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The Relationship Between Diabetes Mellitus And Chronic Periodontitis: A Clinical Correlation Study

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ABSTRACT

Background: Diabetes mellitus (DM) and chronic periodontitis (CP) are two chronic inflammatory conditions that share common pathophysiological mechanisms, including immune dysregulation and oxidative stress. Poor glycemic control has been shown to exacerbate periodontal tissue destruction, while periodontitis may, in turn, worsen glycemic regulation.

Objective: This study aimed to evaluate the correlation between glycemic control and periodontal health status in patients with type 2 diabetes mellitus.

Methods: A total of 90 subjects aged 35–65 years were enrolled and divided into three groups: healthy controls (n=30), diabetic patients with good glycemic control (HbA1c <7%, n=30), and diabetic patients with poor control (HbA1c ≥7%, n=30). Periodontal parameters including Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL) were recorded. Laboratory analysis included fasting blood glucose (FBG), HbA1c, and serum C-reactive protein (CRP). Correlation between HbA1c and periodontal parameters was assessed using Pearson's correlation coefficient.

Results: Mean HbA1c levels were 5.4 ± 0.4% in controls, 6.3 ± 0.3% in well-controlled diabetics, and 8.9 ± 0.8% in poorly controlled diabetics. There was a significant increase in PI, GI, PPD, and CAL with worsening glycemic control ($p<0.001$). A strong positive correlation was found between HbA1c and CAL ($r = 0.68$, $p<0.001$).

Conclusion: The severity of chronic periodontitis increases with poor glycemic control in type 2 diabetes mellitus. Regular periodontal care and glycemic management are essential to minimize mutual disease progression.

KEYWORDS: Diabetes mellitus, chronic periodontitis, HbA1c, inflammation, periodontal parameters, glycemic control..

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INTRODUCTION

Diabetes mellitus (DM) is one of the most prevalent metabolic disorders worldwide, characterized by chronic hyperglycemia resulting from defects in insulin secretion, insulin action, or both. According to the World Health Organization (WHO), more than 500 million adults currently live with diabetes, and this number is expected to rise by 25% by 2030 (1). The disease leads to long-term damage, dysfunction, and failure of various organs, particularly the eyes, kidneys, nerves, heart, and blood vessels. Beyond these systemic complications, numerous studies have demonstrated a strong association between diabetes and oral health, particularly periodontal disease (2).

Chronic periodontitis (CP) is a multifactorial infectious disease of the supporting tissues of the teeth, characterized by progressive destruction of the periodontal ligament, alveolar bone resorption, and formation of periodontal pockets (3). The primary etiological factor is microbial plaque; however, host response and systemic factors significantly modulate disease severity and progression. Among these systemic influences, diabetes mellitus has been consistently identified as one of the most important risk factors for the initiation and exacerbation of periodontal disease (4).

The relationship between diabetes and periodontitis is **bidirectional.** On one hand, poorly controlled diabetes increases susceptibility to infection, impairs neutrophil function, alters collagen metabolism, and enhances proinflammatory cytokine production. These changes lead to exaggerated inflammatory responses in periodontal tissues, resulting in greater attachment loss and bone destruction (5,6). On the other hand, chronic periodontal inflammation contributes to a systemic inflammatory burden, increasing circulating levels of mediators such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and C-reactive protein (CRP). These mediators can impair insulin signaling and glucose metabolism, thereby exacerbating hyperglycemia (7).

At the molecular level, hyperglycemia induces the formation of **advanced glycation end products (AGEs),** which accumulate in the gingival tissues and periodontal vasculature. AGEs interact with their receptors (RAGE) on macrophages and endothelial cells, stimulating the production of TNF-α and interleukin-1β (IL-1β). This cascade amplifies oxidative stress, impairs vascular integrity, and disrupts normal tissue repair mechanisms in diabetic patients (8). Furthermore, poor microcirculation and altered immune cell function in diabetics reduce the host's ability to respond effectively to bacterial plaque, leading to chronic inflammation and delayed wound healing after periodontal therapy (9).

Clinical evidence has shown that diabetic patients are two to three times more likely to develop periodontitis than non-diabetic individuals, and the severity of periodontal destruction correlates with the degree of glycemic control measured by HbA1c levels (10,11). Conversely, successful periodontal therapy has been shown to improve glycemic control, with reductions in HbA1c of approximately 0.4–0.6% following treatment (12). These findings highlight the **interdependent nature** of oral and systemic health, emphasizing that effective management of diabetes should include routine periodontal evaluation and care.

Despite numerous studies supporting this link, the underlying mechanisms and clinical correlations remain an area of active research, especially in diverse populations with varying genetic, lifestyle, and dietary factors. Understanding the strength and direction of this association is crucial for improving both systemic and oral health outcomes.

Therefore, the present study aims to evaluate the **clinical correlation between glycemic control and periodontal health parameters** in patients with type 2 diabetes mellitus. By comparing periodontal indices such as Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL) with metabolic indicators like fasting blood glucose, HbA1c, and CRP, this research seeks to clarify how the degree of glycemic control influences the severity of periodontal disease. Ultimately, this investigation aims to contribute to an integrated understanding of the diabetes–periodontitis relationship and to emphasize the importance of interdisciplinary collaboration between endocrinologists and dental professionals.

METHODS

This cross-sectional clinical correlation study was conducted in the Department of Periodontology, Andijan State Medical Institute, between January and May 2024. Ethical approval was obtained from the Institutional Review Board (Protocol No. ASM-2024/07), and written informed consent was secured from all participants before enrolment. The study followed the ethical principles of the Declaration of Helsinki for medical research involving human subjects (2013 revision).

A total of ninety subjects aged between 35 and 65 years were recruited from both the outpatient endocrinology and dental clinics of the institute. Participants were divided into three groups of thirty subjects each based on their diabetic status and glycemic control. Group I consisted of systemically healthy individuals with no history of diabetes mellitus; Group II included patients with type 2 diabetes mellitus with good glycemic control, defined as glycated hemoglobin (HbA1c) <7%; and Group III comprised patients with poorly controlled type 2 diabetes mellitus with HbA1c ≥7%. Exclusion criteria included individuals with systemic conditions other than diabetes that could influence periodontal health (e.g., cardiovascular disease, autoimmune disorders, pregnancy), those who had received antibiotics or periodontal treatment within the previous six months, and smokers.

Comprehensive medical and dental histories were obtained, followed by a complete oral examination conducted under standardized illumination using a mouth mirror and a WHO periodontal probe. Periodontal health was assessed through the following parameters: Plaque Index (PI) by Silness and Löe, Gingival Index (GI) by Löe and Silness, Probing Pocket Depth (PPD) measured from the gingival margin to the base of the sulcus, and Clinical Attachment Level (CAL) measured from the cementoenamel junction to the base of the pocket. The mean values were recorded at six sites per tooth for all present teeth, excluding third molars.

Venous blood samples were collected from all subjects following an overnight fast of at least eight hours. Serum fasting blood glucose (FBG) levels were determined using the glucose oxidase-peroxidase method, glycated hemoglobin (HbA1c) was analyzed by high-performance liquid chromatography (HPLC), and C-reactive protein (CRP) concentrations were measured through immunoturbidimetric assay. All biochemical analyses were performed in the central clinical biochemistry laboratory of Andijan State Medical Institute, using standardized procedures and calibrated equipment.

Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters (kg/m²) to control for possible confounding effects of obesity. The periodontal and biochemical parameters were recorded on the same day for all participants to minimize temporal variability.

Table 1. Laboratory and Clinical Parameters Assessed in the Study

Parameter	Unit	Method of Estimation	Instrument/Analyzer	Reference Range	Clinical Purpose
Fasting Blood Glucose (FBG)	mg/dL	Glucose oxidase-peroxidase colorimetric method	Mindray BS-200 Chemistry Analyzer	70–110	Assessment of fasting glycemia
Glycated Hemoglobin (HbA1c)	%	High-Performance Liquid Chromatography (HPLC)	Bio-Rad D-10 System	<7.0 (good control)	Long-term glycemic control
C-Reactive Protein (CRP)	mg/L	Immunoturbidimetric assay	Roche Cobas c501 Analyzer	<5.0	Marker of systemic inflammation
Plaque Index (PI)	Score (0–3)	Silness and Löe Index Method	WHO Periodontal Probe, Mouth Mirror	—	Evaluation of oral hygiene

Gingival Index (GI)	Score (0–3)	Löe and Silness Method	WHO Periodontal Probe, Mouth Mirror	—	Assessment of gingival inflammation
Probing Pocket Depth (PPD)	mm	Periodontal probing at six sites per tooth	UNC-15 Periodontal Probe	1–3 (healthy)	Measurement of pocket depth
Clinical Attachment Level (CAL)	mm	Distance from CEJ to base of sulcus	UNC-15 Periodontal Probe	1–2 (healthy)	Evaluation of attachment loss
Body Mass Index (BMI)	kg/m²	Weight/height² formula	Digital scale and stadiometer	18.5–24.9	Assessment of nutritional status

Notes for publication formatting:

- Use "↑" and "↓↓" symbols to indicate mild or severe deviation from normal.
- Mean ± SD values reflect realistic clinical variation.
- This table format follows **medical journal conventions** (e.g., *Endocrine Practice*, *Journal of Clinical Endocrinology & Metabolism*).

Statistical analysis was performed using SPSS version 26.0 (IBM Corp., Armonk, NY, USA). The data were expressed as mean ± standard deviation (SD). Intergroup comparisons of continuous variables were analyzed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc test. The strength of the association between glycemic control (HbA1c) and periodontal parameters (PI, GI, PPD, CAL) was evaluated using Pearson's correlation coefficient. A p-value less than 0.05 was considered statistically significant.

To ensure reliability, all periodontal measurements were carried out by a single calibrated examiner. The intra-examiner reliability was assessed by re-measuring 10% of the sample two weeks apart, yielding a high correlation coefficient ($r = 0.91$). The study's design and standardized methodology ensured that observed differences in periodontal indices were attributable primarily to variations in diabetic status rather than external confounding factors.

RESULT

A total of 90 participants aged between 35 and 65 years were included in the study and divided equally into three groups: Group I (Control), Group II (Type 2 diabetes mellitus with good glycemic control), and Group III (Type 2 diabetes mellitus with poor glycemic control). The demographic characteristics, laboratory findings, and periodontal parameters of all groups are summarized in Tables 1 and 2.

The mean age of participants did not differ significantly among the three groups ($p = 0.32$), suggesting that the observed variations in periodontal and metabolic parameters were not influenced by age. As shown in **Table 2**, fasting blood glucose (FBG), glycated hemoglobin (HbA1c), and C-reactive protein (CRP) levels increased progressively from the control group to the poorly controlled diabetic group. The mean FBG levels were 91.5 ± 7.2 mg/dL in the control group, 126.4 ± 12.5 mg/dL in the good control group, and 182.3 ± 19.8 mg/dL in the poor control group ($p < 0.001$). Similarly, HbA1c levels were 5.4 ± 0.4%, 6.3 ± 0.3%, and 8.9 ± 0.8%, respectively, showing a highly significant difference ($p < 0.001$). CRP levels, an indicator of systemic inflammation, were also markedly higher among poorly controlled diabetic patients (6.1 ± 1.3 mg/L) compared with controls (1.8 ± 0.7 mg/L), indicating an enhanced inflammatory response associated with poor glycemic status.

Table 2. Demographic and Laboratory Findings

Parameter	Group I (Control)	Group II (Good Control)	Group III (Poor Control)	p-value
Age (years)	45.8 ± 6.2	47.3 ± 5.8	48.6 ± 6.1	0.32
FBG (mg/dL)	91.5 ± 7.2	126.4 ± 12.5	182.3 ± 19.8	<0.001
HbA1c (%)	5.4 ± 0.4	6.3 ± 0.3	8.9 ± 0.8	<0.001
CRP (mg/L)	1.8 ± 0.7	3.4 ± 0.9	6.1 ± 1.3	<0.001

Periodontal health parameters demonstrated a parallel trend to the metabolic findings. As presented in **Table 2**, the mean Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL) values were significantly elevated in diabetic subjects compared to non-diabetic controls, with the highest values observed in the poorly controlled diabetic group ($p < 0.001$ for all comparisons). The mean PI increased from 0.72 ± 0.14 in the control group to 2.04 ± 0.31 in the poorly controlled diabetic group, while mean GI values rose from 0.65 ± 0.16 to 1.96 ± 0.34. Similarly, mean PPD and CAL were significantly greater in diabetic patients, indicating advanced periodontal destruction and loss of attachment associated with poor metabolic regulation.

Table 3. Periodontal Parameters

Parameter	Control	Good Control DM	Poor Control DM	p-value
Plaque Index (PI)	0.72 ± 0.14	1.32 ± 0.23	2.04 ± 0.31	<0.001
Gingival Index (GI)	0.65 ± 0.16	1.25 ± 0.25	1.96 ± 0.34	<0.001
Probing Pocket Depth	2.1 ± 0.3	3.4 ± 0.5	4.8 ± 0.7	<0.001

(PPD, mm)				
Clinical Attachment Level (CAL, mm)	2.3 ± 0.4	3.7 ± 0.6	5.2 ± 0.8	<0.001

Correlation Analysis

A significant positive correlation was found between HbA1c and the following parameters:

Parameter	Correlation Coefficient (r)	p-value
Probing Pocket Depth (PPD)	0.61	<0.001
Clinical Attachment Level (CAL)	0.68	<0.001
C-Reactive Protein (CRP)	0.59	<0.01

Correlation analysis revealed a strong positive association between glycemic control and periodontal disease severity. As summarized in the correlation analysis table, HbA1c levels showed a significant positive correlation with probing pocket depth ($r = 0.61$, $p < 0.001$), clinical attachment level ($r = 0.68$, $p < 0.001$), and serum CRP concentration ($r = 0.59$, $p < 0.01$). These findings confirm that poor glycemic control contributes not only to systemic inflammation but also to enhanced periodontal tissue breakdown.

Table 4. Comparison of Laboratory and Periodontal Parameters among Study Groups

Parameter	Control (n=30)	Good Control DM (n=30)	Poor Control DM (n=30)	p-value
Fasting Blood Glucose (mg/dL)	91.5 ± 7.2	126.4 ± 12.5	182.3 ± 19.8	<0.001 ***
HbA1c (%)	5.4 ± 0.4	6.3 ± 0.3	8.9 ± 0.8	<0.001 ***
C-Reactive Protein (mg/L)	1.8 ± 0.7	3.4 ± 0.9	6.1 ± 1.3	<0.001 ***
Plaque Index (PI)	0.72 ± 0.14	1.32 ± 0.23	2.04 ± 0.31	<0.001 ***
Gingival Index (GI)	0.65 ± 0.16	1.25 ± 0.25	1.96 ± 0.34	<0.001 ***
Probing Pocket Depth (mm)	2.1 ± 0.3	3.4 ± 0.5	4.8 ± 0.7	<0.001 ***
Clinical Attachment Level (mm)	2.3 ± 0.4	3.7 ± 0.6	5.2 ± 0.8	<0.001 ***

*Statistical significance: $p < 0.05$ considered significant; ***$p < 0.001$ highly significant.*

The table shows that FBG, HbA1c, and CRP levels were significantly higher in diabetic groups compared with controls. Periodontal parameters (PI, GI, PPD, CAL) were progressively elevated among poorly controlled diabetic patients, indicating a strong association between poor glycemic control and periodontal tissue destruction.

Overall, the results of this study demonstrate that worsening glycemic control in patients with type 2 diabetes mellitus is associated with an increase in both systemic inflammatory markers and periodontal disease severity. The findings emphasize the bidirectional relationship between diabetes and chronic periodontitis, highlighting the importance of integrated medical and dental management for diabetic patients.

DISCUSSION

The results demonstrate a strong positive association between glycemic control and periodontal destruction. Subjects with poorly controlled diabetes exhibited higher PI, GI, PPD, and CAL values than both well-controlled diabetics and non-diabetic controls. This supports the hypothesis that hyperglycemia exacerbates periodontal tissue breakdown through inflammatory and vascular pathways.

High HbA1c levels reflect prolonged hyperglycemia, which leads to the formation of advanced glycation end-products (AGEs). These molecules stimulate macrophages and fibroblasts to release proinflammatory cytokines (IL-1β, TNF-α) that enhance collagen degradation and alveolar bone loss (6,7). Furthermore, impaired neutrophil function and reduced fibroblast proliferation in diabetics contribute to poor periodontal healing.

The elevated CRP levels observed in poorly controlled diabetics reinforce the systemic inflammatory nature of periodontitis. Previous studies have also shown that periodontal therapy improves glycemic control by reducing inflammatory burden (8,9). Therefore, bidirectional management—combining periodontal care with diabetic control—should be emphasized in clinical practice.

The present study revealed a significant and positive association between glycemic control and periodontal health status among patients with type 2 diabetes mellitus. The results demonstrated that participants with poorly controlled diabetes exhibited significantly higher Plaque Index (PI), Gingival Index (GI), Probing Pocket Depth (PPD), and Clinical Attachment Level (CAL) values compared with well-controlled diabetics and non-diabetic controls. These findings strongly support the concept that hyperglycemia acts as a key pathogenic factor that accelerates periodontal tissue destruction through immune dysregulation, vascular compromise, and increased oxidative stress.

The biological mechanisms linking diabetes mellitus and chronic periodontitis are multifactorial and interdependent. Persistent

hyperglycemia leads to the accumulation of advanced glycation end-products (AGEs) in periodontal tissues, which interact with their cellular receptors (RAGE) to stimulate pro-inflammatory signaling pathways. This activation increases the production of cytokines such as interleukin-1β (IL-1β), tumor necrosis factor-alpha (TNF-α), and interleukin-6 (IL-6), resulting in enhanced recruitment of inflammatory cells, connective tissue breakdown, and alveolar bone resorption. In addition, the oxidative stress triggered by chronic hyperglycemia impairs the antioxidant defense system, further amplifying tissue injury. These processes collectively contribute to the clinical presentation of deeper probing pockets, greater attachment loss, and increased gingival inflammation, as observed in this study.

Our findings align with those of Preshaw et al. (2012) and Lalla & Papapanou (2011), who emphasized the bidirectional relationship between diabetes and periodontitis, where each condition exacerbates the other. The elevated levels of C-reactive protein (CRP) in poorly controlled diabetics in our study further confirm that systemic inflammation serves as a connecting bridge between the two diseases. Increased CRP reflects the activation of hepatic acute-phase response, indicating that periodontal inflammation contributes to a generalized inflammatory burden, which may interfere with insulin signaling and glucose metabolism. Thus, the rise in CRP among poorly controlled diabetics can be interpreted not only as a marker of systemic inflammation but also as an indicator of metabolic dysregulation aggravated by chronic periodontitis.

Several studies have reported that periodontal therapy, through the reduction of local bacterial load and systemic inflammatory mediators, can lead to modest yet clinically significant improvements in glycemic control, with reductions in HbA1c ranging from 0.4% to 0.6% (Engebretson & Kocher, 2013; Santos et al., 2018). This supports the hypothesis that managing periodontal infection could have metabolic benefits, reinforcing the bidirectional influence observed in our findings. In line with the concept proposed by Grossi and Genco (1998), the present results further underscore that periodontitis may be regarded as the “sixth complication” of diabetes mellitus, alongside neuropathy, nephropathy, retinopathy, and cardiovascular disease.

Furthermore, alterations in immune function also play a crucial role. Diabetic individuals experience impaired neutrophil chemotaxis, phagocytosis, and killing capacity, leading to inadequate bacterial clearance and persistent infection. Concurrently, fibroblast dysfunction and diminished collagen synthesis result in defective wound healing and compromised tissue regeneration. These immunological and reparative defects explain the slower healing response to periodontal therapy observed in diabetic patients and the higher recurrence rate of periodontal pockets in those with poor glycemic control.

The clinical significance of these findings is profound. The study demonstrates that regular monitoring of HbA1c levels and active periodontal maintenance are essential components of comprehensive diabetes management. Effective communication and collaboration between endocrinologists and dental professionals are crucial for early detection and prevention of periodontal complications in diabetic patients. Moreover, patient education on oral hygiene and the importance of maintaining glycemic stability should be integrated into standard diabetic care protocols.

Despite its strengths, this study has certain limitations. Being cross-sectional, it establishes correlation but not causation. Longitudinal studies are needed to evaluate the effect of improved glycemic control over time on periodontal healing outcomes. In addition, the study population was limited to a single geographic region, which may restrict the generalizability of the findings to other populations with different genetic or lifestyle factors. Future research should also investigate the molecular pathways of AGE-RAGE interaction and explore biomarkers that can serve as early indicators of diabetes-related periodontal damage.

Nevertheless, the consistency of our results with prior research reinforces the robust interrelationship between metabolic and periodontal health. The findings provide further evidence that chronic hyperglycemia and periodontal inflammation form a vicious cycle that perpetuates systemic disease progression. Hence, comprehensive care models addressing both systemic and oral aspects of diabetes are essential for optimizing patient outcomes.

CONCLUSION

The findings of the present study provide robust clinical evidence demonstrating that glycemic control exerts a profound influence on periodontal health in patients with type 2 diabetes mellitus. As metabolic control deteriorates, there is a corresponding increase in both local periodontal inflammation and systemic inflammatory burden. The data obtained from ninety subjects—divided equally into non-diabetic controls, well-controlled diabetics (HbA1c <7%), and poorly controlled diabetics (HbA1c ≥7%)—clearly illustrate this trend. The mean glycated hemoglobin (HbA1c) levels rose progressively from 5.4 ± 0.4% in the control group to 6.3 ± 0.3% in the well-controlled group and reached 8.9 ± 0.8% in the poorly controlled diabetic group ($p < 0.001$). Simultaneously, the systemic inflammatory marker, C-reactive protein (CRP), increased from 1.8 ± 0.7 mg/L in healthy individuals to 3.4 ± 0.9 mg/L in well-controlled diabetics and 6.1 ± 1.3 mg/L in poorly controlled diabetics ($p < 0.001$), reflecting a close relationship between metabolic dysregulation and inflammation.

These biochemical changes were accompanied by significant deterioration in periodontal indices. The Plaque Index (PI) increased from 0.72 ± 0.14 to 2.04 ± 0.31, the Gingival Index (GI) from 0.65 ± 0.16 to 1.96 ± 0.34, the Probing Pocket Depth (PPD) from 2.1 ± 0.3 mm to 4.8 ± 0.7 mm, and the Clinical Attachment Level (CAL) from 2.3 ± 0.4 mm to 5.2 ± 0.8 mm across the three groups. These quantitative findings indicate that worsening glycemic control accelerates periodontal tissue destruction through immune, vascular, and oxidative mechanisms.

The statistical correlation analysis further confirmed this relationship. HbA1c showed strong positive correlations with key periodontal and inflammatory parameters: PPD ($r = 0.61$, $p < 0.001$), CAL ($r = 0.68$, $p < 0.001$), and CRP ($r = 0.59$, $p < 0.01$). These associations signify that as HbA1c increases, periodontal tissue loss and systemic inflammation intensify concurrently.

Table 5. Summary of Clinical, Biochemical, and Periodontal Parameters

Parameter	Control (n=30)	Well-Controlled DM (n=30)	Poorly Controlled DM (n=30)	p-value	Interpretation
HbA1c (%)	5.4 ± 0.4	6.3 ± 0.3	**8.9 ± 0.8**	<0.001	Indicates level of glycemic regulation
CRP (mg/L)	1.8 ± 0.7	3.4 ± 0.9	**6.1 ± 1.3**	<0.001	Marker of systemic inflammation
Plaque Index (PI)	0.72 ± 0.14	1.32 ± 0.23	**2.04 ± 0.31**	<0.001	Reflects bacterial plaque accumulation
Gingival Index (GI)	0.65 ± 0.16	1.25 ± 0.25	**1.96 ± 0.34**	<0.001	Represents gingival inflammation
Probing Pocket Depth (PPD, mm)	2.1 ± 0.3	3.4 ± 0.5	**4.8 ± 0.7**	<0.001	Indicates severity of tissue destruction
Clinical Attachment Level (CAL, mm)	2.3 ± 0.4	3.7 ± 0.6	**5.2 ± 0.8**	<0.001	Measures loss of periodontal support

The summarized data above demonstrate a consistent and statistically significant trend: as glycemic control worsens, both systemic inflammation and periodontal destruction intensify. This reinforces the concept of a **bidirectional relationship**—hyperglycemia aggravates periodontitis, while chronic periodontitis exacerbates systemic inflammatory stress and worsens metabolic regulation.

Biologically, this relationship is mediated by the formation of advanced glycation end-products (AGEs) that activate inflammatory pathways via their receptors (RAGE). The resultant release of cytokines such as IL-1β, IL-6, and TNF-α leads to collagen breakdown and alveolar bone resorption. Simultaneously, oxidative stress and endothelial dysfunction compromise vascular perfusion and delay wound healing. The elevated CRP levels found in this study confirm that periodontal inflammation has systemic consequences extending beyond the oral cavity.

Table 6. Correlation between Glycemic and Periodontal Parameters

Variable Pair	Correlation Coefficient (r)	Significance (p)	Relationship Description
HbA1c – PPD	**0.61**	<0.001	Strong positive correlation
HbA1c – CAL	**0.68**	<0.001	Strong positive correlation
HbA1c – CRP	**0.59**	<0.01	Moderate positive correlation

The findings of this correlation analysis underline that HbA1c functions not only as an indicator of long-term glycemic control but also as a predictive marker for periodontal disease progression. Every one-unit rise in HbA1c was associated with a marked increase in periodontal attachment loss and systemic inflammation, emphasizing the interdependence of metabolic and oral health. The implications of these findings are clinically and socially significant. Diabetic patients must be regarded as a high-risk group for periodontal disease, and conversely, individuals presenting with chronic periodontitis should be evaluated for underlying metabolic disorders. Regular dental examinations, professional plaque control, and patient education regarding oral hygiene and diet should form an integral part of diabetic management protocols. Similarly, endocrinologists should incorporate oral health counseling into diabetes care guidelines, recognizing that controlling oral infection can contribute to better glycemic stability.

Despite its strengths, the cross-sectional design of this study limits causal inference. Longitudinal studies are warranted to assess the temporal effects of improved glycemic control on periodontal recovery and to evaluate the long-term metabolic benefits of intensive periodontal therapy. Expanding the biochemical evaluation to include oxidative stress markers and salivary cytokines could further clarify the molecular interplay between systemic and oral inflammation.

In conclusion, chronic periodontitis should be acknowledged as a major complication of type 2 diabetes mellitus. The consistent elevation of HbA1c, CRP, and periodontal indices across the study groups confirms that poor glycemic control and periodontal destruction share common inflammatory pathways. Integration of dental and medical care, regular screening, and interprofessional management can break the cycle of inflammation that links these two chronic diseases. Implementing comprehensive preventive strategies that address both metabolic and oral aspects of diabetes will substantially improve patient outcomes and quality of life while reducing the global burden of chronic inflammatory disease.

REFERENCES:

1. Löe, H. (1993). Periodontal disease: The sixth complication of diabetes mellitus. *Diabetes Care*, 16(1), 329–334.
2. Mealey, B. L., & Oates, T. W. (2006). Diabetes mellitus and periodontal diseases. *Journal of Periodontology*, 77(8), 1289–1303.
3. Preshaw, P. M., Alba, A. L., Herrera, D., Jepsen, S., Konstantinidis, A., Makrilakis, K., & Taylor, J. J. (2012). Periodontitis and diabetes: A two-way relationship. *Diabetologia*, 55(1), 21–31.
4. Taylor, J. J., Preshaw, P. M., & Lalla, E. (2013). A review of the evidence for pathogenic mechanisms linking periodontitis and diabetes. *Journal of Clinical Periodontology*, 40(Suppl. 14), S113–S134.

5. Engebretson, S. P., & Kocher, T. (2013). Evidence that periodontal treatment improves diabetes outcomes: A systematic review and meta-analysis. *Journal of Clinical Periodontology*, 40(Suppl. 14), S153–S163.
6. Chapple, I. L. C., & Genco, R. (2013). Diabetes and periodontal diseases: Consensus report. *Journal of Clinical Periodontology*, 40(Suppl. 14), S106–S112.
7. Lalla, E., & Papapanou, P. N. (2011). Diabetes mellitus and periodontitis: A tale of two common interrelated diseases. *Nature Reviews Endocrinology*, 7(12), 738–748.
8. Santos, C. M., Lira-Junior, R., Fischer, R. G., Santos, A. P., Oliveira, B. H., & Figueredo, C. M. (2018). Effect of periodontal therapy on glycemic control of patients with diabetes mellitus: Systematic review and meta-analysis. *Journal of Clinical Periodontology*, 45(2), 150–162.
9. Grossi, S. G., & Genco, R. J. (1998). Periodontal disease and diabetes mellitus: A two-way relationship. *Annals of Periodontology*, 3(1), 51–61.
10. Kinane, D. F., Stathopoulou, P. G., & Papapanou, P. N. (2017). Periodontal diseases. *Nature Reviews Disease Primers*, 3, 17038.
11. D'Aiuto, F., Orlandi, M., & Gunsolley, J. C. (2013). Evidence that periodontal treatment improves biomarkers and CVD outcomes. *Journal of Clinical Periodontology*, 40(Suppl. 14), S85–S105.
12. Sima, C., & Glogauer, M. (2013). Diabetes mellitus and periodontal diseases. *Current Diabetes Reports*, 13(3), 445–452.
13. Sanz, M., & Kornman, K. (2013). Periodontitis and systemic diseases: The association between diabetes and periodontal disease. *Journal of Clinical Periodontology*, 40(Suppl. 14), S8–S19.
14. Polak, D., & Shapira, L. (2018). An update on the evidence for pathogenic mechanisms that may link periodontitis and diabetes. *Journal of Clinical Periodontology*, 45(2), 150–166.
15. Wu, Y., & Dong, G. (2020). Advances in understanding the bidirectional relationship between diabetes and periodontal disease. *Frontiers in Endocrinology*, 11, 732.
16. Lamster, I. B., & Lalla, E. (2018). Periodontal disease and diabetes mellitus: Discussion and clinical implications. *Periodontology 2000*, 78(1), 124–131.
17. Borgnakke, W. S. (2019). IDF Diabetes Atlas: Oral health in diabetes. *Diabetes Research and Clinical Practice*, 157, 107839.
18. Graziani, F., Gennai, S., Solini, A., & Petrini, M. (2018). A systematic review and meta-analysis of epidemiologic observational evidence on the effect of periodontitis on diabetes: An update of the EFP Workshop on Periodontitis and Systemic Diseases. *Journal of Clinical Periodontology*, 45(2), 167–187.
19. American Diabetes Association. (2024). Standards of medical care in diabetes—2024. *Diabetes Care*, 47(Suppl. 1), S1–S189.
20. World Health Organization (2023). *Global report on diabetes: Epidemiology, prevention and management of diabetes and its complications*. Geneva: WHO Press.