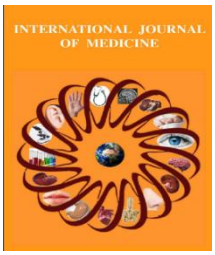

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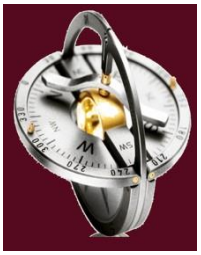

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Original Research

Association of Serum Vitamin D Levels with Glycaemic Control and Diabetic Complications in Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Observational Study.

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Abstract

Background: Vitamin D has metabolic, immunological, and vascular actions that could influence insulin sensitivity and the development of diabetes-related complications. Evidence regarding its association with glycaemic control in type 2 diabetes mellitus remains inconsistent. *Objectives:* To determine serum vitamin D status and examine its association with glycaemic indices and chronic diabetic complications among patients with type 2 diabetes mellitus. *Methods:* This hospital-based cross-sectional observational study included 80 adults with type 2 diabetes mellitus at Government Medical College and General Hospital, Suryapet, Telangana, India, from January to October 2024. Demographic and clinical characteristics, fasting and postprandial plasma glucose, glycated haemoglobin, serum 25-hydroxyvitamin D, and diabetic complications were assessed. Vitamin D status was classified as deficient, insufficient, or sufficient. Associations were analysed using appropriate comparative tests, correlation analysis, and multivariable logistic regression. *Results:* The mean age was 54.6 ± 9.8 years, and 46 (57.5%) participants were male. Vitamin D deficiency was present in 42 (52.5%), insufficiency in 23 (28.8%), and sufficiency in 15 (18.8%) patients. Mean HbA1c was significantly higher in deficient than in insufficient and sufficient groups (9.1 ± 1.4%, 7.9 ± 1.2%, and 7.1 ± 1.0%, respectively; p<0.001). At least one diabetic complication occurred in 44 (55.0%) participants and was more frequent in the deficient group (71.4%) than in the insufficient (43.5%) and sufficient (26.7%) groups. Serum vitamin D correlated inversely with HbA1c (r=-0.48; p<0.001). Vitamin D deficiency independently predicted diabetic complications (adjusted odds ratio 3.12; 95% confidence interval 1.18-8.27). *Conclusion:* Lower serum vitamin D levels were associated with poorer glycaemic control and a greater burden of diabetic complications. Prospective studies are required to clarify temporality and therapeutic relevance.

Keywords: *Type 2 diabetes mellitus; vitamin D deficiency; glycated haemoglobin; diabetic neuropathy; diabetic retinopathy; diabetic nephropathy; cross-sectional study.*

Introduction

Type 2 diabetes mellitus is a chronic metabolic disorder characterised by persistent hyperglycaemia resulting from impaired insulin secretion, insulin resistance, or both. Its clinical importance extends beyond glucose elevation because prolonged metabolic dysregulation progressively damages the retinal, renal, neural, and cardiovascular systems. Contemporary diabetes care therefore emphasises sustained glycaemic control, systematic screening for complications, and management of modifiable cardiovascular risk factors. Glycated haemoglobin remains the principal marker of long-term glycaemic exposure, and values below 7% are generally appropriate for many non-pregnant adults when treatment can be delivered safely.[1]

Vitamin D is a fat-soluble secosteroid with established actions in calcium and bone homeostasis. Its receptor and activating enzymes are also expressed in pancreatic beta cells, skeletal muscle, vascular endothelium, immune cells, and several other tissues. These observations have stimulated interest in possible extra-skeletal effects on insulin synthesis, insulin receptor expression, inflammatory signalling, oxidative stress, endothelial function, and the renin-angiotensin system. Serum 25-hydroxyvitamin D is the accepted indicator of vitamin D status. Concentrations below 20 ng/mL are commonly classified as deficient, levels of 20-29.9 ng/mL as insufficient, and levels of at least 30 ng/mL as sufficient.[2]

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Observational evidence has repeatedly linked low vitamin D concentrations with insulin resistance, incident type 2 diabetes, and unfavourable glycaemic indices, although the direction and independence of these relationships remain debated.[3] Intervention studies have produced heterogeneous findings. Earlier systematic reviews found insufficient evidence for routine vitamin D supplementation solely to improve glucose metabolism, whereas later meta-analyses reported modest reductions in fasting glucose, HbA1c, or insulin resistance, particularly among participants with established deficiency or poor baseline control.[4] Such differences can reflect variation in dose, duration, baseline vitamin D status, body composition, antidiabetic treatment, assay methods, and study quality.

Low vitamin D status has also been associated with microvascular complications. Indian and international studies have described progressively lower vitamin D levels in patients with neuropathy, retinopathy, nephropathy, or combinations of these conditions.[5,6] Proposed mechanisms include impaired neurotrophic support, enhanced inflammation, endothelial dysfunction, activation of the renin-angiotensin system, and altered angiogenesis. Nevertheless, most available evidence is cross-sectional, and residual confounding by age, obesity, duration of diabetes, renal function, physical activity, sun exposure, and glycaemic control remains important.[7]

Data integrating vitamin D status, glycaemic parameters, and multiple diabetic complications in patients attending government tertiary-care facilities in Telangana are limited. The present study was therefore conducted to determine the distribution of serum vitamin D levels among adults with type 2 diabetes mellitus and to assess their association with fasting and postprandial plasma glucose, HbA1c, and chronic diabetic complications. A secondary objective was to identify independent clinical factors associated with the presence of at least one diabetic complication.

Materials and Methods

Study design and setting: This hospital-based cross-sectional observational study was conducted in the Department of General Medicine, Government Medical College and General Hospital, Suryapet, Telangana, India. The study period extended from January 2024 to October 2024. The manuscript was prepared in accordance with accepted reporting principles for observational research.

Study population: Adults aged 30 years or older with a documented diagnosis of type 2 diabetes mellitus were screened during routine outpatient or inpatient care. Patients were eligible when complete clinical records and the required biochemical measurements were available. Patients with type 1 diabetes, gestational diabetes, acute diabetic emergencies, active severe infection, advanced hepatic disease, stage 4 or 5 chronic kidney disease, malabsorption, current treatment with pharmacological vitamin D doses, or medicines known to substantially alter vitamin D metabolism were excluded. Consecutive eligible patients were enrolled until the required sample was reached.

Sample size and sampling: In the absence of a reliable local prevalence estimate, a conservative expected prevalence of 50% was used. At a 95% confidence level and an absolute precision of 11%, the calculated minimum sample was 79.4; this was rounded to 80 participants. Consecutive sampling was used to reduce discretionary selection.

Data collection and clinical assessment: Demographic characteristics, duration of diabetes, smoking status, current antidiabetic treatment, hypertension, and dyslipidaemia were recorded using a structured case-record form. Weight and height were measured using standard procedures, and body mass index was calculated as weight in kilograms divided by height in metres squared. Fasting plasma glucose, postprandial plasma glucose, HbA1c, and serum 25-hydroxyvitamin D were measured in the hospital laboratory using standardised quality-controlled methods. Glycaemic control was categorised as HbA1c <7%, 7.0-8.9%, or ≥9%, consistent with clinically meaningful control strata.[1]

Operational definitions and complications: Vitamin D deficiency was defined as serum 25-hydroxyvitamin D <20 ng/mL, insufficiency as 20-29.9 ng/mL, and sufficiency as ≥30 ng/mL.[2] Peripheral neuropathy was identified from compatible symptoms and clinical sensory examination. Retinopathy was recorded from documented fundus evaluation. Nephropathy was defined by documented albuminuria and/or diabetes-related reduction in estimated glomerular filtration rate. Cardiovascular disease included established coronary artery disease, stroke, or peripheral arterial disease. Individual complications were not mutually exclusive.

Statistical analysis: Data were analysed using [insert statistical software and version]. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequency and percentage. Between-group continuous variables were compared using one-way analysis of variance or an independent-samples t-test. Categorical associations were assessed using the chi-square test or Fisher's exact test when expected counts were small. Pearson correlation coefficients quantified relationships between vitamin D and continuous clinical variables. Multivariable binary logistic regression identified factors independently associated with at least one diabetic complication. Adjusted odds ratios with 95% confidence intervals were reported, and $p<0.05$ was considered statistically significant.

Ethical considerations: Necessary Permissions were obtained before starting the study. Written informed consent was obtained from each participant. Confidentiality was maintained throughout data collection and analysis.

Results

A total of 80 patients with type 2 diabetes mellitus were included, and complete demographic, clinical, biochemical, and complication-related information was available for analysis. The mean age was 54.6 ± 9.8 years (range, 35-74 years); 46 (57.5%) participants were male and 34 (42.5%) were female. The mean duration of diabetes was 8.2 ± 5.1 years, and the mean body mass index was 26.8 ± 4.2 kg/m². Hypertension and dyslipidaemia were present in 38 (47.5%) and 29 (36.3%) participants, respectively. The mean serum vitamin D concentration was 19.8 ± 8.7 ng/mL. Deficiency was identified in 42 (52.5%), insufficiency in 23 (28.8%), and sufficiency in 15 (18.8%) patients. The mean HbA1c was 8.4 ± 1.6%, and only 20 (25.0%) participants had HbA1c <7% (Table 1).

Table 1. Demographic, clinical, vitamin D, and glycaemic characteristics of the participants (N=80)

Characteristic	Category/Value	Frequency or Mean ± SD	Percentage
Age, years	Mean ± SD	54.6 ± 9.8	-
Sex	Male	46	57.5
	Female	34	42.5
Duration of diabetes, years	Mean ± SD	8.2 ± 5.1	-
	<5 years	24	30.0
	5-10 years	31	38.8
	>10 years	25	31.3
Body mass index, kg/m²	Mean ± SD	26.8 ± 4.2	-
Hypertension	Present	38	47.5
Dyslipidaemia	Present	29	36.3
Serum vitamin D, ng/mL	Mean ± SD	19.8 ± 8.7	-
Vitamin D status	Deficient, <20 ng/mL	42	52.5
	Insufficient, 20-29.9 ng/mL	23	28.8
	Sufficient, ≥30 ng/mL	15	18.8
Fasting plasma glucose, mg/dL	Mean ± SD	168.4 ± 48.6	-
Postprandial plasma glucose, mg/dL	Mean ± SD	246.7 ± 69.5	-

HbA1c, %	Mean ± SD	8.4 ± 1.6	-
Glycaemic control	HbA1c <7%	20	25.0
	HbA1c 7.0-8.9%	36	45.0
	HbA1c ≥9%	24	30.0

Glycaemic indices worsened across decreasing vitamin D categories. The mean HbA1c was 9.1 ± 1.4% in the deficient group, 7.9 ± 1.2% in the insufficient group, and 7.1 ± 1.0% in the sufficient group (p<0.001). Fasting and postprandial plasma glucose demonstrated comparable graded differences. Good glycaemic control was observed in 4 (9.5%) deficient patients, 7 (30.4%) insufficient patients, and 9 (60.0%) patients with sufficient vitamin D. The overall distribution of HbA1c categories differed significantly by vitamin D status (p=0.001) (Table 2).

Table 2. Association between serum vitamin D status and glycaemic control

Glycaemic parameter	Vitamin D deficient (n=42)	Vitamin D insufficient (n=23)	Vitamin D sufficient (n=15)	p-value
Fasting plasma glucose, mg/dL	187.6 ± 45.8	155.3 ± 37.6	134.7 ± 31.5	<0.001
Postprandial plasma glucose, mg/dL	278.9 ± 63.4	227.8 ± 49.7	185.6 ± 44.2	<0.001
HbA1c, %	9.1 ± 1.4	7.9 ± 1.2	7.1 ± 1.0	<0.001
HbA1c <7%, n (%)	4 (9.5)	7 (30.4)	9 (60.0)	0.001
HbA1c 7.0-8.9%, n (%)	19 (45.2)	12 (52.2)	5 (33.3)	
HbA1c ≥9%, n (%)	19 (45.2)	4 (17.4)	1 (6.7)	

Values are expressed as mean ± standard deviation or frequency (percentage). The p-value for HbA1c categories represents the overall chi-square test.

At least one chronic diabetic complication was present in 44 (55.0%) participants. Peripheral neuropathy was most frequent, occurring in 28 (35.0%), followed by retinopathy in 20 (25.0%), nephropathy in 18 (22.5%), and cardiovascular disease in 12 (15.0%). The prevalence of any complication was 71.4% in the deficient group, 43.5% in the insufficient group, and 26.7% in the sufficient group (p=0.005). Peripheral neuropathy and nephropathy were significantly associated with vitamin D category, whereas the retinopathy comparison was borderline and cardiovascular disease was not significantly associated (Table 3).

Table 3. Association between vitamin D status and diabetic complications

Diabetic complication	Overall, N=80	Vitamin D deficient (n=42)	Vitamin D insufficient (n=23)	Vitamin D sufficient (n=15)	p-value
Peripheral neuropathy	28 (35.0)	21 (50.0)	5 (21.7)	2 (13.3)	0.011
Diabetic retinopathy	20 (25.0)	15 (35.7)	4 (17.4)	1 (6.7)	0.051
Diabetic nephropathy	18 (22.5)	14 (33.3)	3 (13.0)	1 (6.7)	0.046
Cardiovascular disease	12 (15.0)	8 (19.0)	3 (13.0)	1 (6.7)	0.490
At least one complication	44 (55.0)	30 (71.4)	10 (43.5)	4 (26.7)	0.005

Values are expressed as frequency (percentage). Individual complications were not mutually exclusive. P-values were calculated from the displayed category counts.

Patients with diabetic complications had lower serum vitamin D levels than those without complications (16.3 ± 7.1 versus 24.1 ± 8.6 ng/mL; p<0.001). They were older, had longer diabetes duration, and had higher fasting plasma glucose and HbA1c. Serum vitamin D correlated inversely with HbA1c (r=-0.48; p<0.001), postprandial plasma glucose (r=-0.45; p<0.001), fasting plasma glucose (r=-0.41; p<0.001), and diabetes duration (r=-0.32; p=0.004). In multivariable analysis, vitamin D deficiency, diabetes duration >10 years, and HbA1c ≥9% were independently associated with at least one diabetic complication (Table 4).

Table 4. Comparison, correlation, and regression analysis of factors associated with diabetic complications

A. Comparison according to the presence of diabetic complications

Parameter	Complications present (n=44)	Complications absent (n=36)	p-value
Age, years	57.3 ± 8.7	51.3 ± 10.1	0.006
Duration of diabetes, years	10.6 ± 4.9	5.3 ± 3.7	<0.001
Serum vitamin D, ng/mL	16.3 ± 7.1	24.1 ± 8.6	<0.001
Fasting plasma glucose, mg/dL	181.8 ± 48.5	152.0 ± 43.9	0.005
HbA1c, %	9.0 ± 1.5	7.6 ± 1.3	<0.001

B. Correlation of serum vitamin D with selected clinical and glycaemic parameters

Parameter	Correlation coefficient (r)	p-value
Duration of diabetes	-0.32	0.004
Fasting plasma glucose	-0.41	<0.001
Postprandial plasma glucose	-0.45	<0.001
HbA1c	-0.48	<0.001
Body mass index	-0.18	0.112

C. Multivariable logistic regression for the presence of diabetic complications

Variable	Adjusted odds ratio	95% confidence interval	p-value
Vitamin D deficiency	3.12	1.18-8.27	0.022
Diabetes duration >10 years	4.26	1.52-11.92	0.006
HbA1c ≥9%	3.58	1.27-10.08	0.016
Age ≥60 years	1.74	0.66-4.61	0.264
Hypertension	1.62	0.63-4.17	0.317

Discussion

The present study identified a high burden of low vitamin D status among adults with type 2 diabetes mellitus. More than half of the participants were vitamin D deficient, and fewer than one in five had sufficient concentrations. Deficiency was accompanied by progressively higher fasting glucose, postprandial glucose, and HbA1c values. Serum vitamin D showed a moderate inverse correlation with HbA1c, while patients with at least one diabetic complication had substantially lower concentrations than those without complications. After adjustment for major clinical variables, vitamin D deficiency remained independently associated with diabetic complications.

The observed prevalence is consistent with the frequent coexistence of hypovitaminosis D and type 2 diabetes reported in South Asian and other populations. Mitri et al. described an inverse association between vitamin D status and type 2 diabetes risk, although heterogeneity

across observational studies was considerable.[3] In the present study, HbA1c decreased in a graded manner across deficient, insufficient, and sufficient vitamin D groups. This pattern supports a biological relationship rather than a simple binary threshold, but the cross-sectional design cannot determine whether low vitamin D contributes to dysglycaemia or reflects reduced outdoor activity, obesity, chronic illness, dietary inadequacy, or renal dysfunction.

Our complication findings agree with Bajaj et al., who reported declining vitamin D concentrations with increasing combinations of microvascular complications in Indian patients.[5] Jung et al. also found associations between severe deficiency and selected vascular complications, while Zhao et al. reported independent relationships with diabetic peripheral neuropathy and nephropathy.[6,11] The higher frequency of neuropathy in the deficient group is further supported by Shillo et al., who observed lower vitamin D levels in painful diabetic peripheral neuropathy.[9] Ashinne et al. reported an association with retinopathy among Asian Indians, and Chen et al. prospectively linked higher vitamin D status with lower microvascular risk.[8,12] Broader reviews still describe inconsistency across populations.[7]

Several mechanisms could explain these findings. Vitamin D signalling influences beta-cell function, insulin sensitivity, inflammatory pathways, endothelial integrity, neurotrophin production, and renin-angiotensin activity. Deficiency could therefore amplify metabolic and microvascular injury. Conversely, longstanding diabetes and its complications can reduce mobility, sunlight exposure, nutritional quality, and renal vitamin D handling. The strong independent contribution of diabetes duration and HbA1c in the regression model reinforces the established role of cumulative glycaemic exposure.

Interventional evidence remains mixed. George et al. found insufficient support for routine supplementation to improve glycaemia, whereas Hu et al. and Farahmand et al. reported modest benefits, particularly in deficient participants and short-duration interventions.[4,10,13] A recent systematic review similarly concluded that low vitamin D status is associated with adverse metabolic and complication outcomes, while causal certainty remains limited.[14] These findings support assessment of vitamin D when clinically indicated, correction of confirmed deficiency according to established guidance, and continued prioritisation of evidence-based glycaemic and cardiovascular risk management rather than viewing vitamin D replacement as a substitute for standard diabetes care.

Limitations

This study was conducted at a single tertiary-care hospital with a modest sample, limiting external generalisability. Its cross-sectional design precluded assessment of temporality or causality. Seasonal variation, dietary vitamin D intake, sunlight exposure, physical activity, parathyroid hormone, and detailed renal function were not fully evaluated. Complications were assessed from clinical and documented investigations, introducing classification variability. Residual confounding remained despite multivariable adjustment.

Conclusion

Vitamin D deficiency was common among patients with type 2 diabetes mellitus and showed a clear association with poorer glycaemic control. Serum vitamin D concentrations declined as fasting glucose, postprandial glucose, and HbA1c increased. Patients with deficiency also carried a greater burden of peripheral neuropathy, retinopathy, nephropathy, and overall diabetic complications. Vitamin D deficiency remained independently associated with complications after adjustment for selected clinical factors, alongside longer diabetes duration and marked hyperglycaemia. These findings support clinical attention to vitamin D status in high-risk patients while preserving glycaemic optimisation as the central strategy. Longitudinal studies and adequately powered trials are needed to determine whether correction of deficiency improves metabolic or complication-related outcomes

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