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Original Article

Effect of Serum 25-Hydroxy Cholecalciferol on Lipid Profile and Glycemic Status in Type 2 Diabetes Mellitus

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ABSTRACT

Background: Vitamin D deficiency is frequently reported in type 2 diabetes mellitus (T2DM) and is linked to insulin resistance, inflammation, and adverse cardiometabolic profiles. Its relationship with early glycemic derangement and dyslipidemia in outpatient practice warrants evaluation.
Objectives: To assess serum 25-hydroxyvitamin D status and determine its association with glycemic indices and lipid parameters among recently diagnosed adults with T2DM.
Methods: A cross-sectional study was conducted in the Department of General Medicine, Kamineni Academy of Medical Sciences and Research Centre, Hyderabad, from August 2022 to January 2024. One hundred adults aged 30-60 years with recently diagnosed T2DM on oral hypoglycemic therapy for <3 months were enrolled by simple random sampling. Serum 25-hydroxyvitamin D, fasting and postprandial glucose, HbA1c, and lipid profile were measured using standardized laboratory methods, and group comparisons were performed across vitamin D categories.
Results: Vitamin D deficiency (<=20 ng/mL) was present in 57% and insufficiency (21-29 ng/mL) in 34%. Poor glycemic control was common, with mean fasting glucose 150.9 mg/dL, postprandial glucose 211.5 mg/dL, and HbA1c 8.6%. Dyslipidemia was frequent: elevated total cholesterol (64%), triglycerides (67%), LDL (61%), and low HDL (62%). Lower vitamin D categories showed a graded association with higher glucose indices and a more atherogenic lipid profile ($p<0.001$ for all comparisons).
Conclusion: Suboptimal vitamin D status was highly prevalent in recently diagnosed T2DM and was strongly associated with worse glycemic indices and dyslipidemia. Integrating vitamin D assessment with metabolic profiling can strengthen early cardiometabolic risk stratification in diabetes care.

Keywords: 25-hydroxyvitamin D; cholecalciferol; dyslipidemia; HbA1c; type 2 diabetes mellitus.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major contributor to premature cardiovascular disease and chronic microvascular complications. Even during the early phase of diagnosis, many patients demonstrate a convergence of metabolic disturbances, including poor glycemic control, elevated triglycerides, increased low-density lipoprotein (LDL), and reduced high-density lipoprotein (HDL). Such clustering accelerates atherosclerotic risk and underscores the need for comprehensive profiling beyond plasma glucose alone. Early identification of adjunct risk markers is therefore important in comprehensive care [1].

Vitamin D is traditionally associated with mineral homeostasis, yet its endocrine actions extend to metabolic and immune pathways. Vitamin D receptors and activating enzymes are expressed in pancreatic beta cells, skeletal muscle, adipose

tissue, and immune cells. Experimental and clinical evidence suggests that vitamin D influences insulin secretion, modulates intracellular calcium flux, and affects inflammatory signaling that can impair insulin action [2]. Low 25-hydroxyvitamin D has therefore been proposed as a potentially relevant correlate of impaired glucose regulation in T2DM [1].

Prospective epidemiologic data have reported an inverse relationship between circulating 25-hydroxyvitamin D and the future risk of T2DM. The EPIC-Norfolk cohort and an updated meta-analysis demonstrated that higher baseline 25-hydroxyvitamin D concentrations were associated with a lower incidence of T2DM [5]. A meta-analysis of prospective studies similarly showed that lower vitamin D levels predict incident diabetes across populations, although residual confounding from obesity, diet, and physical activity remains an important interpretive consideration [6].

Beyond glycemia, dyslipidemia is a key driver of macrovascular risk in T2DM. Vitamin D has been proposed to influence lipid homeostasis through hepatic lipid handling, adipocyte biology, and inflammatory cross-talk. A recent narrative review summarized biologically plausible links between vitamin D status and lipid fractions, including potential effects on triglyceride-rich lipoprotein metabolism and HDL function, while also emphasizing heterogeneity across study designs and populations [7].

South Asian populations frequently report low vitamin D levels despite abundant sunlight, reflecting behavioral, dietary, and sociocultural determinants. Studies among individuals with diabetes from regional settings have documented high rates of vitamin D deficiency and have explored its association with HbA1c and metabolic outcomes [9-11]. However, clinic-based evidence in recently diagnosed adults, where metabolic abnormalities are still in an early phase and potentially modifiable, remains limited in many tertiary-care settings.

Objectives of the study were (i) to estimate the distribution of serum 25-hydroxyvitamin D status among recently diagnosed adults with T2DM and (ii) to assess the association of vitamin D categories with glycemic indices (fasting glucose, postprandial glucose, and HbA1c) and lipid parameters (total cholesterol, triglycerides, HDL, and LDL).

METHODOLOGY

Study design and setting

A cross-sectional study was conducted in the Department of General Medicine, Kamineni Academy of Medical Sciences and Research Centre, Hyderabad, India, during August 2022 to January 2024 (18 months).

Study population and eligibility

Type 2 diabetes mellitus patients of both genders aged 30-60 years attending outpatient services were screened. Eligible participants were recently diagnosed T2DM patients receiving oral hypoglycemic drugs for less than three months. Exclusion criteria were: type 1 diabetes mellitus; history of alcoholism; hypertension; thyroid and/or parathyroid disorders; cardiac disease; arthritis; hepatic and/or renal impairment; malignancy; current statin, insulin, vitamin D, oral contraceptive, or steroid therapy; and gestational diabetes mellitus.

Sample size and sampling

Sample size was estimated using a single-proportion approach with 95% confidence level (alpha = 0.05) and absolute precision of 10%. For calculation, an expected prevalence (p) of 30% for vitamin D deficiency among adults with T2DM was assumed based on published regional estimates [10,11]. Using n = Z^2(1-alpha/2) × p(1-p) / d^2, the minimum sample size was 80. After adding 10% to account for non-response, a target of 88 was obtained and rounded to 100 participants. Simple random sampling was applied among eligible attendees until the desired sample size was achieved.

Data collection procedure

After counselling, written informed consent was obtained. A predesigned semi-structured proforma that underwent internal validation was used to record demographic variables and clinical details relevant to eligibility. Participants were instructed on fasting requirements and timing of postprandial sampling. All procedures were performed under standard aseptic precautions.

Laboratory measurements

Venous blood samples were collected for estimation of serum 25-hydroxyvitamin D, fasting blood sugar (FBS), postprandial blood sugar (PPBS), HbA1c, and lipid profile (total cholesterol, triglycerides, HDL, and LDL). Serum 25-hydroxyvitamin D was analyzed by immunofluorescence. Glycosylated hemoglobin (HbA1c) was measured by fluorescence immunoassay. Lipid parameters and plasma glucose were measured using routine standardized laboratory procedures as per institutional protocols. Serum creatinine was assessed to document baseline renal function and to reduce confounding related to renal impairment.

Operational definitions

Vitamin D status was categorized as deficiency (<=20 ng/mL), insufficiency (21-29 ng/mL), and sufficiency (>=30 ng/mL) in line with Endocrine Society clinical guidance [3]. Glycemic categories for FBS, PPBS, and HbA1c were summarized using the study cut-offs specified in the proforma.

Statistical analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 22. Categorical variables were expressed as frequency and percentage. Continuous variables were summarized as mean ± standard deviation, with distribution checked using Kolmogorov-Smirnov and Shapiro-Wilk tests. Comparisons across three vitamin D categories were performed using one-way analysis of variance (ANOVA) for continuous outcomes. Statistical significance was defined as a two-tailed p value <0.05.

Ethical considerations

The study followed institutional ethical standards. Participation was voluntary, confidentiality was maintained, and test results were handled within routine clinical governance.

RESULTS

A total of 100 participants were analyzed. The mean age was 44.11 ± 6.77 years, with most subjects in the 41-50 year group (52%). Males constituted 64% of the cohort. Serum creatinine was <=1.2 mg/dL in 97% of subjects, indicating preserved renal function in the majority (Table 1).

Table 1. Baseline Demographic and Clinical Characteristics of Study Subjects (N = 100)

Variable	Category	n (%) / Mean ± SD
Age (years)	31-40	33 (33.0)
	41-50	52 (52.0)
	51-60	15 (15.0)
	Mean ± SD	44.11 ± 6.77
Gender	Male	64 (64.0)
	Female	36 (36.0)
Serum creatinine (mg/dL)	<=1.2	97 (97.0)
	>1.2	3 (3.0)
	Mean ± SD	0.98 ± 0.16

Glycemic control was suboptimal across the cohort. FBS was >124 mg/dL in 63% and PPBS was >200 mg/dL in 67% of participants. HbA1c exceeded 8.0% in 58%, with an overall mean HbA1c of 8.55 ± 1.35% (Table 2).

Table 2. Glycemic Profile of the Study Subjects (N = 100)

Parameter	Category	n (%)	Mean ± SD
FBS (mg/dL)	<=100	9 (9.0)	
	101-124	28 (28.0)	
	>124	63 (63.0)	150.86 ± 40.71
PPBS (mg/dL)	<=140	12 (12.0)	
	141-200	21 (21.0)	
	>200	67 (67.0)	211.53 ± 45.37
HbA1c (%)	<=6.5	3 (3.0)	
	6.6-8.0	39 (39.0)	
	>8.0	58 (58.0)	8.55 ± 1.35

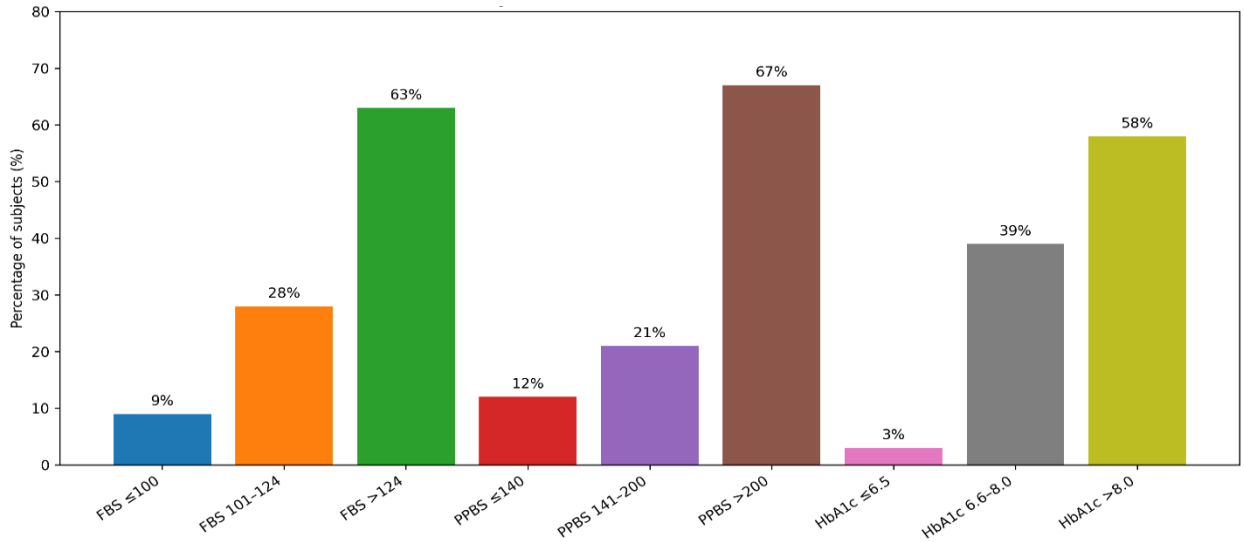

Figure:1 Glycemic Profile Distribution

Dyslipidemia was prevalent. Total cholesterol was >=200 mg/dL in 64% and triglycerides were >=150 mg/dL in 67%. Low HDL (<=40 mg/dL) was noted in 62%, and LDL was >=100 mg/dL in 61% of participants. Vitamin D deficiency (<=20 ng/mL) was present in 57%, insufficiency in 34%, and sufficiency in 9% (Table 3).

Table 3. Lipid Profile and Vitamin D Status of Study Subjects (N = 100)

Parameter	Category	n (%)	Mean ± SD
Total cholesterol (mg/dL)	<200	36 (36.0)	
	>=200	64 (64.0)	198.48 ± 18.34
Triglycerides (mg/dL)	<150	33 (33.0)	
	>=150	67 (67.0)	182.95 ± 59.44
HDL (mg/dL)	>40	38 (38.0)	
	<=40	62 (62.0)	34.61 ± 9.87
LDL (mg/dL)	<100	39 (39.0)	
	>=100	61 (61.0)	102.39 ± 20.74
Vitamin D (ng/mL)	<=20	57 (57.0)	
	21-29	34 (34.0)	
	>=30	9 (9.0)	20.15 ± 6.87

Vitamin D status showed a graded association with both glycemic and lipid parameters. Participants with vitamin D deficiency had higher mean fasting glucose, postprandial glucose, and HbA1c compared with those with insufficiency and sufficiency. Lipid parameters were also more atherogenic in vitamin D deficient subjects, with higher total cholesterol, triglycerides, and LDL and lower HDL. Differences across vitamin D categories were statistically significant for all reported outcomes (one-way ANOVA; $p<0.001$) (Table 4).

Table 4. Association of Serum Vitamin D Levels with Glycemic and Lipid Parameters (N = 100)

Parameter	Vitamin D <=20 (Mean ± SD)	Vitamin D 21-29 (Mean ± SD)	Vitamin D >=30 (Mean ± SD)	p-value
FBS (mg/dL)	179.88 ± 28.03	116.71 ± 12.73	96.11 ± 3.41	<0.001*
PPBS (mg/dL)	245.09 ± 17.46	176.68 ± 25.97	130.67 ± 4.53	<0.001*
HbA1c (%)	9.54 ± 0.85	7.40 ± 0.46	6.67 ± 0.20	<0.001*
Total cholesterol (mg/dL)	210.44 ± 5.56	187.74 ± 15.60	163.33 ± 6.46	<0.001*
Triglycerides (mg/dL)	226.58 ± 34.08	134.44 ± 23.48	89.89 ± 2.89	<0.001*
HDL (mg/dL)	27.70 ± 7.17	43.00 ± 3.16	46.67 ± 1.00	<0.001*
LDL (mg/dL)	117.58 ± 9.65	86.68 ± 10.34	65.56 ± 3.17	<0.001*

DISCUSSION

This study documents a high prevalence of suboptimal vitamin D status in recently diagnosed adults with T2DM and demonstrates strong inverse associations between serum 25-hydroxyvitamin D and both glycemic indices and lipid parameters. The findings indicate that vitamin D deficiency coexists with poor glycemic control and dyslipidemia even early in the disease course.

Using Endocrine Society cut-offs [3], vitamin D deficiency and insufficiency together accounted for 91% of the cohort. Comparable burdens of vitamin D deficiency have been reported in regional diabetes clinic populations, including South Asian and Middle Eastern settings [9-11]. This pattern likely reflects a combination of reduced sun exposure, dietary insufficiency, adiposity-related sequestration, and limited outdoor physical activity, factors that also overlap with T2DM risk behaviors [1].

Vitamin D deficient participants showed markedly higher fasting glucose, postprandial glucose, and HbA1c. Similar inverse relationships between 25-hydroxyvitamin D and HbA1c or fasting glucose have been described in adults with diabetes [4,11]. Mechanistically, vitamin D signaling can influence pancreatic beta-cell function and insulin sensitivity through calcium-dependent pathways, regulation of inflammatory mediators, and effects on adipokine profiles [2]. While causality cannot be inferred from cross-sectional data, the strength and gradient of associations support biological plausibility.

A consistent pattern was also observed for lipid parameters: lower vitamin D categories were linked to higher total cholesterol, triglycerides, and LDL and to lower HDL. Clinical studies have reported comparable associations between vitamin D status, HbA1c, and lipid fractions among T2DM patients [8]. A recent narrative review described plausible biologic pathways connecting vitamin D to lipid metabolism, including effects on hepatic lipogenesis, insulin-mediated lipid handling, and inflammation-driven alterations in lipoprotein metabolism [7]. Given the high baseline prevalence of dyslipidemia in this cohort, vitamin D deficiency could represent an additional marker of risk clustering.

Randomized trial evidence on vitamin D supplementation in T2DM has been variable. Meta-analyses of trials have suggested modest improvements in glycemic indices in selected contexts, particularly when baseline deficiency is present

and adequate repletion is achieved [12,13]. Updated pooled analyses continue to indicate small reductions in fasting glucose and HbA1c in some trial populations, although heterogeneity in dose, duration, baseline vitamin D status, and concurrent therapies remains substantial [14]. These data reinforce that observational associations do not directly translate into treatment effects without careful consideration of baseline deficiency and intervention intensity.

From a practice perspective, outpatient diabetes clinics provide an opportunity to address concurrent nutritional deficiencies and cardiometabolic risk factors. Integrating vitamin D screening with routine metabolic profiling can strengthen risk stratification, support tailored counselling regarding diet and safe sunlight exposure, and guide evidence-based supplementation when deficiency is confirmed.

Limitations

The cross-sectional design limits causal inference. The study was single-center and restricted to recently diagnosed patients on oral therapy, which reduces external validity. Key confounders such as body mass index, diet, sun-exposure behavior, physical activity, and seasonal variation were not quantified for adjustment. Single-time biochemical measurements cannot capture intra-individual variability. Medication adherence and duration of diabetes prior to diagnosis were not quantified.

CONCLUSION

In this outpatient cohort of recently diagnosed adults with T2DM, vitamin D deficiency and insufficiency were highly prevalent and showed strong graded associations with poorer glycemic indices and dyslipidemia. Vitamin D deficient participants had higher fasting and postprandial glucose and higher HbA1c, alongside higher total cholesterol, triglycerides, and LDL and lower HDL. These results indicate that suboptimal vitamin D status accompanies early metabolic dysregulation and cardiovascular risk clustering. Routine assessment of serum 25-hydroxyvitamin D, integrated with standard metabolic profiling, can strengthen early risk stratification and support targeted nutritional correction as part of comprehensive diabetes care. Longitudinal studies in similar settings can further clarify temporal relationships in routine clinical practice today**.**

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