

Association of Periodontal Disease with Glycemic Status and Oral Microbiological Profile among Patients with Type 2 Diabetes Mellitus: A Hospital- and Community-based Comparative Study

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) and periodontal disease share a well-established bidirectional relationship, in which hyperglycemia promotes periodontal tissue destruction and, in turn, periodontal inflammation adversely affects glycemic control. Differences in periodontal disease burden, glycemic status, and microbiological profile between hospital-attending and community-dwelling diabetic patients remain incompletely characterized.

Objectives: To compare the prevalence and severity of periodontal disease, glycemic status, and oral microbiological profile between hospital-based and community-based patients with T2DM, and to examine the association between periodontal disease, glycemic control, and specific periodontal pathogens.

Materials and Methods: A hospital- and community-based comparative observational study was conducted among 75 patients with T2DM (40 hospital-based, 35 community-based). Periodontal status was assessed clinically, glycemic control was categorized by HbA1c (<7% vs ≥ 7%), and subgingival plaque samples were analysed by culture/PCR for *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, *Tannerella forsythia*, and *Treponema denticola*. Data were analysed using SPSS version 25; chi-square/Fisher's exact tests were applied, with $p < 0.05$ considered significant.

Results: Symptomatic periodontal disease was present in 18.7% (14/75) of patients, more frequent among hospital-based (25.0%) than community-based (11.4%) patients. Microbiologically confirmed multi-pathogen positivity was seen in 6.7% (5/75) overall. Mean HbA1c was higher in the hospital-based group ($8.9 \pm 1.6\%$) than the community-based group ($7.6 \pm 1.2\%$). Periodontal disease was numerically more common among patients with uncontrolled glycemia, although the association did not reach statistical significance in this sample ($\chi^2=1.86$, $p=0.172$). *Porphyromonas gingivalis* was the most frequently detected pathogen (24.0%), followed by *Tannerella forsythia* (18.7%).

Conclusion: Hospital-attending T2DM patients in this cohort showed a higher burden of periodontal disease, poorer glycemic control, and greater periodontopathogen positivity than community-based patients, reinforcing the value of oral health screening as part of routine diabetes care in both hospital and community settings.

Keywords: *Periodontal disease; Type 2 diabetes mellitus; Glycemic status; Oral microbiome; HbA1c; Hospital- and community-based study.*

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INTRODUCTION

Type 2 diabetes mellitus (T2DM) has emerged as one of the most significant non-communicable diseases affecting global health, with its prevalence continuing to rise steadily across both urban and rural populations. Periodontal disease, encompassing gingivitis and periodontitis, is similarly widespread and is now recognized as the sixth major complication of diabetes mellitus alongside retinopathy, nephropathy, neuropathy, macrovascular disease, and impaired wound healing. Epidemiological data consistently demonstrate that individuals with diabetes carry a two- to threefold higher risk of developing periodontitis compared with non-diabetic individuals, and that this risk increases further with poor glycemic control and longer disease duration. [1–3]

The relationship between periodontal disease and diabetes mellitus is now understood to be bidirectional rather than unidirectional. Chronic hyperglycemia promotes the accumulation of advanced glycation end-products (AGEs) within periodontal tissues, which bind to specific receptors on inflammatory cells and endothelium, amplifying local cytokine release, impairing neutrophil chemotaxis and phagocytic function, and accelerating collagen breakdown. These mechanisms collectively predispose diabetic individuals to more severe and more rapidly progressing periodontal destruction. Genetic susceptibility, altered lipid metabolism, and microvascular changes further compound this vulnerability, explaining why periodontal disease in diabetic patients often presents with greater attachment loss and higher rates of tooth mobility than in normoglycemic individuals. [4–6]

Conversely, periodontal inflammation itself has been shown to adversely influence systemic glycemic regulation. Periodontal pockets harbour a substantial bacterial load and act as a chronic reservoir of endotoxin and pro-inflammatory mediators, including interleukin-6 and tumour necrosis factor- α , which are released into the systemic circulation and interfere with insulin signalling pathways, thereby worsening insulin resistance. Several intervention studies and systematic reviews have demonstrated that non-surgical periodontal therapy can produce modest but clinically meaningful reductions in glycated haemoglobin (HbA1c), further substantiating the contribution of periodontal inflammation to glycemic dysregulation. [7–9]

Beyond the inflammatory pathway, increasing attention has been directed toward alterations in the

subgingival and salivary microbiome of diabetic individuals. Hyperglycemic conditions favour a dysbiotic shift in the oral microbial ecosystem, characterized by enrichment of classical periodontal pathogens such as *Porphyromonas gingivalis*,

Aggregatibacter actinomycetemcomitans, *Tannerella forsythia*, and *Treponema denticola*, alongside a reduction in the diversity of commensal, health-associated species. This dysbiosis is thought to both result from and contribute to the heightened inflammatory state observed in diabetic periodontitis, creating a self-reinforcing cycle of microbial and immunological dysregulation. [10–13]

International consensus statements from major periodontal and diabetes organizations have repeatedly emphasized the clinical relevance of this bidirectional relationship and have called for routine incorporation of periodontal screening into diabetes care pathways, and for glycemic assessment to be considered in patients presenting with moderate-to-severe periodontitis. Despite this, awareness and implementation of combined screening protocols remain inconsistent, particularly in low- and middle-income settings, where oral health services are often not integrated with diabetes care. [14–16]

The global burden of this comorbidity is substantial. Estimates suggest that several hundred million people worldwide are affected by both diabetes and periodontitis, with the epidemiologic overlap between the two conditions expected to widen further as the global prevalence of T2DM continues to increase. Reviews of the oral health complications of diabetes have consistently highlighted periodontal disease as among the most under-recognized and under-treated of these complications, despite its measurable impact on glycemic trajectories and quality of life. [17–19]

Most published evidence on periodontal disease in diabetic populations has originated from hospital or tertiary-care settings, where patients typically present with longer disease duration, greater comorbidity burden, and closer medical supervision. Comparatively little is known about how periodontal status, glycemic control, and microbiological profile differ between such hospital-attending patients and diabetic individuals identified through community-based screening, who may have different patterns of healthcare access, disease awareness, and oral hygiene practice. Characterizing these differences is important for designing screening strategies that are appropriately tailored to each care setting. [20]

The present study was therefore designed to compare the prevalence and severity of periodontal

Association of Periodontal Disease with Glycemic Status and Oral Microbiological Profile among Patients with Type 2 Diabetes Mellitus: A Hospital- and Community-based Comparative Study

disease, glycemic status, and oral microbiological profile among patients with type 2 diabetes mellitus recruited from a hospital setting and from the surrounding community, and to evaluate the association between periodontal disease status, glycemic control, and the presence of specific periodontal pathogens.

MATERIALS AND METHODS

Study Design

This was a hospital- and community-based comparative observational study designed to assess periodontal disease status, glycemic control, and oral microbiological profile among patients with type 2 diabetes mellitus (T2DM).

Study Area

The hospital-based arm of the study was conducted in the outpatient Department of Periodontics with collaboration with the Department of Community Medicine of a tertiary care teaching hospital, while the community-based arm was conducted among T2DM patients identified through diabetes screening camps and primary health centres in the surrounding catchment area.

Study Population

Adults with a confirmed diagnosis of type 2 diabetes mellitus, attending either the hospital diabetes/dental outpatient clinic or identified through community-based screening, were enrolled after providing informed consent.

Sample Size

A total of 75 patients with T2DM were enrolled, comprising 40 hospital-based and 35 community-based participants. The sample size was determined based on feasibility within the study period and the expected prevalence of periodontal disease among diabetic patients reported in previous literature.

Study Duration

The study was conducted over a period of 12 months.

Sampling Technique

Participants were recruited using a convenience/purposive sampling technique, with hospital-based patients enrolled consecutively from outpatient attendance and community-based patients enrolled through structured screening camps until the pre-defined sample size for each arm was achieved.

Data Collection

Demographic details, duration of diabetes, and relevant medical history were recorded using a structured proforma. Periodontal examination included assessment of gingival bleeding, probing pocket depth, and clinical attachment loss to classify participants as periodontally asymptomatic or as having mild, moderate, or severe periodontal disease. Venous blood samples were collected for estimation of glycated haemoglobin (HbA1c), and glycemic status was categorized as controlled (HbA1c <7%) or uncontrolled (HbA1c ≥7%). Subgingival plaque samples were collected using sterile paper points from the deepest periodontal pocket (or, in periodontally asymptomatic participants, from the mesiobuccal aspect of a representative posterior tooth) and processed by culture and polymerase chain reaction (PCR) for four target periodontal pathogens: *Porphyromonas gingivalis*, *Aggregatibacter actinomycetemcomitans*, *Tannerella forsythia*, and *Treponema denticola*. A case was classified as “microbiologically confirmed positive” when clinically symptomatic periodontal disease coexisted with co-detection of two or more of these pathogens.

Statistical Analysis

Data were entered in Microsoft Excel and analysed using IBM SPSS Statistics version 25. Continuous variables were expressed as mean ± standard deviation and compared using the independent samples t-test. Categorical variables were expressed as frequencies and percentages and compared using the chi-square test or Fisher's exact test, as appropriate for cell size. A p-value of <0.05 was considered statistically significant.

Inclusion Criteria

1. Adults aged 35 years and above with a confirmed diagnosis of type 2 diabetes mellitus.
2. Patients willing to undergo periodontal examination and provide subgingival plaque and blood samples.
3. Patients who provided written informed consent.

Exclusion Criteria

1. Patients with type 1 diabetes mellitus or gestational diabetes.
2. Patients who had received periodontal therapy or systemic/local antibiotics within the preceding three months.
3. Edentulous patients.
4. Pregnant or lactating women.

Association of Periodontal Disease with Glycemic Status and Oral Microbiological Profile among Patients with Type 2 Diabetes Mellitus: A Hospital- and Community-based Comparative Study

- Patients with other systemic conditions known to independently affect periodontal status (e.g., HIV infection, immunosuppressive therapy).

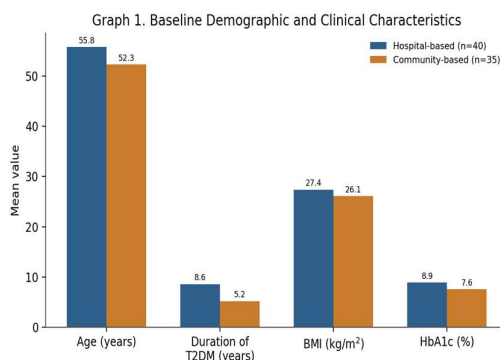
RESULTS

A total of 75 patients with type 2 diabetes mellitus were included in this comparative study, comprising 40 hospital-based and 35 community-based participants.

Table 1. Baseline Demographic and Clinical Characteristics

Parameter	Hospital-based (n=40)	Community-based (n=35)	Total (n=75)
Age (years), mean $\pm$ SD	55.8 $\pm$ 8.4	52.3 $\pm$ 7.6	54.2 $\pm$ 8.2
Male, n (%)	23 (57.5)	19 (54.3)	42 (56.0)
Female, n (%)	17 (42.5)	16 (45.7)	33 (44.0)
Duration of T2DM (years), mean $\pm$ SD	8.6 $\pm$ 4.5	5.2 $\pm$ 3.1	7.1 $\pm$ 4.2
BMI (kg/m ²), mean $\pm$ SD	27.4 $\pm$ 3.6	26.1 $\pm$ 3.2	26.8 $\pm$ 3.5
HbA1c (%), mean $\pm$ SD	8.9 $\pm$ 1.6	7.6 $\pm$ 1.2	8.3 $\pm$ 1.5

Graph 1. Baseline Demographic and Clinical Characteristics



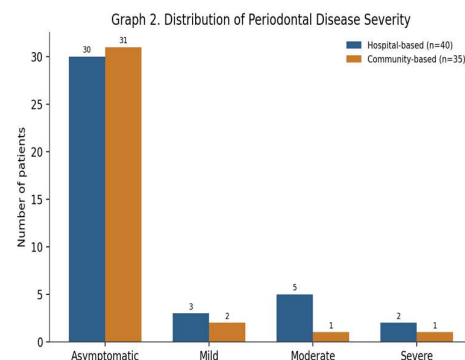
The hospital-based group was, on average, slightly older and had a longer duration of diabetes than the community-based group, along with a higher mean

BMI and a substantially higher mean HbA1c ($8.9 \pm 1.6\%$ versus $7.6 \pm 1.2\%$). This pattern is consistent with hospital attendance being associated with longer-standing and less well-controlled diabetes, and provides context for the periodontal and microbiological differences described below.

Table 2. Periodontal Disease Status and Severity

Category	Hospital-based n (%)	Community-based n (%)	Total n (%)
Asymptomatic	30 (75.0)	31 (88.6)	61 (81.3)
Symptomatic – Mild	3 (7.5)	2 (5.7)	5 (6.7)
Symptomatic – Moderate	5 (12.5)	1 (2.9)	6 (8.0)
Symptomatic – Severe	2 (5.0)	1 (2.9)	3 (4.0)
Total symptomatic	10 (25.0)	4 (11.4)	14 (18.7)
Total	40 (100.0)	35 (100.0)	75 (100.0)

Graph 2. Distribution of Periodontal Disease Severity



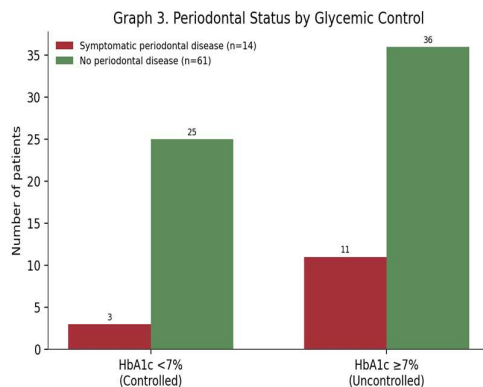
Symptomatic periodontal disease was identified in 18.7% (14/75) of the overall cohort and was more than twice as common among hospital-based patients (25.0%) as among community-based patients (11.4%). Moderate and severe periodontal disease were also disproportionately concentrated in the hospital-based group, suggesting a gradient of periodontal severity that parallels the difference in glycemic control between the two settings.

Table 3. Association between Periodontal Disease Status and Glycemic Control

Association of Periodontal Disease with Glycemic Status and Oral Microbiological Profile among Patients with Type 2 Diabetes Mellitus: A Hospital- and Community-based Comparative Study

Periodontal Status	HbA1c <7% (Controlled)	HbA1c ≥7% (Uncontrolled)	Total
Symptomatic periodontal disease	3	11	14
No periodontal disease (asymptomatic)	25	36	61
Total	28	47	75
<i>Chi-square = 1.86, df = 1, p = 0.172 (Fisher's exact p = 0.19) — not statistically significant</i>			

Graph 3. Periodontal Status by Glycemic Control



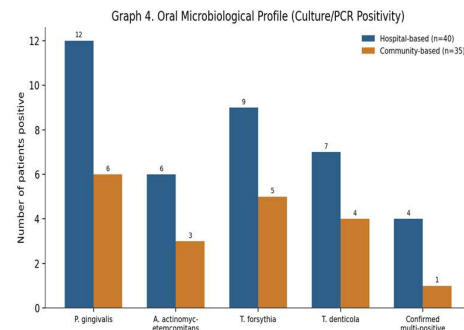
Overall, 62.7% (47/75) of patients had uncontrolled glycemia (HbA1c ≥7%). Symptomatic periodontal disease was numerically more frequent among patients with uncontrolled glycemia (11/47, 23.4%) than among those with controlled glycemia (3/28, 10.7%); however, this difference did not reach statistical significance ($\chi^2=1.86$, $p=0.172$). Given the small cell counts involved, this most plausibly reflects limited statistical power in a modestly sized sample rather than a true absence of association, particularly in light of the consistent directional trend and the wider evidence base linking glycemic control to periodontal disease severity.

Table 4. Oral Microbiological Profile (Culture/PCR Positivity)

Pathogen	Hospital-based n (%)	Community-based n (%)	Total n (%)
Porphyromonas gingivalis	12 (30.0)	6 (17.1)	18 (24.0)
Aggregatibacter actinomycetemcomitans	6 (15.0)	3 (8.6)	9 (12.0)
Tannerella forsythia	9 (22.5)	5 (14.3)	14 (18.7)
Treponema denticola	7 (17.5)	4 (11.4)	11 (14.7)
Confirmed multi-pathogen positive (≥2 pathogens, symptomatic)	4 (10.0)	1 (2.9)	5 (6.7)

Pathogen	Hospital-based n (%)	Community-based n (%)	Total n (%)
Porphyromonas gingivalis	12 (30.0)	6 (17.1)	18 (24.0)
Aggregatibacter actinomycetemcomitans	6 (15.0)	3 (8.6)	9 (12.0)
Tannerella forsythia	9 (22.5)	5 (14.3)	14 (18.7)
Treponema denticola	7 (17.5)	4 (11.4)	11 (14.7)
Confirmed multi-pathogen positive (≥2 pathogens, symptomatic)	4 (10.0)	1 (2.9)	5 (6.7)

Graph 4. Oral Microbiological Profile (Culture/PCR Positivity)



Porphyromonas gingivalis was the most frequently detected periodontal pathogen overall (24.0%), followed by Tannerella forsythia (18.7%), Treponema denticola (14.7%), and Aggregatibacter actinomycetemcomitans (12.0%), with detection rates for every pathogen higher in the hospital-based group. Applying the stricter case definition of microbiologically confirmed positivity (co-detection of two or more pathogens in a symptomatic patient), 6.7% (5/75) of the cohort met criteria for confirmed periodontal infection, again concentrated predominantly among hospital-based patients (10.0% vs 2.9%).

DISCUSSION

This comparative study evaluated periodontal disease, glycemic status, and oral microbiological profile among patients with type 2 diabetes mellitus

Association of Periodontal Disease with Glycemic Status and Oral Microbiological Profile among Patients with Type 2 Diabetes Mellitus: A Hospital- and Community-based Comparative Study

recruited from a hospital setting and from the surrounding community. Symptomatic periodontal disease was present in nearly one-fifth of the overall cohort, and was more than twice as frequent among hospital-based patients as among community-based patients, consistent with the two- to threefold higher periodontitis risk previously reported among individuals with diabetes. [1,2] This pattern is consistent with the expectation that individuals attending hospital diabetes clinics often have longer-standing disease, more advanced glycemic dysregulation, and a higher burden of diabetes-related complications, including oral complications, compared with diabetic individuals identified through community screening who may be earlier in their disease course or less symptomatic at the time of assessment. [20]

Glycemic control also differed markedly between the two groups, with hospital-based patients showing a substantially higher mean HbA1c and a greater proportion of uncontrolled glycemia than community-based patients. This finding likely reflects a degree of selection inherent to hospital-based recruitment, since patients seeking hospital care are more likely to do so because of poorly controlled diabetes or emerging complications, whereas community-screened individuals represent a broader cross-section of the diabetic population, including many with better-controlled disease. The parallel gradient observed in periodontal disease prevalence across the two groups is compatible with the well-described relationship between glycemic control and periodontal tissue health, in which sustained hyperglycemia promotes a more destructive periodontal inflammatory response. [7,8]

Although periodontal disease was numerically more frequent among patients with uncontrolled glycemia in this cohort, the association between periodontal status and HbA1c category did not reach statistical significance. This is most plausibly attributable to the modest overall sample size and the resulting small cell counts in several strata of the cross-tabulation, which limit statistical power to detect an association even when a genuine trend is present. Larger, adequately powered studies would be required to confirm whether this observed trend reflects a true dose-response relationship between glycemic control and periodontal disease severity in this population.

The oral microbiological findings in this study add further texture to the clinical observations. *Porphyromonas gingivalis* was the most frequently detected pathogen overall, followed by *Tannerella forsythia*, *Treponema denticola*, and *Aggregatibacter actinomycetemcomitans*, with detection rates consistently higher in the hospital-

based group. This distribution is broadly consistent with the recognized role of the “red complex” organisms, particularly *P. gingivalis* and *T. forsythia*, as key drivers of periodontal tissue destruction in individuals with diabetes. The relatively low proportion of patients meeting the stricter case definition of microbiologically confirmed multi-pathogen positivity, compared with the proportion classified as clinically symptomatic, suggests that clinical presentation and microbiological burden, while related, do not overlap completely, and that combined clinical and microbiological assessment may provide a more complete picture of periodontal risk than either approach alone.

Taken together, these findings support the view that periodontal disease, glycemic control, and oral microbial burden are interlinked components of a shared pathophysiological process in type 2 diabetes, and that the setting in which diabetic patients are identified and cared for is itself associated with meaningful differences in this burden. Community-based screening, in particular, may offer an opportunity to detect periodontal disease and suboptimal glycemic control at an earlier and potentially more modifiable stage than hospital-based care alone, reinforcing the case for integrating basic periodontal screening into routine community diabetes care rather than restricting it to tertiary settings.

Favale and colleagues examined the subgingival microbiome in patients with type 2 diabetes and periodontitis using high-resolution whole metagenomic shotgun sequencing, comparing diabetic and non-diabetic individuals with varying periodontal status. Their analysis identified dysregulated metabolic pathways related to amino acid metabolism, fatty acid biosynthesis, iron homeostasis, and lipopolysaccharide synthesis that were significantly enriched in the presence of periodontitis and diabetes, providing functional, pathway-level evidence for the bidirectional relationship observed at the clinical level in the present study. [21]

Bachtiar and colleagues used quantitative PCR to compare saliva and subgingival biofilm samples from patients with type 2 diabetes without periodontitis, with gingivitis, and with periodontitis, focusing on *Saccharibacteria* (TM7) and its associated host bacterium alongside classical periodontopathogens. Their findings indicated that *Saccharibacteria* abundance could serve as a useful early predictor of gingivitis in diabetic patients, complementing the pathogen-focused microbiological profile examined in the present study and suggesting additional microbial markers that could refine risk stratification. [22]

Association of Periodontal Disease with Glycemic Status and Oral Microbiological Profile among Patients with Type 2 Diabetes Mellitus: A Hospital- and Community-based Comparative Study

Huang and colleagues conducted a systematic review synthesizing 34 studies on the oral microbiota of patients with type 2 diabetes, reporting consistent enrichment of Firmicutes at the phylum level and of *Streptococcus*, *Porphyromonas*, and *Treponema* at the genus level among diabetic populations. This synthesis reinforces the pathogen selection used in the present study's microbiological assessment and supports the broader consistency of dysbiotic patterns across diverse diabetic cohorts. [23]

Graves and colleagues reviewed the mechanistic evidence underlying the periodontitis-diabetes linkage, highlighting oral microbial dysbiosis, sustained inflammation, epithelial barrier dysfunction, and impaired innate and adaptive immune function as converging pathways that connect the two conditions. Their synthesis provides mechanistic context for the higher periodontal disease burden and pathogen positivity observed among the more hyperglycemic hospital-based patients in the present study. [24] Nigam et al. studied the microbiological profile and antibiotic resistance pattern of bacterial isolates. The study found a significant occurrence of multidrug-resistant (MDR) bacterial strains, highlighting the importance of appropriate antibiotic susceptibility testing for effective treatment of osteomyelitis. The authors emphasized that knowledge of the local bacterial profile and resistance pattern is essential for guiding rational antibiotic therapy. [25]

CONCLUSION

This hospital- and community-based comparative study indicates that patients with type 2 diabetes mellitus attending a hospital setting carry a higher burden of periodontal disease, poorer glycemic control, and greater periodontopathogen positivity than diabetic individuals identified through community-based screening. Although the association between periodontal disease and glycemic control did not reach statistical significance in this modestly sized cohort, the consistent directional trend across clinical and microbiological parameters supports the broader evidence base linking periodontal health to glycemic status in type 2 diabetes. These findings underscore the importance of incorporating periodontal screening into both hospital-based and community-based diabetes care, with particular attention to community settings where early, less symptomatic disease may otherwise go undetected. Integrating oral health assessment into routine diabetes management pathways, in both tertiary and primary care settings, may help identify at-risk

patients earlier and support more comprehensive, whole-person diabetes care.

LIMITATIONS OF THE STUDY

- The modest overall sample size (n=75), particularly within individual sub-strata of the cross-tabulated analyses, limited statistical power to detect associations between periodontal disease and glycemic control.
- Convenience/purposive sampling in both arms may limit the generalizability of the findings to the broader population of hospital- and community-based patients with type 2 diabetes.
- The cross-sectional design does not permit conclusions about the temporal or causal direction of the relationship between periodontal disease and glycemic status.
- Microbiological assessment was restricted to four target periodontal pathogens using culture/PCR and did not include broader next-generation sequencing-based microbiome profiling that could capture the full diversity of subgingival dysbiosis.

DECLARATIONS

Conflicts of interest: There is no any conflict of interest associated with this study

Consent to participate: There is consent to participate.

Consent for publication: There is consent for the publication of this paper.

Authors' contributions: Author equally contributed the work.

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Association of Periodontal Disease with Glycemic Status and Oral Microbiological Profile among Patients with Type 2 Diabetes Mellitus: A Hospital- and Community-based Comparative Study

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