International Journal of Research in Medical Sciences
Sil S et al. Int J Res Med Sci. 2026 Aug;14(8):3459-3464
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20262636

Original Research Article

Periodontitis in diabetic patients: prevalence, glycemic associations and renal implications – a cross-sectional study

Shiladitya Sil[1]*, Saswati Mukherjee[2], Rupa Ghosh[3], Chandreyee Ghosh[4], Soumyajit Dutta[5], Chhanda Biswas[6]

[1]Department of Oral Medicine and Radiology, North Bengal Dental College and Hospital, Thiknikata, West Bengal, India
[2]Department of Periodontics, North Bengal Dental College and Hospital, Thiknikata, West Bengal, India
[3]Department of Orthodontics, Dr. R. Ahmed Dental College and Hospital, Sealdah, Kolkata, West Bengal, India
[4]North Bengal Medical College and Hospital, Thiknikata, West Bengal, India
[5]Department of Dentistry, Ranigunj Institute of Medical Sciences, Raniganj, West Bengal, India
[6]Department of Dentistry, North Bengal Medical College and Hospital, Thiknikata, West Bengal, India

Received: 06 February 2026
Revised: 06 July 2026
Accepted: 27 July 2026

***Correspondence:**
Dr. Shiladitya Sil,
E-mail: shiladitya.sil@gmail.com

ABSTRACT

Background: Diabetes mellitus is associated with multiple systemic complications, including periodontal disease. The bidirectional relationship between diabetes and periodontitis is well established, and emerging evidence suggests periodontal disease may contribute to diabetic nephropathy progression through shared inflammatory mechanisms. This study evaluated the prevalence and severity of periodontitis in diabetic patients and assessed its associations with glycemic control and renal function.
Methods: This observational, cross-sectional study enrolled 203 diabetic patients at IPGMER and SSKM Hospital, Kolkata, India (February 2014–August 2015). Periodontal status was assessed using the community periodontal index (CPI) and classified as no, low-grade, or high-grade periodontitis. Clinical parameters including HbA1c, fasting and postprandial plasma glucose, urine albumin-creatinine ratio (ACR), and estimated glomerular filtration rate (eGFR) were recorded. Statistical analysis used was statistical package for the social sciences (SPSS) ($p<0.05$).
Results: Periodontitis prevalence was 85.2%, with high-grade disease in 52.7% and low-grade in 32.5% of participants. Patients with HbA1c ≥8% had significantly greater periodontal severity ($p<0.0001$). Severe periodontitis was independently associated with higher albuminuria (ACR: 810.94 versus 96.13 mg/g; $p=0.0014$) and reduced eGFR (73.89 versus 93.13 ml/min/1.73 m^2; $p=0.0018$). Diabetes duration, smoking, and BMI were also significantly associated with periodontal severity.
Conclusions: Periodontitis is highly prevalent in diabetic patients and significantly associated with suboptimal glycemic control and renal dysfunction. These findings support integrating periodontal evaluation into routine diabetes management. Prospective studies are needed to determine whether periodontal treatment can mitigate diabetic complications.

Keywords: Diabetes mellitus, Periodontitis, Glycemic control, Albuminuria, Diabetic nephropathy, Community periodontal index

INTRODUCTION

Diabetes mellitus (DM) is a chronic metabolic disorder defined by persistent hyperglycemia arising from defects in insulin secretion, insulin action, or both. Type 2 diabetes mellitus (T2DM) constitutes approximately 90–95% of all cases worldwide and contributes substantially to the global non-communicable disease burden.[1] India bears a disproportionately large share of this burden, rendering diabetes-related complications — including nephropathy, retinopathy, neuropathy, and cardiovascular disease — a major public health concern.[2]

Periodontal disease, a chronic inflammatory condition affecting the tooth-supporting structures, was first characterized by Löe as the "sixth complication" of diabetes mellitus, establishing its clinical importance within the spectrum of diabetic sequelae.[1,3] The relationship between the two conditions is well-recognized as bidirectional: chronic hyperglycemia promotes periodontal tissue destruction, while periodontal infection sustains a systemic pro-inflammatory state that may worsen glycemic regulation.[4,5]

Mechanistically, elevated blood glucose promotes the formation of advanced glycation end-products (AGEs), heightens oxidative stress, and dysregulates innate immune responses, collectively accelerating alveolar bone resorption.[6,7] In the reverse direction, periodontitis drives the systemic release of tumor necrosis factor-alpha (TNF-α), interleukin-1β (IL-1β), and interleukin-6 (IL-6), which can exacerbate insulin resistance and impair glycemic control.[8,9]

Beyond glycemic consequences, emerging evidence suggests that periodontal inflammation may contribute to the development and progression of diabetic nephropathy. Chronic bacteremia and cytokine release from periodontal infection may compromise glomerular endothelial integrity, promoting albuminuria and declining renal function.[9,10] Despite growing international data supporting these associations, population-specific evidence from India remains limited.[11,12]

The present study was designed to evaluate the prevalence and severity of periodontitis in a diabetic cohort and to examine its associations with glycemic control and renal function markers.

Objectives

The study was conducted with the following objectives: to determine the prevalence and severity of periodontitis among patients with diabetes mellitus; to evaluate the relationship between glycemic control, as measured by HbA1c, and periodontal severity; and to assess the association between periodontitis and markers of diabetic nephropathy — specifically urinary albumin-creatinine ratio (ACR) and estimated glomerular filtration rate (eGFR).

METHODS

Study design and setting

This observational, hospital-based, cross-sectional study was conducted at the Department of General Medicine, IPGMER and SSKM Hospital, Kolkata, India, between February 2014 and August 2015. Ethical approval was obtained from the Institutional Ethics Committee (Memo No. Inst/IEC/454, dated 11/01/2014), and written informed consent was secured from all participants in accordance with the Declaration of Helsinki.

Study population

A total of 203 consecutive diabetic patients were enrolled from the medicine outpatient department, emergency department, diabetic clinic, and cardio-diabetic clinic. Eligible participants were aged 15–70 years, had a confirmed diagnosis of diabetes mellitus per American Diabetes Association (ADA) criteria, and were receiving insulin and/or oral hypoglycemic agents. Patients were classified as T1DM if diabetes onset occurred before 31 years of age with exclusive insulin management; all others were categorized as T2DM. Demographic and clinical characteristics are summarized in Tables 1 and 2.

Exclusion criteria included pregnancy, obstructive uropathy, use of medications known to cause gingival enlargement (e.g., phenytoin, cyclosporine, calcium channel blockers), non-cigarette tobacco use, systemic immunosuppressive conditions, uncontrolled hypertension (>140/90 mmHg), diabetes duration under two years, and incomplete permanent dentition.

Periodontal assessment

Periodontal status was evaluated using the community periodontal index (CPI) with a WHO-standard ball-tipped probe (Figure 1). Six sextants were examined for bleeding on probing, presence of calculus, and pocket depth. Based on the highest recorded CPI score, participants were classified as having no periodontitis (CPI score 0), low-grade periodontitis (CPI scores 1–2), or high-grade periodontitis (CPI scores 3–4) (Figure 2).

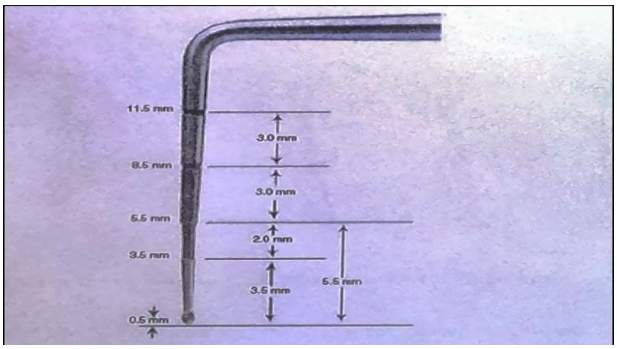

Figure 1: Various marking in the tip of the WHO community periodontal index probe.[13]

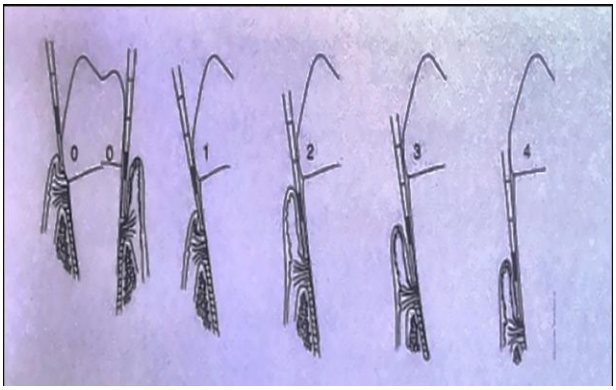

Figure 2: Scores assigned for assessment of the patient's periodontal condition using the WHO community periodontal index probe.[13]

Laboratory investigations

Fasting plasma glucose (FPG), postprandial plasma glucose (PPPG), and glycosylated hemoglobin (HbA1c) — measured by high-performance liquid chromatography (HPLC) — were recorded. Renal function was assessed using serum creatinine, urine ACR from a spot sample, and eGFR calculated by the modification of diet in renal disease (MDRD) formula. Macro-albuminuria was defined as ACR ≥300 mg/g creatinine. BMI was classified as normal (<25 kg/m²) or overweight/obese (≥25 kg/m²) per WHO criteria.

Statistical analysis

Data were analyzed using statistical package for the social sciences (SPSS) version 20.0. Continuous variables are expressed as mean±standard deviation (SD); categorical variables as frequencies and percentages. Between-group comparisons for continuous variables used the independent-samples Student's t-test; associations between categorical variables were assessed using the Chi-square test. A two-tailed p value <0.05 was considered statistically significant.

RESULTS

Demographic and clinical characteristics

Among the 203 participants enrolled, 201 (99.0%) had T2DM and 2 (1.0%) had T1DM. The mean age was 50.57±10.01 years. Males constituted 67.5% (n=137) and females 32.5% (n=66) of the cohort (Table 1). Mean BMI was 21.93±2.69 kg/m², with 18.7% classified as overweight or obese.

Regarding smoking, 63.1% were non-smokers, 25.6% were current smokers, and 11.3% were past smokers. The majority (52.7%) had diabetes duration of 2–5 years; 36.5% had 6–10 years and 10.8% had more than 10 years (Table 1).

Prevalence and severity of periodontitis

The overall prevalence of periodontitis was 85.2% (n=174). High-grade periodontitis (CPI 3–4) was identified in 52.7% (n=107) of participants, while low-grade disease (CPI 1–2) was present in 32.5% (n=66). Only 14.3% (n=29) had no periodontal disease. Periodontitis prevalence and severity increased progressively with age; the highest burden of high-grade disease was observed in the 61–70-year age group, where 86.5% of patients had CPI 3–4 (Table 1 and Figures 3-5).

Table 1: Baseline demographic, clinical and behavioral characteristics of the study population (n=203).

Variable	Category	N (%)
Age (years)	<20	2 (1.0)
	21–40	41 (20.2)
	41–60	123 (60.6)
	61–70	37 (18.2)
Sex	Male	137 (67.5)
	Female	66 (32.5)
Duration of diabetes (years)	2–5	107 (52.7)
	6–10	74 (36.5)
	>10	22 (10.8)
BMI (kg/m²)	<25	165 (81.3)
	≥25	38 (18.7)
Smoking status	Non-smoker	128 (63.1)
	Past smoker	23 (11.3)
	Current smoker	52 (25.6)

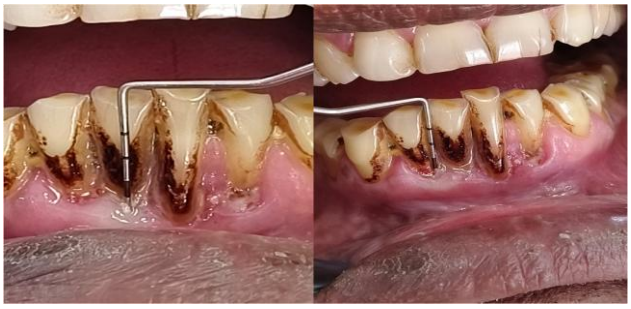

Figure 3: Periodontal pocket depth assessment using the WHO CPI probe in the mandibular anterior teeth.

Figure 4: Periodontal pocket depth assessment using the WHO CPI probe in the mandibular posterior teeth.

Figure 5: Bleeding on probing on walking the WHO CPI probe around the periodontal pocket in the mandibular anterior teeth.

Glycemic control and periodontal severity

A highly significant association was observed between glycemic control and periodontal severity (p<0.0001; Table 2). Among patients with HbA1c <8%, periodontitis was present in 73.0%; in contrast, all patients with HbA1c ≥8% demonstrated periodontal disease. Patients with high-grade periodontitis had significantly higher mean FPG (155.05±59.03 versus 113.97±13.25 mg/dl; p<0.0001), PPPG (229.13±84.94 versus 140.43±18.33 mg/dl; p<0.0001), and HbA1c (7.87±1.46 versus 6.77±0.26%; p<0.0001) compared with those without periodontitis (Table 2).

Duration of diabetes, BMI, and smoking

Patients with periodontitis had a significantly longer mean diabetes duration than those without (7.79±6.00 versus 5.57±2.21 years; p=0.046). Mean BMI was also significantly higher in the periodontitis group (22.27±2.71 versus 19.98±1.55 kg/m²; p<0.0001). Current and past smoking was both significantly more prevalent among patients with periodontitis (Table 3).

Renal parameters and periodontal disease

Patients with periodontitis demonstrated significantly lower mean eGFR (73.89±30.18 versus 93.13±33.46 mL/min/1.73 m²; p=0.0018) and substantially higher mean urine ACR (810.94±1205.15 versus 96.13±87.39 mg/g; p=0.0014) compared with those without periodontal disease (Table 3). These findings indicate a clinically and statistically meaningful association between periodontal disease severity and markers of diabetic nephropathy.

Table 2: Association of periodontitis severity with age, glycemic control and smoking status.

Variable	Category	No PD	Low grade PD	High grade PD	Total	P value*
Age (years)	<20	0	2	0	2	<0.001
	21–40	9	18	14	41	
	41–60	20	42	61	123	
	61–70	0	5	32	37	
HbA1c (%)	<8	30	39	42	111	<0.001
	≥8	0	27	65	92	
Smoking status†	Non-smoker	26	102	—	128	0.010
	Past smoker	0	23	—	23	
	Current smoker	4	48	—	52	

†Smoking data were originally analyzed as periodontitis (PD) versus no periodontitis, not according to low- and high-grade periodontitis separately. Therefore, the smoking section is presented as PD versus no PD, whereas age and HbA1c are presented by periodontitis severity. *Pearson's Chi-square test

Table 3: Comparison of clinical and biochemical parameters between participants with and without periodontitis.

Parameter	PD (mean±SD)	No PD (mean±SD)	P value
Duration of diabetes (years)	7.79±6.00	5.57±2.21	0.046
BMI (kg/m²)	22.27±2.71	19.98±1.55	<0.0001
Fasting plasma glucose (mg/dl)	155.05±59.03	113.97±13.25	<0.0001
Post-prandial plasma glucose (mg/dl)	229.13±84.94	140.43±18.33	<0.0001
HbA1c (%)	7.87±1.46	6.77±0.26	<0.0001
eGFR (ml/min/1.73 m²)	73.89±30.18	93.13±33.46	0.0018
Urine ACR (mg/g)	810.94±1205.15	96.13±87.39	0.0014

DISCUSSION

The present study reports a high prevalence of periodontitis (85.2%) among diabetic patients in a tertiary care hospital in Kolkata, with more than half presenting with high-grade disease. These findings align with established epidemiological evidence. Löe's landmark characterization of periodontal disease as the sixth complication of diabetes established a framework further reinforced by Papapanou's comprehensive epidemiological reviews and Nelson et al's seminal work in Pima Indians.[1,5,11] Indian-specific data from Karoli et al

and Apoorva et al similarly document high periodontal disease burden in diabetic populations attending tertiary care centers, consistent with the present cohort.[14,15]

The dose-response relationship between HbA1c and periodontal severity is a central finding. As comprehensively reviewed by Preshaw et al and Lalla et al and Papapanou, AGE accumulation, oxidative stress, and dysregulated cytokine responses collectively create a pro-inflammatory milieu that amplifies the tissue-destructive response to periodontal pathogens.[5-7] The uniform presence of periodontitis in all patients with HbA1c ≥8% underscores the critical role of glycemic optimization in preventing oral complications.[16,17]

Duration of diabetes was independently associated with greater periodontal severity, consistent with findings by Taylor and Chapple et al Prolonged hyperglycemic exposure results in cumulative microvascular damage, impaired neutrophil function, and altered collagen metabolism — all facilitating disease progression.[4,8] Elevated BMI and smoking further compounded periodontal risk in our cohort. Obesity sustains a low-grade systemic inflammatory state characterized by elevated TNF-α and IL-6, as modeled by Genco et al and documented epidemiologically by Pischon et al and Chaffee.[2,3,18]

The association between periodontitis and renal dysfunction markers is arguably the most clinically significant finding of this study. Patients with periodontitis exhibited markedly higher urine ACR and lower eGFR, indicating a relationship between periodontal inflammation and diabetic nephropathy.[19] This mirrors the prospective work of Saremi et al who demonstrated that severe periodontitis was independently associated with cardio-renal mortality in Pima Indians, and Shultis et al, who reported progressive increases in macroalbuminuria and end-stage renal disease incidence with worsening periodontitis severity.[9,10] The mechanistic rationale is biologically plausible: periodontal gram-negative bacteria introduce lipopolysaccharide (LPS) and inflammatory cytokines into systemic circulation, potentially contributing to glomerular endothelial dysfunction, podocyte injury, and tubulointerstitial inflammation.[20]

These findings carry important clinical implications. Our data support the incorporation of structured periodontal assessment into routine diabetes management, particularly for patients with suboptimal glycemic control or early renal involvement. Intervention data from Preshaw et al and Llambés et al suggest modest HbA1c improvements following periodontal treatment, though larger randomized controlled trials are needed to confirm clinical benefit and to establish whether periodontal therapy attenuates nephropathic progression.[6,21]

Several limitations warrant acknowledgment. The cross-sectional design precludes causal inference. The hospital-based sample may overestimate disease prevalence. The CPI tool does not capture clinical attachment loss and may underestimate severity in adults. The study predates the 2017 World Workshop periodontal classification. Residual confounding from dietary habits, oral hygiene practices, and anti-diabetic drug classes cannot be excluded.

CONCLUSION

Periodontitis is highly prevalent (85.2%) among diabetic patients and is significantly associated with suboptimal glycemic control, longer diabetes duration, higher BMI, and smoking. Importantly, patients with periodontitis exhibited significantly higher urine ACR and lower eGFR, suggesting a clinically meaningful relationship between periodontal inflammation and diabetic nephropathy in this Indian tertiary care cohort.

These findings reinforce the case for incorporating periodontal evaluation into comprehensive diabetic care. Prospective and interventional studies are required to establish causal pathways and to determine whether periodontal therapy can meaningfully reduce the risk of systemic diabetic complications.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Löe H. Periodontal disease: the sixth complication of diabetes mellitus. Diabetes Care. 1993;16(1):329-34.
2. Pischon N, Heng N, Bernimoulin JP, Kleber BM, Willich SN, Pischon T. Obesity, inflammation, and periodontal disease. J Dent Res. 2007;86(5):400-9.
3. Chaffee BW, Weston SJ. Association between chronic periodontal disease and obesity: a systematic review and meta-analysis. J Periodontol. 2010;81(12):1708-24.
4. Taylor GW. Bidirectional interrelationships between diabetes and periodontal diseases: an epidemiologic perspective. Ann Periodontol. 2001;6(1):99-112.
5. Papapanou PN. Epidemiology of periodontal diseases: an update. Ann Periodontol. 1996;1(11):1-36.
6. Preshaw PM, Alba AL, Herrera D, Jepsen S, Konstantinidis A, Makrilakis K, et al. Periodontitis and diabetes: a two-way relationship. Diabetologia. 2012;55(1):21-31.
7. Lalla E, Papapanou PN. Diabetes mellitus and periodontitis: a tale of two common interrelated diseases. Nat Rev Endocrinol. 2011;7(12):738-48.
8. Chapple IL, Genco R; Working Group 2 of the Joint EFP/AAP Workshop. Diabetes and periodontal diseases: consensus report. J Periodontol. 2013;84(4 Suppl):S106-12.
9. Saremi A, Nelson RG, Tulloch-Reid M, Hanson RL, Sievers ML, Taylor GW, et al. Periodontal disease and

mortality in type 2 diabetes. Diabetes Care. 2005;28(1):27-32.
10. Shultis WA, Weil EJ, Looker HC, Curtis JM, Shlossman M, Genco RJ, et al. Effect of periodontitis on overt nephropathy and end-stage renal disease in type 2 diabetes. Diabetes Care. 2007;30(2):306-11.
11. Nelson RG, Shlossman M, Budding LM, Pettitt DJ, Saad MF, Genco RJ, et al. Periodontal disease and NIDDM in Pima Indians. Diabetes Care. 1990;13(8):836-40.
12. Campus G, Salem A, Uzzau S, Baldoni E, Tonolo G. Diabetes and periodontal disease: a case-control study. J Periodontal Res. 2005;40(3):239-45.
13. Ainamo J, Barmes D, Beagrie G, Cutress T, Martin J, Sardo-Infirri J. Development of the World Health Organization (WHO) community periodontal index of treatment needs (CPITN). Int Dent J. 1982;32(3):281-91.
14. Karoli R, Fatima J, Chandra A, Singh G. Periodontal status in patients with diabetes mellitus. Indian J Endocrinol Metab. 2012;16(Suppl 2):S384-6.
15. Apoorva SM, Sridhar N, Shetty A. Prevalence of periodontal disease among type 2 diabetes mellitus patients. J Indian Soc Periodontol. 2013;17(4):528-31.
16. Kumar A, Sharma DS, Verma M. Association between periodontal disease and type 2 diabetes mellitus: a cross-sectional study. Int J Med Sci Public Health. 2014;3(6):714-8.
17. Bhardwaj VK. Periodontal disease and diabetes mellitus: a clinical study. J Craniofac Surg. 2012;23(6):e599-602.
18. Joshi DV, Deshpande N, Deshpande A. Relationship between periodontal disease and glycemic control in diabetic patients. Natl J Integr Res Med. 2010;11(2):80-4.
19. Genco RJ, Grossi SG, Ho A, Nishimura F, Murayama Y. A proposed model linking inflammation to obesity, diabetes and periodontal infections. J Periodontol. 2005;76(11 Suppl):2075-84.
20. Preshaw PM, Heasman PA, Stacey F. The effect of periodontal therapy on glycemic control in patients with type 2 diabetes mellitus. J Clin Periodontol. 2004;31(7):585-92.
21. Llambés F, Arias-Herrera S, Caffesse R. Relationship between diabetes and periodontal infection. World J Diabetes. 2015;6(7):927-35.

Cite this article as: Sil S, Mukherjee S, Ghosh R, Ghosh C, Dutta Biswas C. Periodontitis in diabetic patients: prevalence, glycemic associations and renal implications – a cross-sectional study. Int J Res Med Sci 2026;14:3459-64.