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Serum 25-Hydroxycholecalciferol Levels in Type 2 Diabetic Patients with Periodontitis: A Socioeconomic and Biochemical Analysis

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ABSTRACT

Only a limited number of investigations have focused on how the coexistence of type 2 diabetes mellitus (T2DM) and periodontitis (PD) influences serum Vitamin D concentration, especially in developing nations such as India. This aspect requires more scientific attention. The goal of this study was to explore how serum Vitamin D values relate to periodontal disease in individuals affected by T2DM, and to determine whether socioeconomic and demographic factors have any bearing on Vitamin D concentration or the degree of periodontal breakdown in these patients. This cross-sectional hospital-based analysis gathered participants' medical, dental, and nutritional records and assessed their socioeconomic status (SES). Clinical indices such as the plaque index (PI), gingival index (GI), gingival bleeding sites, probing pocket depth (PPD), and clinical attachment level (CAL) were evaluated across three groups: 1. Individuals with generalized Stage III Grade B PD and T2DM (n = 35); 2. Patients with generalized Stage III Grade B PD only (n = 35); Healthy control participants (n = 35). Laboratory analysis included testing for serum 25-hydroxyvitamin D (25[OH]D) and hemoglobin A1C (HbA1c) levels. Comparisons of periodontal and biochemical markers among the groups were made using one-way ANOVA. Correlations between 25(OH)D, clinical variables, and SES were determined through Pearson's correlation and linear regression models. Participants with Stage III Grade B PD along with T2DM exhibited the lowest mean 25(OH)D concentration (13.54 ± 3.31 ng/mL). A strong negative relationship ($P < 0.01$) was found between serum 25(OH)D levels and periodontal indices — PI (−0.442), PPD (−0.474), CAL (−0.459), gingival bleeding (−0.354), and GI (−0.346). Regression outcomes revealed that increased periodontal damage (PI, GI, PPD, CAL) and higher HbA1c were linked with diminished 25(OH)D values. Conversely, Vitamin D status showed no meaningful correlation with socioeconomic or demographic characteristics. The findings indicate that Vitamin D levels decline when PD and T2DM coexist or even when PD occurs alone. However, SES did not significantly influence Vitamin D concentration in either group.

Keywords: Vitamin D, Type 2 Diabetes Mellitus, Periodontitis

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Introduction

Periodontitis (PD) is a chronic inflammatory condition caused by bacterial infection that progressively destroys the tooth-supporting tissues [1]. It remains one of the most frequently reported oral health issues worldwide [1]. Research consistently supports its association with multiple systemic diseases, including type 2 diabetes mellitus (T2DM), cardiovascular and respiratory disorders, and certain malignancies [2].

Among these, diabetes mellitus (DM) has the most pronounced connection, with poorly controlled cases exhibiting a two- to three-fold higher susceptibility to PD [3]. Globally, DM affects nearly 9.3% of the population,

making it a major metabolic health concern [4]. The interaction between PD and DM is bidirectional—PD is both a consequence of diabetes and a factor that can exacerbate glycemic dysregulation and systemic complications [3].

Vitamin D plays a central role not only in bone metabolism but also in immune modulation and cellular homeostasis [5]. The level of 25-hydroxyvitamin D (25[OH]D) in the bloodstream serves as a key indicator of Vitamin D sufficiency [5]. Recent investigations have suggested a link between Vitamin D deficiency and both PD and T2DM [6–8].

The immune response to bacterial pathogens in PD involves both innate and adaptive pathways, partly regulated by Vitamin D.[9] Since PD is an ongoing inflammatory process driven by microbial imbalance, chronic disease activity may contribute to lower Vitamin D levels and endocrine alterations [9]. Additionally, inadequate Vitamin D impairs insulin secretion and contributes to insulin resistance [10]. Genetic factors, such as Vitamin D receptor polymorphisms, may further explain this metabolic and immune interrelationship [10].

When T2DM and PD occur together, the inflammatory burden may intensify, potentially worsening both diseases. Therefore, individuals suffering from both conditions are likely to exhibit markedly reduced Vitamin D levels compared with those having PD alone or no systemic disease. Although several studies have assessed the Vitamin D–PD association, there is a scarcity of data from developing countries like India, where dietary habits, sunlight exposure, and socioeconomic factors may influence Vitamin D status. Hence, this study aimed to determine serum Vitamin D concentrations in patients with PD and T2DM and evaluate how demographic and socioeconomic variables affect these levels.

The state of Chhattisgarh currently encounters numerous public health challenges, among which improving the oral and dental well-being of its citizens remains a priority [11]. Considerable variations exist in the cultural background and socioeconomic standing (SES) of the population, which in turn contribute to differences in oral health outcomes [12]. Limited educational attainment, restricted income levels, and inadequate access to healthcare and public infrastructure in several regions of Chhattisgarh may further contribute to reduced Vitamin D concentrations among its residents [12].

To date, no prior investigation has been undertaken in this geographical region to examine serum Vitamin D concentrations in individuals presenting with both periodontitis (PD) and type 2 diabetes mellitus (T2DM). Therefore, this research sought to determine the relationship between Vitamin D status and PD among T2DM patients, and to explore the influence of demographic and socioeconomic determinants on Vitamin D concentration and the degree of periodontal destruction in this group.

Materials and Methods

This investigation adopted a cross-sectional hospital-based design. Participants were recruited from the outpatient department of Periodontics after obtaining written informed consent. The study complied with the principles of the Helsinki Declaration (2013) and received approval from the Institutional Ethics Committee, Government Dental College and Hospital, Raipur (IEC: ECR/6488/GDC/CG/2019).

Eligible participants were 35–55 years old, residents of Chhattisgarh, and possessed a minimum of 20 natural teeth. Subjects were classified into three categories:

1. Individuals with generalized Stage III Grade B PD (PD group),
2. Those diagnosed with generalized Stage III Grade B PD along with T2DM (PD–DM group), and
3. Systemically and periodontally healthy controls.

The diagnostic framework for Stage III Grade B PD followed the 2017 American Academy of Periodontology guidelines [13]. Inclusion of T2DM patients adhered to the 2018 ADA criteria: HbA1c ≥ 6.5% (48 mmol/mol) [14].

Participants with systemic diseases influencing Vitamin D metabolism (such as bone pathologies, cancer, cardiovascular or renal disorders), habitual tobacco users, pregnant or lactating women, postmenopausal females, individuals with recent periodontal therapy, or those taking antibiotics or medications affecting periodontal status within the preceding six months were excluded.

The sample size was calculated using data from Joseph *et al.*, ensuring 80% statistical power and a 95% confidence level. The software G*Power (Version 3.1.9.2, Heinrich Heine University, Düsseldorf, Germany) yielded a minimum requirement of 33 subjects per group. After rounding, 35 participants were included in each group, totaling 105 participants.

Prior to clinical evaluation, participants underwent detailed medical, dental, and dietary interviews. Information regarding age, sex, residence, outdoor activity, sun exposure (hours/day), dietary type, physical exercise, dental visitation pattern, family structure, and housing condition was obtained. Body mass index (BMI) was computed as weight (kg)/height² (m²). Socioeconomic status was determined using the Modified Kuppuswamy Scale [16], which assesses education and occupation of the family head, and total family income. Scores (ranging from 3–29) classified families into upper, upper-middle, lower-middle, upper-lower, and lower socioeconomic categories.

Biochemical analysis

Venous blood (5 mL) was collected aseptically from the antecubital fossa into sterile EDTA vials by a trained laboratory assistant and transported for biochemical testing. Serum 25-hydroxyvitamin D (25[OH]D) was quantified using chemiluminescence immunoassay (Advia Centaur, Siemens, Germany), while HbA1c was measured by fully automated HPLC (Bio-Rad Variant II Turbo, Montreal, Quebec, Canada). All analyses were performed by a qualified laboratory technician.

Clinical examination

A single calibrated examiner recorded all clinical periodontal parameters, including plaque index (PI), gingival index (GI), bleeding on probing (BOP), probing pocket depth (PPD), and clinical attachment level (CAL). All teeth except third molars were evaluated using a mouth mirror and a UNC-15 periodontal probe (Hu-Friedy Mfg. Co., Chicago, IL, USA). BOP was determined using the Ainamo and Bay index [17].

PI and GI were documented at mesiobuccal, buccal, distobuccal, and entire lingual surfaces, following the scoring systems by Silness & Löe (for PI) and Löe & Silness (for GI) [18]. PPD and CAL were recorded at six points per tooth: mesiobuccal, buccal, distobuccal, distolingual, lingual, and mesiolingual, and rounded to the nearest millimeter.

Prior to data collection, intra-examiner calibration was performed on five non-study PD patients. Measurements at six sites within one random quadrant were repeated after 24 hours. The intraclass correlation coefficient for PPD was 0.85, and 0.80 for CAL, confirming acceptable measurement consistency.

Statistical analysis

All collected information was first organized in a Microsoft Excel spreadsheet, followed by descriptive statistics to summarize demographic, clinical, and biochemical data. The results were expressed as mean ± standard deviation (SD) for continuous variables, and as percentages and ratios for categorical ones. The Chi-square test was applied to assess relationships among categorical factors.

For continuous measurements—including age, BMI, clinical indices (PI, GI, BOP, PPD, CAL), and biochemical markers (HbA1c and 25[OH]D)—comparisons across the three study groups were carried out using either a one-way ANOVA or the Kruskal–Wallis test, depending on whether data satisfied normality assumptions. Post hoc Bonferroni correction was employed to identify significant pairwise differences between the groups.

Associations among clinical scores, 25(OH)D concentration, and socioeconomic level (SES) were determined using Pearson's correlation coefficient. Additionally, linear regression analysis was conducted to examine the influence of periodontal indices and HbA1c levels on 25(OH)D concentration, as well as to explore the relationship between 25(OH)D and demographic variables, including SES. A P value below 0.05 was considered statistically meaningful. All statistical computations were executed using IBM SPSS Statistics software version 20.0 (IBM Corp., Armonk, NY, USA).

Results and Discussion

The investigation enrolled 105 individuals, distributed equally among three cohorts: 35 participants with generalized Stage III Grade B periodontitis (PD), 35 with PD combined with type 2 diabetes mellitus (PD–DM), and 35 periodontally and systemically healthy controls.

As shown in **Table 1**, the average BMI values recorded for the PD, PD–DM, and control groups were 20.52 ± 1.53, 21.95 ± 1.59, and 20.63 ± 1.36, respectively. The intergroup variation in BMI was statistically significant ($P < 0.001$). In contrast, no significant differences were detected for other demographic attributes such as age, gender, place of residence, sun exposure duration, outdoor activity, exercise level, dietary pattern, dental visit frequency, family setup, housing condition, and SES.

Table 1. Demographic details of study participants

Characteristic	Healthy Control	Periodontitis (PD)	Periodontitis with Diabetes (PD-DM)	P-Value*
Age (years), Mean±SD	40.0±6.49	43.1±8.25	48.5±9.5	0.19
Gender Breakdown (%)†				0.29
Men	22 (62.8)	17 (48.6)	23 (71.8)	
Women	13 (37.2)	18 (51.4)	12 (28.2)	
Residence, n (%)†				0.06
Rural Area	4 (18)	13 (37.1)	2 (5.8)	
Suburban Area	16 (38)	10 (28.6)	11 (31.4)	
Urban Area	15 (44)	12 (34.3)	22 (62.8)	
Outdoor Engagement, n (%)†				0.54
1–5 Times/Week	7 (20)	11 (31.4)	9 (25.7)	
>5 Times/Week	28 (80)	24 (68.6)	26 (74.3)	
Sunlight Exposure (h/day), Mean±SD#	1.53±0.83	1.47±1.10	1.30±0.59	0.52
Physical Activity Level, n (%)†				0.36
Consistent	10 (28)	8 (22.9)	5 (14.3)	
Occasional	25 (72)	27 (77.1)	30 (85.7)	
Body Mass Index (kg/m²), Mean±SD#	20.63±1.36	20.52±1.53	21.95±1.59	0.00**
Dietary Pattern, n (%)†				0.26
Vegetarian Diet	26 (74.2)	22 (62.8)	28 (80)	
Mixed Diet	9 (25.8)	13 (37.2)	7 (20)	
Dental Care Frequency, n (%)†				0.54
Only When Issues Arise	32 (91.4)	32 (91.4)	34 (97.1)	
Every 6 Months	3 (8.6)	3 (8.6)	1 (2.9)	
Household Structure, n (%)†				0.27
Extended Family	11 (31.5)	12 (34.3)	7 (20)	
Single Family	24 (68.5)	23 (65.7)	28 (80)	
Housing Type, n (%)†				0.87
Permanent Structure	22 (62.8)	23 (65.7)	22 (62.8)	
Apartment Building	10 (28.5)	11 (31.4)	10 (28.5)	
Standalone House	3 (8.7)	1 (2.9)	3 (8.7)	
Socioeconomic Status, n (%)†				0.40
High Income	0	6 (17.1)	0	
Upper Middle Income	21 (62)	23 (65.7)	29 (82.9)	
Lower Middle Income	14 (38)	6 (17.1)	6 (17.1)	
Upper Low Income	0	0	0	
Low Income	0	0	0	

**P < 0.05 = significant;* *P < 0.01 = highly significant; †Chi-square test; #One-way ANOVA. PD – generalized Stage III Grade B periodontitis; PD-DM – generalized Stage III Grade B periodontitis with T2DM; SD – standard deviation; n – number of participants; BMI – body mass index; SES – socioeconomic status; kg/m² – kilogram per square meter.

All periodontal measurements—PI, GI, BOP, PPD, and CAL—were markedly higher among both diseased groups when compared with healthy individuals (P < 0.001). Between the two case categories, the PD–DM group exhibited more severe periodontal destruction (P = 0.04 for BOP; P < 0.001 for other indices) **(Table 2)**.

Table 2. Comparison of periodontal variables among study groups

Gum Health Indicators	Periodontitis (PD), Mean (SD)	Periodontitis with Diabetes (PD-DM), Mean (SD)	Healthy Control, Mean (SD)	P-Value*	P1‡	P2‡	P3‡
Gum Inflammation Score	2.10 (0.21)	2.30 (0.28)	1.88 (0.29)	0.000**	0.003**	0.006**	0.000**

Plaque Accumulation	2.10 (1.00)	2.77 (1.21)	0.50 (0.15)	0.000**	0.000**	0.000**	0.000**
Bleeding on Probing	0.85 (0.21)	0.96 (0.23)	0.06 (0.01)	0.000**	0.000**	0.040*	0.000**
Pocket Depth (mm)	6.41 (0.69)	6.84 (0.53)	2.22 (0.30)	0.000**	0.000**	0.004**	0.000**
Clinical Attachment Loss (mm)	4.28 (0.34)	4.74 (0.78)	0.00 (0.00)	0.000**	0.000**	0.000**	0.000**

**P < 0.05 = significant;* *P < 0.01 = highly significant; ‡Bonferroni post hoc correction applied. PD – generalized Stage III Grade B periodontitis; PD-DM – generalized Stage III Grade B periodontitis with T2DM; GI – gingival index; PI – plaque index; BOP – bleeding on probing; PPD – probing pocket depth; CAL – clinical attachment level; mm – millimeters; SD – standard deviation.

Table 3 displays that the mean HbA1c values were 5.50 ± 0.45 in PD cases, 8.67 ± 1.84 in PD–DM cases, and 5.62 ± 0.49 in controls, demonstrating a highly significant distinction among the groups (P < 0.0001). Pairwise evaluation indicated substantial differences between both diseased groups and healthy participants (P < 0.001 each) and also between the two case groups (P < 0.0001).

Regarding serum 25(OH)D, the lowest concentration was seen in PD–DM participants (13.54 ± 3.31 ng/mL), followed by the PD group (15.72 ± 6.60 ng/mL), while the control group displayed the highest mean level (22.0 ± 9.26 ng/mL). The differences across groups were significant (P < 0.001), though post hoc analysis did not identify a statistically relevant difference between PD and PD–DM cases. The occurrence of Vitamin D deficiency was greatest in PD–DM subjects, next in PD-only patients, and least among healthy controls (P < 0.001).

Table 3. Comparison of biochemical indicators between study groups

Biological Markers	Periodontitis (PD), Mean (SD)	Periodontitis with Diabetes (PD-DM), Mean (SD)	Non-Diseased Control, Mean (SD)	P-Value*	P1‡	P2‡	P3‡
Glycated Hemoglobin (%)	5.50 (0.45)	8.67 (1.84)	5.62 (0.49)	0.000**	1.000	0.000**	0.000**
25-Hydroxyvitamin D (ng/mL)	15.72 (6.60)	13.54 (3.31)	22.0 (9.26)	0.000**	0.000**	0.56	0.000**
Proportion (%) of Individuals with Vitamin D Insufficiency	26 (74.2)	35 (100)	17 (48.5)	0.000**	-	-	-

*P < 0.05 = significant; *P < 0.01 = highly significant; ‡Bonferroni post hoc correction used. PD – generalized Stage III Grade B periodontitis; PD-DM – generalized Stage III Grade B periodontitis with T2DM; SD – standard deviation; HbA1c – glycated hemoglobin; 25(OH)D – 25-hydroxyvitamin D; ng/mL – nanograms per milliliter.

As depicted in **Table 4**, 25(OH)D demonstrated a moderate inverse correlation (P < 0.01) with PI, PPD, and CAL, while GI and BOP revealed a weaker but statistically significant negative correlation. Within individual groups, these associations did not reach statistical significance. The relationship between SES and periodontal parameters was also not significant across any of the groups **(Table 5)**.

Table 4. Association between 25-hydroxyvitamin D and clinical indices

Group Analysis	25-Hydroxyvitamin D	Bleeding on Probing	Gum Inflammation Score	Plaque Accumulation	Pocket Depth	Clinical Attachment Loss
All Groups						
25-Hydroxyvitamin D	Pearson Correlation	1	-0.354**	-0.346**	-0.459**	-0.442**
	Significance (2-tailed)		0.000	0.000	0.000	0.000
	Sample Size (n)	105	105	105	105	105
Non-Diseased Control Group						
25-Hydroxyvitamin D	Pearson Correlation	1	-0.098	-0.309	-0.176	-0.086
	Significance (2-tailed)		0.574	0.071	0.313	0.625
	Sample Size (n)	35	35	35	35	35

Periodontitis (PD) Group						
25-Hydroxyvitamin D	Pearson Correlation	1	0.006	0.263	-0.211	0.242
	Significance (2-tailed)		0.971	0.126	0.224	0.162
	Sample Size (n)	35	35	35	35	35
Periodontitis with Diabetes (PD-DM) Group						
25-Hydroxyvitamin D	Pearson Correlation	1	-0.020	-0.236	0.105	0.015
	Significance (2-tailed)		0.908	0.173	0.550	0.932
	Sample Size (n)	35	35	35	35	35

P < 0.01 = statistically significant (two-tailed); negative (−) sign indicates inverse relationship. PD – generalized Stage III Grade B periodontitis; PD-DM – generalized Stage III Grade B periodontitis with T2DM; 25(OH)D – 25-hydroxyvitamin D; PI – plaque index; GI – gingival index; BOP – bleeding on probing; PPD – probing pocket depth; CAL – clinical attachment level; n – number of participants.

Table 5. Relationship between socioeconomic class and periodontal indicators

Socioeconomic Level	Bleeding on Probing	Gum Inflammation Index	Pocket Depth	Clinical Attachment Loss	Plaque Level	Socioeconomic Level
Pearson's Coefficient	0.172	−0.105	0.176	0.296	−0.071	1
Significance (Two-Tailed)	0.324	0.549	0.311	0.084	0.684	
Sample Size (n)	35	35	35	35	35	35

SES – Socioeconomic status; GI – Gingival index; PI – Plaque index; BOP – Bleeding on probing; PPD – Probing pocket depth; CAL – Clinical attachment level; n – number of participants; P<0.05 indicates a statistically significant difference; P – p value

According to the regression outcomes, lower serum 25(OH)D levels were observed when periodontal measures such as PI, GI, PPD, and CAL increased (25[OH]D = 25.793 – 1.247[GI] − 1.580[PPD] − 2.317[CAL] − 0.436[PI]) **(Table 6)**. Additionally, the analysis revealed that a rise in HbA1c was linked with a fall in Vitamin D levels (25[OH]D = 25.394 – 1.247[HbA1c]) **(Table 7)**. Linear regression also showed that socioeconomic and other demographic variables had no measurable influence on serum Vitamin D among study groups **(Table 8)**.

Table 6. Linear regression data showing association between Vitamin D and periodontal indicators

Variable	Unstandardized Coefficients		t	Significance	95.0% CI for B
	B	SE			
Intercept	25.793		5.668	4.550	0.000
Gingival Index (GI)	−3.236		2.636	−1.227	0.223
Plaque Index (PI)	−0.436		0.637	−0.685	0.495
Probing Pocket Depth (PPD)	−1.580		1.326	0.962	0.339
Clinical Attachment Loss (CAL)	−2.317		1.459	−1.588	0.115

P<0.05 = statistically meaningful. GI – Gingival index; PI – Plaque index; PPD – Probing pocket depth; CAL – Clinical attachment level; CI – Confidence interval; t – t value; P – p value; B – Regression coefficient; SE – Standard error

Table 7. Linear regression between Vitamin D and glycated hemoglobin

Variable	Raw Coefficients	Standard Error	t-Statistic	P-Value	95% Confidence Interval for Coefficient
	Coefficient	SE			Lower Limit
Intercept	25.394	2.682	9.470	0.000	20.076
Glycated Hemoglobin	−1.247	0.391	−3.186	0.002	−2.023

P<0.05 = statistically meaningful. HbA1c – Glycated hemoglobin; CI – Confidence interval; t – t value; P – p value; B – Regression coefficient; SE – Standard error

Table 8. Linear regression between Vitamin D and demographic variables

Predictor	Raw Coefficients	Standard Error	Standardized Coefficient (β)	t-Statistic	P-Value
	Coefficient	SE			
Intercept	10.931	10.895		1.003	0.318
Household Structure	−1.506	1.893	−0.088	−0.796	0.428
Residence Type	−0.312	1.422	−0.025	−0.219	0.827
Geographic Area	−0.048	1.135	−0.005	−0.042	0.966
Dental Care Frequency	2.142	1.653	0.139	1.296	0.198
Outdoor Engagement	−0.067	0.498	−0.015	−0.135	0.893
Physical Activity	1.907	2.127	0.095	0.896	0.372
Nutritional Pattern	1.399	0.949	0.163	1.474	0.144
Socioeconomic Status	1.41	0.872	0.764	1.071	0.621

P<0.05 = statistically meaningful. SES – Socioeconomic status; t – t value; P – p value; B – Regression coefficient; SE – Standard error

Extensive literature confirms the two-way interaction between type 2 diabetes mellitus (T2DM) and periodontal disease (PD), as both conditions intensify each other's detrimental outcomes when present together [3, 15]. While T2DM elevates the probability of developing PD, periodontal inflammation may equally disrupt glucose balance [3]. The precise biological pathways—such as those involving cytokine signaling, immune modulation, and neutrophil activity—remain partly unclear, though they appear central to this interconnection [3]. Moreover, prior investigations have pointed to a probable connection between PD, diabetes, and diminished Vitamin D status [15]. In this research, the lowest Vitamin D concentrations (25[OH]D) were found in participants affected by both PD and T2DM, followed by those with PD alone, and the highest levels occurred in healthy controls. A clear negative association existed between serum 25(OH)D and periodontal parameters. However, demographic and socioeconomic conditions had no notable relationship with either Vitamin D status or periodontal health.

Across all three participant categories, no substantial variation was seen in general features including gender, age, residence type, outdoor habits, exposure to sunlight, diet, exercise frequency, dental hygiene visits, or living conditions. This uniformity ensured comparability and controlled experimental conditions.

The mean body mass index for all participants was within a healthy range, signifying absence of overweight cases. Although the intergroup BMI difference achieved statistical significance, the actual numerical variation was small and biologically negligible.

No considerable difference in HbA1c levels emerged between subjects with PD and healthy individuals, similar to the observations by Kebede *et al.* and Wahi *et al.* [19, 20] A slightly lower HbA1c level among the PD group might relate to their marginally reduced BMI [21]. Nevertheless, both cohorts exhibited tendencies characteristic of prediabetes. Declining β-cell efficiency, due to genetic and metabolic pressures, may account for such patterns [22].

The data further indicated that indicators of periodontal tissue breakdown (GI, PI, BOP, PPD, CAL) and HbA1c levels were highest in those with concurrent PD and diabetes. Chronic infections of periodontal origin, primarily due to Gram-negative bacteria, can amplify systemic inflammatory processes, intensify insulin resistance, and thereby worsen diabetic conditions [3]. Persistent inflammation and hyperglycemia contribute to further deterioration of metabolic control and an elevated risk of diabetic complications [3]. These findings reaffirm the mutual influence and cyclical aggravation between PD and T2DM noted in previous scholarly research [2,3].

The study findings indicated that participants with both periodontal disease and diabetes (PD-DM) had the lowest serum 25(OH)D concentrations, followed by those with PD alone, while healthy individuals showed the highest levels. The consistent differences between and within these groups confirmed the study's working premise. Across all groups, including the control group, mean serum 25(OH)D levels were below the standard reference range. Although the healthy group exhibited *insufficient* Vitamin D levels, both PD and PD-DM groups showed *deficiency*. Despite most participants reporting adequate sun exposure and regular outdoor activity, specific

genetic variants—such as overexpression of 25(OH)D-24-hydroxylase that degrades Vitamin D into inactive metabolites—might account for low levels even in healthy individuals [23]. Additional factors, including darker skin pigmentation and vegetarian diets deficient in calcium but rich in phytates and oxalates, can further deplete Vitamin D [23]. The percentage of Vitamin D deficiency was highest in PD-DM participants, followed by PD-only and healthy subjects. Moreover, all periodontal parameters showed a negative association with 25(OH)D levels. Regression analysis demonstrated that higher values of PI, GI, PPD, and CAL were linked to lower serum 25(OH)D. These observations align with earlier studies by Joseph *et al.*, Wang *et al.*, and Agrawal *et al.*, suggesting a potential link between PD, diabetes, and Vitamin D deficiency [15, 24, 25]. The markedly low Vitamin D levels in PD-DM patients may result from chronic inflammation and cytokine overactivity in these individuals.

Persistent periodontal inflammation might also lead to endocrine dysfunction involving Vitamin D metabolism [9]. Intracellular bacteria are thought to influence cytokine production, and these cytokines can inhibit transcription of the 1,25-dihydroxyvitamin-D (1,25[OH]$_2$D)/Vitamin D receptor (VDR) gene in macrophages.[9] To counter this, the body synthesizes more 1,25(OH)$_2$D to activate VDR and induce antimicrobial peptides such as cathelicidin and beta-defensins, which combat bacterial invasion [9]. This rapid conversion process depletes circulating 25(OH)D, resulting in reduced serum levels.[9] These findings correspond with the reports of Dietrich *et al.*, Bhargava *et al.*, and Laky *et al.*, who established a connection between PD and reduced Vitamin D [6, 26, 27].

The current results also reinforce previous studies linking Vitamin D deficiency to Type 2 diabetes [28–30]. Experimental and clinical data associate low Vitamin D with impaired insulin secretion, heightened insulin resistance, and disrupted insulin signaling pathways [30]. Since reduced Vitamin D correlates with chronic inflammation and oxidative stress, the connection between low Vitamin D and insulin resistance may stem from inflammatory cascades and epigenetic modulation of metabolic genes [31].

In this analysis, the plaque index (PI) was significantly higher in PD-DM participants compared with the PD-only group, suggesting heavier bacterial accumulation and increased cytokine response, which may obscure diabetes-related differences. The absence of a group with diabetes but no PD and the lack of PI matching between groups were study limitations. The study also did not account for diabetes duration. Nevertheless, linear regression demonstrated that elevated HbA1c values were consistently associated with lower 25(OH)D, even after adjusting for confounders, confirming a persistent negative relationship.

It remains uncertain whether Vitamin D deficiency is a cause or a result of such health conditions [32]. Mendelian randomization analysis by Ye *et al.* also indicated that the association between 25(OH)D and T2DM is unlikely to be causal [33]. Likewise, it is unclear whether Vitamin D deficiency predisposes individuals to PD or occurs as a consequence of the disease. Some clinical investigations suggest that Vitamin D supplementation may improve insulin sensitivity [34, 35] and short-term interventions indicate potential benefits for periodontal stability when combined with calcium [36, 37]. However, due to weak and inconsistent evidence, general Vitamin D supplementation cannot yet be universally recommended.

Regarding socioeconomic status (SES), no significant relationship with periodontal indicators was observed, contradicting earlier findings by Kim *et al.*, Lee and Han, and Javed *et al.* [38–40]. Regression results confirmed that neither SES nor demographic factors were associated with Vitamin D concentrations, which diverges from studies by Puri *et al.*, Mechenro *et al.*, and Lin *et al.* [41–43] Most participants across all groups were from upper-middle-class backgrounds, with no individuals from lower or upper socioeconomic tiers. This suggests that people from these classes might underutilize government dental care services due to either limited awareness or accessibility issues. The relatively small sample size may also have influenced this distribution, highlighting the need for broader studies involving rural and lower-income populations to better evaluate SES effects on Vitamin D levels.

Other study constraints included the inability to adjust for seasonal variations in Vitamin D, as sampling occurred year-round, and the limited generalizability of findings to other populations beyond Chhattisgarh, India. Additionally, post-treatment Vitamin D evaluation was not performed, and the cross-sectional design prevented establishing causality or underlying mechanisms.

Conclusion

Within the study's boundaries, it can be inferred that serum Vitamin D concentrations decline in the presence of both PD and T2DM or PD alone. The findings underscore the importance of enhancing public and clinical awareness regarding prevention, early diagnosis, and management of periodontal diseases. Nevertheless, no conclusive association was found between socioeconomic class and Vitamin D levels in subjects with PD and T2DM, suggesting that more extensive future studies are necessary.

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