1c.

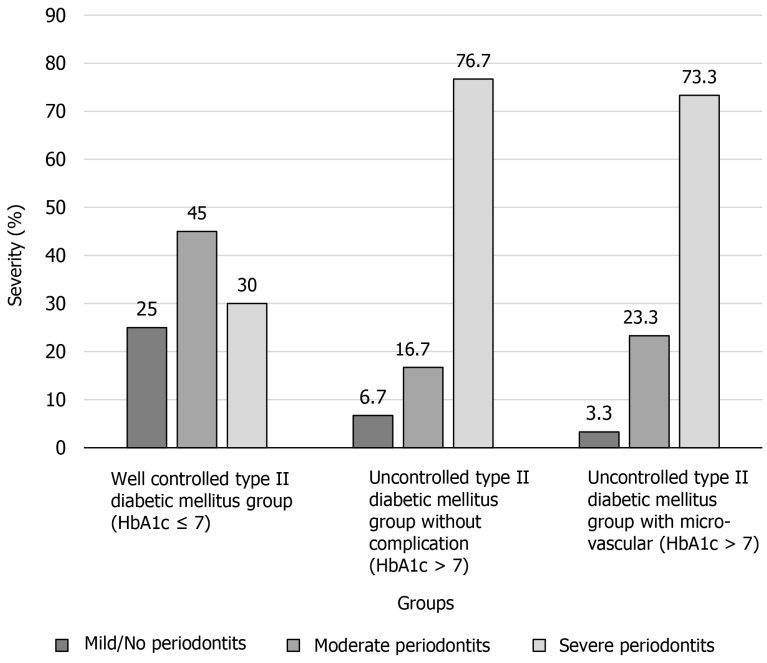

Figure 1 Proportion of periodontal disease severity among groups. HbA1c: Hemoglobin A1c.

lack of a tool that adequately assesses the inflammatory burden of periodontitis. Currently, used tools such as CAL and PPD for grading periodontitis are linear measurements that do not quantify the inflammatory burden caused by periodontitis. So, in this study, PISA was used to assess the inflammatory burden of periodontitis. PISA reflects the surface area of the bleeding pocket epithelium. In this study, the uncontrolled T2DM group with and without microvascular complications had a higher percentage of severe periodontitis and a higher mean PISA than the controlled T2DM group. This is in accordance with the study by Leira *et al*[25] in 2017, which reported a higher PISA in severe periodontitis. From this study, it is evident that a greater estimate of PISA observed in this study could have contributed to the inflammatory link between periodontitis and diabetes. In this study, a positive correlation was obtained between PISA and HbA1c. A dose–response relationship between PISA and HbA1c was observed, and an increase of PISA with 168 mm^2 was associated with

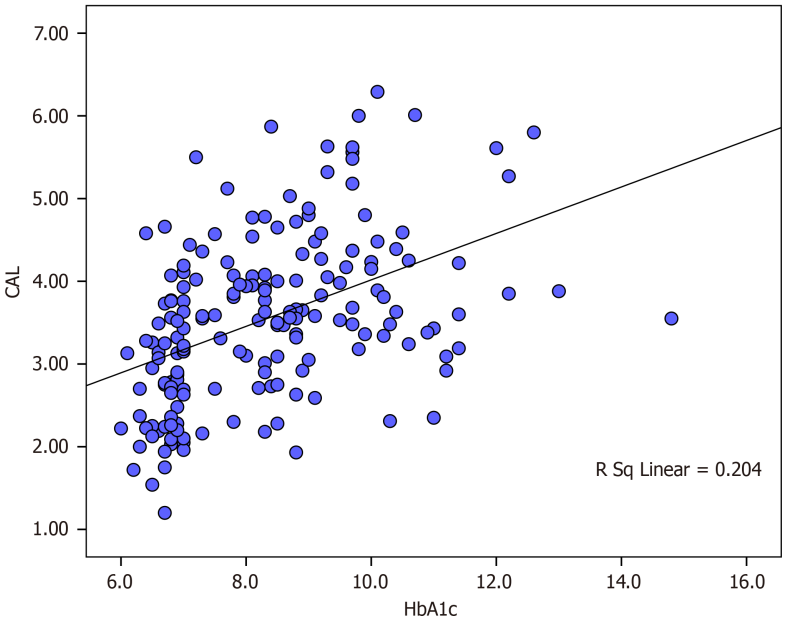

Figure 2 Correlation between clinical attachment loss and glycated HbA1c in well-controlled type 2 diabetes mellitus group, uncontrolled type 2 diabetes mellitus group without microvascular complications and uncontrolled type 2 diabetes mellitus group with microvascular complications. A significant positive correlation existed between the mean clinical attachment loss and HbA1c (Pearson correlation coefficient: 0.451, $P < 0.001$). CAL: Clinical attachment loss; HbA1c: Hemoglobin A1c.

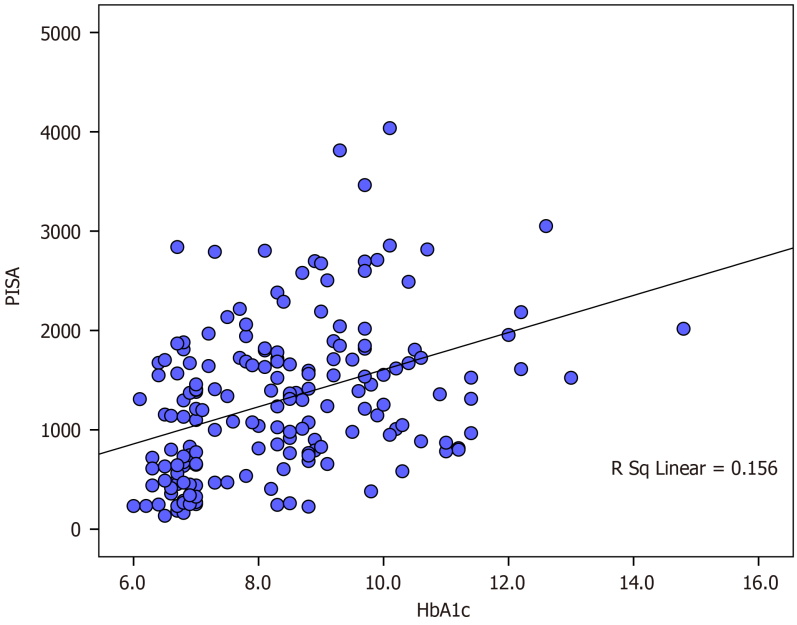

Figure 3 Correlation of periodontal inflamed surface area and HbA1c in well-controlled type 2 diabetes mellitus group, uncontrolled type 2 diabetes mellitus group without microvascular complications and uncontrolled type 2 diabetes mellitus group with microvascular complications. A significant positive correlation existed between the periodontal inflamed surface area and HbA1c (Pearson correlation coefficient: 0.393, $P < 0.001$). PISA: Periodontal inflamed surface area; HbA1c: Hemoglobin A1c.

a 1% increase of HbA1c. Previously, a dose–response association between PISA and HbA1c levels was reported by Nesse *et al*[26] in 2009. They reported that a 333 mm^2 increase of PISA was associated with a 1% increase of HbA1c.

One of the limitations of this study was its small sample size. Moreover, inflammatory markers like interleukin (IL)-1, IL-6, tumor necrosis factor-α, C-reactive protein, MMPs and adipokines were not assessed in this study. Even though our patients had a fair–good OHI-S score, significant positive correlation was found between PISA, CAL and HbA1c. Metabolic control of diabetes may be an important variable in the progression and aggravation of periodontal diseases. Since a bidirec-

tional relationship between diabetes and periodontal disease is well established, high proportion and severity of periodontitis, and increased inflamed surface area in uncontrolled T2DM patients may have contributed to the poor glycemic control and microvascular complications. As it was a cross-sectional study, it is impossible to confirm the direction of the relationship between periodontitis and T2DM with and without microvascular complications. It is necessary to conduct longitudinal studies to show the direction of the relationship between periodontitis and T2DM.

CONCLUSION

The high proportion and severity of periodontitis and increased inflamed surface area in uncontrolled T2DM patients may have contributed to poor glycemic control and microvascular complications. Since a bidirectional relationship exists between periodontitis and diabetes, the periodontal examination is mandatory for patients with diabetes. Proper periodontal therapy can help improve glycemic control and prevent microvascular complications associated with diabetes.

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ARTICLE HIGHLIGHTS

Research background

The bidirectional link between periodontitis and diabetes mellitus (DM) is well established. Periodontitis causes systemic inflammatory burden through inflammatory mediators. The currently utilized tools [clinical attachment loss (CAL) and probing pocket depth (PPD)] are linear measurements that do not exactly quantify the inflammatory burden of periodontitis. Periodontal inflamed surface area (PISA) quantifies the surface area of bleeding pocket epithelium and estimates the inflammatory burden.

Research motivation

Studies relating to the periodontal status of patients with diabetes with and without microvascular complications are scarce. This study assessed the proportion of periodontitis and correlation of PISA with glycemic status in controlled, uncontrolled type 2 DM (T2DM) with and without microvascular complications.

Research objectives

Firstly, to assess the prevalence and severity of periodontitis in T2DM patients (well-controlled T2DM group: [glycated hemoglobin (HbA1c) levels ≤ 7%], uncontrolled type T2DM group: (HbA1c > 7%) without microvascular complications, uncontrolled T2DM group: (HbA1c > 7%) with microvascular complications. Secondly, to assess the correlation between CAL and HbA1c. Finally, to assess the correlation between PISA and HbA1c.

Research methods

This cross-sectional study was conducted by the Department of Periodontics, Government Dental College Calicut, in collaboration with the Department of Internal Medicine & Department of Microbiology, Government Medical College, Calicut, Kerala, India. The duration of the study was 12 mo. In this study, 180 T2DM patients were selected from the Diabetic Clinic of the Department of Internal Medicine and divided into three groups based on their HbA1c as follows: (1) Group I: controlled T2DM group: (HbA1c ≤ 7%); (2) Group II: uncontrolled T2DM group: (HbA1c > 7%) without microvascular complications; and (3) Group III: uncontrolled T2DM group: (HbA1c > 7%) with microvascular complications. Patients were evaluated using a detailed questionnaire about their sociodemographic characteristics, medical history, oral hygiene practice, history of diabetes and drug allergy. HbA1c, fasting plasma glucose and postprandial plasma glucose, PPD, CAL, bleeding on probing, oral

hygiene index-simplified and PISA were assessed.

Research results

The proportion of periodontitis among the controlled T2DM group, uncontrolled T2DM group without microvascular complications, uncontrolled T2DM group with microvascular complications was 75%, 93.4% and 96.6%, respectively. The extent and severity of periodontitis were high in the uncontrolled T2DM group. A significant positive correlation was found between PISA and HbA1c among all patients (r = 0.393, P < 0.001).

Research conclusions

The high proportion and severity of periodontitis and increased inflamed surface area in uncontrolled T2DM patients may have contributed to poor glycemic control and microvascular complications.

Research perspectives

Since a bidirectional relationship exists between periodontitis and diabetes, the periodontal examination is mandatory for patients with diabetes. Proper periodontal therapy can help improve glycemic control and prevent microvascular complications associated with diabetes to some extent.

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