Original Article

# Comparison of the gingival phenotype in diabetic and non-diabetic subjects among patients suffering from periodontal diseases

*Aram M. Sha*[1*]

## Abstract

**Objective:** This study aimed to compare the gingival phenotype (GP) in subjects with periodontal disease and type 2 diabetes mellitus (DM) with that in subjects without diabetes mellitus.

**Methods:** A cross-sectional study was performed involving 182 subjects with periodontal diseases (102 with type 2 DM, 80 without DM), aged 40 to 65 years. Clinical parameters were assessed, including keratinized gingival width (KGW) and gingival thickness (GT), plaque index (PI), bleeding index (BI), probing depth (PD), and clinical attachment loss (CAL).

**Results:** Diabetic patients displayed significantly higher KGW (5.58 ± 0.90 mm vs. 5.12 ± 0.58 mm, p = 0.006), PI (41.19 ± 19.71% vs. 28.38 ± 13.53%, p = 0.001), and BI (42.49 ± 18.16% vs. 28.19 ± 13.80%, p = 0.0001) in comparison to non-DM individuals. There were no significant differences in GT, PD, and CAL between the groups. Sex-based comparisons indicated no significant differences between any of the examined parameters. Correlation analysis and chi-square testing demonstrated substantial relationships between PI and BI (p = 0.0001), BI and CAL (p = 0.04), and PD with CAL (p = 0.0001).

**Conclusions:** Diabetes significantly alters the GP by increasing KGW, plaque, and bleeding indices, whereas other clinical parameters, such as GT, PD, and CAL, remain predominantly unchanged. These findings underscore the importance of managing inflammation and closely monitoring treatment outcomes in individuals with diabetes with periodontal disease.

**Keywords:** *Gingival phenotype, Keratinized gingival width, Gingival thickness, Diabetes mellitus, Periodontal disease.*

*Submitted: December 19, 2025, Accepted: January 21, 2026, Published: April 1, 2026.*

**Cite this article as:** Sha AM. Comparison of the gingival phenotype in diabetic and non-diabetic subjects among patients suffering from periodontal diseases. Sulaimani Dent J. 2026;13(1):40-50.

***DOI: https://doi.org/10.17656/sdj.10220***

1. *Periodontics Department, College of Dentistry, University of Sulaimani, Sulaimani, Iraq.*

** Corresponding author*: *aram.hamad@univsul.edu.iq.*

Published by the College of Dentistry, University of Sulaimani

## Introduction

Periodontal diseases (gingivitis and periodontitis) are chronic, multifactorial inflammatory diseases of the tooth-supporting structures. They result from the interaction between pathogenic bacterial biofilms and host immune-inflammatory responses and are influenced by systemic diseases[1,2]. Among systemic diseases, diabetes mellitus (DM) is considered a major risk factor for periodontal diseases. Periodontitis and DM have a bidirectional relationship: periodontitis can affect glycemic control and lead to diabetic complications; similarly, DM is associated with the increased prevalence and severity of periodontitis[3].

The term "periodontal phenotype", suggested by the 2017 classification of periodontal and peri-implant diseases and conditions, refers to the combination of gingival phenotype (GP) that includes both three-dimensional gingival volumes, such as keratinized gingival width (KGW) and gingival thickness (GT), as well as bone morphotype (the thickness of the buccal or labial bony plate)[4]. Clinically, GP is frequently classified as either thick or thin. Gingival recession, periodontal attachment loss, and compromised esthetics are more likely to occur in a thin GP, which is typically associated with thin tooth morphologies and narrow keratinized tissue[5,6]. On the other hand, a thick GP offers better treatment outcomes by being more resistant to trauma and inflammation, and better outcomes following periodontal surgical procedures[7]. Additionally, a thick GP has conventionally been considered fundamental for preserving periodontal health and improving plaque control. However, it has been reported that even with a minimal KGW, periodontal health may be maintained with proper oral hygiene measures[8].

GP can be evaluated by direct visual inspection, transgingival probing, dental probe transparency, ultrasonic transducer, parallel profile periapical radiography, and cone-beam computed tomography (CBCT)[9]. Nevertheless, more than 50% of patients with the thin-scalloped GP are misclassified, suggesting that thick and thin GPs cannot be distinguished by direct visual evaluation[10]. Although trans gingival probing is a straightforward procedure, it requires local anesthesia to distort the soft tissues. On the other hand, despite being non-invasive, ultrasonic instruments are unable to detect subtle variations in gingival tissues[11]. Recently, CBCT has become a more common method to evaluate the type of GP. However, in addition to the risk of high-dose radiation exposure, it requires technical expertise and is usually part of the cost-benefit analysis[12]. Finally, a straightforward technique for evaluating the GP based on the transparency of the periodontal probe throughout the gingival margin has been introduced. This approach is considered the "gold standard" because it has been used in numerous clinical studies[6,13].

In diabetes, the structure and healing capacity of gingival tissues can be adversely affected by hyperglycemia-induced microvascular changes, impaired collagen metabolism, increased inflammatory responses, and altered neutrophil activity[14, 15]. Reduced GT and KGW may occur in individuals with DM due to these metabolic and circulatory alterations. Many studies have focused on the effects of GPs on periodontal health and disease[16-19]. However, research specifically comparing GP in DM and non-DM individuals with periodontal diseases has not yet been conducted. Therefore, this cross-sectional study was conducted to compare GPs in individuals with DM and those without DM with periodontal disease.

## Materials and methods

### Ethical Approval

Ethical approval for the current cross-sectional clinical study was obtained from the Ethical Committee of the College of Dentistry, University of Sulaimani (ethical approval code: COD-EC-25-0112, on December 14, 2025). Accordingly, a sample of 182 individuals with periodontal diseases, comprising both males and females, was selected from the College of Dentistry-Periodontics Department and the Diabetes and Endocrine Center in Sulaimani city, Kurdistan Region, Iraq, between 21 December 2022, and 25 November 2024. All participants signed written informed consent before participating in the study.

### Study design

A total of 182 individuals with periodontal diseases were classified into type 2 DM (80 subjects) and non-DM (102 subjects) groups (Figure 1). Periodontal disease subjects recruited were defined by the presence of bleeding on probing at >10% of sites for gingivitis or "the presence of interproximal clinical loss of attachment (CAL) at ≥2 teeth or the presence of CAL ≥3 mm at the facial/oral surfaces associated with probing pocket depths (PPDs) ≥4 mm for periodontitis"[20]. DM was verified by HbA1c readings ≥ 6.5% and medical records. To be eligible, diabetic patients had to have had type 2 DM for at least a year and have moderate glycemic control, as indicated by HbA1c levels between 7.0 and 9.0%[21].

### Study criteria

The inclusion criteria for the study were an age range of 40-65 years, individuals with upper and lower anterior teeth, diagnosed as periodontal disease, and those with

diabetes, confirmed as type 2 DM for at least 1 year, with an HbA1c level within the last three months of 7.0–9.0%. Meanwhile, the criteria for exclusion from the study were individuals with long-term use of drugs that influence gingival tissues, such as cyclosporine, calcium channel blockers, and phenytoin, history of periodontal therapy during the previous six months, pregnancy or lactation, smoking or a history of smoking during the previous five years, orthodontic appliances, systemic diseases other than diabetes that are known to impact periodontal health.

**Clinical Periodontal Parameters**

BI and PI were measured to evaluate gingival inflammation and dental hygiene, respectively[22]. Bleeding observed after 20 seconds was recorded as 1, while its absence was 0. Dental plaque presence was coded as 1 and absence as 0. At the same time, PD and CAL were measured as the distances from the gingival margin and cementoenamel junction to the base of the sulcus/pocket, respectively[20].

Twelve teeth per patient, six maxillary anterior (FDI numbers: 11, 12, 13, 21, 22, 23), and six mandibular anterior teeth (FDI numbers: 31, 32, 33, 41, 42, 43), were examined for the clinical examination of GT and KGW.

The UNC-15 probe was used to evaluate the KGW, and its evaluation was performed by measuring the distance from the gingival margin to the mucogingival junction at the mid-labial aspect of all anterior teeth[23].

Based on the visibility of the periodontal probe through the gingival sulcus, a clinical examination was conducted to assess GT. “Colorvue TM Biotype Probe (Hu-Friedy®, Chicago, IL, USA)” was used in the probe transparency method to detect the GP. GT can be classified as thin, medium, thick, or very thick, with the probe's three different-colored resin tips (white, green, and blue)[13]. This technique involved inserting the probe 1 mm into the gingival sulcus of the maxillary and mandibular anterior teeth through the mid-labial surface (Figure 2). The phenotype was considered thin if the white tip was visible, medium if the green tip was visible, and the phenotype was labeled as thick if the blue tip was present, and as extremely thick if neither the green nor the blue tip was discernible[24].

**Examiner Calibration**

A single calibrated examiner performed all clinical examinations. A kappa value of > 0.85 was obtained for intra-examiner reliability for B after calibration on ten subjects not included in the study for the clinical parameters, suggesting strong repeatability[25].

**Statistical Analysis**

Data were compiled in Microsoft Excel and analyzed using SPSS version 26. The Shapiro-Wilk test assessed normality, while descriptive statistics, including mean ± SD and frequency, were calculated. Independent samples t-tests compared mean values of KGW, PD, and CAL between groups, and the Chi-square test examined distributions of GT, PI, and BI. Pearson's correlation was used to assess the correlation between the means of clinical parameters. Statistical significance was defined as a p-value of ≤ 0.05.

## Results

The study population consisted of 182 patients, among whom 80 (43.95%) were non-DM and 102 (56.04%) were DM**.** The distribution of participants by sex showed that 132 (72.5%) were female and 50 (27.5%) were male. The average age of patients with DM was 52.04 ± 7.2 years, whereas in those without diabetes, the mean age was 46.54 ± 6.37 years.

**Comparison of clinical parameters between diabetic and non-diabetic patients**

The mean of GT in DM was 1.81± 0.85 mm, while in non-DM, it was 1.91± 0.89 mm, with no statistically significant difference (p = 0.5). DM subjects had a statistically significantly higher KGW (5.58± 0.9 mm) than non-DM subjects (5.12 ± 0.58 mm, p < 0.006).

Likewise, individuals with DM had a greater PI (41.19±19.71%) than those without DM (28.38 ± 13.53%, p = 0.001). Additionally, there was a significant difference in the BI in DM (42.49 ± 18.16%) when compared to non- DM (28.19 ±13.80, p = 0.0001).

No statistically significant differences in mean PD between DM subjects (1.75 ± 0.75 mm) and non-DM subjects (1.38 ± 0.65mm) were detected (p = 0.07). Similarly, no statistically significant differences in mean CAL of DM (1.48 ± 0.33 mm) and non-DM (1.40 ± 0.28 mm) were observed (p = 0.8).

Overall, these results show that DM patients had considerably higher KGW, PI, and BI values than non-DM subjects. Whereas no statistically significant differences in GT, PD, and CAL were detected between the studied groups (Figure 3).

**Tooth-by-tooth comparison of keratinized gingival width between diabetic and non-diabetic patients**

As mentioned earlier, DM individuals had a mean KGW of 5.58 ± 0.90 mm, while non-DM individuals showed a mean KGW of 5.12 ± 0.58 mm. In DM subjects, the mean KGW per tooth ranged from 4.13 ± 1.18 mm to

6.79 ± 1.18 mm. While in non-DM patients, the range was 4.06 ± 1.09 mm to 6.28 ± 1.09 mm. No statistically significant differences between groups at any tooth site ($p > 0.05$) were detected, and KGW distribution across the dentition was found to be similar for both groups, as shown in Figure 4.

**Tooth-by-tooth comparison of gingival thickness between diabetic and non-diabetic patients**

Again, as presented in Figure 3, DM patients had a mean GT of 1.81 ± 0.85 mm, and non-DM patients showed a mean GT of 1.91 ± 0.89 mm. When evaluated by individual tooth, DM patients had average GT values ranging from 1.74 ± 0.03 mm to 1.84 ± 0.03 mm. In non-DM subjects, average GT values ranged from 1.78 ± 0.11 mm to 2.09 ± 0.11 mm. Although some tooth sites showed numerical differences, statistical analysis found no significant differences in GT between DM and non-DM groups across all teeth ($p > 0.05$ for each tooth position). Thus, GT was similarly distributed in both DM and non-DM subjects, with no significant tooth-specific changes (Figure 5).

**Comparison of clinical parameters between males and females**

A comparison of clinical periodontal parameters between male (n = 50) and female (n = 132) participants indicated no statistically significant differences in any of the parameters examined, as shown in Figure 6.

**Correlation of clinical periodontal parameters**

The darker shading in Figure 7 distinctly highlights the strong correlations (e.g., PI–BI, PD–CAL, KGW–CAL, and age-related correlations), while lighter tones indicate negligible or weaker correlations. Several significant relationships were identified among clinical parameters in the correlation analysis. Age showed a weak but statistically significant positive correlation with KGW ($r = 0.22$, $p = 0.003$), PI ($r = 0.20$, $p = 0.007$), and BI ($r = 0.27$, $p = 0.0001$). However, no statistically significant associations were found between age and GT, PD, or CAL ($p > 0.05$). Regarding the association among periodontal clinical parameters, GT showed no statistically significant correlations with KGW, PI, BI, PD, or CAL ($p > 0.05$). KGW displayed a statistically significant negative correlation with CAL ($r = –0.27$, $p = 0.0001$), while its correlations with PI, BI, and PD were not statistically significant.

Further, the study demonstrated a strong positive association between PI and BI. There was also a strong positive association between PD and CAL ($P \leq 0.01$). BI was moderately correlated with CAL ($P \leq 0.05$). However, there were no statistically significant associations among GT, KGW, and other parameters.

These findings suggest that increased age is associated with higher KGW, PI, and BI. In contrast, the correlations with other parameters showed that inflammatory indices (PI and BI) are strongly associated with periodontal breakdown markers (CAL and PD).

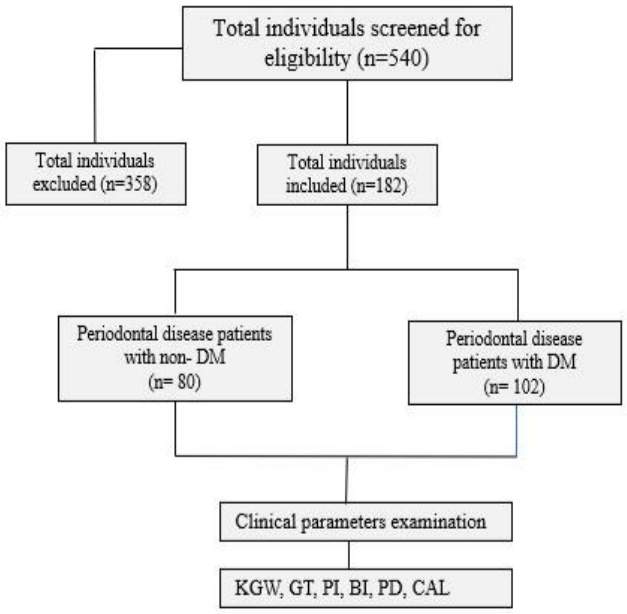


Figure 1: Design of the study.

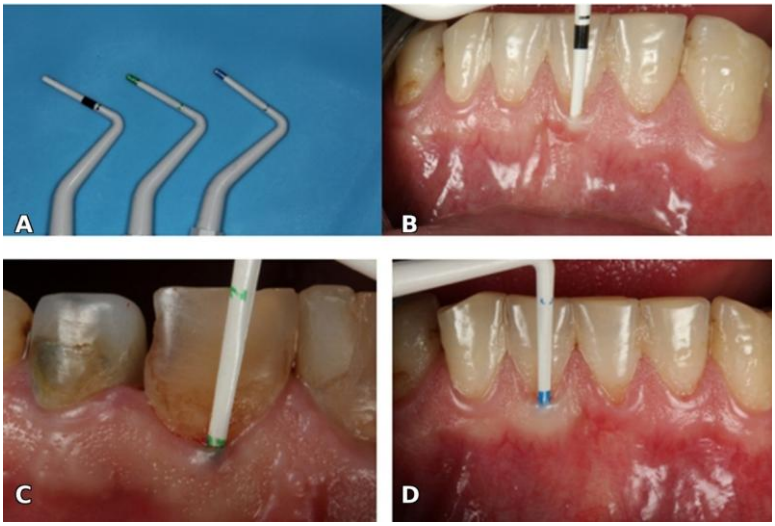


Figure 2: Probe transparency method using Hu-Friedy's Colorvue Biotype Probe for the evaluation of GT (A). The probe was inserted 1 mm into the gingival sulcus at the mid-labial surface (B). The GT was considered thin if the white tip was visible, medium if the green tip was visible, and thick if the blue tip was present, and as extremely thick if neither the green nor the blue tip was discernible (B, C, and D).

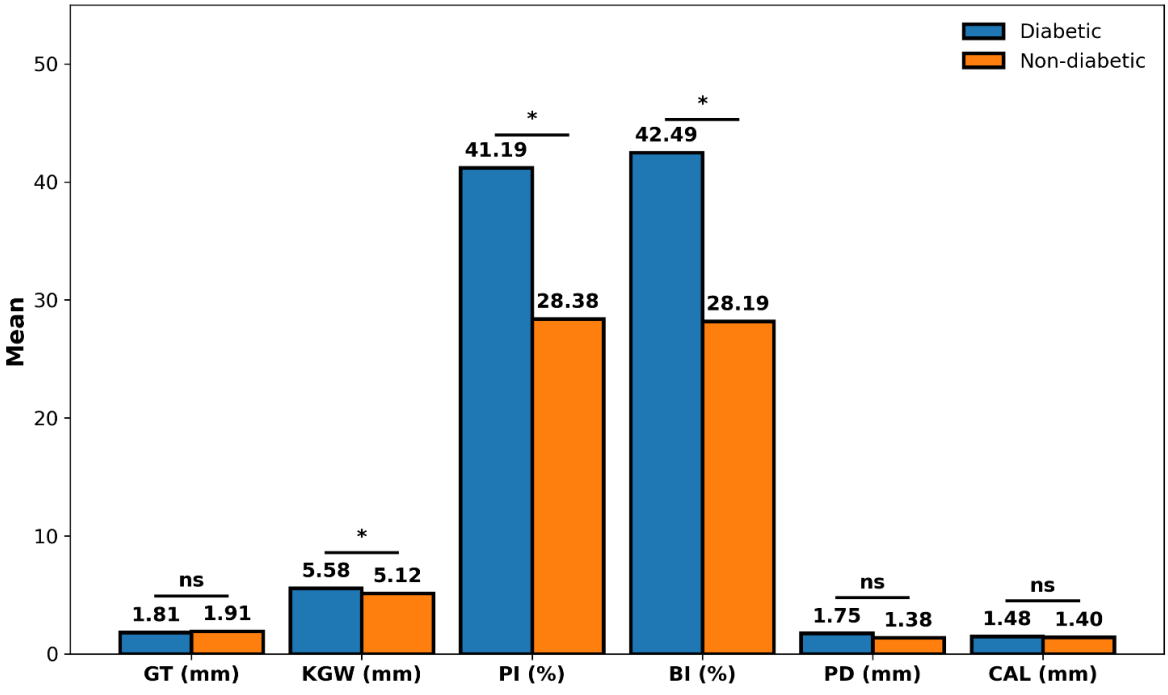


Figure 3: Comparison of clinical parameters between diabetic and non-diabetic patients. ns: non-significant.

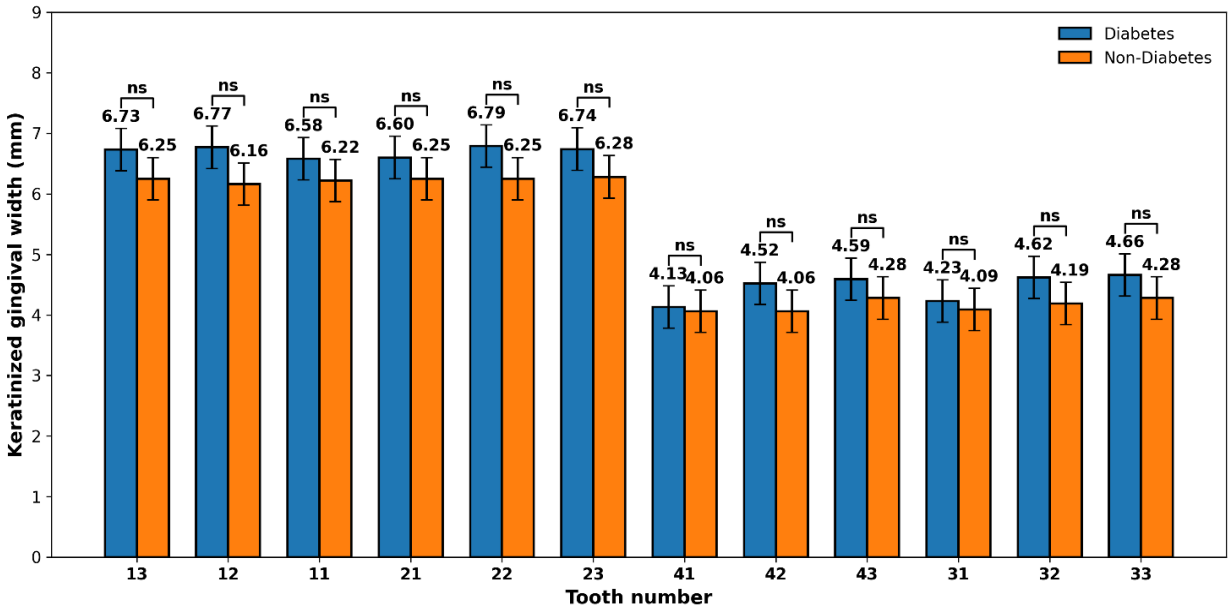


Figure 4: Keratinized gingival width in diabetic versus non-diabetic patients (tooth-by-tooth comparison).

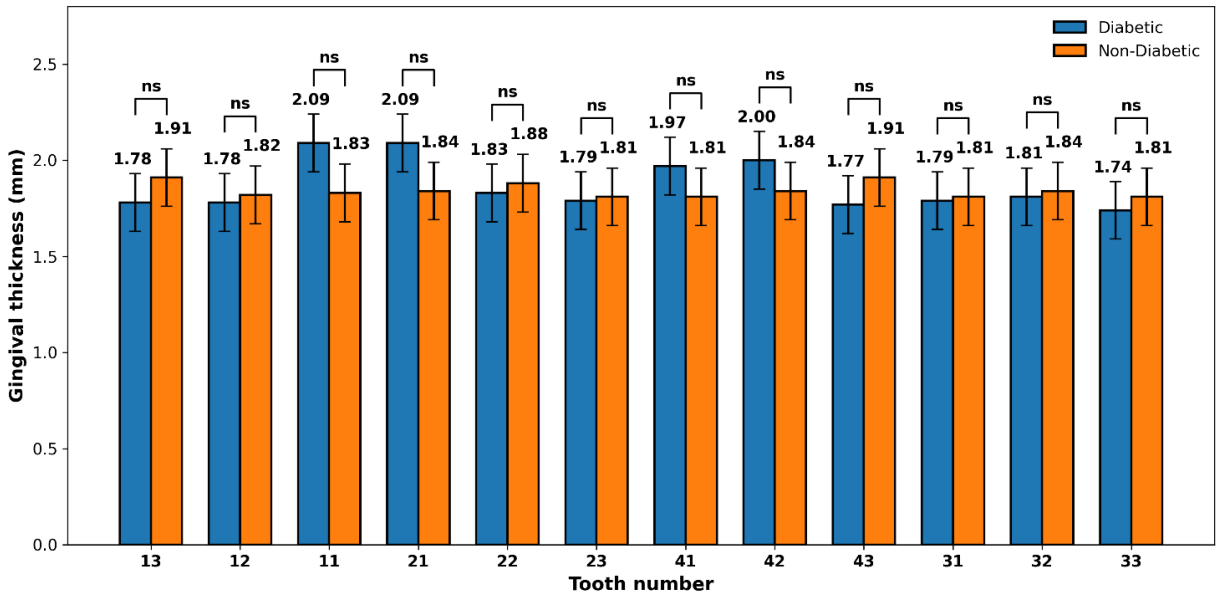


Figure 5: Gingival thickness in diabetic versus non-diabetic patients (tooth-by-tooth comparison).

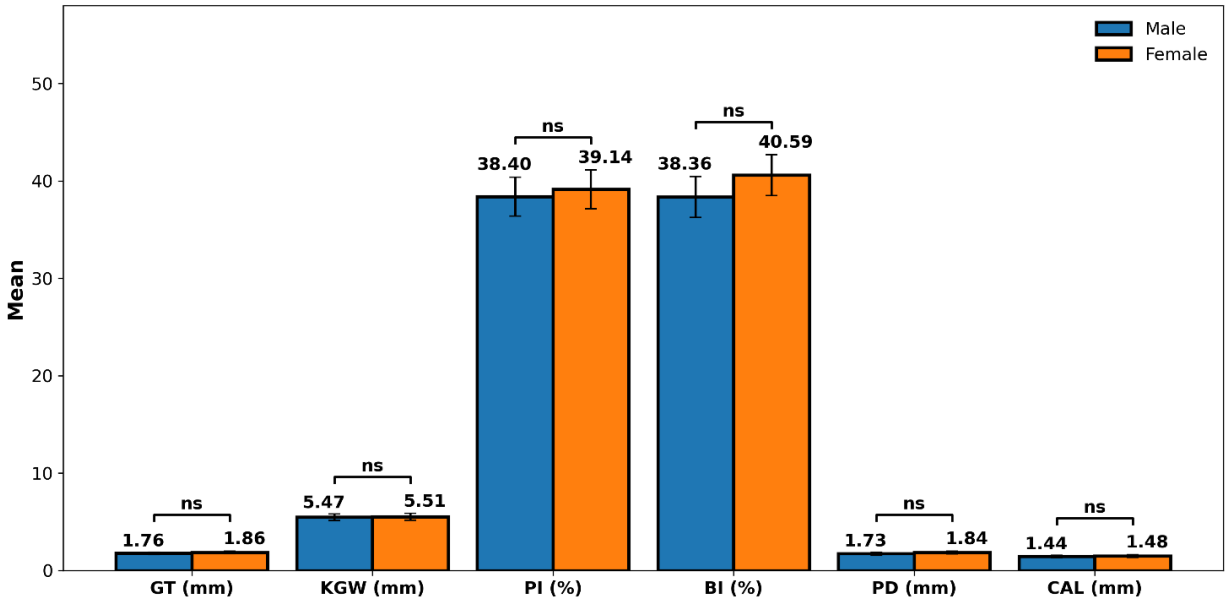


Figure 6: Comparison of clinical parameters between males and females.

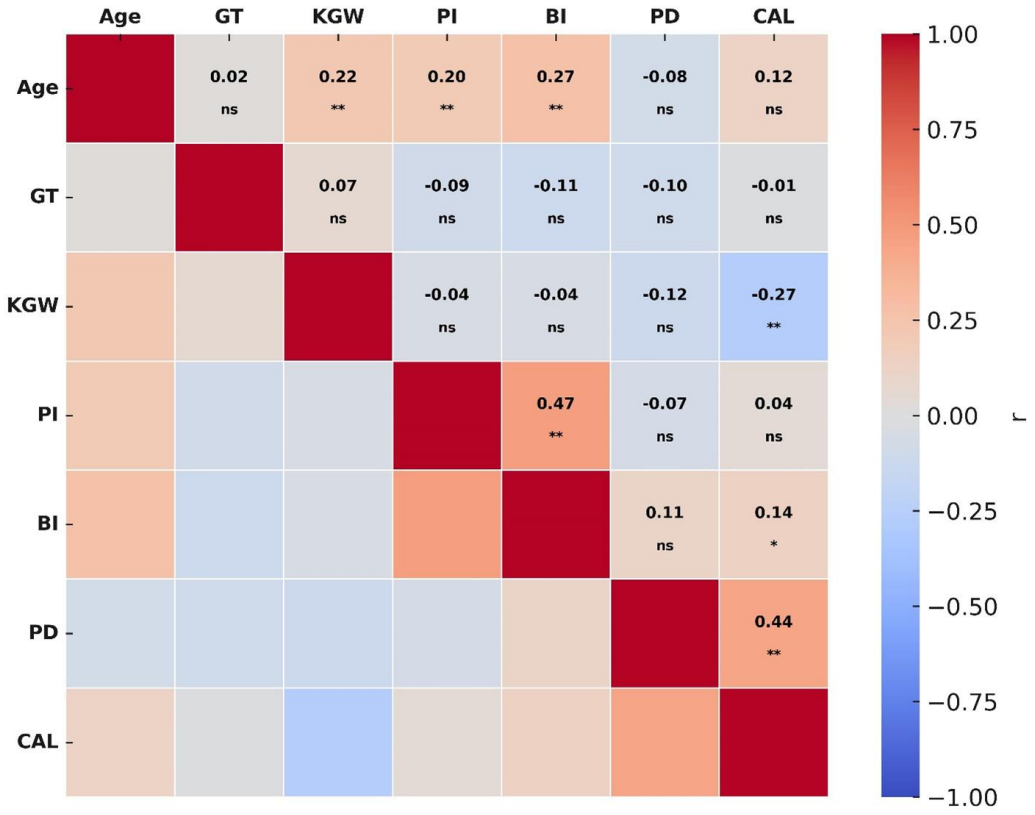


Figure 7: Correlations between clinical parameters.

r: Pearson Correlation; **Correlation is significant at the 0.01 level; *Correlation is significant at the 0.05 level; ns: non-significant

## Discussion

The GP is pivotal in periodontal health and disease progression, as it induces liability to inflammation, tissue response to bacterial biofilm, and outcomes of periodontal treatment[19]. In patients with DM, modifications in the immune-inflammatory response, disrupted collagen metabolism, and microvascular complications contribute to a thinner and more fragile GP, potentially leading to more severe periodontal destruction than in non-diabetic individuals[26,27]. Numerous studies have demonstrated that individuals with DM often have diminished KGW and altered GT. This negatively affects periodontal stability and regenerative capacity[28,29]. In contrast, non-DM individuals usually have a more robust GP. This offers better protection against microbiological and mechanical challenges[9]. Comparing the GP of DM and non-DM individuals gives significant insights. It helps understand the relationship between systemic diseases and the characteristics of periodontal tissues.

The current study demonstrated that DM impacts GP and periodontal health, with the characteristics of these effects differing across clinical and biological parameters.
The rise in KGW among DM individuals contradicts the belief that DM compromises gingival tissue heights. Multiple mechanisms may explain this observation.

Chronic hyperglycemia affects collagen metabolism and microvascular function. This may lead to fibrotic remodeling or an adaptive increase in keratinized tissues[14]. Adaptive reaction may explain the increased width of keratinized gingiva in people with DM. However, Tooth-by-tooth comparisons showed no statistically significant differences. This means the overall rise in KGW may not apply to all anterior teeth.

Recent research shows that GT measurements are sensitive to the specific locations tested. Yildirim Bolat and Lutfioglu[30] found that measuring GT at several vertical levels (sulcus base, 1 mm, and 2 mm apical) gives different results. They demonstrated that averaging multiple points offers a more dependable classification of GP. This may explain the non-significant GT differences between DM and non-DM. The finding also aligns with research showing that GT is more affected by local anatomical features and tooth morphology than by systemic disease[5,6]. Tooth-by-tooth comparisons demonstrated no statistically significant variations in GT. This finding corroborates the notion that GT is a relatively stable parameter, whereas KGW may be more vulnerable to systemic metabolic changes[31].

DM individuals showed significantly higher PI and BI. Contemporary pathogenesis models indicate that periodontal inflammation alters the subgingival environment, promoting a dysbiotic shift. In turn, dysbiosis perpetuates inflammation, creating a circular feed-forward loop. This framework suggests that elevated BI and PI reflect an altered host–biofilm relationship rather than increased plaque quantity alone [32]. This aligns with findings that hyperglycemia triggers heightened inflammatory responses and weakens immune regulation[15]. The strong association between PI and BI highlights the role of biofilm-induced inflammation in periodontal disease. Biomarker research has found increased concentrations of MMP-8, MMP-9, IL-6, and AGE in patients with DM and periodontitis. This supports a molecular link between systemic hyperglycemia and local periodontal inflammation[33,34]. Gregorczyk-Maga, Kania[35] reported changes in the gingival crevicular fluid microbiota and metabolome in people with type 1 DM on continuous insulin therapy. These changes correlated with mild gingival inflammation. The findings support that hyperglycemia and its systemic effects trigger molecular changes. These include microbial dysbiosis, elevated inflammatory mediators, and AGEs, which can affect GP and disease progression[33].

KGW, PI, and BI vary between DM and non-DM individuals. However, many studies, including this one, have not found significant differences in PD and CAL. It may depend on glycemic control, disease duration, and methodological differences[3,36]. These results suggest that inflammatory indices (PI, BI) may be more sensitive predictors of early periodontal risk in individuals with DM than destructive measures such as CAL or PD.

The positive associations observed among PI and BI, PD and CAL, and BI and CAL underscore the interrelationships among inflammatory and destructive periodontal disease. Moreover, age exhibited weak yet significant correlations with KGW, PI, and BI, indicating that aging may progressively affect GP and inflammatory load. These findings align with a previous study indicating cumulative age-related periodontal alterations[1].

There were no significant differences between DM and non-DM subjects by sex. This suggests that systemic metabolic status and behavioral risk factors have a greater impact on periodontal status than sex[9]. This

highlights DM status as a more critical determinant of periodontal health outcomes than sex.

Several limitations should be considered when interpreting these results. The cross-sectional design precludes establishing a cause-and-effect relationship between diabetes status and changes in gingival phenotype or periodontal characteristics. Assessment of the gingival phenotype was limited to anterior teeth, excluding posterior teeth, which may exhibit greater morphological variation. The diabetic cohort comprised only individuals with moderate glycemic control, limiting the generalizability of findings to those with well-controlled or poorly controlled diabetes. Gingival thickness was measured using the probing transparency method, a semi-quantitative approach that may not detect subtle tissue differences despite its clinical utility. Behavioral and socioeconomic factors were not systematically evaluated. Additionally, because the research was conducted at a single center, the findings may not generalize to other populations, underscoring the need for multicenter longitudinal studies.

The clinical implications of these results underscore the need to tailor periodontal care techniques for DM patients by providing tailored oral hygiene measures and treatment. Standardized assessment techniques, including multi-point GT measures, improve accuracy and comparability across studies and clinical settings[30]. Although GT may not be substantially influenced by DM, the persistently high PI and BI values underscore the importance of accurate biofilm management, reinforcement of oral hygiene, and routine professional maintenance to prevent periodontal disease. Future studies should incorporate salivary or GCF biomarkers, such as AGEs, cytokines, and microbiota profiles, that can serve as essential instruments for early identification and customized treatment strategies[37]. The increase in KGW among individuals with DM contradicts established assumptions, raising important questions regarding whether this adaptation serves a protective or pathological role. This observation underscores the need for a prospective longitudinal study to further investigate its implications.

## Conclusion

DM clearly affects GP and periodontal health, primarily increasing KGW, PI, and BI. GT, PD, and CAL appear to be less affected under moderate glycemic control. These findings underscore the need for inflammatory control and thorough oral hygiene in individuals with DM. Future research should explore whether increased KGW is a protective adaptation or a pathogenic response to metabolic stress.

## References


1. Kinane DF, Stathopoulou PG, Papapanou PN. Periodontal diseases. Nat Rev Dis Primers. 2017;3(1):1-14.
2. Zardawi F, Gul S, Abdulkareem A, Sha A and Yates J. Association between periodontal disease and atherosclerotic cardiovascular diseases: revisited. Front. Cardiovasc. Med. 2021; 7:625579.
3. 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.
4. Jepsen S, Caton JG, Albandar JM, Bissada NF, Bouchard P, Cortellini P, et al. Periodontal manifestations of systemic diseases and developmental and acquired conditions: Consensus report of workgroup 3 of the 2017 world workshop on the classification of periodontal and peri-implant diseases and conditions. J Clin Periodontol. 2018;45: S219-S229.
5. De Rouck T, Eghbali R, Collys K, De Bruyn H, Cosyn J. The gingival biotype revisited: transparency of the periodontal probe through the gingival margin as a method to discriminate thin from thick gingiva. J Clin Periodontol. 2009: 36(5):428-33.
6. Kan JY, Morimoto T, Rungcharassaeng K, Roe P, Smith DH. Gingival biotype assessment in the esthetic zone: visual versus direct measurement. Int J Periodontics Restorative Dent. 2010;30 (3)237-43.
7. Fu JH, Yeh CY, Chan HL, Tatarakis N, Leong DJ, Wang HL. Tissue biotype and its relation to the underlying bone morphology. J Periodontol. 2010;81(4):569-74.
8. Wennström J, Lindhe J. Role of attached gingiva for maintenance of periodontal health: healing following excisional and grafting procedures in dogs. J Clin Periodontol. 1983;10(2):206-21.
9. Kim DM, Bassir SH, Nguyen TT. Effect of gingival phenotype on the maintenance of periodontal health: An American Academy of Periodontology best evidence review. J. Periodontol. 2020;91(3):311-38.
10. Lee SP, Kim TI, Kim HK, Shon WJ, Park YS. Discriminant analysis for the thin periodontal biotype based on the data acquired from three-dimensional virtual models of Korean young adults. J Periodontol. 2013;84(11):1638-45.
11. Bednarz W. The thickness of periodontal soft tissue ultrasonic examination—current possibilities and perspectives. Dent Med Probl. 2011;48(3):303-10.

12. Fu JH, Lee A, Wang HL. Influence of tissue biotype on implant esthetics. Int J Oral Maxillofac Implants. 2011;26(3):499-508.
13. Tofiq RK, Hamad AM. Assessment of Gingival Phenotype in Type 2 Diabetic Patients in Sulaimani City. Sulaimani Dent J. 2023;10 (1):11-19.
14. Lalla E, Papapanou PN. Diabetes mellitus and periodontitis: a tale of two common interrelated diseases. Nat Rev Endocrinol. 2011;7(12):738-748.
15. Polak D, Shapira L. An update on the evidence for pathogenic mechanisms that may link periodontitis and diabetes. J Clin Periodontol. 2018;45(2):150-66.
16. Pashova-Tasseva Z, Mlachkova A, Tosheva E. Impact of gingival phenotype on the periodontal disease. Folia Med. 2023;65(3):468-75.
17. Moosa Y, Samaranayake L, Pisarnturakit PP. The gingival phenotypes and related clinical periodontal parameters in a cohort of Pakistani young adults. Heliyon. 2024;10(2):e24219.
18. Alasiri MM, Almalki A, Alotaibi S, Alshehri A, Alkhuraiji AA, Thomas JT. Association between gingival phenotype and periodontal disease severity-a comparative longitudinal study among patients undergoing fixed orthodontic therapy and Invisalign treatment. Healthcare (Basel). 2024;12(6):656.
19. Nagate RR, Chaturvedi S, Al-Ahmari MMM, Al-Qarni MA, Gokhale ST, Ahmed AR, et al. Importance of periodontal phenotype in periodontics and restorative dentistry: a systematic review. BMC Oral Health. 2024;24(1):1-10.
20. Tonetti MS, Greenwell H, Kornman KS. Staging and grading of periodontitis: Framework and proposal of a new classification and case definition. J Periodontol. 2018;89(12):S159-S72.
21. Ainamo J, Bay I. Problems and proposals for recording gingivitis and plaque. Int Dent J. 1975;25(4):229-25.
22. Lang NP, Löe H. The relationship between the width of keratinized gingiva and gingival health. J Periodontol. 1972;43(10):623-27.
23. Kloukos D, Koukos G, Doulis I, Sculean A, Stavropoulos A, Katsaros C. Gingival thickness assessment at the mandibular incisors with four methods: a cross-sectional study. J Periodontol. 2018; 89(11):1300-09.
24. Bland JM, Altman D. Statistical methods for assessing agreement between two methods of clinical measurement. Lancet. 1986;327(8476):307-310.
25. Mealey BL, Oates TW. Diabetes mellitus and periodontal diseases. J Periodontol. 2006;77(8):1289-303.
26. Botero JE, Yepes FL, Roldan N, Castrillon CA, Hincapie JP, Ochoa SP, et al. Tooth and periodontal clinical attachment loss are associated with hyperglycemia in patients with diabetes. J Periodontol. 2012;83(10):1245-50.
27. Păunică I, Giurgiu M, Dumitriu AS, Păunică S, Pantea Stoian AM, Martu M.A, et al. The bidirectional relationship between periodontal disease and diabetes mellitus—a review. Diagn. 2023;13(4):681.
28. Zhang Z, Ji C, Wang D, Wang M, Song D, Xu X, Zhang D. The burden of diabetes on the soft tissue seal surrounding the dental implants. Front Physiol. 2023;14:1136973.
29. Yildirim Bolat S, Lutfioglu M. Evaluation of gingival phenotype: the role of gingival thickness measurements from different vertical gingival levels. Clin Oral Investig. 2025;29(1):87.
30. Barootchi S, Tavelli L, Di Gianfilippo R, Shedden K, Oh TJ, Rasperini G. et al. Soft tissue phenotype modification predicts gingival margin long-term (10-year) stability: longitudinal analysis of six randomized clinical trials. J Clin Periodontol. 2022;49(7):672-83.
31. Abdulkareem AA, Al-Taweel FB, Al-Sharqi AJB, Gul SS, Sha A, Chapple ILC. Current concepts in the pathogenesis of periodontitis: from symbiosis to dysbiosis. J Oral Microbiol. 2023;15(1):2197779.
32. Chen J, Wang H, Bu S, Cheng X, Hu X, Shen M, Zhuang H. Alterations in subgingival microbiome and advanced glycation end-products levels in periodontitis with and without type 1 diabetes mellitus: a cross-sectional study. BMC Oral Health. 2024;24(1):1344.
33. Ebersole JL, Kirakodu SS, Zhang XD, Dawson III D, Miller CS. Salivary features of periodontitis and gingivitis in type 2 diabetes mellitus. Sci Rep. 2024;14(1):30649.
34. Gregorczyk-Maga I, Kania M, Dąbrowska M, Samborowska E, Żeber-Lubecka N, Kulecka M, Klupa T. The interplay between the gingival crevicular fluid microbiome and metabolomic profile in intensively treated people with type 1 diabetes, combined metagenomic/metabolomic approach, cross-sectional study. Front Endocrinol. 2024;14:1332406.

35. Xiang Dd, Sun Yx, Jiao C, Guo Yq, Fei Yx, Ren Bq, et al. Diabetes and periodontitis: the role of a high-glucose microenvironment in periodontal tissue cells and corresponding therapeutic strategies. Stem cell res ther. 2025;16(1),366.
**36.** Sanz M, Ceriello A, Buysschaert M, Chapple I, Demmer RT, Graziani F, et al. Scientific evidence on the links between periodontal diseases and diabetes: Consensus report and guidelines of the joint workshop on periodontal diseases and diabetes by the International Diabetes Federation and the European Federation of Periodontology. Diabetes Res Clin Pract. 2018;137:231-41.