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Serum Vitamin D Levels and Their Association with Glycemic Control in Type 2 Diabetes Mellitus - A Hospital-Based Cross-Sectional Observational Study

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Abstract

Background: Over 77 million adults with in diahaving Type 2 diabetes mellitus and its significant public health issue. Vitamin D deficiency is very common among Indians, and its functioning directly related to β-cell dysfunction and impaired glucose regulation. Although vitamin D deficiency very common among India's despite the country's tropical climate, nothing is known about how it affects glycemic control in North Indian T2DM patients.

Aim: To estimate serum level of Vitamin D [25(OH)D] among adult T2DM patients and to evaluate the association between Vitamin D level and glycaemic control measured by glycated haemoglobin (HbA1c).

Methods: Muzaffar nagar Medical College & Hospital carried out an observational cross-sectional study over the period of eighteen months. The study comprised 100 consecutive people with type 2 diabetes (≥18 years old) on established antidiabetic medication. Individuals with gluco corticoid use, chronic kidney illness, liver disease, or prior vitamin D supplementation were not included. Measurements were made of 25(OH) D serum level, HbA1c, and fasting plasma glucose. Based on HbA1c, glycaemic management was categorised as good (≤ 7%), fair (7.1–8.9%), or bad (≥ 9%). One-way ANOVA, the chi-square test, and correlation (Pearson test) were used to examine the data (SPSS v25).

Results: 54% of them were men, and their average age was 56.75 ± 12.76 years. The mean serum level of 25 (OH) D was 20.65 ± 8.55 ng/mL, which is within the insufficient range, and the mean level of HbA1c was 8.51± 1.15%. The mean HbA1c for the three different Vitamin D classes varied significantly: 8.09 ± 0.82% for the sufficient group and 9.01 ± 1.15% for the deficient group [F (2,97) = 8.260, $p < 0.001$]. Association test revealed that relationship between vitamin D status and glycaemic control categories ($\chi^2 = 21.981$, $p < 0.001$). The variables showed an inverse trend between serum 25(OH)D and HbA1c. The findings also showed a relationship between vitamin D insufficiency and hypertension ($p = 0.049$). BMI, eGFR, and FPG did not substantially correlate with vitamin D.

In conclusion, vitamin D deficiency is commonly observed among T2DM patients in North India and is closely associated with hypertension and poorer long-term glycaemic management. Targeted vitamin D correction and regular screening should be part of comprehensive diabetic management. To establish evidence-based supplementation procedures and verify causality, larger prospective RCTs are necessary.

Keywords: Type 2 Diabetes Mellitus, Hba1c, Glycaemic Control, Insulin Resistance, Hypertension; North India, Vitamin D, 25-Hydroxyvitamin D

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic and radually advancing metabolic disorder characterised by sustained elevation of blood glucose due to gradual β-cell impairment and peripheral resistance to insulin[1,2].

Globally, there were 108 million adults with diabetes in 1980; by 2019, that figure has risen to 463 million, and by 2045, it is predicted to reach 700 million[3]. The "diabetes capital of the world," India, is home to over 77 million of their adults. It is estimated that this burden will rise to 109 million by 2035[4]. The Asian Indian phenotype characterized by central adiposity, higher body-fat percentage at lower BMI cutoffs, and marked insulin resistance places this population at risk for T2DM at younger ages and lower body weight than their Western counterparts [5,6].

A lipid - soluble secosteroid compound peptide is vitamin D. Its function in regulating calcium homeostasis and maintaining skeletal health is well known in nature [7]. Direct exposure of skin to UVB rays stimulates vitamin D production. The main circulating biomarker of vitamin D status, 25-hydroxyvitamin D [25 (OH) D], is produced when liver hydroxylates this vitamin D. This is then changed into the active form of vitamin D, called 1, 25-dihydroxyvitamin D (calcitriol), through renal 1α-hydroxylase-mediated conversion [8]. The Vitamin D receptor (VDR), which is present in nearly every tissue, including smooth muscle of the blood vessels, immune cells, and pancreatic islets, is how calcitriol works. Apart from its impact on the skeleton, vitamin D has an important role in controlling endothelial performance and the RAAS mechanism, both of which are important for circulatory and metabolic health [9,10]. Vitamin D and glucose metabolism are linked through a number of processes. In pancreatic β-cells, where calcitriol controls the transcription of the insulin gene and the intracellular calcium flux required for insulin exocytosis, VDRs are abundantly expressed [11].

Furthermore, vitamin D reduces the impairment of insulin signalling in skeleton muscles and adipose tissue brought on by reactive oxygen species (ROS), increases the level of expression of insulin receptors in peripheral tissues, and reduces inflammatory mediators including

TNF-α and IL-6 that are associated with insulin resistance [12,13,14]. Despite the country's abundance of sunshine, vitamin D insufficiency is very common in India. Contributing variables include urbanisation and indoor lives, air pollution that lowers UVB penetration, cultural clothing customs, and diets low in foods high in vitamin D[15]. Numerous studies conducted globally have found a negative correlation between serum 25(OH) D levels and HbA1c in people with type 2 diabetes [16, 17].

There is still a lack of data from North Indian tertiary care populations, where the Asian Indian phenotype, high rates of vitamin D deficiencies, and persistent, diabetes that is poorly managed coexist. Bibliometric data shows that research on vitamin D and type 2 diabetic conditions is rapidly expanding.

Determining if vitamin D deficiency is a modifiable risk factor for poor glycaemic control in this population directly affects the use of supplements in addition to conventional diabetes therapy and screening procedures. The current investigation examined serum vitamin D levels in adult patients with established type 2 diabetes in order to evaluate the association between vitamin D status and glycaemic management as determined by glycated haemoglobin (HbA1c).

Material and Methods

Study Design and Setting

The general medicine division of Muzaffarnagar Medical College & Hospital (MMCH), Muzaffarnagar, Uttar Pradesh, India, conducted a cross-sectional observational study. The study required 18 months to complete, of which 12 months were spent on patient enrolment and data collecting, while the remaining six months were spent on data assembly, analysis, and publication preparation. The MMCH Institutional Ethics Board accepted these investigations, which were carried out in compliance with the Declaration of Helsinki (Ref. No. MMC/ IEC/ 2024/31, dated 13 – 03 - 2024). Each participant was enrolled only after providing written informed consent.

Study Population and Eligibility Criteria

The study population consists of adult patients having Type 2 Diabetes Mellitus that are eighteen years of age or older and who either visit the general medicine outpatient facility or are admitted to MMCH's medical wards. Based on hospital data, the diagnosis of type 2 diabetes was verified using the American Diabetes Association (ADA) criteria. To find the next 100 eligible patients, consecutive sampling was employed. Individuals with a history of long - term liver disease, chronic renal impairment, continuous glucocorticoid therapy, anti-seizure drugs known to interfere with the metabolism of vitamin D, or participants who had consumed vitamin D supplements during the previous three months prior were excluded.

Data Collection

A standardised case record form was used to document the demographics, length of diabetes, current antibiotics, co morbidity (hypertension, cardiovascular illness, abnormal thyroid, chronic renal disease), and medication history. Each subject was noted for vital signs, blood pressure, pulse rate, systemic examination and general a physical checkup.

Laboratory Investigations and Outcome Definitions

Venous blood was collected aseptically following an overnight fast of at least eight hours Blood glucose concentration was determined using the glucose oxidase technique in fasting patients. Quantification of glycated haemoglobin (HbA1c) was performed by high - pressure liquid chromatography (HPLC). Serum 25-hydroxy vitamin D [25 (OH) D] was measured by

chemiluminescence immunoassay (CLIA). We measured markers of vitamin D-related mineral metabolism, namely alkaline phosphatase (ALP), serum phosphorus, and parathyroid hormone (PTH). Standard work-up consisted of complete blood count, ipid parameters, hepatic function tests and renal function tests to confirm eligibility. Glycaemic management was classified as good (≤7.0%), adequate (7.1–8.9%) or poor (≥9.0%) based on HbA1c. Vitamin D levels were categorised as sufficient (≥30 ng/mL), insufficient (20–29.9 ng/mL), or deficient (<20 ng/mL) according to the Endocrine Society Clinical Services Guideline.

Statistical Analysis

Data was entered into "Microsoft Excel" and then analysed using SPSS wityhn using version 25.0. Categorical variables are shown as frequency and percentage, whereas constant variables with a somewhat regular distribution are shown as mean ± standard deviation (SD). One-way variance analysis was used to compare the mean differences among groups in continuous outcomes for each of the three categories of vitamin D levels. Tukey's HSD was used as the post-hoc test for pair wise comparisons. chi-square statistic was utilized to evaluate correlations across different categories. Pearson's product-moment correlation was calculated to evaluate the linear correlations between blood 25(OH)D and continuous metabolic markers (HbA1c, FPG, BMI, and eGFR

Results

Baseline data of Demographic and Clinical Characteristics

A total of 100 adult T2DM patients were enrolled... mean age 56.75±12.76 years, BMI 27.17±2.84 kg/m², diabetes duration 12.65±6.92 years. Gender: 54% male, 46% female; 50% family history. HTN 59 %, thyroid 40%, CKD 24%, CAD 20%. Only 35% on Vit D supplementation."

Table 1: Baseline Demographic and Clinical Characteristics – Continuous Variables (N=100)

Variable	Mean ± SD	Range
Age (years)	56.75 ± 12.76	35–80
Body mass index (kg/m²)	27.17 ± 2.84	18.9–33.5
Duration of diabetes (years)	12.65 ± 6.92	1–25
Pulse rate (beats/min)	86.19 ± 8.92	70–100

Table 2 presents the glycemic profile of the enrolled patients. Mean fasting blood glucose levels was 160.97 ± 33.57 mg/dL (range 72.3–264.9 mg/dL) and mean HbA1c was 8.51 ± 1.15% (range 4.4–11.3%). Both values were far above the recommended target of ≤ 7.0% for optimum glycemic management according to ADA guidelines, indicating that the majority of patients had poorly controlled diabetes. This finding also reflects the long duration of disease and the typical unacceptable control pattern seen in resource limited settings.

Table 2: Glycemic Parameters (N=100)

Variable	Mean ± SD	Range
Fasting plasma glucose (mg/dL)	160.97 ± 33.57	72.3–264.9
HbA1c (%)	8.51 ± 1.15	4.4–11.3

The vitamin D and related mineral metabolism parameters are collected in Table 3. The cohort as a whole fell into the inadequate range (<30 ng/mL) with a mean serum 25 (OH) D of 20.65 ± 8.55 ng/mL (range: 0.1–37.1 ng/mL). Phosphorus was 3.50 ± 0.51 mg/dL, PTH was 39.56 ± 12.21 pg/mL, and alkaline phosphatase enzyme was 106.71 ± 25.07 U/L. The slight increase in PTH is consistent with secondary hyperparathyroidism, which is frequently linked to vitamin D deficiency.

Table 3: Vitamin D and Mineral Metabolism Parameters (N=100)

Variable	Mean ± SD	Range
Serum 25(OH)D (ng/mL)	20.65 ± 8.55	0.1–37.1
Parathyroid hormone – PTH (pg/mL)	39.56 ± 12.21	15.4–70.4
Phosphorus (mg/dL)	3.50 ± 0.51	2.4–4.9
Alkaline phosphatase (U/L)	106.71 ± 25.07	38.9–170.7

Table 4 shows glycaemic control distribution across over Vitamin D groups. Poor control (≥9%) was higher in deficient group (54.7%) vs only 4.3% in sufficient group, with highly significant association (χ^2=21.981, p=0.001), confirming stepwise improvement with rising Vitamin D levels.

Figure 1: Pearson correlation scatter plot between serum 25(OH)D and HbA1c (r = −0.329, p = 0.001). Dashed line represents the regression line with 95% CI shading.

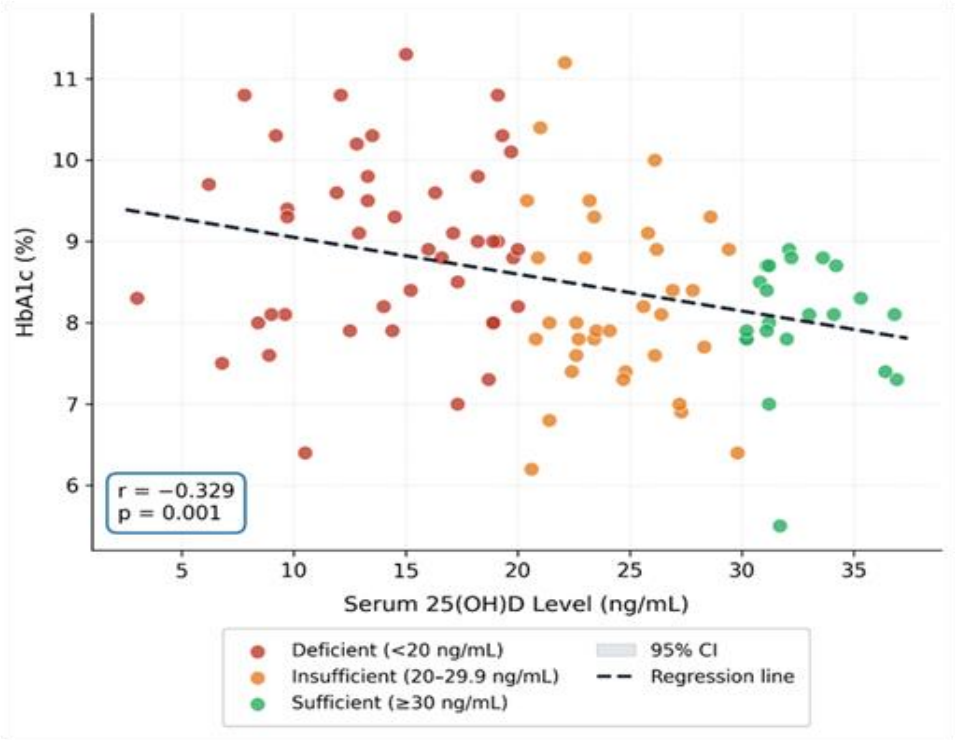


Table 4: Association of Vitamin D Status with Glycemic Control (N=100)

Vitamin D Status	Good (≤7%) n (%)	Fair (7.1–8.9%) n (%)	Poor (≥9%) n (%)	Total n (%)	χ^2, p-value
Deficient (<20 ng/mL)	2 (4.8%)	17 (40.5%)	23 (54.7%)	42 (42.0%)	χ^2=21.981
Insufficient (20–29 ng/mL)	5 (14.3%)	23 (65.7%)	7 (20.0%)	35 (35.0%)	p=0.001
Sufficient (≥30 ng/mL)	2 (8.7%)	20 (87.0%)	1 (4.3%)	23 (23.0%)	

Mean HbA1c across Vitamin D Groups – One-Way ANOVA

Table 5 shows the stepwise grading of mean HbA1c levels by vitamin D level groups. The group with the highest mean HbA1c was the vitamin D deficiency group (9.01 ± 1.15%), followed by the insufficient group (8.20 ± 1.05%) and the sufficient group (8.09 ± 0.82%). A one-way ANOVA revealed that these differences were statistically significant [F(2,97)=8.260, p<0.001]. This gradient has significant therapeutic implications: a clinically significant decrease in the risk of micro vascular and macro vascular problems is linked to the mean difference of nearly 0.9% in HbA1c between the insufficient and sufficient groups.

Table 5: Mean HbA1c Across Vitamin D Status Groups – One-Way ANOVA (N=100)

Vitamin D Status	n	Mean HbA1c ± SD (%)	F-statistic (df1, df2)	p-value
Deficient (<20 ng/mL)	42	9.01 ± 1.15	F(2,97)=8.260	<0.001
Insufficient (20–29 ng/mL)	35	8.20 ± 1.05		
Sufficient (≥30 ng/mL)	23	8.09 ± 0.82		
Total	100	8.52 ± 1.12		

Figure 2: Box plot showing HbA1c distribution across Vitamin D status categories. One-way ANOVA: F(2,97) = 8.260, p < 0.001.

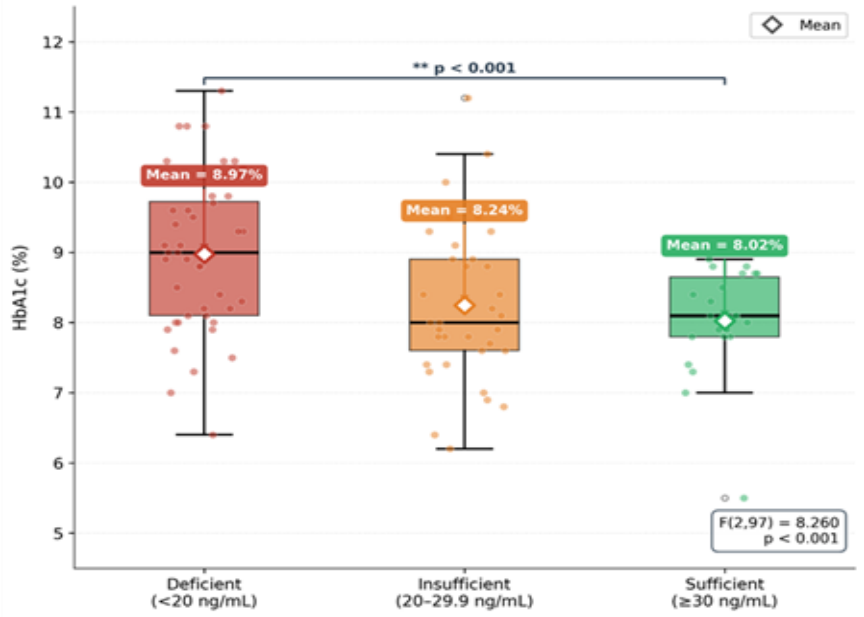
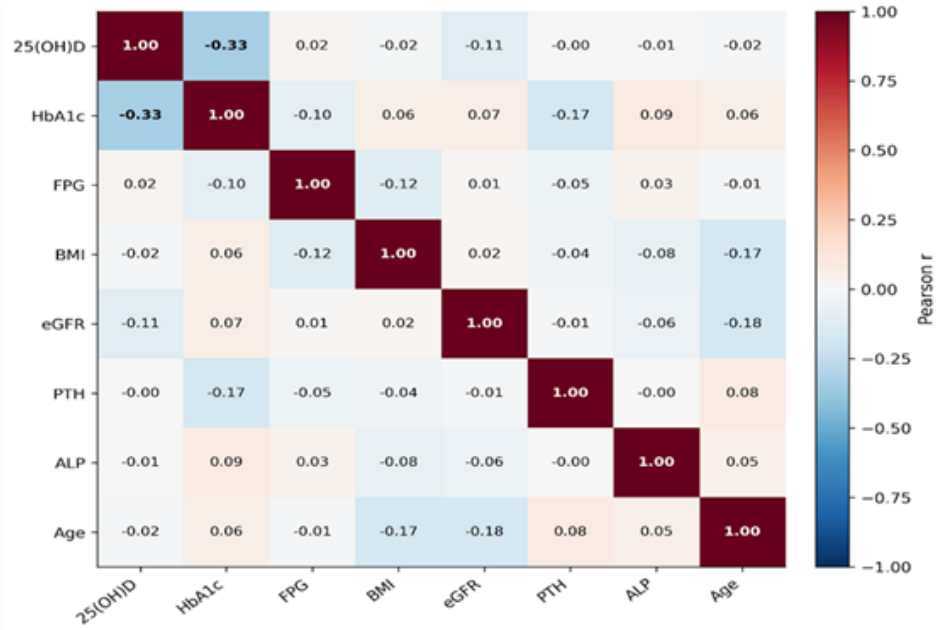


Figure 3: Correlation heatmap of serum 25(OH)D with glycaemic and metabolic parameters (N=100).



Discussion

In this study, 100 adult T2DM patients at a tertiary care facility in western Uttar Pradesh had their blood 25(OH)D level and its relationship to glycaemic therapy evaluated. The cohort's mean age of 56.75 years, mean BMI of 27.17 kg/m² (overweight category), and mean duration of diabetes of 12.65 years are all very typical of the usual North Indian T2DM group described in previous research [20,21,22,23]. Biochemical insufficiency is common in this group, yet only 35% of participants were on any form of vitamin D supplementation. This difference has been previously documented in Middle Eastern and South Asian populations [17,24].

The average HbA1c of 8.51 ± 1.15% was significantly higher than the ADA objective of ≤ 7.0%, which is in line with the poor glycaemic control frequently observed in long-term T2DM patients monitored in public hospital settings with inadequate resources. In a similar diabetic population, A similar negative correlation among 25 (OH) D and HbA1c ($p < 0.001$) was discovered by Abdo et al. (2024) [25].

Vitamin D administration reduced FBG (WMD −0.49 mmol/L) and HbA1c (WMD −0.30%), with the biggest results in individuals with baseline vitamin D insufficiency and HbA1c ≥8%, according to Chen et al.'s 2024 meta-analysis of 39 RCTs (n = 2,982) [26]. This sub-group profile closely resembles the vitamin D-deficient patients in our cohort (mean HbA1c 9.01%). These findings are situated within the larger worldwide body of evidence by recent umbrella reviews by Cheng et al. (2024) and Alzahrani et al. (2024) [24,27].

A substantial stepwise gradient in mean HbA1c among Vitamin D categories was revealed by one-way ANOVA [$F(2,97) = 8.260$, $p < 0.001$]: 9.01 ± 1.15% in the deficient group, 8.20 ± 1.05% in the inadequate group, and 8.09 ± 0.82% in the sufficient group. Given that even modest HbA1c reductions are linked to notable decreases in micro vascular and macro vascular risk, the ~0.9% HbA1c difference between the deficient and sufficient groups is clinically important. Various populations have been reported to exhibit a similar gradient. In a sizable Chinese T2DM population, Zhao et

al. (2020) discovered that vitamin D insufficiency was independently linked to increased HbA1c [18]. In 309 Saudi T2DM patients, Darraj et al. (2019) discovered that VDD was an independent indicator of inadequate glycaemic control using variable logistical regression [17]. In accordance with Salih et al. (2021) and Al Dossari et al. (2019) [28, 29], vitamin D-deficient individuals had significantly elevated HbA1c and a higher proportion of patients in the poor glycaemic control category.

The chi-square test ($\chi^2 = 21.981$, $p = 0.001$) revealed that the distribution of the control of glucose categories was irregular among Vitamin D status groups: 54.7% of patients with inadequate amounts of vitamin D had poor glycaemic control, as relative to 4.3% of participants with adequate vitamin D status. Serum 25 (OH) D and HbA1c had a negative and significant Pearson association ($r = -0.329$, $p = 0.001$), falling within the range of previous observational studies ($r = -0.26$ to -0.74) [30,31,32]. The biological basis for this relationship is explained in detail. Vitamin D regulates the effects of calcitriol on insulin gene synthesis and calcium-dependent exocytosis processes in pancreatic β-cells [11,33], increases the expression of insulin receptors in peripheral tissues [12], and suppresses pro-inflammatory cytokines like TNF-α and IL-6 that cause insulin resistance [13,14]. Since HbA1c is more sensitive to the long-term metabolic impact of vitamin D than a single fasting glucose evaluation and represents chronic glycaemic exposure to the substance over approximately two to three months, there is no statistically significant association between 25(OH)D and FPG ($r = +0.024$, $p = 0.809$) [19,34].

With prevalences of 74.3% in the insufficient group, 57.1% in the lack of category, and 40.9% in the sufficient group, vitamin D insufficiency was substantially linked to the occurrence of hypertension ($p = 0.049$). Mechanisms: Vitamin D is known to improve endothelial function, regulate smooth muscle tone in arteries, and inhibit the rennin – angiotensin - aldosterone system [9,10]. This is supported by the study by Shaafie and Hesham (2016) [35] and the review by Pérez - Hernández et al. (2016) [10]. When taken as a whole, these results strengthen the assumption that reduced vitamin D level influences cardio metabolic risk in T2DM in ways other than glycaemic control.

There are a few restrictions to be aware of. Causal inference is not possible with the cross-sectional design. The single center sampling of 100 patients limits the external validity. Sun exposure, dietary vitamin D intake, and physical exercise were all significant factors that were not thoroughly assessed. However, before causal claims and evidence-based dietary supplements can be made, sufficient powered prospective randomized controlled studies in Indian T2DM samples with multivariable correction for these variables are required. procedures established for this group.

Conclusion

With a mean blood 25 (OH) D of 20.65 ± 8.55 ng/mL, which is within the insufficient range, low vitamin D status was frequently reported in this North Indian tertiary care cohort of T2DM patients. Reduced vitamin D levels were linked to a higher prevalence of hypertension ($p = 0.049$) and showed a negative correlation with HbA1c ($r = -0.329$, $p = 0.01$). From the appropriate to the deficient group, the mean HbA1c started to increase [$F(2,97) = 8.260$, $p < 0.001$]. Therefore, vitamin-D level seems to be a modifiable factor related to glycaemic control in type 2 diabetes.

Patients with poor control may benefit from screening and targeted replacement, especially if hypo vitaminosis

D is established. Due to the cross-sectional design, causal inferences cannot be made from the findings. Adequately powered randomised studies in Indian T2DM populations with multi variable correction for factors like BMI, sun exposure, and nutritional consumption are required before optimal dose and treatment thresholds can be determined.

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