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Original Article

Vitamin D Status and Its Relationship with Glycemic Control in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Background: Hypovitaminosis D is common in people with type 2 diabetes mellitus (T2DM) and has been linked to impaired insulin secretion, insulin resistance, and chronic low-grade inflammation.
Objectives: To determine the status of serum 25-hydroxyvitamin D [25(OH)D] and examine its relationship with glycemic indices in patients with T2DM.
Methods: Adults with T2DM (N = 100) underwent assessment of serum 25(OH)D, fasting blood sugar (FBS), postprandial blood sugar (PPBS), and glycated hemoglobin (HbA1c). Vitamin D status was categorized as deficient (<20 ng/mL), insufficient (20–29 ng/mL), or sufficient (≥30 ng/mL). Group comparisons and Pearson correlation were applied.
Results: Vitamin D deficiency was observed in 62% of participants and insufficiency in 23%, with a mean 25(OH)D level of 18.9 ± 7.4 ng/mL. Mean HbA1c was 8.6 ± 1.4%, and 88% of patients had poor glycemic control (HbA1c >7%). HbA1c levels were highest in the deficient group (9.1 ± 1.3%) and lowest in the sufficient group (7.4 ± 0.9%), with significant differences across categories. Serum 25(OH)D showed a negative correlation with HbA1c, FBS, and PPBS.
Conclusion: Lower vitamin D levels were associated with poorer glycemic control among patients with T2DM in this tertiary-care setting.

Keywords: Type 2 diabetes mellitus; vitamin D deficiency; 25-hydroxyvitamin D; HbA1c; fasting blood sugar; postprandial blood sugar.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) continues to expand as a major non-communicable disease burden, driven by urbanization, sedentary behavior, and dietary transitions. Beyond classic risk factors, micronutrient status has gained attention because it can influence metabolic pathways that shape insulin secretion and peripheral glucose uptake. Identifying modifiable biological correlates of glycemic control remains clinically important, especially in settings where long-term complications are common at presentation[1,2].

Vitamin D, traditionally recognized for skeletal health, also acts as a pleiotropic hormone through the vitamin D receptor expressed in pancreatic beta cells, adipocytes, and immune cells. Experimental and clinical observations suggest that vitamin D can influence glucose homeostasis by modulating beta-cell function, insulin sensitivity, and inflammatory signaling.These biological links have encouraged researchers to explore whether low serum 25(OH)D levels track with poorer glycemic indices in established T2DM, and whether deficiency represents an actionable risk marker within routine care[3].

Population studies from different regions frequently report a high prevalence of vitamin D deficiency among adults with dysglycemia. In India, urban cohort data demonstrate substantial hypovitaminosis D across glucose tolerance categories,

including individuals with T2DM [4]. Recent facility-based Indian studies similarly highlight that vitamin D deficiency is common in people with T2DM, reinforcing the need for contextual data from diverse clinical settings [5]. Limited food fortification, indoor work patterns, darker skin pigmentation, and cultural clothing practices can further constrain cutaneous vitamin D synthesis, making deficiency a persistent public health concern.

Several observational analyses show an inverse association between serum 25(OH)D and HbA1c, suggesting that lower vitamin D status accompanies poorer long-term glycemic control [3,6-8]. However, findings are not uniform because glycemic control is influenced by obesity, diet, physical activity, sun exposure, renal function, and medication patterns. Interventional evidence also remains mixed; meta-analyses of vitamin D supplementation in T2DM report variable effects on HbA1c and related indices, often showing modest benefit in deficient participants or short-duration regimens [9-11]. Large trials and smaller randomized studies provide additional nuance, with some reporting limited glycemic change despite improved vitamin D status [12,13], while others show measurable metabolic shifts under specific dosing strategies [14].

Against this background, generating local evidence from routine tertiary-care populations is valuable for clinical decision-making and for designing targeted supplementation strategies. The objectives of the present study were to (i) estimate the distribution of vitamin D status among adults with T2DM attending a tertiary-care hospital, and (ii) evaluate the association and correlation of serum 25(OH)D levels with FBS, PPBS, and HbA1c.

METHODOLOGY

Study design and setting: This hospital-based observational study was conducted in the Department of General Medicine at Mamata General Hospital, Khammam, Telangana, India. Data collection was completed over six months from March 2025 to August 2025.

Participants and eligibility: Adults (≥18 years) with a confirmed diagnosis of T2DM attending outpatient follow-up or admitted for glycemic evaluation were screened. Patients with type 1 diabetes, gestational diabetes, acute severe illness, chronic liver disease, advanced chronic kidney disease, malabsorption syndromes, granulomatous disorders, or current high-dose vitamin D supplementation were excluded to reduce biochemical and clinical confounding.

Sample size and sampling: A sample size of 100 was achieved using a consecutive sampling approach during the study period, aligned with the planned feasibility for laboratory testing and documentation within routine clinical workflow.

Data collection procedures: After informed consent, demographic details (age and sex), clinical history (duration of diabetes), and treatment details were recorded using a structured proforma. Standard clinical evaluation was performed, and venous blood sampling was done under aseptic precautions. Samples for biochemical analysis were processed in the central laboratory as per internal quality control protocols.

Biochemical measurements: Venous blood samples were collected after an overnight fast for FBS and serum 25(OH)D estimation. PPBS was measured two hours after a standard meal as per clinical protocol. HbA1c was measured using standardized laboratory methodology aligned with routine diabetes monitoring. Serum 25(OH)D was estimated using an immunoassay-based method available in the institutional laboratory, with results reported in ng/mL.

Laboratory definitions: Serum 25(OH)D was used as the biomarker for vitamin D status. Participants were classified as vitamin D deficient (<20 ng/mL), insufficient (20–29 ng/mL), or sufficient (≥30 ng/mL), consistent with commonly used clinical thresholds [1,4]. Glycemic control was categorized as good (HbA1c ≤7%) or poor (HbA1c >7%) for analytical grouping.

Statistical analysis: Data were entered into a spreadsheet and analyzed using standard statistical software. Continuous variables are presented as mean ± standard deviation (SD), and categorical variables as frequency and percentage. Group differences were assessed using one-way analysis of variance for continuous outcomes and the chi-square test for categorical outcomes. Pearson correlation coefficients were computed to assess relationships between serum 25(OH)D levels and HbA1c, FBS, and PPBS. All tests were two-tailed, and a p-value <0.05 was considered statistically significant.

Ethical considerations: The study followed ethical principles for biomedical research involving human participants and was conducted after institutional permission. Confidentiality was maintained through coded identifiers, and test results were communicated to treating clinicians for appropriate care and counseling.

Ethics:

The study protocol was approved by the Institutional Ethics Committee of Mamata Medical College. Confidentiality was maintained and participation was voluntary.

RESULTS

A total of 100 patients with T2DM were evaluated. Baseline demographic and clinical characteristics are presented in Table 1.

Table 1. Baseline demographic and clinical characteristics (N = 100)

Variable	Value
Age (years), mean ± SD	54.8 ± 9.6
Male, n (%)	58 (58%)
Female, n (%)	42 (42%)
Duration of diabetes (years), mean ± SD	7.2 ± 4.1

Serum 25(OH)D assessment showed a high prevalence of hypovitaminosis D, with deficiency in nearly two-thirds of participants (Table 2).

Table 2. Distribution of vitamin D status (N = 100)

Vitamin D category	Serum 25(OH)D level (ng/mL)	n (%)
Deficient	<20	62 (62%)
Insufficient	20–29	23 (23%)
Sufficient	≥30	15 (15%)
Overall mean ± SD (ng/mL)	—	18.9 ± 7.4

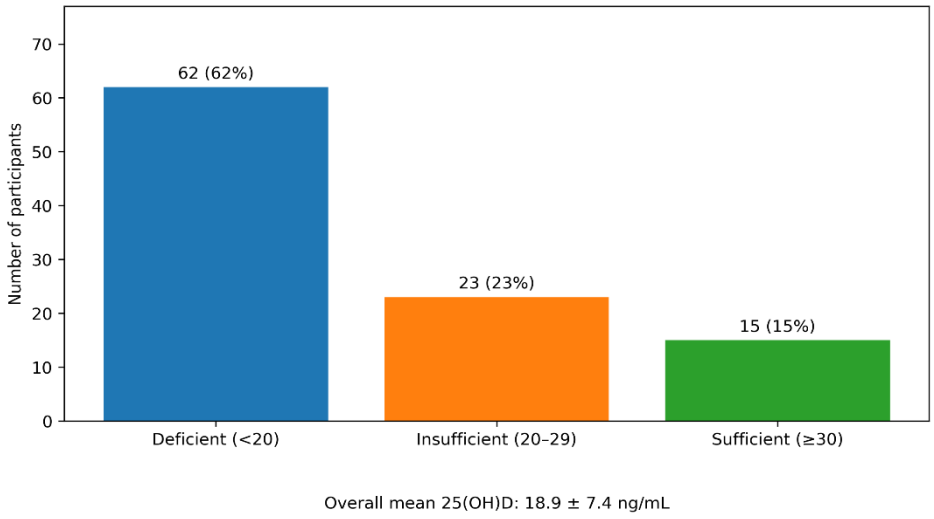

Figure 1: Distribution of Vitamin D Status

Overall glycemic indices indicated suboptimal control in the cohort, with poor control in 88% of patients based on HbA1c (Table 3).

Table 3. Glycemic profile of study participants (N = 100)

Parameter	Mean ± SD	Category	n (%)
Fasting blood sugar (mg/dL)	152.6 ± 41.3	—	—
Postprandial blood sugar (mg/dL)	214.8 ± 48.7	—	—
HbA1c (%)	8.6 ± 1.4	—	—
Glycemic control (HbA1c)	—	≤7% (Good control)	12 (12%)
	—	>7% (Poor control)	88 (88%)

Glycemic indices varied significantly across vitamin D categories, with higher HbA1c, FBS, and PPBS among vitamin D–deficient patients (Table 4).

Table 4. Association between vitamin D status and glycemic parameters

Vitamin D status	HbA1c (%) Mean ± SD	FBS (mg/dL) Mean ± SD	PPBS (mg/dL) Mean ± SD	Poor glycemic control n (%)
Deficient (n = 62)	9.1 ± 1.3	165.4 ± 39.8	228.6 ± 46.2	58 (94%)
Insufficient (n = 23)	8.2 ± 1.1	148.7 ± 33.5	205.1 ± 40.4	17 (74%)
Sufficient (n = 15)	7.4 ± 0.9	132.1 ± 28.4	186.3 ± 35.7	8 (53%)

Statistical notes: HbA1c comparison across vitamin D groups, $p < 0.001$; FBS and PPBS comparison, $p < 0.01$; association with glycemic control, $\chi^2 = 14.62$, $p < 0.001$. Correlation coefficients for serum 25(OH)D: HbA1c ($r = –0.48$, $p < 0.001$), FBS ($r = –0.42$, $p < 0.01$), PPBS ($r = –0.39$, $p < 0.01$).

DISCUSSION

This study demonstrates a high burden of hypovitaminosis D in adults with T2DM in a tertiary-care setting, with 62% classified as deficient and only 15% having sufficient 25(OH)D levels. These proportions are comparable to Indian reports showing widespread deficiency among people with dysglycemia and established diabetes [4,5]. The observed mean 25(OH)D concentration (18.9 ng/mL) also aligns with reports from other regions where indoor occupations, reduced sun exposure, obesity, and dietary insufficiency interact to lower vitamin D stores [6,7].

A consistent gradient was noted between vitamin D categories and glycemic indices. Patients with deficiency had higher mean HbA1c values than those with sufficient vitamin D status, and similar patterns were seen for fasting and postprandial glucose. These findings mirror reports from European and Middle Eastern cohorts where HbA1c has shown an inverse relationship with 25(OH)D levels in people with T2DM [3,6,7]. The negative correlations seen with HbA1c, FBS, and PPBS in the present cohort further support the concept that poorer vitamin D status clusters with worse glycemic control, even though causal direction cannot be inferred from cross-sectional data.

Several biological pathways can explain this association. Vitamin D signaling can influence beta-cell calcium flux and insulin release, and it can also modulate peripheral insulin sensitivity via effects on inflammation, oxidative stress, and adipokine profiles [1,2]. In addition, vitamin D has immunomodulatory functions that can attenuate cytokine-mediated insulin resistance, providing a plausible link between low 25(OH)D levels and higher glycemic burden in chronic metabolic disease [1].

Interventional literature offers mixed but informative evidence for translation into practice. Meta-analyses of randomized and interventional studies suggest that vitamin D supplementation can produce small improvements in HbA1c or related indices, particularly when baseline deficiency is present and dosing is adequate [9-11]. However, trial outcomes vary by regimen, duration, baseline 25(OH)D, and participant phenotype. For example, a randomized controlled trial in vitamin D–deficient patients with T2DM reported no major improvement in insulin sensitivity or secretion despite supplementation [13], while another double-blind trial in individuals with T2DM and obesity documented changes in metabolic markers following supplementation [14]. Taken together, these findings support a targeted approach that prioritizes identifying and correcting deficiency, while acknowledging that supplementation alone is not a substitute for comprehensive lifestyle and pharmacologic management.

Limitations

The cross-sectional design does not establish temporal relationships between vitamin D status and glycemic control. This single-center study used consecutive sampling, which limits representativeness for the broader community. Determinants such as dietary vitamin D intake, quantified sun exposure, physical activity, body mass index, and seasonal variation were not modeled. Parathyroid hormone and mineral profile were not assessed, and vitamin D supplement history was recorded only at screening.

CONCLUSION

In this six-month hospital-based study of 100 adults with T2DM, hypovitaminosis D was highly prevalent, and lower serum 25(OH)D levels were linked to worse glycemic indices. Vitamin D–deficient participants had higher HbA1c, fasting glucose, and postprandial glucose values than those with sufficient vitamin D status, and 25(OH)D correlated inversely with HbA1c, FBS, and PPBS. These findings emphasize that vitamin D status is a clinically relevant metabolic marker in routine diabetes care. Integrating vitamin D assessment with standard glycemic monitoring can help identify patients who need correction of deficiency and reinforce lifestyle counseling as part of holistic risk reduction. Correcting deficiency during follow-up visits is warranted**.**

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